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Conference

A Cultural History of Heredity III: 19th and Early 20th Centuries

Table of Contents

Introduction

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The contributions to this volume were prepared for the workshop "A Cultural History of heredity III: Nineteenth and Early Twentieth Centuries" that took place at the Max-Planck-Institute for the History of Science January 13-16, 2005. The workshop was part of a long-term interdisciplinary project dedicated to the cultural history of heredity. Concentrating in turn on a succession of time periods in chronological order, this series attempts to uncover and relate to each other the agricultural, technical, juridical, medical, and scientific practices in which knowledge of inheritance was materially anchored and in which it gradually revealed its effects.[1] The two previous workshops took place in May 2001 and January 2003, each devoted to what we identified as 'historical epochs' in the history of hereditary thought.[2] What follows is a summary of the results reached so far in our project, of the questions we wanted to address with the third workshop, and the answers suggested to these questions by the contributions collected in this preprint.[3]

In his book "The Logic of Life", François Jacob already pointed out that the concept of reproduction, and by extension, that of heredity were virtually absent from speculations about generation until the eighteenth century. This we found largely corroborated. It may be worthwhile to stress that heredity as a biological concept originated as a metaphor: prior to the nineteenth century it was used as a synonym of "inheritance" in legal contexts only, a sense which it since then has lost. It was only in the early nineteenth century, in medical contexts, that metaphors of heredity began to gain currency. To be sure: Phenomena that nowadays would count as hereditary had by no means gone unnoticed before. It seems, however, to be a simple matter of historical fact that these phenomena were not addressed in terms of inheritance.

The conceptual reason for this was the lack of some fundamental distinctions in pre-modern theories of generation. Before the end of the eighteenth century hereditary transmission was not a domain regarded as separate from the contingencies of conception, pregnancy, embryonic development, parturition, and even lactation. Similarity between progenitors and their descendants was thought to come about as a result of the similarity in the constellation of causal factors involved in each act of generation. Parental organisms were thought to actually make their offspring, without the intervention of a specific hereditary substance transmitted from generation to generation.

So if speculations into generation did not provide the context in which biological heredity originated, what did? When Buffon, Maupertuis, and Kant were addressing heredity in the second half of the eighteenth century, they were referring, alongside the discourses of natural history and breeding, to a discourse of highly idiosyncratic origin, namely the Latin-American system of racial

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1 See http://www.mpiwg-berlin.mpg.de/HEREDITY/ for further information on the project.

2 Contributions to the first two workshops have been documented in the Max Planck Institute's preprint series (no. 222 and 247).

3 A more extensive summary of the resultsfrom the project so far, including a bibliography, has been published as in the Max Planck Institute's preprint series no. 276.

classification, known as *las castas*, and usually presented in paintings arranged in a tabular form. This scheme was primarily based on a classification according to skin color, to a lesser degree also on hair form and eye color. Children resulting from mixed marriages were positioned in this scheme in analogy to the simple mechanism of color mixing, implying "blending" as the causal relation connecting traits of parents with traits of their offspring.

The *castas* classification originated from attempts in early colonial Latin America to find a measure by which legal and social status could be allocated, while colonial society experienced constant flux due to intermarriages and migration. This indicates an important general result of our project: the problem that biological heredity came to address was *not* the constancy of species; it were the patterns and processes that structure communal life at a sub-specific level. This shift of attention was associated with a mobilization of early modern life within various largely independent cultural domains, all of which also saw a growing concern with hereditary phenomena towards the end of the eighteenth century: Breeding new varieties for specific marketable characteristics, the exchange of specimens among botanical and zoological gardens, experiments in fertilization and hybridization of plants and animals, the dislocation of Europeans and Africans that accompanied colonialism, and the appearance of new social strata with their particular pathologies in the context of industrialization and urbanization, all of these processes interlocked in relaxing and severing cultural and natural ties to provide the material substrate for the emerging concept of heredity.

The various domains in which the knowledge regime of heredity took shape during the eighteenth century did not cohere, however, after the model of an overall "influence" that a fundamental idea of heredity gained over them. Rather, conjunctions between them came about by a kind of domino effect that mobilization in one field had on another. The growth of a class that depended on mobile property evoked a culture of leisure collecting and breeding. The import of plants for collection purposes of natural history in turn inspired attempts at their acclimatization for economic purposes. Several, highly specific, and largely independent cultural sub-fields were thus subsequently conjoined to form a field of phenomena which only eventually, in the mid-nineteenth century, came to be addressed by the concept of biological heredity.

Although this makes it difficult, even impossible, to draw a general picture of the historical development that led to the formulation of full-fledged theories of heredity in the mid-nineteenth century, it is possible, in hindsight, to characterize the result of that development. As a point of departure, let us take Darwin's theory of pangenesis. Two aspects of that theory, if compared with premodern theories of generation, are remarkable. First of all, Darwin endorsed a view of heredity that abstracted, to some extent at least, from the personal relation between parents and their offspring. While conceding an inheritance of acquired properties, Darwin believed that the true carriers of the properties to be inherited are not the parents themselves, but submicroscopic entities — "invisible characters" as he called them — which circulate, from generation to generation, among individuals within one and the same species. Secondly, Darwin's theory of heredity shows a peculiar inversion in comparison with early modern theories of generation: while the latter emphasize the vertical dimension of lineal descent — where ancestral organisms bring forth their offspring — Darwin invoked an image where the horizontal dimension dominates, the dimension of a common reservoir of dispositions, passed down from the sum total of ancestors,

redistributed in each generation among individuals, and competing now, in the present, for their realization. We take these two aspects, which accord well with the long term development of Western European kinship systems as social anthropologists have reconstructed them, to be the fundamental hallmarks of modern hereditary thought.

The complex constellation that Darwin evisaged — thousands of generations represented virtually in the microscopic space of each fertilized egg — points to the complexity of the biological concept of heredity. In this respect, one can speak of an "epistemic space" of heredity that came into being in mid-nineteenth century. In contrast to other subjects of biological research, which are determined within individual experimental settings, heredity depended on a vast, spatial configuration of distributed technologies and institutions connected by a system of exchange: botanical gardens, hospitals, chemical and physiological laboratories, genealogical and statistical archives.

It is this "space" and its development during the late nineteenth century that we wanted to explore in the third workshop in our project. It was planned to occupy itself — chronologically speaking — with the latter part of the nineteenth century and the first years of the twentieth. Roughly, this period can be regarded as one in which various attempts were made to thoroughly theorize heredity on the basis of observation, experiment, and statistical analysis, but in different directions. For orientation, we demarcated the period by two publications: Francis Galton's *Hereditary Talent and Character* of 1865 and Wilhelm Johannsen's *Über Erblichkeit in Populationen und in reinen Linien* of 1903. Obviously, it could be asked why we did not take Mendel's 1864 paper and the year 1900, the *annus mirabilis* of the birth — or re-birth — of genetics as our points of orientation. We did *not* do that because we did not intend to focus on the triumphal advent of classical genetics, breeding practice included, in this workshop. Rather, we wanted to locate this event, this advent — the "(re-)discovery" of Mendel — in the much broader spectrum of theories and practices of inheritance that had already been consolidated in such widely different contexts as evolutionary and developmental biology, cytology, bacteriology and epidemiology, eugenics and anthropology by the end of the nineteenth century. What we did do, however, is to ask Rafael Falk to talk to us on Mendel's impact as a sort of introductory lecture, an *exergon* so to speak for — and before — this conference, and then turn to the other topics. We plan to come back, as already said, to Mendel, breeding, and classical genetics in a future workshop.

Following an earlier suggestion from Jean Gayon, we can draw the following scheme for the development of hereditary knowledge in the second half of the nineteenth century:[4] With the rise of heredity as a central biological problem by the middle of the nineteenth century, the question of its material basis and of its mechanism had taken shape. In the second half of the nineteenth century, two major frames were proposed to deal with this question. The first one saw heredity as a *force*, whose strength could be accumulated over the generations, and which, as a measurable magnitude, could be subjected to statistical analysis. This concept was particularly widespread among nineteenth-century breeders, and it influenced Francis Galton and the so-called "biometrical school." The second saw heredity as residing in *matter* that was transmitted over the

[4] For references see our entry on "Gene" in the *Stanford Encyclopaedia of Philosophy* at http://plato.stanford.edu/entries/gene/.

generations. Two major trends can be differentiated in this latter case. One of them conceived of hereditary matter as particulate and amenable to breeding analysis. Charles Darwin called the presumed hereditary particles "gemmules"; Hugo de Vries, "pangenes"; Gregor Mendel, "elements". None of these authors, however, thought of associating these particles with a particular hereditary substance. They were seen as microscopic entities, which, if accumulated en masse, would make the particular traits visible for which they stood. A second category of biologists in the second half of the nineteenth century, to whom Carl Naegeli and August Weismann belonged, distinguished a specific hereditary substance, the "idioplasm", or "germ plasm", which they assumed to be responsible for generational hereditary continuity, on the one hand, and the body substance, the "trophoplasm" or "soma", on the other.

While considerable work has already been done in terms of a history of these ideas, comparatively little effort has been made to explore the links of the above mentioned late nineteenth century developments with the notion of heredity itself. Most studies, which paid attention to the contexts of, e.g., anthropological or medical work have tended to take inheritance as the necessary, yet in itself unquestioned prerequisite, upon which their actors relied to promote their hierarchies of human inequality. With the variety of hereditary theories we encounter around 1900 it seems, however, that the very notion of inheritance was rather deeply troubled at this time.

The workshop was planned to explore the sources for these troubles in a (certainly incomplete) number of fields in which hereditary considerations played a role: anthropology, evolutionary and developmental biology, cytology including microbiology, plant and animal breeding, epidemiology including bacteriology, eugenics, and juridical and sociological concepts of hereditary transmission. Not all of these fields could be covered with equal density, others came into sharper focus during the preparation of the conference, and in putting together the proposals, we had to make our choices. So we ended up with six sessions. The first set the stage of theorizing heredity in the later nineteenth century; the second looked at heredity in the context of evolution and development; the third occupied itself with the physiology of heredity in a broad sense; the fourth brought together aspects of social, psychological, and cultural heritage; the fifth was devoted to heredity and genealogy; and the sixth, finally, thematized heredity and anthropology.

One of the main outcomes of the workshop was that the two dichotomies developed by historians so far with respect to late nineteenth-century theories of heredity, namely soft vs. hard (Ernst Mayr) and blending vs. non-blending inheritance (Robert C. Olby) do not seem to work well to fully capture the variety of theoretical approaches in that time period. There are two reasons for this:

(1) It became apparent that up until the very end of the nineteenth century speculations into heredity either conflated these oppositions, or remained rather indifferent with respect to them. Within the medical community, for example, the belief was widespread that diseases leave their "stamp" upon offspring or that substances like alcohol "poison" the germinal substances. This position reflects a conception of heredity both "soft" and "hard" at the same time — soft, in as much as environmental factors induce a change in the hereditary substance; hard, in as much as this change, once it has occurred, is irrevocably passed on in the germinal line (in obvious analogy to original sin). Likewise, the "blending" of parental characters in the offspring was often seen as

compatible with a view of the hereditary substance as being composed of "non-blending", particulate elements, as notably in Darwin's theory of pangenesis and Galton's theory of the stirp. Both oppositions, "soft" vs. "hard" and "blending" vs. "non-blending" seem to have become serious issues of dispute and dissent only after the onset of Mendelism in 1900.

(2) The oppositions of "soft" vs "hard" and "blending" vs "non-blending" heredity cover up or cut across more fundamental, and more hotly debated, oppositions with regard to the material make-up and causal agency of hereditary material. Was heredity to be conceived as a natural force or as an organic structure? What was the source of the hereditary material, the ancestral organism itself or something passed on independently? Was there a particular substance that formed the germ plasm, and where was it possibly located? And could it be related to particular cellular structures? What, if any, were the elements of the hereditary material, and how did these elements relate to each other? Did they fuse, or just mix? And in what manner did they determine the future organism, directly throughout the individual life-span, or only by determining the first steps of development? Finally, what, if any, where the distinct roles the two sexes played in inheritance?

The nineteenth century did not come up with concluding answers to these questions, as is well known, and the contributions to the workshop showed the considerable breadth of positions with respect to inheritance that persisted well into the first decade of the twentieth century. Yet the very nature of these questions points to a decisive trend in the period studied by the workshop. Inheritance was increasingly seen not as a relation between individual organisms – ancestors and descendents – but as a relation of populations to a shared, germinal substrate. Various contributions pointed to two important, but until now under-researched, sources of models that shaped representations of this substrate: genealogy and epidemiology. Genealogy developed in the late nineteenth century into a tool for analyzing populations rather than individual ancestry, and brought to the fore both the openness of family relations and the quasi-mathematical closure of genetic relationships. In epidemiology, heredity, infection, and vaccination intersected to produce what Jean-Paul Gaudiellière and Ilana Löwy have called the "impossible separation" of horizontal and vertical dimensions in the transmission of diseases.

The reason for the "devaluation of ancestry" in favor of a view that sees (cultural as well as biological) inheritance as a common stock of dispositions seems to lie in the association of heredity with the future rather than the past, with projection rather than with legitimization, that occurred in the context of the all-pervading late-nineteenth century theme of progress. Even where the past entered hereditary discourse, it did so either as a threat to the present, in form of "atavisms", "throw-backs" and "degeneration", or as a "heritage", "stock", or "capital" to be appropriated anew by each generation. Breeding plants and animals provides the obvious model here, and Mendelism with its close connection to the breeding industry took the decisive step to attack hereditary phenomena by "deducing forward", as Raphael Falk put it during the workshop. Studying the development of Mendelism against the background explored by this workshop will be the agenda for the next workshop, which is planned to take place at the University of Exeter in autumn 2006.

Mendel's impact

Raphael Falk

Abstract

Mendel introduced a reductionist methodology *to the study of inheritance. By 1900, reduction of biology to the laws of chemistry and physics was not self evident: Although developmental bottom-up preformation was largely inadequate, organismic epigenetic hypotheses often fall prey to metaphysical assumptions. The achievements of Mendel's reductive research methodology in genetics were increasingly extended to* reduction at the conceptual level*, not only with respect to transmission genetics but similarly in dealing with problems of development and evolution. Genetics became extremely genocentric in its explanatory arsenal, and accordingly it was divorced from the top-down, life-as-organized-systems' notion of embryology. Similarly, evolution was conceptually reduced to population genetics in terms of gene-frequencies. It was, however, the increasing attempts to ground the bottom-up approach at the molecular level that eventually pushed conceptual reductionism to its crisis. Modern developments of molecular and computational methods finally forced, or allowed, genetics and development to apply* reductionist methods to top-down systems' analysis*, thus to close the cycle of the adoption of reductionist methodologies to top-down conceptions.*

Gregor Johann Mendel's personality unfolded and matured in a community of scientifically minded breeders. We are naturally most interested in the scientific impact of Mendel. However, Mendel apparently had also an impact on breeders, as was unexpectedly brought home to me through a story that a bio-ethicist colleague, Dan Wikler, told me a couple of years ago, about his Himalayan holiday in 1993.

> On his tour of northern India Dan met a local scholar, Tshering Dorje, a specialist in pre-Buddhist religions of Tibet, who functioned also as his group's guide. The man came from a valley, named Lahoul, located in the Western Himalaya, north of a pass that leads to Kulu, Manali, and further down to Punjab (fig. 1). The inhabitants of that valley were quite well off ever since a road that connected them with the rest of the world had been built. This allowed them to export the crop of peas that they had specialized in growing and that has been known for its quality. According to the guide, a Moravian missionary, called Francke, introduced pea growing into the area at the end of the nineteenth century. Francke related that he learned the secret of growing peas from his cell mate in the monastery, whose name was Mendel, "who was quite good at growing peas." Dan suspected that the guide did not know much of Mendel and his impact on science.

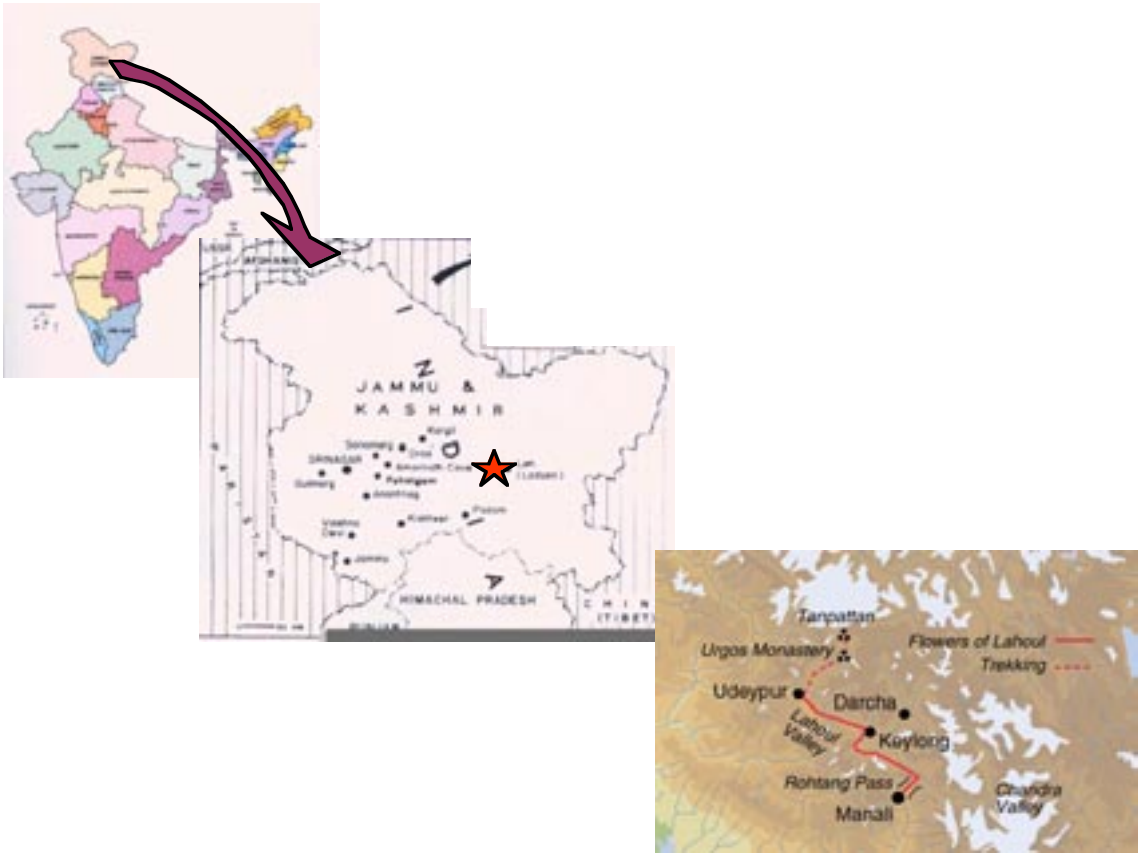

Fig. 1.: The Lahoul valley in the Himalaya.

Now, this story is too good *not* to be true. I tried to follow the story somewhat further: A Moravian missionary by the name August Herman Francke wrote a history of Ladakh (Francke, 1977). He was however too young to be Mendel's cell mate: He was twelve when Mendel died. In his book, *A History of Ladakh* there is no hint to that story, but Moravian missionaries had been active in the area already before Francke: An earlier history of the country was written by another Moravian missionary by the name Karl Marx (of all possible names!). I finally got hold of an e-mail address of the Moravian mission in Ladakh, and got a prompt response from the reverend Elijah Gregan, a pastor of the Moravian church, who turned out to be a cytogeneticist by training. He wrote: "I have never heard of the story that you wrote about Mendel … However, Moravians are well known in the area for having introduced vegetables in the Lahoul and Ladakh valleys." I hope you will agree with me that this adds a piquant and unexpected flavor to the story of the origins of genetics, although unfortunately at the moment it seems not to be true.

Genetics: Coming of age of heredity

Mendel's impact on science, I wish to propose, was by introducing a reductionist agenda to the research of heredity. This notion of Mendel I believe was based primarily on a deep religious conception of the world as governed by laws of nature formulated in numerical principles (Falk, 2001). Mendel, however, did not introduce a particulate material notion into the study of heredity – he talked of *Faktoren***.** As pointed out by Gayon (2000, 71-73), it was Darwin who rejected the prevalent notion of *hereditary force* and promoted a notion of material particles, *gemmules*, and de Vries who turned these particles into *pangenes,* which in 1900 he associated with Mendel's factors. De Vries (1848-1935) was primarily interested in backing up his own hypothesis of *intracellular* development and evolution, rather than in establishing pangenes as foundations of a theory of particulate heredity along Mendel's insight. Notwithstanding, his role in promoting Mendel's insight was crucial, though indirect. As for the other two traditional "rediscoverers" of Mendel's work: The younger Carl Correns (1864-1933), who was less ambitious in formulating universal laws of nature, it must be admitted, did not grasp in due time the potential of Mendel's work as the dawn of a new science of heredity. After reading de Vries's paper, he related that he had obtained similar results, and indeed, was aware that he "had found *something new*," but added "that Mendel's Law of segregation cannot be applied universally," and accordingly, he "did not consider it necessary to establish" his priority (Correns 1900, in Stern and Sherwood, 1966, 120). With regard to the third "rediscoverer" of Mendelian segregation of characters, Erich von Tschermak-Seysenegg (1836-1927), it was noted by Curt Stern that "his publications in 1900 show him not to have been a discoverer of Mendelism but only an experimenter whose understanding … had 'fallen short of the essential discovery'" (Stern and Sherwood, 1966, xi-xii)**.** Or, as pointed out by Bob Olby, his paper "reveals very clearly how Mendelian results could be treated in terms of the orthodox conceptions of heredity in the nineteenth century, *even after reading Mendel's paper*" (Olby, 1985).

Although he never claimed to have had discovered either the Mendelian ratios or their explanation, it was William Bateson (1861-1926, fig. 2) who conceived of Mendel's experimental work as a founding contribution to the new research discipline of heredity and variation, which in 1906 he called *genetics*. Contrary to de Vries, who superimposed his notions of pangenes' function on the Mendelian factors in the service of his theory on the origin of species by mutations (*Die Entstehung der Arten durch Mutation*, de Vries, 1902-03, II, xii), Bateson, like Correns, was interested in the experimental discovery of the nature of these factors of inheritance. Notably, both adhered to material preformationist notions that did not discriminate conceptually between the factors for traits and the traits proper: This was most obvious from their respective notions of dominance-recessivity relationships: Correns interpreted dominance in terms of the *physiological* action of the factors, whereas Bateson attributed it to the *material* presence of the factors of inheritance, as formulated in his Presence-Absence Hypothesis (see Falk, 2001; Rheinberger and Müller-Wille, 2004).

When Wilhelm Johannsen (1857-1927) formally introduced in 1909 the distinction between the "unit character" and the *gene* he was most careful not to attribute any explicit material meaning to his gene-concept (Johannsen, 1909). Johannsen's gene was merely an intervening variable. But with the establishment of the Chromosomal Theory of Inheritance by Morgan

(1866-1945) and his students, the Mendelian units of inheritance became hypothetical constructs, and for at least one of his students, Hermann J. Muller (1890-1967), the genes were discrete material entities, the atoms of inheritance, the physical-chemical properties of which he set out to study (Falk, 1986).

Fig. 2.: William Bateson, the founder of the science of Genetics (on the left) and Wilhelm Johannsen, who segregated the phenotypic traits from the genotypic potential (to Bateson's right).

Thus, I repeat, Mendel's scientific impact was by introducing a *reductionist research agenda*, and genetics became the *prima facie* reductionist life-science. Now, from the privileged perspective of

somewhat more than one century later, I wish to examine the achievements and the shortcomings of the Mendelian reductionism and its conceptual extensions on the science of inheritance.

This, however, must be done in the context of the aggressive developments in the life sciences at the turn of the twentieth century, especially in notions of development and evolution. Although by1900 reduction of biology to the laws of physics and chemistry had already been accepted among physiologists (even though the breakthrough of biochemistry was still decades ahead), this notion still struggled for its application in studies of development and evolution. Mendel's reductionist research methodology thus was to confer on the study of inheritance a special status among the life-sciences, namely that of providing for the extension of reductionism to embryological and evolutionary studies.

I wish to claim that genetics as the science of heredity was never divorced from development and evolution. Rather, we have been witnessing in the nineteenth and the twentieth century a full cycle, from a holist view of heredity as an aspect of development and evolution that necessarily fell back on irrational assumptions, to the higher level of a rational though reductionist view of heredity, back to a holist perspective of heredity at the level of systems' analysis of development and evolution. In the footsteps of Mendel, who heuristically sublimed the details of the characters that he judiciously *selected* for his experiments on transmission between generations, geneticists viewed the specific cases of development and evolution which they happened to study as samples of generalized – methodologically and eventually conceptually – reducible units. Starting with Bateson's presence- absence hypothesis of dominance (Bateson, 1905 (1928)), through Muller's interpretation of dominance as precision of genetic functional adaptation (Muller, 1950), to Beadle and Ephrussi's study of specific discrete gene-effects that culminated in the one-gene – one-enzyme thesis (Beadle & Ephrussi, 1936; Beadle & Tatum, 1941), and to the formulation of the New-Synthesis of evolution in term of populations' gene frequencies (see Provine, 1971), geneticists inferred rules of development and evolution in which the gene provided "a kind of inertia principle" against which effects could be measured (Gayon quoted in Rheinberger & Müller-Wille, 2004).

Genetics: Reducing inheritance with development

Reduction involves the explanation of laws or phenomena in one realm by those of another. Reductionism is the thesis that reductions between two realms are always (or, at least, are always likely to be) successful as explanations. I wish to differentiate between methodological reductionism and conceptual reductionism.

> **Methodological reductionism** is the belief that empirically following single (experimental or observational) variables (other variables being kept constant or randomized) is the effective design to bridge realms.

> **Conceptual reductionism** assumes that phenomena may be reduced to a component or components at a more basic realm, that individually or interactively bridge the phenomenon at the higher realm.

The distinction is one between explanation and resolution. Methodological reductionism may be identified as an epistemological statement, whereas conceptual reductionism is essentially an ontological statement (Sarkar, 1998, 17ff).

Methodological reduction would best correspond to Sarkar's (1998, 43-45) *abstract hierarchical reduction,* which assumes that the rules of one realm are more fundamental than those governing another realm. Conceptual reduction would correspond to his *strong* or *approximate strong reduction*, in which the hierarchy referred to is also a hierarchy in physical space.

Modern science has been *methodologically* reductionist, ever since Galileo introduced the study of one variable at a time, by keeping all other variables as much as possible constant (or, at least, let the other variables vary randomly with respect to the variable studied). It became also increasingly Cartesian, or *conceptually* reductionist.

The achievements of reductionist methods and conceptions in physics, and eventually also in chemistry encouraged researchers to extend reductionism to the life-sciences. But the life sciences of the eighteenth century were demarcated at the outset from physics and chemistry as immanently non-reductionist. In spite of nineteenth century biologists' inroads in extending reductionism to the life sciences (Lenoir, 1982), life remained immune to complete conceptual reduction. Something else was needed, whether it was Caspar Friedrich Wolff's *vis essentialis,* Johann Fiedrich Blumenbach's *Bildungstrieb*, Carl Nägeli's *Vervollkommnungskraft,* Theodor Eimer's *Orthogenesis,* Hans Driesch's *Entelechy*, or Niels Bohr's search for special laws of physics expressed only in living systems, or the modern theories of complexity. The wish to reduce living being to systems that may be completely explained bottom-up, countered the recognition that living being *are* the fundamental entities that should be explained top-down in order to unravel the properties of their components.

The three major conceptual revolutions in the life sciences of the nineteenth century, the cellular theory (1838-1839), the theory of evolution by natural selection (1859), and the theory of particulate inheritance (1865), all reflect the craving for reductionist explanations for living systems. Of the three, Darwin's hypothesis of *The Origin of Species by Means of Natural Selection* was actually a top-down theory. But Darwin's juxtaposition of the organism *versus* its environment, independently of what the organism "wants" or needs – contrary to conceptions up to and including the Lamarckian hypothesis of the organism *in* its environment – inserted a conspicuous reductionist element into his hypothesis. Although Darwin repeatedly tried to back out of this notion of the organism-environment segregation, the reductionist aspect of Darwinism, of 'nature *vs.* nurture,' as phrased by Francis Galton in 1865, was firmly established once formulated by August Weismann (1893) as the germ-plasm theory of heredity.

Of the other two conceptions, Schleiden´s and Schwann's theories of the cell as a universal reductionist idea gained its success with Virchow's 1855 doctrine of *omnis cellula e cellula*, whereas Mendel's reductionist theory gained recognition at the turn of the twentieth century.

But it was in the area of development *and* inheritance in which the reductionist and holist conceptions confronted each other most directly. Wilhelm His and Wilhelm Roux introduced the mechanics of development (*Entwicklungsmechanik*) largely in the hope to repeat the achievements of the physiologists' reductionist methods:

> Developmental mechanics is the doctrine of the causes of organic forms. Form represents the most essential attribute of the organism. … In accordance with Spinoza's and Kant's definition of mechanism, every phenomenon underlying causality is designated as a *mechanical phenomenon;* hence the science of the same may be called mechanics. Since … physics and chemistry reduce all phenomena, … to movement of parts, … the words 'developmental mechanics' agree with the more recent concepts of physics and chemistry, and may be taken to designate the doctrine of all formative phenomena. … In so far, however, as forces or energies are only known by their *effects*, … the problem may be defined as the *ascertainment of the formative modi operandi.* (Roux, 1894, 107-108)

Yet, as Roux himself was conscious of, even if a bottom-up mechanistic methodology was imperative, the conceptual reduction of development was more problematic and controversial:

> … developmental mechanics must from the start be guided by the conviction that *organic structure is mainly due to the operation of components which at present are so complicated as to exceed the limits of our observation.* … Although according to our immediate conception of the matter, even these components depend in the last instance on inorganic *modi operandi*, nevertheless *the complexity of the composition lends them attributes which often differ so widely from those of inorganic modi operandi* that they are not only very *dissimilar* but even *appear to contradict* in part functions of these same inorganic *modi operandi.* (Roux, 1894, 111)

For embryologists the issue of the mechanism of development became part of the discussion of preformationism versus epigenesis, i.e., of the extent of the immanent inheritance of developmental processes. Although a long way from the old notion of Hartsoeker's (1694) *homunculus*, preformation, or the bottom-up view of development as the sum total of the physical and chemical properties of its building blocks was the reductionist notion that prevailed. Those who adhered to the opposite conception of epigenesis, which conceived of the organism as the frame of reference that assimilated the environmental, including the inherited cues in development, could not construe it in terms of additively reducible physico-chemical terms (as preformationist *hoped* was feasible). Thus, epigenesis repeatedly implicated, sometimes inadvertently, scientists with *sensu stricto* meta-physical notions of development.

The predicament of the analytical biologist, confronted with his experimental results obtained in the best tradition of the reductionist methodology, was presented most eloquently by Edmund B. Wilson in his 1893 lecture at the Marine Biological Laboratory in Woods Hall:

> Many leading biological thinkers now find themselves compelled to accept a view that has somewhat in common with the theory of prae-formation … Every one of [the] hereditary characters is … represented by definite structural units in the ideoplasm of the germ cell, which is therefore conceived as a kind of microcosm, … [T]he so-called mosaic theory of Roux and Weismann … is essentially a whole arising from a number of independent self-determining parts, ...
>
> [Yet, a] more decisive result was reached in 1891 by Driesch, … [which] the writer repeated … and found that … [t]he isolated blastomere behaves from the beginning, like an entire ovum of one-half or one-fourth the normal size… .

> These facts are obviously a serious blow to the mosaic theory, and the efforts of Roux and Weismann to sustain their hypothesis in the face of such evidence only serve to emphasize the weakness of their case. (Wilson, 1893, 69-72)

The problem was a conceptual one, rather than one of methodological particularization. It is here that Mendel's accomplishment, of judiciously selecting appropriate markers for his experiments in the 1860s, applying the reductionist *methodology*, without making an explicit statement on the *conceptual* problem, is fully paying off. This was noted already in 1902 by Udny Yule:

> The experimental plants must, Mendel states … "possess constant differentiating characters." … The races for crossing were thus chosen with the greatest care and patience so as to be absolutely distinct; an *A* individual mated with an *A* never producing *a*'s, nor vice-versa, for, as I understand, the whole period of ten years (two years of preliminary trials and eight years of experiment). (Yule, 1902, 222-223)

De Vries, however, from his preformationist perspective, claimed that determinants were universals, for all characteristics, though unlike the Roux-Weismann hypothesis, he did not believe that these determinants were unequally distributed to the different blastomers: According to him differentiation was achieved by activation-inactivation of latent particles, pangenes. This, by the way, was why de Vries *needed* the notion of dominance as if it were one of Mendel's laws, to replace his activation-latency terminology (see Stamhuis, 2003; Stamhuis, Meijer, & Zevenhuizen, 1999).

Once Bateson adopted the Mendelian notion of units of inheritance, a preformationist notion of units of development was for him the consequence, and the Mendelian factors were unit-characters in potency. To the extent that the morphologist's or physiologist's unit character did not agree with those of Mendelian segregation units, the former were not really "unit characters," but rather complex characters. Mendelian segregation was for Bateson a device to assess spurious morphological unit characteristics, such as in the case of chickens' comb morphology: The "walnut" comb was a compound of two unit-characters, the "pea" comb and the "rose" comb (Schwartz, 1998, 2002).[1] Indeed, to a large extent, the dispute between the Mendelians led by Bateson and the Biometricians led by Pearson may be traced back to the Mendelian factors being conceived as preformed unit-characters. Thus, Darbishire's hybridization experiments with mice,

[1] Bateson's ignoring any chromosomal association with Mendelian factors has recently been explained by Patrick Bateson as being due to Bateson's irritation about "the glibness of the explanation offered for the role of chromosomes. The chromosome was portrayed not simply as a structural site of the gene but almost as the gene itself. Worse, the chromosome was treated as the pre-formed version of the character influenced by the gene on that chromosome" (P. Bateson, 2002, 54). Indeed, reviewing Morgan's *Mechanism* William Bateson wrote that "it is inconceivable that particles of chromatin or any other substance, however complex, can possess those powers which must be assigned to our factors. … The supposition that particles of chromatin, indistinguishable from each other and indeed almost homogenous under any known test, can by their material nature confer all the properties of life surpasses the range of even the most convinced materialism" (Bateson, 1916). Bateson may have shared Samuel Butler's rage against machine-aged materialism, crass professionalism, and unabashed utilitarianism (Harman, 2004, 29), but he certainly was looking for a more sophisticated material preformationism. I tend to accept Punnett's response to the question of how he and Bateson missed the tie-up of linkage phenomena with the chromosomes: "The answer is Boveri. We were deeply impressed by his paper 'On the individuality of the chromosomes' and felt that any tampering with them by way of breakage and recombination was forbidden" (Punnett, 1950, 10).

in which there were unexpected variations in fur color, whereas a preformationist principle of dominance predicted uniformity of color for unit characters in the F_1, were taken by Biometricians as evidence against Mendelian determinism, and Bateson had to come forward with various excuses for the variability of fur color of the alleged dominants (Ankeny, 2000, 328). Similarly, William Castle's (1867-1962) explanation of the variation in color-patterns of hybrid rats by a model of allele-contamination in heterozygotes (Castle, 1906) was based on a preformationist notion that did not distinguish between traits and the factors for traits.

> My own experimental studies of heredity, begun in 1902, early led me to observe characters which were unmistakably *changed* by crosses and so I have for many years advocated the view that the gametes are not pure in the sense expressed by Bateson. (Castle, 1919, 126)

Not so for Johannsen, whose notion was one of an essentialist typologist (Roll-Hansen, 1978). His was a top-down view, the essence of which was an Aristotelian genotype, the earthly appearance of which is the phenotype. Johannsen emphasized the status of the genotype as the essential entity, independent of the environment and the circumstances in which it developed.

> Sodann erkennen wir, daß der "Typus" im Quetelet'schen Sinne nur eine *Erscheinung oberflächlicher Natur* ist, welche täuschen kann; erst durch weitere Untersuchungen wird entschieden, ob ein einziger oder mehrere biologisch verschiedene Typen vorhanden sind. Darum könnte man den statistisch hervortretenden Typus passend als Erscheinungstypus bezeichnen oder, kurz und klar, als "*Phaenotypus.*" (Johannsen, 1909, 123)

Johannsen's essentialist notion of the genotype is made most explicit by confronting it with that of Woltereck (1877-1944), who conceived of *the organism in its environment* to be the top-down essence (Woltereck, 1909). Woltereck offered a dynamic, non-typical norm of reaction of the hereditary element of the organism instead of the constant essential genotype. Woltereck's "phenotype curves" are graphical schemes of the degree of a "particular character as it manifests itself under different conditions." Johannsen the essentialist protested:

> Of course the *phenotypes* of the special characters, i.e., the *reactions of the genotypical constituents*, may under different conditions exhibit all possible forms of transition or transgression – this has nothing at all to do with constancy or inconstancy of genotypical differences. … Nobody will assume that there should be *genotypical* transitions here! Pure lines of beans may in one year be different in size … Differences of soil may produce something similar, and it is well known to breeders that some strains of wheat yield relatively much better than others on rich soil, while the reverse is realized on poorer soils. … The genotype-differences are nevertheless constant; the "Reaktionsnorms" of the organisms in Woltereck's cases, … are of course *eo ipso* "constantly different" just as well as the "Reaktionsnorms" of different chemical compounds" (Johannsen, 1911, 145-146).

The genotypes are as constant as are chemical elements, whatever the properties of the compounds these elements are involved in might be. It is important to note, however, that for Johannsen the major distinction was that between the *phenotype* and the *genotype;* his introduction of the "gene" was a bow to Mendel and Weismann, in spite of his holist conception. From the perspective of one decade later he asserted:

> My term "*gene*" was introduced and generally accepted as a short and unprejudiced word for unit-factors … but originally I was somewhat possessed with the antiquated morphological spirit in Galton's, Weismann's and Mendel's viewpoints. From a physiological or chemico-biological standpoint … *there are no unit characters at all!* … We may in some way "dissect" the organism descriptively, using all the tricks of terminology as we please. But that is not allowed in genetical explanation. Here, in the present state of research, we have especially to do with such genotypical units as are separable, be it independently or in a more or less mutual linkage. (Johannsen, 1923, 136-137)

Hence, by his insight Johanssen provided the framework that allowed to severe the Gordian knot of the unit character and the Mendelian factor of Bateson and his school, paving the way for the new science of genetics to become phenomenologically reductionist without being preformationist, or adopting an "instrumental reductionism" without adopting a "material reductionism."

This was taken up by another ardent opponent of Mendelian preformationism, namely Thomas Hunt Morgan. Morgan repeatedly tried to maintain the distinction between methodological reductionism – which he accepted – and conceptual reductionism – which he tried to postpone, if not reject (see, e.g., Falk & Schwartz, 1993). Although Morgan eventually had to give way to the technical hurdles that this position put in his thinking, he maintained the "instrumental reductionist" viewpoint throughout his career (Allen, 1978; Falk, 1986). However, among his students and colleagues, the distinction between the reductionist conception and the reductionist methodology often became rather equivocal.

Thus, by the beginning of the second decade of the twentieth century, when the focus of genetic research moved from Europe across the Atlantic to the United States, and especially to Morgan's "Fly-Room," the lines began to be drawn: Contrary to the Central European tradition of a strong philosophically, worldview-driven research, whether holistic or reductionist (for an extensive discussion, see Harwood, 1987), the American tradition was more a pragmatic, methodological reductionism, which, however, degenerated into conceptual reductionism.

Confronted with the achievements of reductionist genetics of the Morgan school, Johannsen's appeal for a genotypic – we would call it nowadays, genomic – perspective, was to no avail:

> We are very far from the ideal of enthusiastic Mendelians, viz. the possibility of dissolving genotypes into relatively small units, … Personally I believe in a great central "something" as yet not divisible into separate factors. The pomace-flies in Morgan's splendid experiments continue to be pomace-flies, even if they loose all "good" genes necessary for a normal fly-life. …
>
> But however far we may proceed in analyzing the genotypes into separable genes or factors, it must always be born in mind, that the characters of the organisms – their phenotypical features – are the reaction of the genotype in toto. The Mendelian units as such, taken *per se* are powerless.(Johannsen, 1923, 139)

Obviously, Johannsen was increasingly alarmed by genetics becoming more and more conceptually reductionist or genocentric. Instead of characters being "markers" for genes, genes became entities "for" characters. Any attempts to maintain a top-down conception were marginalized.

The genocentric notion probably was at its peak when Oskar Vogt attempted in 1926 to reintroduce essentialist notions of discontinuity of *taxonomic* entities. He and Timoféeff-Ressovsky (1900-1981) in his footsteps, dismissed phenotypic deviations of the pre-determined genetic essences by trivializing the variations as due to phenotypic "penetrance" or "expression" of the mutants (Vogt, 1926). In the same vein, geneticists were deliberating on whether pleiotropy – the production by one particular mutation of apparently unrelated multiple effects – was genuinely "primary," one gene producing fundamentally many products, or "secondary," according to which all the multiple effects of a gene should be reduced to a single effect (see Falk, 2000). As Laubichler and Sarkar (2002) note, these concepts "are invoked when any simple relation between a specific genotype and phenotype break down." They argue, furthermore, that even today "the basic role that 'expressivity' and 'penetrance' play in contemporary human behavioral genetics is ideological: they help maintain a genetic etiology for traits in the face of recalcitrant detail." It is instructive to recognize even in our present conceptions the remnants of these genocentric considerations of pleiotropy, though in the *reverse* direction, namely in the efforts to identify *the* DNA-sequence which may be authoritatively defined as *a gene*. Should the multiple alternative messenger-RNAs extracted from a given DNA-sequence be all considered the products of one gene, or may a DNA-sequence embrace multiple, partly overlapping genes?

In 1910 Morgan asserted that "When we speak of the transmission of characters from parent to offspring, we are speaking metaphorically" (Morgan, 1910), and in 1934, in his book *Embryology and Genetics*, he still continued to claim that "The story of genetics has become so interwoven with that of experimental embryology that the two can now to some extent be told as a single story." He even came up with an important model – not testable at the time – of development through differential activation of the constant hereditary entities, universally present in all somatic cells (Morgan, 1934, 9-10). But by that time Morgan apparently found out that he could not reconcile the old couple of embryology and genetics. As Jane Maienschein noted: "In the end, with his book *Embryology and Genetics* of 1934, he did not even find it profitable to try very hard" (Maienschein, 1987, 92): The "and" in the book's title turned out to be more a dissociate- than an associate-conjunction.

Herman J. Muller's identification of genes as the atoms of inheritance and variation, the physico-chemical properties of which should be elucidated, gained increasing acceptance. As stressed by Allen (2002, 27) "the strong emphasis on discrete and separable units interacting additively, as opposed to synergistically, posed serious problems for understanding developmental processes." Non-genocentirc notions, like Richard Goldschmidt's (1878-1958) top-down philosophy, just as Barbara McClintock's (1902-1992) genomic – sometimes mystic – conception, were politely but emphatically rejected. On the other hand, Lewis Stadler's (1896-1954) conceptual holism yet methodological operationalism (Stadler, 1954) was gracefully acknowledged.

Yet, the involvement of geneticists of the twentieth century in issues of development was not less than that of the biologists in the late nineteenth century who insisted that heredity and development were inseparable (Maienschein, 1987; Sandler & Sandler, 1985). In the footsteps of Mendel, problems of development were skillfully converted to problems of individual gene functions. Haldane (1892-1964) described the function of specific genes in the synthesis of

antocyanines that determine the flower color in *Pelargonium* (Haldane, 1954, 52-58) and Sturtevant (1891-1970) described the function of specific genes in the development of coiling in the snail *Limnea* (Sturtevant, 1923) or of organ differentiation in *Drosophila* (Sturtevant, 1932). Beadle (1903-1989) and Tatum (1909-1975) rightfully saw the direct line from Garrod's (1857-1936) "inborn errors of metabolism," which assigned specific malfunctions in metabolic pathways to specific human diseases, to their experimental evidence that reduced the metabolism of *Neurospora crassa* to that of a "one-gene – one-enzyme" relationships (Beadle & Tatum, 1941).

Significantly, however, Alfred Kühn (1885-1968) expressed a wider awareness of the need to refer to the organism when discussing genes in development. Based on his work on the pigmentation of the moth *Ephestia küniella*, he pointed out that our methodological experimental reductionism should not distract us from a conceptual holistic notion of the organism:

> [Our] apprehension of the expression of hereditary traits is changing from a more or less static and preformistic conception to a dynamic and epigenetic one. The formal correlation of individual genes mapped to specific loci on the chromosomes with certain characters has only a limited meaning. … One trait appears to have a simple correlation to one gene only as long as the other genes of the same action chain and of other action chains which are part of the same node, remain the same. (Rheinberger and Müller-Wille, 2004, quoting Kühn, 1941, 258)

Genetics: The taming of Darwinism

Whereas heredity and development could arguably be viewed as separate or identical disciplines, depending on whether the emphasis was on the physiological aspects of developmental changes or on the generation-stability of development, the relation of heredity and evolution was unequivocal as has been laid down by Darwin's "provisional hypothesis of pangenesis" in *The Variation of Plants and Animals Under Domestication* (1868). Bateson adopted Mendel's hypothesis immediately upon reading de Vries's 1900 paper because it was consistent with his notions of evolution by discontinuous steps. Contrary to Bateson's conclusions that relied on the *a priori* theory of "Mendel's Law of Hybridization," concerning the organization of biological material, Karl Pearson (1857-1936), the student of positivism, argued for a position that was based on statistical observations, ostensibly without prior theoretical assumptions. This, he claimed, was represented in the "Law of Ancestral Heredity" (at least, once the parameters introduced by Galton had been eliminated from it).

However, already in 1902 it was shown by Udny Yule (1871-1951) that *mathematically* one notion could be reduced to the other. Sixteen years later R. A. Fisher (1890-1962) too showed that mathematically the Law of Ancestral Inheritance can be reduced to that of Mendelian Inheritance (Fisher, 1918). Yule and Fisher, however, had different agendas.

A careful analysis of Yule's comments (Tabery, 2004) shows that the dispute between Pearson's biometricians and Bateson's Mendelians was one between an organismic, top-down phenomenological descriptive analysis of the variability of a race (= breeding population) and a bottom-up particulate reductive analytical interpretation of individual (or pure line) character differences in hybridization experiments.

> There has always been a good deal of misunderstanding between biologists … due in great part, I believe, to the fact that [they] do not use such terms as *heredity, variation, variable, variability,* in precisely the same signification. … The employment of quantitative methods necessarily leads to the use of such expression in a more precise signification.
> Quite generally, the statistician speaks of a character as *inherent* whenever the number or "constant" B [in the equation Y=A+B.X] is greater than zero. …
>
> The distinction between continuity and discontinuity of variation, between inheritance of attributes and of variables do not seem to me to be of *necessary* importance for the theory of heredity; … The real and important distinction seems to lie between the phenomena of *heredity* within the race, and the phenomena of *hybridization* that occurs on crossing two races admittedly distinct. (Yule, 1902, 195-199)

Mendelism is concerned with *hybridizations. Heredity* represents the population-aspect of inheritance, whereas *hybridization* is the method to study the inheritance of specific difference characteristics between individuals. The Law of Ancestral Heredity is concerned with *heredity*, it is a law that regards the correlation of variance in one generation of the population with that of another; we would say that it is a law in population genetics. Yule made this most explicit a year later, opening his paper: "The statistical theory of heredity, as developed in the work of Galton and Pearson, concerns itself with aggregates or groups of the population and not with single individuals" (Yule, 1903).

The statistician's notion of "ancestral heredity" is: "will a knowledge of the grandparent's character enable one to increase the accuracy of the estimate" of the character of the grandchildren above that obtained from the knowledge of the character of the parent? "If the answer to the question be in the affirmative, … then there is what may be termed a *partial* heredity from grandparent as well as from parent" (Yule, 1902, 201). The law that may be deduced is "that *the mean character of the offspring can be calculated with the more exactness, the more extensive our knowledge of the corresponding characters of the ancestry*, may be termed the Law of Ancestral Heredity" (Yule, 1902, 202). This is an early version of the breeders' concept of later years of *heritability*, which was formulated in terms of Mendelian genetics as the ratio of genetically caused (additive) variability to total variability of a character in a population (Rieger, Michaelis, & Green, 1991). Noticeably, Yule pointed out that "it is difficult to suppose that the weight attached to pedigree is based on nothing but illusion" (Yule, 1902, 202) – it begs for a hypothesis, and this was provided by Mendel's theory of hybridizations..

Fisher is generally regarded as "the first to successfully put forth a theory of the relationship between biometry, Darwinian evolution, and Mendelian inheritance," and his paper "is considered to be a direct descendent of Yule's earlier suggestion" (Tabery, 2004, 82). However, contrary to Yule's perspective of the demographic fact of the inherited variation in the population for which Mendelism may or may not provide a good theoretical explanation, Fisher was an ardent Mendelian reductionist who endeavored to explain inherited variance of populations from his genocentric perspective. As put by Tabery, "For Fisher the ancestral law was a special case of the Mendelian principle. For Yule, the Mendelian principles were a special case of the ancestral law" (Tabery, 2004, 90-91). Fisher claimed that "if one *first* supposes Mendelian inheritance, *then* one can derive the correlation between relatives, resulting in the ancestral law of heredity"

(Tabery, 2004, 83). In other words, Fisher provided a *conceptual reduction* of biometry's law of ancestral heredity: He took the Mendelian principles of inheritance as the explanatory base and then derived the biometric law of ancestral heredity to show that the statistical law was just a special case of the physiological law (Sarkar, 1998, 106). Fisher was aware that he came upon "the Law of Ancestral Heredity as a necessary consequence of the factorial mode of inheritance" (Tabery, 2004, 83, quoting Fisher, 1918, 421). In the era when genocentric reductionist conceptions gained primacy, Mendelian explanation made the Law of Ancestral Heredity redundant. Thus Fisher laid the foundation for the Modern Synthesis of Huxley and colleagues two decades later, which turned the theory of evolution into a theory of gene frequencies or population genetics.

But Yule, by juxtaposing Pearson's support for the Ancestral Heredity Theory and Bateson's claim for the Mendelian theory, also laid bare important aspects of the way scientific deductions are carried out.

> The fact that although the theory of ancestral contribution to heritage implies the law of ancestral heredity, the converse is not true: the law of ancestral heredity need not in any way imply actual physical contributions of the ancestry to the offspring. The ancestry of an individual may serve as guides to the most probable character of his offspring simply because they serve as indices to the character of the germplasm as distinct from his somatic characters. (Yule, 1902, 206)

As for Yule, Mendel's Laws and their relation to the Law of Ancestral Heredity,

> cannot at least be "absolutely inconsistent" with each other, as Mr. Bateson contends. The Law of Ancestral Heredity is certainly a law of nature of wide generality which cannot be dismissed in such a fashion. Mendel's Laws I assume to be true also. The problem is to delimit their respective spheres, and shew in what way the one type of law may pass into the other, or the two even coexist. (Yule, 1902, 207)

Whereas Pearson and Galton deduce (describe) backwards, to ancestral generations, presumably with no hypothesis implied, Mendel's laws, *ex hypothesis* deduce forward, to future generations (Gayon, 2000). Starting with Galton-Pearson's descriptive law:

> The value of the work of Mendel and his successors lies not in discovering a phenomenon inconsistent with that law [of Ancestral Heredity], but in shewing that a process, consistent with it, though neither suggested nor postulated by it, might actually occur. (Yule, 1902, 227)

No doubt, conceptual reductionism or genocentricity that became the dominant driving force in genetic research was a hypothesis-driven project at the price of excluding other alternative hypotheses. However, the positivists' claim of merely describing the phenomena with no hypothesis involved is misleading and similarly a dangerous illusion. The evolution of science along eras of paradigmatic science, punctuated by periods of scientific revolutions, reflects the struggle inherent in these perceptions. It may, however, be claimed that these alternations of paradigmatic and revolutionary science are only the phenotypes of a more constant scientific-type

that obtains a wide range of norms of reaction of conceptual struggles, which, even if explicitly denied, are constantly there.

Genocentricity and its discontents

I cannot elaborate on the development of the conception of genetic reductionist determinism over the later decades of the twentieth century, and it is out of the scope of this conference. It must, however, be kept in mind that the origins of genocentricity or more accurately hereditary-determinism, so dominant a factor in biological thought and practice throughout most of the twentieth century, may be traced to the late nineteenth century. With the intensification of the interrelationship of science and society, and the appearance of the scientists as professionals in their own right, biological determinism became a weapon, as well as a shield, of the growing community of scientists that considered it their duty to emphasize the social relevance of their science, yet had to face their inability to provide unequivocal answers. Eugenics is, of course, the first that comes to mind. Galton's Law of Ancestral Heredity, much like Darwin's pangenesis, was an *ad hoc* hypothesis, in which environmental effects had to be explained away. Ruth Schwartz-Cowan noted in 1977 that of all things that Galton wrote in *Heredity Talent and Character* in 1865, "he ended his argument against the inheritance of acquired characteristics with a rough statement of the continuity of germ plasm, the principle that we usually associate with the name of August Weismann and with the year 1883, …[as] the result of *sociopolitical rather than biological imperatives*" (Schwartz Cowan, 1977, 142, my emphasis). A few years later Galton created a physiological theory of heredity, the stirp theory that embodied the principle of continuity of germ plasm. "The idea of continuity of germ plasm was absolutely essential for Galton's scheme" of human breeding if it had to compete with that of indoctrination or education in averting the social problems of his time (Schwartz Cowan, 1977, 143). More recently, John Waller pointed out that "heredity came to imply relative fixity and resistance to therapeutic intervention. Moreover, the concept often provided physicians and alienists with a useful rationalization when therapeutics proved impotent" (Waller, 2001, 461). He quoted a poem of John Byrom, probably written in the 1730s, but published in 1894, that beautifully exposed the inclination of scientists to explain away phenomena by hereditary determinism, which was formulated by providing names:

> When our distempers did their name receive,
> (One instance more, good doctors, by your leave),
> Some chronic matters, such as gout and stone,
> That would the fare of no *arcana* own,
> To save their Credit these, the learned dons,
> Cried out, were fix'd hereditary ones:
> If a man's father, grand- or great-grand sire;
> Had the same, 'twas needles to enquire;
> Plain was the case, and safe the doctor's fame;
> The poor old ancestors bore all the blame.

Whichever way we take this, the differentiation of the phenotype and the genotype was superposed on the Weismannian segregation of the germ-line from the soma, and it made sense of "transmission genetics" – a study of heredity independently of development. But not less

significant a consequence of this reductionist, bottom-up disjunction that *Entwickelungsmechanik* forced on embryology was the "remarkable awakening of interest and change of opinion" among working embryologists with respect to evolution. Leo Buss in 1987 in his *The Evolution of Individuality* noted that an "ugly fact" which remained from this bottom-up approach to biology has been that "evolutionary biologists have only rarely been able to make specific predictions regarding the patterns studied by the reductionist research tradition." This situation changed only when "molecular biology has suddenly become a comparative, and inevitably evolutionary discipline. A new 'fossil record,' writ in the genome, is now accessible and being read in a necessary piecemeal fashion." Noticeably, the breakthrough has been technological before it became conceptual (Buss, 1987, vii).

Let me, then, round up my discussion by pointing out that with the presentation of the structural organization of DNA in 1953, the notion of genetic reductionism, and inadvertently also that of genetic determinism, got a strong boost. As Watson and Crick (1953) pointed out at the conclusion of their first paper, "It has not escaped our notice that the specific pairing we have postulated immediately suggests a possible copying mechanism for the genetic material," both for replication and for catalyzing specific gene products.

Notwithstanding, molecular biology, the triumph of genetic reductionist determinism, already carried the seeds of discontent. The detailed power of the molecular analysis challenged the old concepts by confronting them to the utmost. Many human and other organisms' traits which follow Mendelian patterns of inheritance have been labeled "genetic," and many of the genes involved have been mapped, their mutations identified, and screening programs for them instituted. Such a "geneticization," or the tendency to reduce trait differences to genetic ones is expressed in the increasingly popular notion of "the gene for" (Gannett, 1999; see also Nelkin and Lindee 1995). It reflects the extreme deterministic image that genetics acquired. However, an increasing awareness of the more complex traits, ones for which environmental contributions are known to be significant, and whose genetic component could not be attributed to a single gene, emphasized the urgent need for theoretical and empirical methods that would allow a system approach. As it turned out, toward the end of the century, the ambitious technical project of mapping the complete human genome, that had raised many eye-brows among theoretical scientists and philosophers, generated – out of necessity – new tools, empirical as well as theoretical, to meet the challenge. Consequently, we enter the twenty-first century to a large extent with a challenge similar to that of the beginning of the previous century, but this time we are much better equipped to face it both empirically and theoretically.

Comparing the wording of a recent statement by Francis S. Collins, head of the National Human Genome Research Institute at a workshop on *Human Genome Variation and 'Race'* at Howard University, on May 15, 2003, with one of ninety years earlier, which expresses exactly the same notion, by the eugenics-determinist Charles B. Davenport, makes this amply clear:

> In many instances, the causes of health disparities will have little to do with genetics, but rather derive from differences in culture, diet, socioeconomic status, access to health care, education, environmental exposure, social marginalization, discrimination, stress and other factors. Yet it would be incorrect to say that genetics never has a role in health disparities. …

> The question of whether genetics will explain a substantial proportion of health disparities … is largely unanswered … (Collins, 2004)

And:

> In the study of human heredity it has first of all to be recognized that progress will be made only as traits are studied one at a time. The modern science of heredity indeed seeks as the element of study the "unit character." What are unit characters can, however, be told only by breeding experiments in which the true units reveal themselves as relatively, if not absolutely, constant, unalterable, indivisible things. … The first step in the resolution of human traits is, then, a primary rough analysis into fairly simple traits and, second, the study of the behavior of these traits in heredity. …
>
> …The criticism may be made that not all the traits, especially the diseases, given here have any hereditary basis. It is not affirmed that this is the case and yet it cannot be denied that all have an hereditary basis. Even tuberculosis, syphilis, and the plague are the product of a specific germ acting on a susceptible protoplasm and it is this susceptibility that is the inheritable factor. (Davenport, 1912)

To close the circle, we have to return to Roux's emphasize of the need for simple methodologies, and of the caution that must be exerted in interpreting the data as simple concepts:

> Among biologists there is a tendency derived from inorganic sciences, *to regard the hypothetical deductions which appear to us to be the "simplest" as having the greatest probability for the very reason that they seem so simple.* [But a]lthough much has been done on this assumption, and unfortunately must be done, … nevertheless this method must always be applied with great reserve to normal biological phenomena, …
>
> Thus we suppose that we are really simplifying matters when, e.g., we attribute in consequence of functional adaptation many typical and purposive forms to the self-constructive effects of use. The correctness of this *principle* and its application in many cases has long been capable of direct proof. Nevertheless, we observe that many structures which might be the result of this principle, … are already established before there is an opportunity for them to exercise their definite functions. (Roux, 1894, 119)

Only "forward deductions" from experiments that control the causal force, can be "certain." There is no sense (at least in living systems) in isolating one causal force, independently of other causes, but this was the only way that a cause could be studied directly. Roux would have been happy if it were possible "to reproduce *synthetically* in an inorganic way *structures, forms, and processes which resemble as closely as possible*, those of the organic world." But as he noted, "even in cases where it is claimed that such a reduction has been brought about, it appears that the part which the simple components contribute to the formation in question, as compared with that of the coöperant complex components, has been considerably overestimated" (Roux, 1894, 128-129). Embryologists, in view of such a program as Roux's, were caught between holistic concepts of organisms as complex systems that cannot be simplified and studied effectively by the methods of physicists and chemists, and the reductionist need to apply experimental mechanics in order to study "real" causes. Geneticists by having been offered distinct and discrete entities, the genes,

believed that they could successfully apply not only methodological, but also conceptual reductionism. Since then we have advanced in our molecular and computational technologies, and the old dream may again appear luring to the modern genetic engineer. Yet, the same problem may exist in today's studies in genomics which try to follow simultaneously many *individual* causes. Unless a conceptual change takes place, the risk remains that the methodologically isolated forces would be inadvertently identified with the conceptually assumed forces.

The challenge, as I see it, is to sort out the reductionist methods of empirical research – which have been and will be irreplaceable in research in the life-sciences in general, and in genetic research in particular – from the notions of conceptual reductionism that may have had tactical, heuristic and instrumental value, but must be replaced by systems' conception, which are, after all, that unique specificity of life on earth. Griesemer's (2000) suggestion that we should think of both development and inheritance as processes reducible to reproduction may be a step in the direction of maintaining methodological reduction as an efficient research instrument within the framework of organisms as integrated systems.

If we return, for a moment to the 1920s and 1930s, when Niels Bohr and Erwin Schrödinger wondered "What is Life," and physicists, in the footsteps of Max Delbrück flocked into biological research, to discover the specific Laws of Nature hidden in living creatures, we may say now, with some confidence: There are no special laws of the living world, neither *Lebenskraft*, nor *Entelechy*, nor any need for an Anthropomorphic Principle or divine power. It is just the evolution of the material world, as put forward by Darwin, and contrary to that believed by Mendel.

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The Biometric Sense of Heredity: Statistics, Pangenesis and Positivism

Theodore M. Porter

The first commentators on almost every scientific episode are the participants themselves, whose categories of analysis provide a starting point for proper historical studies. But one task of the history of science is to gain some distance from scientific debates and to see them against a wider background in ways the protagonists could not. This generally means, at a minimum, refusing to pass along as historical truth the judgment of the winners. In fact there was no clear winner, or at least no loser, in the controversy between Biometricians against Mendelians. But the statistical program of population genetics has remained mostly distinct from laboratory studies at the level of individual genes, which now usually means molecular genetics, and accordingly, somewhat discrepant histories have also been perpetuated. Add to this the intensity of the controversy at the beginning of the twentieth century and some bitter rivalries within the statistical program involving such leading figures as Karl Pearson, Udny Yule, R. A. Fisher, and Sewall Wright, and we have a landscape littered with polemical arguments to seduce and sometimes mislead the historian.

Blending inheritance or blended characters?

The great evolutionist Theodosius Dobzhansky identified the deficiencies of the theory of blending inheritance in his *magnum opus* of 1937, *Genetics and the Origin of Species*. This concept, he explained, "was based on the assumption that the germ plasms of the parents undergo a sort of amalgamation in the hybrid." They dissolve as dye mixes into water, uniformly. "The amount of variation present in a sexually reproducing random breeding population must be halved in every generation. Given a population which exhibits a large variability at the start, we are bound to observe a progressive, rapid, and irretrievable decay of the variability, until a complete homogeneity is reached." Darwin and his follower were "inexorably driven to this conclusion," Dobzhansky continued, and this is why the new Mendelian approaches after 1900 were so urgently needed to complete the theory of evolution. The "particulate" theory of the gene allowed evolution to be understood in a new way. "Even where the hybrid or heterozygote is intermediate between the parents, no contamination of the genes takes place, and the homozygotes recovered from the hybrids are like the parental races." A particulate germ plasm automatically maintains variability "on an approximately constant level despite the interbreeding."[1]

Historians of science have come to recognize blending inheritance as one of the principal obstacles to an adequate theory of evolution. Robert Olby, for example, explains: "It is now well known that the most unfortunate of the assumptions underlying Darwin's mechanism of evolution was that of blending heredity; i.e. that parental differences are merged in the offspring of bisexual reproduction so that variation is constantly being diminished."[2] Peter Bowler seems to

[1] Dobzhansky (1937), pp. 121– 122.
[2] Olby (1966), p. 55.

adopt a completely different view: "We shall see that Darwinism would function quite effectively with a model of blending inheritance." Yet in his interpretation of the disagreement between biometricians and Mendelians, he sounds very much like Dobzhansky: "In effect, Mendelism undermined Fleeming Jenkin's claim that natural selection was ineffective because new characters will be swamped by interbreeding with unchanged individuals. If heredity is particulate rather than blending a favourable new character will be preserved intact so that its frequency can be gradually increased within the population."[3] And Alfred Sturtevant, the eminent geneticist turned historian of genetics, explained how Darwin's reliance on blending implied that favorable variations would be swamped, retarding or even blocking evolutionary change.[4]

Some other biologist–historians have been more perceptive about blending. Ernst Mayr, in a review, called Olby's treatment of blending inheritance "thoroughly confused," and referred to the "myth," left intact by Olby, that Mendel's greatest contribution was his "concept of particulate inheritance, in opposition to the 'prevailing' idea of blending inheritance." There is, he continued, abundant evidence that Darwin did not support blending inheritance.[5] Michael Bulmer, in a recent biographical study of Francis Galton, comments that this historical interpretation of blending inheritance originated with R. A. Fisher, whose 1930 book *The Genetical Theory of Natural Selection* is the most complete expression of his successful effort to integrate Mendelian genetics into a mathematical version of Darwinian evolution. Bulmer calls the claim misleading and complains that it has "been uncritically accepted by many evolutionary biologists." Dobzhansky, as we can see, was among them, for he too cited Fisher's book as well as an earlier paper in Russian by Sergei Chetverikov on the baleful effects of the blending theory on early Darwinian theories of evolution. Bulmer offers an important distinction between "physical blending of the hereditary particles during fertilization, which is what Fisher meant, and the blending of phenotypes in the sense that offspring are often intermediate between their parents."[6]

Fisher's analysis of Darwin's assumptions about inheritance was based on a reading of manuscripts from 1842 and 1844 and letters to Huxley as well as Darwin's books, and is not without merit as historical interpretation. The conclusions he drew, however, went well beyond Darwin. Since half of all variance will disappear in each generation in a sexually–reproducing population that mates randomly, a huge mutation rate is required to maintain variability. This, he suggested, was why Darwin put so much emphasis on the causes of variation. The concepts Fisher invoked, obviously, are anachronistic. He was more profoundly misleading, however, in leaping directly from Darwin's era to his own, as if nothing but Mendelian genetics had happened since 1859 (or at least since Darwin's *Variation of Plants and Animals under Domestication* in 1868), until Fisher himself stepped heroically forward to demonstrate the need for particulate rather than blending inheritance and to demonstrate the mathematical advantages of the substitution.[7]

The concept of "blending inheritance," unknown to Darwin, was not an actor's category even in the era of Bateson and Pearson. Indeed, Fisher may have coined the phrase. "Blended inheritance," which sounds at first like the same thing, was a well–known biological concept

3 Bowler (1989), pp. 48, 63, 139.
4 Sturtevant (1965), pp. 20– 21.
5 Mayr (1973), p. 141
6 Bulmer (2003), p. 139.
7 Fisher (1930), chapter I.

before 1900. Galton deployed the term in 1889 in his *Natural Inheritance* in a way that suggests something new. The children of a white and a negro, he wrote, "are neither wholly white nor wholly black, neither are they piebald, but of fairly uniform mulatto brown. The quadroon child of the mulatto and the white has a quarter tint; some of the children may be altogether darker or lighter than the rest, but they are not piebald. Skin colour is therefore a good example of what I call blended inheritance." He immediately made it clear that blending implied no theory of heredity, but only described the results of certain crosses. "It need be none the less 'particulate' in its origin, but the result may be regarded as a fine mosaic, too minute for its elements to be distinguished in a general view."[8] A few pages back, Galton had explicitly endorsed a theory of "particulate" inheritance—the word, he said, is "good English" though the quotation marks embracing it implied unfamiliarity. There were no grounds for supposing unfamiliarity when Fisher called for a particulate theory of inheritance in 1930. Galton illustrated particulate inheritance by comparing the formation of an organism to one of those Italian buildings whose elements, stones and columns and pediments, had been pillaged from previous structures, its progenitors.[9]

There was no theory of blending inheritance in the early twentieth century, but only a distinction between those characters that blend in the offspring of hybrids and those that "alternate," which meant to assume one or another discrete form. The entry on heredity in the eleventh edition of the *Encyclopaedia Britannica*, published in 1910, explains that the discrete forms studied by Mendel as well as the saltatory "mutations" made famous by Hugo de Vries pertain "almost exclusively to the crossing of artificial varieties of animals and plants" and "point strongly to the occurrence of alternate inheritance instead of blended inheritance for artificial varieties. On the other hand, in the case of natural varieties it appears that blended inheritance predominates."[10]

This identification of mutations and Mendelian factors with hybridity was widely shared in the first decade of the twentieth century, before T. H. Morgan's fly lab redefined mutation as a change at the level of a single gene. Karl Pearson's student Udny Yule, in his effort to reconcile Mendelian with biometric studies of heredity, argued that Mendelians and statisticians used the same words in different ways. Statisticians, he explained, understand *heredity* as the study of how the divergence of an individual from a racial mean relates to the divergence of its offspring. This was not the object of investigation for Mendelians. "On passing from the Law of Ancestral Heredity to Mendel's Laws, we are passing from a law of intra–racial individual heredity to a series of laws based solely on hybridisation–experiments, and clearly stated by their discoverer as laws of hybridisation only."[11] These remarks suggest that, to allude to a classic paper by Olby, Mendel may have been a Mendelian after all, since the first generation of Mendelians was scarcely less preoccupied with hybrids than he was.[12] In a 1905 symposium in Philadelphia on the mutation theory, William Castle followed Darwin and de Vries in their use of artificial selection as a source of insight into biological evolution. Nature and breeders alike rely overwhelmingly on mutations.

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8 Galton (1889), p. 12.
9 *Ibid.*, pp. 7– 8.
10 Mitchell (1910), pp. 353–354.
11 Yule (1902), pp. 196, 222. See Tabery (2004).
12 Olby (1979).

The latter do not simply wait for these to happen, but work actively to create them through cross–breeding.[13]

Blending and swamping

As Dobzhansky's argument exemplifies, the significance of blending inheritance has from the beginning been associated with an argument that, absent some mechanism of preserving variants, they would disappear due to "swamping" as exceptional individuals bred back into the parent population. In 1867, Fleeming Jenkin offered what is still often regarded as a damaging critique of Darwin's theory of evolution by natural selection. He argued that indiscriminate mating would swamp the effects of any new variant, which would ineluctably be averaged away in subsequent generations unless the exceptional individual could find other similarly–endowed specimens with which to reproduce.[14] In the era of Fisher and Dobzhansky, Mendelian genetics was celebrated as the first adequate response to this conundrum.

It is, in its modern form, a curious argument, since it seems to suppose that variants leading to new species must be discrete and exceptional, yet subject to attenuation through continuous processes of mixing or blending. Jenkin, who understood the argument better than most of his interpreters, did not apply it to continuous variation of the sort that would be found in every generation. Although he doubted the capacity of new species to arise through selection on continuous variation, this was for other reasons. He argued that such variation would run up against limits, and could not support the substantial changes of form required for a new species. This doctrine of limits to variation was a common and enduring reaction to Darwin. Skeptics of Darwinian evolution in the early twentieth century, when the mutation theory of de Vries was all the rage, distinguished between continuous or "fluctuating variations," which go nowhere, and mutations, which can provide a new stable center for a species. Some, including Castle, made the distinction virtually a matter of definition: "mutations are permanent, variations transitory;" and some naturalists sympathetic to Darwin such as Asa Gray as well as opponents like Louis Agassiz believed that small variations were mere oscillations around a normal state which could not be inherited.[15] De Vries and his contemporaries did not rely on the argument of swamping to discredit evolution by selection on small variations.

Neither did Jenkin, who instead used his swamping argument to deny the possibility that new species could arise through discontinuous variation or "sports." More than forty years ago, Peter Vorzimmer showed that Darwin perfectly understood this aspect of Jenkin's argument, and that if confirmed him in his gradual shift away from any reliance on sports and toward a belief in nature without leaps: *Natura non facit saltum.* Saltations, which he already regarded as too rare and too often infertile to be the main engine of species change, now appeared unlikely to perpetuate their own kind even when they did survive and reproduce. The objection applied most forcefully to sports that blended, and Huxley held out hope that new species might form as a result of nonblending ones. Darwin, however, emphasized more than ever the gradual change that could

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13 Castle (1905).
14 Jenkin (1867).
15 Castle (1905), p. 524; Vorzimmer (1963), p. 380.

occur as selection acted on those small favorable variations that should appear relatively abundantly in every generation.[16]

Galton, progenitor of biometry, wrote of blending and discrete inheritance as the radical alternatives and argued that few if any traits conform entirely to either extreme. He minimized the force of Jenkin's critique, even for the case of hybridization as in the imagined presence of a lone white individual within a wholly black population, concluding "that the establishment of a somewhat rare variety as that of white men naturally suited to thrive and multiply in tropical climates" was not such an improbability as some supposed. While he recognized that "mutually–exclusive heritages" such as eye color will be distributed differently from blended traits, his example was about hybridization (at least in a loose sense). Nowhere did he consider that blending must lead to collapse of variation within the race or species, as argued by Fisher and Dobzhansky, and there is no hint of a suspicion that he thought micro–particulate inheritance was needed to preserve variability.[17] Karl Pearson, similarly, worked out a descriptive account of gradual biological change, a detailed quantitative elaboration of evolutionary mechanisms that could work on continuous variation even under conditions of indiscriminate mating. He argued that it did not depend on any particular mechanism of heredity. Like Darwin, he supposed that biological variation was natural and fundamental, and not at all exceptional in the way that Fisher understood mutations to be. Pearson's solution was in some ways a graphical version of Darwin's, translated however into a statistical idiom of frequency distributions that was mostly alien to Darwin.[18] Pearson's version was enhanced to include the quantitative effects of heritability and differential fertility as well as survival. Not the appearance of mutant individuals but a slowly shifting frequency distribution was the mark of evolutionary change for Pearson. The raw material of evolution appeared like statistical clockwork in each generation.[19]

Pangenesis and statistics

Although Darwin placed increasing emphasis in the 1850s and 1860s on continuous variation, he was not in any straightforward way an advocate of blending inheritance. Bulmer shows how he restated Jenkin's (flawed) quantitative demonstration that a singular variation, even if advantageous, must disappear over time, so that half the offspring would possess the variation and half not rather than, as in Jenkin's version, having the variation attenuate by half in each successive generation.[20] The "Provisional Hypothesis of Pangenesis" that he published in his big book on variation in 1868 was itself, after all, a particulate theory. His gemmules, to be sure, were very numerous, perhaps one for every cell in the body, and examples like the blending of skin color in mulattoes convinced him that these gemmules must merge as they form such tissues, so that in the next cycle of reproduction they would be intermediate in color.[21] Others, most notably his cousin Galton, rejected all blending in favor of a mosaic theory of the relations of gemmules. Galton's model of hereditary transmission was thus inspired by Darwin's and used the same terms, but his

16 Vorzimmer (1963). See Bulmer (2004) for some clarifications and corrections.
17 Galton (1887), pp. 400– 402.
18 Gayon (1992), pp. 95– 112.
19 Pearson (1900), chapters X–XI.
20 Bulmer (2004), p. 290.
21 Olby (1966), pp. 70– 71.

gemmules did not blend. On the contrary, Galton conceived mechanisms by which gemmules could join forces to produce visible features recognizably derived from one ancestor or the other. In terms of his architectural analogy, this involved the columns or porticos that were passed along intact to a new structure. In statistical terms, this insight provided the basis of his biological theory of correlation, which was based on gemmules that tended to stay together.[22]

More fundamentally, the statistical study of heredity was anchored from the beginning in the theory of Pangenesis. In 1870, after all, statistics was the quantitative science of human populations, and Galton depended on analogies between gemmules and individuals in society in order to imagine a statistics of human heredity. Much of the historiography of heredity links the quantitative biometric approach to a strong descriptive positivism which dismissed as meaningless the idea of genetic elements. Pearson's *Grammar of Science* is Exhibit One for the prosecution,[23] but Pearson never rejected genetic units, and sometimes worked at mathematical methods to try to count them. As Ida Stamhuis has shown, the association of Pangenesis with statistics was not limited to Britain, but was also central to the work of de Vries in Amsterdam, both before and after he became familiar with Mendel's work.[24]

Was Mendelian genetics required for an adequate theory of evolution? Bowler, as we have seen, suggests that it was, that Mendelism at last enabled biologists to escape Jenkin's critique. But in the same breath he explains how T. H. Morgan and coworkers came to the crucial insight that evolution might occur through the accumulation of small mutations so as to appear continuous.[25] This last formulation, absent the language of mutations, is indistinguishable from the biometric version of evolution. In 1909 (repeated as late as 1925), Wilhelm Johannsen identified Galton's pathbreaking statistical methods, more than Mendel's discoveries, as the basis for a hereditary mechanism that could make sense of Darwinian evolution.[26] And in fact the two were in many respects compatible, as the leading biometricians generally recognized. The Darwinian gemmules on which rested Galton's statistics of heredity could readily be replaced by Mendelian genetic elements without fundamentally revising the statistics.

In principle, statistical approaches favored a quantitative positivism, in which phenomena were counted and analyzed, and the underlying mechanisms put aside. As we will see, Pearson often proceeded this way, sometimes to the chagrin of his allies as well as the bewilderment of his opponents. The statistical study of heredity, however, was initially not at all positivistic, and an ontology of heredity particles was long connected to it. In our terms, the hypothesis of Pangenesis was perhaps more like a model than a theory, particularly as wielded by Galton. Nobody had ever detected a Galtonian gemmule or a pangene of de Vries, and nothing was known about the physiological processes that might produce them. Galton undertook, unsuccessfully, to demonstrate their presence in the bloodstream through transfusions among rabbits. Otherwise they were mostly theoretical entities, whose presumed properties were inferred in a somewhat circular fashion from the phenomena they were designed to explain, rather than tangible objects susceptible to laboratory manipulation.

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22 Porter (1986).
23 The classic statement is by Norton, e.g. (1975a) and (1975b).
24 For example, Stamhuis (2003).
25 Bowler (1989), p. 138– 139.
26 Johannsen (1926), p. 5. On Johannsen see work by Nils Roll–Hansen.

Like Darwin, Galton supposed there must be a very large number of gemmules, enough to permit reliable statistical conclusions to be drawn. He used an extended analogy with processes of political representation to develop the statistical aspects of heredity. The gemmules in a newly–fertilized egg would undergo first a segregation or "election" to determine which would form themselves into the embryo, and, later, a second segregation to sort out the ones that would be transmitted by this individual to the next generation. There was, he explained, no need for a special mechanism to assure that every part of the body was represented; the familiar regularities of statistics would take care of this. When he first outlined this model, Galton regarded the transmission of this genetic inheritance from generation to generation as a random process, akin to fair games of chance, like drawing balls from an urn. The sorting that determined which of these elements should "represent" the whole population of gemmules by determining the bodily characteristics of the offspring, on the other hand, was subject to the elective affinities of the gemmules. Such affinities were required to assure that a tall individual would tend to be more or less uniformly so rather than having very long legs but very short arms.[27]

As Galton's program of experimental breeding got underway, and again when he applied his analysis to a burgeoning collection of records of family traits, he revised his account of these biological processes and of the statistical methods appropriate for dealing with them. His first important mathematical result on heredity, based on pea–breeding experiments, was a law of "reversion," a quantitative relationship between parent pea seeds and their descendants, which he announced in 1877. The plant grown from a large pea seed tended itself to produce large peas, though these were on average less exceptional than their parent. Using deviations from the mean of the whole population as his measures, he found that the average deviation of the offspring from a given size class of parent was a constant fraction of the deviation of the parent. Given that there was also considerable variation among offspring of parent peas from every size class, there were mathematical reasons that the fraction must be less than one if the overall variability is stable from generation to generation. But there remained, he thought, a biological aspect to the question: why were the offspring seeds on average less exceptional than their parents? His answer followed from his hereditary model. According to his version of Pangenesis, the gemmules that formed a given individual derived not only from their parents, but also from grandparents and great grandparents. The more remote these ancestors, the more numerous they must be, and hence the more similar to the general population. This shift toward the mean of the "race" was literally a reversion in the biological sense, a partial return to the traits of these more remote ancestors.[28]

A decade later, when he revisited these questions of inheritance armed with a mass of human records, he was able to apply the same mathematical formulation. He changed the word, however, to reflect a new understanding of the biology of heredity. "Regression," his new term, might involve the influence of ancestral forms, but designated now an innate tendency to return to type, which would correspond with the mean for the population. He explained to the Anthropological Section of the British Association in 1885: "The type is an ideal form towards which the children of those who deviate from it tend to regress…. The stability of a type would, I presume, be measured by the strength of its tendency to regress."[29] Or, in terms of Pangenesis as political

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[27] Galton (1873).
[28] Galton (1877); Porter (1986), p. 270– 296.

analogy, the affinities that formed an individual out of genetic materials meant that the selection of gemmules to be expressed in the offspring was not random, but favored the prevailing type, the dominant party, or the race, from which most gemmules would have derived. This made Darwin's scheme of evolutionary change, without leaps, problematical, because small variations would tend to return to the racial mean unless displaced by a sport or pushed away to a new point of stability by strong and insistent selective pressures. Regression, in this version, was closely allied to the biological correlation of parts, which was about harmonies or similarities among different organs or parts of a single organism. In 1888, just as he was finishing his book *Natural Inheritance*, Galton saw in a flash that the correlation of measurable traits could be understood with the same mathematics as he had developed for regression. As so often in his mathematical work, he came to this realization in the context of a specific quantitative problem, but it also made biological sense to him, for correlation and regression both followed from the same structure of elective affinities among gemmules.[30]

The strange positivism of Karl Pearson allowed, and was perhaps even favorable to a hypothesis of Pangenesis, on condition that the gemmules be regarded only as conceptual, and not projected into the world of sensations. Although Pearson's working methods were often positivistic in the sense of not presuming any particular theory of heredity, he did not reject genes on epistemological or metaphysical grounds. He criticized Mendelism rather for its failure, as he saw it, to meet the standard of adequate agreement with the results of measurement and calculation. Genes and Mendelian factors, too, were scientifically legitimate, but only as handy formulas that are valuable to the extent they lead to correct conclusions. Already in 1900, before Mendel meant anything to him, he complained of August Weismann's projection of Darwin's gemmules "into the phenomenal world," as if we can see or touch them. We should not succumb to mechanistic delusions that would transmute these inventions of mind into material explanations or causes of life.[31] But Pangenesis was Pearson's preferred model of hereditary processes, as it was Galton's. He never acquired the same enthusiasm for Mendelian units, but there is simply no basis for the supposition that he rejected them on grounds of positivist philosophy.[32] Although his quantitative research program was indeed positivistic in important respects, this did not exclude physiological mechanisms of heredity. The statistical "law of ancestral heredity" was potentially consistent with a plurality of models of heredity.[33]

By 1900, when Pearson issued this complaint, he and his biological ally W. F. R. Weldon were already somewhat embattled over the proper uses of statistics in biology. The rapid rise of Mendelism, beginning that same year, sharpened the antagonisms. Pearson repeatedly emphasized that Mendelism was, in principle, scientifically legitimate, and should be judged according to its fruits. The gene, in this respect was no different from the pangene or the gemmule, except that some bold Mendelians were soon announcing a host of one–gene traits. For Pearson, as for many of his antagonists, eugenics gave particular urgency to the new science of heredity, and

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29 Galton, (1885).
30 Galton (1886). See also Galton (1889); Galton (1888); Galton (1890).
31 Pearson (1900), pp. 335–337, from chapters that appeared only in the second edition of this work.
32 I thus take issue with Norton (1975). MacKenzie offers the beginning of a critique in (1981), pp. 140–141; Porter (2004), p. 269.
33 Provine (1971), p. 25.

Pearson thought that irresponsible Mendelian claims "in the name of eugenics" were casting discredit on it. He argued that one–gene traits were very rare, and he devoted almost all of his own efforts to statistical investigations of complex quantifiable traits. The logic of his program reduced Mendelian efforts to a very minor role in evolutionary or eugenic studies, which mostly would be subsumed into the statistics. In the 1890s he worked out methods for ascertaining the number of gemmules that determined a limb or organ from the specific form of the asymmetrical frequency curve, and in the early twentieth century he tried to determine the number of genes required for such traits, as evidence that biometry was a necessary tool even for Mendelians engaged in studies of evolution and eugenics.

The graphical basis of the science of heredity

The gap between biometry and Mendelism was not mainly one of philosophy, but of skills and practices. The statistical study of heredity was graphical and sometimes highly mathematical, and did not involve tight experimental control. It was characteristic of Pearson's statistics, and a point on which he differed from his successor and bitter antagonist R. A. Fisher, that not too much was made of the difference between observation and experiment. Fisher also took a more sympathetic interest in Mendelism, as indeed did Pearson's student, with whom however he was for some years at odds, Udny Yule. Although Pearson did not reject Mendelism, it was for him a narrow and rather uninteresting special case of a program that proceeded with other tools using other sorts of data. Like Galton, and for similar, mostly eugenic reasons, Pearson was very much concerned to distinguish environmentally–conditioned traits from those inherited biologically. But his preferred methods for this work involved instruments of measurement and quantitative analysis, not laboratory interventions. The distinctive strengths of zoology and botany, displayed in efforts to connect mechanisms of heredity with careful microscopy and investigation of cellular physiology, meant little to the biometricians.

Pearson's biometric style owed much to Galton, whom he learned to appreciate as a result of collaborations with his University College colleague Weldon. This much is well known, and should not be minimized. In my recent book on Pearson I try to show that his quantitative program derived also, and no less fundamentally, from his role as a professor of applied mathematics, which in practice meant engineering mathematics, and that his Biometric Laboratory drew inspiration from an Engineering Laboratory established by another University College colleague, Alexander Kennedy. There students were trained in the use of instruments to trace and measure curves, and in graphical methods to solve mathematical problems. Pearson originally took up statistics in the form of economic and social statistics as an important case of subjects that could be handled graphically. When, thanks to Weldon, he came to see evolution as one such subject, his excitement about this project grew uncontrollably, yet for three more years he still thought of statistical biology as a chapter in graphical statics. Some of the most fundamental concepts of Pearson's new statistics derived from engineering graphics. Standard deviation, for example, was mathematically equivalent to swing radius in mechanics, and could be calculated on paper using the very same graphical methods.[34]

[34] Porter (2004).

Pearson outlined his graphical vision for the study of heredity and evolution most systematically in his final set of Gresham lectures in 1893 and 1894, and then again in the chapters on evolution he included in the second edition of his *Grammar of Science*, published in 1900. Galton, too, had relied heavily on graphical presentation, and Weldon used graphs not only to summarize data but also to solve problems. A double–peaked graph was probably responsible for Weldon's original intuition that the Naples crabs he had been studying were undergoing incipient speciation, and he used graphical techniques to separate the compound curve, as he thought, into two simple ones. This was the problem that fired Pearson's enthusiasm for the statistical study of evolution and the subject of his first "Contribution to the Mathematical Theory of Evolution" in 1894. There was a sense in which Pearson's statistical practice was always informed by a geometrical or graphical sensibility, even in writing that took the form of vast algebraic tangles. The graphical structure was most emphasized in relatively popular presentations, as in *The Grammar of Science*, where he constructed a hypothetical frequency distribution of an unspecified trait and showed how it might change due to differential rates of mortality and then reproduction as functions of this trait. The last stage of the analysis involved heritability, the percentage of individual exceptionality that was passed along to the next generation, and the graphical exercise concluded with a new frequency curve representing the offspring generation. The difference between parental and offspring curves was a direct indication of evolutionary change, in a form that should not be compromised by interbreeding and did not depend on any particular mechanism of hereditary transmission. Pearson published this work in 1900, just before Mendelism became an issue for him.[35]

Pearson's research papers for the *Philosophical Transactions of the Royal Society*, and later for *Biometrika*, were rarely graphical to the same extent. He came to see that his comparative advantage lay in explicit, algebraic solutions, which few if any biologists could duplicate or even follow. But the relation to mechanisms of inheritance was the same. Pearson's assumptions about heritability were of a quantitative kind, and did not involve any clear assumptions about how traits were passed from parents to offspring. He could not accept, and indeed actively worked to undermine, Galton's ideas about stability of type. Mendelian genes presented no particular problem, provided that he was allowed to work on them collectively, not individually. Reconciling Mendelism or Pangenesis with biometry was of moderate interest to him, but was far from central to his research agenda. He needed only to assume that most measurable traits—most traits that mattered for survival and reproduction—were composites of many elements and so could be handled statistically. It was not that genes were inherently unknowable or illegitimate, but that he expected more mileage out of a statistical research program, one that depended on counting and measuring. His studies could be called positivistic in a different, though related, sense, because Pearson was mostly uninterested in the biological details. Pangenesis, too, did not remain very important for Pearson's biometry, but it had played a considerable role in the creation of the statistical study of heredity, and the rather indirect significance that it retained was supportive of

35 Pearson (1900).

biometry rather than antagonistic to it. When he discussed physical models of heredity they were always particulate, and theories of blending inheritance, if indeed there were any such in biological work on heredity in the early twentieth century, had no role whatsoever in Pearson's biometry.

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Sources of Johannsen´s Genotype Theory

Nils Roll–Hansen

The rejection of "soft inheritance" was a crucial step in the founding of classical genetics, according to Ernst Mayr. He acknowledges Wilhelm Johannsen's central role in clarifying the distinction between "genotype" and "phenotype", in coining the term "gene", and in setting up his classical selection experiment on beans demonstrating a striking hardness of the genotype for quantitative characters. But Mayr nevertheless claims that a clear grasp of the distinction was not achieved till the middle of the 20th century, with the discovery that "the genotype consists of DNA. […] In the early years of genetics considerable confusion continued, from which even Johannsen was not exempt" (Mayr 1973, 1982: 782–783). This view is also reflected in the analyses of William Provine (1971), Frederick Churchill (1974) and Garland Allen (1979). However none of these authors have discussed Johannsen's pre–1903 work, a main reason being that most of it was published in Danish.

Wilhelm Johannsen started his education in the best primary and secondary schools of Copenhagen. But his father could only afford one son at the university and Wilhelm took vocational training as a pharmacist. The final part of this training was a one year study at the University of Copenhagen, where he became a favourite student of Eugenius Warming, professor of botany. 1880 Wilhelm passed his final exams as pharmacist, and in 1881, 24 years old, he was appointed assistant at the Carlsberg Laboratory. His job was to make investigations of barley in the chemistry section headed by the analytical chemist Johan Kjeldahl, famous for his method of analyzing organic nitrogen. Under this "unusually liberal boss" Johannsen had ample opportunity to pursue advanced biological studies including periods in Germany and Paris (Johannsen 1910). His other senior colleague was the botanist and yeast specialist Emil Chr. Hansen, known for his "pure lines" of yeast cultures. In 1887 Johannsen left Carlsberg in prospect of a lectureship in plant physiology at the Royal Veterinary and Agricultural College. In 1905 he was called as professor ordinarius in plant physiology to the University of Copenhagen where he spent the rest of his career.

Aristotle, Bacon and Claude Bernard

Some characteristic traits of the young plant physiologist appear in an early survey article printed in a popular agricultural magazine (Johannsen 1883). With inspiration from Claude Bernard he praised the experimental method and aimed for the basic physiological principles of life common to plants and animals. Johannsen saw experimental natural science and natural history as united in a comprehensive theory of nature ("naturlære") (p. 333). He demanded stringent method both in experiment and logic, and pointed to the perennial threat of Bacon's idols to all good science (p. 360). Already, Wilhelm Johannsen was an independent thinker. An editorial preface (p. 331) warned against some of his claims: That Liebig's law of minimum factors is useful in practical plant cultivation (p. 356), that heat is the movement of tiny particles (p. 347), and that the idea of

a "vital force" should probably be banned from science (p. 353). Throughout his career Johannsen was highly active as a popular science writer with clear opinions on controversial topics.

The Aristotelian bent of Johannsen is evident in his popular article on "the soul of plants" ("Om Planternes Sjæl") of 1889. He claims that plants do have a sense of touch ("Berøringsfølsomhed"), exemplified by the mimosa, and thus a soul on the level of the senses (Johannsen 1889: 32). Rephrasing Charles Bonnet he said: "I do not claim to have proved that plants really have consciousness ("Bevidsthed"); but it has certainly not been proven that they do not." (p. 181). He rebuked Aristotle mildly for his sharp division between the plant and animal kingdoms (p. 163), and quoted Claude Bernard in conclusion: "There is only one kind of life, only one physiology for all living beings" (p. 182).[1]

A study of the development of the endosperm in barley was Johannsen's first substantial scientific publication (Johannsen 1884). The search for causes of the difference between "mealy" ("melet") and "glassy" ("glasset") barley grains – the first considered to be of superior quality, was one path leading him toward the general problems of variation and heredity.

By 1895 Johannsen had developed a well rounded general approach to the study of heredity, with Hugo de Vries and Francis Galton as main sources of inspiration. His views were published in a small popular book, *Heredity* ("Arvelighet"), and in the third edition of Eugenius Warming's textbook *General Botany* ("Den almindelige Botanik") where Johannsen wrote the chapters on cytology and physiology.

Heredity in evolution and breeding

It is Darwin's eternal claim to fame that he led the idea of organic evolution to victorious breakthrough, wrote Johannsen, but variation and heredity should now be studied in their own right and not merely as subordinate topics to the evolution of species. "Evolution needs theory of heredity; but not vice versa", he claimed (Johannsen 1896: 12). He recommended the study of pre–Darwinian authors, for instance, the successful practical breeder Louis Vilmorin (p.13). Johannsen presented the normal variation curve of Quetelet and Galton, which described how "the various properties of individuals belonging to a species or race vary around an average" expressing a "type", and went on to the diverging variation curves of William Bateson and Hugo de Vries. A curve with two peaks is with great probability ("stor Sikkerhed") due to a mixture of two types (p. 19). Johannsen stressed the fundamental role of causal processes at the individual level in his discussion of statistical methods in breeding. He warned against "German dogmas" like the law of correlation claiming that certain traits were linked and could not be separated. Statistics present average numbers but "do not necessarily say anything decisive about the individuals" (p. 48–50).

Recent developments in cytology a main input to Johannsen's early thinking on heredity. He referred to the work of August Weismann, de Vries and Oscar Hertwig. Weismann's speculations on the "continuity of the germ–plasm" was essentially a development of Galton's stirp theory. This idea expressed a perspective on heredity and evolution that was basic to Johannsens thinking from the 1890s on. With two simple drawings he illustrated the difference between the Galton–

[1] I am translating from Johannsen's Danish. He gives no reference.

Weismann view on the one hand and the orthodox Darwinian and Lamarckian ideas of pangenesis and inheritance of acquired characters on the other (Johannsen 1896: 77, figure 4). For Johannsen the Galton–Weismann idea represented what Imre Lakatos would have called the hard metaphysical core of a research program that eventually produced an empirically well–founded theory of the genotype, or of "hard heredity" in Mayr's terminology.

Weismann's detailed speculations on determinants and the role of reduction division were firmly rejected by Johannsen as excessively preformationist. Weismann's misleading idea was to "have all possibilities and causes of the later development of the individual compressed into the egg, which accordingly would be at least as complicated in its composition as the fully grown individual" (Johannsen 1896: 73).[2] Like Oscar Hertwig (1894) Johannsen held an epigenetic view. He held that the egg did not contain "germs of all possible *later properties* of the grown individual, but at most certain decisive dispositions ('Anlæg') with respect to the *earliest steps* of development". The implication was that "differences in the sex cells could … be expressed through diverging reactions … to conditions of development introduced at an earlier or later stage."[3] The sound core of Weismannism was an old idea, "first formulated relatively clearly by Galton more than 20 years ago," according to Johannsen (1896: 75).

Inheritance of acquired characters

Johannsen nevertheless emphasized Weismann's importance in stimulating the science of heredity. His criticism of claims about inheritance of acquired characters had generally been very skilful and appropriate, but had its limitations. For instance Weismann's criticism of Brown–Sequard's experiments of producing hereditary epilepsy in guinea pigs was not convincing (Johannsen 1896: 83). In Johannsen's view these experiments of Brown–Sequard, Pasteur's development of an anthrax vaccine, and Schübeler's experiments on reversible hereditary change of barley subjected to arctic and temperate climates gave strong evidence that individually acquired characters could influence heredity directly (p. 91).

For Johannsen a moral implication of such inheritance of acquired characters was that each individual human being had responsibility to future generations for its conduct of life, "a responsibility that would be obliterated by Weismann's teaching!"[4] In particular, Johannsen was thinking of a damaging effect of alcohol on heredity (Johannsen 1896: 93–94).

In *General Botany (Den Almindelige Botanik),* the text–book co–authored with Warming, Johannsen carefully summarized the most recent discoveries about the behaviour of the nucleus and the chromosomes in reduction division and fertilization. Strasburger, Weismann, and de Vries take these results to show that "the nucleus alone is the carrier of hereditary properties."

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[2] The whole sentence in Danish: "Disse oppfattelsers fælles ledende, urigtige Grundtanke er den, at Weismann vil have alle Muligheder for og Aarsager til Individets senere Udvikling pressede sammen i Ægget, som saaledes maatte blive mindst lige saa indviklet sammensat somselve det ferdige Individ."

[3] The whole sentence in Danish: "Antage vi derfor ikke i Ægget særligt existerende Smaakim til alle mulige *senere Egenskaber* hos det voxne Individ, men i det højeste visse afgjørende Anlæg med hensyn til de *allerførste Trin* af Udviklingen, saa vil ikke desto mindre Forskjelligheder mellem Kønscellerne dog kunne yttre sig ved forskjelligartede reaktioner – I dette Ords videste forstand – overfor Udviklingsfaktorene, være sig paa et tidligere eller senere Trin I Udviklingen."

"The idea is seductive and not improbable," commented Johannsen, but it has not been proved (Warming and Johannsen, 1895: 153–154). There is a lively current debate about the importance of inheritance of acquired characters in the transformation of species, wrote Johannsen. "Probably we come closest to the truth by assuming that both natural selection and direct adaptation is causing transformation" (p. 507). Fifteen years later Johannsen and Warming had split radically on this question. Warming had become a convinced neo–Lamarckian and Johannsen a Mendelian selectionist.

Pedigree Breeding

In the mid–1890s Johannsen engaged in breeding high quality barley for brewing. The results confirmed his suspicion that certain Darwinian theories of evolution were an obstacle to plant breeding. For brewing large grain size was generally a positive quality. High nitrogen content was advantageous for some purposes and not for others. The popular idea of correlation, of a tight link between properties, was mistaken in this case, argued Johannsen. Such claims were often supported by population averages, "average numbers of material arbitrarily collected from all over the world," as he wrote. If individual selection was combined with hybridization the possibilities of combining wanted properties in many different ways were much larger than indicated by the "law of correlation". "So let us face our tasks without hesitation and beware of German dogmas!" (Johannsen 1898a: 68–78).[5]

Johannsen pointed to the "theory of heredity and plant breeding before Darwin" (Johannsen 1898b). In particular Louis Vilmorin was a good guide to methods of pedigree breeding and effective use of hybridization as means to increase hereditary variation (Johannsen 1898b: pp. 345–346, 456; 1899: 556). Johannsen cited Hertwig (1898) on the lack of experiments that unequivocally demonstrated the inheritance of acquired characters. However, he also agreed with Hertwig that "a certain degree of heredity for characters developed by external factors" had to be assumed. This phenomenon might be important in the natural evolution of species, but for breeding it was irrelevant: "Nowhere is the difference between theory of evolution and breeding work clearer than precisely in this respect"(Johannsen 1898b: 321).[6]

In 1899 Johannsen presented reduction division and fertilization as the basis for understanding heredity. For instance, in distinguishing variation due to internal and external causes he saw "internal" variation as proceeding "quite independently of external conditions" and due to "the 'imperfect accuracy' of cell division" (Johannsen 1899a: 452–455).[7] Vilmorin's old

[4] The whole paragraph in Danish: "Vi komme altsaa til den Opfattelse, at Individets Livsvilkaar og, hva der naturligvis altid vil staa i Sammenhæng dermed, Individets hele Livsførelse maa kunne faae Indflydelse – om end aldri saa lidt – paa afkommets Beskaffenhed, ogsaa naar vi se helt bort fra Smitteoverførelse. Dette er paa ingen Maade saaledes at forstaa, at en eller anden, være sig god eller slet Egenskab, som man har "erhvervet" sig, just behøver at vise sig hos Afkommet *i samme Skikkelse* eller *i samme Retning*. Men paa forskjellige direkte og indirekte Maader kan en Indflydelse, som den nævnte, tænkes virksom. Individet – det enkelte *Menneske* holde vi os til i dette Øjeblik – faa da i alt Fald et vist Ansvar overfor den kommende Slægt med hensyn til sin Livsførelse, et Ansvar, som efter Weismanns Lære udviskes!"

[5] " – lad os gaa lige løs paa vore Opgaver og vogte os for tyske Dogmer!" (Johannsen 1898a: 78).

[6] "Men ingensteds træder Forskjellen mellem Udviklings–Læren og Forædlings–Arbeidet klarer frem, end netop paa det her berørte Omraade."

[7] "Vi antager altsaa, at der kan skje Variation ganske uafhængig af ydre Kaar og nærmest betinget af Celle–Delingens 'ufuldkomne Nøyaktighet' – om man vil tillade mig Brugen af dette Udtryk."

principle that the "hereditary power" of seemingly similar individuals or races could be very different, and that therefore the breeding value of an individual should judged by the qualities of its offspring rather than by the quality of the individual itself, thus had a cytological basis. The importance of this principle was now being rediscovered (Johannsen 1899a: 542), in Johannsen's own experiments, in the breeding successes of Hjalmar Nilsson at Svalöf, as well as in Hugo de Vries' experiments with hereditary malformations in plants (p. 547).

Johannsen suggested that one reason why Vilmorin's correct idea had been neglected was his mistaken ideas about fertilization. Like his contemporaries Vilmorin thought that a flower was usually fertilized by its own pollen. But we now know that fertilization by foreign pollen is the rule. Because of this a reliable pedigree is often impossible to establish. Special methods like artificial pollination would have to be used for Vilmorin's principle to be generally effective. This was the case for important cross–fertilizing plants like rye and sugar beet. However, for self–fertilizers like peas or barley there was no doubt about the "father" and the principle was directly applicable (Johannsen 1899a: 543; Johannsen 1899c: 170–171). Johannsen had worked mostly with barley, and for his classical selection experiment he picked beans, another self–fertilizer.

The analysis of "variability"

In the fourth edition of *General Botany* the treatment of evolution and heredity was reorganized and substantially expanded. The heading of the twelfth and last section of the book was "Theory of Descent" and its one and only chapter, No. 58, was called "Natural Kinship; Variability, Heredity and Phylogenesis".[8] Starting with a discussion of biological systematics Johannsen noted that traditional Linnean species had turned out to contain numerous varieties or subspecies. Some of these were sufficiently stable to be considered independent systematic "types". In Johannsen's view these "small" species deserved, more than the Linnean "large" species, to be considered the basic systematic units (Warming and Johannsen 1900: 666).

Five kinds of variability ("Variabilitet") were listed (p. 667):

1) The polymorphism of traditional Linnean species.
2) Differentiation of forms characteristic of the progeny of hybrids (this was further discussed in a subchapter on laws of Mendelian segregation, pp. 679–683).
3) "*(I)ndividual* or *fluctuating variability*" in the strict sense. [9]
4) Differences developed under "*divergent external conditions*".[10] (In contrast to de Vries Johannsen wanted to keep a distinction between individual variation and modifications due to the growth environment ("Voksestedsmodifikation"), Johannsen 1902: 558.)
5) So–called *mutations* by which individuals representing new stable forms, species or subspecies, suddenly appeared.

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8 "Naturlig Slægtskab; Variabilitet, Arveligehd og Fylogenese".
9 "Den saakaldte *individuelle* eller *fluktuerende Variabilitet, Variabiliteten i snævrere Forstand*."
10 "De ofte iøjenfallende Forskelligheder, som, endog efter Utsæd af samme Frøprøve, vise sig mellom Individer, der udvikle sig under *stærkere afvigende ydre Kaar*."

The first and fifth kinds, species polymorphism and mutation, were directly related to basic systematic groups, and thus by definition hereditary. The second kind, variation following upon hybridisation, was recognized as capable of producing new stable forms by recombining properties of the parent forms. The fourth kind was not hereditary, or at least only partly. But the third kind, "individual variability", was contentious with respect to its contribution to the formation of new species. Johannsen gave special attention to this third kind, individual variation, defining it as the regularly occurring variation with respect to quantitative measurable properties among closely related individuals belonging to a "subspecies, variety, or cultural form" (Warming and Johannsen 1900: 668).[11]

The stability of biological types

In the mutation theory of Hugo de Vries "small" species did not originate by gradual continuous transitions but through sudden larger or smaller changes, "mutations". On this theory individual variations "had absolutely no significance for the development of new forms", according to Johannsen. And he thought it likely that in the Darwinian tradition individual variation had been overestimated ("overvurdert") with respect to the formation of new types (p. 686–687). By simple investigation of an individual different from the average of the population it was not possible to decide if it represented a different biological type. In particular this was so for quantitative properties. The progeny of the individual had to be studied, wrote Johannsen. If the progeny, on average, has the same character as the parent it represents a mutation, i.e., a different basic systematic class. But if the progeny regresses toward the mean of the population we have to do with an "individual variation" (Warming and Johannsen 1900).[12]

Thus the purpose of Johannsen's classical selection experiment on beans[13] was to test the law of regression as formulated by Galton and his followers Karl Pearson and Frank Raphael Weldon, using Vilmorin's method of analysing the offspring. Johannsen bought sixteen pounds of Princess beans, "well developed" and of "uniform" quality, grown in Fyn in 1900 (Johannsen 1903: 15). In *General Botany* we find a table showing correlation between width and length of 12000 beans (Warming and Johannsen 1900: 669). This analysis was presumably part of preparations for the first planting made in the spring of 1901.[14]

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[11] "Herved forstaas det Forhold at Individerne af en given, snævrere Slægtskabskreds (Underart, Afart, Kultrurform), 'variere' regelmæssigt med hnesyn til de Egenskaber, som overhovedet kunne udtrykkes i Tal, d.v.s. kunne grupperes omkring typiske Værdier paa lovbestemt maade."

[12] "Først Afkommeets Forhold kan afgøre Sagen: faar Afkommet, gjennomsnitlig set, samme Præg som de paagædende Individ, da var det Mutation: men slaar Afkommet, gjennomsnitligset, tilbage mot den oprindelige Type, da var det det individuell Variation hos det nævnte Individ. Man forstaar heraf den store principielle Btydning, som en gennemført særskilt Undersøgelse af det enkelte Individs Afkom har for Arvelighedslæren og for Planteforædlingen."

[13] Published in 1903 as "On heredity in Populations and Pure Lines" ("Ueber Erblichkeit in Populationen und in reine Linien").

[14] The preface of Warming and Johannsen 1900 is dated June 1901.

The bean selection experiment and the Biometricians' reaction

The sharp distinction that de Vries had made between the concepts of "statistical variation" and "mutation" was undoubtedly a substantial step forward, wrote Johannsen in a short "preliminary review" of *The Mutation Theory* (Johannsen 1901: 8).[15] The central question of the bean experiment was apparently sharpened also by Johannsen's reading of Weldon's review in *Biometrika.*

Till now, wrote de Vries in the preface to *The Mutation Theory*, the origin of species has been studied by comparative methods. It has been the general opinion that this phenomenon is inaccessible to direct observation and at least to experiment (de Vries 1901: p. III).[16] It was the ambition of de Vries to change this. But Weldon's review was very critical: Firstly de Vries had misunderstood Galton's law of regression, and secondly his results fitted perfectly with Pearson's interpretation of the law. According to Weldon the successive increase and decrease of average number of seed rows on corn heads through selection formed "a fairly conclusive proof" that the theory of mutations was mistaken (Weldon 1902: 369).

Weldon put the burden of proof squarely on his opponents: "A clear proof that Professor Pearson's view of the facts of regression is wrong … is absolutely essential …"[17] With a high degree of self confidence Weldon dismissed the attempts of de Vries and Bateson "to distinguish between 'variations' and 'mutations', or between 'normal' and 'differential' variations". Their attempts rested on a view "which a little knowledge of the statistical theory of regression will show to be wholly imaginary" (Weldon 1902: 374). This was the challenge that Johannsen picked up in his bean experiment.

In a 1902 paper on "The plurality of forms of organisms" ("Organismernes Formrigdom") Johannsen further developed his views on the nature of "variability" with special focus on "individual variability". The general question was whether hereditary change in the evolution of species is caused by the direct influence of living conditions, independently of selection, or through mutations (Johannsen 1902: 548). In a note at the end Johannsen remarked that recently the British "biometric" school headed by Pearson had mercilessly revealed the deficiencies in the arguments of Bateson and de Vries. Johannsen nevertheless had no doubt that mutations do occur, though the border between mutations and individual variation might be less sharp than they thought. "Presumably research of the near future will bring some clarification", he added optimistically (p. 565).[18]

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15 "Denne skarpe Adskillelse mellem Begreberne statistisk Variation og Mutation er utvivlsomt en stor Vinding, og med Mutationslærens Sprog kan man godt som de Vries, sige om en given Underart, at den er 'völlig konstant und höchst variabel': Typen er fast, men Svingningen om Typen ere sterke."

16 The first paragraph runs as follows: "Die Lehre von der Erstehung der Arten ist bis jetzt eine vergleichende Wissenschaft gewesen. Man glaubt allgemein, dass dieser wichtige Vorgang sich der Beobachtung und mindestens der experimentellen Behandlung entziehe."

17 The whole sentence: "A clear proof that professor Pearson's view of the facts of regression is wrong although it is in accord both with the theory of chance and with the results of the numerous statistical studies of inheritance which he and his pupils have made during the past seven years, is absolutely essential, if the view held by Professor de Vries is to be maintained."

18 "Jeg nærer dog aldeles ingen Tvivl om, at Mutationer forekommer, særlig har de Vries Kulturer overbevist mig. Men muligens er Grænsen mellom utvivlsom Mutation og blot og bar individuell Variation næppe altd saa skarp som de Vries og Bateson er tilbøjelige til at antage. Ventelig vil den nærmeste fremtids Forskning her bringe mere Klarhed tilveje."

Results of the bean selection experiment

Analysis of the harvest of the second planting, in 1902, brought much clearer results than Johannsen had expected. It turned out that within each of the types that he had isolated as pure lines[19] there was no hereditary effect of selection. In other words, there appeared to be complete regression to the type of the line. If Johannsen's interpretation was tenable this was just the kind of disproof of the biometric law of regression that Weldon had asked for.

The results appeared so clear cut and convincing, as well as important for the study of heredity, that Johannsen found it right to publish after only two growing seasons. The limited task of this publication, wrote Johannsen in the concluding discussion, is only "to throw light on the Galtonian regression between parents and children" (Johannsen 1903: 64). He had used a material of similar nature to that of Galton, namely beans instead of sweet peas. Like Galton he measured seed in successive generations. But in contrast to Galton he also introduced the pedigree principle. He traced the offspring of each individual first generation parent bean and thus made it possible to test for the presence of distinct types within the population. Galton had taken the whole population to represent a biological unity while Johannsen found that it consisted of many distinct and stable biological types. For the population as a whole Galton's law of regression was confirmed, but within each pure line it did not hold. The hereditary type of each line was stable, claimed Johannsen, completely unaffected by selection (Johannsen 1903: 57).[20] Thus he had shown that de Vries was right in claiming constancy for the elementary biological types. The Biometricians' law of regression had been refuted in so far as it was taken to imply a continuous change of heredity. Within pure lines of self–fertilizing plants like peas or beans "individual variations" were not hereditary.

Not surprisingly this conclusion did not please Pearson and Weldon. Their reviews in *Nature* and *Biometrika* brushed off Johannsen's results in much the same way as Weldon had rejected de Vries experiments the year before: Johannsen had misunderstood the law of regression and in fact his results were a confirmation of it (Pearson 1903; Weldon and Pearson 1903).

Johannsen's controversy with the Biometricians stimulated his development of the concepts of "genotype", "phenotype" and "gene" which were first introduced as the basis of a comprehensive theory in his 1909 German textbook (Johannsen 1909: 130). But this is another story (Roll–Hansen 1989). The purpose of this paper has only been to trace the history and background of Johannsen's concept of stable elementary biological types, leading up to his classical selection experiment of 1901–1902.

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19 A "pure line" as defined by Johannsen is simply the offspring in successive generations from one single parent. With a regularly self–pollinating organism like beans this was easy to achieve and effects of hybridisation in earlier generations would also be minimal.

20 "Indem ich aber nicht dabei stehen blieb, die Populationen als Einheiten zu betrachten, sondern mein Material in seinen 'reinen Linien' auflösen konnte, hat es sich in allen Fällen gezeigt, *dass innerhalb der reinen Linien der Rückschlag sozusagen vollkommen gewesen ist: die Selektion innehalb der reinen Linien hat keine Typenverschiebung hervorgerufen*".

Cloncluding remarks

The role of pedigree selection in Johannsen's early work on heredity is striking. Extensive discussions of the implications of recent cytological discoveries for theories of heredity are also clear evidence of his interest in causal factors and processes at the level of the individual organism. His recurring illustration of the Galton–Weismann stirp versus the Darwin–Lamarck pangenesis model for transmission of heredity fits this interpretation of his theory of heredity.

Thus it appears a mistake when Frederick Churchill interprets Johannsen's concepts of phenotype and genotype as primarily statistical when introduced in 1909. According to Churchill "Johannsen was a chemist and statistician first", and his thinking was basically statistical and "vertical". It started from the description of population averages, "phenotype", and positing "genotype" as a theoretical entity with no commitment in cytology. Only later did Johannsen, according to Churchill (1974: 28–30), come around to the cytologically based "horizontal analysis" that was characteristic of mature classical genetics, and as it was formulated for instance by the Drosophila school of T.H. Morgan around 1915. I believe the preceding account shows that "horizontal analysis" examining "directly the genetic composition sundered either conceptually or physically from the *Erscheinungsphenomena*" (Churchill 1974: 30) was an essential part of Johannsen's research project on biological heredity from the beginning. This approach was well founded by 1895 and lead up to his 1903 paper and the genotype–phenotype distinction of 1909.

Garland Allen, referring to Churchill, also claims that "Johannsen's eye was focused on the entirety of a population and its range of variation. He was not concerned about distinguishing genotypic from phenotypic variation within the individual organisms, but only within large populations" (Allen 1979: 198). This is a description that fits the biometric view well but seems quite opposed to that of Johannsen. Ernst Mayr in his 1982 book, *The Growth of Biological Thought*, still takes Churchill's paper to be authoritative on Johannsen's view of the genotype. When William Provine (1971: 96–97) found the Pearson–Weldon criticism of Johannsen's selection experiment to be "powerful", and held that its neglect by the biological community in general was not justified, the reason may be a similar misinterpretation of Johannsen.

To sum up: It seems that the standard interpretation of Johannsen, as promoted for instance by Mayr, Churchill, Allen and Provine, fails to grasp the difference between him and the biometricians.

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Inheritance of Acquired Characters: Heredity and Evolution in Late Nineteenth-Century Germany

Wolfgang Lefèvre

INTRODUCTION

The assumption of inheritance of acquired characters was a widely shared assumption up to the first half of the twentieth century. Today, inheritance of acquired characters is generally dismissed by biologists. More precisely, it is regarded as being definitely ruled out by the central dogma of molecular genetics as established by Francis Crick in 1958 (Crick 1958): DNA makes RNA makes protein, and never the other way around. Although, probably, little risk would be taken by predicting further revivals of inheritance of acquired characters, in this paper, I will regard the history of inheritance of acquired characters as a closed one.

Conway Zirkle traced back this history to classical antiquity, and one can be sure that the assumption of inheritance of acquired characters is far elder than any written record. However, Zirkle's study documents not only how broadly this assumption was held in all historical periods since antiquity. It also testifies that none of these periods lacked critical voices that doubted or even plainly dismissed inheritance of acquired characters. By this, Zirkle's study shows thirdly, and probably involuntarily, that inheritance of acquired characters was an indifferent assumption, that is, an assumption that could be held and challenged without raising the need, neither on part of its adherents nor of its critics, to subject it to serious tests.

It was no simple task to decide definitely whether or not acquired characters can be transmitted. This became clear when the indifferent status of inheritance of acquired characters ended in the last decades of the nineteenth century and serious efforts were made to prove its groundlessness. "There are three ways," wrote Ernst Mayr, "to refute an inheritance of acquired characters. The first is to show that the mechanisms by which it is supposed to operate are impossible. This was primarily Weismann's approach. There is nothing in the structure and division of cells that would make an inheritance of acquired characters possible. […] A second way to refute an inheritance of acquired characters is by experiment. […] Beginning with Hoffmann and Weismann, such experiments were conducted up to the 1930s and 40s and the results were uniformly negative. The third way of refuting the theory of the inheritance of acquired characters is to show that the phenomena that are claimed to *require* the postulate of an inheritance of acquired characters can be explained equally well or better on the basis of the Darwinian theory. Much of the evolutionary literature of the 1920s, 30s, and 40s was devoted to this third approach" (Mayr 1982, 699ff.).

With this summary, Mayr conjures up the theoretical developments before the age of molecular genetics that undermined inheritance of acquired characters step by step. The probably most important, if not even revolutionary, of these developments consisted in the devaluation of the role of the parental organism in the biological transmission process. This role was reduced to that of a mere conveyer and re-combiner of an inheritance that predates it. From different quarters, Francis Galton's "ancestral line" and August Weismann's "continuity of the germ

plasm" (*Keimplasma*) laid the ground for an understanding of the biological transmission process that left little space, if any at all, for inheritance of acquired characters.

That this would be the final outcome, was of course in no way clear in the 1880s when these developments started. At this point of time, it was as open a question as in the past whether or not inheritance of acquired characters can be asserted or must be denied. Yet, what had apparently changed then was the indifferent state of this issue. Now there were biologists like Weismann who did no longer regard inheritance of acquired characters a matter of individual convictions but wanted the question to be answered definitely. One may ask therefore what did transform the assumption of inheritance of acquired characters which had be held so long despite doubts and even complete rejections, into one that must be proved or dismissed? What did lead to an end of the indifferent state of this issue? Or, to put it differently, why did the assumption of inheritance of acquired characters become an urgent point on the agenda of biology in the 1880s? Ernst Mayr, pondering Weismann's motives for his sudden turn against inheritance of acquired characters, indicated an answer that seems worth to be elaborated. He wrote that it is not clear "whether Weismann had first become convinced of the invalidity of the theory of an inheritance of acquired characters and then adopted the germ-track theory or vice versa. The fact is that he already cites in his 1883 paper [Weismann 1883] so many lines of argument against soft inheritance that one can well imagine that this general conviction preceded the proposal of a specific mechanism. This interpretation is strengthened by the fact that Weismann was a strict selectionist already in the 1870s and presumably had simply no need for an additional mechanism" (Mayr 1982, 701). With the last sentence, Mayr draws our attention to the fact that, since the 1870s, the issue of heredity played a significant role in the debate about evolution. It has, indeed, the appearance that inheritance of acquired characters got then a new state because of its bearing for the understanding of Darwin's theory of evolution.

The goal of my paper is to shed some light on exactly the connection between inheritance of acquired characters and evolution as established by the historical actors in the last quarter of the nineteenth century. However, rather than offering a narrative of the debates by which this connection was brought about by the historical actors, the paper's focus will be on an analysis of the strategic significance inheritance of acquired characters had within the contemporary framework of competing theories of evolution. In a way, what I present could be regarded a kind of a "rational reconstruction" of this significance, a reconstruction, however, that does not rest on an universal logic of scientific reasoning but on the internal "logic" of the historical theories involved. My paper is ordered as follows: In a very short first part, Darwin's own relation to inheritance of acquired characters is addressed. In part II, the bearing is discussed inheritance of acquired characters had for the understanding of evolution. In part III, this discussion is exemplified through a sketch of Ernst Haeckel's Neo-Lamarckian theory of evolution. In the fourth part, finally, the significance of inheritance of acquired characters for conceptions of evolution is shown from a different point of view, namely by citing two contemporary alternatives to Haeckel's theory of evolution that dismissed inheritance of acquired characters.

I. Darwin and inheritance of acquired characters

Today, the assumption of inheritance of acquired characters is equated with a Neo-Lamarckian approach to evolution. However, this assumption was neither first conceived nor given a specific meaning by Jean Baptist Lamarck or one of the known Lamarckians. Rather, as already said, it was a widely shared, though also periodically challenged, assumption from times immemorial up to the end of the nineteenth century and beyond. Against this background, it is in no way remarkable that Charles Darwin himself assumed an inheritance of acquired characters.

Darwin devised an elaborate theory of heredity, his renowned or notorious "Pangenesis" theory, and published it in the second volume of *The Variation of Animals and Plants under Domestication* in 1868. Although all of his assumptions on heredity were dismissed by the subsequent developments of biologists' understanding of the transmission process, and although not at all original as regards its central physiological hypothesis, Darwin's theory must be regarded a landmark in the history of heredity theories. For it constituted the very first attempt at a really comprehensive elucidation of the transmission process, based, as is typical of Darwin, on an abundance of empirical material. It is still a first source if one wants to know what nineteenth-century biologists, breeders, physicians etc. knew or assumed about heredity.

At the centre of his theory is the supposition and explanation that and how every "unit" of parental organisms contributes to the formation of the sexual products of the two sexes that constitute the germ when united and contain all that is bequeathed to the offspring. Assuming in this way a direct dependency of the hereditary material on all parts of the parental organisms, this theory entails inheritance of acquired characters quite naturally. If Darwin, on the base of this theory, had wanted to exclude inheritance of acquired characters, he would have been forced to invent a suitable complementary hypothesis. But with inheritance of acquired characters in its indifferent state characterized above, Darwin felt no need to dismiss this assumption. On the contrary, his theory of evolution could even take advantage of inheritance of acquired characters and did so in fact occasionally.

There is a long tradition of criticizing Darwin for being not enough a Darwinian but too much a Lamarckian evolutionist. I will not go into this judgment that obviously lacks a historical spirit.[1] Instead, I want to state the remarkable fact that Darwin, despite his assumption of inheritance of acquired characters, based his theory of evolution not on Lamarckian adaptations but on random variations. It was probably his familiarity with the experiences of professional breeders what prevented him from a Lamarckian approach. The fact that Darwin didn't built upon Lamarckian adaptations needs clarification, and the following section will try to provide it.

II. Inheritance of acquired characters and evolution

Looking back to the first decades after the appearance of Darwin's *On the Origins of Species* in 1859, one gets the impression that it took a considerably long span of time before biologists came to terms about what constitutes the very core of Darwin's theory of evolution. This core became conceivable, or one may even say: was shaped, in a clarification process in which alternative understandings of evolution emerged. Lamarckism or Neo-Lamarckism[2] was one of those

[1] For a brief discussion of Darwin's theory that places it historically, see (Bowler 1989) 54ff.

alternatives, which contributed a lot to a more adequate understanding of what distinguishes Darwin's theory from other conceptions of evolution. Ironically, Neo-Lamarckism became first such a clarifying alternative when Weismann attacked inheritance of acquired characters, one of its central elements.

It was a shared presupposition of both, Darwinism[3] and Neo-Lamarckism, that the historical evolution of plant and animal forms was essentially a result of adaptations to changes in the conditions of life in the course of the historical development of the Earth's surface. That is to say, both, Darwinism and Neo-Lamarckism, regarded evolution as a process of such adaptations. And it was this shared presupposition by which Darwinism and Neo-Lamarckism distinguished themselves from conceptions of evolution that assumed an inner-organismic formative drive as motor of evolution. Darwinism and Neo-Lamarckism also shared the presupposition that changed conditions of life do not cause directly adaptive modifications of organisms.

However, as is well known, Neo-Lamarckians conceived of changed conditions of life as an indirect cause of adaptive modifications. They assumed that organisms react on such changes by changes of their behaviour; that such behavioural changes entail new uses of their organs; that new use, or for that matter disuse, of organs leads to physical modifications of organs and structure; and, finally, that such modifications are transmitted to the offspring and engender, in the course of the succession of generations, modified organisms that are better adapted to the new conditions of life. In our collective memory, the elongated neck of giraffes stands for this Lamarckian explanation, which I will call adaptation by use and disuse hereafter. As is obvious, this explanation of adaptation by use and disuse stands and falls with the assumption of inheritance of acquired characters. This assumption, thus, was rightly equated with a Neo-Lamarckian conception of evolution because the latter cannot be thought without the former.

For Darwinians, too, conditions of life have a share in the development of adapted organisms. It is exactly these conditions that assign adaptive value to organismic modifications and, thus, prove to be ultimately responsible for the selection of variations in the "struggle for existence." However, Darwinism does not presuppose that variations of an adaptive value are directly or indirectly induced by new conditions of life. Rather this conception of evolution assumes that "natural selection" acts upon variations that are essentially random. It presupposes no other causes for variations of an adaptive value than for variations of no such value. In other words, Darwinism does not presuppose any correlation between the (then) unknown causes of variation and the demand for adaptation to changed conditions of life. Variations of an adaptive value are random in that their coming into being has nothing to do with this value.[4]

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2 The label "Neo-Lamarckism" was coined by contemporaries — see, for instance, (Wagner 1908) 121ff. For the problems that made Lamarckism periodically, and not only before 1900, attractive, see (Beurton 2001).

3 Already his contemporaries labelled Charles Darwin's theory of evolution "Darwinism." — Notwithstanding the uncountable modifications induced in this theory by biologists during the past 145 years, its core – speciation on the base of minimal random variations and natural selection — also constitutes the core of modern Darwinism. Modern Darwinism is to Darwin's theory as classical mechanics is to Newton's theory: Both cannot be found in the writings of their respective patron saint and owe to him nevertheless their decisive conceptual features.

4 For the development of Darwin's understanding of variation, see chapters 3 and 4 of (Vorzimmer 1972). For ambiguities of Darwin's understanding of variation, see chapter 3 of (Gayon 1998).

Against this background, it becomes understandable why radical challenges of inheritance of acquired characters like Weismann's didn't strike Darwinism although Darwin, like most of the contemporary biologists, assumed this inheritance. As became clear in the course of the debate, Darwinism did not stand and fall with inheritance of acquired characters because its explanation of evolution built upon random variations and not on adaptations by use and disuse. In other words, it was Weismann's attempt at a definite dismissal of inheritance of acquired characters that clarified that Darwin's Pangenesis theory was not an integral part of his theory of evolution. As regards the issue of heredity, this theory presupposed only something well-confirmed by all breeding experiences, namely that, as a matter of fact, hereditable variations exist. This simple fact did neither imply nor suggest nor exclude any specific theory of heredity. It had been compatible with the assumption of inheritance of acquired characters, as Darwin's Pangenesis shows, and proved to be in no way at odds with the new understanding of heredity that began to surface with Galton's statistical theory of heredity and Weismann's (and others') cytological distinction between germ and soma plasm. No additional efforts were needed to reconcile it with the understanding of heredity as a process in which a hereditary substance is transmitted and recombined by the parental organisms that remains unchanged by modifications these organisms may undergo. While the development and elaboration of this new understanding of heredity was to undermine inheritance of acquired characters and, thus, the *raison d'etre* of Neo-Lamarckism, it constituted no challenge to a Darwinian theory of evolution that presupposed random variations.

The differences between Darwinism and Neo-Lamarckism with respect to the evolutionary significance of inheritance of acquired characters discussed here are clear for us in hindsight, that is, after nearly 150 years of debates about evolution. But they were far from being clear for biologists in the last decades of the nineteenth century. As already said, the clarification of what is essential for Darwinism and what not took shape only in fierce arguments about evolution which were characteristic of the first 60 years after the appearance of *The Origin of Species*. Particularly in Germany in the 1870s, when evolution was already accepted by a lot of biologists, and probably by the majority of the younger ones, the discussed differences between a Darwinian and a Neo-Lamarckian understanding of evolution were almost not recognizable. For, here, this understanding was dominated by a special theory, that of Ernst Haeckel.

III. Inheritance of acquired characters in Haeckel's theory of evolution

Haeckel presented the understanding of evolution he had arrived at under the impression of Darwin's *Origins* already in 1866, namely in his *Generelle Morphologie*. This was a book for professional biologists, particularly for comparative anatomists. Two years later, under the title *Natürliche Schöpfungsgeschichte*, he published essentially the same theory of evolution in a fashion apt for addressing a broader audience. This book shaped the initial view of Darwinism in Germany, not only that of interested non-experts but that of experts as well.

Haeckel's understanding of evolution was embedded in a fundamental theory of the formation of organismic structure. According to this theory, organismic structure results from and is determined by the mutual action of two general "physiological functions," that is, the mutual action of inheritance (*Vererbung*) und adaptation (*Anpassung*) (Haeckel 1866, II 167f.).

On this base, Haeckel distinguished two kinds of a formative drive (*Bildungstrieb*) that bring about organismic structure: an inner formative drive (*innerer Bildungstrieb*) that determines an organism's development from within according to inherited dispositions; and an exterior formative drive (*äußerer Bildungstrieb*) that modifies its development in reaction to the outer conditions of life (Ibid. 168). The mutual action of "Vererbung" and "Anpassung" also constitutes the physiological base of evolution: "Die ganze unendliche Mannichfaltigkeit der organischen Formen wird also in letzter Instanz lediglich durch die Wechselwirkung dieser beiden physiologischen Functionen, der Anpassung und der Vererbung hervorgebracht" (Haeckel 1866, II 169). Natural selection, "der Kampf ums Dasein," on the other hand, is understood by Haeckel as "Summe der besonderen Verhältnisse, unter denen diese Wechselwirkung überall stattfindet, und von denen sie in hohem Maasse begünstigt wird" (Ibid.). In Haeckel's view, this conception rendered adequately the basic conception of Darwin's theory of evolution ("Grundgedanke(n) von Darwin's Selections-Theorie." — ibid. 167). It may be obvious, though, that the problems Haeckel's conception tried to solve were not those of Darwin.

With respect to what was discussed so far, one particularly striking feature of Haeckel's theory of evolution is the inferior role individual variations play in its frame, that is, the inferior role of exactly that kind of variation on which Darwin had based his theory. For Haeckel, individual variation (*individuelle Abänderung* – ibid. 202) is just one out of three instances of what he called "indirecte oder potentielle Anpassung" (ibid.), an adaptation that was taken to be of less significance for the formation of structure than the "directe oder actuelle Anpassung" (Ibid 207ff.). The latter is nothing else than adaptation by use and disuse. Adaptations by use and disuse, not individual random variations, form the centre of Haeckel's theory of evolution. Having above all the animal kingdom in mind as may seem natural for a comparative zoological anatomist, Haeckel assigned particular importance to the active behaviour with which living beings respond to changing conditions of life:

> Indem sich der thierische Wille den veränderten Existenzbedingungen durch andauernde Gewöhnung, Uebung u. s. w. anpaßt, vermag er die bedeutendsten Umbildungen der organischen Formen zu bewirken. (Haeckel 1868, 190)[5]

It is, thus, hardly possible to miss the central role that adaptation by use and disuse played in Haeckel's theoretical edifice. However, it could play this role only when inheritance of acquired characters was taken for granted. And indeed, Haeckel did take inheritance of acquired characters as an established fact.

In his *Generelle Morphologie*, Haeckel dedicated a sizable chapter of nearly 140 pages to the issue of evolution.[6] About twenty pages of this chapter deal with the issue of heredity.[7] In the

[5] The stress Haeckel laid on volition has an almost personal tinge as may be taken from the following passage: "Der Umfang meiner ganz ungeübten Oberarme hatte sich innerhalb eines Zeitraumes von anderthalb Jahren durch fortgesetzte energische Turn-Uebungen fast genau verdoppelt. Dieses enorme Muskelwachsthum und die damit verbundene Uebung der Willens-Vorstellungen wirkte nun mächtig zurück auf die übrigen Vorstellungen meines Gehirns und insbesondere auf diejenigen des Denkens. Ihnen verdanke ich zum grossen Theile (zum großen Theile allerdings auch anderen cumulativ einwirkenden Ursachen), daß die in meinem Gehirne vorherrschenden dualistischen und teleologischen Irrthümer immer mehr den monistischen und causalen Vorstellungen wichen und ihnen zuletzt vollständig das Feld liessen" (Haeckel 1866, II 213).

centre of his argument, one finds the distinction between "conservative" and "progressive" inheritance, and both kinds of inheritance are given the form of a law. The law of conservative inheritance reads:

> Jeder Organismus vererbt dieselben morphologischen und physiologischen Eigenschaften auf seine Nachkommen, welche er selbst von seinen Eltern und Vorfahren ererbt hat. (Haeckel 1866, II 178)

And the law of progressive inheritance reads:

> Jeder Organismus vererbt auf seine Nachkommen nicht bloss die morphologischen und physiologischen Eigenschaften, welche er selbst von seinen Eltern ererbt, sondern auch einen Theil derjenigen, welche er selbst während seiner individuellen Existenz durch Anpassung erworben hat. (Ibid. 178f.)

Subsequently, a couple of more specific laws follow for each of the two kinds of inheritance. In case of conservative inheritance, these specific laws concern 1.) continual inheritance, 2.) interrupted inheritance (traits are not expressed in each generation), 3.) sexual inheritance (the expression of so-called secondary sexual characters), 4.) mixed inheritance (blending of non-sexual characters), and 5.) abbreviated inheritance (succession in which inherited characters show up in ontogenesis). How interesting ever these laws of conservative inheritance may be, in our context, the specific laws of progressive inheritance are naturally of particular concern. These laws are the following:

> 6. Gesetz der angepassten oder erworbenen Vererbung: Alle Charactere, welche der Organismus während seiner individuellen Existenz durch Anpassung erwirbt, und welche seine Vorfahren nicht besassen, kann derselbe unter günstigen Umständen auf seine Nachkommen vererben. (Ibid. 186)

> 7. Gesetz der befestigten Vererbung: Alle Charaktere, welche der Organismus während seiner individuellen Existenz durch Anpassung erwirbt, und welche seine Vorfahren nicht besassen, werden um so sicherer und vollständiger auf alle folgenden Generationen vererbt, je anhaltender die causalen Anpassungs-Bedingungen einwirkten, und je länger sie noch auf die nächstfolgenden Generationen einwirken. (Ibid. 187)

> 8. Gesetz der gleichörtlichen Vererbung: Alle Organismen können die bestimmten Veränderungen irgend eines Körpertheils, welche sie während ihrer individuellen Existenz durch Anpassung erworben haben, und welche ihre Vorfahren nicht besassen, genau in derselben Form auf denselben Körpertheil ihrer Nachkommen vererben. (Ibid. 188)

> 9. Gesetz der gleichzeitlichen Vererbung: Alle Organismen können die bestimmten Veränderungen, welche sie zu irgend einer Zeit ihrer individuellen Existenz durch Anpassung erworben haben, und welche ihre Vorfahren nicht besassen, genau in derselben Lebenszeit auf ihre Nachkommen vererben. (Ibid. 190)

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6 Chapter 19 of the second volume with the title "Die Descendenz-Theorie und die Selections-Theorie" — (Haeckel 1866) II 148-286.

7 (Haeckel 1866) II 171-190; in (Haeckel 1868), see the lectures 8 and 9.

If one asks on which empirical material or what kind of investigations Haeckel rested these laws, one may be surprised to find that he thought it sufficient to add to each of them merely some remarks. To the first and most fundamental law of progressive inheritance, for example, he added the following assertions and remarks:

> Gleichwie alle von den Voreltern ererbten, so können auch alle neu erworbenen Eigenschaften der Materie durch die Vererbung fortgepflanzt werden. Es giebt keine morphologischen und physiologischen Eigenthümlichkeiten, welche das organische lndividuum durch die Wechselwirkung mit der umgebenden Aussenwelt erwirbt, mit einem Worte keine "Anpassungen", welche nicht durch Vererbung auf die Nachkommenschaft übertragen werden könnten. Dieses grosse Gesetz ist von der höchsten Wichtigkeit, weil darauf unmittelbar die Veränderlichkeit der Arten, die Möglichkeit, dass verschiedene neue Species aus einer vorhandenen hervorgehen, beruht. Wir kennen in der That keine einzige, in die Mischung, Form oder Function des Organismus eingreifende Veränderung, welche nicht unter bestimmten (uns gewöhnlich ganz unbekannten) Verhältnissen auf wenige, oder auf viele Generationen hinaus vererbt werden könnte. Am leichtesten geschieht dies, wenn die Veränderung sehr langsam und allmählich erfolgt (wie z. B. bei Erwerbung chronischer Krankheiten, die viel leichter als acute vererbt werden). Am schwersten dagegen tritt die Vererbung der Veränderung ein, wenn die letztere ganz plötzlich (z. B. traumatisch) erfolgte.[8] Gewöhnlich springen die Fälle, wo eine plötzlich aufgetretene Veränderung auf eine oder mehrere Generationen vererbt wird, sehr deutlich dann in die Augen, wenn die betreffende Veränderung eine "monströse" ist, d. h. einzelne Theile des Organismus in ungewöhnlicher Zahl, Grösse, Form oder Farbe entwickelt zeigt, so z. B. die Fälle, in denen sechs Finger an jeder Hand mehrere Generationen hindurch beim Menschen vererblich blieben, ferner die berühmten Stachelschwein-Menschen aus der Familie Lambert, wo eine eigenthümliche schuppenähnliche monströse Hautbildung von Edward Lambert an (1735) sich durch mehrere Generationen auf die Nachkommen vererbte, und zwar bloss auf und durch die männlichen Nachkommen. Auch die häufigen Fälle von erblichem Albinismus gehören hierher, ferner die Fälle, wo ein einzelner Schafbock oder Ziegenbock mit keinem oder mit 4 - 8 Hörnern geboren wurde, und nun diesen individuellen Charakter auf seine Nachkommen übertrug.
>
> Viel wichtiger, als diese monströsen, auffallend vortretenden Abänderungen, welche durch die angepasste Vererbung übertragen werden, sind die unscheinbaren und geringfügigen Abänderungen, welche erst im Laufe von Generationen durch Häufung und Befestigung ihre hohe Bedeutung für die Umbildung der organischen Form erhalten. Die gesammten Vorgänge der künstlichen Züchtung liefern in dieser Beziehung für das Gesetz der angepassten Vererbung eine lange Beweiskette. (Ibid. 186f.)

That's all he regarded necessary for making his case.

When assessing these remarks, one has certainly to allow for the fact that Haeckel was first of all a comparative anatomist and not a physiologist or cytologist.[9] One has, furthermore, to take into account that he could obviously expect his — professional! — audience to accept such

[8] "Gewöhnlich werden bekanntlich traumatische oder durch Verwundung entstandene Veränderungen nicht vererbt. Um so wichtiger ist es, die Fälle aufzubewahren, in denen dies doch bisweilen geschieht. So wurden kürzlich, wie mir Herr Hofrath Stöckhardt als sicherer Gewährsmann mittheilte, auf einem Gute in der Nähe von Jena mehrere schwanzlose Kälber geboren, deren Vater der Schwanz beim unvorsichtigen Zuschlagen eines Thores eingeklemmt und abgequetscht worden war." (Original note by Haeckel.)

cursory expositions. This proves again that the issue of inheritance of acquired characters was still in its indifferent stage described earlier.

However, as a consequence of this, Haeckel unknowingly ran a high risk in treating the assumption of inheritance of acquired characters that cursory, although this assumption had strategic significance for his theory of evolution. Involuntarily, he contributed by this assumption's promotion to an urgent topic of biological thought in a twofold way: as a strategic element of a prominent theory that stands and falls with it; and, at the same time, as a weakly defended element that suggested itself for being challenged by critics of this theory. In any case, in Germany, the sustained challenge of the old assumption of inheritance of acquired characters that led eventually to its dismissal began in the 1880s with challenges of Haeckel's theory of evolution. Two extremely different such challenges may briefly be addressed in the final part of this paper.

IV. Dismissals of an inheritance of acquired characters

a. August Weismann

It is probably not possible to miss the advantages a Neo-Lamarckian theory of evolution would have if it only were compatible with the body of approved biological knowledge, and particularly with genetics. If one could, like Haeckel, presuppose inheritance of acquired characters and, based on it, adaptation by use and disuse as the centre of evolution, it would no longer be a matter of chance whether modifications of structure develop that are adapted to changed conditions of life. Rather, such modifications would be induced just by those changed conditions through the organism's reaction they provoke. In contrast to this, a Darwinian theory of evolution may appear rather implausible and unintuitive. Regarding the historical development of organismic forms as result of natural selection of random variations, this theory demands to embark in an evolution without guarantees and safety net. It comes therefore without surprise that the idea of a historical evolution of species had first success, not in a Darwinian, but in a Neo-Lamarckian fashion.[10] The rapid victory of the idea of evolution among German biologists since the late 1860s was due exactly to the fact that not a Darwinian theory had been presented to them. What Haeckel successfully offered as Darwinism was, as is clear in hindsight, a Neo-Lamarckian theory of evolution.

What is surprising against this background, is the fact that there were at all biologists who read Darwin's theory differently and attacked exactly the Neo-Lamarckian elements of Haeckel's theory. The best known of these biologists is certainly August Weismann. It is, however, not clear to me what made him start his critique of Haeckel and try to restore a "pure" Darwinism that seems to be purer than that of Darwin himself. At least his contemporaries conceived of it as a radically new theory of evolution and called it Neo-Darwinism. The centrepiece of Weismann's critique was his categorical dismissal of inheritance of acquired characters. This rejection and its cytological underpinning count rightly as a decisive step towards modern genetics.[11] Instead of elaborating this well-known story, I want to point to a less known contemporary challenge to

[9] The physiological theory of heredity he designed nevertheless in later years, his "Perigenesis" hypothesis (Haeckel 1876), reminds, all differences notwithstanding, of Darwin's Pangensis in that the Perigenesis, too, entails inheritance of acquired characters by implication. See (Stubbe 1965) 156ff.

[10] For the success of non-Darwinian theories of evolution in the last decades of the nineteenth century, see (Bowler 1983) and (Bowler 1988).

Haeckel's theory, namely two orthogenetic theories of evolution[12] which also dismissed inheritance of acquired characters.

b. Orthogenesis

A key to an adequate understanding of Haeckel's theory of evolution can be seen in the fact that he developed it in the frame of his *Generelle Morphologie*. From its very beginnings, his conception of evolution was part and parcel of an "Organische Formen-Wissenschaft" as the subtitle of the work spells out the meaning of "morphology." This science claimed to establish general laws and comprehensive and consistent explanations of the formative processes of organismic structure. The enterprise focussed particularly on actual or seeming regularities of such formative processes as observed by embryologists and anatomists in comparative studies. By such regularities, above all one law of development seemed to be suggested, namely that of a development from simple to complex, from homogeneous to articulated organismic structure with a differentiation of function among its parts and organs.

It was not this view of order in the variety of organismic forms that distinguished Haeckel from his colleagues. Embryologists like Karl Ernst von Baer and comparative anatomists like Richard Owen had tried to establish laws of formative processes. However, the regularities, or "logic," of organismic forms these biologists investigated was understood by them in a way that is usually called that of an idealist or transcendental morphology.[13] What distinguished Haeckel's attempt from these conceptions was his materialist approach. He presented a morphology that claimed to provide a "mechanical"[14] explanation of the laws that rule the formative processes. And in this morphology, Darwin's theory as understood by Haeckel turned out to be the key to such a mechanical explanation.

In the late 1860s, under the impact of Darwin's theory, many biologists were prepared to accept a real, historical evolution as a possible or even likely rationale of the regularities and laws that seemed to connect organismic forms. Yet, what they did reject was Haeckel's idea that such a lawful development of organismic forms could be explained through a process of adaptations to outer conditions of life, which apparently changed without perceivable regularities. This rejection seems well grounded. For these biologists, Haeckel was linking two issues that couldn't be linked in this way: namely, on the one hand, adaptation to external conditions of life and, on the other, laws of development of structure that, in the view of these biologists, require inner-organismic causes for their explanation.

In what Haeckel had tried to combine, namely laws of formation of structure and adaptation, an increasing number of contemporary biologists saw a clear alternative: If, as for Darwinists, all formation of structure was ultimately brought about by adaptations to external conditions of life,

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11 For the question of whether Weismann's later theory of "germ selection" contradicts these celebrated achievements, see, for instance, (Ridley 1982) 63ff.

12 The term "orthogenesis" (*Orthogenese*) was coined by the zoologist Wilhelm Haacke. — For characteristic features of these theories of evolution, see (Mayr 1982) 528ff. And, for a contemporary view, (Wagner 1908) 223ff.

13 For Owen, see for instance (Rupke 2001) 252; for von Baer, see for instance (Muzrukova 2001) 307.

14 See the subtitle of the second volume of (Haeckel 1866): "Kritische Grundzüge der mechanischen Wissenschaft von den entstehenden Formen der Organismen."

no laws that rule those formation processes could be assumed.[15] And, vice versa, if these processes are ruled by such laws, they cannot be explained by adaptations to external conditions. Rather, increase of complexity of organismic structure, ever more specialised differentiation of function, in short, the tendency of a progressive development in the realm of organismic forms, seemed to suggest an inner-organismic formative drive. The assumption of such an inner force of development which, in this situation, became attractive to many biologists constituted the core of orthogenetic theories of evolution, which began to challenge Haeckel's theory from another quarter since the 1870s.

I cannot go into the different orthogenetic theories of evolution proposed in the last decades of the nineteenth century. I have to confine myself to naming just two well-known biologists as proponents of this variety of evolutionary thinking, namely Carl Wilhelm von Naegeli und Albert von Koelliker.

The two men worked on quite different fields — Naegeli mainly on physiology and anatomy of plants, Koelliker mainly on zoological morphology and embryology — and can in no way be regarded adherents of a certain school. Rather, their research agenda as well as general orientation was quite different. And so are the orthogenetic theories of evolution each of them proposed.[16] There are nevertheless remarkable congruities between their theories besides the shared core assumption of all orthogenetic theories, that is, the assumption of an inner-organismic cause that rules the formative processes of organismic structure. Each of the two men recommended his theory as a truly mechanical-physiological, that is, as a non-teleological one. Both men shared furthermore the conviction that evolution does not necessarily entail a common origin of species and held that each species developed in an independent process of evolution. Finally, though probably not very surprisingly, both men dismissed inheritance of acquired characters.

Dismissal, or at least marginalization, of inheritance of acquired characters was characteristic of almost all orthogenetic theories of evolution,[17] as was dismissal or marginalization of adaptation by use and disuse, of natural selection, and also of Haeckel's biogenetic law. All of these items belonged to theories of evolution that conceive of it as essentially a process of adaptation to changing conditions of life. They were hall-marks of theories that regarded organismic structure as evolving in reaction to external conditions, that is, hall-marks of exactly those theories that

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15 In this paper, I cannot go into the issue of undeniable optimisations of "natural technology" (Marx) in the course of evolution. Such optimisations cannot be explained by adaptation processes that take place alone or foremost between living beings and inorganic nature. Rather, first adaptations between living beings themselves assign to them a "teleonomic" character (Mayr) on which such optimisations rest — see for instance (Lefèvre 1984) 252ff. and 260ff.

16 Naegeli published an elaborate version of his orthogenetic theory first in 1884 (Naegeli 1884); yet he presented outlines of it in several talks and articles since 1865 (Naegeli 1865). Koelliker's orthogenetic theory can be found in part A of (Koelliker 1872). — It is, by the way, remarkable how many botanical physiologists and anatomists endorsed any form of ortogenetic theories. It may suffice to name Eugen Askenasy (Askenasy 1872) and above all Julius Sachs (Sachs 1894) who had long been the most devoted Darwinist among German botanists — see (Höxtermann 2001) and (Junker 1989) 233ff. The latter book is of general interest as regards the reception of the theory of evolution among German botanists.

17 Apart from Naegeli's and Koelliker's theories, one should name those of Wilhelm His and Alexander Wilhelm Goette — see (Montgomery 1972) 102f. A noteworthy exception is Theodor Eimer's orthogenetic theory, which assumed a "stammesgeschichtliches Wachsen" that develops organismic structure in reaction to environmental factors but is restricted by inner forces and tendencies — (Eimer 1897).

adherents of orthogenetic theories strived to overcome. As regards inheritance of acquired characters, proponents of orthogenesis did not doubt, deny, and dismiss this assumption for special genetical arguments. Rather inheritance of acquired characters was dismissed because it had become part and parcel of rejected theories of evolution.

V. Conclusion

In closing, I would like to sum up my paper by four statements.

First, the question of inheritance of acquired characters was put on biologists' agenda of urgent issues immediately when heredity itself became a major biological topic. (For this, I am assuming that heredity was not such a topic before the last third of the nineteenth century.)

Second, biologists tried to decide definitely whether or not inheritance of acquired characters is possible just when first foundations for an answer were laid in physiology, particularly in cytology. The case, thus, seems to confirm Marx's dictum that mankind never poses a problem before the means for its solution are at hand.

However, thirdly, it has the appearance that, in the beginning, inheritance of acquired characters became a fiercely debated issue not so much in the framework of competing theories of heredity but, so to speak, as a collateral damage of a war between competing theories of evolution. Inheritance of acquired characters was (and is) an element of strategic importance for Neo-Lamarckian approaches to evolution.

Fourth and last, against this background, it seems not that surprising that the periodical revivals of inheritance of acquired characters in the twentieth century were often indicative not of new insights in heredity but of lasting difficulties and debates in the framework of evolution.

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Darwinism versus Evo–Devo: a late–nineteenth century debate

Jeffrey H. Schwartz

Abstract

In his notebooks and culminating in the two volume publication on domestication of plants and animals (Darwin 1868), Charles Darwin developed a theory of inheritance, pangenesis, that fit his worldview: through a continuum of variants and intermediate forms, individuals and species graded one into another both syn– and diachronically. Pangenesis accommodated Darwin's fundamental assumptions: use–disuse, natural selection (as a factor in producing variation and then choosing from the resultant variants), and blending inheritance. He conceived of "gemmules," which were constantly thrown off from every part of an organism's body throughout its entire lifetime, as the recorders of everything that impacted an individual during its lifetime. By way of bodily "fluids," gemmules were conveyed to an individual's sex organs, to be passed on en masse *to offspring, combining upon fertilization with those contributed by the other parent. It was an ingenious idea, especially in its attempt to explain all known phenomena, including the appearance of new or different features (such as sexually dimorphic ones) later in life. Blending inheritance expanded the realm of possible variation in the next generation.*

The thrust of Darwin's theory of pangenesis was a justification of his view that the gradual nature of evolutionary change and continuous variation tied all life together in a seamless web. Ironically, the very examples Darwin enumerated for the origin of new breeds or varieties of domesticated plants and animals, and their stabilization through inbreeding and breeding with parental strains, lend themselves not to a model of gradual change and continuous variation, but to a saltationist one, in which morphological novelty emerges abruptly and yet its bearers remain capable of reproducing with the original stock.

Victorian saltationism, as articulated by St. George Mivart in (Mivart1871) in On the Genesis of Species, *envisioned alterations in development as the basis of major and instantaneous change, but it did not deal with heredity. Mivart pointed out that a major problem with Darwin's gradualism was that features critical to an organism's survival and reproductive competence would be useless unless they were functional from the beginning. In addition, as Huxley (Huxley 1860) had argued well before Mivart, natural selection played no role whatsoever in producing change. Since it was obvious that some hereditary process connected successive generations, perhaps saltationists thought it unnecessary to speculate on a mechanism of inheritance because the idea of novelty arising through alterations of developmental processes was not explained by gemmule, germ–plasm, or any other available model of inheritance. Interestingly, the contrasts between Darwin's theory of heredity and Mivart's emphasis on organismal change being due to altering developmental processes, is being replayed in the opposing views of present–day Darwinism and evo–devo.*

Introduction

A consequence of delving into the history of evolutionary thought is often seeing how a scholar, when confronted with examples of biological phenomena that could lead to diametrically opposed and contradictory models or theories, chooses one set as representing the reality of nature and regards the other as irrelevant. In such instances, the obvious question is: Why? What were the reasons behind the decision? In this regard, an interesting case is the contrast between Charles Darwin (especially his "evidence" for natural selection and gradual evolutionary change as being necessary for the origin of species and the assumptions underlying his theory of inheritance, "pangenesis") and the saltationists (especially as represented by St. George Mivart).

Darwin's bias

Although one cannot ignore *On the Origin of Species* (Darwin 1859 et seq.; herewith referred to as the *Origin*), for this discussion I shall focus on *The Variation of Animals and Plants under Domestication* (Darwin 1868; herewith referred to as *Variation*). The rationale for this emphasis is that, in *Variation*, Darwin not only argues that the character of domesticated plants and animals and the process of domestication via artificial selection make perfect analogues for the character of natural species and the process of evolution via natural selection. In this work Darwin also attempts to articulate a theory of inheritance that embraced his bias toward gradualism.

Since the basics of Darwin's assumptions as presented first in the *Origin* are well known, I shall summarize them only briefly. 1) Variation is essentially infinite and, in a perfectly preserved synchronous and diachronous world, one would be able to observe continuous variation and insensible and infinitesimal gradation between individuals, sexes, and species. 2) At any point in time, natural selection both produces and then chooses from the resultant more fit or better adapted variations. 3) The consequences of use and disuse contribute significantly to the emergence of variability. 4) Evolutionary change proceeds gradually through the accumulation of continually produced, infinitesimally small variations.

In arguing the first assumption, Darwin had to confront the discontinuities between extant species and the "gaps" in the paleontological record between extinct species as well as between extinct and extant species. This absence of evidence was explained neontologically and paleontologically by invoking the extinction or elimination of individuals that would have formed a graded series of intermediates between one species and another. The paleontological conundrum was also addressed from a taphonomic perspective: There had been intermediates, but the deposits containing them no longer existed. With the problems of discontinuity seemingly dealt with, Darwin was then free to attend to other matters, such as the origin of species:

> The differences between natural varieties are slight; whereas the differences are considerable between the species of the same genus, and great between the species of distinct genera. How do these lesser differences become augmented into the greater differences? How do varieties, or as I have called them incipient species, become converted into true and well–defined species? (*Variation*, vol. 1, p. 5)

Although he had already committed himself in the *Origin* to answering this question by invoking a gradualistic model of change, Darwin was clearly aware of examples, not only from nature, but also from plant and animal domestication, that could have led him to formulate a different model of evolutionary tempo. For example, in volume 1 of *Variation* (pp. 92–94) he commented on how the niata breed of cattle had appeared suddenly, in the course of one generation, and then described in detail how they differed from common cattle in numerous aspects of their anatomy. For example:

> In fact, on comparison with the skull of a common ox, scarcely a single bone presents the same exact shape, and the whole skull has a wonderfully different appearance. (p. 94)

Although they appeared suddenly, and their features were so different from those of common cattle, niata cattle could breed successfully with one another, as well as with common cattle. In the latter case, Darwin even discussed the specific characters that were often dominant in offspring when a niata cow was mated with a common bull, and vice versa.

Nevertheless, as profound as the differences between niata and common cattle were, and even with the former being reproductively unimpaired, Darwin rejected the case of niata cattle as being representative of how novelty might arise in the wild. His argument was that these cattle were not as adaptable as common cattle when environmental conditions occasionally and drastically changed for the worse, as in periods of drought. As such, Darwin (*Variation*, vol. 1, p. 94) concluded, "[this] shows us…how natural selection would have determined the rejection of the niata modification had it arisen in a state of nature."

But niata cattle were not the only example of the sudden appearance of marked novelty in animals or plants without loss of reproductive viability of which Darwin was aware. Indeed, it was precisely because these "monstrosities" could mate successfully with one another (as well as with "normal" individuals) that breeders could perpetuate the novelty. For instance:

> [The sprouting–broccoli] variety is a new one, and bears the same relation to common broccoli, as Brussel–sprouts do to common cabbages; it suddenly appeared in a bed of common broccoli, and was found faithfully to transmit its newly–acquired and remarkable characters (vol. 1, p. 342).

> Domestic breeds often have an abnormal or semi–monstrous character, as amongst dogs…some breeds of cattle and pigs,–several breeds of fowl,–and the chief breeds of pigeon. In such abnormal breeds, parts which differ but slightly or not at all in the allied natural species, have been greatly modified. This may be accounted for by man's often selecting, especially at first, conspicuous and semi–monstrous deviations of structure (volume 2, p. 408).

Even in terms of so–called atavistic structures, Darwin knew not only that they re–appeared suddenly, but also that they were morphologically recognizable structures. They were not mere hints of features that had been present in their bearers' ancestors. As he noted in volume 1 of *Variation*:

> Horses have often been observed, according to [the French paleontologist] M Gaudry, to possess a trapezium and a rudiment of a fifth metacarpal bone, so that "one sees appearing by

> monstrosity, in the foot of the horse, structures which normally exist in the foot of the Hipparion [an allied and extinct animal]" (p. 52; comments added).

Yet, in the face of numerous examples of the sudden appearance of novelty in domesticated plants and animals, Darwin argued that they were not reflective of what really occurs in nature:

> There is a much more important distinction between our several breeds, namely, in some having originated from a strongly–marked or semi–monstrous deviation of structure, which, however, may subsequently have been augmented by selection; whilst others have been formed in so slow and insensible a manner, that if we could see their early progenitors we should hardly be able to say when or how the breed first arose…[But] it is certain that the ancon and mauchamp breeds of sheep, and almost certain that the niata cattle, turnspit, and pug–dogs, jumper and frizzled fowls, short–faced tumbler pigeons, hook–billed ducks, &c., suddenly appeared in nearly the same state as we now see them. So it has been with many cultivated plants. The frequency of these cases is likely to lead to the false belief that natural species have often originated in the same abrupt manner. But we have no evidence of the appearance, or at least of the continued procreation, under nature, of abrupt modifications of structure; and various general reasons could be assigned against such a belief.
>
> On the other hand, we have abundant evidence of the constant occurrence under nature of slight individual differences of the most diversified kinds; and we are thus led to conclude that species have generally originated by the natural selection of extremely slight differences. (vol. 2, pp. 409–410)
>
> Some naturalists boldly insist that species are absolutely distinct productions, never passing by intermediate links into one another; whilst they maintain that domestic varieties can always be connected either with one another or with their parent–forms. (vol. 2, p. 409)

But does demonstrating "abundant evidence of the constant occurrence under nature of slight individual differences of the most diversified kinds" necessarily contradict inferring from observing the sudden appearance of novel features in domesticated organisms that this process also occurs in nature? Does demonstration of "abundant evidence of the constant occurrence under nature of slight individual differences of the most diversified kinds" necessarily lead only to the conclusion that "species have generally originated by the natural selection of extremely slight differences"?

The answer to both of these questions is, I believe, no. But by taking the position he did, Darwin conflated the existence of individual variation, which reflects slight degrees of difference in the expression of a particular feature or array of features, with the advent of the feature itself. And it was this unfounded conflation of two entirely different biological phenomena that informed his belief in continuous variation and continuity via an insensible gradation between species through time and at any point in time. Accordingly, and without any justification or demonstration, Darwin could then claim that "[v]ariations often pass into, and cannot be distinguished from, monstrosities; and monstrosities are of little significance for our purpose" (vol. 1, p. 322). Indeed, for the myriad examples Darwin gives in the *Origin* and in *Variation* of "monstrosities" and of slight variations of a feature or features, none supports an assumption of

gradation. Darwin merely asserts that this is the case, just as, in the same quote, he can so easily declare, "monstrosities are of little significance for our purpose."

But Darwin persists in volume 2 of *Variation* in asserting the reality of insensible gradation [e.g. "…we so incessantly see in species of the same group the finest gradations between an organ in a rudimentary and perfect state, that we are led to believe that the passage must have been extremely gradual" (p. 308)]. He also attempts to explain why sudden change, at least in wild species, could not occur: "It may be doubted whether a change of structure so abrupt as the sudden loss of an organ would ever be of service to a species in a state of nature; for the conditions to which all organisms are closely adapted usually change very slowly" (p. 308).

This is an interesting approach to denying the possibility of sudden change because, here, Darwin focuses on the abrupt *loss* of structure, not, as with virtually every other example he musters in the *Origin* and *Variation* in support of gradual change, the *emergence* of structure. He then states with unfounded assurance that abrupt change not only would not benefit wild species, but also that it could not occur because organisms adapt gradually to their slowly changing surroundings. Yet Darwin knows that sudden, non–reproductively disruptive change occurs. He also knows, at least from the example of niata versus common cattle responding to drought, that the environment can change abruptly, without provoking visible organismal change at the same time. But, here and elsewhere (see quote further above) he draws a line of distinction between domesticated and wild species, and seeks to justify this distinction by asserting that no one has observed abrupt change in nature. This, of course, is an assertion without basis, because, by definition (and observation in domesticates) there is nothing to observe if change occurs suddenly. There is only the presence of something previously unknown to you whose origin would be a mystery.

Thus, while on the one hand, Darwin would like his audience to believe that, as artificial selection can gradually change the character of a domesticated species or breed, so, too, can natural selection act on wild species. On the other hand, Darwin asserts without justification that the sudden appearance (not loss) of novelty in domesticated species – that is, the emergence of "monstrosities" – has no bearing on insights into the workings of the organisms in the "wild." Still, by asking us to accept the analogy between artificial and natural selection as agents that gradually alter the character of species, and to deny any biological significance to the sudden appearance of novelty in domesticates (and thus natural species), Darwin perpetuates the misconception that individual variation in the expression of a feature is somehow relevant to the origin of the feature itself. That, by artificially shifting the bell curve of expressed variation of a feature, this serves as evidence that, under natural conditions and with enough time, a feature can be transformed into something entirely different.

Darwin's theory of pangenesis

Without doubt, the climax of Darwin's formulations in *Variation* was the model of inheritance that he called "Pangenesis." In many ways, it was quite elegant (see review in Schwartz, 1999). Through pangenesis Darwin could explain, for instance, features that appear early in development as well as those that emerge later in life (e.g. differences in secondary sexual characters). The idea was simple. All parts of an organism issue small particles, which Darwin identified as gemmules,

throughout the individual's life. Consequently, the entire life history of an individual, from conception until death, is recorded in a trail of gemmules. Individually unique events, such as those that result from use or disuse, would also be recorded in the gemmules of the affected part or parts and thus affect the pool of potential variation. Traveling by way of some unspecified bodily fluid, gemmules accumulate in an individual's sex organ. Upon mating, parental gemmules blend, thereby producing additional sources of variation. Gemmules could also become latent and not expressed over a series of generations. But at some later time, they could become active again, which would account for atavisms.

Pangenesis solidified Darwin's ideas on blending inheritance (hinted at in the *Origin*, although clearly expressed in his notebooks; see Schwartz 1999) and also provided a previously unspecified mechanism for the transmission of acquired characteristics resulting from use or disuse. The theory also increased the number of ways in which individual variation could be produced. Indeed, pangenesis seemed to be able not only to account for the entirety of an organism's being, but also to provide the fodder necessary for natural selection to slowly transform one species into another.

The opposing saltationist view

Three years after the publication of Variation, St. George Mivart, one of England's leading comparative morphologists, published *On the Genesis of Species* (Mivart 1871, herewith referred to as *Genesis*). This was the saltationist's response to Darwin's notions of gradual change, the role of natural selection, the essence of variation, and the viability of pangenesis. Although in his review of the *Origin*, Huxley (Huxley 1860) was clearly strongly opposed to gradualism and a role of primacy of natural selection in producing change, Mivart makes it appear as if his fellow saltationist was not fully committed to this position:

> Professor Huxley seems now disposed to accept the, at least occasional, intervention of sudden and considerable variations. In his review of Professor Kölliker's criticisms, he himself, says, "We greatly suspect that she" (*i.e.* Nature) "does make considerable jumps in the way of variation now and then, and that these saltations give rise to some of the gaps which appear to exist in the series of known forms." (*Genesis*, pp. 103–4)

In mounting his case for saltationism, Mivart paralleled Darwin in compiling a massive array of examples from the plant and animal worlds. But instead of focusing on the minutiae of individual differences, Mivart called attention to the major ways in which species differ from one another, whether it be in the stamens and anthers of flowers, the pincers or antennae of beetles, the configuration of the vertebrate versus invertebrate eye, the presence of mammary glands in mammals, feathers in birds, and baleen in whales, or differences between organisms in their reproductive anatomies. In contrast to Darwin's rejection of sudden change solely on the basis of the argument in *Variation* on the lack of benefit of losing of an organ, Mivart emphasized the emergence of novel structure. Not, of course, that the loss of structure could not be novel – consider the reduced numbers and generations of teeth in mammals compared to reptiles, of toes in modern horses, and of limbs in snakes. But, inasmuch as Darwin himself presented examples of "gain" rather than "loss" (e.g. the vertebrate eye) in both the *Origin* and *Variation*, it was

appropriate that Mivart do the same, although his interpretation of the requisites for the appearance of novelty was vastly different.

Time and time again Mivart discussed a remarkable trait and then raised the question: How could such a functionally important feature have evolved gradually, through an insensible gradation of intermediates, to its present state? How, for instance, could mammals not only have survived, but multiplied in number and become diverse, if the first mammal had merely possessed a vestige of a mammary gland, which, in turn, produced only a drop or two of milk? How could sexually reproductive organisms have persisted generation after generation if, initially, their reproductive organs were merely a hint of their necessary functional states? Turning Darwin's argument of purpose on its head, Mivart asked: How could anything but the fully formed version of a feature, particularly one that was essential for sustenance of life or procreation, be beneficial to an organism? Using flatfish (soles, flounder) as one of many examples leading to doubting Darwin's assumption, Mivart questioned the advantage of selection causing one of the fish's eyes to be dragged gradually from one side of its head, across the rough sand of the ocean floor, until it reached its present position near the eye that had been on the opposite side of the body. Clearly, on various levels, the notion of gradual change did not make biological sense.

But a saltational model for the advent of novelty – especially if functionally integral to the survival of an individual – was not only biologically sensible, it was also compatible with the pattern of life as illustrated in the fossil record.

Indeed, Mivart (pp. 129–130) quotes from Fleming Jenkin's devastating review of the *Origin* ("It is really strange that vast numbers of perfectly similar [fossil] specimens are so great; but it is also very strange that the specimens should be so exactly alike as they are, if, in fact, they came and vanished by a gradual change"), and then proceeds with his own argument: "The mass of paleontological evidence is indeed overwhelmingly against minute and gradual modification…[H]ad such a slow mode of origin, as Darwinians contend for, operated exclusively in all cases, it is absolutely incredible that birds, bats, and pterodactyles should have left the remains they have, and yet not a single relic be preserved in any one instance of any of these different forms of wing in their incipient and relatively imperfect functional condition!"

Even the bird–like fossil reptile, *Archaeopteryx*, which had been discovered in 1861, and which Darwin would use in the sixth and last edition of the *Origin* in an attempted refutation of *Genesis*, did not pose a problem for Mivart (p. 131): "But even supposing all that is asserted or inferred on this subject to be fully proved, it would not approach to a demonstration of specific origin by *minute* modification. And though it harmonizes well with 'Natural Selection,' it is equally consistent with the rapid and sudden development of new specific forms of life."

In contrast to Darwin, who invoked forces external to the organism as the primary provocateurs of change (e.g. as in gradual environmental change, or use–disuse), Mivart hypothesized the source of novelty as lying primarily internally, within the cells of the organism itself. In formulating this theory, Mivart turned to the inert inorganic world for an analogy relevant to the living one. In apparent anticipation of some of Waddington's (Waddington 1940) ideas many decades later, Mivart (*Genesis*, p. 114) suggested:

> Judging the organic world from the inorganic, we might expect, *a priori*, that each species of the former, like crystallized species, would have an approximate limit of form, and even of si-

> ze, and at the same time that the organic, like the inorganic forms, would present modifications in correspondence with surrounding conditions; but that these modifications would be, not minute and insignificant, but definite and appreciable, equivalent to the shifting of [a] spheroid on to another facet for support.

Mivart (pp. 114–115) then quotes from a Mr. Murphy ("Crystalline formation is also dependent in a very remarkable way on the medium in which it takes place…And [as] the Rev. E. Craig found that [different chemicals affected copper crystal growth]…[t]he changes take place not by the addition of new crystals, but by changing the growth of the original ones."), after which he comments: "These, however, may be said to be the same species, after all; but recent researches by Dr. H. Charlton–Bastian seem to show that modifications in the conditions may result in the evolution of forms so diverse as to constitute different organic species."

In contrast to Darwin's efforts to explain away the "gaps" in the fossil record, Mivart (p. 143) expands his model to incorporate them:

> Now all these difficulties [e.g. the absence of fossils in old strata and of intermediate forms] are avoided if we admit that new forms of animal life of all degrees of complexity appear from time to time with comparative suddenness, being evolved according to laws in part depending on surrounding conditions, in part internal–similar to the way in which crystals (and, perhaps from recent researches, the lowest forms of life) build themselves up according to the internal laws of their component substance, and in harmony and correspondence with all environing influences and conditions. [comment added]

But, what is the internal element of Mivart's saltational theory? After all, one could claim that Darwin's theory of pangenesis embodied an internal component to eventual evolutionary change. The difference between the two scholars lies in Mivart's thinking in terms of novelty emerging as a result of alterations in the regulation of an organism's development:

> Altogether, then, it appears that each organism has an innate tendency to develop in a symmetrical manner, and that this tendency is controlled and subordinated by the action of external conditions, and not that this symmetry is superinduced on *ab externo*. In fact, that each organism has its own internal and special laws of growth and development.
>
> If, then, it is still necessary to conceive an internal law or "substantial form," moulding each organic being, and directing its development as a crystal is built up, only in an indefinitely more complex manner, it is congruous to imagine the existence of some internal law accounting at the same time for specific divergence as well as for specific identity.
>
> A principle regulating the successive evolution of different organic forms is not one whit more mysterious than is the mysterious power by which a particle of structureless sarcode develops successively into an egg, a grub, a chrysalis, a butterfly, when all the conditions, cosmical, physical, chemical, and vital, are supplied, which are the requisite accompaniments to determine such evolution. (pp. 186–7)
>
> [T]he new forms must be produced by changes taking place in organisms in, after or before their birth, either in their embryonic, or toward or in their adult, condition. (p. 233)

Reminiscent, at least in spirit, of Wright's (Wright 1932) "shifting balance theory" (represented by the topographic map of differing gene combinations) and Waddington's (Waddington 1940) "epigenetic landscape," Mivart (pp. 228–9) frames his theory of developmental reorganization in terms of a rapid transition from one stable state to another:

> The conception of such internal and latent capabilities is somewhat like that of Mr. Galton…according to which the organic world consists of entities, each of which is, as it were, a spheroid with many facets on its surface, upon one of which it reposes in stable equilibrium. When by the accumulated action of incident forces this equilibrium is disturbed, the spheroid is supposed to turn over until it settles on an adjacent facet once more in a stable equilibrium.
>
> The internal tendency of an organism to certain considerable and definite changes would correspond to the facets on the surface of the spheroid.

Equally interesting is how Mivart's (*Genesis*, p. 230) language anticipates Bateson's (Bateson 1894) "undulating theory" or "theory of repeated parts":

> [A]s the atoms of a resonant body may be made to give out sound by the juxtaposition of a vibrating tuning–fork, so it is conceivable that the physiological units of a living organism may be so influenced by surrounding conditions (organic and other) that the accumulation of these conditions may upset the previous rhythm of such units, producing modifications in them–a fresh chord in the harmony of nature–a new species!

Mivart (p. 231) then asks: "Are new species now evolving, as they have been from time to time evolved? If so, in what way and by what conceivable means?" To which he responds:

> [W]e…saw that minerals become modified suddenly and considerably by the action of incident forces…
>
> We have thus a certain antecedent probability that if changes are produced in specific manifestation through incident forces, these changes will be sensible and considerable, not minute and infinitesimal.
>
> Consequently, it is probable that new species have appeared from time to time with comparative suddenness, and that they still continue so to arise if all the conditions necessary for specific evolution now obtain. (p. 236)
>
> [A]n internal law presides over the actions of every part of every individual, and of every organism as a unit, and of the entire organic world as a whole. It is believed that this conception of an internal innate force will ever remain necessary, however much its subordinate processes and actions may become explicable.
>
> That from such a force, from time to time, new species are manifested by ordinary generation just as *Pavo nigripennis* appeared suddenly, these new forms not being monstrosities but harmonious self–consistent values… (p. 239)

Countering Darwin's argument against saltation in *Variation* that only a solitary individual would be the bearer of a novelty, Mivart (p. 236) hypothesizes that "as the same causes produce the same

effects, several individual parent forms must often have been similarly and simultaneously affected." As for Darwin's theory of inheritance, Mivart (p. 216) dismisses it with a quote from Delpino: "Thus, in Pangenesis, everything proceeds by force of unknown elements, and we may ask whether it is more logical to prefer a system which assumes a multitude of unknown elements to a system which assumes only a single one?"

Darwin's rebuttal

Given Mivart's rejection of virtually all of Darwin's assumptions – with the major exception of allowing that natural selection plays a role in eliminating monstrosities, rapidly eliminates antecedent species, and "favours and develops useful variations, though it is impotent to originate them or to erect the physiological barrier which seems to exist between species" (*Genesis*, p. 240) – it is not surprising that Darwin harshly criticized the former scientist the following year in the sixth (and, as it turned out, last) edition of the *Origin*.

In this edition of the *Origin*, published in 1872, the year after *Genesis*, Darwin doggedly maintained the theme of "natura non facit saltum" and devoted thirty pages specifically to Mivart's objections to his theory and his objections to Mivart's.

> Mr. Mivart believes that species change through an "internal force or tendency," about which it is not pretended that anything is known. That species have a capacity for change will be admitted by all evolutionists; but there is no need…to invoke any internal force beyond the tendency to ordinary variability, which through the aid of selection by man has given rise by graduated steps to natural races or species.
>
> Mr. Mivart is further inclined to believe, and some naturalists agree with him, that new species manifest themselves "with suddenness and by modifications appearing at once." For instance, he supposes that the differences between the extinct three–toed Hipparion and the horse arose suddenly. He thinks it difficult to believe that the wing of a bird "was developed in any other way than by a comparatively sudden modification of a marked and important kind…This conclusion, which implies great breaks or discontinuity in the series, appears to me improbable in the highest degree. (*Origin*, 1872, p. 239)

Although Mivart did hypothesize an "internal force" as a generator of sudden change, his model was at least consistent with the observation of the abrupt appearance of novelty in domesticated plants and animals, the discontinuity (i.e. lack of seamless continuity) between apparent species, and the gap–riddled pattern of life history as recorded in the fossil record. In contrast, Darwin attempted to dismiss the relevance of all three of these observations, appealing, as he stated in the quote above, to a sense of probability (or, in this case, improbability). Also as seen in the quote above, Darwin pushed the envelope of credulity, not only by making it seem that the origin of new breeds of domesticated plants and animals is typically by "graduated steps," but also by adding the claim that this process has led to the emergence of new species of domesticates. In the former assertion, Darwin neglected his own recognition of the reality of niata cattle and other examples of the sudden origin of novelty, while, in the latter, he clearly entered the realm of the imaginary.

Darwin's (Darwin 1872) most relevant objection to Mivart's theory is found in his questioning the expectation that more than one individual will emerge with the same novelty. Although this

suggestion is logically consistent with the argument for change via an internal force that causes developmental reorganization, Darwin (p. 240) makes light of it: "Hence in order that a new species should suddenly appear in the manner supposed by Mr. Mivart, it is almost necessary to believe, in opposition to all analogy, that several wonderfully changed individuals appeared simultaneously within the same district." Of course, "analogy" is the key word here, inasmuch as Darwin promotes the case of gradual change in domesticates through artificial selection. But, upon reflection, Darwin's counterargument merely reiterates the essence of the variation–natural selection argument that Fleming Jenkin demolished in his review of the first edition of the *Origin* (see Schwartz 1999): "This difficulty [the sudden appearance of many individuals with the same novelty]…is avoided on the theory of gradual evolution, through the preservation of a large number of individuals, which varied more or less in any favourable direction, and of the destruction of a large number which varied in an opposite direction" (Darwin 1872, p. 240; comment added). Relying on repetition rather than validation, Darwin states outright: "That many species have been evolved in an extremely gradual manner, there can hardly be a doubt" (p. 240) for "when we look to the special parts of allied species, instead of to distinct species,…numerous and wonderfully fine gradations can be traced, connecting together widely different structures" (p. 241).

Again, the question must be raised: What is the basis of these assertions? If Darwin can take issue with Mivart's hypothesis on the grounds that there is no evidence of an "internal force," are we then expected to take his word for the existence of "numerous and wonderfully fine gradations…connecting together in widely different structures"? Where is the demonstration of the reality of gemmules, of the effects of use and disuse, of blending inheritance, or of the power of natural selection to produce novel structures? Indeed, it is here we can appreciate Delpino's objection to pangenesis: Why should we invoke a multitude of unknowns when an alternative theory predicts only one mechanism?

But Darwin (Darwin 1872, p. 242) continues his attack on Mivart, concluding with an appeal to embryology:

> It is notorious that the wings of birds and bats, and the legs of horses or other quadrupeds, are undistinguishable at an early embryonic period, and that they become differentiated by insensibly fine steps. Embryological resemblances of all kinds can be accounted for…by the progenitors of our existing species having varied after early youth, and having transmitted their newly acquired characters to their offspring, at a corresponding age. The embryo is thus left almost unaffected, and serves as a record of the past condition of the species. Hence is it that existing species during the early stages of their development so often resemble ancient and extinct forms belonging to the same class. On this view of the meaning of embryological resemblances…it is incredible that an animal should have undergone…momentous and abrupt transformations; and yet should not bear even a trace in its embryonic condition of any sudden modification; every detail in its structure being developed by insensibly fine steps.

On the face of it, this passage would appear to echo von Baer's (Baer 1828) laws regarding the commonality of embryonic stages among vertebrates until the point at which each kind of animal veers off onto the ontogenetic path that will mold it into the adult of its taxon, replete with its specifically distinctive features. But it is obvious that Darwin's invocation of ontogeny is actually

a foil for gradualism, using the smoothly transitional nature of organismal growth and development as supposed evidence of the gradual nature of evolutionary change. Future suggestions aside as to how novelty could become imbedded early on in ontogeny (e.g. de Beer's, 1930, theory of clandestine evolution), what is interesting about Darwin's dismissal of such a possibility and his focus on "structure being developed by insensibly fine steps" is that the only avenue he leaves open along which change can occur is by *adding* stages to the end of an individual's ontogeny. This, of course, is precisely the primary interplay between ontogeny and phylogeny that Haeckel (e.g. 1866) envisioned when he formulated the Biogenetic Law ("ontogeny recapitulates phylogeny"). Both Darwin and Haeckel envisioned links between adult individuals, with change occurring at the terminal stage of ontogeny.

Wherefore Thomas Henry Huxley?

Darwin's intensified adherence to notions of gradual transformational change from *Variation* to the last edition of the *Origin*, and his assault on Mivart in the latter work, is both interesting and curious given the reviews he received of the first edition of the *Origin*, not only from Fleming Jenkin, but also and especially from his intellectual defender, Thomas Henry Huxley. For, in his review of the *Origin*, which was published in 1860 (and reprinted thereafter, e.g. in Huxley 1876, *Lay Sermons, Addresses, and Reviews*, hereafter referred to as *Sermons*), Huxley was anything but restrained in his criticism of Darwin's rejection of rapid morphological change. For example, on p. 257 in *Sermons*, in the reprinted review of the *Origin*, Huxley writes:

> We do not speak jestingly in saying that it is Mr. Darwin's misfortune to know more about the question he has taken up than any man living.
>
> But this superabundance of matter must have been embarrassing to a writer who, for the present, can only forward an abstract of his views; and thence it arises, perhaps, that notwithstanding the clearness of the style, those who attempt fairly to digest the book find much of a sort of intellectual pemmican–a mass of facts crushed and pounded into shape, rather than held together by the ordinary medium of an obvious logical bond: due attention will, without doubt, discover this bond, but it is often hard to find.
>
> Again, from the sheer want of room, much has to be taken for granted which might readily enough be proved; and hence, while the adept, who can supply the missing links in the evidence from his own knowledge, discovers fresh proof of the singular thoroughness with which all difficulties have been considered and all unjustifiable suppositions avoided, at every reperusal of Mr. Darwin's pregnant paragraphs, the novice in biology is apt to complain of the frequency of what he fancies is gratuitous assumption.

In apparent heed of his own criticism of Darwin's unrestrained use of example, Huxley keeps his reference to individual cases to a bare minimum. Nevertheless, he does refer to one of Darwin's own citations: the abrupt appearance of a "monstrosity" that became the basis of a new breed of sheep, the Ancon or Otter sheep. He does so (p. 265) to demonstrate not only that it "appears to have arisen in full force, and…*per saltum*," but also to argue that "[i]t was no case of what is commonly called adaptation to circumstances; but, to use a conveniently erroneous phrase, that

variations arose spontaneously." But, while Darwin would say that such demonstrations in domesticated animals are not applicable to wild species, Huxley takes an opposing view:

> Varieties then arise we know not why; and it is more than probable that the majority of varieties have arisen in this 'spontaneous' manner…But however they may have arisen, what especially interests us at present is, to remark that, once in existence, varieties obey the fundamental law of reproduction that like tends to produce like, and their offspring exemplify it by tending to exhibit the same deviation from the parental stock as themselves. (p. 266)

> If a variation which approaches the nature of a monstrosity can strive…to reproduce itself, it is not wonderful that less aberrant modifications should tend to be preserved even more strongly; and the history of the Ancon sheep is, in this respect, particularly instructive. (p. 267)

Anticipating Mivart's *Genesis*, Huxley used Darwin's argument for rejecting monstrosities – in this case the Ancon sheep – as a reflection of nature in the "wild" as the basis for coming to a diametrically opposed conclusion: monstrosities are biologically, and therefore evolutionarily, instructive. Further like Mivart, Huxley (e.g. see pp. 266–271) rejected natural selection as playing a role in the emergence of novel features. Unlike Mivart, however, Huxley did not speculate on how novelty is produced "spontaneously" – a fact that seems incongruous given Huxley's (Huxley 1863) emphasis only a few years later on development and the emergence of differences between taxa. Instead, Huxley took the approach of questioning on philosophical grounds the validity of Darwin's claims. For example, on pp. 294–295 of *Sermons*, he writes:

> Inductively, Mr. Darwin endeavours to prove that species arise in a given way. Deductively, he desires to show that, if they arise in that way, the facts of distribution, development, classification, &c., may be accounted for, *i.e.* may be deduced from their mode of origin, combined with admitted changes in physical geography and climate, during an indefinite period. And this explanation, or coincidence of observed with deduced facts, is, so far as it extends, a verification of the Darwinian view.

> There is no fault to be found with Mr. Darwin's method, then; but it is another question whether he has fulfilled all the conditions imposed by that method. Is it satisfactorily proved, in fact, that species may be originated by selection? that there is such a thing as natural selection? that none of the phaenomena exhibited by species are inconsistent with the origin of species in this way? If these questions can be answered in the affirmative, Mr. Darwin's view steps out of the ranks of hypotheses into those of proved theories; but, so long as the evidence at present adduced falls short of enforcing that affirmation, so long, to our minds, must the new doctrine be content to remain among the former–an extremely valuable, and in the highest degree probable, doctrine, indeed the only extant hypothesis which is worth anything in a scientific point of view; but still a hypothesis, and not yet the theory of species.

> After much consideration, and with assuredly no bias against Mr. Darwin's views, it is our clear conviction that, as the evidence stands, it is not absolutely proven that a group of animals, having all the characters exhibited by species in Nature, has ever been originated by selection, whether artificial or natural.

And on pp. 297–8,

> …Mr. Darwin's position might, we think, have been even stronger than it is if he had not embarrassed himself with the aphorism, "*Natura non facit saltum*," which turns up so often in his pages. We believe, as we have said above, that Nature does make jumps now and then, and a recognition of the fact is of no small importance in disposing of many minor objections to the doctrine of transmutation.
>
> …Our object has been attained if we have given an intelligible, however, brief, account of the established facts connected with species, and of the relation of the explanation of those facts offered by Mr. Darwin to the theoretical views held by his predecessors and his contemporaries, and, above all, to the requirements of scientific logic. We have ventured to point out that it does not, as yet, satisfy all those requirements; but we do not hesitate to assert that it is as superior to any preceding or contemporary hypothesis, in the extent of observational and experimental basis on which it rests, in its rigorously scientific method, and in its power of explaining biological phaenomena, as was the hypothesis of Copernicus to the speculations of Ptolemy. But the planetary orbits turned out to be not quite circular after all, and, grand as was the service Copernicus rendered to science, Kepler and Newton had to come after him. What if the orbit of Darwinism should be a little too circular? What if species should offer residual phaenomena, here and there, not explicable by natural selection?

But others did come after Darwin. Even though Darwin was a well–known naturalist prior to 1859, if we use the publication of *On The Origin of Species* as the public emergence of Darwin into the realm of evolutionary theory, we must recognize Huxley and Mivart as subsequent major critics of the credos of natural selection and gradual transformation. Curiously, though, it is Mivart who Darwin publicly attacked, and who was arguably a primary provocation for the 1872 revision of the *Origin*. Of no less import than Darwin's determined adherence to gradual transformation in this latter work was his clinging to the theory of pangenesis. Indeed, as he clearly appears to have dug in his heels on gradualism and a rejection of saltationism and the importance of "monstrosities" for understanding evolutionary change in reaction to Mivart, he seems to have done the same with regard to pangenesis and his cousin Galton's (Galton 1871) experiments that failed to support it.

By the late nineteenth century, with no less energy than Darwin brought to bear on his theories, Bateson (Bateson 1894) and de Vries (de Vries 1889) were arguing with force and conviction for the sudden origin of novelty as well as for decoupling the origin of novelty from selectionist scenarios, and relegating the role, if any, of natural selection to the survival of species. Although inspired by Darwin's theory of pangenesis, de Vries' theory of intracellular pangenesis was actually a rejection of the former notions of inheritance. And, certainly, in 1903, the capstone year of this workshop, Morgan, a trained embryologist, was as vocal as any scientist in rejecting the hyperbole and circularity of Darwinism as being of any evolutionary import.

Why, then, did the questioning of Darwinian explanations of smoothly transformational change become submerged until the recent advent of "evo–devo" thinking? Ironically, it was through the work of Morgan, who in just over a decade went from lambasting Darwinism and Mendelism in 1903 as metaphysical flights of intellectual fancy to melding the two disciplines into

the population–genetics thinking that informed the evolutionary synthesis (see review in Schwartz 1999).

Conclusion

Although Darwin is often lauded for embracing embryology and development in his theory of gradual change via natural selection, his perspective was clearly at odds with Mivart's. It may be true that both scholars envisioned a source external to the individual as playing some role in the emergence of organismal change. But it is equally obvious that only at this vague level might we seek a favorable comparison between these two scientists. Otherwise, for Darwin, biologically real novelty lies only in the minutiae of individual difference, which, in turn, derives from any number of sources that, often through use or disuse, leave their marks on an individual. The notion that use–disuse can engender change by causing elements of a *postnatal* individual to become altered, and that this effect can then, via gemmules, be passed on to future generations, fits the envisioned role of natural selection: Both concepts externalize the ultimate source of organismal modification. Blending inheritance is, therefore, the only aspect of Darwin's theory of pangenesis that might conceivably be regarded as representing an internal component of an individual's biology. With the substitution of Mendelism and population genetics for blending inheritance and gemmules in early formulations of neo–Darwinism (Morgan, e.g. 1916), Darwin's theory of evolution by means of natural selection seemed to be unassailable (e.g. Simpson 1962).

Although use–disuse arguments were supposedly purged from Darwinism (by way of singling out Lamarck as the lone advocate of such lunacy; see Burkhardt 1977), it is obvious from the language of Darwinian explanation still in vogue that, in essence, they were not (e.g. see examples in Schwartz 2004, 2005). Even though Darwinism today claims a basis in genetics, the emphasis is not only on the incorrect notion of there being "genes for things" – similar to the idea that selection chooses features to serve a purpose – but also and contradictorily on the similarly biologically unreal notion that selection can direct the course of genetic change by selecting behavioral or morphological traits that anticipate their benefit to an individual. One might thus characterize present–day Darwinism as the "vacuum theory of evolution" (Schwartz 2005, in press).

In contrast, Mivart's emphasis on internal reorganization affecting an organism's development represents an entirely different biological perspective. Although hypothesizing an "external force" as the initiator of a process of change, whatever course organismal change takes is rapid and random with regard to the circumstances in which the altered organism finds itself. Most importantly, Mivart seats organismal change in the context of an internal restructuring of developmental processes. At one point in time, the developmental organization of an organism is in equilibrium, as is the spheroid lying on one of its facets. In order for change to occur, this equilibrium must be disrupted. Ultimately, the spheroid will come to rest in equilibrium on another facet; that is, developmentally, the organism will be in equilibrium in a different or novel state of organismal organization. If the resultant novelty is ill suited to its bearer's circumstance, the individual will most likely not survive. Even if one chooses to equate the "elimination" of individuals with "natural selection," this process or phenomenon is involved neither in the

production of novelty, nor in the differential selection of individuals that are either more fit than others or supposedly fulfilling a particular adaptive trajectory.

With the exception of incorrectly predicting that more than one individual will emerge with the same novelty because they will respond similarly to the same provocation, Mivart's focus on development is unexpectedly compatible with the emphasis in evolutionary developmental genetic theory ("evo–devo") on novelty resulting via the differential recruitment of regulatory molecules in different signal transduction pathways (e.g. Carroll et al, 2005; Gilbert and Bolker 2001; Raff 1996; Maresca and Schwartz, n.d.; Schwartz 1999). That is, in contrast to the Darwinian population genetics model of continually changing "genes" or "genomes" underlying the emergence of minute variations of a phenotypic trait, modern cell and molecular biology have demonstrated that cell and DNA stability or homeostasis is the rule. The potential for change thus occurs when this "equilibrium" (to use Mivart's term) is disrupted and new pathways of molecular communication become available. As Gilbert and Bolker (Bolker 2001, p. 451) put it:

> Embryologists now recognize receptors and signal transducing molecules as components of the competence apparatus that enable certain cells to respond to specific inducers. These signaling pathways are the bases of embryonic induction, which is in turn the core of organogenesis. If macroevolution involves changing morphological features, then the alteration of signal transduction pathways becomes critical for any discussion of large scale evolution.

In a very basic way, then, it might not be inappropriate to delineate the beginning of a "Darwinism"–"evo–devo" debate in the late nineteenth century, between one of Victorian England's leading comparative anatomists, St. George Mivart, and Darwin himself.

Postscript

Given Darwin's seemingly career–long entrenchment in gradualism and rejection of saltationsim, it is with some surprise to read entry 130 in his Red Notebook, which according to Herbert (Herbert 1980), was written sometime during March, 1837:

> The same kind of relation that common ostrich bears to (Petisse. {lesser or Darwin's rhea} & diff kinds of Fourmillier {antbird}): extinct Guanaco {llama} to recent: in former case position, in latter time. (or changes consequent on lapse) being the relation. – As in first cases distinct species inosculate, so must we believe ancient ones: [(] not <u>gradual</u> change or degeneration, from circumstances: if one species does change into another it must be per saltum – or species may perish. = This <inosculation> representation of species important, each its own limit & represented. – Chiloe creeper {thorn–tailed Rayadito}: Furnarius {ovenbird}. <Caracara> Calandria; inosculation alone shows not gradation. {comments added}

Reading this passage and then those as well as other notebooks that followed is a frustrating experience inasmuch as there is no obvious reason for Darwin to have abandoned saltational ideas as completely as he did. Indeed, as the quotes above from *Variation* make clear, Darwin had before him the basis of a saltational theory that was even supported by evidence of heredity: not only could "monstrosities" interbreed successfully, they could also reproduce with "normal," parental–type individuals, and thereby perpetuate their novelties. Observations to the contrary, Darwin's

constant assertions of a seamless web having existed among living species as well as between descendents and their extinct ancestors might betray a non–biological concern. Namely, were he to embrace saltation, the door would remain open for his religious contemporaries to invoke special creation to explain the abrupt appearance of species in the fossil record as well as the discontinuities between extant taxa. It is, therefore, perhaps a bit ironic that an intellectual enterprise – neo–Darwinism – that went and continues to go out of its way to denegrate and discredit alternative, saltation–like theories for the origin of novelty was built on such an imaginary foundation.

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Message in a Bottle: The Business of Vaccines and the Nature of Heredity after 1880

Andrew Mendelsohn

Introduction

Before the 1880s there was no consensus on the nature of microorganisms. Some students of bacteria saw constant species. Others saw a flux of form and function. Each *saw* in the sense of both literally seeing through the microscope and perceiving in relation to rival biological theories or classifications. Differences in view were thus at once descriptive and theoretical. Violent debates raged. Research projects arose to great influence and just as quickly came crashing down. It is usually said that the transformists/"unitarians" lost (the botanist Carl Wilhelm Nägeli, the surgeon Theodor Billroth, many lesser–knowns) and the "Linnaeans" won (the botanist Ferdinand Cohn, the physician Robert Koch, their followers) – and that this made possible a science of bacteriology at all, relieving biology of the notion that, in Nägeli's oft–ridiculed formula, all microscopic life was a single species in continual flux of form and function, producing sometimes souring of milk, sometimes butyric fermentation in sauerkraut, sometimes the aging of wines, sometimes cholera, and so on. That is the standard story.[1]

Yet in fact during the 1880s, as I have argued recently, far from one view winning out over the other, a consensus was rapidly achieved in which neither of these views prevailed. Instead, bacteria were seen to vary *within* species.[2] How could this have happened? How could an international and notably French–German consensus on a supremely controversial and uncertain topic – bacterial species – have been so rapidly forged? This historical problem generates the following essay.

My answer is, in a word, vaccines. I shall argue in the first part of this paper that an unusually powerful model of species and variation – a model in the sense of both exemplary case and research object – was inadvertently provided by early vaccines, notably the anthrax vaccine invented by Pasteur in 1880–81 and then mass–produced and distributed, manipulated in laboratories as well as used on farms around the world.[3] In this process, as I shall argue in the second part of the paper, heredity (at least in the realm of microscopic life) came to be located within and redefined by an enterprise of control and testing, production to an exact standard and reliable distribution. This enterprise was akin to yet differed in revealing ways from animal and plant breeding, which in this period became all–important to the making of a biological science of heredity. The resultant new meaning of heredity suggests historical change related but not equivalent to the well–known shift from "soft" to "hard" heredity, or from heredity as force to heredity as structure.[4]

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1 For "unitarians" *versus* "Linnaeans," see Mazumdar 1995, which revises the standard story by showing that the two traditions long continued their conflict but in a series of other research domains, such as immunology and serology.

2 Mendelsohn 2002.

3 Comments welcome as to whether the history of science offers earlier examples of a standardized, mass–produced and distributed research object, as distinct from instruments. The answer would be a definite yes if (some?) chemical reagents count as research objects rather than instruments.

I. Manufacturing Variation

In a series of papers in 1880 and 1881, Louis Pasteur reported that he had produced weakened or "attenuated" cultures of the microorganisms of fowl cholera and anthrax, which conferred immunity to these diseases when inoculated in animals. He called these cultures of attenuated virulence "vaccins." This work is celebrated as the beginning of immunology and artificial immunization. Yet it also had other profound if less remembered implications. Bacteriological workers in many countries began manipulating the virulence of their cultures and soon enough other microbial properties such as morphology, pigment formation, colony form, capacity to liquify gelatine, and sporification, by altering the recipe of the culture medium or the time–span between resowings in fresh medium, or the temperature, or the exposure to air, and so on. They interpreted many of the observed changes in form and function as biological variation.

Thus much early medical bacteriology became a science of what the preeminent French veterinary scientist Auguste Chauveau called "experimental variation."[5] Entire chapters of the standard German and French bacteriological handbooks were soon devoted to variation or "variability."[6] This is remarkable on two counts. First, the received view has been that a dogma of species constancy made bacterial variation unthinkable or at best heretical until the early twentieth century. Second, biology at large could boast nothing comparable. In the early 1880s, when Wilhelm Roux and other academic scientists had barely begun to preach a new biology as experimental laboratory science; when questions of species, variation and inheritance were still being pursued largely through observation in field and museum; when Mendel would not be rediscovered and classical genetics begin for another 20 years, dozens of physicians more or less remote from academic biology and working in mostly medical and public health laboratories around the world, were building a system of sustained cellular–level, *in vitro* experimental research on what they and their contemporaries saw as biological variation and indeed mechanisms of evolution.[7]

What was so special about virulence work? How did it allow observers to see change in bacterial properties as variation *within* species, rather than contamination or "transformism"? There are two answers to this question. Variable virulence modelled variation *within* species because it was observed to correlate with variable severity of what remained clinically distinct, or specific, diseases: bacteria that were attenuated or augmented in their virulence and morphologically sometimes less than uniform could still be used to provoke a predictable set of clinical effects in animals, varying only in their *severity*.[8] And secondly, fully attenuated, no longer pathogenic organisms did not have to be seen as transformed into different species because they exhibited their original species identity by acting as specific vaccines when injected into animals.[9] (Total loss of pathogenicity was no minor species issue: recall that these organisms were often even named after their associated diseases, as in *Bacillus anthracis*.) Thus the identity and stability of

4 See Gayon 1995.
5 Chauveau 1889b, 789.
6 The most important of these was Kruse 1896b.
7 See Mendelsohn 2002, 18–26.
8 Pasteur 1880c, 324–26.
9 Chauveau 1889a–b.

biological species was guaranteed not by a botanical classification system and rules and skills of biological observation, but by clinical and vaccinal practices and effects. Microbial variation existed as a *biological* phenomenon not despite, but because of its medical, even clinical identity. This is not meant as a subtle point. Scientific knowledge took shape through a practical matrix in a way akin to the role of the steam engine in the origins of thermodynamics, or telegraphy in the rise of electromagnetic field theory, a comparison on which I shall elaborate below.

All this was accomplished, inadvertently, more by the distribution of vaccines than by the distribution of journal articles reporting methods and results, of instruments and visual representations, or even of skill and tacit knowledge through circulation of laboratory personnel.[10] Pasteur's celebrated announcements and publications of 1880–81 certainly did not convince his rival founder of bacteriology Robert Koch or lead to experimental confirmation. Koch and his disciples were unable to achieve attenuation in their own laboratory: they reported culturing anthrax bacilli on gelatine for fifty generations and on a potato surface for nine months, through over one hundred generations, without observing any alteration in virulence. They charged that so–called "attenuated" cultures were in fact merely contaminated with common non–pathogenic organisms.[11] Evidently, experimenting on virulence was neither practically nor conceptually coherent and plausible enough to be replicated, much less to serve as a model case for variation in other bacterial characteristics. On the theoretical side, there was no agreed–upon concept for the mechanism of hereditary change. Bacteriological researchers were soon able to use a wide range of often incompatible concepts: variation, progressive modification, acclimatization, race formation, adaptation, degeneration, transformation, and not least, selection in the Darwinian sense. This smorgasbord of biological concepts and theory could not provide the coherence of the phenomenon. Moreover, "variation" was itself a heterogeneous category at the time. Biologists hotly debated the origin and nature of variations, as well as the kinds of variation (continuous *versus* discontinuous). Bacteriologists drew on a concept that was itself ambiguous. Thus it was not the case that the virulence and vaccine model allowed bacteriologists to assimilate their phenomena to some settled standard 'biology' of their time. On the practical side, there was no standard unit of measure, no "meter" or "ohm" of virulence.[12] Nor was there a uniform method of attenuation: all sorts of things were done to bacterial cultures and over a range of times.

The anthrax vaccine, on the other hand, *was* uniform. You could buy a bottle on the market – "käuflich im Handel" as Koch put it[13] – and test it on animals, or try to alter it by passing it through animals in series, or use it as the known starting point for an experimental manipulation in culture. Following the spectacular success of the public trial at Pouilly Le Fort in 1881, requests for vaccine poured into Pasteur's laboratory.[14] His associate Charles Chamberland began large–scale production. Sales and distribution were handled by a commercial agent, Boutroux, in Paris. Tens of thousands of sheep and cattle were vaccinated in France in 1882 alone. Within 10 years 3.3 million French sheep and 438,000 cattle had been vaccinated. In Italy, the government provided

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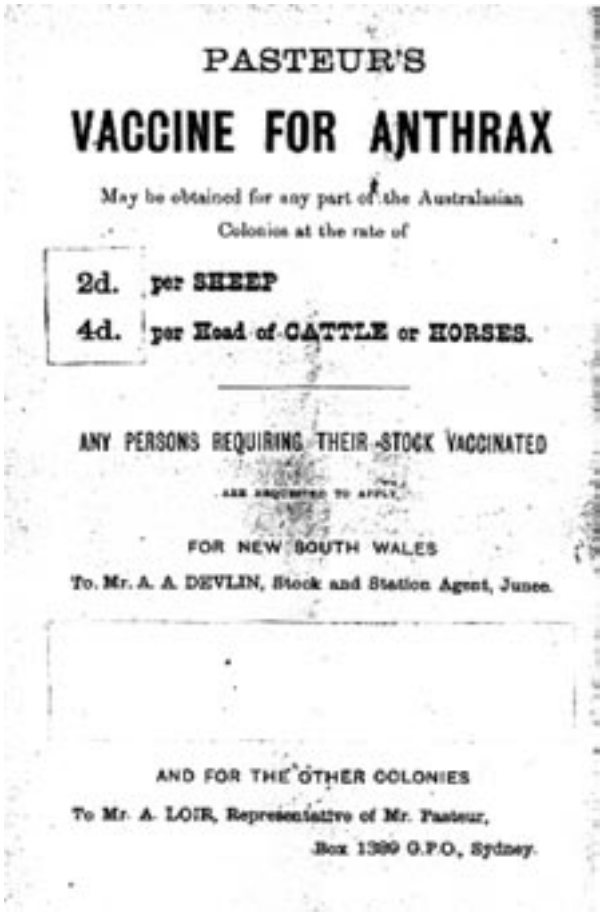
10 Cf. Collins 1985; Latour 1987.
11 Koch 1881, 200; Gaffky 1881, 121–126; Loeffler 1881, 134–141.
12 See Kruse 1896a, 299, for one attempt to define a scale of six "Virulenzstufen" by delimiting sets of microscopic pathological changes in animals.
13 Koch, Gaffky and Loeffler 1884, 247.
14 See Pasteur's published correspondence for July 1881; Valery–Radot, 3:220.

Pasteur's vaccines free of charge. Production laboratories were established in places near and far – Austria, Spain, South America, Russia, Australia.[15] (See figure 1.)


PASTEUR'S

VACCINE FOR ANTHRAX

May be obtained for any part of the Australasian Colonies at the rate of

2d. per SHEEP

4d. per Head of CATTLE or HORSES.

ANY PERSONS REQUIRING THEIR STOCK VACCINATED

ARE REQUESTED TO APPLY

FOR NEW SOUTH WALES

To Mr. A. A. DEVLIN, Stock and Station Agent, Junee.

AND FOR THE OTHER COLONIES

To Mr. A. LOIR, Representative of Mr. Pasteur,

Box 1389 G.P.O., Sydney.

Fig. 1. Loir, Adrien, Pasteur's Vaccine of Anthrax in Australia: as a preventative against Cumberland Disease in sheep, cattle and horses, published c.1891, Sydney, Back Cover Advertisement.

Boutroux distributed the bottles to farmers, but also to scientists. (Researchers also obtained from Boutroux the Pastorian vaccines for fowl cholera and swine erysipelas, though these played a far lesser role in the story told here.) Reference to Boutroux crops up everywhere in the relevant bacteriological, medical, and veterinary journal literature of the 1880s. For the scientists, Boutroux's agency and all the work by Chamberland and his assistants that went into those bottles – and of course the procedures and skills detailed in instruction manuals and diagrams (see figure 2.) – made the bottles' contents a model arguably unprecedented in its distribution, stability, and unequivocality as witnessed by the dramatic and repeatable set of effects produced by following the instructions and inoculating from the bottles into animals.

[15] M'Fadyean 1894, 327.

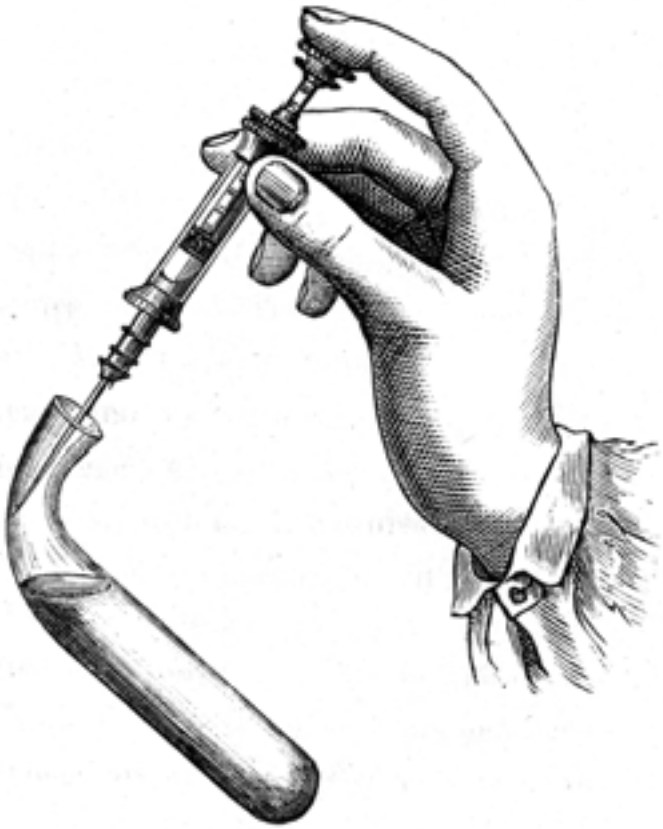


Fig. 9.

Fig. 2.

Only after attenuated cultures began to be distributed and used as vaccines did phenomena of attenuation become 'real' and powerful enough to be recognized and studied by Koch and his associates, rather than dismissed as mere contaminations. Before distribution of the vaccine, even a less sceptical, English bacteriologist who did manage to achieve sporeless anthrax cultures harmless even to mice nonetheless could not confirm that this attenuation was "transmitted" to the next generation.[16] As late as 1888, researchers in the laboratory of Koch's colleague Carl Flügge at Breslau, working on the nature of the attenuation process, were unable to attenuate swine erysipelas bacilli and obtained them instead as vaccines from Boutroux.[17] It is remarkable that Koch and his associates first failed to replicate Pasteur's results and then succeeded *after* the vaccine was publicly tested at Pouilly Le Fort and hit the international medical marketplace. Koch and his disciples obtained vaccines from Boutroux in Paris, an especially well thermostatically controlled incubator from the firm of Wiesnegg also in Paris, which could maintain a temperature

[16] Klein 1883, 9, 64 (paper based on author's 1881 report to Local Government Board).
[17] Smirnow 1888, 244.

"without the slightest oscillations" for weeks, and proceeded to attenuate fully one of their own anthrax cultures and inoculate it into sheep. Now Koch saw and represented a biological phenomenon of hereditary change. He affirmed that "the high scientific importance" of Pasteur's discovery of attenuation lay in the fact "that the new properties are also preserved in the progeny [*Nachkommen*] of the attenuated bacilli." This was "of the highest interest not only for etiological research, but equally for biological science."[18] Hans Buchner, student of the transformist Nägeli, glimpsed why Pasteur's experiments had become, even for Koch, real phenomena of hereditary modification of microorganisms, whereas Buchner's own similar earlier results had been ignored or rejected. The reason was "that Pasteur's protective vaccinations, which are now being practiced – even if at first only in trial form – in nearly all European countries, made it impossible even for Koch to continue to doubt the existence of an attenuated anthrax bacterium."[19] Pasteur's vaccine business distributed not only prophylaxis against disease, but also inadvertently this message in a bottle. Though to the eye nothing but clear liquid in a bottle, the mass–manufactured vaccine culture served as a model in several senses: (1) as an object for demonstration, available virtually off the shelf as needed to show variation within bacterial species; (2) as an exemplary case rendering variation of other microbial characteristics plausible and investigable without casting doubt on the rigor of one's methods or the existence of bacterial species; (3) as a live research object to be worked upon *in vitro* or *in vivo*, whose uniformity, purity, known production method and initial properties made it manipulable in predictable yet exploratory ways.

The bottles were not, of course, at sea. They were part of wider human organization. There is an illuminating analogy to the physical sciences. Like the early physical standards laboratories of the same period, vaccine laboratories helped make a world in which local science could become global. They distributed a standardised material that could be tested and manipulated – with predictable outcomes – in other laboratories as well as used on farms. And with this material, a set of techniques and gestures (see figure 2). In this sense, the Pastorians' vaccine laboratory and their commercial agent and distributor, Boutroux, became like the Cavendish Laboratory at Cambridge, which James Clerk Maxwell did not wish to become a "manufactory of ohms" (the unit of electrical resistance) but which in effect did.[20] Yet unlike the calibration and distribution of electrical measurement devices, the manufacture and distribution of vaccines fundamentally recast the nature of the phenomenon. It changed what sort of descriptive and theoretical statements could plausibly be made. Nature had to be rewritten: the harmless "hay bacteria" species which Buchner had so recently transformed out of anthrax bacteria were now – with no admission of error or retraction of previous claims – "my attenuated anthrax bacteria."[21] Microscopical biology's mountainous intellectual terrain of theoretical divides and commitments and antagonisms was ironed flat.

The explanandum in most history and sociology inspired by Bruno Latour is: How and why does science work? By contrast, my explanandum here is the changing specific content of a science. The successful anthrax vaccine, one of Latour's main *explananda* in his studies on Pasteur, is my *explanans*. In this sense, the distribution, use and monitoring of vaccines, the network of vaccine

[18] Koch 1882, 216–18, 227–28; see also Koch, Gaffky and Loeffler 1884, 233.
[19] Buchner 1883, 411.
[20] Schaffer 1992; Latour 1987.
[21] Buchner 1880; Buchner 1882, 253.

producers and consumers, functioned more as telegraphy in relation to the rise of electromagnetic field theory than the Cavendish in relation to the ohm. Historian of physics Bruce Hunt has shown field theory to have arisen through telegraphy as it was made to work and put to work in British projects of empire and trade.[22] A new biology of microscopic life, I suggest, emerged through vaccines as these were developed and produced in the 1880s and '90s for animal industry, hygiene, and colonialism. Animal vaccines' potential relevance to the "success of colonization" was apparent as early as 1881, when the French Association for the Advancement of Science met in Algiers and thus, in the words of its president, followed in the footsteps of "French arms" to "take possession, in its turn, of this land Africa," a continent which science and the *oeuvre civilisatrice* were rendering nothing less than "France, extended across the sea." Somewhat less predictably, the president continued his address with the question: "What is a virus?" Auguste Chauveau, director of the prestigious National Veterinary School at Lyon and the most important researcher on attenuation outside of Pasteur's group, launched into a detailed account of Pasteur's discoveries (and those of his own research school) culminating in "the permanent and transmissible attenuation" of pathogenic microorganisms.[23]

These spheres of activity, by the same token, provided bacteriology with conceptual tools. From colonialism and its zoological and anthropological sciences, bacteriologists took the concepts of acclimatization and race; from medicine, those of "degenerate" and "abnormal"; from agronomy, as we shall see below, the breeder's concept (and aesthetic–economic ideal) of "fixed" varieties. These terms were transfered from man (and animals) to bacteria, which themselves were already seen as growing in "cultures" and "colonies."[24] At a joint meeting of the five French academies, Henri Bouley explained that in Pasteur's vaccine cultures, attenuation became "a race character" and thus these microbes are "races degenerated from their original power and become beneficial by their very weakness."[25] Evoking contemporary anthropological stereotype, Emil von Behring, in the year he received the first Nobel Prize in medicine, argued that changes of culture medium and environment yielded "statistically almost more abnormal than normal diphtheria bacilli" and dismissed the idea of conformity to a "normal type" as the equivalent of expecting the human race to conform to the "Apollo Belvedere."[26] Agronomy, medicine, and colonialism were not only the matrix, but became also the content of bacteriology. Using the language of acclimatization, race, degeneracy, and abnormality, bacterial change could be described in terms that left species untouched; all these terms came to be ordered under variation.[27]

Neither a celebrated experiment, nor a type–specimen located in one laboratory or museum, nor the application of any biological theory or classification system, but rather an increasingly global scientific and commercial matrix of practice and meaning, of vaccine bottles and bodily

[22] Hunt 1991. With the difference that empire and telegraphy created what Hunt called a "market" for the *reception* of field theory already created by Faraday, whereas I am arguing that the new biological content of bacteriology and the nature of its phenomena was created through vaccine–making and the vaccine enterprise.

[23] Chauveau 1881, 482–83, 490.

[24] A good early example of frequent use of the term bacterial "colonies" is Eberth 1872.

[25] Bouley 1882, 547; see also Chauveau 1885, 355. The language of race continued to be used routinely in technical publications in the twentieth century; e.g., Pfeiffer 1903, 37: "Rassen des Choleraerregers," "Cholerarassen."

[26] Behring 1901, 81–82.

[27] See Mendelsohn 2002, 18–24.

effects, sustained the phenomenon of bacterial hereditary change or "variation" and consensus about it. Without that matrix, variation within bacterial species would not have been merely a neglected topic or heterodox viewpoint. It would not have – indeed, had not – existed as an object of inquiry. Students of bacteria had not seen or conceived of such a thing: they had seen either immutable species or protean mutability. Likewise, later students of bacteria, working within a different matrix, would *stop* seeing it. In the early twentieth century, the unifying vaccine model was displaced by the rise of routine bacteriological diagnosis for endemic diseases and the application to bacteria of the mutation theory of Hugo de Vries.[28] Bacterial "variation" began to fragment into myriad phenomena: mutation and dissociation, cyclogeny, phage effects, and transformation of type, and eventually enzyme adaptation and transduction.[29]

II. Fixing Heredity

In the second part of the paper, I turn from hereditary change to heredity itself. In modeling variation, vaccines modeled hereditary change and thus phenomena of inheritance, at least implicitly. Implicitly, because bacteriologists rarely contributed to the explosion of scientific writing on heredity that characterised the last decades of the nineteenth century. In the absence of such writings, what I offer here is, instead, the intellectual history of practices. The historian can interpret what the hereditary meant and how it changed by looking at practice and its rationales: not *how* things were done (as in much of the past twenty years' work on scientific practice, material culture, tacit knowledge, skill, training), but *which* things were done and *why*.

Why exactly did bacteriologists regard attenuation and return to virulence as hereditary changes rather than merely physiological adaptations? – as in Claude Bernard's experiments on the physiological adaptability of animals to lack of oxygen under a belljar, which Pasteur liked to cite.[30] The simple answer seems to be time – the multiple generations through which microorganisms could be observed to pass and over which a given degree of virulence or attenuation could be found to persist. At first, in Pasteur's earliest attenuation work on fowl cholera, and among other researchers circa 1880–82, persistence from one cultivation or "generation" to the next was indeed the criterion for inheritedness; it showed that "the bacillus having become modified by time, transmits to its offspring this acquired mitigation."[31] As late as 1882, reporting his own experiments, Koch gave no other criterion for inheritance than the regular, stepwise (*Stufen*) character of the attenuation process over the passage of time and thus bacterial generations.[32] Soon enough, however, time ceased to be an adequate criterion. Increasingly, modification was more consistently and confidently judged hereditary if it persisted *under changed conditions.*

Why? In 1884, for example, in their major research paper on anthrax vaccination and immunity, Koch and his associates now reported on anthrax cultures attenuated in his laboratory

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28 This theory–practice relationship has been explored by Olga Amsterdamska 1987.
29 For an overview, see Brock 1990.
30 Pasteur 1876, 241; Duclaux 1920, pt. 6.
31 Klein 1883, 65 (based on his government report of 1881). See Pasteur 1880d. Modification and persistence over many "generations" was also the criterion for those claiming species transformation; see Buchner 1880.
32 Koch 1882, 217. See also *Jahresbericht 1882*, 1883, p. 13; *Jahresbericht 1883*, 1884, p. 15.

and then cultivated at room temperature for two years, during which time they never regained their virulence. From this he concluded that the attenuation was "passed on from generation to generation [*von Generation zu Generation weiter vererbt*]."[33] Without much ado, Koch made persistence under *changed* conditions the test for – and thus in effect the nature of – heredity. Others cited him in agreement.[34] Now, persistence under changed conditions was in fact a paradoxical criterion: if changing the conditions created the hereditary variety in the first place, changing the conditions again or restoring them, would not test inheritance, since it ought simply to yield a new hereditary variety (or the original); this was in fact something Pasteur had explicitly shown he was able to do, namely return attenuated anthrax or fowl cholera microorganisms to virulence by passaging them through animals, beginning with the weakest possible ones and moving in series to more and more robust creatures.[35] But the objection was not made. And no paradox was perceived. Why not? The answer in a word, again: vaccines. What does make sense of this criterion of heredity is to suppose that it was modeled on – or indeed synonymous with – vaccine safety and efficacy.[36]

Thus the question: Are modifications of virulence hereditary? was in fact increasingly being asked as: Can we produce safe and effective vaccines? Will there be accidents? Might vaccines, injected into animal bodies, regain their virulence and thus become a danger? Sure enough, the criterion of persistence under changed conditions (such as when cultured as usual or when injected into an animal) first appears in Pasteur's publications at precisely the point at which he asks whether the attenuated fowl cholera microbe could be "a true vaccine, comparable to the cowpox vaccine." And the criterion was formulated at first as a test not for inheritedness, but simply for useability as a vaccine.[37] In practical vaccine research contexts, this criterion was emphasized and, as we saw with Koch and his associates, became the criterion for inheritedness.[38] By the same token, in theoretical contexts, in research projects designed to "explain" the process of attenuation rather than develop vaccines, the criterion of persistence under changed or usual conditions was not emphasized.[39]

Despite the confidence of Pasteur's various pronouncements, these life–and–death questions could not be resolved quickly in the laboratory or by public vaccine trials such as the celebrated demonstration at Pouilly Le Fort. They remained the questions of the day in the veterinary societies, journals and government bureaux.[40] And these became criteria for judging the merits of various methods of vaccine–making, a hotly contested arena.[41] Koch and others reported that

33 Koch, Gaffky and Loeffler 1884, 236.
34 For example, Beumer 1887, 1–2.
35 Pasteur 1881, 336.
36 The terms vaccine "safety" and "efficacy" are taken from M'Fadyean 1894, 331; for another example, see Flügge 1888, 209: "Gefahrlosigkeit und Sicherheit." An alternative reason for changing the criteria would be to meet the objection that persistence of a given degree of attenuation could indicate merely that each new generation of organisms was undergoing the same purely physiological change in response to conditions, rather than inheriting the degree of attenuation. But I have not found this argument in the sources.
37 Pasteur 1880a, 299–300. Interestingly, at this early stage, Pasteur refers not to safety but to the "fear," once had by Jenner concerning cowpox, that one would have to return always to the original preparation.
38 See, for example, Beumer 1887, 1–2.
39 Flügge 1888, 208–15.
40 Börner 1882, 698; Rózsahegyi 1882, 27; Koch, Gaffky and Loeffler 1884, 261.
41 Geison 1995, chap. 6.

cultures attenuated using Pasteur's young and hapless rival Toussaint's method of heating at relatively high temperatures (47 or 50 or 53 degrees celsius) tended eventually to return to virulence. Cultures attenuated using the Pastorians' method of heating at only 42–43 degrees celsius over longer periods kept ("bewahren") their attenuated state even "in later generations" and in the spores. The lower the temperature, "the more securely [*sicherer*] the physiological varieties seem to keep their [new] properties."[42] "More securely," *sicherer*: at work here was the traditional conception of heredity as a force, as varying in strength, as having degrees.[43] I shall return to this point below and in the conclusion.

These were issues of vaccine production. There were also issues of vaccine distribution. Pasteur's production and distribution chief, Chamberland, outlined these too in a chapter on "variation in the virulence of the anthrax vaccines." Might vaccines become too weak and thus be ineffective and unsafe? The biological stability of varieties was synonymous with successful "conservation."[44] The "theory of virulence" one former Koch student presented, years later, to the master on his sixtieth birthday was equally a method of "conserving virulence *in vitro*" rather than by costly and time–consuming continual animal passage.[45] The terminology of *conserving* and *conservation* was at once the language of the technical and social entreprise of storage and distribution and a language for describing the bacterial cultures themselves: "they will conserve their own virulence."[46] Chamberland aimed at a system in which bottles of vaccine could be "expedited throughout the world, as far as the farthest countries, retaining [*gardant*] their preservative properties," a network of global biological transport. He reported that the Pasteur team studied the conditions "of conservation" and the causes of vaccine instability throughout 1882. They concluded that the vaccine must be "fresh" or "recently prepared," or if the second vaccine, then veterinarians should put it "in a cool place [*au frais*], in a cellar for instance," without opening the tubes and using within 12–14 days. Vaccines could, however, be prepared with "minute" rigor such that they would last two or three months. Ultimately, however, the goal would be to build myriad "little factories" in far–off countries.[47]

When accidents did occur, it was said, for example at a meeting of the French veterinary society in 1882, that Pasteur's "vaccines were not fixed as he had hoped."[48] The pages of Chamberland's 1883 chapter on "Variation in the virulence of the anthrax vaccines" bore the header "Relative fixity of the vaccines."[49] *Fixity*, the breeder's concept and aesthetic, economic ideal of "fixed" varieties, is a keyword for understanding how the vaccine model changed the meaning of heredity in the world of microscopic life. The currency of the term in these discussion may have come from the veterinary context of anthrax vaccination, but presumably also from the Pastorians own prior twenty years' work in agricultural science and industry, which had made

42 Koch, Gaffky and Loeffler 1884, 250, and see 252 ("Varietäten").
43 The same terminology occurs in Haeckel's laws of heredity: "um so sicherer und vollständiger auf alle folgenden Generationen vererbt" (Haeckel 1866, 187).
44 Chamberland 1883, chap. 30.
45 Pfeiffer 1903, 48: "Virulenzkonservierung im Reagenzglase."
46 Chamberland 1883, 282; see also Pasteur 1880a, 299–300 and 1880c, 327, and reports in veterinary journals, such as Bouley 1881, 405; Rózsahegyi 1882, 24; *Jahresbericht, Jahr 1881*, 1882, 10.
47 Chamberland 1883, 282, 295–96.
48 Société centrale de médecine vétérinaire, séance 8 juin 1882, "Sur certains accidents consécutifs à la vaccination charbonneuse," reprinted in Chamberland 1883, 290.
49 Chamberland 1883, 284–295.

them well aware of the theory and practice of animal and plant breeding, especially concerning grape vines and silkworms. For an attenuated fowl cholera culture to be a vaccine, Pasteur wrote in his first paper on attenuation, "It would be necessary, if I may so speak, that it were fixed in its own variety [*fixé dans sa variété propre*] and that one would not at all be constrained always to go back to its original preparation when one wishes to use it."[50] Later, anthrax inoculation "accidents" showed that given batches of anthrax vaccine were not "absolutely fixed," not yet "true races with fixed characters." With Jenner's vaccine, one did not have to fear "these atavistic returns to the primitive virulence." A safe vaccine was "a family of virus in which the attenuation is fixed by heredity."[51] *Fixée par l'hérédité*, "fixed *by* heredity": again, as in Koch's conception, heredity was a force. Yet it was also capable of transcending its own nature as a phenomenon of degree to achieve fixity, the cessation of degree.

Despite bacteriologists' adoption of the keyword *fixed/fixity*,[52] the relation between the business of vaccines and that of animal or plant breeding was not straightforward. Whereas the bacteriologists came to imply that variety was truly hereditary only when it was "fixed," for the breeder, to fix a trait was not to make it hereditary but to stabilise hereditary change (or maximize the force of heredity) and thus prevent regression or "atavism." The term *fixed* connotes inalterability, but in fact it belonged to the conception of heredity as a force ranging from weak to strong. As Darwin glossed: "fixedness of character, or strength of inheritance."[53] The French plant breeder and leading seed company family Vilmorin acknowledged that the constancy of a breed depended on continued selection,[54] and Hugo de Vries emphasized this as the "universal experience of breeders."[55]

Vaccines were different. Unlike the breeder whose pedigree animals or seeds passed into the hands of buyers equally interested in maintaining the product, the vaccine–maker had to let go his product into the wild. His control ceased at the point of inoculation into millions of animal bodies. Thus constancy of characters after cessation of controlled culture was not a happy exception or a distant ideal, but an immediate life–and–death necessity. Thus although productive yield and profitability or economic viability of plant and animal breeds were roughly analogous to safety of vaccines, bacteriologists' pursuit of vaccine safety and efficacy was turning the question of whether something was hereditary into a yes/no question, rather than a question of degrees of strength, or indeed degrees of "certainty" or "uncertainty."[56] Fixed *versus* unfixed, rather than fixity as a high degree, Darwin's "strength of inheritance."

Speaking with science studies or history and sociology of science, we could say at this point that vaccine safety and efficacy constructed, produced, constituted heredity as fixity and in a new, absolute sense. Yet this language would be too socially and morally neutral: the stability in

[50] Pasteur 1880a, 299; see also Pasteur and Thuillier 1883, 531: "chacan de ces états [de virulence] est suceptible d'être fixé par la culture"; Smirnow 1888, 242: "Es gelang mir, auf diese Weise drei gut unterscheidbare Virulenzstufen zu fixiren."

[51] Chauveau 1885a, 617, 620–621; see also Cornil and Babès 1890, 242.

[52] See the introduction to the most important literature review on "variability": Kruse 1896, 476: "*Befestigen* lassen sie [die Varietäten] sich durch Wiederholung der Züchtung in alten Kulturen" (emphasis added).

[53] Darwin 1888, 2:47.

[54] Gayon and Zallen 1998, 260

[55] De Vries 1906, 787.

[56] For "certainty" and "uncertainty," see Gayon 1995, 64.

question was not just technical or epistemic stability, but safety. The nature of heredity, at least among the microscopic organisms, was coterminous with securing against accidents, shouldering responsibility for animal welfare and industry, minimising the potential for lawsuits or disputes with angry farmers or governments, ensuring commercial success for a vaccine over its competitors. It was the flip–side of these practical, medical, economic goals and the system of techniques and organisation designed to make those goals realizable. In this sense, the nature of heredity has a social and technical history. To quote from an introductory section in the most important bacteriological handbook around 1900: When "the artificially induced loss of virulence [becomes] a lasting property of the strain, transmitted from generation to generation . . . we call such strains *vaccins*."[57] Shorn here of its medical meaning, the word *vaccins* could be equally a biological term.

To conclude: Are the contours of this story limited to the history of microbiology, or is there wider significance for the history of heredity? In 1866 phenomena of "fixity" (*Befestigung*) and methods by which organisms could be "'pure' cultured" (*"rein" fortgepflanzt*) still belonged to but one of nine "laws" of heredity outlined by Ernst Haeckel, and they remained firmly within the conception of heredity as force.[58] After 1880 the pursuit of vaccine fixity, of safety and efficacy, exemplified a shift from heredity as a force, a phenomenon essentially of degree, toward heredity as a phenomenon essentially of presence and absence – a world in which Johannsen's "pure lines" and genotype/phenotype divide, Mendel's laws, and the search for units of heredity would soon make the most sense and command the chief attention.[59] For the history of biology, breeding has most obviously been important in two ways: (1) in the formulation of Darwin's theory partly through the analogy to artificial selection; (2) in the making of Mendelian genetics as a science whose main practice was the breeding practice of hybridisation – using or making "true" varieties to cross with one another and keeping records of the results. But the vaccine story suggests another relationship of practical breeding–like enterprises to biological science in this period. Here was one important area of science in which heredity was freed from environment and atavism alike and coming literally to mean permanence. Bacteriologists routinely contrasted "temporary" and "permanent" modifications.[60] And this permanence was not found through observation of the workings of nature in the wild or even the research laboratory as such, but created through a practical enterprise of making things reliable, context–independent, accident–free. Heredity would be like a safe, effective vaccine.

The vaccine story may be an example of how agricultural and other enterprises of biological stabilization and standardization in this period contributed to this shift in the meaning of heredity – a shift that was independent of hybridization and the origins of Mendelism; independent of the "hardening" of heredity through the rejection of inheritance of acquired characteristics by August Weismann and others; independent of cytology and of biological theories of subcellular structures

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57 Wassermann 1903, 248.
58 Haeckel 1866, 187–88: "Gesetz der befestigten Vererbung." I thank Wolfgang Lefèvre for directing my attention to Haeckel's heredity laws.
59 This shift is related but not identical to the shift from "force" to "structure" identified by Gayon 1995.
60 See, for example, Smith 1894. For a biologist's language of "temporary" versus "durable" characters and "permanency," see de Vries 1906, 774, 786.

and units of heredity. This would not be merely another example of how scientific phenomena are stabilized at any given historical moment. For here was a historical change that was itself a process of stabilization, a shift away from ambiguity and degree. "True" heredity would seem to be less an ideal or proposition or a perception of nature as such, than something whose existence was warranted or predicted by such enterprises as vaccine–making, animal and plant breeding or domestication, perhaps also human breeding. If the setting into motion of people and objects through natural–historical and agricultural projects, trade, colonialism, industrialisation and urbanisation was a key determinant of the history of heredity in the early modern period,[61] in the 19th century an ever increasingly man–made world of controlled and stabilized variety would seem to be crucial. No wonder it seemed to Wilhelm Johannsen, who displayed his debt to breeders such as Vilmorin, "daß die Verhältnisse der reinen Linien das eigentliche Fundament der Erblichkeitslehre sein müssen, selbst wenn man in den meisten Populationen – vor allem in der menschlichen Gesellschaft – überhaupt nicht mit reinen Linien zu tun haben kann."[62] His pure line methodology was a scientific expression, a research–program residue, of this man–made world. A similar contrast had impressed itself upon Darwin, writing of inheritance:

> If animals and plants had never been domesticated, and wild ones alone had been observed, we should probably never had heard the saying, that 'like begets like.' The proposition would have been as self–evident as that all the buds on the same tree are alike, though neither proposition is strictly true. For, as has often been remarked, probably no two individuals are identically the same. . . . The saying that 'like begets like' has, in fact, arisen from the perfect confidence felt by breeders, that a superior or inferior animal will generally reproduce its kind . . .[63]

Darwin continued and nuanced the picture in an important way: "Inheritance is not certain; for if it were, the breeder's art would be reduced to a certainty, and there would be little scope left for [his] wonderful skill and perserverance." And yet in the end, amidst the ambiguities of the "wild," of what Johannsen called "most populations," one could observe that, "Hard cash paid down [for prize animals], over and over again, is an excellent test of inherited superiority."[64] Those "excellent test[s]" – hard cash paid again and again, vaccines safely injected by the millions – made clear heredity's nature for contemporaries in ways that biological theorizing and experimenting alone could hardly do. It was by no means the only nature of heredity being revealed at this time.

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61 Müller–Wille and Rheinberger 2004, 13.
62 Johannsen 1903, 9. Cf. Gayon and Zallen 1998, 244, 260, who challenge the similarity between fixed breeds and pure lines.
63 Darwin 1888, 1:531–32.
64 Darwin 1888, 1:534.

Quite the opposite, de Vriesian mutationism (with its own roots in a different horticulture) was both an argument against the reality of breeders' stable varieties and an attempt to reestablish biology on the basis of genetic instability.[65] Heredity by 1903 bore a Janus face, reliable and mutable.

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[65] Undermining the breeding analogy to the origin of species was necessarily as important to de Vries as establishing it had been to Darwin; de Vries 1901–03, 1:4–9, 86–87, passim; de Vries 1906, Lecture 27.

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The Chromosomal Theory of Heredity and the Problem of Gender Equality in the Work of Theodor and Marcella Boveri[1]

Helga Satzinger

In the early years of the twentieth century Theodor Boveri (1862—1915) and Walter Sutton (1877—1916) proposed the chromosomal theory of heredity. In his paper 'Ergebnisse über die Konstitution der chromatischen Substanz des Zellkerns', published in 1904, Theodor Boveri summarised the results of two independent fields of research: experimental cytology on the one hand, which was his field of expertise, and Mendelian hybridisation experiments on the other. At the end of his 130-page paper he came to the conclusion: 'The probability becomes extraordinarily high that the characters traced in Mendelian experiments are actually bound to specific chromosomes'.[2] Working independently, Sutton and Boveri correlated the behaviour of chromosomes during cell cleavage, germ cell development, and cell fusion in fertilisation to the recently rediscovered laws of Gregor Mendel.[3] In doing so, they laid a cornerstone of modern genetics, as our textbooks on the history of biology tell us. The localisation of the hereditary material in the chromosomes became the precondition of all gene-mapping projects of the twentieth century, the first one starting in 1911 with the famous fruit fly *Drosophila melanogaster* and a recent one being the HGP with humans.[4]

Yet, upon a closer examination of how Boveri established the chromosomal theory of heredity, things do appear confusing. First of all, Boveri did not call his theory the 'chromosomal theory of heredity', he gave no name for his combination of two fields of research. In 1904 he was proposing the 'theory of chromosomal individuality', attributing to each chromosome a specific relevance

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1 This paper is part of an extended work on the history of genetics from the perspective of science and gender studies. I want to thank Staffan Müller-Wille, Karin Hausen, Christiane Eifert and my colleagues at the Wellcome Trust Centre for the History of Medicine at UCL for critical and supportive comments.

2 Boveri (1904), p. 117: '... so wird die Wahrscheinlichkeit, daß die in den Mendelschen Versuchen verfolgten Merkmale wirklich an bestimmte Chromosomen gebunden sind, ganz außerordentlich hoch.' [Translation: H.S.] Translating Boveri's texts into English is difficult, both because the meaning of certain terms changed over the years, and because some German forms of conceptualisation escape an appropriate one-to-one translation. The terminology of genetics was developed in the first decade of the twentieth century, but Boveri did not use it: he used his terms derived from cytology and from early Mendelian work in Germany. Especially for the mathematically defined entities, which were called Mendelian 'genes' after 1909, a translation is not possible without changing the meaning in an a-historic way. Boveri most frequently used the term 'Anlagen', which I do as well in this paper, adding the English expression 'disposition' as an indicatory term in brackets. The only contemporary, but still very late translation of a text of Theodor Boveri was done by his widow Marcella Boveri in 1929: Boveri, Theodor: The Origins of Malignant Tumors. London, Baillière, Tindall & Cox, 1929, German: 1914. Here the term 'inheritance factors' is used for the German 'Erbfaktoren, über die wir durch die Mendelforschung unterrichtet sind', thus linking this hereditary unit directly to the experimental approach of the Mendelians. Marcella Boveri's translation, not using the term 'gene' but 'inheritance factors', indicates a conceptual difference between the 'heredity', she and her husband were investigating and the transmission of 'genes', the Mendelian geneticists were after. This will become clearer, as I hope, at the end of this paper.

3 The terms 'mitosis' for cell fission and 'meiosis' for the cell divisions during germ cell development were coined in 1905. See: Churchill (1970), esp. p. 445.

4 Rheinberger and Gaudillière (2004); Gaudillière and Rheinberger (2004).

for the inheritance of certain characters. This difference in the theory's name signifies an important difference in the understanding of heredity that Boveri had in mind, compared to that of his contemporaries and even that of recent genetics. Second, well equipped with new experimental results, Boveri called his own theory into question shortly before his untimely death in 1915. Since 1902 he had claimed that the chromosomes imprinted parental properties onto the next generation's cell or organism, as the substrate of the hereditary material.[5] In 1915 he expressed his doubts: 'Experience does not teach us what the substrate that contains the paternal [!] 'Anlagen' ['dispositions'] might be.'[6] Thus, the cell nucleus probably no longer could be seen as the carrier of the 'Anlagen', but as a factor enabling development only ['Entwicklungsfaktor']. Boveri came to the surprising conclusion — and he did it explicitly in the face of the rapidly growing science of genetics — that 'Our knowledge of heredity itself ... amounts to nearly nothing.'[7]

These doubts of Theodor Boveri reflect a very complicated process in the generation of knowledge in cytological research, which aimed at an understanding of heredity. This research used very sophisticated experimental approaches and microscopic techniques to illuminate the interplay of the cell's plasma and its elements on the one hand and the chromosomes in the cell's nucleus on the other. Both plasma and chromosomes took part in the process of heredity — the question was how they interplayed.

The creation in 1902/1904 of the scientific fact that bound hereditary 'Anlagen' to chromosomes was a decision taken in favour of the chromosomes as the decisive entity in the cell. In the same move the cell's plasma became the supportive element, active only in the ontogenetic realisation of an organism. This decision had far-reaching consequences for the science of heredity and the understanding of the functions of the cell. It helped to create a split into two disciplines, genetics dealing with the chromosomes, and embryology investigating the plasma and development. It was reinforced in further developments in genetics, social and experimental, which centred the experimental approach on the chromosomes, the genes in the chromosomes, and the DNA as their basic structural unit. Needless to say, the belief in an identifiable, stable, Mendelian hereditary unit was crucial for the belief in the feasibility of eugenics and plant and animal breeding, and it is still alive in the current understanding in medicine of a 'gene for' a certain disease.

By using the word 'decision' I am implying that there were other options available around 1900. Because the chromosomal theory of heredity was not accepted immediately by Boveri's

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5 'In diesen väterlichen und mütterlichen Kernelementen [i.e. den Chromosomen] müssen wohl die dirigierenden Kräfte liegen, welche dem neuen Organismus neben den Merkmalen der Species [sic] die individuellen Eigenschaften der beiden Eltern kombiniert aufprägen.' Boveri (1902), p. 35.

6 'Welches das Substrat ist, das die väterlichen Anlagen enthält, darüber lehrt die Erfahrung nichts. Den Kern läßt sie nur als Entwicklungsfaktor, nicht als Träger der erblichen Anlagen erkennen.' Boveri † (1918), here p. 467. Published posthumously and in an unfinished state by his widow, Marcella Boveri. The manuscript reached the publishing journal on 10 April 1917; p. 417. - The word 'Anlage' in embryology refers to an undifferentiated cellular structure out of which a certain organ or limb may develop. In this sense it also can mean a potential of a given structure, whereas the 'Entwicklungsfaktor' refers to a function only, enabling the development. Both notions have their difficulties, as it was not known how at the microscopic and submicroscopic level the development — not growth — of an organism took place.

7 'Über die Vererbung selbst ... wissen wir so gut wie nichts.' Ibid., p. 417.

contemporaries due to various scientific and some idiosyncratic reasons, we can say that options existed.[8] The impression of a certain fragility of the theory is underlined by the fact that, in the years 1914/1915, Boveri himself found it difficult to stick to his own theory, which he had developed ten years earlier and still praised in 1913, a fact overlooked by current historiographers and biologists. Evidence for the importance and activity of cell plasma had been provided by Boveri himself in the years before and after 1902/1904, when he was studying heredity in cell fission and embryonic development in the flatworm *Ascaris* and in sea urchin embryos. Thus, data on the plasma's importance were available at the time of the chromosomes being made the site of the 'Anlagen'; they were there at the beginning of 'the century of the gene'. The cytoplasm and its activities were not discovered by geneticists at the end of the twentieth century in the form of '"postgenomic" metabolic pathways' or 'multiple systems of inheritance'.[9] It was there in the founding years of genetics and it is currently rediscovered by developmental geneticists praising Boveri as their long forgotten founding father.[10]

In this paper I shall argue that the problem of gender equality was one important cultural and social factor in the making and stabilisation of the chromosomal theory of heredity. This social problem was a factor that supported the decision in favour of the chromosomes as the hereditary material and hampered the possibilities to formulate the interaction of cell plasma and chromosomes as a kind of co-operation, most likely non-hierarchical, by using the experimental evidence available in 1915.

Historians of biology have shown for the 1920s and later that the relations of cell nucleus/chromosomes and cell plasma were understood according to highly contested political concepts, such as the monopoly of power exercised by the nucleus upon the plasma, the 'Kernmonopol'.[11] According to recent science and gender studies, the chromosomal theory of heredity is an example of how the order of the cell's elements follows a gendered hierarchy. Analysis of the scientific language and the metaphors used reveals the familiar Aristotelian, hierarchical dichotomy between male form and female matter: on the one hand, the (male) active, controlling, imprinting chromosomes or genes, represented by the sperm; on the other, the female, passive, obedient cell plasma, the egg.[12]

But, in considering the making of the gendered order of the cell before World War I, something else becomes apparent. The decision in favour of the chromosomes as the carriers or place of the 'Anlagen' reflects an uneasiness. Cytologists based their understanding of hereditary processes on the investigation of cell fusion in fertilisation and subsequent cell fissions. Since 1875 fertilisation was understood within the paradigm of cell theory, thus entailing a highly alarming connotation. Former theories of fertilisation saw a male force active on female matter, thus providing a hierarchical order of unsurpassable eternal and cosmic dimensions.[13] At the new

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8 Thomas H. Morgan, Hans Driesch, and Oskar Hertwig, just to mention three important contemporary scientists, did not immediately accept the chromosomal theory of heredity in the decade before World War I. Cremer (1985); Gilbert (1978).

9 Keller (2000), p. 9.

10 Moritz (1993); Moritz (1996).

11 Harwood (1993), pp. 315—350; Sapp (1987).

12 The Biology and Gender Study Group (1989); Keller (1995). Keller extends the argument to the 'gene action talk', in which the gene even became the only representative of life itself.

13 See Lesky (1951), pp. 125-159, on the canonisation.

material level of cells only, the male tended to be much smaller than the female, if not irrelevant. In this situation the chromosomes defined as the hereditary substance helped to rescue gender equality for the male. It was in no way a feminist move to claim gender equality at the level of cells and chromosomes; it was a move to regain at least some male influence in the realm of generativity and heredity in a situation in which matter mattered.

Contemporaries noticed that a negotiation of the social gender order was taking place in cytology. In 1909 the Prague scientist and historian of biology Emanuel Rádl saw the 'philosophy of gender' [die Philosophie des Geschlechts], 'the subject of deepest thoughts throughout the ages, culminating in the science of the chromosomes'.[14] Rádl criticised Boveri, Hertwig and other cytologists for their claim that there was equality and no fundamental difference between men and women. The situation appeared to be even worse: Rádl uttered the fear that man himself was in danger. The research of Jacques Loeb had shown that fertilisation and egg development could be initiated by chemicals only. In Rádl's words, 'Some potassium chlorate or everyday salt taken from the kitchen may substitute the male of the Echinide, of the worms, the starfish and other animals, if not the human male himself.'[15]

Before looking more closely into Boveri's formulation of the chromosomal theory of heredity, some remarks on his social background are relevant.[16] His work is the work of a creative couple in the sciences.[17] Theodor Boveri was born the son of a medical doctor in 1862 in a small town of northern Bavaria. He studied in Munich, where he got his doctorate title, beginning with ancient history and philosophy, then changing to anatomy. Again he changed to the Institute for Zoology under the directorship of Richard Hertwig, and continued his cytological work on the cell in fertilisation and development. In 1893 he became professor for zoology and comparative anatomy at the University of Würzburg, primarily teaching medical students. The institute became an internationally renowned place to do the latest research in cytology, and several female scientists of the first generation worked here and completed their doctoral dissertations. Theodor Boveri gained such a high reputation in the German scientific community that he was assigned to become the director of the newly founded Kaiser Wilhelm Institute for Biology in Berlin-Dahlem in 1911/1913. The institute and the composition of its staff was planned by Boveri, but in the end he did not take up its directorship. Since 1897 he had been married to the U.S.-American Marcella O'Grady (1863—1950). After their marriage they co-operated scientifically all his life, with Marcella Boveri remaining in the shadows of her husband. The daughter of a Boston architect, she was the first 'woman to graduate with a concentration in biology' at the Massachusetts Institute for Technology. She studied comparative zoology and embryology at Bryn Mawr College, carried out research at the Marine Biological Laboratory (MBL) Woods Hole and received an appointment as a teacher of biology at Vassar Women's College in 1889. Here she became a full professor in 1893 and developed a new curriculum. In 1896 she left for a sabbatical year with

14 Rádl (1909), p. 498. 'Die Philosophie des Geschlechts, welche zu allen Zeiten den Gegenstand tiefsten Nachdenkens bildete, kulminiert heute in der Lehre von den Chromosomen.'

15 Ibid., p. 501. 'Ein wenig Chlorkali oder Küchensalz ersetzt, wenn nicht geradewegs den Mann, so doch das Männchen der Echinide, der Würmer, der Seesterne u.a. Tiere.'

16 Baltzer (1962); Neumann (1998).

17 Wright (1997). See, for comparison, the case studies of several marital co-operations in: Pycior, Slack, and Pnina (1996).

Theodor Boveri at the Würzburg Institute in order to do a PhD dissertation, which she published in 1903. She stayed, raised one daughter and returned to the United States in 1927 to work as a biologist, teaching at the Albertus Magnus Women's College in New Haven.[18] In the life of the Boveris the woman was not restricted to a purely female domain: she took part in his professional work, understood what he was doing, and co-operated in the experiments and other scientific activities. In addition, she was responsible for the running of the house, with a child and servants.[19] Marcella and Theodor Boveri lived at a time when women in Germany were struggling hard for regular access to academic training and independent income and for an improved legal standing. Women in Germany faced a severe step back as in the years around 1900 a new German Civil Code was passed, which made the situation for women much worse than before. It gave husbands control over their wives: they had the final decision in any respect, they controlled women's means and property, they decided on all matters concerning the children, on her professional activities and so on. The women's campaign for suffrage ended in 1919, with the new Weimar constitution giving women the right to vote.[20] The women's movement was an important social force in the time of the Boveris, and its repercussions can be seen in their professional life. The scientific work of Theodor Boveri has to be regarded as the work of a married couple, thus creating some problems for the historiographer. How to talk of a work that is authored by one person only, but created by two from a certain time, from 1897, onwards? The publications bear his name only. How to escape 'the ~~Mathew~~ Matilda Effect in Science', which attributes to the known male scientist the contributions of his collaborator?[21] I would like to pay tribute to Marcella Boveri's contribution to the shared work. She was active in the performance of the experiments, but she did not publish under her own name, with one exception only.[22] Marcella Boveri even did not finish her late husband's last and incomplete paper, she cannot be viewed as his invisible co-author, and it does not seem legitimate to attribute every sentence that Theodor Boveri authored to her as well.[23] For lack of a better solution, I refer to Theodor Boveri when I am discussing the papers he put his name on, thus risking a continuation of her 'silencing by his pen'.[24] I use the phrase 'the Boveris' when I am referring to their common work and not explicitly to him as the author of a specific statement in a paper.

All the usual elements that take part in the creation of a scientific fact can be found in the making of the chromosomal theory of heredity. There was a good deal of experimental and observational material involved, women's work, inductive and deductive modes of reasoning, several hypotheses carefully combined, with the most important ones left to be tested further in complicated experiments. Scientific enemies had to be fought in the development of the new theory [e.g. Oskar Hertwig], and scientific authority had to be accumulated by building bridges to promising new fields of research, such as the Mendelian hybridisation experiments.

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18 Wright (1997), pp. 629-636.
19 Ibid. pp. 638-643; Boveri (1982), pp. 10-50.
20 See, e.g., Bleker (1998); Hausen (1986); Special Issue 'Universität — Frauen — Universitäten', Feministische Studien 20/1, 2002; Gerhard (1990), here pp. 137-324.
21 Rossiter (1993).
22 Boveri (1903).
23 Boveri † (1918).
24 Pycior, Slack, and Abir-Am (1996), p. 6.

The above-mentioned paper of 1904, 'Ergebnisse über die Konstitution der chromatischen Substanz des Zellkerns', presents a combination of the Boveris' own observations and experimental results and the results of twenty years of cytological research, undertaken by an international group of male and female cytologists and zoologists. Most of them came from Germany, the United States and Belgium. Nearly all of them also had worked for some time at marine laboratories, like the Stazione Zoologica in Naples; they used insects, sea urchins, frogs, *Ascaris* and other species as animal models.[25]

The paper starts with the interpretation that the number of chromosomes was a characteristic one for each species and that the chromosomes appeared in a reliable and constant way in every cell division. This 'persistence of chromosomes' was a hotly debated issue of the time and not at all agreed upon by all cytologists.[26] This finding was combined with the results of two of the sea urchin experiments Theodor Boveri had begun in 1889 and continued with Marcella Boveri.[27] In the so-called Merogonie experiment, egg cells were deprived of their nucleus and fertilised with one or two sperm of another sea urchin species. In the early experiments made before 1902, the resulting larvae did not show maternal properties, thus proving relevance of the sperm's nucleus. The second experiment used sea urchin eggs that had been fertilised simultaneously by two sperms; it showed abnormal developments due to the wrong number of chromosomes in the various cell lineages derived from the fertilised egg. Both experiments used the variation of the number and quality of chromosomes in fertilised eggs and its effects on the developing embryo. The experimental results allowed the conclusion that each chromosome had its own relevance for the development of the new organism. This interpretation was called the 'theory of chromosomal individuality'. Attributing a specific quality to each chromosome and describing the regular reduction of chromosomes during germ cell development, it was possible to see a parallel between the behaviour of chromosomes and the Mendelian 'Anlagen'. Most of the experiments stabilising the 'theory of chromosomal individuality' were performed in a sophisticated way in the years to come until the final ones led to some destabilisation.[28]

The gender problem lay in the apparent size difference of the germ cells, the maternal ones contributing much more material to the offspring than the paternal ones. As parthenogenesis showed, the egg cell made a different and a much greater contribution to heredity than the spermatozoon did.[29] Theodor Boveri had elaborated on that explicitly in a short paper of roughly 40 pages, 'Das Problem der Befruchtung' [The Problem of Fertilisation].[30] The paper had been published in 1902 and made the first step to the chromosomal theory of heredity, linking chromosomes to the Mendelian 'Anlagen' as the parental properties [Eigenschaften] were situated in the chromosomes.

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25 Some names should be mentioned: Edmund B. Wilson, Thomas H. Morgan and his wife Lillian Morgan, C. E. McClung, Nettie Maria Stevens, Kristine Bonnevie, Oskar Hertwig, Yves Delage, Eduard van Beneden.

26 See, Cremer (1985).

27 The first paper on a series of experiments of 25 years is: Boveri (1889).

28 Boveri (1910); Boveri (1908).

29 Boveri (1904), p. 112. '... und es hat ... die Eizelle ... eine andere und ungleich viel größere Bedeutung bei der Vererbung als die Samenzelle.'

30 Boveri (1902). The paper derived from a talk at the Versammlung Deutscher Naturforscher und Ärzte in 1902. All of the following quotations are from this paper.

The understanding of fertilisation was the starting point for the understanding of heredity in cytology. The Boveris' research can be characterised as the 'investigation into processes, which cause the generation of a new individual with specific properties from the parental procreative substances.'[31] Hence, research into heredity had to begin with the germ cells. The paper on the problem of fertilisation began with the proud declaration that the untranslatable 'uralte menschheitsgeschichtliche Problem' finally was solved. Everywhere in organic nature, even at the level of protozoons, two sexes co-operated in the creation of their offspring. This, according to Theodor Boveri, was new.[32] In his eyes the co-operation occurred in a reciprocal way, as both germ cells were dependent on each other. Both germ cells had a potential for cell cleavage and development, but they were inhibited. Through co-operation they could overcome this inhibition, through co-operation they supplemented each other in their intrinsic urge to procreate and combine different properties, through co-operation they had enough plasmatic and nutritive substances necessary for the building of the embryo.[33]

This order of the germ cells resembles quite clearly the ideal of a German middle-class/ bourgeois couple around 1900, practising the model of the 'Arbeitspaar' [working couple] in a gendered division of labour, like the Boveris themselves. The unquestioned purpose was the raising of children and the common production of its economic basis, enhanced by the dowry of the woman. At the level of biological reasoning, Theodor Boveri derived his concept of the reciprocity of the male and female germ cells from evolutionary thinking. Boveri was no Darwinist in the sense of seeing selection as one important force of evolution. According to Boveri, the evolution of the organisms was a progressive process driven by intrinsic forces, the 'bildnerische Elementargesetzlichkeit', leading from the primitive 'Urzustand' [primordial state] to utmost complexity.[34] Attempting to find a reason for the existence of male and female germ cells, Boveri described a line of development starting with single cells procreating via cell fission. Then copulation developed between two equal cells. At the evolutionary state of colonies of 16 cells, a differentiation of copulating cells developed — and in the case of the flagellate *Eudorina elegans*, the first egg and sperm cells were to be found, characterised by their difference in size.

The 'Urzustand' was the self-sufficient cell, procreating through growth and subsequent fission. Bees and other insects procreating partially by parthenogenesis exhibited this property of the egg cell. The egg cell in higher animals still had this self-sufficiency, albeit a bit hampered by inhibition. Boveri compared the egg cell to a perfect clock that was missing the spring. In 1902 he had to give up his earlier idea that the centrosom of the sperm caused the cell cleavage of the (fertilised) egg cell, as this process was not found to be a general one in all organisms. Thus, Boveri abandoned the concept of a male induction of embryonic development, which he saw as being in perfect line with the Aristotelian notion of the female providing the matter and the male giving the activating stimulus for the movement of the matter.[35] Having lost this possibility of explaining a

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31 Es ging um die 'Erforschung jener Vorgänge, ... durch die aus den elterlichen Zeugungsstoffen ein neues Individuum mit bestimmten Eigenschaften hervorgeht.' Baltzer (1962), p. 81.

32 The long tradition of not seeing two sexes or more existing in the plant kingdom had been overcome in the 16th century; now the protozoons had it as well.

33 Theodor Boveri followed August Weismann's line of argument that amphimixis, the combination of different parental properties in the offspring, was the purpose [Zweck] of fertilisation. Boveri (1902), p. 36.

34 Ibid. p. 38.

universal biological difference between the male and the female, he tried to create a full reciprocity of male and female germ cells: 'One could say the spermatozoon is fertilising the egg, one could also say that the spermatozoon is being fertilised by the egg.'[36] Both cells needed each other to get procreation started. However, Boveri had to concede that it was always the egg cell that started development: there was parthenogenesis only and no androgenesis. No development of an embryo started from the sperm. Having to admit a generative difference in germ cells, Boveri posed the question: 'How does it come that the properties of the sperm are not suppressed within the egg? How do they cope with the properties of the egg, which exceeds the sperm in size by the thousands and millions?'[37] Boveri sought rescue in the mere statement that the sperm could cope with the egg, though he did not know how. He referred to the experience that 'generally the father's influence on the constitution of the child equalled that of the mother's'.[38] This equal influence was guaranteed by equality at the level of chromosomes. Boveri saw his work in the tradition of others, starting with Carl Naegeli's postulate of an 'Idioplasma'. Naegeli had claimed that a substance was present in every cell in a very small quantity, which derived from equivalent substances in the egg and the sperm cell. This substance provided an equal force of heredity ['gleiche Vererbungskraft'] of both parents, despite the enormous differences in their material contributions for the gestation of a child.[39] After 1900 the Mendelian laws provided an experimental approach to prove parental equality in heredity. This claim of equality, however, entailed a specific definition of heredity. In a rather circular reasoning, heredity now only dealt with properties characterised by their binary difference in both parents. In other words, only the inheritable differences between members of a species could be dealt with and localised in the chromosomes. All the general properties of an individual, like the inheritable features of the mammalians, the properties of the genus and higher classificatory groups, were not included in this definition.[40]

For Boveri, the chromosomes of the sperm incorporated the sperm's equal influence on the offspring's properties in this narrow sense of inheritable properties. Describing the behaviour of chromosomes after the sperm's integration into the egg, he rhapsodised in a pseudo-religious language: 'Indiscernible the grown nucleus of the sperm stands face to face with the nucleus of the egg; in fullest equality in size, form and number ['Gleichheit nach Größe, Form und Zahl'] the paternal and maternal nuclear elements ['Kernelemente'] lie close to each other. They are passed on in the same combination to the daughter cells and, as we may suppose, to all the cells of the new individual. All this happens with unsurpassable, painstaking care. In these paternal and maternal nuclear elements lie the directing forces, which in combination imprint onto the new organism not only the properties of the species but the individual characters of the parents.'[41]

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35 Ibid. p. 23.
36 Ibid. p. 34.
37 Ibid. p. 35.
38 'Zahllose Erfahrungen ... lehren, daß der Vater auf die Konstitution des Kindes im allgemeinen ebenso viel Einfluß hat, wie die Mutter.' Boveri (1902), p. 35.
39 Boveri (1904), pp. 102-103.
40 The paradigmatic example for the species-specific properties lying in the chromosomes was derived from the crossbreeding of a horse and a donkey. It made a considerable difference in the offspring if the maternal animal was the horse or the donkey, thus showing the influence of the maternal cell plasma.

Boveri was so fascinated by the notion of two equal sets of chromosomes in the fertilised egg that it took him some time in the following years to accept the findings of Nettie Maria Stevens (1861-1912) on chromosomal sex determination. According to this theory, two types of male germ cells existed, which differed in the number of chromosomes, thus determining the sex of the offspring.[42] Stevens had worked with Boveri in his institute in Würzburg in 1903, and she was the one who immediately saw the applicability of Boveri's chromosomal theory of heredity to the problem of sex determination. Boveri did not — he was trapped in the creation of gender equality at the level of chromosomes. As late as 1908 he revised his interpretation that there always were two completely equal nuclei in the germ cells.[43]

In the following years, Theodor Boveri argued as a convinced Mendelian geneticist. In 1913 he praised the results of the latest research, which enabled the localisation of certain characters onto the sex chromosome: 'sex and colour blindness — what could be more different? Nonetheless, there is nearly no doubt that the 'Anlagen' for both characters are located in the same chromosoma.'[44] He thus was on the same track as his scientific enemy Thomas H. Morgan, who, with his group of PhD students had started mapping *Drosophila* genes onto sex chromosomes.[45] The Boveris, however, favoured a different experimental approach: heredity was to be investigated in embryonic development. The qualities of the germ cell's nucleus were to be analysed here. This approach was called in analogy to the spectral analysis of light in physics: 'Embryonalanalyse des Zellkerns' ['Embryonic analysis of the cell nucleus']. Heredity was to be investigated in fertilisation and ontogenesis; the methods used were a combination of cytological analysis and hybridisation experiments, including interspecific crossbreeding of sea urchins. In co-operation with his wife, Marcella O'Grady, Theodor Boveri designed experiments to change the chromosomal constitution of the egg cell and fertilised egg to observe and interpret their abnormal development. He planned to use a device that was to remove single chromosomes from a cell, and various other techniques were applied to influence a cell such that the relation of plasma and chromosomes was changed. These experiments aimed at the understanding of the interplay of chromosomes and cytoplasm at various stages of embryonic development.

The last Merogonie experiments did not provide the desired result that the chromosome was the site of the 'Anlagen'. Marcella Boveri published her husband's last paper in its unfinished form in 1918 under the very unpromising title: 'Zwei Fehlerquellen bei Merogonieversuchen und die Entwicklungsfähigkeit merogonischer, partiell-merogonischer Seeigelbastarde'.[46] If this paper contained at least some dynamite to shake the chromosomal theory of heredity, then most probably nobody took notice — with one exception perhaps: Richard Goldschmidt (1878-1958).[47] For the geneticists after World War I, there was no longer a need to take notice of the

41 'In diesen väterlichen und mütterlichen Kernelementen müssen wohl die dirigierenden Kräfte liegen, welche dem neuen Organismus neben den Merkmalen der Species die individuellen Eigenschaften der beiden Eltern kombiniert aufprägen.' Boveri (1902), p. 35.

42 Brush (1978).

43 Boveri (1909).

44 'Geschlecht und Farbenblindheit, was könnte verschiedener sein. Und doch können wir ... kaum zweifeln, daß die Anlagen für beide Eigenschaften in dem gleichen Chromsoma lokalisiert sind.' Boveri (1913), p. 16.

45 See, Kohler (1994); Gilbert (1978).

46 'Two sources of artefacts in merogonic experiments and the potential for development of merogonic, partially merogonic sea urchin bastards.'

results of a paper that promised to deal with methodological problems in a very complicated experimental system using sea urchin development, as it no longer was in use in the research into heredity.

The main and unexpected results of Boveri's last paper are that embryonic development is divided into two phases. In the first phase, the plasma alone is active; the chromosomes step in at a later stage. In addition, chromosomes and plasma need to be compatible with each other in order to enable proper development. As mentioned, the results were such that Boveri doubted his own proposal of 1904, that the chromosomes might be the material substrate of heredity. But this last, unfinished paper shows as well that these new and severe doubts were outweighed by the desire to believe, in disregard of the new findings, that the substance within the cell nucleus actually was the hereditary substance [die 'Vererbungssubstanz'].[48] Boveri did not reformulate the interaction of plasma and chromosomes in heredity according to his and his wife's new findings. It makes no sense to speculate what Boveri might have done had he lived longer. He ended with drawing a line between the 'exakte Vererbungslehre', the newly developed genetics, on the one hand and the research into the processes of ontogenesis on the other. The one approach used hybridisation experiments, the breeding of pure lines, it applied Mendel's laws and the notion of a gene; the latter asked how the constellation in the zygote leads to the 'Erbeffekt' [Charakter] that the geneticists ['Vererbungsforscher'] deal with. For Boveri, only the latter equalled 'heredity itself', the 'Vererbung selbst'.

In the work of the Boveris the interaction of plasma and chromosomes in the fertilised egg and the developing organism was conceptualised in various ways. In the same move in which gender equality at the level of the chromosomes was introduced, a gendered hierarchy between the chromosomes and the plasma was established. In the years 1902 and 1904, Theodor Boveri appreciated the Aristotelian notion of fertilisation as female matter set in motion by a male activating impulse, but he had to concede that this solution was not a general one and dropped it. The Aristotelian hierarchical dichotomy between form and matter could, however, be applied to the chromosome-plasma relation by interpreting the chromosomes as entities that contained 'conducting forces to imprint paternal properties onto the egg cell and the organism of the next generation'. Theodor Boveri was criticised by colleagues who saw him as advocating the autocracy of the nucleus within the cell.[49] Boveri replied that the cell plasma and the nucleus were dependent on each other and that neither could exist alone; he used his opponents' metaphor: the huge crowd of workers necessary for the autocrat's existence was present in the cell plasma and inherited by the plasma.[50] His own analogy was that of the brain and the body to exemplify the mutual dependency of chromosomes and plasma, or of the plan of an architect and the construction workers building the house.[51] Obviously, mutual dependency was not an equal dependency. It entailed a clear hierarchy following the Aristotelian model, which ensured a gender hierarchy as

47 Goldschmidt who followed his own track in conceptualising the genes and the chromosomes became the head of one department at the Kaiser-Wilhelm-Institute in Berlin in 1914. He appreciated Boveri's work until the end of his own life.
48 Boveri (1918), p. 468.
49 Boveri (1904), p. 103.
50 'Vor allem wird eben im Protoplasma das ganze Heer des Arbeitsvolks vererbt, ohne welches selbst ein Alleinherrscher, wenn wir einmal dieses Bild gebrauchen wollen, nicht existiert.' Ibid, p. 113.
51 Ibid, p. 103.

well. The order of cell nucleus and plasma followed this double hierarchy. At the level of gender, it parallels the legal situation of a married couple according to the civil code of the time. The man had the legal power to make all decisions, he was the head of the household, and all the negotiations and co-operations made to keep the family going were left invisible between husband and wife.

In 1914/15 there was no new conceptualising of the nucleus-plasma relation possible, albeit the possibility had to be considered that the chromosomes were not the decisive forces but enabling the specific 'Gestaltung' of the developing organs of an organism only. The cause for its concurrence with the specific 'Gestalt' of the parents could lie in other parts of the cell.[52] It is even much more remarkable that the Boveris did not re-conceptualise the plasma-chromosome relationship as Theodor and Marcella Boveri provided important findings on the interplay of the plasma and the cell nucleus, findings Theodor Boveri is praised for nowadays by developmental geneticists.[53]

1. The centrosoms and spindles organised the distribution of chromosomes during cell cleavage. They organised the positioning of the hereditary material/chromosomes within the cell and thus 'decided' on their fate during development.
2. Substances and processes in the plasma were important for the first phase of embryonic development before gastrulation.
3. Even more dramatic: the plasma was able to reorganise the chromosomes; it could change their size and composition. This process, called 'Chromosomendiminution — chromatin diminution', was observed for the first time by Boveri in 1887 in *Ascaris*, and examined in extensive cell lineage studies later on. Boveri showed in 1899 that only the germ cells did not undergo chromatin diminution. For him, this was a necessary finding to support Weismann's germ line theory and his own theory of chromosomal individuality. The reorganisation of the chromosomes by the plasma during ontogenesis was crucial, as a process was needed which could explain an unequal distribution of hereditary material during ontogenesis so that cell differentiation could occur. It was a process of circular reasoning due to which chromosomal diminution could not happen in the germ line.

Let me end with a kind of a-historic thought experiment using a bricolage of the Boveris' findings of the plasma's own activities. It would have been a very radical move to claim that the plasma could rearrange the chromosomes of the germ line as well.[54] Boveri had postulated that chromatin diminution happened in species other than *Ascaris*, even if it was not visible under the microscope — so why not in the germ line, where one could not see it as well? Obviously, there was no way of conceptualising an interaction between plasma and chromosomes in heredity which was a co-

[52] The nucleus (Kern) was only to be recognized 'als ein Organ ..., welches dazu da ist, die Ausbildung des Larvendarms, des Skeletts usw. zu ermöglichen' while 'die *spezifische* Gestaltung dieser Prozesse, d.h. die Übereinstimmung mit der spezifischen *Gestalt* der Eltern, in anderen Teilen der Gameten ihre Bedingungen haben werden.' [emphasis by Boveri]. Boveri (1918), p. 467.

[53] Moritz (1993).

[54] See for comparison the fate of the contemporary experimental system in hereditary research using the worm *Planaria*. *Planaria* did not have a visible germ line thus providing reasons for a completely different understanding of heredity as a process of metabolism, not linking it to a specific structure in the cell. The scientists using *Planaria* did not gain the social authority to efficiently compete with the Morgan group in the US. Mitman and Fausto-Sterling (1992).

operative, non-hierarchical one, which would have made the egg cell much more powerful than the chromosomes of the sperm, and which would have resulted in a completely new understanding of 'heredity' and its science.

For the social gender order there was obvious progress attached to the knowledge of genetics. In 1934 the Norwegian geneticist Otto Mohr enthusiastically praised it, claiming that the times of pure male genealogy were over: 'one of the most far-reaching achievements of modern biology is the definite establishment of the fact, that men and women are genetically equivalent'.[55] For genetics, however, it might not be seen as a success story.

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[55] Mohr (1934), p. 207.

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Hugo de Vries's transitions in research interest and method

Ida H. Stamhuis[1]

I said, no- one deserves the title botanist who is not also a physicist and a chemist.
(Hugo de Vries looking back to the beginning of his research career around 1870)

Assumptions in which way the hereditary characteristics are determined through the composition of their carriers are not yet made; this elaboration of the theory of heredity is unnecessary for the time being.[2]
(Hugo de Vries in 1889 about hereditary particles)

To get an impression of the context in which the modern ideas of heredity were embedded, I will concentrate on Hugo de Vries (1848–1935), the person who put his stamp on the emerging science of genetics, yet soon distanced himself from it. I will examine the pre–Mendelian stages in De Vries's scientific development. 'Context' will in this case mean that I discuss Hugo de Vries's early scientific interest and methodology, which was not in heredity and evolution but in plant physiology, and that I will connect that to his later research interest and methodology. Heredity was not a general concept from the beginning, but would slowly emerge; I will try to make plausible that something parallel was true with respect to the methodology that had to be used to study the concept of heredity.

Looking at Hugo de Vries's early work in plant physiology and comparing that to the – for him – most important later work in heredity and evolution, his *Intracellular Pangenesis (Intracellulare Pangenesis)* and his *Mutation Theory (Die Mutationstheorie)*, the contrast in research methodology is striking: the first is what I will call 'reductionist', because an explanation of biological phenomena is sought with the help of physics, chemistry and quantitative reasoning, whereas the second does not try to do so, and is qualitative.[3] In this essay my aim is to discuss the methodological aspects of De Vries's first choice for plant physiology and then his transition from plant physiology to heredity and evolution. I will try to gain some understanding as to why he chose to work in these two fields in botany with such radically different approaches, and to discuss how he dealt with this contrast. I will address this problem by showing that originally it was not

[1] I am grateful to Erik Zevenhuizen, who is writing a biography (in Dutch) on De Vries, for his critical response to my ideas, especially on the physiological foundation of De Vries's ideas on heredity, and for his meticulous comments on the whole text. I thank my Free University colleagues (Ab Flipse, Teun Koetsier, Frans van Lunteren and Wijnand Rekers) for their comments during a discussion meeting. Thanks to Daniel Carroll for his correction of the English.

[2] See the more elaborate quotations and references in the sections 'Change of Research Focus" and "Change of Research Method".

[3] Reductionism has been extensively written about. Here I will take reductionism to mean the explanation of biological phenomena with the help of a so–called 'underlying' less complex field of knowledge: chemistry, or physics, or not an 'underlying' but nevertheless less complex field of knowledge: mathematics. In the case of mathematics a characterization like 'modelling' is perhaps better, but to make clear that in all cases the effort is to try to explain biological phenomena with the help of a simpler field of knowledge, I use one concept in all cases (Jongeling 1997).

obvious that the methods in these two fields would be so different. For De Vries the original methods turned out to be unsuccessful and ultimately he would no longer consider these as useful.

To gain deeper insight into De Vries's research choices, it will also be helpful to sketch his background: the milieu he came from, his secondary school time, and the atmosphere at the university in Leiden, where he studied botany. I will start by dealing with these aspects. After discussing his change of research focus, I will include a short characterization of his theory of heredity and I will discuss a number of criticisms levelled at De Vries's *Intracellular Pangenesis*, because he did not explain these ideas within the framework of his plant physiological work. In addition I will show that he tried out a quantitative model of heredity, but had to abandon it due to the resulting inconsistencies. In the next section I will argue that in the 1890s he again tried out a reductionist approach, namely a statistical/probabilistic one. And although this approach resulted in 1896 in the rediscovery of the Mendelian Laws, for De Vries it was not a complete success, since that approach was not compatible with his theory of heredity.

I discuss only an aspect of the background of De Vries's research choices. There is an erudite essay about De Vries's transition from plant physiology to genetics (written in Dutch) by Bert Theunissen, from which I will now extract a few observations.[4] Theunissen discusses the views of De Vries and other Dutch scientists in that time on the importance of science for the progress of society, because that may have influenced their research choices. Their opinion was that science should have a civilizing influence. Moreover science could help to solve problems of society, both social and economic. According to De Vries, botany could very well serve that goal. Within botany the study of heredity was a possible choice, but other choices were also possible. That De Vries nevertheless chose heredity was for other reasons. Theunissen suggests that for De Vries the reason for leaving the field of plant physiology, the collaboration with his colleague at the University of Amsterdam, the physical chemist Jacobus H. van't Hoff, will have been a decisive factor. Aided by De Vries's work on osmosis, Van't Hoff was able to formulate his theory of dilute solutions for which he and Svante Arrhenius would later receive a Nobel Prize. De Vries subsequently felt that he had to redefine the demarcation between each other's sphere of activity. Why, then, did De Vries choose to focus on genetics? Theunissen points, just as I will do, to De Vries's early interest in this field. Theunissen also argues that De Vries's ultimate aim, to be able to control mutations, was in accordance with the more general idea of Dutch scientists of the 'improvability' of society with the help of science.[5] But this would motivate De Vries later in his life than the period I discuss in this paper.

For my discussion of De Vries's research choices and methods, I will mainly rely on existing scholarship. In addition, this material will be augmented by the rich primary resource represented by the preserved 450 letters De Vries exchanged with his colleague and friend Jan Willem Moll (1851–1933).[6] Little, is available in English about De Vries's youth and student time, but in Dutch a number of biographies have been written, which will be useful for my aims.[7] A contemporary

4 (Theunissen 1992).
5 'improvability' is a translation of the Dutch 'maakbaarheid'.
6 (Stamhuis 1995). The collection of letters is in the University Library in Groningen. Moll's letters are copied in three copy books, to which I refer as CB1, CB2 and CB3.
7 (Van der Pas 1970) and (Zevenhuizen 1998a) are biographies in English; (Heimans 1948), (De Veer 1969), (Smit 1980) and (Visser 1992) in Dutch.

biography was published in 1900 by one of his first pupils, the later professor of botany in Utrecht, Friedrich A.F.C. Went (1863–1935).[8] We can use this text, supplemented with modern biographies, to gain deeper insight into his boyhood, student time, the time spent as a researcher in plant physiology and his turn to heredity. Another interesting source is a short biography written by his friend and colleague Jan Willem Moll on the occasion of his seventieth birthday.[9] Moll had been a colleague and friend of De Vries since 1872 and commented upon many concepts of De Vries' scientific publications. Although Moll's comments on De Vries's work were sometimes very critical, to the outside world he always defended and supported him. For the discussion of the significance of the reductionist approach in De Vries' work on heredity, I draw on two of my own publications.[10]

Life Sketch

Hugo de Vries was born in 1848 into a family of intellectual and political distinction. His father was successively a member of the 'Raad van State', an important national advisory council for the government, a minister of Justice and a member of the 'Tweede Kamer' (Parliament); Hugo's uncle was professor of Dutch language at Leiden. His maternal grandfather was an archaeologist. So his family was intellectual, but more in the humanities than in the natural sciences.

Notwithstanding this family interest, it is told that even as a primary school pupil Hugo was interested in nature. He lived in Haarlem near dunes, woods and the sea. He regularly took long walks in these woods and dunes and studied the flora native to these natural environments. At the age of twelve he participated in a competition initiated by the Dutch Society of Agriculture to make a collection of hundred dried plant species from the surroundings of Haarlem.[11] He started to compose a herbarium and was awarded an honourable mention. During holidays his family made trips to various parts of the country and it is said that on these occasions his brother made drawings and Hugo looked for plant species. During his time at secondary school he and his family moved to The Hague, the Dutch administrative capital, because of the political career of his father. At secondary school the classics played an important role, and Hugo would later say that he was not happy with the neglect of modern languages and the natural sciences.

In 1866 the period of university study came (fig. 1). That he would go to university was obvious given his milieu, but not his wish to study natural sciences, especially not botany; he registered to study natural philosophy ('philosophia naturalis'). Apparently his interest in natural history was not in accordance with his family's interest. Moreover, there were no 'distinguished' jobs after such a study, the available few in secondary school teaching not being considered 'distinguished'. Moreover, botany was connected to agriculture and, according to his biographer Went, in this period of the upcoming industrial revolution, industry enjoyed a higher standing in his family than agriculture.[12] Ultimately, though, Hugo's family accepted his choice.

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8 (Went 1900).
9 (Moll 1918).
10 (Stamhuis et al. 1999); (Stamhuis 2003).
11 'Competition' is a translation of the Dutch 'prijsvraag'.
12 (Went 1900), 266.

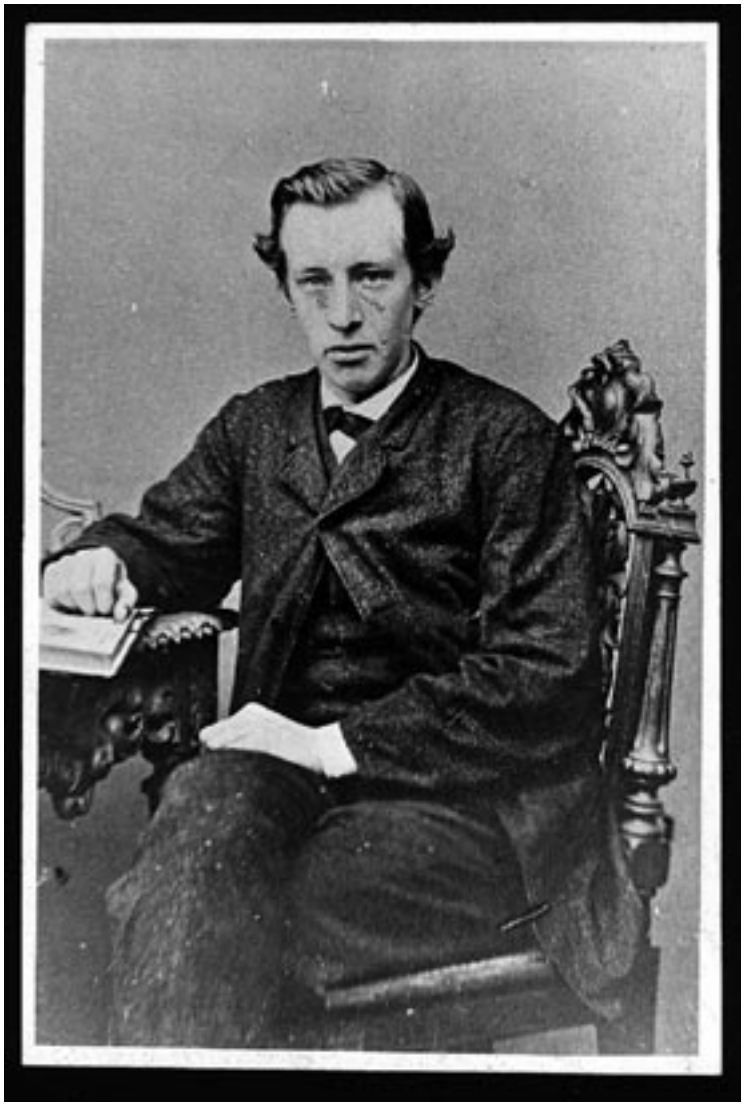

Fig. 1. Student Hugo de Vries.(Archive Hugo de Vries, University of Amsterdam).

In 1859 Darwin had published the *Origin of Species.* De Vries's teacher, the professor of botany W.F.R. Suringar, did not agree with Darwin's ideas on evolution. Hugo de Vries therefore did not hear about it from his teacher but read Darwin's book for the first time in 1868, his second year of study, in a German translation he had bought at an auction. According to Went, he and his fellow students discussed it, abandoned the idea that species could not change into others, and became convinced adherents of Darwin's evolutionary theory.[13]

In the meantime Hugo de Vries encountered another way of practicing botany than he learnt from his teacher Suringar, who was working in the field of systematics and wanted Hugo to study and describe lichens. In the same year as he studied Darwin's work, Hugo studied the new book

[13] (Went 1900), 267–268; (Visser 1992), 160–161.

Lehrbuch der Botanik by the German Julius Sachs, in which the new plant physiological approach of botany was discussed. De Vries also found an opportunity to apply this new method, because the University of Groningen held a competition entitled "What is known of the effect of warmth on plant roots?" He participated in this contest and did the necessary experiments at home, because at the university in Leiden there was no opportunity to carry out these kinds of investigations. He used his answer in the Groningen Prize Contest, for which he was awarded the Gold Medal in 1869, for his thesis, and was awarded his PhD in 1870.

To explain the physiological interest of De Vries notwithstanding his education in systematics by Suringar, his biographer Went informs us that De Vries was not only interested in the outside but also in the inside of the plant: in its anatomy and vital functions.[14] I would add that Hugo might have been an ambitious young man, who will soon have noticed that his teacher's interest was old fashioned. He may well already have had greater ambitions. The most obvious opportunity to do something which was new and which was considered of real scientific value was the new plant physiology. The practitioners of plant physiology could be found especially in Germany, the country on which Dutch scientists were mainly oriented. Moreover, Sachs had discussed an applicable research program in his book. De Vries would later also refer to the teaching of his physics professor Rijke; this professor will have played a stimulating role in choosing a research field in which physics was essential.

After gaining his PhD, Hugo de Vries left for Germany. His first visit to the old Wilhelm Hofmeister in Heidelberg was not successful, but subsequently, in 1871, he went to the young Julius Sachs (1832–1897) in Würzburg. That was the beginning of a fruitful relationship with Sachs and with the new botany that Sachs represented (fig. 2).[15] However, De Vries also had to earn a living and he therefore spent the next four years as a secondary school teacher in Amsterdam. During this period he frequently spent his entire summer break in Würzburg. In April 1875 he was commissioned by the Prussian Ministry of Agriculture to write monographs on agricultural plants. He was able give up his job as a teacher and settled in Würzburg. In 1877 he became a 'Privatdozent' at the University of Halle, and published his Habitilationsschrift *About the mechanical causes of cell stretching (Über die mechanischen Ursachen der Zellstreckung).* De Vries noticed that the professor of botany in Halle was not happy with his presence. Moreover, student interest in his lectures was minimal; it is therefore not surprising, that De Vries was not very enthusiastic about the job in Halle.[16] He was therefore happy when in the next year, in 1878, he was appointed lecturer at the University of Amsterdam where he became a full professor in 1881.[17]

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14 (Went 1900), 267.

15 Letter from De Vries to Moll, 24–04–1875 from Würzburg "I am now busy with micro- chemical work" (Ik ben thans druk met microchemischen arbeid bezig); see also the letter of 28–06–1875.

16 (Went 1900), 271; (Visser 1992), 161–162; Letter from De Vries to Moll, 22–05–1877 from Halle "Gradually I start to notice (…) that it will not be easy for me (..). Don't tell anyone about this." (Ik begin langzamerhand te merken (…) dat ik het hier niet gemakkelijk zal hebben (…) vertel hiervan maar aan niemand iets).

17 Letter from De Vries to Moll, 09–09–1877 from Den Haag "I have been proposed for the post of lecturer in plant physiology in Amsterdam. Salary 1500 Dutch guilders." (Ik ben tot lector in de plantenphysiologie te Amsterdam voorgesteld. Tractement Fl. 1500). Letter 19–09–1877 from Würzburg "This morning I received the official announcement of the Municipal Executive of Amsterdam about my appointment" (Heden morgen ontving ik de officieele mededeling van B. en W. van Amsterdam omtrent mijn benoeming).

In 1896 he also became the director of the Hortus Botanicus of the university. In the Netherlands he became a 'Man of Significance', even before the so–called rediscovery of Mendel in 1900 and the publication of his *Mutation Theory* (in 1901 and 1903).[18] Notwithstanding various job offers from Dutch and foreign universities, he remained in Amsterdam until his retirement in 1918.

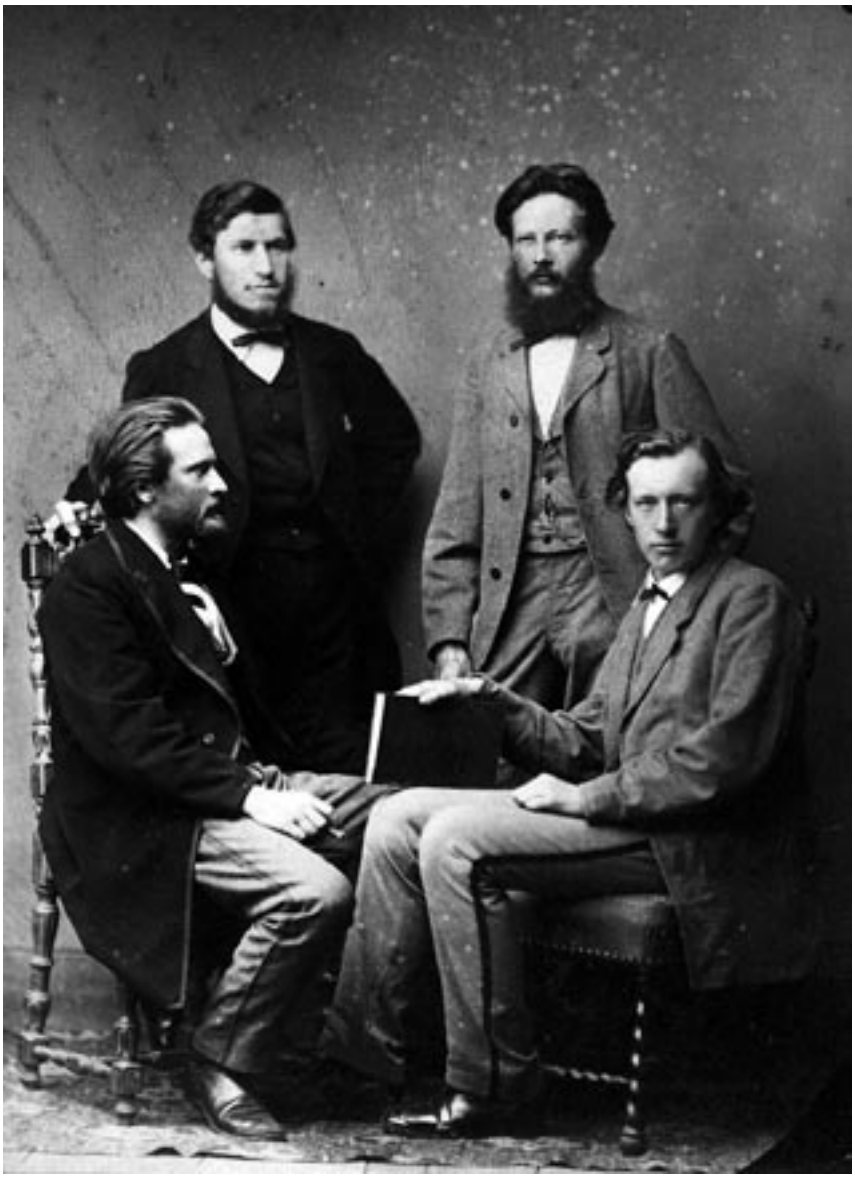

Fig. 2. Julius Sachs and Hugo de Vries left and right at the front. (Archive Hugo de Vries, University of Amsterdam).

[18] Went published in 1900 his biography of De Vries in the series 'Mannen en Vrouwen van Beteekenis in onze Dagen' (Men and Women of Significance in our Days).

Change of research focus

I will now discuss his research interest and focus attention on the apparently great transition from plant physiology on the one hand, to heredity, evolution and mutations on the other.[19] It is striking that De Vries's colleague and friend Moll distinguished three stages in De Vries' work, of which the first two are in plant physiology and the third in heredity, evolution and mutations.[20] De Vries's pupil Went did not set De Vries's work on plasmolyses apart from his other plant physiological work, for example on the movements of plants, as clearly as Moll did. Moll gave De Vries's work on plant physiology more weight than we would expect. Moll stated that this last work took place during a so–called preparatory period, and his work on plasmolyses during De Vries's second period, which Moll characterizes as 'very important for science'. It is strange that Moll more or less distinguished two *periods* of plant physiological work, since from the list of De Vries's publications it follows that these two kinds of work cannot be distinguished chronologically. An explanation may be that De Vries's chemical physiological work on turgor, plasmolyses and isotonical coefficients (fig. 3) made a greater impression than his work on the growth of plants, the movement of plant vines, the causes of tree rings and of transport in the plant, and that for that reason Moll felt the need to clearly distinguish between the two.[21]

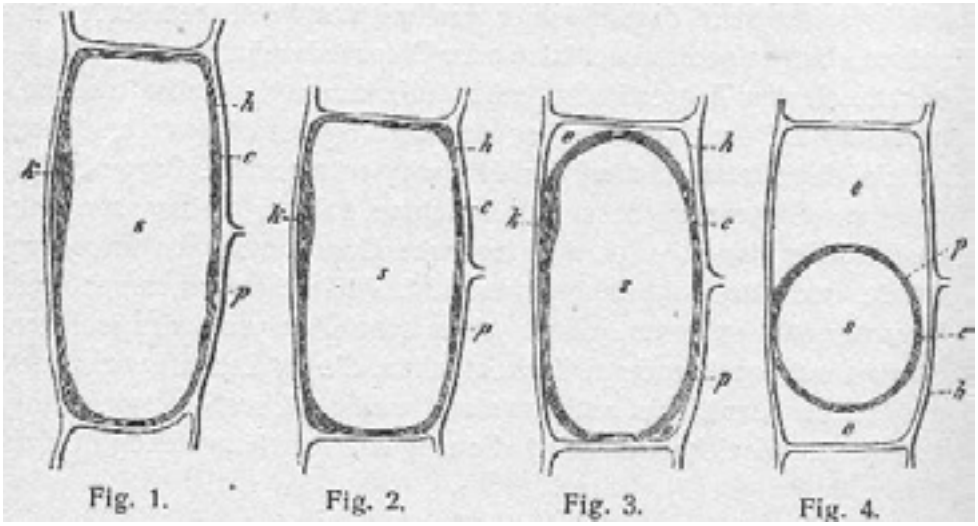

Fig. 3. An example of De Vries's reductionist plant physiological work. Cephalaria leucantha. 1) Turgescent young cell; 2) In 4% nitric acid solution; 3) In 6% nitric acid solution; 4) In 10% nitric acid solution. (De Vries 1918-1927) Vol. 1, 396. (Library Free University Amsterdam).

Went as well as Moll discuss De Vries's work on heredity and evolution as something different from plant physiology. Went asserted that as soon as De Vries became acquainted with Darwin's

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19 See also (Visser 1992) and (Theunissen 1992).
20 (Moll 1918).
21 (Moll 1918), 2. About difference in prestige of different methods in plant physiology see (De Chadarevian 1996).

ideas on evolution in 1867, they made a strong impression on him.[22] To support this view, Went pointed to the theses that De Vries added to his dissertation on 1870 in which he defended evolutionary theory, although he knew that his supervisor would oppose them. One of these theses was: The hypothesis of Pangenesis [...] cannot explain the variability of the species.[23] Went even stated that "since that time the great questions of variability and heredity absorbed De Vries's mind and never released him. Although it is true that (…) through the influence of Sachs, for a time he went into an entirely different field of investigations."[24] Before Went started to discuss De Vries's work on 'heredity and variability' he stated[25]

> Certainly few have studied Darwin's work so thoroughly as De Vries. When he was conducting his investigations in the field of mechanical physiology, (…) his mind also remained engaged in the great questions of the theory of descent, and he soon collected all facts in this field that he encountered in the literature. As soon as he had his own field for experiments at his disposal, directly some of the experiments were dedicated to questions of variability and heredity. We thus see that De Vries was driven more and more in this new direction, especially since during the academic year 1881–82 he gave a course in the theory of descent for students of botany. I believe that all who attended those lectures – not only philosophers, but also medical students from various academic years – will not easily forget the clear way in which such a rich subject matter, which was difficult to gather, was discussed by a convinced adherent of Darwinism.

Went must have attended these lectures.

Much later, in an interview published in 1935, De Vries himself commented upon his original views on the role of physics and chemistry for physiology and botany, and his later transition of research focus.[26] He said:

> I felt in that time that physics and physiology were so closely connected that they were only different communications about the same events, and I also said that to Hofmeister. I said, no–one deserves the title botanist, who is not also a physicist and a chemist. Then I had something in my head that had not yet a name. You must realize that Van't Hoff and Arrhenius were then still schoolchildren. And names like physical chemistry, chemistry, biochemistry, colloids, cell physiology and the like were not used until thirty to forty years hence. (...)When I came back in Amsterdam, I wanted to continue with what is now termed physical chemistry of the cell. I felt it as a kind of responsibility towards my esteemed teacher of physics Rijke.

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22 (Went 1900), 167–268; (Visser 1992), 160.

23 "De hypothese van de pangenese kan de variabiliteit der soorten niet verklaren."

24 (Went 1900), 268. "Sedert dien tijd hebben de groote vraagstukken van erfelijkheid en variabiliteit zich meester gemaakt van den geest van De Vries en hem niet meer losgelaten. Wel is waar betrad hij (…) onder den invloed van Sachs, een tijdlang met zijn onderzoekingen een geheel ander veld."

25 (Went 1900), 289. "Weinigen hebben zeker Darwin's werken zoo grondig bestudeerd als De Vries. Ook toen hij de onderzoekingen op het gebied der mechanische physiologie verrichte, (…) bleef zijn geest zich bezig houden met de groote vraagstukken der afstammingsleer en hij verzamelde spoedig alle feiten, hierop betrekking hebbende, die hij in de literatuur aantrof. Zoodra hij beschikken kon over een eigen terrein voor proefnemingen, werden dadelijk eenige van die proeven gewijd aan vragen over erfelijkheid en variabiliteit. Zoo zien wij De Vries meer en meer gedreven in deze nieuwe richting, vooral sedert hij in den cursus 1881–82 een college voor botanici gaf over de afstammingsleer. Ik geloof, dat al degenen die dit college volgden – en het waren niet alleen philosophen, maar ook medici uit verschillende studiejaren – niet licht de heldere wijze zullen vergeten, waarop een zoo rijke stof, die moeilijk bijeen te garen was, door een overtuigd voorstander van het Darwinisme werd behandeld."

> But nobody knew exactly what I intended; the child had no name, the institute was not equipped. For students of medicine, I had to teach general physiology and theory of heredity, and this confronted me with my old love, evolutionary theory. Only during holidays was I able to do experimental cytology. However Ms. De Vries had three young children and wanted to spend the summer in the country. So I abandoned osmosis (...) and I started with what is now termed genetics.

He suggested that he ceased work on plant physiology after his appointment in Amsterdam in 1878, but that is not correct. Although he had also started with experiments in variability and evolution on a modest scale, he continued to publish in plant physiology until 1889, the year of the publication of his *Intracellular Pangenesis*. His correspondence with Moll clearly reflects that after 1889 his physiological work abruptly came to a halt. In 1887 and 1888 he wrote about the tonoplast, about permeability of the protoplasm, about isotonical coefficients, plasmolyses and permeability, about the influence of gravitation on protoplasm and the lowering of the freezing point of raffinoses.[27] In 1887 he started to write about sunflower seeds, about numbers of rows of seeds in corn ears, about conifer seeds and about monstrosities in plants.[28] In a letter dated May 19th 1888 his interest in heredity and variability became more explicit: he wrote about August Weismann's work, stating that he appreciated Weismann's germ plasm theory.[29] In September 1888 he mentioned for the first time his intended booklet on heredity and in October 1888 he sent the first part to Moll for comment.[30] Subsequently we find no further references to plant physiological work.

I think that it is well founded to conclude that in his youth De Vries loved to walk in nature and to study plants in the countryside. This passion endured throughout his lifetime (fig. 4). He was however not satisfied to carry out investigations in the old fashioned systematics, but was ambitious and eager to work on new and modern botanical questions. Notwithstanding the opinion of his teacher Suringar, he became a convinced adherent of Darwinian thinking.

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[26] Quotation from the *Prager Presse*, June 2, 1935. The German part I copied from (Theunissen 1992), 112; the Dutch part I copied from (De Veer 1969), 18.
"Ich fühlte schon damals dass Physik und Physiologie irgendwie nahe zusammenhängen, dass sie nur verschiedene Berichte über ein und dasselbe Geschehen vorstellen und habe es auch Hofmeister gesagt. Niemand sollte Botaniker heissen, der nicht zugleich Physiker und Chemiker ist, sagte ich. Ich hatte damals etwas im Kopfe, das noch keinen Namen trug. Van't Hoff und Arrhenius waren damals noch Schulbuben, müssen Sie wissen. Und Namen wie Physikalische Chemie, Biochemie, Kollide, Zellphysiologie und dergleichen mehr sind erst nach dreissig bis vierzig Jahren geprägt worden. (...). Als ich nach Amsterdam kam, wollte ich zuerst selbstverständlich in dem, was man heute Physikalische Chemie der Zelle nennt, fortfahren. Ich fühlte es übrigens halb als Gebot meines geliebten verstorbenen Lehrers [P.L. Rijke]. Aber niemand wusste, wass ich eigentlich vorhabe, das Kind hatte keinen Namen, das Institut war nicht eingerichtet, rings herum stand zu den Versuchen nichts Lebendes zur Verfügung:"
"Voor de medicijnstudenten moest ik college geven in de algemene fysiology en erfelijkheidsleer, en dit confronteerde mij weer met een oude liefde, de evolutietheorie. Alleen in de vacanties zou ik experimentele cytologie hebben kunnen doen. Maar mevrouw De Vries had drie kleine kinderen en wilde de zomer buiten doorbrengen. Aldus heb ik de osmose verlaten (...) en ben begonnen aan datgene wat men tegenwoordig genetica noemt."

[27] Letters from De Vries to Moll, from Amsterdam, 07–01–1887; from Amsterdam 13–12–1887. Letter from De Vries to Moll, from Amsterdam 12–01–1888; from Amsterdam 24–02–1888, Letter from De Vries to Moll, 04–03–1888 from Amsterdam. De Vries informs Moll that research of Arrhenius affirmed the previous results of De Vries.

[28] About sunflower seed and corn: Letter from De Vries to Moll,: from Amsterdam 20–09–1887, 27–10–1887; Letter from De Vries to Moll, from Amsterdam 27–03–1888.

However, for the time being he was absorbed in successful plant physiological work in which Sachs became an influential factor and of which the Groningen Prize Contest marked the beginning. He may at that time have had the impression that there was not yet an interesting Darwinian research program. However, when circumstances made it easy to grow plants and his mind became again engaged in heredity and evolution because he lectured in it, and perhaps because it became clearer how to do interesting work in heredity and variability, he did not hesitate to change his research focus and to start to investigate questions of heredity. Theunissen also pointed to the possible roles of the promising applicability of this new field and the need for the redefinition of the demarcation line between him and his colleague, the physical chemist Van't Hoff. After the publication of his *Intracellular Pangenesis* his change of research focus was complete.

Fig. 4. Hugo de Vries with students on a botanical excursion. (Archive Hugo de Vries, University of Amsterdam).

[29] Letter from De Vries to Moll, from Hilversum, 19–05–1888; again on Weismann's work in a letter dated 07–06–1888 from Hilversum.
[30] Letter from De Vries to Moll from Amsterdam 04–10–1888.

De Vries's theory of heredity

To understand the discussion of the change of De Vries's research method, it is necessary to know something of the content of his theory of heredity.[31] I will sum up the most important aspects. According to *Intracellular Pangenesis* the properties of visible characters depend on small invisible particles, which De Vries called 'pangenes'. They are either inactive or active, and they are able to grow and multiply in both states. Their activity is dependent on the type of cells in which they are located. There are two kinds of cell lines. There is a line of cells from the fertilized egg cell to the germ cells of the newly formed organism. This cell line is called the germ line; the other lines are called somatic lines. The cells of the somatic lines develop into the cells forming the organs of the organism. De Vries stated that pangenes are usually inactive or latent in the germ lines, and develop their greatest activity in the somatic cells. Differentiation of organs is due to the fact that individual pangenes, or groups of pangenes, develop more strongly than others.

In the nucleus of each cell of the organism all types of pangenes of the individual are present, in the cytoplasm only those that will become active. In the nucleus most pangenes remain latent. They become visible and active only in the cytoplasm. Pangenes multiply in the nucleus, partly for the division of the cell nucleus, and partly in order to be transported later to the cytoplasm.

There has to be transport from the nucleus to the cytoplasm. Transmission of pangenes is the function of the nucleus, their development the function of the cytoplasm. In the nucleus, most pangenes only have to multiply. In the cytoplasm pangenes will continue to multiply (usually more so than in the nucleus). Sometimes they will change there from active to latent, or sometimes the other way around. Some will become active immediately after their arrival, others later.

The development of a character requires a minimum number of active pangenes of the same kind. When the number of pangenes becomes larger, the expression of the character will become stronger. If the numbers of certain pangenes are reduced, the corresponding visible property will be only weakly developed; if their number is strongly reduced, they become latent. Therefore, the number of similar pangenes is very important. In the cytoplasm, this number is decisive for the function of the various organs, in the nucleus for the hereditary force.

Pangenes do not represent morphological parts of the organism, or cells, or parts of cells, but specific individual characters. They can vary independently of each other. De Vries stressed the independence of hereditary properties. Hybridisation is performed in order to transfer properties from one variety to another. Therefore the study of hybrids is of great importance. Hybrids show that the nature of a species is not unitary. Properties of hybrids can be as clearly distinguished and are as constant as those of pure species.

Heredity must have a material basis, which can be none other than the living protoplasm. In every cell division two new pangenes arise which are identical to the original one. In every cell division all types of pangenes present go, as a rule, to both daughter cells. Exceptions to this rule are the starting point of the emergence of varieties and species.

There are two kinds of variability. Firstly, the numbers of pangenes may vary in what is called 'fluctuating variability'. Secondly, during successive cell divisions, pangenes may change their nature slightly, or even considerably.

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[31] (Stamhuis et al. 1999), 243–244.

Change of research method

In this section I will explore the thesis that Hugo de Vries's *Intracellular Pangenesis* can be considered as a transition; in various cases a physiological approach was suggested, but it had to be abandoned, because it was not possible to explain the complicated hereditary phenomena in that framework (fig. 5).

Fig. 5. Drawing of *Rubus fruticosus laciniatus* from *The Mutation Theory* (The original German edition, Vol. 2, p. 689) The caption is: 'The distinguishing feature of the variety, the deep indentation of the leaf edge, expresses itself in the leaves as well as in the petals. Both phenomena are expressions of the same material carrier of the internal character'. (Archive Hugo de Vries, University of Amsterdam).

Let me start by making clear that it is not true that for his *Intracellular Pangenesis* De Vries abandoned a physiological approach, or that he put it aside. Quite the contrary is the case. Throughout the booklet he was very clear that his theory had a physiological basis and that his hereditary carriers, which he called pangenes, were morphological particles. However, since it was mostly not yet possible to connect the hereditary characteristics with the physiological material basis, his opinion was that, to enable progress in the field of heredity, it would be better to put considerations about that basis aside for the time being. I will refer to a few passages of his booklet in which he put this view into words. In the introduction to this booklet he stated that he had to "explore the basic ideas of pangenesis." His starting point was that "the physiology of heredity" could be investigated during the "microscopic investigation of the cell division and the

fertilization, and the morphological substrate." He continued: "One must not try to explain the morphological details of these processes, because our knowledge is still far too restricted." De Vries's opinion was that if such an explanation was nevertheless possible, then that should of course be given, but that was only seldom the case.[32] In the chapter in which he discussed the hypothetical carriers of species characteristics, he asserted that[33]

> an assumption about the nature of these hypothetical units (…) must be connected to their relation to the hereditary characteristics. Assumptions in which way the hereditary characteristics are determined through the composition of their carriers are not yet made; this elaboration of the theory of heredity is unnecessary for the time being.

That he always realized that his theory had a physiological basis is also clear from the fact that later he talked at various times about the "physiology of heredity".[34]

Although he explicitly formulated his opinion in his booklet on heredity of 1889, his readers would not be satisfied with it. They would criticize aspects of his theory that he could not explain physiologically or that were even implausible from a physiological point of view. I will cite a few examples. Moll commented upon a concept version of *Intracellular Pangenesis* in 25 written pages and an accompanying letter.[35] In the accompanying letter he criticized what he called the "hypothesis of intracellular pangenesis itself."[36] Moll's main criticism concerned what he considered to be an inconsistency in De Vries's hypothesis. De Vries had argued that the pangenes moved from the nucleus to the other parts of the cell in protoplasm streams, the normal route of transport in the plant.[37] Moll's comment was[38]:

> But now the reader will proceed further on this road. He remembers that H. de V. is himself the man who considers the protoplasm movements as the means of transport for food through the entire plant. He thinks of the ppl [protoplasm] connections between the cells, a fact which is as well established as the many facts you have used, and therefore he feels forced to assume a transport of pangenes through the entire plant and thinks that in this way he acts completely in your spirit.

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32 "den Grundgedanken der Pangenesis (…) auszuarbeiten" "die Physiologie der Erblichkeit" "die mikroskopische Erforschung der Zelltheilung und der Befruchtung und das morphologischen Substrat". "Nicht die morphologischen Einzelheiten jener Vorgänge soll man zu erklären suchen, dazu ist unsere Kenntniss noch viel zu beschränkt." (De Vries 1889), 6–7.

33 "Eine (…) Annahme über die Natur jener hypothetischen Einheiten (…) bezieht sich auf ihre Beziehung zu den erblichen Eigenschaften. In welcher Weise diese durch den Aufbau der Träger bestimmt werden, darüber werden bis jetzt keine Annahmen gemacht, denn auch dieser Ausarbeitung bedarf die Theorie der Vererbung vorläufig nicht." (De Vries 1889), 36.

34 e.g. (De Vries 1897), 63.

35 Copy of the letter from Moll to De Vries: CB1, 168 ff., October 15, 1888. See (Stamhuis 2003), 125–130.

36 "de eigenlijke hypothese der intracellulaire pangenesis zelve": copy of the letter from Moll to De Vries, CB1 168 ff., October 12, 1888.

37 (De Vries 1889), 141.

38 "Maar nu gaat de lezer op dien weg als vanzelf verder. Hij herinnert zich dat H. d. V. zelf de man is, die de protopl–bewegingen als middel van vervoer voor voedsel enz. door de geheele plant beschouwt. Hij denkt aan de ppl verbindingen tusschen de cellen onderling, een feit even vast staande als vele feiten die je gebruikt hebt en hij ziet zich dus genoodzaakt een vervoer van pangenen door de geheele plant heen aan te nemen en meent daardoor zelfs zeer in je geest te handelen."

De Vries' earlier statements about transport in the plant by protoplasm streams implied that pangenes could not only move from the nucleus to the protoplasm, but also from the protoplasm to the nucleus and even from one cell to another. Moll stressed that accepting this consequence would mean that De Vries's theory of heredity did not essentially differ from Darwin's provisional *intercellular* theory of pangenes. "He [the reader] will think that it would have been better if you had made it clear from the start that you accept Darwin's pg [pangenesis] entirely and now want to show that the more recent results are entirely in accordance with it." [39] Moll formulated his opinion rather confrontationally: "There is no reason to assume that pangenes are able to *leave*, but not to *enter* the nucleus." But although this reasoning was in accordance with his physiological work, De Vries did not accept this conclusion. In Moll's words: "In one word, this closure of the nucleus to its former inhabitants is an auxiliary hypothesis, which you frame because you have said from the start that you don't want to have anything to do with 'movable pangenesis' and with heredity of acquired characters, and you feel now that without this hypothesis you return to these ideas." We do not have de Vries's immediate reaction to this criticism, but in his booklet he discussed the problem briefly.[40] In a section entitled "Comparison with Darwin's Transport Hypothesis" ("Vergleichung mit Darwin's Transporthypothese") he admitted that on the basis of the physiological knowledge available to Darwin there had been no reason to doubt the possibility of transport of a material carrier of hereditary information from one cell to another. He went on to say that, especially thanks to Weismann's convincing arguments that acquired characters are not hereditary, it had become clear that such transport was very improbable. So he put the physiological approach aside in favour of hereditary evidence.

An example of the critical reception of *Intracellular Pangenesis* is a review in the *Botanische Zeitung*[41] in 1881 written by Georg Albrecht Klebs, who had studied with Julius Sachs in Würzburg.[42] The criticism centred around the nature of pangenes. Klebs concluded that pangenes must be a kind of imprint of the cell organs, but he complained that it was not explained how they were able to perform their activity. He wondered what exactly the difference was between active and inactive pangenes and upon what that difference was based. Nor was it explained how it was possible that the functioning of a pangene depended entirely on its location: in the cell nucleus it caused the force of heredity and in the protoplasm it was responsible for the physiological activity. In addition, Klebs found the idea of the latency of pangenes unclear, and remarked that the claim that the level of expression of characters depended on the number of pangenes led to dubious consequences. Klebs criticized De Vries because he had simplified a complex issue and Klebs wrote that it was not difficult to formulate a few questions that would be difficult to answer. For example, De Vries's theory did not explain the essence of an organism, the connection of all its parts, and their union into one entity. De Vries did not explain much at the cell level either, for example, how

39 "Hij zal dus meenen, dat je beter gedaan hadt met voorop te zetten, dat je Darwin's Pg geheel aanneemt en hier aan wilt toonen, dat de latere resultaten van het onderzoek daarmede geheel in overeenstemming zijn." "Er is geen enkele reden om aan te nemen, dat pangenen wel *uit*, maar niet *in* den kern kunnen gaan." "In één woord deze geslotenheid van den kern voor zijn gewezen inwoners is een hulphypothese, die je opstelt, omdat je van den beginne al gezegd hebt, dat je met de bewegelijke pangenesis en erfelijke verworven eigenschappen niets te maken wilt hebben en nu voelt dat je er zonder die hypothese weer toe terugkeert."

40 (De Vries 1889), 142–146.

41 (Klebs 1889).

42 See (Stamhuis 2003), 137–138.

the cell is generated and how pangenes influence each other in the cell. Because of these deficiencies, Klebs concluded, De Vries' theory would probably not be generally accepted.

The criticism of another commentator, Cornelis Adrianus Pekelharing, professor of physiology at the medical faculty of the University of Utrecht, shows parallels with Klebs's criticism. De Vries had discussed his ideas with Pekelharing and on that occasion his concept of latent characters had also proved to be rather controversial.[43] With reference to that occasion he wrote to Moll:[44]

> I cannot compromise on P.'s objection against latent characters, it makes it even more difficult to say what I mean. I sense that I am usually unsuccessful in this anyway. That the nucleus is the storehouse of *all* types of pangenes was what I meant to say, for example. However, I didn't manage to say it. I wish rather that I had not started it.

The basis of the criticism by Pekelharing, Klebs and Moll was that the claims of De Vries's theory were not explained physiologically. Moll asked how it was possible that pangenes can leave a nucleus but cannot return to it, or move to other cells. It was a well–known physiological fact that transport within the plant takes place by protoplasm streams, which stream into and out of the nucleus and from cell to cell. Movement only from the nucleus to the cell could not be explained. Pekelharing's and Klebs's criticisms of the latency of characters can be understood analogously. De Vries needed the concept of latency. He had to argue that all types of pangenes are present in all cells. For the explanation that nevertheless cells develop differently, he needed the concept of latency. However, the physiological questions remained unanswered: how it was possible that in some cells a pangene (or character) did not develop activity while in other cells it became active; how some pangenes in the same cell became active while others remained latent; and also, how it was possible that their activation depended on their position within the cell: in the nucleus or in the protoplasm. De Vries did not explain what physiological processes were responsible for this; the only reaction he could give was that these concepts could explain important hereditary phenomena and that therefore he believed that they were true.

De Vries had already come to the conclusion that in some cases he could not use the usual physiological approach to explain hereditary phenomena. In his booklet on heredity he had also put that into words. However, others confronted De Vries with it, because they experienced De Vries's argumentations as unsatisfying, inconsistent and incomplete.

Calculating offspring character values

In Moll's detailed comment on a concept version of Intracellular Pangenesis there is a passage, which shows that De Vries tried out a quantitative model of heredity, but that he could not

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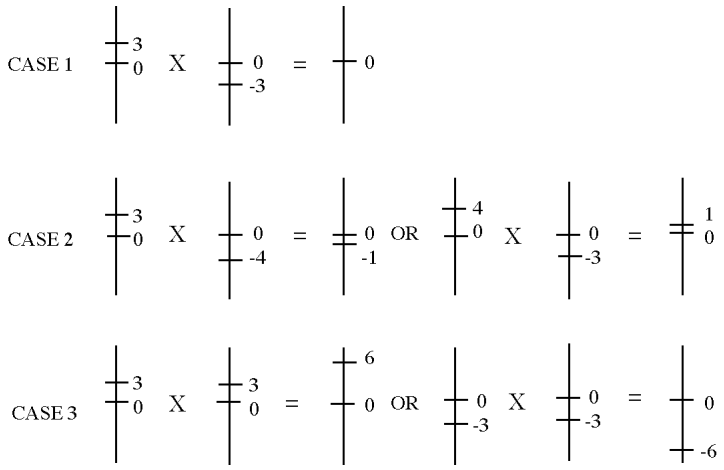
[43] In his detailed comment on *Intracellulare Pangenesis* Moll also referred to Pekelharing's criticism of the latency of characters. Copy of comment from Moll to De Vries, especially 170 and 172.

[44] "P's bezwaar tegen latente eigenschappen kan ik niet toegeven, ook maakt het het nog veel moeilijker om te zeggen wat ik bedoel. Ik bespeur dat dit toch veelal mislukt. Dat de kern is de bewaarplaats van *alle* soorten van pangenen, en dat de questie van de inactiviteit bijzaak is, was bv mijn bedoeling. Doch 't was mij niet gelukt dat te zeggen. 'k Wilde wel dat ik het niet begonnen was." Letter from De Vries to Moll, November 1, 1888.

maintain it, because it resulted in inconsistencies.[45] Unfortunately we do not have De Vries's concept at out disposal, only Moll's response. The relevant parts of Moll's comment are:[46]

> I am not sure whether this calculation contributes to clarity. If 1 on p. 54 is right, then 2 must be right, as I changed it above, and then at 3 p. 55 reinforcement has to take place. If this is not in accordance with the facts, then that is proof that your formula is wrong and then I would rather delete it.
>
> On second thoughts it is like this. In case 2 one gets an individual with the character in question above or below the zero point. In case 3 with the character above the zero point. Like this:



> I think that these are the results of your calculation. Your further calculation on p. 55 I do not understand. Doing that you forget that by zero point (see p. 54) you mean the mean character if all individuals of the culture are mixed. And if you want to stick to this, I don't see what

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45 See (Stamhuis et al. 1999), 244–246.

46 "Of deze berekening nu wel tot de duidelijkheid bijdraagt weet ik niet. Als 1 op p. 54 waar is, dan moet 2 waar zijn, zooals ik het boven veranderde, en dan moet bij 3 p. 55. versterking plaats hebben. Als dit nu niet met de feiten overeenkomt dan is het een bewijs dat je formule verkeerd is en dan zou ik die liever weglaten.

Bij nader inzien is het aldus. In geval 2 krijgt men een individu met de bedoelde eigenschap meer of minder dan het nulpunt. In geval 3 met de bedoelde eigenschap hooger dan het nulpunt. Aldus:

[And then the three cases of crossing schemes follow]

Dit zijn dunkt mij de uitkomsten van je som. Your further calculation on p. 55 I do not understand. With that you forget that with the zero point (see p. 54) you mean the mean character if all individuals of the culture are mixed. En wil je je daaraan houden, dan zie ik niet in wat een verdere berekening meer geeft dan het bovenstaande, terwijl de lezer moeite zou hebben je te volgen.

(.....) Je doel is uit de feiten der bevruchting te halen dat de erf. eig. onafhankelijke grootheden zijn. (.....)

Eindelijk de berekenings–?tirade? is meer een 3e (p. 54) onderwerp om de *Anwendung* v.d. voorstell. der zelfstandigh.v.erf.eigensch. op het fixeeren duidelijk te maken:" Copy of Comment Moll, CB1, 175–176.

> more a further calculation will give than the preceding one, whereas the reader will have difficulties in following you.
>
> ——
>
> (.....) Your aim is to derive from the facts of fertilization that the hereditary characters are independent entities. (.....)
>
> Finally the calculation –?tirade? is rather a 3rd (p. 54) subject to make clear the *Anwendung* (application) of the notion of the independence of the hereditary characters on the fixation.

In the passages quoted, Moll used elementary arithmetical methods such as adding, subtracting and averaging. First the mean of the character value of the whole culture was calculated and that became the reference point, and was therefore characterized as zero. Individual values of that character became the differences with respect to this mean value. The character value of the individual offspring was the sum of the values for that character of both parents. Therefore, a hybridization of an organism with a character value 3 units above the average and another with a character value 3 units below the average would result in offspring with the mean value. Moll's interpretation of De Vries's calculations as resulting in an additive hereditary theory has strange consequences: offspring of parents with values higher than the mean value will have an even higher value for that character than both parents, and offspring of parents with values lower than the mean similarly an even lower value. It is clear from the first passages of Moll's comment, in which he wrote that in case 3 reinforcement had to take place, that this consequence was clear to him. Moll knew that this consequence was definitely not in accordance with the facts. He was successful in conveying his objections against such an approach; De Vries took Moll's objections to heart and did not include the quantitative model in his book.

The conclusion of the preceding two sections is that De Vries's publication of his theory of heredity was surrounded by ideas that a reductionist explanatory framework was advisable; physiological or additive. This turned out to be impossible. De Vries's hereditary ideas could not yet be reduced in this way. Notwithstanding, De Vries did not yet abandon a reductionist approach. In the next section his efforts to introduce statistical and probabilistic thinking in his work will be discussed.

Again a quantitative approach of heredity: rediscovery of the Mendelian laws

Klebs's conclusion of his review of De Vries's *Intracellular Pangenesis* was that a general acceptance was not probable because of the large gaps and deficiencies in the hypothesis. Klebs's prophecy came true; De Vries's theory was considered too speculative. As I have discussed elsewhere, De Vries responded by developing a sizable research program of hybridization and other experiments with the aim of making his ideas on heredity more convincing.[47] The experiments that he performed resulted in a huge amount of data, of which it was not directly clear how to interpret them. During the period of his physiological work De Vries had encountered the efforts of L.A.J.

[47] (Stamhuis et al. 1999); (Zevenhuizen 1998b).

Quetelet, F. Galton and W.F.R.Weldon to apply statistics to biological data and to make the normal distribution play an important role.[48]

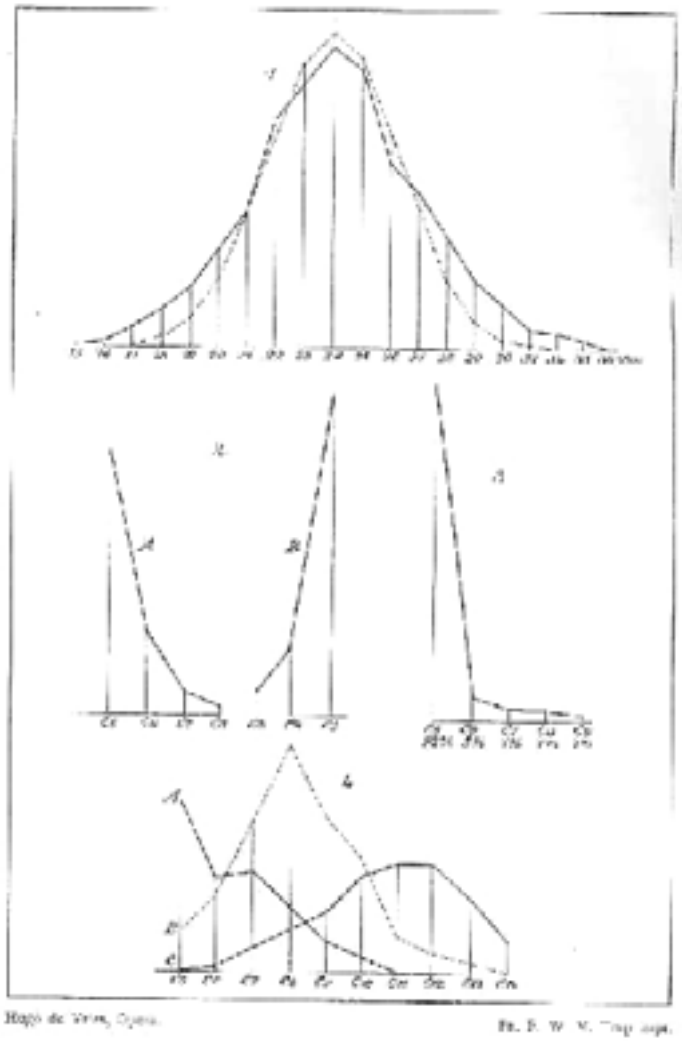

Fig. 6. An example of the statistical interpretation of De Vries´ research results. De Vries called the curves Galton curves and the underlying law: Law of Quetelet and Galton. 1) Curve of the number of fruits of *Oenothera Lamarckiana*; 2) A. One sided curve of number of petals of *Caltha Palustris*; B. One sided curve of number of petals of *Weigelia Amabilis*; 3) One sided curve of number of petals of *Ranunculus bulbosus*; 4) *Ranunculus bulbosus.* A) One sided curve of number of flowers; B) Symmetrical curve of the number of flowers. C) Symmetrical curve of number of flowers. (De Vries 1918-1927) vol. V, 505. (Library Free University Amsterdam).

De Vries started to try out statistics to interpret the results of his experiments (fig. 6). To that end he had to design his experiments accordingly. Through this approach in 1896 he encountered the

48 (Zevenhuizen 1998b), 431–435.

laws that after 1900 would be named the Mendelian laws. He became persuaded that these laws were just what he needed, because the independence of distinct hereditary factors was central to them. Therefore, initially the great popularity of Mendel served him very well.

Notwithstanding, because he was absorbed by the writing of the first volume of the *The Mutation Theory*, he paid little attention to these laws in 1900.[49] In the course of time the experimental foundation of his pangenes theory was no longer the only aim of these experiments; criticism of Darwin's selection theory became another central point. Although he did not use the terminology of his pangenes theory in *The Mutation Theory,* it is clear from the way he expressed himself that he always had this theory at the back of his mind and that he tried to combine the two theories. He was more or less successful in bringing his ideas on pangenes and on mutations in line with each other, but it was more difficult to mould the Mendelian laws into the framework of his ideas on heredity and mutations. When his attempts to describe Mendelian crossings in terms of pangenes and mutations are examined precisely, it becomes obvious that he became entangled in a number of contradictions. As discussed elsewhere, he described these crossings in terms of his pangenes theory in two different ways, but in both cases unsolvable inconsistencies emerged.[50] He did not admit this explicitly, but some of his remarks convey the impression that he was more or less aware that Mendel's laws on the one hand and his theories on pangenes and mutations on the other hand could not be reconciled.

Moreover, it remains unclear how his "normal" fluctuating variability could be fitted in with the quantitative approach of Mendelism, because he did not confront them with each other. He may not have found Mendelian genetics important enough to feel obliged to bring all kinds of concepts of his theories in line with it. However, it is not easy to imagine how the concept of fluctuating variability, in which numbers of the same pangenes can decrease or increase rather randomly, could be made compatible with paired Mendelian hereditary factors.

The incompatibility between De Vries's ideas on heredity and mutations and the statistical formulated Mendelian laws may have caused De Vries's need to relativize the importance of the Mendelian laws, as he did in several passages of *The Mutation Theory* and elsewhere.[51] I will give a few examples from *The Mutation Theory*. De Vries remarked that the splitting of the hereditary factors does not only occur in the case of Mendelian crossings, but also in hybrids where the splitting is not always complete.[52] De Vries formulated the opinion that the deeper, the so–called systematically higher, characters are generally transferred to the next generation without changes and therefore without splitting.[53] In addition, polyhybrids are possible whose elementary characters are connected so strongly that they cannot be split.[54] In general, only the phylogenetically younger characters, or the *race* characters, obey Mendel's splitting rules, although even this is not the case with all of them.[55] De Vries hypothesized that the occurrence of Mendelian crossings pointed to the termination of the mutation capacity within a family.[56] And

[49] (Stamhuis 1995), 20–22.
[50] (Stamhuis et al. 1999), 259–264.
[51] (Meijer 1985), 220–223; (Theunissen 1994), 247–248; (Stamhuis 1995), 19–24.
[52] (De Vries 1903), 74.
[53] Ibid, 79.
[54] Ibid, 115.
[55] Ibid, 140–141.
[56] Ibid, 458.

he apparently did not think of these kinds of organisms as the most interesting ones. He, moreover, emphasized that the possibility has always to be taken into account that phenomena occur that do not obey these hybridization laws.[57] He could also not accept the rule that, when the offspring of Mendelian crossings possess the recessive character, the following generations remain forever constant, but stated that when it was allowed that this constancy was not absolute, an influence of an earlier hybridization could remain present in a latent state.[58] Somewhere in the offspring the dominant character could re–emerge. All of these assertions are more or less incompatible with the Mendelian laws.

Although with hindsight we may conclude that he was very successful with this new attempt to use a reductionist framework, it was not the way he ultimately experienced it. The Mendelian framework was not in accordance with his ideas on heredity and evolution. It is plausible that this led him to consider the Mendelian framework as not being of essential importance.

Conclusion

In this paper I have mainly restricted myself to a discussion of the methodological aspects of Hugo de Vries's research choices, and showed that it was not clear from the beginning by which methodology heredity should be investigated. I intended to link Hugo de Vries's later ideas and methods in heredity and mutations to the context of his early ideas and methods in plant physiology, and see whether a thread can be discerned in his scientific development. A conclusion is that this ambitious young man, who from his early youth showed a focused interest in botany, was from the very beginning of his career not only interested in plant physiology, but also in heredity and evolution. That he chose to work in physiology was because a research program in plant physiology was more easily attainable for him. He was however also steeped in Darwin's publications on evolution and heredity. When it became clearer to him how to approach the problems of heredity and when the circumstances became favourable, he did not hesitate and started to work in that field. This work was first mainly theoretical, but later he started a sizable experimental program, first in variability and heredity but later – through his work on mutations – also in evolution.

We see that on numerous occasions the approach he had become used to in his work in plant physiology was connected to his new work. He himself stated that his ideas on heredity had a physiological basis, although it was not yet known how to explain hereditary phenomena physiologically. Notwithstanding De Vries's statements on this topic, others criticized his new ideas on heredity because De Vries did not explain them in the way as they were used to in his plant physiological work. He himself tried to apply quantitative methods, first an additive model in his original theory of heredity of 1889 and later, in 1896, a probabilistic approach in the interpretation of his hybridization and other experiments. Although this last effort was very successful in hindsight, in both cases De Vries found these approaches unsatisfactory. In all cases the reductionist argumentations could not satisfactorily answer the questions in which he was interested. The problems of heredity and evolution that he studied could not yet be moulded in such a reductionist way. Therefore he distanced himself from the applicability of these

[57] Ibid, 486–487.
[58] Ibid, 525–526; see also 537–538.

approaches. In the case of physiology he stated explicitly that for the time being they could not be used for his hereditary theory. In the case of the probabilistic Mendelian laws he relativized their significance, but in the background their incompatibility with his own theories will have been a factor in this diminution.

I will close with the following observation. In the discussion of De Vries's transition from plant physiology to heredity and evolution we can also take into account that for De Vries there is an additional reason why the contrast between the two research approaches will not have been so sizable as we experience it now. During the period of De Vries' plant physiological investigations, Darwin was also working in the same field. In 1862 he published *Fertilisation of Orchids,* in 1875 *Insectivorous Plants*, in 1877 *Forms of Flowers* and in 1880 *The Power of Movement in Plants.* Although De Vries's mentor in plant physiology Julius Sachs and Darwin criticized each other's approach, they worked on the same kind of plant physiological problems.[59] At the end of the 1870's Hugo de Vries corresponded with Darwin, and in 1878 visited him.[60] In their correspondence and during their meeting they discussed their similar plant physiological investigations: among others about their work on climbing plants. So De Vries knew that Darwin, who had formulated the theory on evolution for which De Vries strongly admired him, was also engaged in plant physiological problems. It is not improbable that the physiological interest of Darwin was a factor that diminished De Vries' feeling of the contrast between plant physiology at the one hand and heredity and evolution at the other.

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[59] (De Chadarevian 1996), 17–41.
[60] (Van der Pas 1970). See also (Theunissen 1992), 101.

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Herbert Spencer's two editions of the Principles of Psychology: 1855 and 1870/72. Biological heredity and cultural inheritance

Snait B. Gissis

Introduction

My paper is a report on one aspect of my project that addresses Spencer's role in the process of *explicitly* evolutionising the human sciences in the second half of the 19th century. Introducing an evolutionary component in the human sciences immediately confronts the issues of biological heredity and of cultural inheritance, — whether clearly distinguished or not, whether intertwined or not, whether specific mechanisms for them can be adduced or not,— and thus introduces a significant, if not crucial, element into these fields of knowledge.

My presentation is in many ways an *internalist* one. I will not delineate genealogies of ideas nor draw contextual filiations in Spencer's *Principles of Psychology* (PP) as much of this work has been done, and redone, rather thoroughly from various aspects during the past three decades, particularly by Robert M. Young, J. D. Y. Peel, Roger Smith, C. U. Smith, Robert Richards and Rick Rylance. My paper falls within the 'evolution bent' in reading Spencer, but perhaps with a difference in the significance attributed to the evolutionary tradition within which Spencer was located, as I have given an interpretation which throws light on Spencer's position within the human sciences of the second half of the 19th century. In an attempt to combine the various parts of my reading of the two editions of PP from the perspective of biological heredity and cultural inheritance *I have used Spencer's Lamarckism* as my needle and thread.

Let me first very briefly point to some elements in the context of Spencer's enterprise. The period between the two editions was eventful. It witnessed several developments that had important political repercussions.[1] Similarly, several notable intellectual and cultural controversies took place during the period. Also, a number of books that brought a new perspective to bear on social institutions and on diverging human capabilities were published.[2]

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[1] Eg: 1846 – Repeal of Corn Laws. 1850 – Telegraph cable laid under English Channel. 1851 – Great Exhibition ("Crystal Palace"). Population of United Kingdom at 21 million. 1855 – Livingston discovers Victoria Falls. – Civil Service Commissioners appointed. 1857–8 – The Mutiny in India. 1858 – First Atlantic cable laid. 1860 – Budget–changes to Free trade. 1861 – Albert dies; Victoria retires into mourning. 1861–5 – American Civil War. 1867 – Second Reform Bill: enfranchises many workingmen; adds 938,000 to an electorate of 1,057,000 in England and Wales. (Disraeli's legislation)– Factory Act– extends restrictions on working hours for women and children to all factories– South African diamond fields discovered. – Fenian Rising in Ireland. 1869 – Suez Canal opened. 1870– Forster's Elementary Education Act establishes School Boards starts a system of state elementary schools. 1871 – University Tests Act removes religious tests at Oxford and Cambridge. – Trade unions legalized. Newcastle engineers strike for a nine–hour day. 1872 – Public Health Act– creates sanitary authorities with medical officers. 1873 – Population of the United Kingdom at 26 million (France 36 million). 1875 – Artisans' dwellings Act encourages slum clearance. 1876 – Victoria named Empress of India. – Compulsory school attendance in Great Britain. 1877 – Transvaal annexed.

I would suggest that some of these were relevant not only to Spencer's later writings on sociology ('The Study of Sociology', 'Principles of Sociology') but also to the collectivist aspect of his work on psychology.

Being a time of debates and controversies, the period from the 1850's until the mid 1870's was rich in newly founded publication organs for the various sides in those debates (over 170 new periodicals were started).[3] This meant that both the publication of the 1st and the 2nd edition were not singular events but part of an ongoing activity in which the issues discussed in Spencer's books formed one thread in a yarn in which things scientific, political, social and biological, not to mention religious, were intimately interwoven. [4]

The two editions of the 'Principles of Psychology'

Spencer published the PP1 in 1855. It consisted of one volume of some 620 pages; He issued the second edition first as sections to subscribers and then as volumes between 1870–72. The three volumes that made up the second edition totalled around 1800 pages. The 3rd edition appeared in 1880 and was identical to the second except for one chapter of some 50 pages set almost at the end, called 'congruities'.

The PP in its first edition was Spencer's second book. He had published his *Social Statics* in 1851. Given that his essays just before and after issuing the first edition dealt with aspects of progress and evolution, the question arises why he did decide on psychology as his project. His testimony does not answer that question,[5] and thus one is left to guess. I view it as an attempt to lay down the foundational assumptions that would enable him to pursue his enterprise of refashioning the contemporary human sciences. PP 55' is foundational in the following sense: it dealt with 'human nature', 'human understanding', and the requirements for humans 'to associate together' — the ever recurring themes of 18th- and early 19th- centuries philosophers — but with a difference: those parts that dealt with philosophy were based on physiology, and the parts that dealt with psychology or 'the social' were based on an evolutionised physiology. It aimed to encompass all the known functioning of the human mind and of the human psyche, but it did so by situating humanity as a component of a natural continuity. The basic analytical units, the mechanisms, the generalisations, the laws, all would have to apply to living organisms at large *in*

2 Eg: Major works in British anthropology written between the appearance of the 1st and the 2nd edition of 'Principles of Psychology': Maine – Ancient law 1861; Lubbock – Prehistoric Times 1865; McLennan – Primitive Marriage 1865; Tylor – Researches into the early History of Mankind 1865; Lubbock – The Origin of Civilization and the Primitive Condition of Man 1870; Maine – Village Communities in the East and West 1871; Tylor – Primitive Culture 1871.

3 Among the new periodicals some had been very short–lived, others survived until well into the early 1880's. I have noted down some of those which either published articles or reviews on Spencer's work, or dealt with issues similar to those discussed in his books of those years: The Leader 1850, The Saturday Review of Politics, Literature, Science and Art. 1855, The National Review 1855, The Oxford and Cambridge Review 1856, Bentley's Quarterly Review 1859–60, Macmillan's Magazine 1859, National Reformer 1860, Cornhill Magazine 1860, Home and Foreign Review 1862, The Reader 1863, The Fortnightly Review 1865, The Contemporary Review 1866, The Academy 1869, Fraser's Magazine 1870, The New Quarterly Magazine 1873, The Nineteenth Century 1877; The Natural History Review 1854, The Naturalist 1864 , Nature 1869; The Geologist 1858, Geological Magazine 1864, A Quarterly Journal of Science 1871; The Anthropological Review 1863, The Journal of the Anthropological Institute of Great Britain and Ireland 1872; Asylum Journal of Mental Science 1853 continued by Journal of Mental Science 1859–1875, Mind 1876, Brain 1878 , Journal of Physiology 1878.

order to apply to humans. It 'naturalised' human behaviour by putting it on a par with the behaviour of any living organism. The patterns and the biological mechanisms of heredity that would apply to living nature would apply to human perception and consciousness, as well as to unconscious, purposeful, planned and spontaneous human activities; to thought, feelings, desires, and to both individual and social behaviour. Thus, to create and establish a new psychology meant to lay the foundations for sociology, anthropology, politics and ethics.

Spencer's position on heredity when applied to all these fields had far reaching implications. In both editions, in so far as Spencer employed Lamarckian assumptions, there was an *intentional* effort to *blur* the distinction between biological heredity and cultural inheritance. The hereditary mechanism resorted to a delineated, alternating–rhythmic pattern between the two. The important feature was in the choice of 'basic units', which were neither solely mental nor solely physiological (and their process of recombining, etc was a hybrid one too). He resorted to the method of analysis characteristic of the empiricist–sensational associationist schools– i.e. *to choosing basic units which were assumed to be the most elementary, primitive, while all other elements were regarded as combinations and compoundings of these units.* In contradistinction to his predecessors, the evolutionary mechanism he had adopted operated on these basic units and their compoundings resulting in processes of *complexification*. Let me give as an example Spencer's discussion of the emergence of consciousness in the two editions. Both editions indicated the significance of 'change' as a constitutive element in the explanatory framework. In the 1st edition such a basic unit was what Spencer defined as 'resistance' – 'action by direct contact, being the primary action, and at the same time the simplest and most definite… the sensation of resistance, through which this fundamental action is known.. …(resistance) is the only species of external activity which we are obliged to think of as objectively and subjectively the same'(1st edition p 269–270) – a hybrid unit. [6] In the 2nd edition Spencer gave an example of the most elementary physiological–neural unit, once again a hybrid, in discussing consciousness: 'which state of

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[4] This becomes apparent when one looks at the list of major writings by Darwin, Lyell, Spencer and Wallace between the beginning of the 50' and mid 70's: Spencer *Social Statics* 1850; Spencer *A Theory of Population , deduced from the General Law of Animal fertility* 1852; Spencer*Development hypothesis* 1852; Spencer*Principles of Psychology* 1855; Spencer*Progress, Its Law and Cause* 1857; Spencer*Transcendental Physiology* 1857; Wallace *On the Tendency of Varieties to Depart Indefinitely from the Original Type* 1858; Darwin *On the Origin of Species by Means of Natural Selection.* 1859; Spencer *The Social Organism* 1860; Lyell *The Geological Evidence of the Antiquity of Man* 1863; Wallace *The Origin of Human Races and the Antiquity of Man* 1864; Wallace *The Origin of the Human Races and the Antiquity of Man deduced from the Theory of Natural Selection* 1864; Spencer *Principles of Biology* 1864; Darwin *The Variation of Animals and Plants under Domestication* 1868; Walllace *The Malay Archipelago* 1869; Wallace *Sir Charles Lyell on Geological Climates and the Origin of Species* 1869*;* Wallace *Contributions to the Theory of Natural Selection* 1870; Darwin *The Descent of Man and Selection in relation to Sex* 1871; Darwin *The Origin of Species by Means of Natural Selection.* 6th edn 1872; Spencer *Principles of Psychology* 2nd edn 1870–72; Darwin *The Expression of the Emotions in Man and Animals.* 1872; Spencer *The Study of Sociology* 1873; Spencer *Descriptive Sociology* 1874*;* Spencer *The Comparative Physiology of Man* 1875; Wallace *The Geographical Distribution of Animals* 1876

[5] Spencer himself, in his *Autobiography*, noted that 'Psychology underlies sociology; and there had to be specified a number of those more special truths of psychology which have to be handed in to sociology as part of its data' (*Autobiography,* vol ii, p. 240). Similarly he stated 'After dealing with general psychology, it became requisite to enter upon the special psychology of man in preparation for sociology. Certain traits of human nature are presupposed by the ability to live in *the associated state*…' (Duncan, *Life and Letters of Herbert Spencer*, vol ii , p.355, my italics)

consciousness caused by a blow, may be taken as the primitive and typical form of the nervous shock… something of the same order as that which we call a nervous shock is the ultimate unit of consciousness;…'(2nd edition vol I p. 151). As Duncan indicated, Spencer in the 2nd edition '…ventured the hypothesis that sensations of all kinds and by implication higher feelings of all kinds result from the compounding and re compounding…so that there is an ultimate element of mind…'(Life and letters, ed. Duncan, vol ii, 348) . Human competences (or 'tendencies' or 'latencies') were neither solely biological nor solely mental–cultural. They were pre–organised moulds, inherited as neural modifications which functioned as neural–psychological patterns for organising experience and behaviour. At the same time they were open to further functional–adaptational modifications by that experiencing, which in its turn would be biologically inherited. Thus biological heredity was cultural inheritance; and heredity was conceptualised on the boundary between the individual and the collectivity (the 'species', the 'race'). The Darwinian mechanism of natural selection played a very subsidiary role. Darwin was read through Lamarckism (which generally agrees with P. Bowler's argument) and the role of variations in Spencer's discussion of Darwinian mechanism was almost merely rhetorical. In fact, having both mechanisms helped Spencer draw a distinction between human 'mental' evolution and evolution at large.

The central role of environment and of adaptation to its changing conditions could, theoretically, be interpreted in two major ways:

I. As a facet of a hard–core biological determinism that would overrule whatever effects social changes of environment could have. The capabilities of the individual would then be conceived as merely a reflection of the conditions of existence of the species and thus be bindingly hereditary. Biological determinists could then apply concepts of biological purity to race, to class.

II. The role of environment would be expressed through the weight of the formation and the transmission of socially and culturally functional, adaptational patterns such as habits, customs, traditions, and thus it could highlight 'progress' as an open ended endeavour.

As Spencer conceived of biological evolution as a movement from homogeneity to heterogeneity, and understood it as complexification, it could, to a large extent be identified with 'progress' when applied to humans. Even the distinction between white and non–white races was not wholly a determinist one in PP. Lamarckism offered a double perspective on ethics and on society at large. It emphasized the overall importance of the milieu in shaping present and future generations through the inheritance of acquired characteristics, with 'use–repetition–habituation' as a major explanatory mechanism. This implied the possibility of shaping the future: present changes could be bequeathed as prospective biological traits to be further elaborated in the future. The seeming determination of the present by the past was one consequence of such thinking, the other being the moulding of the present in light of a projected utopian future. But there was a price to be paid. What made possible this seemingly magic alternation between the cultural and the biological, which allowed for hybrid basic units, which made room for psychology as a unique

[6] Spencer's explanations of how space and time, motion and matter , were constituted derive from the analysis and then extrapolation of that hybrid basic unit). In choosing effort–resistance as a basic unit Spencer aligned himself in more than one sense with the psychological programme of the ideologues and its derivatives. In fact, this programme had been one of the principal sources of influence upon Lamarck's later work on Man in the 'Systeme analytique' and in some dictionary items he had written in the second decade of the 19th century.

science of the individual, which sanctioned the bridging between the solely experienced and the absolutely innate without ascribing to Kant's transcendentalism and its vagaries, was the role assumed by the collectivity in Spencer's theory. The Spencerian individuals contained the collective within themselves as their adaptational mode. This was the collective inheritance transmitted as biological heredity. It formed the moulds which fashioned and shaped the individuals' experiencing, behaviour and thought.

The changes were truly feasible only on the level of collectivities because though modifications were inherited by individuals, they were effected and inherited as social habits and psychological patterns (e. g. in the *corollaries*– cooperation). Thus the 'future perspective' of Lamarckism was wholly dependent on its being a social, collective rather than an individual construction.

Evolutionising meant accepting the priority of the collectivity at least on the methodological and epistemological levels. In that sense Durkheim made explicit the implicit cost of Lamarckian evolutionising in the human sciences. The later crisis of neo–Lamarckism both in biology and in the human sciences helped turn collectivist assumptions into a methodological anathema.

I would further suggest that in the period between the late 1840s and early 1870s, ((in some sense until the implications of Galton's *Hereditary Genius* (1869) sank in) evolutionising psychology meant to discuss physiology and heredity in conjunction. In the more general context of 'progress' it meant gradual racialisation both in terms of imperial policies and in terms of social class determination on the one hand and of human developmental potentialities from a psychological point of view on the other hand. In this interim period individuals from the British middle class could gain a measure of immortality as their experiencing became that of the collectivity *jointly,* and the collective patterns became part of each individual *singly*. Moreover, behavioural patterns– habits– of the individuals were conceived as shapers of the cultural history of the collectivity. But it is doubtful, that it was a way to overcome the self–interested discreteness of individuals in most of political and economical theories of the time.

The 1870's can be viewed as a fairly clear dividing line between those who would investigate the brain only, and those who would discuss categories of thought culturally and philosophically only (e.g. the Neo–Kantians). The mixing of modes of discourse, of metaphors and analogies which Spencer used — philosophical, medical, biological, general–scientific — would not have been possible later on. Thus, in more than one sense, a comparison of Spencer's two editions affords today's reader an insider's gaze into the passage from one phase to the other, and not just in terms of evolutionary models used, or conceptions of heredity.

As is well known, from the mid 1860s until the late 1880s Spencer's writings were translated and disseminated all over Europe and the USA. They were reviewed, discussed and emulated. His writings had an impact on the human sciences of that era in a way which has not yet been adequately delineated. The period between his 1st and 2nd edition of PP was roughly the time in which anthropological work became more clearly divided into what we now call physical and cultural anthropologies. The last third of the 19th century was also the period where one finds more writings on society which intended to produce a synthesising oeuvre that encompassed an analysis of modern society, its structure, its dynamics, its peculiarities and what was then termed 'its ills' or 'its pathologies'. This usually came together with a genealogical narrative, often couched in evolutionary terms and intended as a causal explanation. I would claim that the underpinning

of at least some of the then more influential sociologies and cultural anthropologies was a peculiar combination of evolutionised biology with Lamarckian hereditary mechanism. This amalgam was first encountered and put to usage for that purpose in Spencer's psychology. Quite a few of those who used the rhetoric of Darwinian 'natural selection' did in fact read Darwin along the same lines as Spencer, i.e., through some version of Lamarckism. The 'Lamarckian amalgam' made it possible to deploy the biological reservoir of problématiques, models, metaphors and analogies both for legitimation and for constitution. It was of great significance to fields of knowledge that were in the process of establishing themselves as autonomous and later as academic disciplines of the human sciences. One of the main assets of this Lamarckian amalgam was its hereditary mechanism which in many extant versions at the time allowed for cultural inheritance to be biologised and vice versa. The 'Lamarckian amalgam' catered also for the urgent need to present these theories not only as 'diagnoses' of the past and the present, but also as suggestions for potential cure, 'prescriptions' for the future, based on 'scientific analysis'.

By evolutionising psychology Spencer gained hitherto untapped resources for psychological analysis, which provided numerous examples and illustrations. These were the materials authored by social and anthropological thinkers. Just as a Lamarckian evolutionist might use examples for the process of complexification from organic nature, Spencer used examples from 'social nature' as described by social thinkers, and particularly by anthropologists. In this connection recall that by the turn of the 18th century the then existing traditional, principally non- European societies had already been presented as earlier phases or stages in human progress (at times dubbed 'human civilisation'). By the middle of the 19th century this was collapsed with a rather rigid view on the capabilities and potentialities of whites of European origin as compared to non–white groups, ordinarily described as 'the lower races'. The portrayal and analysis of 'the poor', 'the lower classes' in general and of women were often conducted using the same rhetoric, and the same categories, attributes and localisation on the scale of progress. Though many of the anthropological and sociological theories of that period appeared to be methodologically individualistic, their implicit assumption was in fact collectivist in the above stated sense. Spencer's psychology was the first to boldly espouse this mechanism for the purposes of describing 'human nature' and 'human society', and thus the first to harbour this discrepancy between claimed and de facto methodological assumptions: this had significant implications in his and in other contemporary theories.

In a more detailed paper on which this paper is based I go into a rather detailed comparison of the two editions: first their overall plan, and then to issues related to the methodology and to the rhetoric of psychology as a new science. This serves as a background to my discussion of Spencer's evolutionary mechanisms and their consequences: in terms of evolutionary models used, Lamarckian evolutionism, and the meaning and usage of Darwinian 'natural selection'. Here, I shall just briefly highlight each of these latter issues.

Psychology was constituted by Spencer in the 1870s edition as a 'relational science', a science that dealt with the *relationship of* the relations among outer phenomena *with* those among inner phenomena, i.e. with the relationship between environment, the organism and its physiological and 'mental' reactions to the stimuli of the environment. In both editions, each category used by

Spencer in the description, analysis and explanation of psychology became constituted as a hybrid one – neither solely mental in the traditional sense nor solely physiological.

The main feature of the evolutionary mechanism was perceived as adaptation between organisms and their environment (which for Spencer included 'social environment'). Inheritance of acquired patterns of action was its principal mechanism. 'Adaptation' was then transposed into Spencer's psychological discourse as the continual process of adjustment, conformation, correspondence, and the fitting of 'internal and external relations.'[7] The actual term 'adaptation' was used in the 1st edition only in relation to complex organisms (which, presumably, were fully active in this process). Organisms were graded according to the degree of 'correspondence' between the internal and the external environment; life itself was defined in relation to it.

In the 1st edition the continual adaptation was to be achieved through processes of differentiation, integration and thus coordination; the transposition of this model unto the domain of psychology was supposed to explain the evolution of distinctions between 'outer' and 'inner' within the states investigated by psychology, up to and including the emergence of consciousness and the explanation of intelligence. Consequently, various crucial conceptualisations such as those of space and time were presented as developing in an evolutionary manner both in the individual and in culture in general.

In the 1870s edition, evolutionary processes at large, and those which were dealt with by psychology in particular, were characterised as a passage or movement from homogeneity to heterogeneity, along the lines suggested in various contemporary essays of Spencer.[8]

In the 2nd edition there were two, rather than one, mechanisms of evolution called on to explain how change could be brought to bear on organisms and what the change consisted of. The one often called 'direct equilibration' was the Lamarckian mechanism – in which the organism was also active. The other often called 'indirect equilibration' was the Darwinian natural selection which referred both to the survival of the more adjusted or better fitted and to their predominance in terms of reproduction. Spencer thought that the Darwinian mechanism of natural selection could work in evolution in general but would not suffice to explain what he considered the extraordinary development and complexification of the neural structures/the mind in the higher organisms (i.e. in humans). In this, he aligned himself with both evolutionists and non–evolutionists during the 1860's who had adopted for various reasons somewhat similar positions.

However, the main mechanism for solving problems[9] — methodological, epistemological as well as structural — was the usage of certain presuppositions of Lamarckism, namely:

01. a graded continuity of the organic world;
02. the evolutionary process was developmental and gradual;
03. a directedness of the evolutionary change towards growing complexification by way of adaptation via increasing differentiation, specification and coordination of these. Spencer

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7 e. g. PP 1855, pp. 366–375.

8 'T*ranscendental physiology*' (1857, 'National review,' under the title 'The Ultimate Laws of Physiology') and elaborated in his book '*First Principles*'(1862). Most of Spencer's interpreters in the 2nd half of the 20th century related this to his reading of von Baer particularly via Carpenter's '*Principles of Physiology* ' (1851).

defined this (after von Baer when discussing the development of the embryo) as going from homogeneity to heterogeneity, with increasing definiteness;

04. the environment, whether external, internal, or for Spencer also social[10], affected changes directly through adaptation;
05. a predominant role was assigned to the environment as a process of 'posing problems and difficulties' to the actively reacting organisms;
06. the mechanisms of use and disuse and habituation in the organisms 'process of adaptation;
07. the organic entities discussed were to be considered as differentially self– organising;
08. the results of the process of adaptation of the individual organisms in the form of what Spencer called 'functional modifications' ('acquired characteristics' or in Lyell – 'peculiarities') were biologically transmitted inter–generationally;
09. the gradual change in individuals of a species('race' in PP) occurred in such a fashion that all members of the group, ignoring individual differences, turned out to share the same changes, or more precisely the same results of the gradual process of change. (this assumption was actually at the core of Lamarck's 'transformism');
10. all presuppositions applied equally to the whole organic nature and to humans, as 'life under all its forms has arisen by a progressive unbroken evolution and through the immediate instrumentality of what we call natural causes.'

Let me exemplify what sort of 'work' evolutionary Lamarckism was supposed to perform and at what cost. Already in the 1855 edition Spencer used habituation– habit formed as a result of repetition– as an important explanatory tool. Habituation allowed the performer (the reasoner, the speaker) to make significant shortcuts, because stages in the 'mental' process 'merged' and 'sank to the unconscious,' and the 'shorter version' became the ordinary one. One of his more detailed examples[11] was an analysis of how a child learned to classify and to read. Here evolution and development became conflated, and Spencer argued that this process of 'merging and sinking,' whereby the activity of those specific parts of the reasoning, classifying, estimating etc. were converted into an 'automated' one, started at early infancy and thus became 'organic.'

Conceptions of space and time were the *developmental* product of experiencing rather than its condition. Consciousness itself was defined as a consequence, an emergent consequence, of experiencing changes, rather than its precondition. Consequently, an infant's perception would differ from that of a child, and a child's from that of an adult. The sense of necessity of seeing the world this way rather than another, was consequent upon the 'shortcut mechanism' of 'merging and sinking'.

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9 As is well known, Spencer came from a dissenter family from Derby, and his father participated in the 'Derby philosophical society.' By his own testimony Spencer learned his Lamarckism as a young man by reading Lyell's presentation of it and its refutation (particularly the first chapters in the second volume of the '*Principles of Geology*'). One would assume that he had also read Chambers' 'Vestiges', he mentioned reading Rymer Jones, William Carpenter, Henri Milne–Edwards etc. From the late 1840s on he lived in London and was close to some of the radical figures of the 1830s. One can surmise that he was familiar with the extant versions of Lamarckism, catastrophism and other in–between contemporary narratives of evolution, as well as with the natural theology arguments against it.

10 e.g. PP 1870-72, p. 381

11 PP 1855, pp. 205–207.

For Spencer human adaptation was distinguished by the ability to learn and pass on gathered and organised information to other members of the collectivity. This process was accompanied by a parallel gradual extension of the relevant environment.[12] (In certain contexts, human evolutionary adaptation was called 'progress' and was perceived as a highly complex hybrid.)

In the then contemporary context reflex action and instinct were difficult notions to explain for both those who were committed to innate ideas and those who were not, particularly without assuming divine power. Spencer claimed that if his mechanism of accumulative–repetitive–experience was regarded as developmental *and* evolutionary, then it could explicate both. Reflex action was thus defined as the basic/primary hybrid unit, as a simple one, the compounding of which would be called 'instinct'.[13] As such the results of its action were to be passed on through hereditary transmission i.e. a biological heredity of habituated psychological patterns of experiencing acquired through the life of an individual.

The thrust of Spencer's argument concerning instinct and reflex action meant that every mental procedure had to be in some sense a learned one . Spencer was fully aware of the fact that this explanation could not suffice, given that it had to provide for the universally necessary and permanent character of both instinct and reflex action, also given individual idiosyncracies, diversity of environments etc. I suggest that Spencer *therefore* resorted to collectivist assumptions, and these became a major underpinning of the whole system. Experience and behaviour became manifestations of collective hereditary transmission, working equally on all possible generations of individuals of the relevant group. What was transmitted was not solely a psychological–cultural pattern. Rather, in line with the notion of hybrid units, a changed biological pattern, neural in this case, was hereditarily transmitted. This biological pattern, which he called 'tendency' (or 'latency') collectively provided the members of the collectivity with modified competences through which their experiences would be organised. This 'tendency' moulded the physical and psychological behaviour of individuals.[14]

These competences in their turn were the enabling preconditions for further individual accumulation of experience and thus for learning and communication. They allowed for further minute gradual modifications having had already been organised in specific moulds common to all members of that very general collectivity (humans in our case).[15]

Experience and learning processes very gradually formed and fashioned biological-psychological competences — the mechanism which operated via a collective biological heredity.

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12 PP 1855, pt. 3, ch. 8. One of the hallmarks of this process in humans was the combined ability to forge both specifications and generalisations which mutually generated relevant activities .

13 It seems that Spencer took over some of the features of reflex as propounded by Laycock, e. g. it being a mediator between organism and environment, its automatic character, its pervasiveness at all levels of the neural system. In Laycock it appeared within the context of discussing addiction (alcoholism). See also the two appendices at the end of vol I of Spencer's 2nd edition: 'on the actions of anaesthetics and narcotics, Consciousness under chloroform' pp 631–640. It is worth noting that throughout the 19th century discussions of addiction were a locus of a 'mixed discourse' on heredity—biological–psychological.

14 (pp. 526–538).

15 In a footnote which discussed perception of space: '….being inherited by the infant in a proximate form, is progressively modified by the daily activities that accompany development, until it reaches complete form.' Concurrently there were, Spencer argued, modifications that had to do with the overall adjustment of the organism on the basis of the 'ancestral one' (PP 1870-72, vol II, ch. 1, p. 193)

In the cases of instinct and reflex action the moulding, which originally might have been more elastic, became inflexible through biological transmission of gradually hardened patterns. Presumably this inflexibility had its adaptational uses. Spencer's account of instinct was just one instance of the fundamental mechanism of the new psychology, applicable to organic nature at large, to the development of an individual from infancy to maturity, and to the progress of humanity (as response to specific needs which produced that pattern in the first place). Once this form of hereditary transmission was established as an explanatory tool, it could be used as an explanation for more flexible patterns of behaviour as well. Spencer went into great length in explaining the necessary level of development of the neural system in order for such a pattern to be formed and to be transmitted. He also drew a fine distinction between automated cognition, behaviour which developed over the life span of individuals, and behaviour that was so to say, 'ready–organised' at birth. Conversely, (hereditary) instincts could, according to Spencer, become a component of some more complex activity, in which case the isolated repetitive character would become distorted, experience and action less automated, until it lost both its character and its adaptational role. The latter process seems an extrapolation of the concept of reflex as a simple automated unconscious event, stretching it to cover conscious and complex ones. Purposive behaviour could be completely physiologised in this manner. E.g. feelings, which were called 'inborn' or 'innate sentiments' in the philosophical discourse, were explained by the same mechanism of experience, repetition, habituation, inter-generational, biological, hereditary transmission, moving from the level of individual heredity to the level of collective heredity. Thus Spencer treated 'will' as a natural continuation of the same. One can hardly think of more significant concepts than 'will' and 'reason' in the Victorian ethos and in Victorian culture (for both believers and non–believers). In their diverse interpretations, these encompassed the moral code and the 'self forming' ideals of the ascending British middle class.[16]

The decomposition of sensationalist notions, of a utilitarian world view, of Kantian transcendental epistemology, of certain facets of associationism, of faculty psychology, all depended on a specific mode of biologising and evolutionising. This mode was expressed in the notion of compounding and complexifying of hybrid basic units and the notion of collective hereditary biological, neural transmission of psychological and cultural moulds of experiencing.

A brief note on Spencer's use of 'natural selection'

There were three contexts in which 'natural selection' was used, with or without mentioning variations:

1. in the 'biological' volume– when discussing aspects of development of the neural system. There it was posited as the main mechanism for its primary development, through the production of certain variations. But even there Spencer specified that in higher organisms the causal productive mechanism was the Lamarckian one (e.g. PP 1870-72, vol. I, pp. 526, 614–15).

[16] As well as in the medical discourse of that time; In this case Spencer's analysis undermined the separateness and distinctness of the particular chain called the 'ego'– but at the same time performed a salvational act by anchoring it, once again, in collective experience and biological heredity. Instead of the traditional 'will' and 'freedom of will' Specner suggested to his audiences the laws of growing adaptation.

2. in the chapter on 'Pleasures and Pains'. There Spencer assumed the hybrid basic units, and the other mechanisms discussed earlier. These accounted for the *description* of pain and pleasure, but not for their functions as tools for adaptation within the evolutionary psychological context. Utilitarians viewed pleasure and pain as instruments with the help of which to rear/raise and control individuals and lead them towards the 'right' modes of behaviour in the social–economic sphere. For Spencer pleasure and pain were more of a total gauge of the organism's (including the human) state of affairs — how it coped with vicissitudes and perturbations and the continual changes in its environment.

As indices of adaptation pleasure and pain would be found to function better in those individual organisms which lived longer, reproduced more, and thus replaced the other 'less–adjusted'. This was how Spencer viewed natural selection in that context (PP 1870-72, vol. I, pp. 279–281). This was also the kind of context in which he would use the expression 'survival of the fittest'. 'Habitat' and 'migrations' also made their first appearance in that context. It is worth noticing that Spencer distinguished there between organisms with foresight (humans and 'some of the highest allied races', i.e. apes) and other species. He then went on to produce a narrative of human evolution. This narrative was based on the familiar 18th century division of four general stages: hunting–gathering, pastoral, agricultural, and urban–industrial big societies. The relevant issues concerning natural selection are: Spencer's emphasis on 'the social' as a type of environment which called for adaptation; the failure of successful adaptation(in terms of psychology) of humans in the passage from the second to the third stage and more pronouncedly from stage three to stage four ('They were thereby cut off from activities like those of the men whose characters they inherited, and were forced into activities in which their inherited characters furnished no incentives'(282)).

3. The third case in the 2nd edition with 'natural selection' in the foreground was in the 'Corollaries' section, at the end of Vol II–2. The chapter on sympathy and sociality was mentioned earlier and the question here is whether the inclusion of natural selection made a significant difference in Spencer's analysis. This chapter was one of Spencer's ideologically laden ones in the PP. It was explicitly stated there that cooperation and mutual aid were the higher aims of social evolution, and this was juxtaposed with a sharply formulated criticism of military societies. According to Spencer, military societies encouraged selective sympathy and sociality. (In some of his later books such as '*Principles of Ethics*' one finds a 'reverberation' of the distinction found in the 'Corollaries' between sympathy exercised towards one community and its withdrawal from anyone outside that community.) One can barely regard that as an adoption of a Darwinian group selection, which because of the inner contradictions it created, hampered their own evolution towards the desired ends. We may perhaps call it, anachronistically, 'cognitive dissonances'. The occurrence of sentiments of sympathy and sociality and their increase was related to other social structural factors (power structure, family structure, societal type), spatial diffusion as well as to relative degree of intelligence. The sentiments were formed and acquired through the Lamarckian mechanism, while the survival of the fittest played the role of an additional factor ensuring the smooth functioning of the sentiment.

Clearly, the most important discussion of that in the biological volume of the 2nd edition was on 'pleasures and pains', concluding the second section –the inductions of psychology– in vol. I. Both feelings were explained in terms of the adaptational strategy of the organism. Throughout the

evolutionary process pain indicated a perturbation to the adaptational equilibrium between the organism and its surroundings, while pleasure indicated a growing adjustment. Spencer did not explain the development of such gauges on the basis of hereditarily transmitted biological transformation. Rather, he presented them as a result of natural selection: the benefit which accrued thereupon would be for the collectivity ('race'), in terms of its long term survival.[17] The role Spencer assigned to variations, which were central to Darwin's mechanism, with which he certainly became acquainted shortly after the publication of the *Origin*, was in my opinion mostly rhetorical. He mentioned them whenever the aspect dealt with was predominantly biological. Furthermore, even there, it was relegated to dealing with simpler structures and organisms. It is not clear whether the variations 'did' anything in his employment of 'natural selection'. There were, however, a couple of clear instances. One was related to the development of the nervous system, and thus clearly related to the biological aspect of the discussion. The other occasion in which natural selection was conjoined with variations was an interesting one. This was when Spencer's previous dictum on pleasure and pain as indicators of adaptation seemed to be contradicted by phenomena which he defined as '… a seemingly abnormal desire to dwell on that which is intrinsically painful.' This he found in certain modes of pity, defined as a very peculiar mixture of pain with pleasure, – e.g. watching the suffering of another – and he joined them with what he called 'all the bodily appetites.' These according to him included the sexual drive and the parental instinct. In that context he said: '…(that feeling of pity etc.) is not one which has arisen through the inherited effect of experience, but belongs to a quite different group, traceable to the survival of the fittest simply – *to the natural selection of incidental variations.*' (vol. II, p. 623, my emphasis). What did he make of these variations? In a letter to Darwin in 1871 he wrote: 'though I have endeavoured to show that instinct is compound reflex action, yet I do not intend thereby to negate the belief that instincts of some kinds may arise at all stages of evolution by the selection of advantageous variations. I believe that some instincts do arise: and especially those which are operative in sexual choice' (Life and Letters, ed. Duncan, vol. I, p. 196). In 1899 he wrote a sort of 'intellectual biographical sketch' called 'The Filiation of Ideas', in which he referred to psychology, to the inheritance of functional modifications, and argued that 'Though, nowadays, I see that the natural selection of variations in the nervous systems has been a factor, and *in the earliest stages,* perhaps the most important factor…' (Life and Letters, ed. Duncan, vol. II, p. 324, Spencer's italics) he would still stick on to his Lamarckian view etc. This fits in with the first case.

[17] Incidentally the 'Corollaries' section was the most political one in the whole of the two editions. There Spencer's position on policies and economies of war was stated bluntly. All in all, in those chapters and in the chapter 'On pleasures and pains' Spencer offered a grim view of modern military industrialised society, and gave a first approximation to what would, twenty years later, turn into two versions of binary sociological typology: that of Durkheim and that of Toennies. Durkheim in '*De la division du travail social*' and Toennies in '*Gemeinschaft und Gesellschaft*' suggested a typology of traditional versus modern societies. Schematically, and put somewhat unfairly, Durkheim criticised certain aspects of the traditional societies, particularly their homogeneity and lack of space for individual freedom, and thus called their type of solidarity 'mechanical.' While Toennies extolled their community of beliefs and solidarity of ties and support; he characterised them as 'organic'; Durkheim saw modern societies as complex (differentiation, division of labour etc.), they allow space for individual autonomy and idiosyncracies, and have a solidarity and cooperation which emerges from the webs of interdependencies among their members (thus he called them 'organic'); Toennies viewed modern societies as devoid of human solidarity, based solely on contractual ties, capital and gain oriented, socially isolating their members and he characterised them as 'mechanical'.

In his autobiography, when he mentioned the universal biological transformation in PP, he wrote that at the time he was committed to the Lamarckian mechanism ' …and was unconscious that in the absence of indirect adaptation effected by the natural selection of favourable variations…' the explanation proffered could not suffice (*Autobiography* vol. I, p. 502): '…what Mr Darwin called 'natural selection' might more literally be called survival of the fittest…'; Also 'some individuals in a species are so constituted that their moving equilibria are less easily overthrown than those other individuals, and these are the fittest which survive' (*Autobiography* vol. II, p. 100).

In the 'Corollaries' section of the 2nd edition, where social issues such as socialty, sympathy, egoism and altruism were discussed, Spencer emphasised the inaptness of natural selection in coping with adaptational situations in which complex organisms such as humans were involved. He claimed that Lamarckian mechanisms were much too slow for the fast pace of change in social environments as the alternation between the biological and the cultural needed more time to get usefully established. These were regarded as significant factors in the explanation furnished for the ills of modern civilisation.

I would argue that though Spencer did acknowledge and accept that there was another evolutionary mechanism beside the Lamarckian he used, it played a very subsidiary role in his PP. Put more generally, one could perhaps say that Spencer viewed the mechanism of natural selection as one that did a 'negative work', rather than the creative one which Darwin attributed to it. This brings me to reiterate my claim that Spencer's edifice of PP cannot be understood without taking into account his brand of Lamarckism.

To conclude–

I have argued that in both editions Spencer's innovative stance stemmed from a particular evolutionising of an amalgam of tenets from the then current mental and physiological theories of psychology. One of the consequences of this was that it became possible for Spencer to posit hybrid categories– neither solely physiological nor solely mental, and to intentionally blur the demarcation between biological heredity and social, cultural and psychological inheritance. This whole edifice could be coherently sustained if the underpinning of the blurring or hybridising became collectivist, while the rhetoric – political, ideological, and scientific – remained individualistic, and thus could provide legitimation for a new science.

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Writing Heredity: Emile Zola's Rougon-Macquart and Thomas Mann's Buddenbrooks

Ulrike Vedder

I.

„Writing heredity": I have bracketed these two terms to explore the relations between two sets of concepts: heredity and its theorisation, on the one hand; and the writing process, respectively literacy, on the other. In doing so, I do not intend to present Zola as a theorist of heredity who assimilated the theories of his age more or less accurately; nor do I intend to present Thomas Mann as a genealogist who 'applies' theories of degeneration to his characters. Instead, I shall take Zola as an example to examine how writing and different media operate in theorising heredity; I will focus on the last novel of Zola's cycle *Les Rougon-Macquart*, which circles around Docteur Pascal, a physician and scholar of heredity; subsequently, and by way of brief comparison only, I will bring into view the written media that appear in the context of the genealogy of Mann's *Buddenbrooks* by considering their role in establishing, respectively abolishing such genealogies.

II.

Le Docteur Pascal (1893) is the twentieth – and final – novel in Zola's *Les Rougon-Macquart* (1871-93). As is well established, the cycle is subtitled *Histoire naturelle et sociale d'une famille sous le Second Empire*. The subtitle is striking in several respects, such as its opposition of nature and culture – that is bridged by a plain and simple 'and'; moreover, we note Zola's use of the term *histoire naturelle* to refer not just to the human species, but also to a single genealogical group. His time indication is striking, too, in that *histoire naturelle* does not envisage any *longue durée,* but merely the nineteen years of the Second Empire. Zola had already consigned the Second Empire to history with the publication of the first volume in 1871 and brought it to a narrative conclusion in volume 19, *La Débâcle* (1892), that offers an account of the defeat suffered in the Franco-Prussian War of 1870/71. So if he intends another volume to close the cycle a second time, it functions to render an account of the family's demise – and of a feeble new beginning – following the downfall of empire – beyond all political coordinates, focusing fully on the family's 'histoire naturelle'.

Heredity

Whereas Balzac's *Comédie humaine* had focused on the synchronic 'cataloging' of the social species on the lines of zoological species, Zola's „histoire naturelle" can be considered a change within the species, that turns on heredity as its pivot. Given its lawfulness, as attested by science, heredity – as a biological term – has the potential to structure Zola's voluminous serial novel, quite unlike mythical fate that would amount to little more than a ridiculous organisational principle in the face of the cycle's expansiveness – and that would lack modernity, moreover. And as Zola describes his plans for the cycle in a preliminary note, „Il ne faut pas user du mot fatalité, qui serait

ridicule dans dix volumes. Le fatalisme est un vieil outil". *Les Rougon-Macquart* – initially conceived as a cycle of ten novels and later amounting to no less than twenty – thus unfolds the question how ancestral disposition takes effect on descendants and which variants emerge in the various constellations. Such structuring by the laws of heredity aims at the underlying, deeper dimensions of a genealogical order, whereas unstructured, unordered diversity reigns on the surface, among the individuals observed, that is; as Zola suggests, it calls for hereditary analysis: „individus, qui paraissent, au premier coup d'œil, profondément dissemblables, mais que l'analyse montre intimement liés les uns aux autres".[1]

In the *Préface* to the entire cycle, Zola formulates his programme to make visible the underlying, submerged 'liaisons'; he also names the technique for doing so: „Je tâcherai de trouver et de suivre [...] le fil qui conduit mathématiquement d'un homme à un autre homme."[2] This thread, which interconnects human beings and marks a path toward cognisance, opens up a broad field of meanings: (1) To begin with, the thread as a plaited connection evokes three concepts: the rhizomatic network or mesh; the hierarchized family order, for instance as a genealogical (that is family) tree; and Wittgenstein's supposedly linear, yet by all means brittle conception of family ressemblance. (2) The thread as a means of orientation refers to the labyrinth, and (3) the thread as a steering mechanism evokes an author holding all the strings; as Zola notes in the *Préface*, „je tiendrai tous les fils".[3] Zola thus bases his twenty-novel cycle on the concept of lawful heredity as a prestigious model sanctioned by science. He adopts it as a narrative technique of authorial control, and strives to realise the programmatic verve of the preface throughout the entire cycle, in which heredity proves to be an extraordinarly productive generator of novels: „Je dois le suivre strictement, il est en même temps ma force et mon régulateur."[4] The novel concluding the cycle is about the theory and technology of such literary generativity, that converts life into writing and generates life through writing – albeit lives that exist but on paper and whose veins convey but ink. This last novel, which Zola himself referred to as „conclusion scientifique de tout l'ouvrage",[5] elaborates once more the interrelations between the various family members – over five generations – that have appeared in each of the novels by seeking to systematise and theorise them.

The interrelations are organized along hereditary – and not along psychological or juridical lines, for instance – in accordance with a notion of heredity that is conceived particularly in pathological terms.[6] A scene set in an asylum where Adelaïde is an inmate makes this point clear. Adelaïde, 105 years old, is the family's 'great mother', „leur mère à tous"[7]; not only do both branches of the family go back to her – the legitimate Rougons and the illegitimate Macquarts – but so does the transmission of hereditary phenomena of degeneration: „la lente succession des accidents nerveux et sanguins".[8] At the asylum, Charles, Adelaïde's debilitated great-great grandson, sits across from his grandmother, who is paralysed and undead, and has fallen into a

1 Zola (1960), p. 3.
2 Ibid.
3 Ibid.
4 Zola (1961), p. 799. Gumbrecht focusses on Zola's problems to separate the synchronic (social) and the diachronic (biological) perspective (cf. Gumbrecht (1978), p. 41).
5 Ibid., p. 799f.
6 Cf. Föcking (2002).
7 Zola (1967a), p. 1105.
8 Zola (1960), p. 3.

profound silence over the years; Charles unifies various impairments of previous generations of his family in that its central nervous and celebral deficiency to exert itself in a „débordement des appétits“[9] in a compulsive-obsessive manner – reaching as far as *décadence* and madness, illness and murder – has assumed the physiological features of a haemophiliac. Adelaïde und Charles look remarkably alike, which makes them appear as if they bracketed their lineage – „la chaîne se déroulait, dans son hérédité logique et implacable“[10] – and their deaths coincide: Whereas Charles gently bleeds to death, the apathetic aged woman is stricken by shock – because the steady flow of blood reminds her of the bloody deaths of her lover and her grandson – and dies of cerebral apoplexy. In this spectacular scene, the diversification that unfolds in the course of the cycle's novels thus becomes compressed to absolute closeness [closure], which is accounted for in hereditary terms.[11]

The fact that the family destroys itself through the continued transmission of hereditary impairment reveals just how close(d) it is: „la famille brûlera comme une matière se dévorant elle-même“, as Zola notes in the *Plans préparatoires.*[12] This closeness takes effect, too, with respect to Pascal Rougon, the principal protagonist, even though he positions himself outside the family: Not only does he not bear his family's name and is addressed simply as Docteur Pascal, but he acts as his family's external positivist observer and scientific investigator, studying it „avec l'attention d'un naturaliste surprenant les métamorphoses d'un insecte“.[13] He makes use of his family as a generalisable „exemple à la science“,[14] that is as material for his work on theories of heredity. He has been observing and recording his family's hereditary development for thirty years, under implicit and explicit reference to various contemporary theories: : „Il était donc allé des gemmules de Darwin, de sa pangenèse, à la périgenèse de Haeckel, en passant par les stirpes de Galton. Puis, il avait eu l'intuition de la théorie que Weismann devait faire triompher plus tard, il s'était arrêté à l'idée d'une substance extrêmement fine et complexe, le plasma germinatif, dont une partie reste toujours en réserve dans chaque nouvel être, pour qu'elle soit ainsi transmise, invariable, immuable, de génération en génération.“[15] Prosper Lucas' *Traité philosophique et physiologique de l'hérédité* (1847-50) enters Pascal's theory of how heritable information [genetic material] determines the human being and shapes his classification of four cases of heredity: „l'hérédité directe“, „l'hérédité indirecte“, „l'hérédité en retour“, „l'hérédité d'influence“. The fifth case, „l'innéité“, completes the schematic that has the structural advantage of allowing the classification of all human beings without fail. Pascal classifies himself as a case of „innéité“, a family member unimpressed by the laws of heredity, thus defining himself as an uninvolved exception, as an objective and dispassionate witness, as a scientific documentarian („un de ces cas fréquents qui font mentir les lois d'hérédité“).[16]

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9 Ibid.
10 Zola (1967a), p. 976.
11 But this spectacular hereditarian continuity is irritated by the narrative and its allusions to royal images (Charles is cutting out kings and queens out of paper, he has a royal air, as a haemophiliac he alludes to royal families etc.), that contradict the scientific claim of the novel. Cf. Warning (1999), p. 248.
12 Zola (1967b), p. 1741.
13 Zola (1960), p. 301.
14 Zola (1967a), p. 1015.
15 Ibid., p. 946. Cf. Schmidt (1974).
16 Zola (1967a), p. 66f.

And yet he is involved in his family and its laws on several levels. Indeed, the fact that he has chosen his own family as the material basis of his research ties him to it. His critical state of health that will lead to his death confronts him with his family affiliation, too, as it also compels him to look for the cause of his impairment in the hereditary ailments of previous generations. As such, this reveals Docteur Pascal as a rather ambiguous figure, whose detached indifference toward his object of study collides with his involvement in „le legs inévitable de sa terrible ascendance".[17]

Yet it is Pascal's incestuous relationship with his niece Clotilde Rougon – which remains surprisingly uncommented – that manifests the family's closeness in the most far-reaching manner. The novel offers a euphoric description of their work on Pascal's research, their great love, the celebration of their union and their life together, resulting in the birth of a new generation – albeit not until after Pascal's death. Although both characters are demarcated from the Rougon-Macquart – Pascal has brought over Clotilde from the inner circle of her family to live with him and has thus 'corrected' her hereditary disposition („tu as corrigé mon hérédité"[18]) – their choice of lover ties them nonetheless to the confines and bonds of their family. Phantasmatic and narrative aspects of their incest are superimposed here. On the one hand, incest does not have the connotations of *décadence* and hereditary decline, but authorizes itself through health and nature. The greatest possible closeness between Pascal Rougon and Clotilde Rougon therefore aims at making new the Rougon family, which their child personifies. It is not the combination of difference that generates the new, but the doubling of resemblance, of the near-to identical. This comes to a head in that the child born of this incestuous association is elevated and consigned to the realm of myth: It appears in the last chapter (after Pascal's death) as a redeemer, a saviour of the human species, as a Messiah even.

Another narrative function that incest has here is to cover the incompleteness, brittleness, the „fêlure" that the greatest possible family closeness brings to a head, threatening the Rougon-Macquart family and its underlying theory of heredity time and again. In Zola's seventeenth novel *La Bête humaine*, there is mention of the „fêlure héréditaire" – of the cleft, of the fissure, of the void – from which the protagonist, who is losing himself, ensues, „des trous par lesquels son moi lui échappait". Gilles Deleuze has referred to this "fêlure héréditaire", this hereditary decomposition as the „grande hérédité", the proper heritage of the Rougon-Macquart that is, that the account of the everyday hustle and bustle might well conceal, but that nonetheless inscribes death in the novels themselves.[19] The „fêlure héréditaire" undermines both the scientific and aesthetic perfection of the family's genealogical tree – which I shall turn to in a while – as much as the devoted vitalism with which Pascal and Clotilde evoke „la vie" evermore and factor out death. Insofar, their incest can be read as an attempt to conceal the fatal void with the greatest possible proximity and closeness.

Following these comments on the key significance of heredity for the novel, I would like to consider the relevance of writing and of written media to heredity and its theorising.

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17 Ibid., p. 1164.
18 Ibid., p. 1154.
19 Deleuze (1972).

Writing

Pascal develops his theories of heredity in a number of ways: through observation and by keeping records of his observations, by collecting documents in files, and by distilling and visualizing these in a genealogical tree. His filing cabinet contains a wealth of files, manuscripts and notes that require ordering and need to be assigned meaning, respectively pulled together, to make sense. The material calls for a structure that satisfies both scientific and aesthetic requirements. Structuring this material will not result in a definite form, such as a printed scientific treatise, but will rather assume the shape of continually reworked manuscripts striving to grasp new aspects of heredity theory. Such openness to change, for which the file („le dossier") is an indispensable medium (as I shall explore in more detail below), reveals at once the mobility of the object of writing, namely life, that „vie" that Zola evokes incessantly in the novel. Designing a book of life by deciphering the book of life: this is not only the objective of Docteur Pascal, physician and scientist, but also that of Docteur Pascal the documentarian and author.

Insofar as Pascal's writing starts out from observation(s) and experience(s), that is from empiricism, it is closer to narrating than to theorising. In this respect, we come across that quintessential scene in which he explains his research to Clotilde, recapitulating his entire family and its heredity interrelations in the process. He does so by tracing his finger along the genealogical tree and offering a detailed and passionate account. The knowledge that the tree formalizes is thus not codified once and for all, but can be transposed into oral accounts, scientific treatises and – last but not least – into novels. This scene amounts to a *mise en abyme* of the entire cycle, since not only do the titles of all twenty novels occur verbatim here, but so, too, does an abridged version of the novels' storylines based on their principal characters. Just as the cycle is mirrored in this scene, so too is its personnel – Pascal und Clotilde – mirrored in the family's history. The literary self-reference that is apparent in this *mise en abyme* thus produces an overwhelming mirror effect that centres attention in a compelling manner on heredity, conceived as a theorising of self-reference (in that heredity produces family resemblance, for instance, which in turn provides evidence for heredity).

Docteur Pascal's hypothesising, described recurrently, can be transferred onto the novel itself, respectively the entire cycle, which can be conceived as a hypothetical enactment. These are writings that have no place in scientific-academic writing because they are unable to articulate any final knowledge; rather, they 'test' thinking and knowing in the novel's fictional space, that thus assumes the character of a laboratory.[20] The openness of this fictional space corresponds with the openness of the question about heredity, which partly explains Pascal's passionate[21] preoccupation with it, „parce qu'elle restait obscure, vaste et insondable, comme toutes les sciences balbutiantes encore, où l'imagination est maîtresse."[22]

The way in which heredity is written conditions the way in which it is conceived. Here, it is „l'imagination" that marks out the fictional space in which scientists come by their first hypotheses and that both scientists and poets inhabit: „le domaine des poètes autant que des savants".[23] The poets, however, form the „avant-garde". This is able to discover unknown

[20] Cf. Preiss (1983), p. 126.
[21] Pascal, „amant discret de la science" (Zola (1960), p. 72).
[22] Zola (1967a), p. 947.

territories and new solutions on account of the power of literature to access the world: „ils découvrent les pays vierges, indiquent les solutions prochaines“ (1008).[24] The *mise en abyme*-scene has shown that Docteur Pascal is both a scientist *and* a narrator, respectively an author.[25] On the same terrain, poets have other means and technologies than scientists, even if the media they apply in their writing intersect, as the cases of the file and the genealogical tree illustrate, to which I shall now turn.

Files

Pascal's filing cabinet, one of the most essential objects not only of his study, but of the entire novel, has pivotal status. The key to it is a central object of desire. It wanders from pocket to pocket to be kept as safe as possible from view; eventually, it finds its ultimate hiding place – under the dead Docteur Pascal's pillow. The key functions as a constant pointer to the cabinet, behind whose doors the files of the Rougon-Macquart family have been collected to be deciphered for the invisible laws of heredity. Even if there is a confusion of papers, a „pêle-mêle“, in the depths of the cabinet, the family files have been put into methodical order: „toute une série d'énormes dossiers s'alignaient en bon ordre, classés méthodiquement. C'étaient des documents divers, feuilles manuscrites, pièces sur papier timbré, articles de journaux découpés, réunis dans des chemises de fort papier bleu, qui chacune portait un nom écrit en gros caractères.“[26] Sundry papers – documents, handwritten sheets, lettered stamping paper and newspaper cuttings – are gathered between strong blue file covers that have been labelled with large letters. Here, a bureaucratic ambition steps alongside the scientific one, emphasizing the materiality of the medium under reference to different qualities of paper and various functions of registration. In a narrative perspective, files and dossiers are specific insofar as they report the history of their own genesis in logging the entries of the documents they contain, for instance. They operate additively, absorbing everything indiscriminately and unresistingly; through the internal mobility of their contents, however, they enable synoptic readings under differentiated keywords, ever new readings that is. This, however, requires that the filing cabinet is not merely a labyrinthian repository, but an item of furniture permitting the ordering, relocation and further use of files, thus making it possible to couple them back to the user. In Docteur Pascal's case, alphabetic and chronological order compete against each other, depending on whether he is interested in individual specifics or connections relevant to heredity and succession.[27]

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23 Ibid., p. 1008.

24 „Ah! ces sciences commençantes, ces sciences où l'hypothèse balbutie et où l'imagination reste maîtresse, elles sont le domaine des poètes autant que des savants! Les poètes vont en pionniers, à l'avant-garde, et souvent ils découvrent les pays vierges, indiquent les solutions prochaines. Il y a là une marge qui leur appartient, entre la vérité conquise, définitive, et l'inconnu, d'où l'on arrachera la vérité de demain...“ (Ibid.)

25 Cf. Borie (1981), p. 140: „[...] en se proclamant docteur, Pascal ne dit pas toute la vérité, dans la mesure où il est aussi l'archiviste et, pourquoi se le dissimuler, le créateur de sa redoutable famille. Des 'Rougon-Macquart' et de leur auteur, ce dernier roman offre un tableau en abîme: l'écrivain y figure au milieu des siens, entouré de ses personnages asservis, comme le chasseur de ses trophées.“

26 Zola (1967a), p. 919.

27 Cf. ibid., p. 1008.

The medial conditions of the file just mentioned lead Docteur Pascal to ever new theorising of the laws of heredity, whenever he attends to the files and recombines the documents gathered therein. This results in a correspondence of the media format – the files – and their subject (heredity), because combining is a procedure that is both file-specific and concerns heredity at one and the same time. This becomes apparent when Docteur Pascal is taken ill and asks himself which case of heredity applies to him: „les dossiers répondaient par toutes les combinaisons possibles. Elles se présentaient si nombreuses, qu'il s'y perdait".[28] In his constant rereading and recombining of files he loses his way in different heredity cases, which yield different forecasts for him depending on their combination. After all, files have a retrospective orientation, like the modelling of the laws of heredity; their forecasting power, moreover, is highly doubtful.

The filing cabinet is emptied by Pascal's mother, in the night he passes away. She has been wanting to destroy his records for years to be able to make public her family's fame above reproach and beyond blemish, to erase the dark stains that have tarnished her family's history. Now she goes about burning all the papers in the open fireplace. In consigning the documents to the fire, she separates the files out into individual sheets, though not to read them, but because separate sheets burn better than convoluted paper. Destroying the files and therefore family memory thus replaces reading in order to erect another memory in place of the mobile papers, namely the endowment of a home for senior citizens through which she intends to monumentalize – and petrify – family fame. While the ceremonious laying of the foundation stone of the „Asile Rougon" is underway in the last chapter, Clotilde sits in Pascal's former study with her baby, as his will has bequeathed his estate to her. She is stowing baby clothes away in what used to be Pascal's filing cabinet, and comes across some half-burnt remnants of files. Even though these are barely legible, so that the research that had preoccupied Pascal for years is destroyed and science is thrown back by twenty years, the remnants provide Clotilde with an opportunity to furnish an account of her own. Figures, memories, stories – that is living literature – arise from these remnants: „les phrases se complétaient, un commencement de mot évoquait les personnages, les histoires. [...] Et chaque débris s'animait" (1215).[29] The wreckage – „débris" – reveals that it is not a matter of completeness when it comes to furnishing a narrative from the archive. Every narrative is always only a version of the archive, whereas the archive is an open labyrinth through which a number of paths could lead. Narrating depends on which guiding thread ('fil conducteur') is chosen.

Genealogical tree

Alongside the illegible remnants of the files, the family's genealogical tree has survived the auto-dafé unharmed, although it has become unreadable without the files. Whereas the files furnish an account of how they came into existence – as I have discussed above – the genealogical tree more or less distills their essence, operating as a document that has no knowledge of its genesis – which is even more amazing as this is a matter of a family lineage drawn out in schematic form. Nevertheless, the family's genealogical tree as it is published in *Les Rougon-Macquart* can be made visible in its genesis: in the different models of trees that Zola tries out during his working process.

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[28] Ibid., p. 1033f.
[29] Ibid., p. 1215.

Following an outline by Zola, the physician Georges Pouchet drew an "arbre mathématique"[30] (fig. 1) as an attempt to calculate the hereditarian relations (1:1 in the first generation, 1:2 in the second, 1:4 in the third generation etc.). According to such a law of proportion the relationship between the first and the fifth generation, between Adelaïde and Charles should be 1:16 – and not appear as if they bracketed their lineage. Another possibility is not a diagrammatic but a written tree (fig. 2) that can even be read from the left to the right, from the legitimate to the illegitimate branch that is. In 1878, Zola published a genealogical tree with the eighth novel *Une page d'amour*. But as the cycle progressed the writings overlapped the frame, more persons and more characteristics were added, and what Zola had called "ma force et mon régulateur", „sans me permettre d'aller ni à droite ni à gauche",[31] gained its own growing dynamic (fig. 3). With the last novel *Le Docteur Pascal* finally a last genealogical tree – completed on Pascal's deathbed – was published in 1893 (fig. 4).

When the novel refers to the relationship between the files and the genealogical tree – „cet Arbre généalogique [...] dont les volumineux dossiers n'étaient que le commentaire"[32] – the notion of the commentary brings into view the genealogical tree as a sacred text or a legal text requiring indefinite interpretation to make it meaningful and keep it alive. The genealogical tree functions to authenticate the scientific character of the novels and to exercise authorial control over them. Moreover, the scientific technology involved in the genealogical tree as a medium refers not merely to the function of representing knowledge, but also to that of generating it. That the temporal dimension of genealogy and heredity is translated into a spatial arrangement in the genealogical tree renders it possible to visualize the relations between the living and those long deceased, thus giving postulated heredity a visual structure. Changes in the succession of generations, that are not free from the coincidental, are captured and codified by the coherence and regularity of the genealogical tree. Its representativeness, moreover, implies a mode of thinking and a recording in terms of hierarchical deductions: The trunk explains the branches that in turn explain the leaves,[33] so that the complexity-enhancing possibilities of the tree's ramified crown remain retraceable back down to the tree's roots within the framework of heredity theory.

As a schematic representation of genealogy and lineage, the genealogical tree has narrative functions, too, even though it differs from the family bible or chronicle that are closer to narration, as Mann's *Buddenbrooks* illustrates. In Pascal's genealogical tree, entries are reduced to names, dates and heredity-diagnostic keywords – a compression of knowledge in written and diagrammatic form that has to be unfolded through narrative to be made meaningful, productive and transmittable. Docteur Pascal's „testament scientifique"[34], in which he verbalizes the findings of his research (in the presence of his colleague) on his deathbed, is beyond recovery for science, because only the genealogical tree itself has survived, and not Pascal's spoken commentary or his files.

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30 Ibid., p. 1008.
31 Zola (1961), p. 799.
32 Zola (1967a), p. 929.
33 „Le tronc explique les branches qui expliquent les feuilles" (Ibid., p. 1019).
34 Ibid., p. 1178.

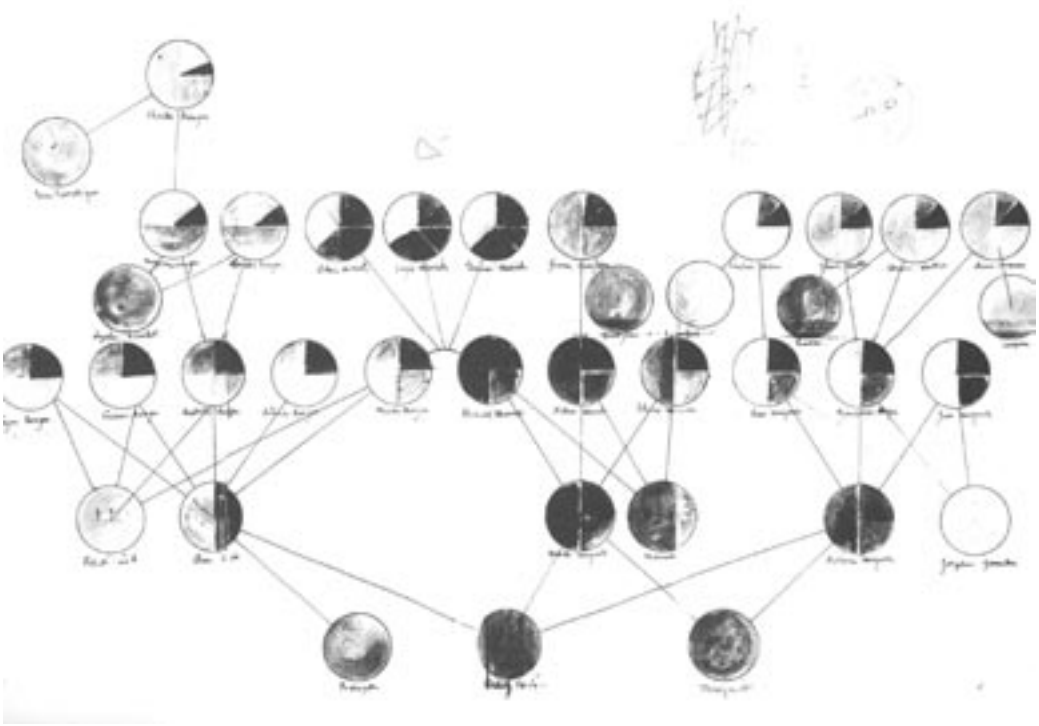

Fig. 1: Les Manuscripts et les dessins de Zola. Vol. 1: L'Invention des lieux. Edited by Olivier Lumbroso. Paris 2002, p. 537

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Fig. 2: Ibid., p. 533.

Fig. 3: Ibid., p. 535.

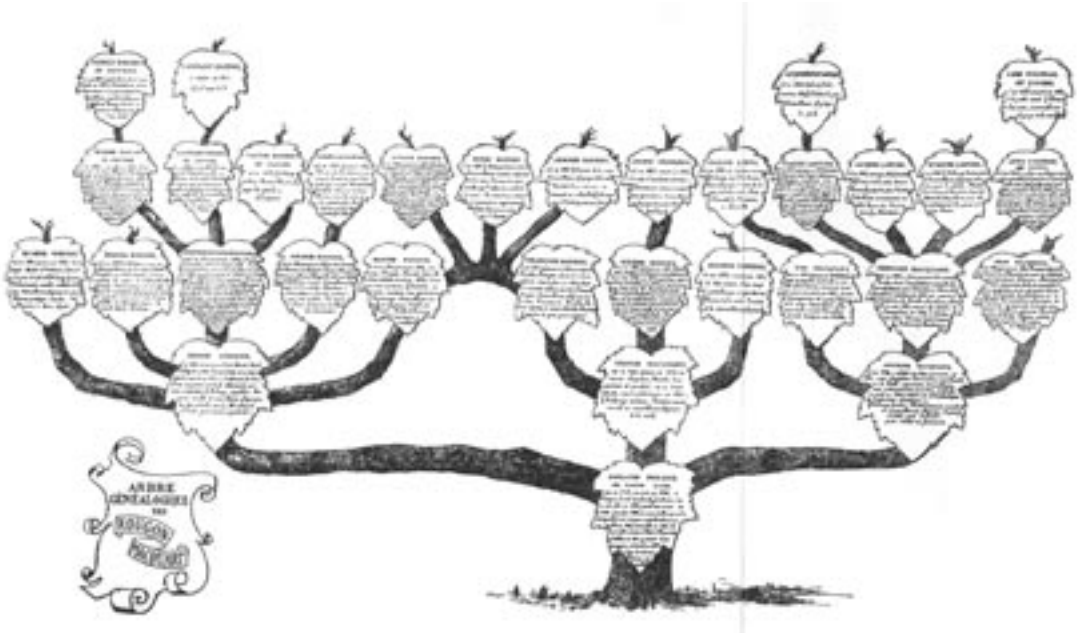

Fig. 4: Zola (1967a), between p. 912 and 913.

The genealogical tree, however, has gaps, too, despite Pascal's efforts to attain its perfect beauty and completeness: „pas un trou".[35] The gaps are of a systematic kind. They include positing Adelaïde as the great mother, without recording her ancestors – and their illnesses – however. As is generally known, every origin, including that of a *histoire naturelle*, results from cultural norming, as the 'root' of the genealogical tree makes particularly clear; here, all hereditary impairments originate in Adelaïde's „névrose originelle" that – of all dates – dates back to the revolutionary years around 1790. Another gap opens up when Pascal is able to enter his own position in the tree, but does not know how to locate the path of succession, and is hence unable to classify himself as a heredity case.[36] Just as little is he able to already classify the descendants of the youngest generation, which impairs both the explanatory force of the genealogical tree in scientific terms and its aesthetic guise: „Le pis était, pour la beauté de son Arbre, que ces gamins et ces gamines étaient si petits encore, qu'il ne pouvait les classer."[37]

Pascal updates his genealogical tree to his last breath: When he enters his date of death as he is dying, recording overtakes reality – and it is precisely reality that recording brings into being because the strain that writing involves accelerates Pascal's death. He also enters the birth of his child for the coming year, although Clotilde is only two months pregnant and her bereavement poses a threat to her pregnancy. Yet she does give birth – not only because she receives the deceased's last letter in which he summons her home, but also because the child has already been entered in the „arbre généalogique". A „genealogical imperative" emanates from the genealogical tree – an imperative that conjoins the linear narration in the epic novel with the idea of ancestors and descendants and with thinking in terms of origins and processes[38] and that requires a future that is not void: a narrating, in other words, that glosses over the „fêlure héréditaire", the fatal void, and attempts to suspend it. The pathos of the novel's ending ensues from this function in stylizing the child as the forthcoming hero and Messiah. At the same time, this stylization alludes to the notion of immortality. The genealogical tree should not just be a tree of knowledge – in its two meanings as knowledge and as sexual encounter as in the *mise en abyme*-scene featuring Pascal und Clotilde – , but also that other tree, the one located in paradise that has remained forever beyond human reach (cf. Genesis 3, 22): the tree of life.

And thus the unborn child is entered as „l'enfant inconnu" and becomes a new space in the genealogical tree to be written on, a placeholder of the future: it is entered in order not to have to accept the end of the family, nor the termination of writing at the close of the last novel; its entry, in other words, occurs in order to suspend death. In dealing with death, the files are closed, the last date of death is entered in the genealogical tree, and the chronicle is ruled off. In the novel, however, death is deferred because the debris yields narratives, and because the letters of the dead still reach their addressees, and because the wills that have been drawn up only begin to take effect gradually.

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35 Ibid., p. 1006f.
36 „Pourquoi, mon Dieu! l'Arbre ne voulait-il pas lui répondre, lui dire de quel ancêtre il tenait, pour qu'il inscrivît son cas, sur sa feuille à lui" (Ibid., p. 1034).
37 Ibid., p. 1018f.
38 Cf. Tobin (1978).

III. Vista

By way of a brief vista, I would like to turn to Thomas Mann's novel *Buddenbrooks. Verfall einer Familie* (1901). In the face of Hanno Buddenbrook's death, the last descendant of a wealthy mercantile family of Lübeck, the belief in immortality is evoked on the last page of this novel, too. Here, however, this is delegated to Sesemi Weichbrodt, a character delineated with unmistakable irony. The „Decline of a family" is reported from a narrative distance, quite unlike the high tones in which *Le Docteur Pascal* is narrated. The narrative distance in Mann's novel, which deprives the demise of the Buddenbrooks of all hope for a new beginning, takes effect on how the family's decline unfolds over four generations. Whereas Zola is intent on establishing the causal nature of the illness, Mann makes no reference to any clearly diagnosable hereditary impairments. Instead, it is rather a decline that is psychologically motivated and delineated in social terms that neither bookkeeping tricks nor legally determined succession can delay: „die Psychologie ermüdenden Lebens".[39] Following the end of Zola's large-scale naturalist project of reproducing reality in a „roman scientifique", Thomas Mann places emphasis on a culturally determined, multicausal history of decline, whose ironic depiction allows for distance to the characters, their afflictions and their self-deception through pre-formed discourses.

Mann also subjects the family chronicle to this procedure. The chronicle represents the family's genealogical self-insurance: a representative, gilt-edged booklet in which Jean Buddenbrook makes a verbose entry of the birth of his fourth child. In doing so, „kaufmännische Schnörkel"[40] („mercantilistic curlicues") and sentimental sentences blend with the god-fearing lines over and over. As he turns back the pages, Jean Buddenbrook reads previous entries about himself with „damp eyes" („mit feuchten Augen").[41] The emotion which grips him as he reads these entries time and again is an expression of a circular sentimental preoccupation with himself, a stagnating self-referentiality, which is in turn a symptom of the Buddenbrook's increasing incapacitation and morbidness. Since the booklet is organised along chronological lines and is hard-backed, no stimulating recombinations are possible – unlike with Pascal's files and their collection of loose sheets. There is no reference system other than the lapse of time; the genealogy remains codified in its media format, and is only readable in its linearity. Lacking any possibility of being reordered, its end is inevitable, however; thus the final stroke that Hanno dreamfully draws under the chronicle as a child proves to be a clear-sighted forecast, even if his father slaps him the face for this show of disrespect.

It might come as a surprise that the media that document and convey reflection on heredity and decline in Zola and Mann – files, genealogical tree and chronicle – are all old writing media. Newer technical media, which would be imaginable too, such as photography, are absent. Which has nothing to do with the authors being out of touch with modernity, but rather casts light on the fact that the assertion of a family and hereditary association is a discursive-symbolic construction. The nature of visual media to furnish evidence, such as photography (employed around 1900 to perform facial anthropometry), proves its value only in an argumentative context – given that it is not merely a question of stating the proliferation of resemblance, which does not

39 Mann (1981-86), p. 10.
40 Mann (1960), p. 53.
41 Ibid., p. 56.

necessarily result in a kinship order. Both novels put on view the power of this discursive construction, without which family heredity and decline would be inconceivable: Zola´s does so in a programmatic and identificatory manner, whereas Mann's is derisive and distanced.

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Heritage – Appropriation – Interpretation: The Debate on the Schiller Legacy in 1905

Stefan Willer

This new year sees a jubilee of one of Germany's most renowned poets and playwrights: Friedrich Schiller, who died 200 years ago, in May 1805. In the first weeks of 2005, there was already much ado about Schiller in German newspapers; there is going to be a significant increase of Schiller stagings in German theaters; German publishers have started new editions of Schiller's works; in March 2005, the Berlin Academy of Arts will arrange a 24 hours-reading of Schiller's works with chancellor Schröder as first lecturer; and so on and so forth. Much of this boom reminds of the celebrations one-hundred years ago, when in 1905 Schiller was commemorated for his 100th obit. That remembrance gave rise to large-scale discussions on the justification of claiming Schiller for various cultural and political interests – discussions which were centred on the notions of cultural property and inheritance.

When I, some months ago, proposed to Staffan Müller-Wille and Hans-Jörg Rheinberger to do a paper on this Schiller debate, I mainly thought of it as of a historical matter. Strangely enough, the actuality of this jubilee only occured to me several weeks after I had written down my proposal. The reason for my denseness probably lies in the urgency and the emphasis of the 1905 debate: a kind of articulating the desire for cultural property which seemed to me fairly outdated. I know better now. To name but one example: Two weeks ago, German journalist Burkhard Müller strongly emphasized Schiller being part of our inheritance. In an article in the *Süddeutsche Zeitung* entitled "Schiller and the Future", he argues that Schiller's quest for the 'ends' of History in his famous lecture *Was heißt und zu welchem Ende studiert man Universalgeschichte?* (What is, and to what end do we study, Universal History?) still has an afterlife, an aftermath, that it is thus a legacy and heritage. In his article Müller says explicitly that Schiller bequeathed us this question: "Diese Frage hat uns Schiller vererbt"; and he also asks what it means to be Schiller's heir, "[wenn] wir ihn heute beerben."[1]

It would not have been likely to find formulations like these in a West-German newspaper ten or fifteen years ago. In Germany, 'Erbe' as a concept of cultural tradition, in the decades before 1989, was mainly regarded as a domain of Marxist aesthetics and, above all, of the official cultural and educational policy and bureaucracy of the GDR in its proclaimings of the progressive, humanistic, classical and socialist heritage.[2] The more these socialist traces of the concept of 'Kulturerbe' fall into oblivion, the easier it becomes in German discourses to connect to the semantics of cultural heritage which is well-established in English as well as in French speaking countries – *patrimoine culturel* – and hence in the globalized canon of cultural values, the 'world cultural heritage', or simply 'world heritage'.

But this global concept is far from being self-evident. Its history goes back to the 19th century in which the 'invention of tradition' played a decisive role in the self-conception of nations as

1 Müller (2004), 15.
2 Cf. Dautel (1980); Willer (2005).

ontological entities.[3] This political impact of tradition has to do with a monumentalization of culture which is characteristic of the 19th century. This is not only true for sites, buildings, and museums, but also for literary culture with its rather vague material status. The great critical editions of literary lifeworks were often regarded as national affairs and had by 1900 established themselves as monuments of cultural legacy. As for the 'Weimarer Klassik' in Germany, it worked as an example for this kind of monumentalization. A wide range of Goethe and Schiller editions were available, the literary estate of both writers was stored in the 'Goethe and Schiller Archive' in Weimar since the 1880s. In the Schiller celebrations and discussions of 1905, you can see that the concept of cultural heritage and inheritance is ubiquitous – testifying that in the course of the 19th century this idea had become more and more prominent.

The aim of my paper is therefore to substantiate the importance of the history of cultural heritage in the context of "A Cultural History of Heredity". Concepts of handing down knowledge and art within a society are closely linked to biological theories about the transmission of traits and, especially, to juridical and economical regulations concerning the transfer of properties. I will concentrate on this latter aspect in the following considerations as it displays the contested and controversial status of heritage. Official jubilees and celebrations of cultural tradition generate a scene on which these controversies can be staged. Speaking of a 'scene': Maybe the playwright Friedrich Schiller was such an important figure for the German cultural heritage of the later 19th century precisely because of his singular ability to create theatrical conflicts which were both rhetorically and philosophically powerful. It was Schiller's theatrical pathos along with the main subject of his poetry – individual freedom confronted with tyranny or social restraints – that established and renewed his importance for what was claimed by opposite parties as their own tradition.

More generally, the importance ascribed to the particular poet Friedrich Schiller shows to which extent cultural tradition – and creation of cultural value – are based on the instance of 'the author'. The modern copyright as conceived in the late 18th century sees the author not only as the producer of artefacts, but also as the person owning the intellectual property of his work. At the same time – around 1800 –, reformed codes of law like the *Code civil* or the *Allgemeines Preußisches Landrecht* granted proprietary rights on a large scale, which rendered necessary, among others, a new configuration of laws of succession, the case of succession being a specific form of transferring and acquiring property in which economy immediately touches the lives of the juridical subjects. The case of succession means death and change of proprietorship at the same time. This is also true for the author subject in his status as originator and owner of his works. If property is ascribed to such things as poems and dramas – but also to letters and diaries, as soon as they were composed by an author in the modern, emphatic meaning of the word –, then, of course, the *death of the author*[4] gains importance for the question of his intellectual property.

But what exactly happens when the intellectual proprietor deceases? One of the main questions debated in the beginning of the 19th century is: if, how and when his proprietary right is transferred to the general public. For there seems to be a consent that there is no possibility for the author's descendants to inherit this proprietary right. In 1819 a copyright commission of the

[3] Cf. Hobsbawm and Ranger (1983).
[4] Cf. the 'classical' post-structuralist papers by Barthes and Foucault: Barthes (1967); Foucault (1969).

German Federal Assembly ('Bundesversammlung') formulates that the descendants of an author "cannot inherit the spirit by which a work is produced and by which alone it can be perfected, following its own peculiarity" ("[dass] sie den Geist, aus welchem ein Werk hervorgegangen und durch welchen es nach seiner Eigentümlichkeit allein vervollkommnet werden kann, nicht erben können")[5] – at which it is significant that in German *peculiarity*, 'Eigentümlichkeit', and *property*, 'Eigentum', are etymologically and conceptually very close to one another.[6]

Nevertheless there are considerable objections to transfer this 'Eigentümlichkeit' of an author into public 'Eigentum' as soon as he is dead. For among the characteristics of the modern author there is also the need of doing business with his works. This is where the publishers come into play. They participate in the intellectual property on a contract basis, and they want to safeguard their proprietary rights – especially the protection against reprints – as long as possible. Speaking of Friedrich Schiller, there was a relevant litigation referring to this very problem of publisher's copyright and public interest in the 1850s. Finally the publisher Cotta managed to have his print privilege prolonged so that just in 1859, the year of Schiller's 100th *birthday*, it was impossible to do free reprints of his works. This was all the more important as the public interest in 1859 was already substantial, Schiller becoming a kind of national hero and the Schiller celebrations having a considerable symbolic impact on the German nation building which was to be put into practice with the 'Reichsgründung' of 1871. However, the publisher's intervention shows that concrete economical interests could decisively interfere in the appropriation of an author and his works by the general public; moreover, it puts into question whether the author, even in his living years, can really be regarded as the owner of his works.

On this background, some verses of Goethe, related to Schiller's posthumous fame, seem rather idealistic and a little too optimistic: "Schon längst verbreitet sich's in ganze Scharen, / Das Eigenste, was ihm allein gehört." (Long since, his most personal property is being spread out in crowds, resp. into the crowd.) Goethe himself plays a specific role in inheriting Schiller's property. Not only did he have a strange affinity to the skull of his late friend which he had exhumed to keep it inside his own house some years after Schiller's death,[7] he also tried (in vain) to complete Schiller's unfinished last play *Demetrius*, and he wrote a lengthy *Epilogue* to Schiller's famous (and likewise lengthy) poem *The Bell*, from which the last quotation was taken.[8] All of these symbolic actions were part of a technique of mourning[9] whose aim was, as Goethe himself puts it in a letter to Cotta from June 1805, to represent not what was lost but what remained: "ich werde in diesem Sinne weniger das, was wir verloren haben, als das, was uns übrigbleibt, darzustellen versuchen." (*MA* 6.1, 903) For Goethe, Schiller's remains, or remainder, were obviously not the dead poet's eternal belongings, but that which could be appropriated. In Goethe's *Epilogue*, this appropriation is even backdated into Schiller's living years: for several times, the sentence "Denn er war unser" (For he was ours), is repeated as a kind of refrain throughout Goethe's stanzas.

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5 Quoted from: Bosse (1981), 127-128.
6 Cf. Plumpe (1979).
7 Cf. Schöne (2002); for further details and connexions regarding the history of cranioscopy and brain research cf. Hagner (2004), 70-78.
8 Goethe (1985-1998), vol. 11.1.1., 297-300, 300. Cf. the poem's first version, vol. 6.1, 90-92. (Subsequent references to the *Münchner Ausgabe* are cited in the text using the abbreviation *MA* with volume and page number.)
9 Cf. Horn (1998), 107-129.

Some months after Schiller's death, his poem *The Bell* and Goethe's *Epilogue* were staged for a solemn commemoration which was repeated several times in the following years in Weimar. Goethe promoted these celebrations and repeatedly added new verses and stanzas to his *Epilogue*. This was the beginning of the bourgeois Schiller remembrance which culminated in the jubilees of 1859 and 1905. The significance of this remembrance for a cultural history of heredity of the 19th century lies in the fact that Schiller not only seemed so likely to be appropriated – by surviving contemporaries as well as by posthumous followers –, but that at the same time his life, works and death altogether were interpreted as a last will and testament for posterity as a cultural community. To quote once again Goethe's *Epilogue* – which by the way was one of the most-quoted texts in the 1905 Schiller celebrations –, it is the poet's fatherland that has to execute his sacred last will: "Oh! möge doch den heil'gen, letzten Willen / Das Vaterland vernehmen und erfüllen." (*MA* 6.1, 92) And, according to Goethe, it is especially in the act of celebrating that posterity can somehow effect a balance of values: it reimburses, as it were, the cultural heritage it received by paying tribute to the dead poet. "So feyert ihn! Denn was dem Mann das Leben / Nur halb ertheilt, soll ganz die Nachwelt geben." (What life only gave to him by halves, posterity shall give in full.) (*MA* 11.1.1, 299)

Most of the official speakers in the dozens of 'Schillerfeiern' celebrated in 1905 in many German cities, universities, schools and clubs, see their task in elucidating "in which sense Schiller has to be regarded as a national poet."[10] This formulation comes from the speech of Eugen Kühnemann, a philologist who was at that time director of the Royal Academy in Posen/Poznan (a city in a region which was Polish up to the end of the 18th century and then again after the first World War). The print version of the speech says that the return on sales of the booklet is "to be applied to the erection of a Schiller bust in Posen" – intended as a visible sign for the "spiritual unity" of the "greater Germany" which Kühnemann conjures explicitly at the "eastern boundaries of the fatherland", as he puts it.[11] More drastically, one professor Max Hecht promotes "The Idea of a Schiller Memorial in Königsberg" in Eastern Prussia which in his vision would work as an outpost against Slavian barbarism and a bullwark against the "assailing Polishness".[12] In spite of this aggressive way of defending national possessions, Kühnemann in his Posen speech declares his fellow citizens the "glücklichen Erben" (lucky heirs) of the national idea of the 19th century for which Schiller is seen as a forerunner.[13] Indeed, the interpretation of the present as a matter of inheritance is at the core of the national-conservative claim of Schiller's legacy. Many of the speakers see quite clearly that the historical Schiller could not at all be interpreted as a nationalist author; but they affirm that he ought to be read like this *today*. Kühnemann says, "It is the essence of the great figures of intellectual history that they are appropriated anew, time and again, and that every age has its own vision of them."[14]

'Appropriation' is a plainly programmatic concept that is not only valid in the nationalist context. In appropriating Schiller, the issue is neither the proprietary right of the author (so that you would have to respect his own intentions), nor the perpetuation of unalienable and

[10] Kühnemann (1905), 25.
[11] Ibd., 5.
[12] Hecht (1905).
[13] Kühnemann (1905), 25.
[14] Ibd., 6.

unchangeable cultural values, but a kind of actualization in which these values can be converted and thus valorized anew. It is this very conversion which was understood as an accumulation of cultural capital. To put it in the words of Erich Schmidt, renowned German philologist, in his Schiller speech at the Berlin University: The Germans of the beginning 20th century shall follow Schiller, but not as "satte Erben" (saturated heirs), but as "Mehrer des Reichs" – a rather strange formulation, meaning literally 'those who augment the Empire', but obviously understanding 'Reich' also in the sense of 'Reichtum' and thus speaking of an augmentation of cultural wealth and value.[15] This kind of actualization is the concern of many speakers, as can be seen in titles like *Schiller und die Deutschen der Gegenwart* (Kühnemann), *Schiller und die Gegenwart* (Schiller and the Present),[16] *Schiller und die neue Generation* (Schiller and the New Generation),[17] *Schillers Bedeutung für das Maschinen-Zeitalter* (The Meaning of Schiller in the Age of Machines).[18]

This kind of actualization and appropriation is essentially related to the case of succession, thus, to the death of the author, which in these speeches works as a pathos formula for the vitality of his afterlife. Kühnemann says, "He bequeathed us his own life as an exquisite national possession"; and he emphasizes the meaning of the fact that it is not a birthday but an obit which is celebrated: "Whenever the nation celebrates the anniversaries – birthdays and obits – of its great men, it manifests a feeling of duty to take an interest in them just like in the next of our kin. But we do not think of their infirmity and mortality, but of their work. And in that sense celebrating an obit is even more meaningful than celebrating a birthday. For it is not important to us that the man existed but what he achieved for us."[19] So, in spite of Schiller's 'idealism' often claimed by conservative speakers as a monument against contemporary materialism, the concept of inheritance ultimately effects a materialistic interpretation of intellectual history – that is, it uses the capitalistic logic of 'achieving' a performance by making it convertible into cultural value.

It seems obvious that the political Left was highly suspicious about any conservative claim of Friedrich Schiller. In fact there was a large debate on the Schiller legacy among socialist and social democratic writers and theorists as well. This was not only a defiance to a national-conservative image of Schiller, but also a response to the inner-socialist discussion on 'revisionism', meaning the contested influence of bourgeois thinking on socialism. Falling back on bourgeois cultural traditions had been, ever since the beginning of the socialist movement, a nuisance and a necessity at the same time. It was especially the bourgeois pathos of liberty, with Schiller being the most illustrious protagonist in German literature, that had a great impact on agitational speakers like Ferdinand Lassalle, whose lectures to proletarian audiences, given in the 1860s, sometimes took several hours – and could therefore use a great deal of pathos.

For Marx and Engels, on the other hand, Schiller's pathos was not to be separated from its bourgeois interpretations in the later 19th century, either as a means of escaping reality or as an encouragement of nationalism. So it was the celebrations of 1859 that had the most important impact on Marx's and Engels's idea of Schiller – who appears to be one example of "tradition weighing on the brains of the living like a nightmare", as Marx put it in his famous essay *The 18th*

15 Schmidt (1905), 6-7.
16 Windelband (1905).
17 Fulda (1905).
18 Kammerer (1905).
19 Kühnemann (1905), 10, 7.

Brumaire of Louis Bonaparte.[20] Still Marx and Engels were far from condemning bourgeois tradition as such, but they drew a distinction which proved efficient, namely, a dualism of bourgeois utopism on the one hand, and bourgeois ideology on the other. In Friedrich Engels's words from 1872, the task was "to conserve what is really worth-while being conserved of the knowledge – science, art, social manners etc. – handed down by history, but not only to conserve it, but to turn it from a monopoly of the ruling class into a common good of the entire society."[21] To be really sure what is worth-while being conserved, one has to draw the distinction in every single case of cultural tradition. So the important thing here is the value judgement. It is obvious that even here the connection between handing down and evaluating is a matter of inheritance. In one of his late writings from 1888, Engels explicitly uses this notion when he says that the German proletarian movement is "heir to classical German philosophy".[22]

By the first decade of the 20th century, the concept of inheritance was already well-established among socialist writers. It not only worked to distinguish wanted from unwanted cultural relics but also to designate the 'bequest' of Marx and Engels, the socialist 'classics', as they were soon called. So an important figure of that trend could be characterized by a fellow combatant as the "executor of Marx's and Engels's legacy" just because he intended to "bind the German working class to classical poetry, to Lessing, Schiller and Goethe, with untearable ties".[23] This is what Rosa Luxemburg wrote about the left wing social democrat Franz Mehring who was one of the protagonists in the Schiller debate of 1905.

So it is a definitely bourgeois notion of property transfer and accumulation of wealth that is used as an affirmative concept of marxist cultural theory. Nevertheless, the socialist and social-democratic contributions of 1905 aimed at bourgeois Schiller remembrance in a very critical way. A social-democratic newspaper speaks of a "Schillerkult",[24] thus referring to the solemnity of most of the celebrations, but also to the industry of Schiller monuments and busts – plus kitsch objects for domestic use like brooches, needles and drinking-glasses. More basically, the cultural technique of celebrating as such is made dubious. The writer and politician Kurt Eisner points at what he calls "Schiller-Baalsdienst" (Schiller idolatry) with a pun: bourgeois Germany, he says, "feiert Schiller, um von Schiller zu feiern."[25] This pun uses the double meaning of 'feiern' in German which means 'celebrating' as well as 'resting from work'. So, what Eisner says is that the bourgeoisie celebrates Schiller in order to rest from the effort of really understanding him, whereas the way of the working class should be working on, or, working up Schiller.

Franz Mehring's writings played an important part in popularizing this idea. In 1905 he published several articles on Schiller and, above all, his book *Schiller. Ein Lebensbild für deutsche Arbeiter*. In this book – a 150 page survey of Schiller's life and works – Mehring declares his own partiality from the first lines on. He says that he is attempting a reading of Schiller apt to the class struggle of the present, and that he is about to picture Schiller "von der sicheren Warte" (from the certain standpoint) of the working class.[26] Of course, as a marxist, Mehring does not think that

[20] Marx [1852] (1969), vol. 8, 115.
[21] Engels [1872] (1969), vol. 18, 221.
[22] Engels [1888], vol. 21, 307.
[23] Luxemburg (1984), 104.
[24] [N.N.](1905), 97.
[25] Eisner (1905), 24.

this partiality is in any contradiction to historical objectivity, but that it is the only possibility to analyze and come to terms with historical contradictions as such. In the case of Schiller, Mehring argues that his famous idealism is not in contradiction to reality – as his bourgeois followers claim –, but that it indicates contradictions within reality. According to Mehring, the actualization of Schiller for the class struggle must be based on the finding that Schiller's actual solutions, namely the invention of an 'aesthetic state' and the concept of 'play' to overcome the contrarieties of reality, are false, but that the highness of his conviction ("Hoheit der Gesinnung"[27]) is right. In this very distinction – or, in this 'distinguishability', if the word may be allowed – lies the marxist concept of cultural inheritance.

In the end of his "Lebensbild"-book, Mehring summarizes that the working class cannot regard Schiller as an infallible teacher or forerunner, but still has an inviolable right to claim his bequest, and will always pay honour to it: "was ihr [der Arbeiterklasse] von seinem Erbe gebührt, das hält sie in unantastbaren Ehren."[28] In this sentence, the distinction between the 'inheritable' and the 'non-inheritable' is confirmed: you only have to pay tribute to what is due to you, 'was einem gebührt'. In his short article "Schiller und die Arbeiter", written also in 1905, Mehring puts even more clearly that "the modern working class accepts Schiller's inheritance only with critical reservation" by separating "what in him is still alive and what has died off".[29]

Thus an objection is raised against the concept of "Nachruhm", posthumous fame, which Mehring himself takes into account in his Schiller-book. When he comes to the end of his "Lebensbild", he uses heroic words on dying and afterlife not at all dissimilar to the corresponding passages in the official and semi-official 'Festreden': "While his body crumbled into dust, his great name lived on." And Schiller is quoted with two verses articulating that earthly life vanishes, while the dead last forever: "Denn das irdische Leben flieht, / Und die Toten dauern immer."[30] From a socialist point of view this concept of afterlife had to be judged as insufficient or even false. Since Marx's early writings, the socialist movement was definitely sceptical about any combinaton of historical memory on the one hand and eternity on the other. In his already cited *18th Brumaire of Louis Bonaparte* Marx had suggested to do away will all kinds of what he called "welthistorische Totenbeschwörungen" (historical conjurations of the dead).[31]

So, the internal separation of Schiller's bequest into a living and a dead part was essential. It is only in this sense, Mehring continues in his article on "Schiller und die Arbeiter", that modern workers may utter Goethe's phrase, "For he was ours."[32] And Mehring adds two more famous lines by Goethe, Faust's often quoted maxim on inheritance: "Was du ererbt von deinen Vätern hast, / Erwirb es, um es zu besitzen." (To really possess what you have inherited from your fathers, you have to earn it.) (*MA* 6.1, 553) This again means appropriation – but in a specific way. Mehring's use of this concept is not so much based on the desire of having 'the complete Schiller' at his disposal, but rather on a selection, which also means: a fragmentation of this cultural

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26 Mehring [1905a] (1961), Vol. 10, 91.
27 Ibd., 241.
28 Ibd., 240.
29 Mehring [1905b] (1961), Vol. 10., 280.
30 Mehring [1905a] (1961), 235. Cf. Schiller (1958), 426.
31 Marx [1852] (1969), 115.
32 Mehring [1905b] (1961), 280.

tradition. Speaking in terms of inheritance: Following Mehring, one does not have to decide whether to accept the inheritance or to waive it altogether, but one can decide which parts of it are acceptable. As a juridical practice, this might seem somewhat arbitrary, and it was not at all undisputed as an aesthetic practice. Marxist aesthetics since the 1930s made strong efforts to conceptualize appropriation in a more dialectical way, without simply recurring to political actualization.[33]

For Mehring, anyway, the internal separation of the heritage produced a distance between the testator and the heir which was crucial for the heir's self-legitimation. And it was also for reasons of self-legitimation that the socialist concept of cultural inheritance was at the same time an attempt to disinherit those that had been in possession before. As the journalist Karl Korn put it, the working class was to "enter upon the inheritance of the intellectual heroes that was forfeited by the bourgeoisie."[34] The bourgeoisie of course struck back. Several Schiller speeches of 1905 emphasize that Schiller will never be the hero of the proletariat but will always remain the poet of the middle classes.

With a hundred years of historical distance, this contention can be seen as a negotiation of cultural values at a wider range. Moreover, it shows that these values have to be negotiated to be valorized at all. This idea is also displayed in contemporary economical and philosophical value theories. Georg Simmel's *Philosophy of Money* for instance (first published in 1900) is based on the perception that values have no causes within the things evaluated but that they are expressed and generated in comparison to other values. Values as such are downright relative; evaluation quite simply is relativity. In Simmel's words: "Only relativity generates the objects' value in the objective sense, because it is only in relativity that the things are kept in distance from the subject."[35] Speaking of the relativity of values in the context of cultural inheritance, I think it is not just a pun to refer to the double sense of relation. By relating yourself to Schiller, you became, as it were, Schiller's relative and thus were entitled to be his heir. In this perspective, even the most enthusiastic contributions to the debate ultimately do not refer to 'eternal' or 'immaterial' values, but they operate relatively and economically.

Relations of values need "Träger" (carriers), as Georg Simmel puts it. The main carrier substance he deals with is money, "the embodied relation of economic values."[36] But Simmel regards art as well as a "structure of comparing values."[37] When it is about cultural inheritance, one could say that this structure is generated by a comparison between the old and the new. The creation of new cultural values requires an estimation of how the difference between innovation on the one hand, and tradition, the old, the existing on the other will be evaluated at a certain point of time. Thus you can estimate why and how this difference is likely to be stored in the cultural memory.[38]

So the idea of converting and augmenting cultural values by appropriating texts written by the dead poet Friedrich Schiller reveals its economical logic. It is by relating oneself to elements of the

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33 Cf. Franz (2000), 190.
34 Korn (1907/08), 414.
35 Simmel [1900] (1989), 135.
36 Ibd., 130.
37 Ibd., 163.
38 Cf. Groys (1992), 47.

classical tradition that one's own contribution can be evaluated and valorized in relation to the existing one. All the more, in the threefold ratio of the act of inheritance – *someone* inherits *something* from *someone* – the active part is shifted to the heirs. But still the testator's perspective has to be taken into consideration. Even though appropriation is affirmed throughout this debate, none of the contributors would have wanted to play the part of the legacy hunter. This is where interpretations and ways of reading intervene. For of course it is in reading Schiller that he is made heritable for the various heirs claiming his legacy. So, which is the way of reading an author that is most likely to legitimate the reader as his heir?

Franz Mehring criticizes in his Schiller book that the national-conservative reception is characterized by deliberate misreadings, and especially: misquotings, for instance by using words of the sovereigns in Schiller's dramas to make statements about current problems of government. In the earlier socialist agitational rhetoric, on the other hand, classical quotations sometimes were even changed in their wording to make them applicable to the appropriator's political aims. However it is striking that in most of the contributions of 1905 the policy of quoting Schiller does not seek the plain evidence of substantial statements in favour of – or against – certain political, cultural or pedagogical programs, but that they rather look for hints concerning the business of appropriation itself. This is why, I think, Goethe's Schiller-*Epilogue* is so often quoted and why so many speakers recur to the Schiller celebrations of 1859 – as if the 1905 celebrations were a kind of frame in which preceding acts of remembrance could be observed. Among the Schiller quotations themselves, quite clearly those are preferred that deal with questions of death and remembrance, of posthumous fame and of aftermath. Eugen Kühnemann's already mentioned speech closes in an exemplary way by quoting a Schiller epigram that displays the difference between mere physical procreation of the species (the genus), which is said to be the business of "millions", and the tradition of humanity, which is only ensured by the happy few: "Millionen beschäftigen sich, daß die Gattung bestehe, / Aber durch wenige nur pflanzet die Menschheit sich fort."[39]

In conclusion: The meaning ascribed to inheritance in this German cultural-political debate of the early 20th century makes it evident that the history of cultural heritage plays an important role in a cultural history of heredity. The 'Schiller cult' of 1905 shows to which extent the culture of inheritance can be regarded as a way of negotiating with the dead. The interesting part of these Schiller commemorations is not that they confirm the fact that there were conflicts between conservative and socialist interpretations of the classical tradition. Beyond this divide I have tried to argue that every definition of cultural heritage is based on appropriation, because it is the very concept of property that permits the translation from juridical and economical to aesthetic transactions of bequest.

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[39] Schiller (1958), vol. 1, 303.

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From pedigree to database. Genealogy and Human Heredity in Germany, 1890–1914

Bernd Gausemeier

The 1911 Dresden Exhibition for Hygiene gave a considerable boost to the German eugenics movement. Only one year after its foundation, the German Society for Racial Hygiene was able to present itself in a highly successful show for the popularization of medical knowledge.[1] The section organized by the Society confronted visitors with an impressive array of illustrations showing the progress of the biological sciences – diagrams on hybridization experiments or on the mechanisms of cell division and fertilization. The eugenicists' preferred exhibit, however, were pedigrees visualizing the impact of heredity on human life – family trees showing the transmission of night blindness, musicality, tuberculosis or 'moral insanity'. The use of pedigrees was also a major topic in the international meeting of eugenicists connected with the exhibition. In a keynote talk, the genealogist Hans Breymann expressed that he wasn't completely happy with what he had seen in the showrooms.[2] With mild sarcasm, Breymann stated that the medical profession had led genealogical method into a state of babylonic confusion. While generations of genealogists had worked to standardize and to simplify the method of pedigree–keeping, medical practitioners were excessively creative in inventing new genealogical systems. This situation, Breymann argued, would not only make the collected material incompatible, it would also create a serious problem for the propagation of eugenics: methodological ignorance might repel the hobby genealogists who formed a considerable potential of aides for the case of eugenics. Bringing genealogical method to eugenicists and interesting genealogists in problems of heredity would, in his eyes, provide eugenics both with a solid scientific groundwork and with more social acceptance.

The most urgent common task for medical researchers and genealogists, according to Breymann, was the establishment of comprehensive collections for medical family research. He bemoaned that there were excessive public expenditures for "the cure and accomodation of degenerates", but no support for the scientific study of human heredity that would help to control the spread of mental diseases. Since state institutions showed more interest in "deep sea research and polar expeditions than for this most profound problem of humanity", he called for concerted action of genealogists and scientists.[3]

Breymann was neither confronting his audience with unheard ideas nor were his proposals as unpopular as he pretended. Among psychiatrists, there was a vivid discussion on the use of genealogical collections for the study of mental diseases. Around 1910, there were several schemes to reorganize and to standardize the family histories collected in asylums and for setting up regional surveys of 'burdened' families. The psychiatrist Robert Sommer suggested to complete such efforts with a national central office for psychiatric family research within the public health administration; other doyens of German psychiatry like Alois Alzheimer and Emil Kraepelin voiced similar ideas.[4]

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1 von Gruber/ Rüdin (1911); Weindling (1989), p. 230; Weingart et al (1992), p. 206.
2 Breymann (1912).
3 Ibid., p. 28.

The collection of family histories had been a part of psychiatric practice since the early 19th century, but it was not before the 1890s that German genealogists developed interests in this aspect of their occupation. After then, however, genealogical associations quickly built up contacts with the medical profession. This trend was spearheaded by the organization Breymann presided over, the *Zentralstelle für deutsche Personen– und Familiengeschichte* (German central office for family history). Its establishment in 1903 reflects the boom of family research in late 19th century Germany. Leading genealogists felt the need to coordinate the collecting activities of the countless clubs and societies. Shortly after ist foundation, the *Zentralstelle* sought to join forces with medical scientists. In 1908, its representatives reached an agreement with psychiatrists and eugenicists (among them the leader of the German Racial Hygiene movement, Alfred Ploetz) that assigned it as a central collecting point for genealogical material that might contribute to the understanding of "heredity, degeneration and regeneration".[5] This scheme exemplifies how dramatically the role of genealogy changed around this time. It was no longer regarded as an aristocratic pastime or as an auxiliary method for political history. In 1913, it was no longer unusual that a manual of genealogy contained contributions on psychiatric and anthropological applications and on the uses of family research in the social sciences.[6]

The change that is visible here did not only consist in an increasing use of genealogical method by medical researchers interested in hereditary transmission. The field of genealogy as a whole acquired a new meaning. In his *History of Sexuality*, Michel Foucault has devoted considerable attention to the redefinition of genealogy in the 18th and 19th century. While *ancien regime* genealogy represented the old descent and the strong alliances of a family, the pedigree of the bourgeoisie contained the ancestors' diseases and anomalies: "The care for the pedigree became the concern about heredity."[7] Foucault relates this change to an essential shift in the history of modern society: While "classic" genealogy was associated with a historical regime *(dispositif d'alliance)* under which the distribution of wealth and of social prestige depended on the politics of marriage, the new form corresponded to a regime *(dispositif de la sexualité)* defined by the values of individual health and the control of reproduction. Thus, the metamorphosis of genealogy reflects profound structural changes in family, kinship and society. The two regimes should be understood as overlapping rather than successive historical formations, and the same applies to the different forms of genealogy. Pedigrees were used to visualize hereditary phenomena already in the 18th century, but this was only a first step in a long process of structural transitions.

All notions about the transmission of physical features and diseases are necessarily based on genealogical knowledge. However, the rise of hereditarianist ideas in 18th and 19th century medicine was not necessarily associated with the use of pedigrees. Medical practioners usually treated the hereditary aspect as part of the nosographical description – in other words, the family history was an integral part of a narrative explaining the genesis of a disease.[8] Comprehensive collections of genealogical material emerged primarily in psychiatry. In the first half of the 19th century, asylums began to keep regular records about of their patients' familial background.[9] In

4 Sommer (1913), pp. 394f; Rüdin (1911), p. 571.
5 Zentralstelle (1908), pp. 106f.
6 Heydenreich (1913).
7 Foucault (1983), pp. 149f.
8 Nukaga (2003).

the same period, criminologists tried to reconstruct the ancestry of delinquents in order to understand the emergence of "moral degeneration".[10] Systematic surveys of alleged hereditary phenomena were closely connected to the rise of medical and penal institutions. Their genealogical collections allowed the definition and registration of menacing sub–populations, the insane and the criminal.

The establishment of the *Zentralstelle* represents a different quality of institutionalized genealogy. The genealogists pursueing this project wanted to incorporate the specific collections of asylums, prisons and hospitals, as well as aristocratic or bourgeois family archives, into a comprehensive genealogical network of the whole population. This idea implied the redefinition of genealogy from a private into a public affair. Moreover, the limits between 'normal' and 'abnormal' genealogies were to be dissolved. It seems obvious that these plans were essentially a consequence of the growing political influence of the eugenics movement. Yet, the redefinition of genealogy can be related to a whole complex of epistemic shifts in the human sciences.

"Scientific genealogy"

Only some years before the establishment of the *Zentralstelle*, German genealogists were virtually untouched by the discussions about the hereditary transmission of diseases or mental qualities. One of the first scholars to raise the topic in genealogical journals was the lawyer Stephan Kekulé von Stradonitz, son of the eminent chemist August Kekulé, and a co–founder of the *Zentralstelle*. But the most influential voice calling for a reorientation of the discipline was the historian Ottokar Lorenz (1832–1904), whose 'Manual of the entire scientific genealogy' (1898) made a lasting impact both on genealogists and scientists.

Lorenz saw his manual as a reaction to the revival of genealogy in large parts of German society – which was, according to his view, unfortunately only taking place in private associations, not in the historical departments of the universities. But it was not his main idea to support the reconstruction of aristocratic customs. The largest parts of his book dealt with new developments in hereditarianist medicine, psychiatry and psychology. Given the mass of material compiled in this field, this is hardly surprising, but Lorenz saw a much deeper need to discuss the biological aspects of family research. He held that the achievements of modern science demanded a complete redefinition of the scope and the methods of genealogy. Genealogy, Lorenz stated, had always been concerned with the phenomenon of human heredity, but "the high ... standard of present natural science ... allows a totally different degree of certainty and comprehension for genealogical research than it would have been possible in earlier times of human observation."[11] Lorenz was referring to the advances of cytology of the 1880s and 1890s that had brought about the chromosome theory of heredity. Even though the mechanisms of transmission were still obscure, the basic principles of sexual reproduction seemed to be firmly established: "Nothing today can be regarded as more secured by the exact ... investigation of the cell ... than the complete equivalence of the germ–plasms emanating from both sexually different individuals, and accordingly genealogy has, in its own domain, to regard and to evaluate the paternal and the

9 Jacobi (1834), p. 312.
10 Becker (2002), p. 342.
11 Lorenz (1898), p. 338

maternal lineage as the basic elements of all examinations of the individual as well as of family, kin, people and species."[12] What he regarded as the pivotal result of modern biology, thus, was the microscopic 'proof' for the biological equivalence of the sexes. Lorenz stressed this simple fact because he regarded it as a crucial problem that a majority of genealogists only considered the male lineages of a family. The first step towards a modern genealogy had to be the emancipation from the aristocratic idea of patrimony.

However, the appeal to the exact sciences had deeper implications. Lorenz made special reference to August Weismann, though he frankly admitted that he only knew the renowned zoologist's ideas from secondary sources. Apparently he only used Weismann's name as a synonym for experimental research on fertilization and cell division. Despite his own recurrent statement that genealogists should never meddle in unsettled scientific discussions, he situated himself in one of the most vivid biological debates. Weismann's theory of the continuity of the germ plasm, which was most influential in the biological discussion, met considerable resistance in the medical community. The outright rejection of an inheritance of aquired characteristics challenged the prevailing view on the nature of hereditary disease. Weismann's most eminent opponent Rudolf Virchow regarded the germ plasm doctrine as a symptom for a problematic development: since experimental biology had successfully emancipated itself from mother medicine, it had ceased to listen to the medical practitioner's experience.[13] Virchow's critique reflects that the study of heredity was, at the end of the 19th century, in a highly ambivalent situation. On the one hand, the mechanisms of heredity seemed to become a tangible object of exact scientific examination. For pathologists and psychiatrists, on the other hand, the term "heredity" simply stood for the occurence of certain maladies in a family. Distinguishing between "direct", "collateral" and "atavistic" forms of heredity, they were lacking a clear–cut concept to analyze the mode of transmission itself. Lorenz's program of "scientific genealogy" implied the promise that sophisticated genealogical methods would help to overcome this problem.

However, Lorenz was not primarily dealing with pathological, psychiatric and psychological studies because he wanted to provide scientists with a new understanding of heredity. He considered it necessary to interest historians and sociologists in this field because they usually neglected the impact of heredity on human life. Nevertheless, most of his references to hereditarianist literature were marked by an acerbic criticism. Lorenz especially accused psychiatrists of being too careless in postulating hereditary influences. In his view, this biased perspective corresponded to shortcomings in genealogical methodology: medical family studies were too often based on an inadequate use of *pedigrees*. Pedigrees represent the descendants of a single ancestor. Medical pedigrees departing from a sick person and tracing the spread of the disease in the following generations unavoidably suggested the multiplication of the evil seed – in other words, they produced a picture of increasing degeneration. Similarly, Lorenz warned to derive any conclusions about a hereditary burden from isolated cases of madness in the ancestry – after all, who would not find "some well developed fools" among his forefathers?[14] The adequate tool of a biological genealogy, Lorenz argued, was not the pedigree, but the ancestral chart

[12] Ibid., p. 347.
[13] Virchow (1886), p. 1.
[14] Lorenz (1898), p. 387.

(*Ahnentafel*), the genealogical system showing the complete ancestry of a living person. The ancestral chart was the proper form to represent the total "germ plasm" of a given individual. On this basis, it would be possible to survey the number of certain diseases in the ancestry and to assess if a hereditary influence could reasonably be assumed.

"Scientific genealogy", thus, was not primarily defined as a method to detect more hereditary diseases, but rather as a way to constrain the growing influence of "hereditarianist superstition" (*Vererbungsaberglauben*) in the public.[15] In the eyes of the conservative historian, the fear of degeneration was a modernist folly stirred up by fashionable authors like Zola and Ibsen. For Lorenz, the idea that mankind was riding a biological avalanche was a complement to the excessive belief in progress. Accordingly, he took a reserved attitude towards eugenics. His sceptical remarks show that it was clearly not his idea to boost the acceptance for genealogy by jumping on the eugenic bandwagon. On the contrary, he was repelled by radical ideas about "race improvement". He ridiculed psychiatrists who hoped "that the better organized society of the future will, counselled by psychiatry, generally bar burdened persons from marriage".[16]

However, psychiatry was by no means dominated by the careless hereditarianism Lorenz criticized. By the end of the 19th century, heredity was commonly regarded as an important factor in the genesis of mental diseases, but there was a widespread awareness that its influence could hardly ever be measured exactly. The place of heredity in etiology was rather challenged than strenghthened as the advances in bacteriology provided new insights about many diseases. Further, the fear of degeneration did not find unanimous support among psychiatrists. Robert Sommer was an influential voice in the medical discussion raising quite similar objections like Lorenz. He strongly condemned the "pessimistic world–view" of the "doctrine of *décadence*" that was often propagated by the misleading use of pedigrees.[17] Sommer also demanded a strict distinction between the terms *Heredität* and *Vererbung*. The occurence of similar symptoms in one family *(Heredität)* made it probable that hereditary transmission *(Vererbung)* was involved, but it could not be regarded as a proof in the proper sense of the word. This scepticism, however, did not restrain but rather reinforce the interest in human heredity. Both Lorenz and Sommer called for more methodic family research in psychiatric institutions. Sober work with these genealogical data would support the insight that the powers of heredity were not an inescapable fate but a controllable phenomenon.

But what could the genealogist actually contribute to the study of heredity? Lorenz did not propose a clear guideline how to interpret the collected material. He even doubted that heredity followed a definite, intelligible law. Yet, he claimed that proper genealogical method would enable scientists to demonstrate certain regularities of human heredity, particularly for what he considered to be its central question – the problem of latency or "atavism". The reappearance of hereditary traits after several generations seemed especially important to Lorenz because he regarded it as the main source for the modern "horror" of hereditary diseases. A comprehensible explanation why the forefather's maladies suddenly struck a family, he reasoned, would help to chase off the "ghosts" of heredity that haunted the bourgeois society.[18]

[15] Ibid., p. 438.
[16] Ibid., p. 437.
[17] Sommer (1901), p. 67.
[18] Lorenz (1898), p. 438.

In fact, Lorenz used the term atavism for all irregular forms of transmission. And the only case he discussed in detail was not even about the problem of latency, but about the constancy of a "family type". As a historian with profound knowledge about European dynasties, he chose the most prominent example of physical characteristics prevailing in royal families – the Habsburg dynasty and its protruding lip. Basically, Lorenz' approach did not differ from the ideas prevailing in medical family research. Every ancestral generation represented the total germ plasm of an examined person – for example, each grand–grandparent contributed 1/8 of the present generation's genetic material (this simple fractional arithmetic was, in fact, not in full accord with Weismann's ideas). Yet, Lorenz stressed a phenomenon that is especially striking in royal genealogies: due to marriages within the family, the actual number of ancestors in higher generations differs from the theoretical one. In other words, almost no ancestral chart (not only in the notoriously incestuous nobility) would display 16 or 32 different individuals in the fourth or fifth generation, but rather an increasing number of "multiple" antecedents. For Lorenz, this "loss of ancestors" was a crucial aspect that was usually disregarded in the study of human heredity. The Habsburg family was an ideal case to demonstrate how a certain trait (the lip) became a constant family characteristic through repeated intermarriage. Lorenz showed that striking cases of the lip appeared whenever marriages between Habsburg royals and women from side lineages of the dynasty occurred.[19] In so far, he did not primarily trace an hereditary defect, but rather used a physical trait to describe how a dynasty maintained its biological and social identity.

It was surely not only a result of monarchist sentiments that the Habsburg case has been discussed by several other scholars before and after Lorenz. Royal genealogies provided a favorable material for family studies: they could be followed over a long time span, there were excellent family records and, above all, portraits. Medical researchers dealing with a more common kind of people were aware that informations on a patient's ancestors were for the most part insecure. The crucial question for hereditarianists was how medical data of the living generations could be recorded in a standardized form. But what notions of heredity formed the conceptual basis for such collections?

Robert Sommer stated that organized family research was necessary in order to understand the "familial relations of mental diseases and their distribution in the whole country".[20] Still at a time when he had accepted Mendelism as the theoretical key to human heredity, his primary question was not how certain diseases were transmitted but how they could be classified. Psychiatrists were used to see the nervous diseases as complex system of various clinical patterns. Accordingly, Sommer hoped that exact statistics about the familial and regional distribution of disorders would provide new insights into their "family relations". Heredity, in this perspective, was rather a method than the object of investigation. Psychiatrists were usually not inclined to regard complex mental disorders as Mendelian units. The first scientist to follow this approach was the ardent racial hygienist Ernst Rüdin, who built up a large psychiatric family register at the German Research Institute for Psychiatry after World War I. Rüdin also introduced a genealogical method that differed distinctly from the ideas of Lorenz or Sommer. Instead of an exhaustive examination

19 Ibid., p. 407.
20 Sommer (1913), p. 394.

of selected family histories, Rüdin used broad samples of "short" genealogies (at most three generations) for the statistical calculation of Mendelian ratios.[21]

Such a statistical use of family research was clearly beyond Lorenz's scope. Generally, his approach to heredity was descripitve, not analytical. Despite his references to experimental biology, he did not really understand heredity as a phenomenon that might be explained by a single principle. In so far, he was a typical representative of late 19th century hereditarianism. In Théodule Ribots influential book *L'hérédité psychologique*, for example, heredity still appeared as closely linked to reproduction – i.e. the circumstances of procreation were discussed as a possible influence on the qualities of the offspring. Unlike Ribot, Lorenz was not inclined to render romantic ideas about the extraordinary talent of children begotten in extramarital love affairs.[22] Yet, he allusively accepted the predominance of the paternal germ plasm.[23] In fact, he had not completely dissociated himself from the patrilineal concept of heredity he pretended to refute. With reference to such inconsistencies Wilhelm Weinberg, a pioneer of Mendelian statistics, later criticized Lorenz' book as an overrated and insufficient contribution to the study of human heredity.[24] But this critique missed both the original aim and the actual impact of 'scientific genealogy'. Lorenz did not primarily define genealogy as a scientific tool to analyze human heredity, but as a way to understand human society and history.

History

Lorenz used pathological and psychiatric examples in order to demonstrate the problems and possibilities of studies about heredity. As an historian, he was more interested in the transmission of "normal" psychological qualities. Yet, even though he claimed it to be unquestionable that individual characters were for the most part shaped by hereditary factors, he was rather unsatisfied with the scientific treatment of this problem. When it came to the inheritance of mental qualities, he was even more sceptical in questions of hereditary diseases. He directed especially harsh critizism against the most influential author on this topic, Francis Galton. Galton's compilations of families with certain professional or intellectual preferences were, in his eyes, hardly a proof for the hereditary nature of 'talent', but a highly redundant affirmation of the well–known fact that a pear–tree doesn't yield apples.[25] Lorenz was especially unhappy with the superficial equation between talent and vocation: after all, the fact that a judge had a number of judges among his relatives gave little evidence if he was particularly good in his job. Lorenz's limited esteem for Galton does not reflect a distrust in his conclusions, but an aversion against purely statistical method. For Lorenz, understanding human heredity was inextricably linked with detailed genealogical case studies, which were absent in Galton's work. A convincing proof for the transmission of mental qualities was, in his eyes, a matter of meticulous historical critique. The excessively cited pedigree of the Bach family, for example, appeared to him as a questionable evidence for hereditary artistic genius since it represented a typical form of professional tradition.

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21 Rüdin (1911).
22 Ribot (1884), p. 175.
23 Lorenz (1898), p. 407.
24 Weinberg (1911).
25 Lorenz (1898), p. 425.

Lorenz considered it more fruitful to follow cases in which outstanding talent emerged from an unlikely social background.

Lorenz did not primarily conceive genealogy as a tool to demonstrate the preponderance of nature over nurture. The influences of education and social customs were an integral part of his concept of family research. He envisaged (royal) family studies combining political history and hereditary psychology into a form of historical characterology – a model study about medieval Saxon royalty was provided by his pupil Ernst Devrient.[26] Lorenz himself did not confine himself to the discussion of specific case studies. His program of "scientific genealogy" was about larger political issues.

Much more than about the ghosts of degeneration, Lorenz worried about a phantom that scared the majority of German politicians and academics: Socialism. He explicitly defined "scientific genealogy" as a weapon in the struggle against egalitarian ideas. The modern contempt for genealogy itself was, in his words, a typical effect of "the social–democratic doctrine which thinks to be able to disengage itself from the natural foundations of human existence". However, the "scientific spirit" of the present age and a growing "genealogical awareness" would repel these pernicious influences. The current revival of genealogy, Lorenz emphasized, was not about a return to an aristocratic class society, but would evoke a new consciousness that the specific qualities of families and individuals were the product of "genealogically developed characters." "Under this banner, scientific genealogy now fights the social doctrines like aristocracy fought democracy in former times."[27] Biological knowledge was to replace the old aristocratic pedigree–consciousness.

Lorenz did not only want to use his genealogical arms against Marxism. He first developed his ideas on genealogy in a 1891 book devoted to the celebrated historian Leopold von Ranke. With the master of Historism as his patron, he attacked all idealistic notions of social progress. Contemporary historiography with its preference for ideas and institutions, he complained, displayed an "insurmountable disgust for the marriage–bed and for births" and described historic events "as if they had happened on the moon".[28] Apparently it was time to bring "real life" and "real people" back into history. As human life was a biological process taking place in families, the old auxilliary disciplin of genealogy had to be considered as the starting point for all historical reflections: "Genealogy is the immediate certain fact in terms of natural history, the given element of historical events."[29]

But what concept of history resulted from this heraldic motto? Clearly, the genealogic turn implied a highly conservative perspective. Kekulé, usually in full accordance with Lorenz, explicitly referred to Treitschke's catchword "great men make history" – then, it was up to the genealogist to show how great families made great men.[30] Still, Lorenz offered a little more than the prospect of a biologized form of dynastic chronologies. His basic idea was that the succession of generations formed the true basis of all historic change. Thus, generations were the units determining the rhythm of history. Lorenz called it a "natural law" that a generation (in the male

[26] Devrient (1897).
[27] Lorenz (1898), p. 18.
[28] Lorenz (1891), p. 188.
[29] Ibid., p. 257.
[30] Kekulé (1900), p. 114.

lineage) comprised 33–35 years, and he claimed that this regularity explained the historical identity of a period. In a similar fashion like the influential philosopher Wilhelm Dilthey, he attributed the ideas and political aims of a generation to common juvenile experiences. Lorenz, inevitably, perceived generations primarily as generations of monarchs which formed the character of a period. His concept of generation, thus, was clearly rather a sociological than a biological one, nevertheless it served an anti–sociological objective. According to Lorenz, the generational perspective illustrated that "ideas do not possess men, but men possess ideas, and accordingly inherit them or reject the inherited ones."[31] Generational history was to emphasize the role of deliberately acting people and to reject all notions of a transcendental progress in history. But the concept of generation also implied that historicity was largely determined by biological conditions.

While Lorenz stressed that the coherence of generations was shaped by common memory, he attributed the constancy of national histories to biology. He described the existence of nations and classes as a result of inbreeding or, in his own special terminology, "loss of ancestors". Not only the aristocracy secluded itself by establishing rigid regulations for marriage, also rural societies and premodern urban groups were formed by restricted possibilities for mating. But there were also limits for generative seclusion. Lorenz stressed that every genealogist was aware that even royal family trees showed an increasing number of common elements after some generations. No period in (European) history knew a total, caste–like separation, "there has always been a complete mixture among people, which ascends and descends between the generations, like the waves in the sea."[32] Yet, he regarded the inhibition of connubial mixture as one of the basic laws of human existence: "There is a tendency rooted in human nature to reduce the number of ancestors. The law of attraction between the kindred and the coequal sometimes becomes abandoned ..., but on the whole it is ineradicable, since love prospers best with loss of ancestors and equality of birth (*Ebenbürtigkeit*)."[33] All social grouping, then, was the result of more or less conscious inbreeding. Whenever this law was violated and distinctions between classes and ethnic groups broke down, the inevitable result was decay and revolution. The insights of genealogy, Lorenz proclaimed triumphantly, disproved the idea of complete equality of all human beings as a socialist reverie. The most unthinkable offence against natural order was equality between white and coloured races: A total mixture of races, he warned, would bring about the end of civilization.

Lorenz was not simply providing a vindication of racial segregation and class hierarchy. He proposed a perspective on human society which was consequentially based on the idea of selection. Genealogy was the adequate method to visualize how social stratification, i.e. social order, was produced. Developing Lorenz' ideas further, the historian Armin Tille defined genealogy as a social science in its own right. According to Tille, the basic idea of genealogy was that "the individual man is an intellectual *abstraction*" while kinship formed the "constant basic element of society."[34] Due to this anti–individualistic perspective, the genealogical approach was closely related to organicist concepts in contemporary sociology. Tille explicitly referred to Ferdinand Tönnies' influential dichotomy between community and society, claiming that

[31] Lorenz (1891), p. 255.
[32] Lorenz (1898), p. 316.
[33] Ibid., p. 334.
[34] Tille (1913), p. 376.

genealogy provided the appropriate tool to analyze how "organic" communities were generated. He particularly recommended socio–genealogical studies on the formation and transformation of classes and professional groups.

There were even more clear–cut ideas about the sociological application of genealogical databases. Robert Sommer proposed a very ambituous agenda for psychological family research. Besides his psychiatric research, Sommer worked in the field of experimental psychology, with a special interest in aptitude tests. The approved methods of individual psychology, he claimed, had to be complemented by an examination of the tested person's family. The combination of family research and psycholoigcal assessment would generate a new, exact social psychology. Sommer regarded it as the crucial problem of modern society that many people were not choosing the vocation adequate to their inborn ability. He held that it was the "task of social psychology, and of a psychological social policy which has to be derived from it, to guide the natural dispositions of the individuals, as far as possible, into those professions where they can develop the greatest effectivity in the interest of society."[35] Sommer conceded that his science was still far from the diagnostic knowledge needed to accomplish this aim. However, his objectives clearly show that the idea of genealogical databases was closely related to a technocratic vision of society. Sommer hoped that sophisticated collections of family records were to provide a tool for the professional selection of individuals. On the whole, this implied a vision of control over the process of selection that shaped human society.

There was also a strong affinity between the idea of "scientific genealogy" and the interest in population development. Fears about stagnating birth rates as a consequence of urban life began to spread in the 1890s. Kekulé strongly suggested that genealogists should turn to demographic problems since "in principle, all questions about population development are genealogical questions".[36] In fact, the genealogical perspective implied a novel concept of population. Rather than just comprising the living inhabitants of a nation, the genealogical concept of population included the past generations. This idea was most clearly expressed in the plans for a national genealogical register propagated by Lorenz's followers. The genealogical network they envisaged was to represent the complete ancestry of the nation. Rather than as an association of citizens, the population would appear as an organic entity – a virtual body of the people or, as the German eugenicists expressed it, a *Volkskörper*.

"Genealogical awareness"

The plans for a national genealogical register, thus, had a double meaning. On the one hand, it was to serve eugenicists to identify abnormal hereditary dispositions, on the other hand, the reconstruction of common ancestry promised to strenghten the sense of national community. The latter aspect was related to the fact that such large collections could only be accomplished with the cooperation of amateur genealogists. As Breymann had stressed, this mobilization implied the chance to popularize biological thinking. In a similar form, this idea had been expressed by Lorenz. Though he emphatically claimed that genealogy was a science, he decidedly did not want

35 Sommer (1907), p. 6.
36 Kekulé (1900), p. 109.

to turn it into a method for experts. It was up to the "scientific genealogist" to advise laymen how to collect the right material for their descendants.[37] Lorenz regarded the interest in family history as a part of the human condition and an indicator of cultural standard. The growing popularity of genealogy, thus, was a sign of true social progress. Lorenz hoped that due to "raising living conditions", more and more people would develop an interest in their own descent or, as he called it, "genealogical awareness". Genealogical awareness, he hoped, was to become an effective weapon against all socialist and egalitarian evils. This idea was eagerly taken up by his followers. As one co–founder of the *Zentralstelle* put it with monarchist zeal, genealogy had a "high moral value" since it formed "a strong bulwark against subversive activities of all kinds" by strenghthening the sense for family and fatherland and therefore supporting a state–loyal attitude.[38] Yet, Lorenz had also emphasized that the practice of family research would help to popularize the insights of modern biology. With the gradual integration of genealogical organisations into the eugenics movement, this aspect became more and more predominant. Sommer developed this idea with a remarkable consequence. Like Lorenz, he strictly rejected all proposals for compulsory eugenic measures, labelling them as true expressions of contemporary moral degeneration. All the more, the "racial decay" of modern society had to be fought through eugenic education. And the most effective way to spread the awareness of imminent degeneration was "the general penetration of the people with the ideal of natural aristocracy".[39] The idea of aristocracy, Sommer demanded, had to be reconstructed in it original sense: as a feeling of responsibility for the own family's specific qualities. The practice of genealogy, thus, was the best way to make people internalize the ideas of eugenics.

After World War I, this aspect gained importance. The position of genealogy as a method for exact studies on human heredity became increasingly questionable. On the one hand, it had turned out that most hereditary phenomena were not explicable in simple Mendelian terms, on the other, twin research emerged as a new standard method of human genetics. In 1930, the eugenicist H.W. Siemens stated that family research had not yielded the scientific results once hoped for – and most likely never would. Nevertheless, he stated that it was indispensible for the cause of eugenics, since the popular practice of genealogy created an emotional connection to family and people, accordingly providing the most "unobstrusive" and continuous way of propaganda for racial biology.[40]

Genealogy, after all, has never only been a method for the science of heredity. As a practice that is deeply connected with the self–definition of individuals and collectives, it imposes a specific dynamic on the study of human heredity. Genealogy provides the thinking about heredity with a moment of historicity, but it also brought about a biologization of history. Lorenz defined genealogy as "the bridge on which historical and natural science meet", and he was right to stress the importance of this encounter.[41] The emergence of a hybrid field between history, sociology and biology proved to be most momentous for the early 20th century human sciences. The

[37] Lorenz (1898), p. 139.
[38] Ueltzen–Barkhausen (1905), p. 10.
[39] Sommer (1907), p. 221.
[40] Siemens (1930).
[41] Lorenz (1898), p. 26.

genealogical perspective did not only foster selectionist and organicist concepts of history and society. It also changed the scope of eugenics. Only the idea that population was an organic body and that the hereditary abilities of families, nations and races were the true basis of historical development, provided eugenics with its particular authority and assertiveness.

Epilogue: Total genealogy

The high time of genealogical collections was to come after World War I, when eugenics became an integral part of public health policy. Representatives of the *Zentralstelle* were involved when the *Reichsgesundheitsamt* discussed the foundation of a National Institute for eugenics and human heredity in 1923.[42] These plans eventually led to the establishment of the Kaiser Wilhelm Institute for Anthropology, Human Genetics and Eugenics, an institute not devoted to genealogical collections. Such databases flourished in Ernst Rüdin's genelogical department at the *Deutsche Forschungsanstalt für Psychiatrie*, which assembled the largest collection of psychiatric family histories in Germany. With support from the state of Saxony, the eugenicist Rainer Fetscher built up a database of the "inferior" which was designed to comprise all Saxon convicts and their families. The Bavarian ministry of Justice established a Criminal Biological Record Office in 1924, which had the right to access prisoner's files, but also to school and parish records.[43] The anthropologist Walter Scheidt called for the replacement of classic anthropological race typologies by "population biology", a method based on a complete genealogical survey of rural populations. Scheidt was also an outstanding figure among those eugenic visionaries who demanded a national genealogical register, as he proposed a central statistical office assembling all medical and juridical records on the whole population and its complete known ancestry.[44] While such plans never materialized, Scheidt's concept of local population studies became seminal for the program of a "racial survey of the German people" carried out in the early 1930s. But the heyday of "population genealogy" was still to come under National Socialism. The Nazi Peasant's League (*Reichsnährstand*) and the Nazi Teacher's Association launched a project aiming at the total working–up of all German parish registers up to the 17th century. The material was to be issued in "kinship books" for every German village designed to raise the rural population's awareness of common racial descent. The second – and more important – plan was to create a comprehensive database by using a filing system developed by Scheidt.[45] Card indices would allow to trace every individual's ancestry, including the accessible information about their health. *Reichsnährstand* officials hoped to link up these files with the material collected in the eugenic "inventory" campaign of the psychiatric asylums. They were also in touch with the Nazi authorities who were trying to detect people of "gypsy" descent. Originally, the *Reichsnährstand* had started the systematic registration of genealogical sources for similar reasons: it was meant to simplify the procedures connected with the proof of "purely Aryan" descent. The project, thus, combined the

42 Weingart et al (1992), p. 241.
43 Weindling (1989), p. 385.
44 Scheidt (1930).
45 Klenck/Kopf (1937).

idea of pure descent and the eugenic vision of total control over genetic defects. In Foucauldian terms, the conjunction between the myth of the blood and the control of heredity was carried to the extreme. Total genealogy was both to create a sense of racial aristocracy in all "racially pure" Germans and to defend the racial community against all threats to the "hereditary health".

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Bismarck the Tomcat and Other Tales: Heredity and Alcoholism in the Medical Sphere, The Netherlands 1850–1900

Stephen Snelders, Frans J. Meijman and Toine Pieters

1. Introduction

A tale

Once there lived in Paris a baker who was well known and appreciated in his neighbourhood for his very excellent pies and cakes. His business was therefore thriving. Not only his customers enjoyed the baker's products, so did various rodents whose habitats overlapped those of the humans. To assert his dominance over these animals the baker had taken in another lodger: an Angora cat. The fierceness of this tomcat earned him, in these days after the catastrophal defeat of the French by the German armies, the name Bismarck.

Bismarck justified his name and turned out to be an excellent rat–catcher. Not only rats were to his liking, though. Bismarck also took a fancy to the delicious pastry produced by the baker. This in itself was not a problem. But unfortunately the baker, who had grown up in a culture where the use of alcohol was considered normal and necessary, also made pastry of which this drug was one of the ingredients. Especially his rumcake was (deservedly) famous. It was also appreciated by Bismarck the tomcat. After consuming one of these cakes the people of the neighbourhood would see Bismarck walking and tip–toeing in a pleasantly drunk condition. They did not realize that this was the first step in a fast deterioration of Bismarck's physical and psychological condition. Having taken a liking to the rumcakes, Bismarck became, in short, a dipsomaniac. And where in the beginning his reaction to the alcohol had been one of pleasant drowsiness, now he would react less favourably to the drug. He became ill–tempered, more and more wild, and finally mad until he started to attack, not the rodents, but the humans, and had to be put out of his misery.

This late nineteenth century story reads as the reverse of Egar Allan Poe's *The Black Cat*, the story of the decline and fall of an alcoholic who ends up attacking (and is revenged upon by) his cat. Admittedly Poe's story is far more chilling. But then his story was literature and published in a literary journal. However, this originally German tale of the tomcat Bismarck was meant to serve the progress of science. It was used instrumentally in the March 1901 edition of the Dutch medical weekly, the *Geneeskundige Courant voor het Koninkrijk der Nederlanden* ('Medical Journal for the Kingdom of the Netherlands') to support the claim that alcoholism presented a most serious health problem that deserved imminent medical attention.[1] In an allegoric sense the story warns against the dangers of alcohol abuse, and against the low threshold between an 'innocuous' use of alcohol in daily life and the degeneration into madness.

Around 1900 it was generally accepted that chronic alcoholism could be inherited or transmitted to descendents as morbid nervous predispositions. Together with tuberculosis and syphilis, alcoholism was regarded as a major cause of degeneration and as such defined as a public

1 *GC* 55 (1901), no. 7.

threat that should be curbed by public health measures. In a number of European and American countries support was mounting for 'hard–line' policies of eugenics, such as marriage restrictions and involuntary sterilizations. However, as we will discuss in this paper, other scenarios of fighting the 'alcoholism peril' were also enacted. To understand how these scenarios took shape in Dutch medicine in the second half of the nineteenth century, we need to take into account the diversity and fluidity of medical and public debates around degeneration and heredity.

2. Research focus and questions

This paper reports on preliminary findings of the on–going research project on 'Heredity and Concepts of Illness and Health in the Netherlands in Modern History'.[2] The focus here is on the second half of the 19th century. The paper investigates the roles played by notions of heredity in Dutch medicine, by analysing discourses on prevention and treatment of alcoholism and alcohol abuse in the Dutch medical literature.

It is commonplace in the historical literature to relate medical positions on alcoholism in the 19th century to evolving concepts of degeneration and heredity.[3] Two recent volumes are exemplary of the general sort of accounts of nineteenth century approaches to degeneration and hereditary diseases such as alcoholism. Craig Heron, in his wonderful history of alcoholism in Canada, writes about the last decades of the century:

> The growing numbers of physicians who believed inebriates had inherited their "craving" for alcohol shared the environmentalist concern that these degenerates be confined under medical care at an early stage, in the hope of weaning them from their destructive habit. Yet most of those in the medical profession who believed in the crucial importance of heredity saw little hope for drunkards in the end and took limited interest in them. Doctor's optimism about institutional treatment for any kind of mental–health problem was waning by the end of the nineteenth century. Prevention became more important than cure.[4]

The same position is held by John W. Crowley and William L. White in their history of the first U.S. asylum for alcoholics, the New York State Inebriate Asylum in Binghamton, which opened in 1864. They write: "Whereas 'made' drunkards were capable of responding to the care of moralists and reformers, 'born' drunkards should be placed in the hands of professional authorities who would control them by legal or medical means. The appropriate object of such control was the congenital dipsomaniac, whose chances of full recovery were deemed to be small but whose threat to society was deemed to be large."[5]

In both case studies alcoholism shows double faces: cause and product of degeneration, vice and malady.[6] The readings above are in line with the idea, that medical hereditarianism in the 19th century transforms from 'soft' into 'hard', i.e. that it became increasingly deterministic and fatalistic with concomitant implications in medical and public domains. According to some

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2 Earlier findings are reported in Snelders (2003); Snelders and Pieters (2003); Idem (in press).
3 For an overview of the rise of this relationship between alcoholism and degeneration, cf. Bynum(1984); Sournia (1990).
4 Heron (2003), 143.
5 Crowley and White (2004), 77.
6 White (2003).

authors this happened because this hardening conveniently explained the failure of medicine to find cures for the problem of alcoholism and other diseases.[7] But the thesis of a 'hardening' hereditarianism has also been explained by what has been called 'the hardening face of nature' in scientific and cultural understandings. "Medical hereditarianism assumed much stronger form in the 19th century, when nature turned from a benevolent, purposeful entity to assume the harsh face of physical necessity."[8] These claims are supported by evidence from the medical literature of the period. Within this perspective, the tale of Bismarck the tomcat can be read as representing the serious social and biological problem of the incurable, degenerative hereditary disease alcoholism, as represented in one of the higher mammals.

We consider alcoholism to be an exemplary case to study the dynamics of medical hereditarianism, since alcoholism was considered to be an important cause and consequence of degeneration, shown in mental diseases. As Gianna Pomata has written: "It is significant that most of the 19th century doctors' interest in hereditary diseases shifted from gout, the patrician malady, to insanity – a disease considered to be endemic at the other end of the social ladder."[9] We therefore focus on the questions how, to what extent, and why knowledge of inheritance was anchored in medical concepts and practices around alcoholism. By studying five leading Dutch medical journals, this paper looks for changes in the medical conceptualization and practical approaches around alcoholism. The main goal is the closer inspection of the fore–mentioned assumptions regarding the transformation of hereditarianism, in 19th century Dutch medicine. The paper makes a distinction between two analytical levels. First there is the level of theory and conceptualization within medicine; second, we distinguish the level of application and practical approaches. Medicine is a field in which a logical coherence between these two levels often seems to be lacking: it is a science as well as an art.[10] In medical practice 'elastic' approaches dominate, given the unruly nature of medicine. The semiotic status of hereditarianism, that we label 'plastic', involves flexible boundaries between the respective influences of 'nature' and 'nurture'. Connecting concepts of hereditary disease to that of individual constitutions did not necessarily have to entail a desire on the part of physicians to rationalize and to excuse their inability to treat a range of persistent chronic maladies, including alcoholism. It could also justify an elastic approach in treatment, undisturbed by hereditary determinism. Discourses of conceptualization as well as practices of treatment and prevention have both to be contextualized. On both these levels more broader cultural themes and social beliefs, in this case especially that of degeneration, become apparent.

In this paper a contribution to the historical assessment of the connections between heredity and alcoholism in the medical domain between 1850 and 1900 will be made by a focus on an unexplored source: medical journals and publications from the Netherlands. The Dutch are not regarded as pioneers of medical and political activities in this field. It might therefore be that a focus on the Netherlands shows developments more representative of overall developments in the Western hemisphere. In following and analyzing the evolution of ideas and views of Dutch medical doctors on heredity and alcoholism we will particularly focus on diffusion patterns as well

7 Weiss (1987), 19; Dowbiggin (1997), ix–x; Waller (2002); Idem (2003).
8 Pomata (2003), 151.
9 Ibid., 150.
10 Weatherall (1995).

as differentiation and transformation of: 1. medical thought about hereditary transmission; 2. concurrent medical treatment and prevention practices regarding alcoholism.

All the five major Dutch medical journals were selected for research. These are:

1. The *Nederlandsch Tijdschrift voor Geneeskunde* ('Dutch Journal for Medicine'; in this paper abbreviated as *NTvG*). This journal appeared weekly and was (and still is) the major journal for Dutch physicians, in the nineteenth century being the journal of the Dutch Society for the Advancement of Medicine. It was created in 1857 after a fusion of nearly all the existing medical journals, with one significant exception (see no. 2). *NTvG* was politically oriented to the liberals; it counted leading hygienists who advocated public health policies among its contributors.
2. The *Geneeskundige Courant voor het Koninkrijk der Nederlanden* ('Medical Journal for the Kingdom of the Netherlands', in this paper abbreviated as *GC*) was the one medical journal of importance that remained outside the fusion to *NTvG*. First published in 1847 and appearing weekly as well, it was politically more oriented to the conservatives.[11]
3. The *Psychiatrische Bladen* (Psychiatric Papers' in this paper abbreviated as *PB*) started publication in 1883 and were devoted to subjects of psychiatry and neurology, as reflected in the change of its name in *Psychiatrische en Neurologische Bladen* ('Psychiatric and Neurological Papers', in this paper abbreviated as *PNB*) in 1897.
4. The *Geneeskundige Bladen uit Kliniek en Laboratorium voor de praktijk* ('Papers from Clinic and Laboratory for Medical Practice', in this paper abbreviated as *GB*) appeared for the first time in 1894. They aimed to present the contemporary discussions in clinical and laboratory research.
5. Finally, the *Tijdschrift voor Sociale Hygiëne en Openbare Gezondheidsleraar* ('Magazine for Social Hygiene and Public Health', in this paper abbreviated as *TSH*) was started in 1899 as the 'mouthpiece' of the hygienist movement. Its first two volumes can only be taken into account in our analysis.

3. Concepts

At the start of the 20th century, Dutch doctors in general took the concept of hereditary predisposition to alcoholism for granted. Research reports in the Dutch medical journals translated from the German, French, and British literature, that appeared over the course of the 1890s, and were based on family–tree research and statistical studies of the inmates of asylums, seemed to establish beyond doubt certain facts. Alcoholism (or dipsomania) had a strong tendency to be hereditary. However in most cases it changed form into other mental diseases. Inmates of asylums were to a large, but debated degree (42,6 % according to one French study) hereditary insane due to the influence of alcoholism. The hereditary influence of alcoholism expressed itself in different forms: directly as delirium tremens and periodic bouts of alcohol

[11] On the Dutch medical press until 1857, see Delprat (1927). On *NTvG*: Idem (1932). Unless mentioned otherwise, references to *NTvG* are to the second series, starting in 1865. On the politics of *NTvG* and *GC*: Houwaart (1991), 224.

abuse; indirectly in other psychoses; and biologically in 'deprivation of the progeny' and extinction in the third and fourth generation. This pathological form of heredity could be transmitted by inheritance due to chronic alcohol abuse of one or both of the parents, or due to alcohol intoxication of the germ–plasm during sexual intercourse, or due to what we would call foetal conditioning by an alcoholic mother.[12] The following excerpt from an article by the physician J. Kat, an active member of the temperance movement, on the influence of alcohol on posterity was fairly typical of the articles in medical journals around 1900.

> *'Das grauige Kapital* [the drab capital, i.e. the influence of alcohol abuse] shows on the basis of health statistics produced by researchers in all parts of the world, that retardation, imbecility, idiocy, epilepsy, neurasthenia, criminality, are for the greatest part grounded in alcohol use of parents before and during the conception.'[13]

A similar but in terms of degenerative heredity more articulate view can be found in the writings of Amsterdam gynaecologist Hector Treub. Treub´s 1900 New Year speech to Amsterdam doctors, subsequently printed in *GB,* has been regarded as an early example of eugenic thought in the Netherlands.[14] According to Treub, there existed five 'laws of heredity':

1. direct inheritance from the parents;
2. inheritance of traits from an earlier generation, i.e. atavism.
3. indirect or collateral inheritance from a collateral family line.
4. 'initial inheritance' from the condition of the parents during cohabitation. For example a drunk parent increased the chance for idiocy in the child.
5. inheritance of influence, or telegony: for instance a white woman cohabitating with a negro would give birth to a mulat. If she later cohabitated with a white man, the child would again be a mulat.

As far as this fifth law is concerned Treub was rather sceptical. In his speech he mainly engaged himself with the first three laws and he came to some far–reaching conclusions, as we will see. In fact, his expositions were meant to stress the necessity of medical investigation and advice on heredity before marriage. This might indeed seem to point to a hard form of hereditarianism, emphasizing genetic determinism and fatalism. But when we study these 'laws' more thoroughly on the level of conceptualization then the fourth law appears to be deviating from the others. According to this law hereditary characteristics could be acquired during lifetime. Treub's example can be read as a plastic and more 'soft' form of hereditarianism.[15]

Before further discussing the nature of Treub´s hereditarianism and whether or not Treub's concept of heredity was typical of Dutch medical thought around 1900, we will explore the transformations of medical thought about hereditary transmission since the 1850s. The underpinning of Treub's laws of inheritance was based on the results of a research method that had been popular for a long time: family–tree research. For instance, Treub used a family–tree taken from the most influential medical authority on heredity in the second half of the 19th century, French psychiatrist Bénédict–Augustin Morel. Treub used this tree to show how chronic

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12 For example: *NTvG* 27 (1891) II, 328–330; Ibid. 31 (1898) II, 554; Ibid. 34 (1898) II, 496; Treub (1900).
13 *NTvG* 40, I (1904), 108.
14 Noordman (1989).
15 Treub (1900).

alcoholism could lead in the following generations to idiocy and madness.[16] Treub's exposition was neither new as far as the methodology was concerned nor with regard to the research data on display.

The investigation of family trees had been pioneered by Franz Wilhelm Lippich in Laibach. In a study published in 1834 Lippich had given a statistical analysis of two hundred alcoholic patients, and found their offspring to be generally more unhealthy than the general population of Laibach.[17] Morel's treatises on degeneration (1857) and mental illness (1860) stimulated similar studies. His own family–trees famously represented the hereditary degeneration caused by alcoholism: in the first generation moral stupefaction, brutalisation, sapping of the body; in the second: hereditary drunkenness, mania and palsy; in the third: hypochondria, suicidal tendencies; and in the fourth finally: mental defections, idiocy, and premature decease, ultimately leading to extinction. In Morel alcoholism shows its double faces as vice *and* malady, as cause *and* consequence of hereditary predispositions and vicious environments.[18]

But did Morel offer anything sensationally new in the conceptualization of alcoholism? The impact of his work may have been more due to the elaboration of existing notions than to a revolutionary new approach. Even before Morel published his volumes, leading Dutch psychiatrist J.N. Ramaer emphasized in 1852 that inebriety was the cause of hereditary mental diseases, primarily idiocy. Ramaer referred to Lippich's evidence, but also to the knowledge of this hereditary degeneration among the ancient Greeks.[19]

What seems to happen over the course of the next half a century is that this theory of degeneration is repeated again and again, occasionally confirmed by new family studies. We did not find any divergent opinions. In Dutch medical literature from the 1850s until Treub's lecture Morel was again and again cited as authority and evidence for the double faces of alcoholism. In his public health manual of 1872 the leading hygienist Ali Cohen incorporated the views of Morel.[20] So did twenty years later psychiatrist Pierre F. Spaink in his 1892 monograph on alcoholism.[21] Jan Broers in his M.D.–thesis on alcoholism, morphinism and chloralism, the first Dutch medical treatise on the broader subject of addiction, lamented that especially chronic alcoholism had the 'important disadvantage' that posterity had to suffer for the sins of its ancestors (in Morelian degeneration vice becomes the disease of the descendants, and disease becomes the vice).[22] *GC* regularly referred in its columns to the effects of a 'Morelian degeneration', for instance in 1870 by publishing the research results and conclusions of Morel's pupil Doutrebente on hereditary madness, or three case–studies of another French physician, Taguet, in 1877.[23] Neither *GC* nor *NTvG* ever doubted the scientific truth of Morelian degeneration, nor did any of the other magazines.

The discussions about evolution and heredity that arose after the publication of *The origin of species* in 1859 did not exert any influence on this. Family–tree research done after Morel was

16 Ibid., 34.
17 Bynum (1968), 175–176.
18 Morel (1857); Idem (1860). On Morel: Coffin (2003).
19 Ramaer (1852), 97–100.
20 Ali Cohen (1872), 156.
21 Spaink (1892), 19–20.
22 Broers (1886), 125.
23 *GC* 24 (1870), no. 10; Ibid. 31 (1877), no. 49, 50, 51.

regularly cited and always confirmed his conclusions: only the exact percentage of the expression of predisposition wavered, but this was not taken in any sense as invalidating the theory. *GC* reported in 1884 on German research, in which among idiots in c. 10 % of the patients evidence was found of chronic alcohol abuse or dipsomania.[24] In 1899 the news was brought that a research in Bonn had uncovered 709 descendants of a 'well–known' alcoholic woman who had died in 1800. Of these descendants 462 had become murderers, criminals, beggars, or prostitutes, and had cost the German government six million francs.[25] Leading articles once more explained the degenerative effects of alcoholism on succeeding generations in 1895 and 1900.[26] Psychiatrist A. Tellegen did not doubt in *PB* in 1884 the hereditary consequences of alcoholism, but recognized that figures on the hereditary etiology of madness differed extremely, from 4% to 90%! But research had one severe problem: persons could have the predisposition to madness, but die before this predisposition was expressed.[27] *NTvG* also endorsed the relationship between hereditary degeneration and alcoholism by citing family studies. R. Demme from the University of Bern saw Morel confirmed in 1890 in his study of twenty families, in which one or both of the parents were alcoholics, over a period of 12 years. Of 57 children, only 10 (17,5 %) had had a normal development. 25 had died in the first weeks after birth, and 22 showed congenital defects: defective physical development, chorea, epilepsy, idiocy. In a group of 10 families with moderate drinkers as parents, 50 of the 61 children had grown up healthy (81,9 %).[28] In 1895 *NTvG* reported the conclusions of French psychiatrist Legrain: in his asylum 42,6 % of the inmates were hereditary insane due to the influence of alcoholism.[29] This research was in 1898 confirmed by the study of 1200 cases of hereditary insanity by Farquharson in Britain. Dipsomania had according to Farquharson a strong tendency to be hereditary, although in most cases it changed form into other mental diseases.[30]

To the reader of *GC* in 1901 then, the story of Bismarck the tomcat might have had implications that went beyond the moral decline of one individual cat. Most likely, Bismarck's decline symbolized the fate of a whole race of dipsomanic cats. It would have been as recognizable to a reader of *GC* in the 1850s. Which makes us wonder, how medical thought about alcoholism and degeneration relate to the dynamic and controversial discussions about biological evolution in this period.

Connecting degeneration theory and evolutionism started in the Dutch medical literature in the early 1880s. This means that the impact of Darwin on the medical framing of alcoholism and degeneration was almost non–existent in the 1860s and 1870s. Darwin seems not to be relevant in any way to this framing, for which Morel was sufficient authority. The theory of degeneration was as acceptable to those physicians who showed a positive stand towards the new evolution theories,

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24 *GC* 38 (1884), no. 6, 7.
25 Z., 'De afstammelingen van een alcoholist', *GC* 40 (1899), no. 40.
26 Niermeijer, 'Alcohol en alcoholisme', *GC* 49 (1895), no. 31; A.N.J. Hazedoes van Almkerk, 'Alcoholisme en de houding van medici te dien opzichte', *GC* 54 (1900), no. 10.
27 A.O.H. Tellegen, 'Eenige beschouwingen over krankzinnigheid, hare oorzaken en hare behandeling', *PB* 11 (1884), 5–46.
28 S.K. Hulshoff, review of R. Demme, 'Ueber den Einfluss des Alkohols auf den Organismus des Kindes', *NTvG* 27 (1891) II, 328–330.
29 *NTvG* 31 (1895) II, 554.
30 *NTvG* 34 (1898) II, 496.

as to their more traditional and religiously minded colleagues. We must remember that Morel himself was not a Darwinist in any sense. He was a Catholic who framed degeneration in terms of the 'Fall of Man'.[31] The great authority for the physician on these matters in the 1880s was not any (Neo–) Darwinian biologist, but one of their own, the German pathologist Rudolph Virchow.

Virchow was not only the dominant figure in establishing and transforming German laboratory medicine in the second half of the 19th century. He was a physician profoundly interested in relating clinical research to medical practice. Virchow was moreover one of the leaders of the German liberals and his political activities almost led him into a duel with Bismarck – the statesman, not the cat.[32] *NTvG* discussed in 1882 a lecture of Virchow on Darwin and anthropology, in which he cautioned against a rushed generalization and transmission of Darwin's theories from animals to humans.[33]

The great challenge to conceptualizations of alcoholism and heredity in the medical domain was provided by August Weismann´s new concept of the 'germ plasm'. Weissman´s theory had a major impact on the redefinition of the concepts of heredity at the end of the 19th century. Starting from the question of how the germ plasm with the inherited characteristics could reproduce itself, Weissman conceptualized the soma, the body, as a mere transport vehicle for the germ plasm. In doing so he separated the problem of heredity and the problem of growth and differentiation. Changes in the soma were not transmittable to the germ plasm. Weismann 'proved' in 1883 that traits required during one's lifetime could not be inherited by descendants. However this proof did not have the impact in the medical domain historians of biology have often accorded to it.[34]

W. Koster discussed the matter in 1886 in *NTvG*. He called Weismann's *Bedeutung der sexuellen Fortpflanzung für die Selektionstheorie* a 'phantastic–speculative evolution theory', that seemed to contradict established pathological and clinical knowledge. Virchow gave acclimatisation as an example of the inheritance of acquired characteristics, while Weismann was of the opinion that the acclimatised individual was already, by chance, adapted to his new environment. Koster thought that Weismann's idea of the continuity of the germ plasm explained much, for example the inheritance of the 'Jewish type'. But if Weismann was right there could not exist 'infectious and hereditary' diseases. However, syphilis and tuberculosis were regarded as ample proof for the claim of the hereditability of these common diseases.[35]

A year later, a leading article in *GC* clearly stated that under specific conditions acquired characteristics could be inherited. Why this happened was still a mystery; that it happened, beyond doubt. Weismann notwithstanding, the article informed its readers that: "The acquired character [developed by education, environment, etc, the 'envelope' of the true, inherited character] later changes again into the inherited character, because it is inherited by the descendants". *GC* specified that not the characteristics themselves, but the predisposition to develop them was inherited. Expression depends on circumstances. The more often characteristics occurred in

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31 Huertas (1992).
32 Ackerknecht (1953).
33 *NTvG* 18 (1882) I, 823–824.
34 On the discutable idea that Weismann disproved the inheritance of acquired characteristics: Bowler (1988).
35 W. Koster, 'Ontwikkelingsleer en ziektekunde', *NTvG* 22 (1886) I, 341–349.

family–trees, the more chance that they would return in later generations. A person with a powerful imagination could transfer his acquired characteristics more easily to his descendants: the example given was that of an alcoholic father. Inheritance of equal characteristics occurred, but more often polymorphism, the unequal distribution of dispositions. Expression of these predispositions was dependent on circumstances: for example shock, misery, strain. Under the right circumstances the predisposition could even express itself as genius, as in the case of Schopenhauer. Morel's family–trees once again figured prominently to demonstrate the mechanisms and patterns of hereditary transmission.[36]

This perspective of plastic expression was compatible with existing medical traditions. It did not conflict either with the new theory of evolution as expressed by Darwin. It was recognized that Darwin himself believed in the inheritance of acquired characteristics.[37] But even to Weismann's ideas the medical community was not unfavourably disposed. In 1891 Zwaardmaker, one of the editors of *NTvG*, called the theory of Weismann a hypothesis of great value, a progress in the direction of a mechanical explanation of nature. There was much that seemed to support Weismann, such as the occurrence of atavisms and of morphological characteristics that were insurmountable for the individual, but not for the species as a whole. Still, the theory was held far from proven.[38] Koster in turn undertook in 1893 a review of Weismann. There is no evidence for the inheritance of acquired characteristics in a positive sense, he wrote, but Weismann did not deny inheritance in a negative sense: agents as alcohol or virus syphiliticum could damage sperm cells and lead, when sperm and egg mingled, to a spontaneous poisoning of the germ plasm.[39]

This 'inheritance in a negative sense' became in the course of the 1890s an equally satisfactory and convenient explanation for the hereditary degeneration caused by alcoholism as the inheritance of acquired characteristics had been. What we have here is a typical form of medical eclecticism, producing a workable explanatory tool that met a need, based on doctor's experiences and seemingly proven by empirical family–tree studies. Possible inconsistencies between the fore–mentioned biological concepts were noted but amended and adjusted to produce a medical argument consistent with a perspective of plastic expression of hereditary predisposition.[40] It is of importance to look at the adjustment and amendments of knowledges of heredity by doctors from a functionalist perspective, and not from the perspective of consistency with developments in the scientific sphere.

At the end of the century there was consensus about the three possible mechanisms by which alcoholism could be inherited and which explained both Treub's 'laws of inheritance' and the onset of the degeneration process, i.e. the transformation of a metabolic disorder into morbid nervous dispositions. As the authoritative Swiss researcher and temperance activist Auguste–Henri Forel explained in 1892 on the Fourth International Congress against alcohol abuse, alcohol itself was a toxic agent that led to degeneration of progeny.[41] Alcohol abuse of the mother could

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36 'De overerving van zenuw– en zielsziekten', *GC* 41 (1887), no. 22, 23, 24.
37 'De overerving van verworven eigenschappen', *GC* 43 (1889), no. 43.
38 *NTvG* 27, (1891) I, 418–420.
39 *NTvG* 29 (1893) II, 293–311
40 For similar developments in France: Pinell (2001).
41 Le Rütte Jr., 'Het vierde internationale congres tot wering van het misbruik van sterken drank', *PB* 11 (1892), 220–221.

lead to poisoning of the foetus in utero. *Blastotoxie* occurred when a child was conceived while one or both of the partners in the sexual act were drunk. A third mechanism, *Blastophtorie*, functioned when one of the parents was a chronic alcoholic whose germ plasm was seriously poisoned by the alcohol.[42]

While in accordance with neo–Darwinism, this plastic perspective on hereditary transmission also fit into Morelian degeneration schemes. Even the positive implications of the inheritance of acquired characteristics were saved: in the course of generations healthy living could restore the vitality of the germ plasma.[43] J. van Rees, physician and leading prohibitionist, explained in 1902 in a propaganda brochure against alcohol use that the germ plasm could regenerate in the third or fourth generation, on the condition that it was mixed with undamaged plasm.[44] Forel furthermore considered, as *NTvG* explained its readers in 1895, that man had a 'plastic' disposition: the expression of the hereditary disposition could take different forms, depending on opportunity and exercise, and did not have to take a pathological form per se.[45]

Given the plastic nature of attempts by Dutch doctors at adjusting theories on heredity to medical problems this raises the question to what conclusions regarding alcoholism treatment policies the transforming thoughts about hereditary transmission led.

4. Approaches: public health

Dutch doctors regarded alcohol abuse and alcoholism both as an individual and a public health problem. In particular the consumption of strong liquor, especially the Dutch *jenever* (geneva), was high on their agenda. Quite a number of physicians, however, considered fermented drinks such as beer and wine as nutritious and stimulating health. The concerns about the consumption of jenever were not a new feature of the 1850s, but had been around since at least the 18th century.[46] N.B. Donkersloot, psychiatrist and editor of *GC*, in 1854 put the abuse of strong liquor on a par with the state lottery as one of the two great disasters that had overcome the Netherlands. Not only was the use of strong liquor an individual health problem, since it badly affected physical health and was the cause of mental and moral aberrations. It was also a public problem, since it meant economic misfortune for consumers (who spend their money on drink) and destroyed religion and higher morality. Donkersloot pleaded for jenever prohibition, a position he would keep advocating in *GC* until his death in 1890.[47]

It is easy to see that the Morelian twist to the degeneration story fitted Donkersloot like a glove, and that it continued to be acknowledged in his articles in *GC*. It is also remarkable that Donkersloot's views on jenever were exactly the same as those of one of the leading hygienists, L. Ali Cohen, almost two decades later. In his public health manual of 1872 Ali Cohen named the use of jenever as *the* chief source of the misery of the Dutch people. He expanded the usual description of jenever's health hazards with the consequences for posterity as described by Morel.[48] Just as

42 On these views: Finzen (1977), 31.
43 Ibid., 33.
44 van Rees (1902).
45 *NTvG* 31 (1895) I, 324.
46 For analyses of the British 'gin craze', one of the first modern drug scares: Warner (2002); Dillon (2003).
47 Donkersloot (1854).
48 Ali Cohen (1872), 155–160.

Donkersloot, Ali Cohen was unlike most of his colleagues also concerned about the physiological effects of fermented alcohol: already in 1863 he referred in *NTvG* to French research that had shown that beer and wine were 'false' nutritients, since they diminished the use of real nutrition. Its only value lay in its therapeutic use in medicine, as a stimulant.[49] The question of the nutritious and therapeutic value of alcohol was one of the few questions regarding the effects of the drug that physicians continued to discuss in the medical press.

By the 1890s these discussions had become tied up with the discussions whether physicians should advocate moderation, as the majority obviously did, or whether they should preach abstinence. These discussions could take fierce forms, for instance at the 4th International Congress against alcohol abuse in The Hague in 1893, with Forel as foremost spokesman for the extremists.[50] Two years later at the next international congress the issue was even more hotly debated. W.P. Ruijsch reported that the moderates (such as himself) were treated as traitors by their opponents.[51] The psychiatrist Spaink was saddened by the exaggerations of the advocates of prohibition. Alcohol had its use as medication, he thought, although he prohibited its use in his own asylum in Apeldoorn.[52]

Hardly anyone questioned the public health hazards of chronic alcohol abuse. Ali Cohen was a contributing editor to *NTvG*, and the hygienists allied themselves politically to the liberals, while Donkersloot was the editor of the more conservative *GC*. But their position on the alcohol problem was the same, and Ali Cohen too advocated preventive and repressive measures in this 'war on alcohol'. These strong opinions of prominent members of the medical profession contributed to the enactment of the first law on alcohol use (*Drankwet*) of 1881. This law regulated to some extent the trade in distilled liquor by preventive and repressive measures (such as limitation of the number of pubs and a prohibition of liquor sale to children younger than 16) and made public inebriety an offence. Until the First World War more than 4000 men and women would end up doing forced labour in state prisons for this offence.[53]

To the temperance movement this law was totally inadequate, but this does not concern us here. What is relevant is that the whole spectrum of sides within the medical profession, that is the sides that expressed themselves in the medical press, were of the same opinion concerning the necessity of state regulation in the fight against alcohol abuse. However *NTvG* never got around to discussing the exact nature and desirability of the 'preventive and repressive' measures. Prohibition was certainly not one of them, partly because Dutch physicians were clearly too fond of their own alcohol intake. In 1900 the diatribe of Hazedoes van Almkerk against any use of alcohol in *GC* was coincidentally illustrated with an advertisement for a wine seller.[54] In 1901 on the general meeting of the Dutch Society for the Advancement of Medicine the representative of the city of Dordrecht argued that it was not the task of the Society to prohibit alcohol use. He evoked laughter when he added: "This representative has at least not noticed anything of this these

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[49] *NTvG* First series, 7 (1863), 664–665; Ibid. Second series 1 (1865) I, 523–533. On 19th century therapeutic use of alcohol: Paul (2001).
[50] *NTvG* 29 (1893) II, 321–323.
[51] *NTvG* 31 (1895) II, 551–552.
[52] *NTvG* 31 (1895) I, 92–93, 326.
[53] Van der Stel (1995), 156.
[54] A.N.J. Hanedoes van Almkerk, 'Alcoholisme en de houding van medici te dien opzichte', *GC* 54 (1900), no. 10.

days [at the social gatherings surrounding the meetings]."[55] The majority of Dutch physicians did not intend to confuse the fight against alcoholism with their personal enjoyment of life. *GC* took more stringent positions. To its editors, the hereditary degeneration caused by alcohol abuse was the 'damnation of the human race', and of more importance to the medical profession than bacteria research, sources of malaria, adulterated food, or miasmas.[56] Measures against public inebriety, as would become law in 1881, were not the right method, although Donkersloot did advocate fines and, on second offence, loss of citizen's rights.[57] Donkersloot consistently advocated that the only radical measure would be to make alcohol a prescription drug. Other, less radical measures, would be a state monopoly on the sale of alcohol and the close of the jenever pubs on Sunday and Monday morning.[58] Donkersloot and his collaborators advocated now and then in *GC forced* abstinence for alcoholics.[59] After his death his successors continued in the 1890s to warn against the dangers of alcohol and alcoholism. They advised physicians to give a good example and make propaganda against alcohol abuse. They advocated alcohol as a prescription drug, warned against the degeneration of progeny, and against the introduction of absinth.[60] The tale of Bismarck the tomcat was only another example of the tradition of the fight against alcoholism upheld by *GC*.

But as with the degeneration theories this tradition had already been around in the 1850s. Did the link between degeneration and the advocacy of public health measures show any new transformations in the second half of the 19th century?

We can indeed pinpoint changes in health policy strategies. In the 1880s and 1890s some voices are heard in the medical literature that seem to point to more support for eugenic policies based on genetic determinism and fatalism. These voices run parallel to the 'hardening' of hereditarianism in the biological sciences. But as we have seen, this parallel development should not seduce us into making any causal connections. Possible policy implications of the various biological theories are discussed only once, by Koster in 1886 in *NTvG*. According to him, the inheritance of acquired characteristics provided some hope that by a policy of (social) hygiene a better species could be created, whereas Weismann´s theories only led to prospect of a Spartan State, in which the inferior had to be eliminated. Koster´s line of argument here shows rather close similarities with a neo–Lamarckian perspective on positive eugenics.[61] But on the not the hereditarianism hardens, but the conclusions of some doctors concerning public health and prophylaxis. However, before the 1900s these voices remain a minority with no political impact.

A first eugenic voice (although not in any way connected to a 'eugenic movement') can be heard in *PB* in 1884. It is based, not on hard hereditarianism, but on the 'proven' inheritance of

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55 *NTvG* 37 (1901) II, 153.
56 'Erfelijke dronkenschap', *GC* 31 (1877), no. 49, 50, 51.
57 *GC* 24 (1870) no. 23; 'Beteugeling der dronkenschap', ibid. 34 (1880), no. 40, 41, 42, 43, 44.
58 'Het alkoholisme, zijn verspreiding, werking op het persoonlijk en maatschappelijk organisme, en de middelen om het te bestrijden', *GC* 33 (1879), no. 5, 6, 7.
59 N.B. Donkersloot, review of B.W. Richardson, *Volksonderwijs over alcohol*, *GC* 33 (1879), no. 26; Ibid. 42 (1888), no. 5, 49.
60 C.W. Bollaan, review of Spaink, *Over alcoholismus*, *GC* 46 (1892), no. 38; Ibid. 49 (1895), no.2; Niermeijer, 'Alcohol en alcoholisme', ibid. no. 31; absinth: ibid. 48 (1894), no. 12.
61 Koster, 'Ontwikkelingsleer en ziektekunde', *NTvG* 22 (1886) I, 341–349.

acquired characteristics. Psychiatrist Tellegen draws a provisional conclusion from this theory. Physicians should advise before marriage on the heritability of certain diseases in the family–trees of the couple. Significantly, he adds, this is only done seldom.[62] Actually the only thing Tellegen seems to do is to point out and advocate a practice that is current among some physicians, a practice that was regarded as consistent and normal for those familiar with Morelian degeneration and the inheritance of acquired characteristics: advice before marriage.[63] It is new that it is mentioned, but far from new as part of the private world of medical practice. When Spaink in 1892 suggests that confirmed drunks should not be allowed to marry this seems a continuation of what physicians (or anybody else with common sense) had always thought.[64] Treub has in 1900 basically the same position as Tellegen, since he asks for a medical advice before marriage that is not legally binding.[65] There is some echo of this idea, when *GC* reports on the French idea of having a heredity report in the military passport.[66] But that seems to be all, apart from one voice, which we would like to argue speaks the language of 'modern eugenics'.

G. Jelgersma, psychiatrist and editor of *PNB*, in 1897 reviewed the magazine of the temperance movement in an editorial. He is sympathetic and recommends the magazine. But he also has his doubts about the goals of the temperance movement. Is prohibition of strong liquor indeed the adequate method to significantly reduce the number of mentally insane (with as much as one–third, as the temperance activists claim) and of criminals and beggars (for the greater part)? The figures from those 'dry' American states where prohibition rules suggest otherwise. This should not be surprising, since alcohol abuse is from a scientific point of view as much consequence as cause of insanity. Where the 'weak' are not able to get alcohol, they ruin themselves by other means: in 'dry' Iowa the use of opium has increased. And now we hear the first and only voice in the medical press before 1900 that sounds distincty 'social–darwinist' in our popular sense. Prohibition would be counterproductive, since it would only keep the weak alive and allow them to reproduce themselves. It would interfere with natural selection. Jelgersma therefore feels that it is the *état maladif*, the pathogenic predisposition that is the cause of alcohol abuse and insanity. This pathological heredity should be controlled – by legislation of reproduction.

For sure, Jelgersma does not deny the efficacy of anti–alcohol propaganda since there is also another form of alcohol abuse with a non–hereditary etiology: the great number of people who suffer from the increased demands and stress of modern society, and who seek escape and relaxation in drink. For those the activities of the temperance movement are very well suited. But as for the hereditary predisposed: they should not be allowed to reproduce.[67]

As said before, Jelgersma's voice here sounds as a first trumpet blast of eugenic thought. It is however interesting that his discourse can also easily be constructed as a 'modern' fin–de–siècle remake of more traditional views on the necessity of medical advice before marriage, just as Morelian degeneration could for its purposes in the medical domain as well be explained by the inheritance of acquired characteristics as by negative inheritance. Personal distaste of a physician

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62 Tellegen 'Eenige beschouwingen', *PB* 11 (1884), 5–46: 18–25.
63 On the survival of this practice in the United States: Pernick (1996).
64 Spaink (1892), 26–27.
65 Treub, 'Huwelijk en ziekte', *GB* 7: 29–48: 44–45.
66 *GC* 56 (1902), no. 28.
67 G. Jelgersma, review of *De Wegwijzer. Maandblad voor geheel–onthouding*, *PNB* 1 (1897), 287–294.

for an alcoholic or an addict in general could as well be justified by his immoral behaviour and danger for the public community in the 1850s as for his Darwinian unfitness and danger for the public community in the 1890s.

Of course, what is new is Jelgersma's plea for legislation, but in the Dutch context this is a very solitary position before 1900 – even Treub does not want to go that far. What is of primary interest here is that 'eugenic' ideas of regulation of reproduction (by regulating marriage) are actually already around and even well–accepted by physicians in the second half of the 19th century. It remains however 'invisible' because it does not, or only now and then, extend to demands for public legislation. We must remember here that eugenic thought in various countries (but *not* the Netherlands) primarily made its essential impact on state legislation in the political, social and economic crises of the Interbellum.[68]

5. Approaches: individual health

In practice, doctors would have to deal more with individual patients and their treatment options than with public health strategies. What can we say about the dynamics in conceptualizations around heredity and alcoholism and individual health approaches?

First of all, we must discount one important historiographical notion about the treatment of alcoholism in the 19th century: the myth of therapeutic pessimism. If anything, the case of alcoholism and heredity shows that the idea of a 'hardening' hereditarianism as explanation of therapeutic failure can not be generalized. Estimations of recovery percentages under the right therapeutic regime ran as high as 40% (and as low as 25–30%) at the end of the century, a figure given by not extremely optimistic authors, and a figure that in the year 2004 is unsurpassed by modern addiction treatment methods.[69] Of course we should not take these figures at their face value, but they clearly indicate something else than therapeutic pessimism.

This does not mean to say that we cannot find any such pessimism in the medical press. In 1888, P. Wellenbergh reported in *PB* on his visit to the psychiatric hospital in Graz, Austria, run by the eminent degeneration specialist Richard von Krafft–Ebing. According to the latter, psychiatrists should make a distinction between madness that is not hereditary and can be cured in 70 to 80 % of the cases, and paranoia, that is the expression of a hereditary predisposition and can only rarely be cured. The implications for the alcoholic seemed evident.[70] Spaink was of the opinion that the symptoms of alcoholism were more severe in hereditary alcoholics. This was logical, because they were already insane or alcoholic before they had had their first drink; after their first drink, they had to become alcoholic.[71] However this did not lead Spaink to therapeutic pessimism concerning his patients with a hereditary predisposition. A.N.J. Hanedoes van Almkerk, medical supervisor of the first Dutch asylum for inebriates in Hoog–Hullen, described his patients in the most pessimistic and abhorrent terms: 'inferior', 'incongruency of the brain parts', 'Aztec skulls' in a denigratory sense, predisposed children with the attitude of wild animals.

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68 This argument, based on a comparative literature review on Britain, the United States, Germany, Sweden, Russia, and the Netherlands, is developed more fully in Snelders and Pieters (2003).

69 *NTvG* 25 (1889) I, 563, 636; Spaink, *Over alcoholismus,* 72.

70 *PB* 6 (1888), 96–103.

71 Spaink (1892), 16–17, 26.

But at the end of his article he claimed to cure 75% of his patients that stayed for more than one year of treatment, and 25% of those that stayed for a shorter duration.[72] His abhorrence for degenerates did not led him to pessimism in his work, although we must add that not all alcoholics in Hoog–Hullen were of course thought to be hereditary predisposed to their condition. It was reported from Hoog–Hullen that, because 60% of its patients were in their thirties, it was not probable that hereditary conditions were the main cause of their alcoholism.[73]

Our thesis is that discovering hereditary antecedents in the family–tree of an alcoholic did not lead into pessimism because physicians generally thought that the expression of the predisposition was plastic, or fluid, dependent on environment and circumstances. These fluctuations in individual predisposition could be combined with the idea that treatment should be "a psychiatric treatment in accordance with the individuality of the patient".[74] It also combined with the possibilities of prophylaxis. The physician could, according to Ruijsch, cure where possible, and prevent by his influence in the families of his patients: by calmly and quietly explaining that alcohol is not necessary for everyone, by banishing it from children's diets, by pointing to alcohol abuse in cases of illness, and to its negative consequences especially for those predisposed to alcoholism.[75] In general, explained *PB* in 1888, a hereditary predisposition to insanity in individuals could be countered with the provision of a healthy wet–nurse for young children (in case of insanity or alcoholism in the mother), caution against the appearing of 'brain congestions', and a sensible education.[76]

Plasticity of genetic expression and elasticity of treatment theoretically opened the way for, not therapeutic pessimism, but the possibility of the reversal of degeneration. The readers of *GC* could read in 1870 about the research results of Morel's pupil Doutrebente, one of whose conclusions was: "*It is beyond doubt that races can regenerate themselves* [emphasis added by *GC*], i.e., that through the influence of a harmless factor at least part of the descendants can climb to a higher position."[77] Thirty years later the same position was phrased by Van Rees in the terminology of negative inheritance: the damaged germ plasm could regenerate in the third or fourth generation if mixed with new, undamaged plasm.[78] We can therefore subscribe to some extent to the conclusion of the study of Claus Finzen on alcoholism and degeneration in the German language scientific literature around 1900: many psychiatrists gave precisely their attention to the prophylaxis and treatment of alcoholism because alcoholism was seen as the main cause of mental diseases, and work in this field seemed to hold great promises of success.[79]

Apart from the propaganda effect, what did treatment approaches consist of? Basically, alcohol abuse, delirium tremens, and dipsomania were treated with the same approach, consisting of abstinence, diet, and exercise.[80] Once again, this was nothing new. Sir Walter Scott described

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72 Hanedoes van Almkerk, 'Alcoholisme en de houding van medici', *GC* 54 (1900), no. 10.
73 *NTvG* 37 (1901) I, 1333.
74 Broers (1886), 30.
75 *NTvG* 31 (1895) II, 714.
76 Van Deventer, 'Eenige opmerkingen over de psychiatrische behandeling van krankzinnigen', *PB* 6 (1888), 27–28.
77 'Erfelijke krankzinnigheid', *GC* 24 (1870), no. 10.
78 Van Rees (1902).
79 Finzen (1977), 31.
80 Broers (1886), 30; Spaink (1892), 71.

in 1830 a medical treatment of a patient with alcoholism resulting in delirium tremens: "a gentle course of medicine (...) retire to [the patient's] own house in the country, observe a temperate diet and early hours, practicing regular exercise, (...) avoiding fatigue."[81] We can safely assume that this method would have been in use for centuries. The historian W.F. Bynum has concluded on 19th century medical approaches: "(…) alcoholism and alcohol–related problems could be treated by relatively simple measures like a wholesome diet and complete abstinence from alcoholic beverages. The alcoholic on occasion could be reformed and returned to society, hence the prognosis, even if often perceived to be bleak, was not so grave as that of many [other] asylum patients."[82] Furthermore, the second half of the 19th century witnessed extensive experiments and interventions with psychopharmacological medication. In these fifty years an astounding collection of drugs were tried out, especially to fight the effects of delirium tremens. In the 1850s, opium is still the chief medication in cases of delirium tremens and withdrawal symptoms. The idea is to artificially induce sleep, during which the "anomalies of the brain and nervous system" will automatically disappear.[83] Opium therefore assisted the body in curing itself. Another drug in use was digitalis. Later in the century chloral hydrate, morphine, lupuline (in the 1870s), strychnine (in the 1880s), and other medication are in use, including purgatives and nausea cures (in which all food and drink are dosed with alcohol). Continuously more or less favourable reports are published in the medical journals.[84]

The great problem for physicians was not a lack of (more or less) effective treatment methods. The great problem was that the generally accepted *best* treatment method required total abstinence and a retreat from daily life to recover and cure. But most actual or potential patients did not have their own country house to effect their cure. In 1891 a special asylum for inebriates, Hoog–Hullen in Eelde, was opened, directed by a medical supervisor.[85] But neither this initiative nor changing thought about heredity led to significant changes in the overall medical approaches to alcoholism before 1900. Far from being helpless against alcoholism in its various forms, the physician of the second half of the 19th century had an impressive armoury of methods to help and cure his patients. Of course there are no reliable figures on the efficacy of these methods, but there was no cause for *a priori* therapeutic pessimism, nor did conceptualizations around heredity lead to that consequence.

6. Conclusions

The Dutch medical journals of the second half of the 19th century show that doctors in the Netherlands integrated transforming knowledge around heredity in a flexible and fluid way in their conceptualizations and approaches of alcoholism. On a conceptual level, their goal seems not to have been theoretical consistency with scientific knowledge production, but its amendment and adjustment in the construction of workable explanatory tools. Morelian degeneration, neo–

[81] Scott (2001), 19. On British medical approaches to alcoholism in the 18th and early 19th centuries: Porter, 'The drinking man's disease'.
[82] Bynum (1985), 63.
[83] *NTvG* 1 (1857), 355, 594.
[84] E.g., *GC* 24 (1870), no. 3; *GC* 32 (1878), no. 1; *GC* 33 (1879), no. 26; *GC* 34 (1880), no. 41; *NTvG* 20 (1884) I, 203.
[85] Van der Stel (1995), 189–194.

Lamarckian inheritance of acquired characteristics, Darwinian evolution, or Weissmannian poisoning of the germ plasm could and were all used to produce these tools. They could be fitted to explain doctor's experiences of phenomena of hereditary degeneration, of plastic expression of predispositions, and even of the possibilities for hereditary regeneration. In short, the transfer of knowledge from the scientific to the medical sphere was by no means hierarchically downward.

On the level of approaches, knowledges of heredity did not connect to a therapeutic pessimism. The fight against alcoholism, based on a plastic concept of heredity, and incorporating elastic and pragmatic treatment and prevention practices, was offering hope for individual cures, as well as being an instrument in the long–term regeneration of the population. We must be hesitant to characterize public health strategies connected to heredity around 1900 as primarily concerned with collectives or the 'race', and not with individuals.

Although not static, the medical domain in the Netherlands before 1900 does not come under the grip of a hardening hereditarianism. How Bismarck the tomcat would have fared under medical supervision in the subsequent decades will have to become clear in follow–up research.

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Abbreviations in footnotes:

GB: Geneeskundige Bladen uit Kliniek en Laboratorium voor de praktijk

GC: Geneeskundige Courant voor het Koninkrijk der Nederlanden

NTvG: Nederlandsch Tijdschrift voor Geneeskunde

PB: Psychiatrische Bladen

PNB: Psychiatrische en Neurologische Bladen

TSH: Tijdschrift voor Sociale Hygiëne en Openbare Gezondheidsleraar

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A Changing Landscape in the Medical Geography of 'Hereditary' Disease: Syphilis, Leprosy, and Tuberculosis in Hawai'i (1863-1903)

Philip K. Wilson

It has been argued that during the second half of the 19th century a 'hardening' occurred in beliefs about the nature of heredity.[1] 'Inherited' disease had long been viewed as the result of both nature and nurture. Appropriately nurturing against the hereditary predisposition to a disease could, it was argued, diminish the likelihood of the disease ever being expressed. This longstanding 'soft hereditarian' view of disease was later eclipsed by more 'hard hereditarian' claims that inherited disease was solely dependent upon a non-malleable nature.

The current quest for better understanding the hereditary nature of particular diseases is hardly new.[2] Considerable attention has been directed towards the meaning of heredity in relation to disease within the context of Europe, England, and the United States. However, the extent to which these beliefs changed elsewhere around the globe and whether this change was concurrent with that in Euro-America has been little explored. This paper begins to uncover some nineteenth-century viewpoints about 'hereditary' disease in a different location – Hawai'i.

Remote though it is, Hawai'i's potential as a critical colonial outpost garnered the attention of Germany, England, France, and the United States during the nineteenth century. European explorers had opened new vistas to the eyes and to the mind, portraying their newly found 'specimens' as savages in primitive cultures. Ethnographers busily concocted labels of new races and varieties of humans within newly updated classification schemes. Missionaries focused upon all of the Polynesian Islanders as prime targets for Christianization. Governments envisioned these islands as colonial outposts which, when acquired, would strengthen their fortunes and futures. Euro-American Imperialism consequently provoked many native population groups to reinforce their respective cultural beliefs and traditions, including their beliefs and practices related to health and disease.

Of the many Pacific islands ethnographically grouped at the Polynesian Islands within the triangulated zone of Hawai'i, New Zealand, and Easter Island, a cultural heredity-mindedness was particular apparent in three of them: Tahiti, Tonga, and Hawai'i. With particular interests in the disease patterns and healing traditions from the Native Hawaiian cultural perspective, I am interested in examining notions of heredity in relation to disease within this particular island group. My particular goal is to ascertain the extent to which views of the heritability of disease in Hawai'i conformed with the general 'hardening' of hereditarian views expressed in the Euro-American medical writings during the second half of the nineteenth century. Adding the perspective of heredity from earlier Hawaiian perspectives will add a further dimension into our worldview of beliefs about the inheritance of disease.

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1 Carlos López-Beltrán (1994) described that a malleable view existed in the "soft hereditarianism" beliefs of the early nineteenth century in contrast to the more objective qualifications of a nature-based, "hard hereditarianism" later in the century.

2 For example, Wilson (2003a and 2002a).

To begin reassessing just what natives and newcomers may have viewed as "hereditary" regarding both native and foreign disorders suffered by people in Hawai'i during the nineteenth century, the existing literature must be questioned anew. Primary source material is crucial. The nineteenth-century published medical and public health writings, ethnographical accounts, scientific expedition reports, and travelogue memoirs help to capture contemporary anthropological perspectives of the Hawaiian people. These writings supplement the native perspectives from cultural luminaries including David Malo, Samuel Manaiakalani Kamakau, and John Papa Ii that were compiled at the time and are now available in English translation.

Introduction

Hawai'i faced a number of significant disease outbreaks during the 1800s. Venereal disease began devastating the native population soon after it arrived via James Cook's sailors and continued, despite missionary attempts of Christian cleansing, to ravage Hawai'i for many years. The *Okuu* (probably what Westerners' viewed as Asiatic cholera) ravaged Hawai'i beginning in 1804.[3] Measles was introduced to the islands in 1848. Smallpox arrived in Oahu in 1853 claiming at least 5000 lives, then returned in force in the early 1880s.[4] Tuberculosis became endemic among the Hawaiian island populations during the nineteenth century, just as it did across the globe. And leprosy, possibly first identified in Hawai'i as early as 1819 by members of a French scientific expedition, later became a disorder that troubled not only the afflicted, but also their families as it precipitated a legal segregation and isolation of the diseased to Kalawao (and later Kalaupapa) on Moloka'i. Those who manifested signs of the leprous taint were gathered and hauled off to the settlement at Kalaupapa in the name of quarantine – for life. Finally, what has been characterized as the bubonic plague swept through the islands in 1899, taking a further toll upon the inhabitants at the close of the century.

The "infectious" diseases mentioned above were frequently described in the 19th century by Euro-American physicians, missionary settlers, expeditionary voyagers, and itinerant travelers – all *haole* (white foreigners), *hapa* (part) *haole*, or "*Hawaiianized haole*" – who provided critical insights into these diseases from various perspectives.[5]

However, most of the scholarship to date has yet to cover the entire spectrum of illness and disease as experienced in nineteenth century Hawai'i. Indeed, I have noticed little mention of "inherited" or "hereditary" disease in recent writings about the Hawaiian *kânaka* (people) of the 1800s.[6] This paucity of information is puzzling. For when one peruses nineteenth-century Euro-American medical writings and patient narratives, one finds that disease – or at least the propensity to disease – was commonly viewed as being hereditarily transmitted. This claim is true

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3 Schmitt (1970).

4 Greer (1969).

5 We owe much of our historical contextualization of these emerging infectious diseases of nineteenth-century Hawai'i to the scrupulous research and widely read writings of O.A. Bushnell and C. S. Judd. Their devotion to advancing our understanding and treatment of disease in Hawai'i, both past and present, was unprecedented. Their intellectual approach to explaining the birth and significance of new diseases has been replicated in the work of others, including Diamond (1996) and Igler (2004).

6 See, for example, Green and Beckwith (1926), Larson (1962/63), and Blaisdell (1989).

even for the so-called "infectious" diseases mentioned above. It is also puzzling in that among the Polynesians, the Hawaiian islanders were a hereditary conscious group.

Pedigrees, in the form of epic genealogical chants have long been a part of Hawaiian culture. Kinship was, in the nineteenth century, an important concern for the *kânaka maoli* or *'ôiwi* (people of the bone) as well as for the *ali'i* (ruling families). Hawaiians had established families of ruling chiefs as a class distinct from common islanders.[7] But the Hawaiian *kânaka* (people) became increasingly worried throughout the nineteenth century about the repeated threats to the hereditary rule of the islands. Ascribing to a belief that the "nation lived while his [Kamehameha's] descendants lived and ruled," both natives and rulers remained focused upon the struggle to maintain this hereditary rule. Thus, thinking about the possible hereditary nature of diseases was, to some extent, a natural extension of the hereditary-mindedness among the *kânaka maoli.*

A complete reassessment of hereditary disease within Hawaiian heritage is, however, beyond the scope of this paper. For now, I want to focus upon some ideas about heredity in Hawaiian history regarding three particular diseases common among Hawaiians and Euro-Americans during this period: syphilis, leprosy, and tuberculosis. Particular attention will be devoted to the timing and the extent to which hereditary conceptualizations of these diseases may have been modified by the Germ Theory, a new explanation of disease that gained considerable support in at least some parts of Europe and the United States during the second half of the nineteenth century.

Prior to turning to each of these diseases, it may be helpful to identify, using a few examples, several traditional Hawaiian cultural beliefs concerning heredity, healing, and disease.

Traditional Hawaiian beliefs concerning heredity and disease

Inheritance has been a traditional concern among native Hawaiians in regards to health and disease. In Hawai'i, ancestry appears to have been as important for the pedigree of the healer as for that of the sufferer. For example, Kamakau recounted that only if the *kahuna lâ'au lapa'au* (medical healer) is "an upright person" – one who is pure and clean of person and deed, who "obeys the laws of that land as well as those of the *akua* (god or goddess)," – only then will he be "guided properly by true revelations of his spirit guides" in that the "secret things of his ancestors" will be revealed to him.[8]

Knowledge that certain illnesses were "inherited in a family through generations," was a common belief to all *kânaka maoli* (native Hawaiians). The "secret things" that medical *kâhuna* received from their ancestors included wisdom about treating diseases that the an *'aumakua* (deified ancestor) or a *kumupa'a* (ancestral deity) passed along hereditarily. These diseases were known to be "very resistant." They did not "respond to [regular] medicines" and there were "not many *kâhuna* who are able to work on [them]." Healing "could not be done by outsiders or strangers because their voices and appeals would not be heeded by the *'aumakua*." Since these diseases had been introduced through familial lines, it was thought to be more "the work of the family" than of the *kâhuna* in overcoming them.[9]

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7 Goldman (1970), p. 234.
8 Kamakau (1964), p. 95.
9 Kamakau (1964), p. 97.

Newborn babies were generally inspected by a "*kahuna* of predispositions" (*kahuna pa'ao'ao*). If a baby was found to be ill, it was imperative for the *kahuna pa'ao'ao* to ascertain whether or not it was "an ailment that afflicted the ancestors, coming down from them and afflicting the parents and then the child." For if such ailments were not promptly and properly treated, the child was likely to suffer from "a more severe ailment" in adulthood.[10] Untreated hereditary predispositions were believed to hold the capacity for changing into adult diseases including generalized states of emaciation, localized swellings, or specific disorders including tuberculosis and dysentery.

Hereditary diseases represented a particular grouping of disorders that were commonly understood around the world as being capable of manifesting themselves throughout many generations. However, within Hawaiian cultural belief, a hereditary disease could be manifested for all future generations. Since a "relationship" had been forged "between *'aumakua* and descendants," the Hawaiian people became "actual children (*keiki ponoi*) of the gods." Therefore, a hereditary influence over illness and disease held the potential to last forever.[11]

However, the meaning of "forever" in regards to inheritance in Hawai'i was challenged during the later nineteenth century as islanders faced repeated threats to the hereditary rule of the islands. After the death of Hawai'i's ruler, Kamehameha IV (Liholiho), in 1863 until the defeat of the Home Rule Party in 1902, Hawai'i encountered turbulent confrontations by outsiders regarding the hereditary succession of its rulers.

It was also during this period that many new claims were being tested in Hawai'i as to the inheritance of particular diseases. Therefore, focusing upon this time frame of threat upon the Hawaiian belief of hereditary rule, I will look briefly at the meaning of heredity in regard to what have been deemed as Hawai'i's "destructive trinity of diseases": syphilis, leprosy, and tuberculosis.[12]

Syphilis

It could be easily assumed from standard historical accounts that describe syphilis arriving *de novo* on the Sandwich Islands with Captain James Cook's men in 1778, and its quick and wide-spread devastation, that contemporaries viewed syphilis as a contagious, infectious disease. Such a statement, however, is only part of the story. Taking a broader perspective on this disease and the Hawaiian people offers new insights into the history of Hawai'i during the 19th century in terms of island rule, the new-found wealth of sugar, immigration related to agricultural needs, concerns of 'the other' consequent to immigration, the changing demography of the island populations, and – most critical to our interests – changes in how people in Hawai'i understood the etiology of this island scourge.

William Ellis, the surgeon's second mate aboard Cook's ship, HMS Discovery, conjectured that it was only since the arrival of his ship that the island natives had become diseased "in a violent degree." He identified European voyagers as the underlying "cause of this irreparable injury."[13] Alonzo Chapin, one of a series of nine missionary physicians sent to serve the medical needs of the

[10] Kamakau (1964), pp. 101-102.
[11] Kamakau (1964), p. 99.
[12] Mouritz (1916), p. 58.

Hawaiians, claimed in 1838 – a half-century after Cook's men established contact between the Hawaiians and "the other" – that

> Those, who have the credit of the discovery of the islands, and of exhibiting first to the astonished gaze of the simple and ignorant natives, some of the ingenious and useful implements and commodities of enlightened lands, and who sailed ships so enormous in size as to have been regarded as floating islands, inhabited by supernatural beings, must also receive credit of having introduced among these islanders . . . the vilest and most loathsome disease ever sent as a punishment for transgression.[14]

Chapin, in remarks of 1850, cited syphilis as "the most prominent cause of the decrease in population" of Hawaiian natives.[15] Though census tabulations of this era leave room for speculation as to the precise population counts, there is general agreement among scholars that the number of native Hawaiian declined sharply between 1778 and the second half of the nineteenth century.[16] While the Hawaiians "died off," what one literary scholar described as a "strange cosmopolitan society of Chinese, Japanese, Portuguese, half-breed and Americans sprouted in their stead."[17] Many native Hawaiians hurled scorn against the largest immigrant population at mid-century, Chinese men in particular, as being "unchaste" and "whose cohabitation with Hawaiian women was doing nothing but contributing to their [the Hawaiian] continued infertility."[18]

This so-called "sliding way of death" prompted considerable reaction.[19] On one level, the government expressed concern to "cease or at least limit" the importation of Chinese men.[20] Such efforts, however, were also seen by many authoritative figures as counterproductive to the need of new plantation labor as sugar increasingly became the unofficial, corporate King of the Islands. Another King, Kamehameha IV, worked on another level to develop public health measures against syphilis. "Our first duty is that of self-preservation," the King claimed. "Our acts are in vain

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13 Ellis (1782), pp. 73-74, as cited by Stannard (1990), p. 329. For other broad analyses of Cook's impact upon the island from an infectious diseases perspective, see Stannard (1989), A. Bushnell (1993), and O.A. Bushnell (1993). Ralston (1984) also provides a helpful overview of key cultural changes within this time period.

14 Chapin (1838), reprinted excerpts, Halford (1954), p. 291. These nine physicians were Thomas Holman, Abraham Bletchley, Gerrit Judd, Dwight Baldwin, Alonzo Chapin, Thomas Laton, Seth Andrews, James Smith and C.H. Wetmore.

15 Chapin (1850), p. 93. Views expressed by many foreigners who traveled to or were at least temporarily relocated in the islands was consistent with that of many activists promoting Hawai'i's reclamation of sovereignty. This view is also apparent in blanket statements by historians who claim, the "white man's presence was killing the Hawaiians." Daws (1973), p. 74.

16 State statistician Robert C. Schmitt (1968) provided a descriptive analysis of census taking. His numbers show a decline of the percentage of "native" Hawaiians among the people on the Hawaiian islands from 95.8 % in 1853 to 28.5 % in 1896. In identifying what contemporaries as well as later scholars viewed to be the likely causes for this decline, Schmitt (1968), p.159, that the "role of syphilis has been mentioned frequently." Haunani-Kay Trask (1993), University of Hawai'i Professor of Hawaiian Studies and an outspoken advocate for Hawaiian sovereignty, argues that an even more drastic decline occurred beginning from a considerably larger initial native Hawaiian population, pp. 6-7. A revised edition was published in 1999 by the University of Hawaii Press.

17 Carré (1930), p. 237.

18 Osorio (2002), p. 174.

19 Judd (1977) attributed the use of this term to Ozzie Bushnell, p. 593.

20 Osorio (2002), p. 174.

unless we can stay the wasting hand that is destroying our people." Consequently, the Queen's Hospital was opened in 1859, devoted primarily to care "for sick and indigent Hawaiians," in which a syphilitic ward was opened within its first year.[21]

But what of heredity and syphilis? Infection, regardless of its source, was not the only cause associated with the spread of syphilis.[22] On one level, discussion of hereditary syphilis was propagated among islanders through the medical textbooks that Western-trained missionary physicians added to their collection while working on the islands.[23]

Physicians during this era diagnosed syphilis based upon the visualization of signs. Hereditary syphilis often appeared in newborns as a particular constellation of signs.[24] It was also these visible manifestations that foreign visitors "inspected" and noted in their written characterizations of syphilis amongst the Hawaiians. But to the Hawaiians, the signs of syphilis held meanings beyond that routinely noted in the medical literature.

As noted earlier, syphilis had been particularly tied to the arrival of Captain Cook. According to Hawaiian cultural belief, Cook personified the white god Lono. Lono had once been present among the Hawaiians, but had gone away. Cook's appearance was commonly touted as Lono's return.[25] Upon Cook's arrival, Hawaiian's claimed, "Now our bones shall live" because "our *ʻaumakua* (ancestral spirit) has returned."[26] Within the genealogical tradition of Hawaiian gods and priests, Cook's own death had been a foretold event. According to some contemporary accounts, Cook's (Lono's) revenge or wrath for his death would be delivered via later personifications of Lono who, in turn, were connected to Cook through their godly or priestly inheritance. Accordingly, some Hawaiians perceived that the syphilis spreading amongst them was the physical manifestation of Cook's revenge being delivered unto them through the hereditary lines of the god Lono.

Some Hawaiians explained this hereditary transference or transmission of disease in terms of *mana*. *Mana*, according to one leading structural anthropologist of Hawaiian culture, was "the creative power" that Hawaiians describe as "making visible what is invisible, causing things to be seen, which is the same as making them known or giving them form."[27] Views attributing disease to supernatural, spiritual causes were consistent with the aims of the *kahuna lâʻau lapaʻau* (medical healer) and the Hawaiian missionary. The spirits underlying disease would, Hawaiian

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21 Halford (1954), p. 238.
22 Wilson (2003b), pp. 18-21.
23 Such nineteenth-century textbooks, according to Halford (1954), pp. 131-132, included Benjamin Bell's *Treatise on the Venereal Disease*, Alphée Cazenave's *On Cutaneous Diseases*, René Theophile Hyacinthe Laennecs' *On Diseases of the Chest*, M-F. Xavier Bichat's *Epitome of Physiology, Anatomy, and Pathology*, Samuel Solomon's *Guide to Health*, John Armstrong's *On Fevers*, François Magendie's *Physiology*, and John Mason Good's *Study of Medicine*. It may be helpful to note that the distinctions twenty-first-century medical writers draw between the terms "hereditary" and "congenital" as applied to disease were not so clear in the nineteenth century. See Wilson (2003b), pp. 18-19.
24 For an over view of the importance of "reading" marked children during this era, see Wilson (2002b).
25 In *How Natives Think: About Captain Cook, For Example*, cultural anthropologist Marshall Sahlins (1995) elaborately analyzed this view of Cook as Lono, defending his interpretation against that postulated by Gananath Obeyesekere (1992). Ozzie Bushnell has also incorporated the Hawaiian view of Cook as Lono in his well received fictionalized historical account, *The Return of Lono: A Novel of Captain Cook's Last Voyage*, a work that that originally appeared as *Peril in Paradise* (1956).
26 Kamakau (1961), as cited by Sahlins (1981), p. 7.
27 Sahlins (1981), p. 31.

healers claimed, remain active for generations and could return along specific lines of inheritance to inflict successive members in a family's lineage.

Christian missionaries also kept the hereditary thinking about syphilis alive as they spread their word to the peoples of Hawai'i. Underlying disease, promiscuously-spread disease in particular, lay original sin. This disease, an William Bliss, one nineteenth-century traveler to the Islands noted, represented the "iniquity of the parents . . . visited upon the children, even to the third and fourth generations."[28] Christianizing the islands would, the missionaries hoped, cleanse the people by blocking this inheritance of original sin. Such reform would, it was argued, sway the trend away from the demise of the native population. With this frame of mind, missionaries – and missionary physicians – focused particularly on Christianizing the *kua'âina* (rural folk in the hills and valleys) whom they perceived had been "beyond [the] reach of procurers and poxed foreigners," who had "refused to tolerate intimacies with foreigners," and who had "no taint of venereal diseases. For it was the *kua'âina*, the missionaries claimed, that held great potential for the "preservation of the [native] race."[29]

Alcoholism has, as I have argued elsewhere, been one of the "bad habits" frequently associated with the spread of syphilis.[30] Alcoholism was also a disorder that, throughout the nineteenth century, was often claimed to have a hereditary etiology.[31] This view, common in Euro-America, also gained support among the Hawaiians. Lunalilo, while prince and king, was known to be "constantly tempted to indulge his inborn craving for drink," in that he was "lacking the constitution" to resist.[32] The apparent high incidence of alcoholism in Hawai'i was also exacerbated by environmental factors. For instance, many *kânaka maoli* in the nineteenth century who grew increasingly concerned about the depopulation of the native population and the threat of the complete disappearance of the "Hawaiian race" appear to have frequently turned to alcohol to palliate their concerns.

By the early twentieth century, heredity was still supported in etiological thinking about syphilis in prominent Western writings. Even the Don of medical authorities, Sir William Osler, argued that cleansing "parents before marriage" remained the "most certain prophylaxis" against the hereditary spread of syphilis – that "most tragic form of the disease."[33]

Missionaries, physicians and native Hawaiians agreed that, uncontrolled syphilis would continue its wrath through its hereditary spread to future generations. Uncontrolled, syphilis was also thought to be able to transform or metastasize into another disease – leprosy.[34]

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28 Bliss (1873), p. 76.
29 Halford (1954), p. 198.
30 Wilson (2003b).
31 Bynum (1984). In Wilson (2003a), I have demonstrated that Dr. Erasmus Darwin, grandfather of the naturalist Charles Darwin, was a leading advocate for temperance and documented many pedigrees of intemperance (i.e., alcoholism) in the early 1800s that ran through English families – including his own.
32 Gordon-Cumming (1883), vol. 2, p. 224.
33 Osler and McCrae (1915), vol. 2, p. 200.
34 George L. Fitch, physician to the Kalaupapa leper settlement from 1882 to 1884 widely endorsed this view. Other physicians who spent time in Hawai'i – as did others across the globe – noted the great difficulty in differentiating signs of leprosy from those of syphilis. Samuel Kneeland (1873), p. 405, depicted leprosy as "inseparably mixed with syphilis." Frank Enders (1877), p. 719, viewed leprosy "not as a disease *sui generis* but [as] an offspring of syphilis." He reaffirmed this view, p. 719, citing the beliefs of other physicians that "the eradication of syphilis from these [Hawaiian] Islands, would eventually cause the disappearance of leprosy."

Leprosy

Leprosy, a disease known for centuries throughout Europe, became a major health concern closely intertwined with Hawaiians during the mid 19th century. As Nathaniel Emerson, the physician son of a Hawaiian missionary, observed, the "roots" of leprosy are "very deep and are intricately interwoven with the whole fabric of the community."[35]

Native Hawaiians came to believe that they held some special susceptibility to leprosy that was not seen in the *haoles* living on the islands. According to one government physician, George Fitch, the *haole* has "acquired a kind of hereditary immunity to leprosy as a result of centuries of exposure in Europe."[36] Even the licentious *haole*, who readily contracted syphilis were deemed to be "protected" from what Fitch viewed as the "fourth stage of its consequence" – leprosy.

Missionary physician, Dwight Baldwin, noted its presence on the Hawaiian island of Maui as early as 1840. However, the Berlin-educated physician, William Hillebrand is typically credited with having noted its first appearance in Honolulu in 1848. The introduction of this "fresh item of the infinite curse which has come upon this [Hawaiian] race," was blamed, by many, upon the Chinese.[37] Indeed, it soon became colloquially knows as *ma'i Pâkç* (the Chinese disease).

The afflictions of leprosy became well known to people on all the Hawaiian islands.[38] Its manifestations were remarkable. One well known piece of historical fiction recounts an 1893 episode on meeting lepers. The lepers were

> monsters – in face and form grotesque caricatures of everything human. They were hideously maimed and distorted, and had the seeming of creatures that had been racked in millenniums of hell. Their hands, when they possessed them, were like harpy-claws. Their faces were the misfits and slips, crushed and bruised by some mad god at play in the machinery of life. Here and there were features which the mad god had smeared half away, and one woman wept scalding tears from twin pits of horror, where her eyes had once been. Some were in pain and groaned from their chests. Others coughed, making sounds like the tearing of tissue. [Some] were idiots, more like huge apes marred in the making, until even the ape were an angel. They mowed and gibbered in the moonlight, under crowns of drooping, golden blossoms. One, whose bloated ear-lobe flapped like a fan upon his shoulder, caught up a gorgeous flower of orange and scarlet and with it decorated the monstrous ear that flip-flapped with his every movement.[39]

For a decade after its first description in Honolulu, leprosy was hardly mentioned in the growing number of widely read travel accounts of Hawai'i. Mark Twain, for example, omitted any reference to leprosy in his well known *Letters From Hawai'i* because, according to historian A.

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35 Nathaniel Emerson to Rudolph Meyer, January 3, 1889, as cited by Daws (1973), p. 78. Insightful overviews of leprosy within the Hawaiian peoples are found in many works, including Stoddard (1895), Thompson (1897), Mouritz (1916), Weymouth (1938), esp. pp. 153-183, Farrow (1954), Wellman (1968), O.A. Bushnell (1963, 1968), Kalisch (1973), esp. pp. 500-501, Judd (1984), Gussow (1989), Veith (1992), pp. 300, 302, 304, Merwin (1998), Castillo (1992), and Inglis (Forthcoming).

36 Daws (1973), p. 133.

37 Bishop (1966), p. 223.

38 Kalisch (1973), p. 501.

39 London (1914 edition), pp. 50-51. Although the word "leper" connotes strong stigmatization and disrespect today, I have maintained its usage throughout this paper when I believe that it is important to contextualize nineteenth-century vernacular.

Grove Day, Twain "did not wish to frighten off the business men who would be his most important readers."[40]

However, among the people of Hawai'i, both native and *haole*, leprosy hardly held a threat to visiting foreigners. Although the mode of its transmission was unclear, most initially viewed that it did not spread in a contagious manner. Writings of the period strongly suggest that physicians and Hawaiian deemed that leprosy was "somehow transmitted along hereditary lines,"[41] for the "leprous taint" was observed to run "strong in many families."[42]

Such a view was consistent with the concurrent claims half-way across the globe in the leprosy asylums in Norway. There, Daniel Cornelius Danielssen and Carl Wilhelm Boeck had concluded from their studies that leprosy was passed along familial lines.[43] This view was also consistent with the Hawaiian missionaries' Biblical account of inheriting leprosy as a curse. The curse of Elisha seemed to be "irrevocably fixed" upon the Hawaiian just as the leprosy of Naaman was cursed to "cleave unto thee and unto thy seed forever."[44] Yet within this framework of thinking, another Biblical message resonated within the Hawaiian population. For similar to the treatment of lepers in the Biblical era, measures were soon undertaken in Hawai'i to segregate the lepers from the "clean." To briefly review this action, we turn again to Dr. Hillebrand.

William Hillebrand remains a complex figure in the Hawaiian history of the 1860s. Regarding leprosy, he seems to have served several masters. It was Hillebrand who initially claimed that the leprosy in Hawai'i owed its importation to the Chinese "coolie" labor force. Such medical concerns, however, did not prevent him, in 1865, from acting, as the King's appointed Commissioner, to procure additional coolie plantation labor. That same year, Hillebrand prompted the Legislative Assembly to establish a leprosy Hospital and Detention Center in Kalihi that would help to better identify and contain the lepers in Honolulu. Once detected, the lepers were, according to the 1865 segregation "Act to Prevent the Spread of Leprosy," sent off to the newly acquired governmental land at Kalawao, on the eastern side of the Hawaiian island of Moloka'i.

The history and lore of the lepers at Kalawao, and later after the transfer of this settlement to Kalaupapa on the northern side of Moloka'i has been retold – frequently embellished – in much Hawaiian history and literature. Most common is the tale of Father Damien, a Belgian Catholic priest of the Sacred Hearts, who arrived at Kalaupapa in 1873 to offer spiritual and physical care for several hundred lepers secluded there, and who, eleven years later, was diagnosed with and subsequently died from leprosy.[45] However, Kalaupapa also represented a cornerstone for the study of a reputed hereditary disease.

Leprosy was, as noted above, known to afflict members of different generations within one family, but curiously this pattern of transmission appeared only in some families – even when looking at just the native Hawaiian community. Such transmission could, of course, have been explained by contagious spread among members of a household that often included several

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40 Day (1977), p. 65.
41 Daws (1973), p. 6.
42 Bishop (1966), p. 223.
43 Rokstad (1964), p. 65.
44 Bliss (1873), p. 97.
45 Emmett Cahill (1990) offers a photo journalistic overview of the history of this settlement.

generations of family members living closely together. However, more often than not, leprosy appeared only in a few individuals of one family's generation, sparing most of the family members, all of whom had been in relatively similar contact with each other. Moreover, many *kôkua* (caregivers) voluntarily worked amongst the lepers in the isolated community at Kalaupapa, yet they never contracted leprosy. Was some form of protection from this disease hereditarily transmitted? Or, was it a weakened physical constitution among the afflicted that had been passed along family lines?

It initially startled hereditarian thinking when news reached Hawai'i from Norway regarding Gerhard Henrik Armauer Hansen's 1873 isolation of rod-shaped bodies routinely observed microscopically in the mass of bacteria present in nodules of leprous patients.[46] However, neither Hansen nor his contemporaries could demonstrate a clear causal relationship by inoculating "clean" animals or humans with material taken from a leprous nodule. Thus, Hansen's findings prompted little change of thinking in Hawai'i – at least initially.

Leprosy was becoming a "national blight" for Hawai'i. Forty-eight Protestant ministers, both Hawaiian and *haole*, who gathered at the 1873 Hawaiian Evangelical Association meeting conjectured that "our Hawaiian people will become in a very few years, a nation of lepers."[47] Indeed, even members of a later generation within a leprous family were at times among those sequestered and sent off to Kalaupapa. The Kalaupapa containment had not reduced leprosy's spread on the other islands. Based upon this information, the Hawai'i Legislative Assembly concluded in 1873 that leprosy was not a contagious concern, and they attempted to reverse their earlier decision and vowed to send no more lepers to Moloka'i.[48] However, commercial interests prevailed as other authoritative figures within the Hawaiian community wished to keep lepers out of sight from the *malihini* (foreign observers) visiting Honolulu. These *malihini* were, after all, the observers whose future financial investments would continue to bolster the economic growth of the islands.

In 1882, according to Harvard Professor of Dermatology, James C. White, the medical world had "almost universally" adopted the opinion that "leprosy is not contagious, and that it is endemic mostly because it is hereditary."[49] Although most medical authorities agreed with White's claim, emphasizing that it continued to "appear in the descendents of . . . [particular] families," others argued that this finding alone "proves nothing *a priori*, for the . . . continuance [of a disease] among relations may [also] be used . . . as the best evidence of its communicability by contagion."[50]

Alas, this "unquestionable doctrine" continued to be questioned. The "important point to be determined," they argued, is "the proof of [contagion], not the dis[proof] of [heredity]." Maintaining this secluded population of lepers allowed physicians during the last two decades of the nineteenth century to use Kalaupapa as an experimental study site from which they hoped to

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46 Hansen's initial article (1874) is available for English readers as ""Causes of Leprosy" (1955). See also Feldman (1965), pp. 412-416. In the twentieth century, leprosy was officially renamed "Hansen's Disease."
47 Daws (1973), p. 63.
48 Osoro (2002), p. 177.
49 White (1882), p. 435.
50 White (1882), p. 435.

ascertain the precise hereditary underpinning of leprosy. Hawai'i provided the case study site whereby the "recent introduction of leprosy into an insular nation" and the sequestration of most of those with the diseases into a further isolated community provided "that virgin field for observation so essential for the proper study" of the transmission of leprosy.[51]

Much was at stake over the Kalaupapa findings regarding leprosy. On one level, marriageability between lepers as well as between individuals from 'clean' and 'unclean' families depended upon these observers' conclusions. On another level, the entire usefulness of the settlement was in question. One figure at the center of this question was George Fitch. It was Fitch who, in 1882, had become Hawai'i's greatest proponent of drawing the interconnections between leprosy and syphilis. Leprosy, he argued, was the fourth stage of syphilis. His view, according to one observer, became "quite a popular topic of discussion amongst the laity and the medical fraternity in Honolulu."[52] It also exacerbated the already present diagnostical dilemma that existed between these two disorders.

Fitch drew upon the findings at Kalaupapa as well as authoritative reports from around the globe for his 1884 Report to the Board of Health. He construed the information in this report as thoroughly supportive of a hereditary transmission of leprosy. "There appears no more need . . . for restricting the liberty of lepers" by sequestering them in perpetual quarantine at Kalaupapa than for "restricting the liberty of those with the gout." Segregation, he continued, "except in so far as it prevents [the] hereditary transmission of the disease has absolutely no effect towards checking it."[53]

Going against the tide, though unsupported in his belief, was Harvard dermatologist James White. His view was based upon the observation that just as in the case of the "syphilization" of Hawai'i, leprosy appeared far too quickly for it to be exclusively caused by heredity. For if heredity was acting as the only factor, he argued, "it would have required several generations to have accomplished such results."[54] Additional support that this was certainly not the case in Hawai'i, some surmised, came from the finding that no word existed in the Hawaiian language for this disease. Nor could, as Morell Mackenzie argued, "ancestral proclivity" alone explain the "sudden outbreak of the diseases [across the globe] in races [that were previously] altogether free of it."[55] Also going against the tide were the Hawai'i-based physicians, Eduard Christian Arning and Arthur Mouritz. Arning, a highly skilled bacteriologist, arrived in Honolulu in late 1883 from Germany. Mouritz, an Oxford-educated physician, came to Hawai'i and served as a resident physician and Medical Superintendent to the Leper Settlement at Kalaupapa from 1884 to 1887.

Arning acknowledged that a "disposition" to leprosy, a "certain weakness to resist its attacks," "may possibly be transmitted by heredity," but he remained firm that "leprosy itself was not congenital."[56] In 1884, he designed an experiment that would, he argued, prove once and for all, that leprosy could also spread via contagion. This involved the inoculation of a condemned

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51 White (1882), p. 435.
52 Mouritz (1916), p. 54.
53 Cited in Mouritz (1916), pp. 384, 396.
54 White (1882), p. 438.
55 Mackenzie (1889), p. 938. For an elaborate contemporary description of specific global sites affected by the recurrence of leprosy, see Tebb (1893).
56 Arning 14 November 1885 report to Gibson, cited in Mouritz (1916), p. 326.

prisoner, Keanu, whose sentence the Hawai'i Privy Council commuted from hanging to life imprisonment so that he might serve as a subject for Arning's experiment. Keanu, a strong man who showed no signs of leprosy or other illnesses, had the leproma (a leprous filled swelling) that had been removed from a young leper colony resident and sutured over an incised site on his forearm. Arning removed material from Keanu's body near the inoculation site for a number of months. To his great disappointment, no bacterial signs of leprosy were found nor did Keanu develop any visible signs of leprosy before Arning returned to Europe in 1886.

The resident Kalaupapa physician, Mourtiz, watched over Keanu after Arning's departure and, a year later, diagnosed him as a "confirmed leper." Arning presented this case at the First Congress of the Society of German Dermatologists held in Berlin in 1889. Shortly thereafter, however, doubts over Arning's findings surfaced. Leprosy was, it was noted, endemic in Hawai'i, and, as Mouritz argued, some of Keanu's relatives were also known to be inflicted.[57] Moreover, Mouritz had not succeeded in any of over a hundred similar attempts to inoculate healthy Hawaiians with the disease.[58]

Arning's later writings show more than disappointment. Although he had once viewed Hawai'i as a natural laboratory for the study of leprosy, he now saw it much differently. For in Hawai'i, "the intense feeling which everything connected with leprosy necessarily evokes in so small and terribly afflicted community, cannot favour the slow and tedious process of purely scientific work." And, no small obstacle in and of itself, there one also had to contend with the "character of the natives."[59]

Concern mounted on all sides of leprosy's etiology for the remainder of the century. Foreign dignitaries and visitors, such as M.G. Bosseront d'Anglade, Commissioner and Consul of France, noted in 1893 that there are "very few native families who are not affected" with leprosy – both the disease and its stigmatization – as well as by the separation of their family members to Moloka'i. In his travelogue, he admitted his inability to absolutely ascertain whether leprosy was contagious. The "combination of two conditions appears necessary for leprosy to develop," he concluded. First, "prolonged contact with other affected persons," and second, "individual susceptibility to disease." The susceptibility, he argues, is seemingly inherited. He cited an example of the inherited passage of this susceptibility in the case of a "healthy Kanaka man who during ten years at the [leprosy] settlement had successively married four women patients, [and] begotten leprous children by each of his wives, and yet never gave any indication that he himself had become a leper."[60] Thus, for many, as for Bosseront d'Anglade, the ambivalence over what precisely guided leprosy's spread kept the matter controversial for some time.

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57 Keanu's familiar linkage to this disease was also raised by another resident physician at Moloka'i, Sidney Bourne Swift. Tebb (1893), pp. 127-128.

58 Daws (1973), pp. 142, 234-235. For an overview of the attempts to inoculate humans with leprosy, see Dubois (1952).

59 Arning to Nathaniel Emerson, 10 May 1886, as cited by Daws (1973), p. 278. Keanu was retained at Moloka'i, a "punishment," according to California University pathologist, D.W. Montgomery, that was "ten times more severe than the death penalty. Physician William Jelly concurred, noting "had [Keanu] known what leprosy is, [he] would without hesitation, have preferred the guillotine, the garrote, or the hangman's noose" to his fate in Kalaupapa. Tebb (1893), pp. 124-135.

60 Bosseront d'Anglade (1987).

It should be noted that by the century's end, we do find heredity being eclipsed by contagion as the chief explanation for Hawai'i's leprous population. However, this turnabout resulted more from politics than from medicine. New immigration channels would be opened if Hawai'i was formally annexed to the United States. Fear of exporting leprosy together with coolie labor loomed, reaching unprecedented levels in California in 1897.[61] When, as New York public health authority Prince A. Morrow argued, it is considered that "more than ten percent of the Hawaiian race are affected with leprosy," he conjectured that "it becomes a serious question as to what will be the effect of the absorption of this tainted population upon the health interests" of the United States if the islands are annexed to them.[62] The Hawaiians themselves are not the real problem, he argued, for each of them is "essentially insular in his tastes and habits and shows little disposition to leave his native shores." Rather, it is the labor forces who, having interbred with the native Hawaiians, are the likely ones to carry the "seeds of . . . [this] deadly contagion [i.e., leprosy]" with them when imported to the Western shores of the United States.[63] "All of these facts," he concluded, "should be carefully considered and their importance from a sanitary point of view carefully weighed by our legislative authorities before deciding upon the [U.S.] annexation of Hawaii with its leprous population."[64]

Hawaiian sovereignty came to a close soon after the U.S. Congress's passage of an act to Annex the Hawaiian Islands to the United States on July 7, 1898. Political turmoil had begun with the overthrow of the Hawaiian Constitutional Monarchy in 1893 and the self-designated provisional government to oversee the new U.S. protectorate. It remained strong through the time that pro-expansionist U.S. President, William McKinley ushered in the annexation bill. "Among the early results of annexation," the physician Burnside Foster argued, will "undoubtedly be a largely increased immigration to as well as emigration from the islands." Many who "either know or suspect that they have [leprosy] will undoubtedly escape to this country while those from this country who settle in Hawaii will be thrown into more or less intimate relations with the already infected but unrecognized lepers." Thus, it would "certainly seem worthwhile," Foster continued, "for the United States to take this question immediately at hand, and to appoint a commission of bacteriologists properly equipped and with every facility for the study of the leprous problem. England has gained the eternal gratitude of humanity for her Jenner and her Lister, France for her Pasteur, Germany for her Robert Koch. Shall not America . . . gain further glory by striking leprosy from the calendar of human afflictions?"[65]

Tuberculosis

The rise of bacteriological thinking in the nineteenth century, promulgated primarily via the work of Robert Koch, diverted the medical gaze, focusing it more upon germs than germ cells. Tuberculosis, one of the most common diseases of the nineteenth century, is among the most critical emerging infectious diseases across the globe in our era. To combat medicine's diminished

[61] Gussow and Tracey (1970), p. 439.
[62] Morrow (1897), p. 582.
[63] Morrow (1897), p. 588.
[64] Morrow (1897), p. 590.
[65] Foster (1898), p. 305.

stronghold over tuberculosis, current investigations focus upon uncovering changes in the genetic evolutionary processes between the tubercular microbe and anti-microbial agents as well as identifying inheritable alterations that have influenced the germ's pathogenicity. Curiously enough, in the nineteenth century, the heredity of tuberculosis was also being investigated. But unlike the present focus upon the etiological agent itself, earlier genetics studies were inferred from tuberculosis' transmission along family lines.

Divergent thoughts existed surrounding the hereditary nature of tuberculosis during the 1800s. Medical authorities who purported that this "white plague" was hereditary offered as evidence its frequent appearance in multiple members and successive generations of the same family. Some argued that "from the beginning of medical time it was considered that tuberculosis ran in families; that heredity had much to do with the occurrence of the disease."[66] Within the early nineteenth century, we find a number of prominent European, British, and American physicians endorsing its hereditary basis. Antoine Portal "maintained categorically" that tuberculosis was "hereditary and had nothing to do with contagion." Gaspard Laurent Bayle argued that heredity was the "chief aetiological factor" underlying this disease. René-Théophile-Hyacinthe Laënnec, whose innovation of the stethoscope was targeted towards the study of respiratory diseases acknowledged that although tuberculosis "appeared to be contagious is some countries, this did not seem to be the case in France" – his homeland. Gabriel Andral, William Cullen, Sir John Forbes and Sir James Clark also supported this theory.[67]

Prominent Boston medical authority, Henry I. Bowditch wrote to a general reading audience in 1869 that "Undoubtedly it is true that public opinion considers consumption [i.e., tuberculosis] as hereditary, and medical experience seems to support this view. We presume that there is scarcely a physician anywhere who would not admit the truth of this belief." Yet, he adds, "no physician would dare to say that . . . consumption would *necessarily* be transmitted from parent to child."[68]

However, Robert Koch's 1882 discovery of a bacillary germ as what he deemed the etiological agent responsible for tuberculosis provoked serious rethinking about this disease among the medical community and the general populace. According to biomathematician and eugenicist Karl Pearson's later reflections, following Koch's discovery, the "idea of infection dominated" and consequently, an "immediate neglect" arose regarding any reputed "hereditary factor" underlying this disease.[69]

Although it is true that Koch's discovery "immediately" spread throughout the globe, we are only beginning to fully appreciate that his pronouncement did not immediately convert all 'hereditarians' into supporters of the germ theory.[70] Indeed, many physicians, especially older physicians, remained skeptical of any germ-based etiology, and they continued to practice believing tuberculosis to be hereditarily transmitted.[71] When confronted with findings suggesting

66 Lewis (1923), vol. 1, p. 178.
67 Keers (1978), p. 53, reviewed these proponents' beliefs. Additional supporters of a hereditary passage of tuberculosis are found in Castiglioni (1933). For a helpful outline of the changing perceptions of this disease, see Burke (1955).
68 Bowditch (1869) as reprinted in Rosenkrantz (1994), p. 63.
69 Pearson (1912), p. 3.
70 Worboys (2001) and Carter (2003), p. 196.
71 Ott (1996), p. 128. For more on the persistence of hereditarian thoughts, see Wilson (Forthcoming).

that they disregard heredity as the primary cause of tuberculosis, they typically replied with something like the following argument.

> How can we account for the cases where the parents, having died of Consumption, the children are necessarily attacked on arriving at a certain age, with a severe type of the disease? And, moreover, there are several instances . . . where the children, who happened to be scattered in various parts of the world, were yet attacked and succumbed to the fell disease at almost the same age?[72]

However, the hereditarians were divided over precisely what they deemed to be heritable regarding tuberculosis. Whereas a large faction had once argued that "the disease" itself was inherited, many physicians began to assert during the late 1800s that it was something about this disease that was inherited. In other words, a hereditary tendency, predisposition, or diathesis to disease existed.[73] Diathesis, the term commonly used at this time, referred to the "inherent liability to consumption which 'ran in families' and was handed down from one generation to another."[74] Indeed, the extent to which the germ theory predominated medical thought regarding tuberculosis during this period remains unresolved.

In Hawai'i, we find it present in successive generations of missionaries, natives, and chiefs.[75] There, tuberculosis "vied with syphilis" as the "singularly most remarked upon disease . . . during the first half of the nineteenth century."[76] However, as the century progressed, tuberculosis, part of what Mouritz characterized as Hawai'i's "destructive trinity of diseases" (together with syphilis and leprosy), had come to overshadow the other two such that it remained in "almost full possession of the field."[77]

When we look to Hawai'i during the two decades following Koch's discovery, we find the most vocal support for a hereditary transmission as being the primary explanation of tuberculosis among the island population. Most typically, the discussions closely followed descriptions of leprosy.

The connection between these two diseases was not merely coincidental, for considerable clinical conundrums arose because leprosy, at least in its early forms, mimicked the signs of tuberculosis. Some physicians considered the "tuberculosa" or "tuberculous condition" that formed on the face, legs, arms, and trunk of lepers as affiliated with an inward pulmonary tuberculosis.[78] However, over time, the manifestations of leprosy became more distinguishable, and it did not involve the lungs.[79]

Tuberculosis also held a particular relationship with syphilis on the islands. It was widely held that an individual demonstrating "defects of development" or having parents whose diseases

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72 Williams (1882), p. 618.
73 Ackerknecht (1982).
74 "Tuberculosis" (1911), vol. 27, p. 357.
75 Middleton (1971), p. 452.
76 Stannard (1990), p. 342.
77 Mouritz (1916), p. 58. This finding is remarkable considering that in the mid-1800s, several physicians noted the Hawaiian's "entire exemption from pulmonary tuberculosis." Gulick (1855), p. 197.
78 Kneeland (1873), pp. 404-405, and Tryon (1883), p. 447.
79 Although sanitaria were developed on four of Hawaiian Islands, tubercular patients admitted themselves voluntarily. They never suffered the stigmatization commonly felt among the leprous patients.

"stamp[ed] sufficient weakness upon the[ir] offspring" frequently gave rise to children with a diminished resistance to tuberculosis. Prominent among such parental-derived diseases was syphilis.[80]

And again, as seen in beliefs about syphilis and leprosy, those determined to Christianize the Hawaiian islands integrated Biblical wisdom with medical reports. Dr. Bowditch provided Hawai'i's missionaries considerable support through popularizing tuberculosis as follows.

> It is often seen that the "diseased conditions of the parents, sometimes, alas! Due to their own or to their ancestors' previous excesses, [create] tender bodies of [newborn] children . . . so tainted that life becomes a burden. We have often seen in such [tuberculosis] cases the terrible vindication of the power of the old Mosaic law, 'For the sins of the fathers are visited upon the children . . . unto the third and fourth generations.'"[81]

One additional connection may be gleaned regarding tuberculosis and inheritance in the Hawaiian islands. King Lunalilo's tuberculosis brought an end to his reign in 1874, only thirteen months after he had been elected King by the Hawaiians upon the death of Kamehameha IV. Between the time of his death and the U.S.'s takeover of control of the Hawaiian islands, the hereditary lineage of rulers successively lost ground. Interestingly, it was over this same time that the germ theory fought to overtake hereditary beliefs in the transmission of diseases. Perhaps the end of hereditary rule in Hawai'i parallels, in ways that have yet to be uncovered, the transference of power from the hereditarians to the germ theorists regarding disease.

Hawaiian highlights expanding our perspective of hereditary disease

Perhaps Hawai'i is marginalized and remote to most cultural worldviews, both then and now. Still, this reflection upon a seemingly remote Hawaiian culture during the last half of the nineteenth century reminds us that in the views of some folk and physicians, "heredity" and "inheritance" as related to disease were, at times, quite distinct from concurrent views expressed in Euro-Ameican writings. For example, long before Westerners sought to identify the hereditary substance within humans, Hawaiians already envisioned a hereditary substance, *mana*, (spiritual power), as a contributory factor to health and disease. If, for example, the flow of *mana* was disrupted, disease would likely follow. Spirits, ghosts, wraiths and astral bodies are ancestral, and thus, as Hawaiian heritage reminds us, their influence must be considered in discerning a culture's view of heredity as an etiological factor of disease.

Similar beliefs are found and may be contextualized within other Polynesian Island cultures. Anne E. Becker analyzed Fijian culture, noting that the spirit, often ancestral, is often attributed as the cause of an illness.[82] Although Fiji lies just outside of the typical island range denoted as the Polynesian group, it has long been populated with a large percentage of Polynesians whose belief systems have come to influence Fijian culture.

If visual detection was important for a culture to identify the presence of a hereditary disorder, then the sufferer must be visible. If, however, we follow the suggestions presented in some early

[80] Osler and McCrae (1915), vol. 1, p. 317.
[81] Bowditch ([1869] 1994), p. 65.
[82] Becker (1995).

nineteenth-century writings, we are left uncertain as to whether these diseased individuals were actually kept alive for long. "Deformed, sluggish, or ill-tempered babies were unceremoniously put out of the way," it has been argued. Children with "deformities and trying illnesses of one sort or another," many of which could have been brought on through inheritance, were frequently disposed of quickly or "placed out of sight and sound . . . and allowed to languish and waste away."[83] Despite what some modern readers may think of such practices, if indeed they were as common as nineteenth-century authors lead us to believe, we should also appreciate that such practices were condoned within the moral and religious framework of native Hawai'i at that time and discuss them within that historical context.[84]

Expanding research into these and related areas will allow us to recapture more about the entire spectrum of illness and health as viewed by Hawai'i's *ka po'e kahiko* (the people of old). Such views can complement and strengthen the Western-centered narratives that have dominated decades of the telling of the Hawaiian past were applied to states of disease within different cultural perspectives across the globe. Such efforts will allow us to better contextualize the significant prior contributions regarding "infectious" and "contagious" diseases within Polynesia with an even broader worldview of disease and history.

This focus upon Hawai'i exemplifies our need to be aware that specific cultural contexts drive specific and precise meanings into such polysemic words as "inheritance" and "heredity." It also helps us appreciate the need to consider comparative cultural views as we conceptualize the meaning that heredity held during earlier periods. It remains to be determined just how contemporaries of the nineteenth century viewed immigration and racial mixing as altering, perhaps exacerbating, hereditary influences upon disease within these new mixes of peoples in Hawai'i. This area of inquiry will be covered in my next foray into heredity, disease, and Hawai'i as I look at Hawai'i through the eyes and minds of early 20th-century Anglo-American eugenicists.

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[83] Lee (1966), pp. 172, 178.

[84] Tobin (1994). Cultural anthropologist George M. Foster (1976) provided timeless reminders to historians of the need to be ever cognizant of the comparative ethnographical contexts when pursuing historical work regarding "other" cultures.

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How Cultural Is Heritage?
Humanity's Black Sheep from Charles Darwin to Jack London

Marianne Sommer

Charles Darwin's *black sheep* of my title was one of several near-synonyms of what was frequently referred to as *atavism*. Other related concepts were *reversion (to type)* or *throwback*, and *arrest of development* or *stagnation*. It seems that reversion was an important concept within plant breeding to refer to the reappearance of traits of the wild type.[1] Used by evolutionists such as Herbert Spencer (1820-1903), Charles Darwin (1809-82), and Ernst Haeckel (1834-1919), atavism meant the complete or partial reversion of a plant or animal to an ancestral state. However, at a time when the mechanisms of heredity were unknown, concepts such as atavism had to remain equally diffuse. In addition, when applied to humans they became colored by socio-cultural attitudes of the time.

Clearly, atavism was about questions of heredity from the start. Yet, possibly indicative of a general indeterminacy, the concepts of reversion and stagnation were not restricted to any particular theory of heredity or human evolution, associated with an emphasis on the so-called Lamarckian mechanism of the inheritance of acquired characteristics or Darwinian spontaneous variation and natural selection. Rather, atavisms regarded as caused by either of the two processes are often difficult to tell apart.

For example, in a 'Lamarckian' framework, it was possible to see an impoverished environment as the cause of regression or stagnation, which in effect looked similar to the 'Darwinian' notion that an environment in which there were no selective pressures, or the wrong ones, might lead to regression or stagnation. In fact, within non-scientific discourses, the latter was sometimes seen as being possible within one individual.[2]

However, in my talk I have argued that reversion and arrest were intimately related to recapitulation theory, the belief that the individual in its embryonic development repeats stages its species passed during evolution. Michael Hagner has therefore asked the fundamental question of why atavism did not come to preoccupy non-biological discourses earlier, at the time of the recapitulationism of *Naturphilosophen* and transcendentalists such as Lorenz Oken, J. F. Meckel, and Etienne Serres.[3] Indeed, as has been pointed out by Jonathan Harwood, the concept of atavism works particularly well within a linear-developmental view of evolution. At this point, I may only offer a few tentative remarks on the subject, deferring a more detailed discussion to a future opportunity.

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1 The plant-breeding origin of the concept of reversion was stressed by Roger Wood. Where atavism is concerned, an article in *Scientific Gardening* of 1833 referred to Joseph Duchesne (1549-1609), when explaining atavism as designating the phenomenon that children often resemble one of their grandparents more than their parents (*OED* online, etymology).

2 Even so, the difference between 'hereditary' and 'acquired' atavism became increasingly important for example in connection with criminality and homosexuality (e.g. Nye, 1976, who deals with acquired and inborn criminality; Williams, 1990, who discusses acquired versus congenital homosexuality; Pizer, 1961, who sees Frank Norris' Vandover as acquired and McTeague as hereditary atavism (see below)).

3 On these see for example Gould, 1977, pp. 39-52; Ospovat, 1976.

Obviously, the notion of atavism was bound up with evolutionism, which in the Anglophone world mainly under concern here did not come to dominate either scientific or popular discourses until after Darwin's *On the Origin of Species* (1859). At that time, a new kind of recapitulationism, in accordance with Darwinian evolutionism, became widespread. However, as I discuss elsewhere, the tree-like structures that came to be proposed as representations of phylogenies did not in all cases completely turn away from a linear sequence. This seems to have been particularly true for anthropology, where the acceptance of human antiquity and the turn towards evolutionism added the parameters of time and development to a racial hierarchy already in place in physical anthropology.

Incorporating the new insights from comparative ethnology and prehistoric archaeology, an inevitable series of ever higher cultural and anatomical stages came to be seen as mandatory passages for all human races and civilizations in their evolution. This progression from savage to civilized, or primitive to modern, was analogized to individual embryonic development. As a consequence, fossil and living 'non-white races', children, the insane, etc., were interpreted as occupying stages of the human evolution of mind and body which Western man had passed in his ascent to the apex of nature and culture. The 'savage races' were understood as both simultaneously, offshoots of the line leading to the modern 'civilized races' and stages through which the latter had passed in their evolution. They were projected back in time, so that a *scala naturae* structure was essentially maintained.[4]

Thus, evolutionary progressionism had been accompanied by the counter-concepts of reversion and stagnation from the beginning. However, towards the end of the century, the general self-assuredness was increasingly troubled by a fear of regression and degeneration, both of which could signify the return to a primitive state.[5] This was motivated among other things by the notion that struggle was essential to progress, combined with the observation that growing sections of Western societies were excluded from the fight for survival – a threat epitomized in such dandy and effeminate figures as Oscar Wilde.[6]

In the latter half of the nineteenth century until well into the twentieth, atavisms and their close relatives came to haunt philosophical, literary, sociological, and psychological discourses as the shady side of the progressionist paradigm. The wider cultural appeal of the biological concepts might exactly have been enhanced by their insufficient differentiation and imprecise definition within an evolutionary biology uncertain about the underlying mechanisms of hereditary continuity and change. They may have been symptomatic of a growing uncertainty, their pervasiveness signifying an attempt at ostracism and control over what was perceived as the non- or anti-progressive. While in this sense they seem to have functioned as an instrument for securing the status quo, atavism was also employed as a way of conceptualizing the duality of the human being – heir to animal instincts and cultured mind.

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4 Sommer, 2005.

5 Within a cyclic model of history, in which senility signified a return to an infantile condition, both degeneration and regression could indeed indicate a return to an ontological or phylogenetic earlier and lower stage.

6 On progressionism and fears of degeneration see for example Bowler, 1989, 1993. For one of the most instructive documents of the fin-de-siècle culture and its concerns see Nordau, 1968 (1895).

The atavistic disposition could therefore also be perceived as lurking within everyone. Somewhat paradoxically, the belief that anyone of us may fall victim to partial or full atavism was sometimes accompanied by a more positive interpretation of the phenomenon. Indeed, atavistic traits or events could become instruments of self-discovery, a means of integrating the individual with the common racial heritage, or of integrating the altruistic and civilized with the more selfish and animal aspects of personhood; a trend that would culminate in the new psychology at the dawn of the twentieth century.

Although this is already a longer version of the talk I gave in Berlin, the paper offers no thorough discussion of atavism and similar concepts. Most of the questions that have been raised during the workshop have to remain unanswered for the time being. A closer analysis of the relation between degeneration and regression, as well as of the development of the concept of atavism over time in the contexts of progressionism, imperialism, theories of racial supremacy, growing nationalism, fear of miscegenation, eugenics, Mendelism and the new genetics, etc., will have to be put aside.

Indeed, as Manfred Laublicher has pointed out, within a framework of hard heredity and Mendelian laws of inheritance, the meaning of atavistic traits and individuals must have changed considerably. However, these new insights were not incorporated into evolutionary anthropology until well into the twentieth century and neo-Lamarckian use-inheritance remained a favorite explanation for change. Also popular discourses in America as well as Britain drew from diverse expressions of evolutionism.[7]

The paper represents work-in-progress and the above attempt at a rough contextualization is meant as an invitation to continue the discussion initiated in Berlin. The paper aims at reconstructing hypothetical passages along which the concepts under concern might have traveled for one particular example – the American fiction writer, journalist, and adventurer Jack London (1876-1916). With the birth of modernism, old and new political, artistic, philosophical, and scientific ideas clashed. London felt caught in-between and struggled against paradoxes in life and writing. He appears to have seen in the notion of atavism a conceptual tool for overcoming opposites of old and new; first and foremost within the human being.

1. Books as tools of the trade: The production of atavisms in Jack London's laboratory

To identify some of the ideas that converged in the notion of atavism, I will approach the subject through London's prehistoric fiction novel *Before Adam*, and discuss some of the possible scientific and non-scientific influences on its creation. This approach lends itself to an attempt at a wider cultural understanding of atavism for two reasons. The first is due to London's writing practices. London referred to his farm at Glen Ellen, California, where he lived and worked, as a "writing laboratory", in which his more than 15'000 books served as "tools". London read and wrote according to a strict schedule. He had a sophisticated ordering system for his notes, references, newspaper clippings, correspondence, etc. However, the books he bought and that

[7] E.g. Bowler, 1986, pp. 190-198; Oldroyd, 1980. For a critique of the biological notion of atavism see Montagu, 1938, 1945.

were sent to him by far overextended the space of his library and colonized the house, including the barn.

London's laboratory, like those of so many of the scientists he read, functioned as a hub, where objects and information from all over the world were accumulated, analyzed, and synthesized into something new. London himself was an excessive reader, but also an adventurer and explorer, undertaking 'field research' at the places where his stories would be situated. It must be ascribed to London's passion for sailing that he used the following analogy when explaining the function of his books as tools of his trade:

> I regard books in my library in much the same way that a sea captain regards the charts in his chart-room. It is manifestly impossible for a sea captain to carry in his head the memory of all the reefs, rocks, [...] harbors, points, lighthouses [...] of all the coasts of all the world; and no sea captain ever endeavors to store his head with such a mass of knowledge. What he does is to know his way about in the chart-room, and when he picks up a new coast, he takes out the proper chart and has immediate access to all information about that new coast. So it should be with books.[8]

And London, in good white-suprematist fashion, conquered many new intellectual coasts, such as evolution, psychology, political economy, travel, navigation, philosophy, drama, poetry, and fiction. His claim to factuality, one suspects, was influenced by the American and French naturalists he studied, first and foremost Émile Zola (1840-1902). Indeed, when accused of plagiarism from *The Story of Ab* (1897) by Stanley Waterloo, London self-confidently replied that *Before Adam* presented rather something of a reply, as *The Story of Ab* was unscientific.[9] It is therefore possible to ask what kinds of tools London could have used in his laboratory in the creation of his supposedly scientific *Before Adam*. In doing so, I will limit myself to three of the coasts London explored, evolutionary biology, literature, and psychology.

The book's preoccupation with atavisms and arrests of development constitutes the second reason for choosing this approach. The novel was first published as a series in *Everybody's Magazine* from October 1906 to February 1907 (Figure 1). The main story evolves around three stages of hominization present at the same time in the Mid-Pleistocene. The Tree People are closest to modern anthropoid apes and as their name suggests, they live in trees. The Folk are physically and culturally more advanced than the Tree People. They are in the process of descending from the trees, have less body hair, are capable of rudimentary communication, and have tools. While the members of the Folk are more intelligent but less strong than the Tree People, they also lack the foresight and purpose of the Fire People, who are anatomically modern humans.

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8 Letter to *The North American*, 30 March 1913, reproduced in Hamilton, 1986, pp. 1-2. David Mike Hamilton, 1986, has investigated and catalogued London's library and the annotations found in books. While many of the books I will discuss in this paper are explicitly mentioned by Hamilton, in other cases it is only known for certain that London was acquainted with the author.

9 Hamilton, 1986, p. 26.

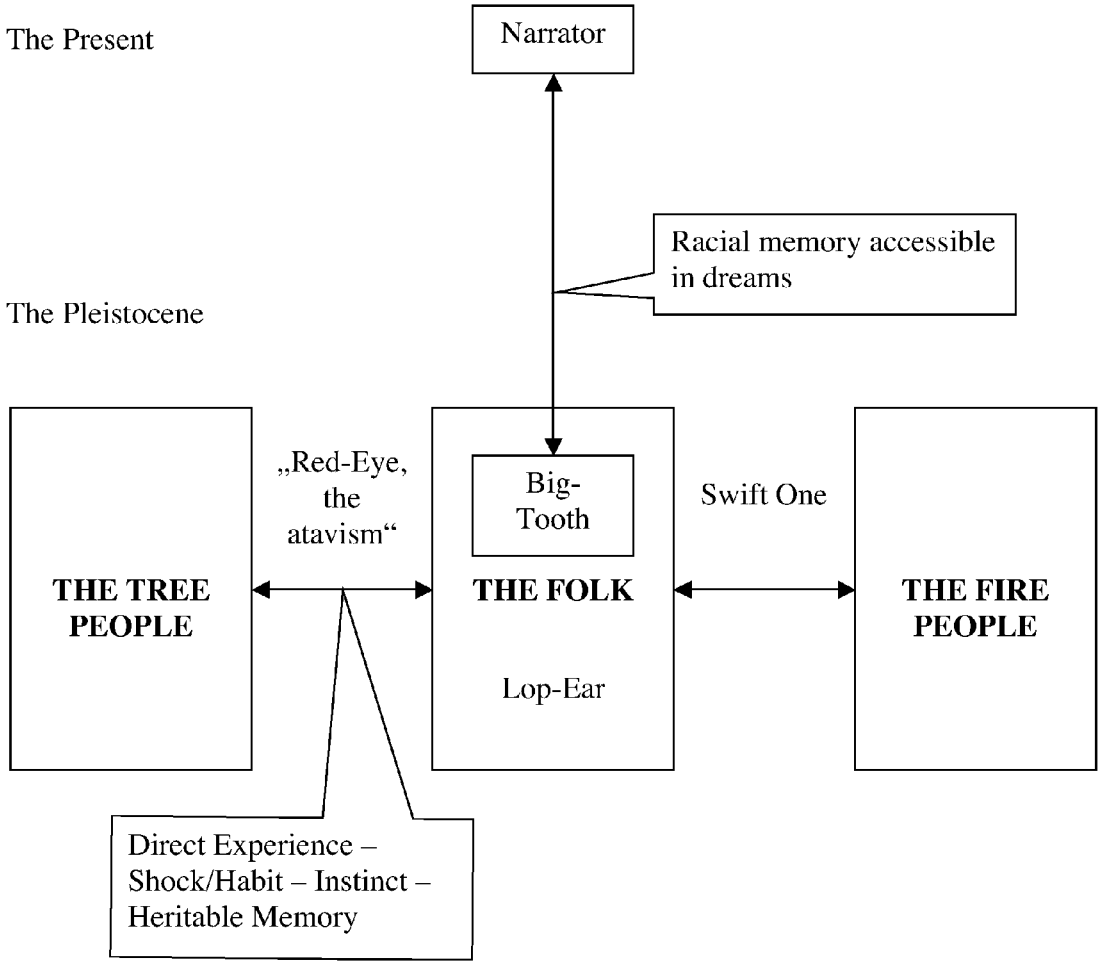

Figure 1: The Structure of Jack London's *Before Adam* (1906-1907)

The three stages of human evolution are no truly independent side-branches of the human family tree; rather, the less progressive groups are cases of arrest of development, or stagnation. However, the fact that London had three different stages of human evolution live simultaneously would not have looked so odd to the contemporary reader. Evolutionists in general conceptualized 'the lower human races' as having stagnated at points which represented evolutionary stages in the white man's prehistory.[10] Similarly, more primitive fossil hominids were often interpreted as arrests of development in undemanding environments at the peripheries of the earth.[11] Like the contemporary 'savages' of London's time, the Tree People and Folk are 'living fossils'.[12]

[10] Stocking, 1982, Ch. 6.
[11] Sommer, 2005.
[12] London, 2000 (1906-1907), e.g. pp. 171, 224-225. In the story of *Before Adam*, London also described the prehistoric equivalent to the colonization and marginalization of 'savages' by 'civilized Europeans' underway at his time. The Folk are gradually being displaced and extinguished by the Fire People, who due to their increase in numbers have to gain new territories.

Furthermore, the three groups are interconnected by missing links such as "Red-Eye, the atavism". Although Red-Eye is one of the Folk, he represents a throwback to the earlier stage of human evolution occupied by the Tree People.[13] Finally, the world of the Pleistocene hominids is accessed through the dreams of a man of London's time. The narrator's dreams about the life of his prehistoric ancestor Big-Tooth are resurfacing chunks of racial memory. In a way then, the narrator, too, is an atavism; one might say a *psychological atavism*: "[…] some strains of germplasm carry an excessive freightage of memories – are, to be scientific, more atavistic than other strains; and such a strain is mine."[14]

2. Tools from biology: The concepts of atavism/regression/reversion and arrest/stagnation

There can be no doubt that books on evolution played a central role in the making of London's atavisms and evolutionary stagnations. By 1900, London had studied Spencer, Darwin, Thomas Henry Huxley (1825-95), and Haeckel among others.[15] In the evolutionary theory of the Prussian biologist Haeckel, the so-called Biogenetic Law took center stage, according to which an individual's ontogeny was essentially the shortened and accelerated repetition of its phylogeny.[16] Thus, in its embryonic development, the individual recapitulated the paleontological history of its phylum. This is of relevance, because as I will show the notions of atavism and stagnation were intimately related to the belief in recapitulation theory.

With the spread of the Darwinian theory, the view of ontogeny of the Prussian-Estonian embryologist Carl Ernst von Baer (1792-1876) as a process of differentiation and individuation was applied to the view of evolution as another system of divergent development.[17] Thereby, the ideal *(Naturphilosophie)* and/or non-evolutionary (Cuvier, Owen, von Baer) notion of archetypes of taxonomic groups such as fish, reptiles, birds, and mammals were turned into real common progenitors, even if their fossil bones had not yet been found.[18] Darwin, too, integrated von Baerian embryology and a view of evolution as a process of divergence into a recapitulationist framework. Already in the notebooks of the late 1830s, Darwin embraced recapitulation theory; in *On the Origin of Species* (1859) he argued:

> As the embryonic state of each species and group of species partially shows us the structure of their less modified ancient progenitors, we can clearly see why ancient and extinct forms of life should resemble the embryos of their descendants, – our existing species […] Embryology rises greatly in interest, when we thus look at the embryo as a picture, more or less obscured, of the common parent-form of each great class of animals.[19]

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[13] Another example of a connecting link is Big-Tooth's partner the Swift One, who is related to the Fire People. Her father or mother might belong to that 'higher race'. In general, there is a certain exchange between the three stages of development (London, 2000 (1906-1907), p. 135).

[14] London, 2000 (1906-1907), p. 20.

[15] E.g. Hamilton, 1986, pp. 8, 14.

[16] E.g. Haeckel, 1898 (1868), p. 190.

[17] Baer, Carl Ernst von. *Über Entwicklungsgeschichte der Thiere. Beobachtung und Reflexion.* Königsberg, Teil 1: 1828, Teil 2: 1827.

[18] Haeckel, 1898 (1868), Vortrag 13, pp. 314-315 on the three-fold parallelism between comparative embryology, the classification system, and comparative phylogeny, all of which resulted in a tree. Spencer, too, argued for the threefold parallelism (e.g. Spencer, 1897 (1866), Part II, Ch. 2, and Part III, Ch. 5).

Thus, for the 'Darwinian bulldog' Huxley, the pronounced similarity of human and ape embryology was the strongest evidence in favor of their close kinship – too close, as we will see, for humans to be quite safe from the ape within:

> Startling as the last assertion [that it is only in the last stages of development that the young human being presents marked differences from the young ape] may appear to be, it is demonstrably true, and it alone appears to me sufficient to place beyond all doubt the structural unity of man with the rest of the animal world, and more particularly and closely with the apes.[20]

To turn now to the notion of atavism, which Haeckel defined in the *Natürliche Schöpfungsgeschichte* as an organism that represents a throwback to the state of a long lost *ancestral* generation,[21] we find a possible explanation for the phenomenon in Darwin's *The Variation of Animals and Plants Under Domestication.* At the outset of the chapter on "Reversion or Atavism", Darwin gave several terms for the phenomenon besides atavism and reversion, such as throwback, *pas-en-arrière, Rückschlag,* and *Rückschritt.*[22] Here, Darwin conjectured that in sexual reproduction, the characters of both parents would exist in the offspring in a double state, once blended and once kept apart. The usual case of character expression would be through blending of the parent variants.[23] However, there were cases where certain traits were predominant, as he called it, in which the plant or animal offspring may revert to a character thousands of generations remote: "From these several facts it must be admitted that certain characters, capacities, and instincts, may lie latent in an individual, and even in a succession of individuals, without our being able to detect the least sign of their presence."[24]

The reversion of characters could be catalyzed by a change in living conditions or by hybridization, or simply happen for no apparent reason. With regards to human races, Darwin speculated that "[…] the degraded state of so many half-castes is in part due to reversion to a primitive and savage condition, induced by the act of crossing [between two races, both low in the scale], even if mainly due to the unfavourable moral conditions under which they are generally reared."[25] Already from these considerations it seems that, particularly where human atavisms were concerned, there was neither a precise definition of reversion, nor were partial and full reversions clearly distinguished.

While so far there seems to be no necessary connection between atavism and recapitulation theory, there was such a connection for the concept of arrest of development. Darwin was well aware of the fact when he wrote in *The Descent of Man:* "[…] it is hardly credible that a complex part, arrested at an early phase of embryonic development, should go on growing so as ultimately

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19 Darwin, 1964 (1859), pp. 449-450. London, 2000 (1906-1907), p. 172, also followed the von Baerian model.

20 *Man's Place in Nature* (1863), in Huxley, 1894, p. 92. On recapitulation theory see Gould, 1977; Ospovat, 1976; Russell, 1916; on Darwin's recapitulationist ideas see for example Darwin, 1964 (1859), pp. 449-450, and Richards, 2002, pp. 526-533.

21 E.g. Haeckel, 1898 (1868), p. 186.

22 Darwin, 1998 (1883), p. 1.

23 Darwin spoke of characters "blending together" (e.g. Darwin, 1998 (1883), p. 11).

24 Darwin, 1998 (1883), p. 29. This notion seems similar to Haeckel, 1898 (1868), pp. 184-186, "unterbrochene oder latente Vererbung". Like Darwin, Haeckel thought that a return to the wild often caused an animal or plant species to revert to earlier generations.

25 Darwin, 1998 (1883), p. 21.

to perform its proper function, unless it had acquired such power during some earlier state of existence, when the present exceptional or arrested structure was normal."[26] But how could such an arrest of development then unambiguously be distinguished from an atavism?

In many cases it could not: "When a structure is arrested in its development, but still continues growing, until it closely resembles a corresponding structure in some lower and adult member of the same group, it may in one sense be considered as a case of reversion."[27] As an ontological model of evolution suggests, a more primitive phylogenetic stage might be reached either through reversion, or by stagnating in one's embryonic development. Thus, Darwin conceded that microcephalous idiots in as far as their brains resembled those of apes, like many other cases of arrest of development, might just as well be classified as reversions. Thereby both arrest of development and atavism were placed within a recapitulationist framework, which turned them into catchalls for the ape-like, the primitive, the 'savage', the childish, and the 'idiotic':

> *Arrests of Development.*– […] It will suffice for our purpose to refer to the arrested brain-development of microcephalous **idiots** […] Their skulls are smaller, and the convolutions of the brain are less complex than in normal men. The frontal sinus, or the projection over the eyebrows, is largely developed, and the jaws are prognathous to an "*effrayant*" degree; so that these idiots somewhat resemble **the lower types of mankind**. Their intelligence, and most of their mental faculties, are extremely feeble […] They often ascend stairs **on all-fours**; and are curiously fond of **climbing up furniture or trees**. We are thus reminded of the delight shewn by almost all **boys** in climbing trees […] and several cases have been published of their bodies being **remarkably hairy**.[28]

Obviously, in this discussion of the phenomenon of arrest of development, supposedly phylogenetically and ontogenetically earlier and lower stages, such as 'non-white races', children, and apes (and animals in general), were brought in to characterize the arrested or reverted state of microcephalous idiots. Again, it seems as though the atavistic trait of a microcephalous brain rendered the affected individuals atavistic in general morphology and behavior.

London's atavism in *Before Adam*, called Red-Eye, closely corresponds to the concept within evolutionary theory (Figure 2). Although Red-Eye lives among the Folk, he represents a regression to the evolutionary lower and more apish type of the Tree People: "He [Red-Eye] was a monster in all ways. Physically he was a giant […] He was abominably hairy […] Furthermore, it was a rare individual among us who balanced himself with his knuckles when walking. Such an individual was an atavism, and Red-Eye was an even greater atavism."[29] As becomes clear from both London's description and Charles Livingston Bull's (1874-1932) illustration of Red Eye in

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26 Darwin, 1874 (1871), p. 54.
27 Darwin, 1874 (1871), p. 54.
28 Darwin, 1874 (1871), pp. 52-54 (since Darwin used italics, I have marked keywords bold). Darwin based his speculations on the microcephalous condition on Carl Vogt (1817-95), who argued that fossil hominids, 'lower extant races' such as 'the Negro', and microcephalous idiots represented missing links between the living white races and the recent great apes. The microcephalous idiot was interpreted as arrest of development *(Entwicklungshemmung)*, "[…] welche eine der Stationen bezeichnet, die der menschliche Embryo nothwendig durchlaufen muss und welche durch die Mischung menschlicher und afflicher Charaktere jetzt noch in ihrer Abnormität die Zwischenbildung bezeichnet, die früher in normaler Bildung bestanden haben kann" (Vogt, 1863, p. 278).
29 London, 2000 (1906-1907), pp. 56-59.

aggressive bearing, he is marked by the primitive morphological traits enumerated by Darwin; but he also betrays a primitive mind.

Figure 2: "It was Red-Eye"; Illustration by Charles Livingston Bull, American wildlife artist, chief taxidermist at the National Museum in Washington D.C.

This is further emphasized by the introduction of yet another term, that of *the monster,* which reconnects the notion of atavism to the older view of monstrosities as the outcome of embryology gone awry, and which brings the very negative connotations to the fore. In *The Expression of the Emotions in Man and Animals,* where Darwin strongly relied on scales of mental capacity of animal-primitive/savage/insane/child-white adult male, he included lengthy opinions that expressed the belief that recapitulation theory could explain the insane. The 'idiot' and the insane were linked to a brute state of humankind dominated by lower instincts:

> Dr Maudsley, after detailing various strange animal-like traits in idiots, asks whether these are not due to the reappearance of primitive instincts – 'a faint echo from a far-distant past, testifying to a kinship which man has almost outgrown'. He adds, that as every human brain passes, in the course of its development, through the same stages as those occurring in the lower vertebrate animals, and as the brain of an idiot is in arrested condition, we may presume that it 'will manifest its most primitive functions, and no higher functions'. Dr Maudsley […] asks, whence come 'the savage snarl, the destructive disposition, the obscene language, the wild howl, the offensive habits, displayed by some of the insane? Why should a human being, deprived of his reason, ever become so brutal in character, as some do, unless he has the brute nature within him?' The question must, as it would appear, be answered in the affirmative.[30]

As in the scientific literature, where the stagnations at or reversions to earlier evolutionary stages might affect an individual in its entirety, Red-Eye's regression to ape anatomy in *Before Adam* coincides with a reversion to ape mind and behavior. Red Eye is driven by animal instincts:

> Red-Eye was an atavism. He was the great discordant element in our horde. He was more primitive than any of us. He did not belong with us, yet we were still so primitive ourselves that we were incapable of a cooperative effort strong enough to kill him or cast him out. Rude as was our social organization, he was, nevertheless, too rude to live in it. He tended always to destroy the horde by his unsocial acts. He was really a reversion to an earlier type, and his place was with the Tree People rather than with us who were in the process of becoming men.[31]

The atavism appears as the epitome of all threats to progress, and the term came to encompass all kinds of undesirables. In analogy to animal breeding, Darwin recognized the need of a society to dispose of such destructive elements as the insane and the criminal through confinement or execution. Other problems were seen as to a certain degree self-regulatory since the violent or intemperate, the restless, or the profligate tended to die prematurely, to emigrate, or to bear few children. In any case, they were not to hand on their dispositions to the next generation:

> In the breeding of domestic animals, the elimination of those individuals, though few in number, which are in any marked manner inferior, is by no means an unimportant element towards success. This especially holds good with injurious characters which tend to reappear through reversion, such as blackness in sheep; and with mankind some of the worst dispositions, which occasionally without any assignable cause make their appearance in families, may perhaps be reversions to a savage state, from which we are not removed by very many generations. This view seems indeed recognised in the common expression that such men are the black sheep of the family.[32]

Black sheep as a designation for throwbacks, reversions, atavisms, arrests, or stagnations seems more than any other to convey the multiple meanings of the phenomenon referred to. Like black sheep, atavisms were not only about physical appearance, but also alluded to mental and behavioral traits. They were the unwanted elements in society at large. As indicated by the quote from *Before Adam* above, the Folk cannot heed Darwin's advice of isolating atavistic individuals,

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30 Darwin, 1998 (1872), p. 241.
31 London, 2000 (1906-1907), p. 111.
32 Darwin, 1874 (1871), p. 211.

because they have not yet developed the high degree of sociality needed for such a common effort. As the examples from Darwin and London show, the atavism was generally driven by (even) lower instincts than his fellow creatures. Among civilized people, the atavism particularly lacked the latter's highly developed social and moral instincts. Judged by contemporary readers, Red-Eye's unsocial behavior would amount to the criminal – he even kills his own wives.

The link to the criminal anthropology of the Italian Cesare Lombroso (1838-1909), who had even reviewed Zola's *La bête humaine* (1890, *The Human Beast*) for its 'scientific accuracy' with regards to criminal types,[33] seems thus an obvious one. As is well known, for Lombroso, the stigmata of the born criminal represented atavistic features of an evolutionary past. Thus originated the belief that what was regarded as criminal in civilized societies might be normal behavior among 'savages', animals, and for children, a belief instantiated by Red-Eye, who ends up completely at ease among the apish Tree People.[34] Not surprisingly then, London kept a file on 'crime and criminals' and read Enrico Ferri's *Criminal Sociology* (1899).[35]

3. Tools from literature: Instincts as missing links between biology and culture

From the above observations it appears that for the atavism as social-misfit and criminal, instinct theory played a central role. Darwin and Spencer, particularly in the editions of *The Principles of Psychology* published after *The Descent of Man*, provided similar explanations for the evolution of social instincts and a moral sense.[36] They saw the origin of the complex social and moral instincts that distinguish humanity in lower and more common ones. They agreed that the lower instincts, which represented compound reflex actions, were the result of habitual (stimuli and associated) behaviors and thoughts that had impacted brain anatomy. Through the mechanism of the inheritance of acquired characteristics, the modifications in brain structures were passed on to the next generation.

Darwin and Spencer regarded sympathy as the basis of altruistic behaviors. Through the capacity to empathize, the individual would learn to suffer from the community's condemnation and feel happy when praised. With the possibility to reflect on one's actions conscience arose, which would strengthen the social over the selfish instincts through habit. They also agreed that group selection had catalyzed the development of the highest instincts of sociality and morality. While humans would have obeyed the social imperatives with regards to their own tribe, in a primitive state, they would not have done so towards other tribes – a quality which would not have been judged as amoral. Where they differed was in the way they thought these higher instincts had arisen in the first place. While Darwin in *The Descent of Man* saw the main factor in the gradual addition of small and spontaneous but hereditary variations to the lower instincts, Spencer's

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33 Lombroso, 1891.

34 London, 2000 (1906-1907), p. 242. See also Lombroso, 1895.

35 Lombroso's *L'Uomo delinquente* (1876) was not translated into English until 1911; but his ideas were made known through Havelock Ellis' *The Criminal* (1892) and Arthur MacDonald's *Criminology* (1893) (Pizer, 1961).

36 In *On the Origin of Species* (Darwin, 1964 (1859), Ch. 7 – case of neuter insects), Darwin seems to have ascribed more power to spontaneous variations and natural selection in the development of instincts than in *The Descent of Man*. On the other hand, Spencer increased the role of selection after the first edition of *Principles of Psychology*, so that their instinct theories converged over time (Young, 1990 (1970), pp. 186-190).

instinct theory in *The Principles of Psychology* stressed the impact of the individual's will and effort through the inheritance of acquired characteristics.[37]

Generally, that mechanism lay at the heart of Spencer's Synthetic Philosophy that advocated for a universal law of progress guiding biological, psychological, and social evolution.[38] It ensured that individual or cultural achievements would lead to the corresponding advance in anatomy and mind. Also here, an integrating factor was the von Baerian theory of embryology as a process of increasing differentiation, which Spencer regarded as analogous to an increasing distribution of labor and overall complexity in the organism and in society. Spencer's emphasis on the individual's self-determination made the existing social hierarchy inevitably a just one. By analogy, so was the hierarchy of races, since the stage of development of each race reflected its individuals' combined effort or lack thereof.[39]

The 'Spencerian' combination of the individual's self-responsibility and a ruthless struggle for survival found wide entry into literary creations. Clearly, Spencerian philosophy was part of the bedrock of London's worldview. Among London's favorite authors whose books functioned as tools of his trade were Frank Norris (1870-1902), O. Henry (William Sydney Porter 1862-1910), and Robert Louis Stevenson (1850-94), all of whom dealt with atavistic phenomena in their writings, and might have fought with the threat of regression in their real lives.[40] As in *Before Adam,* the reversions to lower types they expounded were primarily due to a loss of the particularly human instincts.

The Jekyll-Hyde case can clearly be interpreted as atavistic (*Dr. Jekyll and Mr. Hyde* 1886). Hyde is described as ape-like, of small and muscular stature, hairy, and as a troglodyte. Jekyll represents the normal state of the human being who has risen above his ape ancestry through the acquisition of among other things highly developed moral and social instincts that work as checkers of the selfish animal drives. Similar to London's Red Eye, the violent, uncontrolled, wanton, and above all criminal, Hyde is the disrupting element among the civilized circle of London gentlemen. In accordance with his animal instincts, Hyde has no conscience: "his every act and thought centered on self; drinking pleasure with bestial avidity from any degree of torture to another; relentless like a man of stone."[41]

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37 Spencer thought that the more complex the organism, the smaller the effect of natural selection. In humans, changes in morphology and behavior were therefore seen as nearly exclusively the result of use-inheritance, as natural selection was hindered from eliminating the unfit. However, between human societies, group selection was considered to be still at work (Spencer, 1897 (1866), Vol. I, Part III, Ch. 13; also note that Spencer, 1897 (1866), Vol. I, Part II, Ch. 9, explained 'spontaneous variation' as the effect of the recombination of individual direct adaptations). On instinct theory see Spencer, 1895 (1855), Vol. I, Part IV, and Vol. II, Part IX, Chs. 5-8; Darwin, 1874 (1871), Chs. 3-5.

38 Both Spencer and Haeckel criticized August Weismann (1834-1919) for his rejection of the inheritance of acquired characteristics. Haeckel saw particularly with regards to the evolution of instincts no other satisfactory explanation than that they were psychic habits turned heritable (Spencer, 1893, 1893; Haeckel, 1898 (1868), p. 192).

39 On Spencer see also Bowler, 1989, pp. 37-39, 153-154, 157-158, 194-195; 1993, pp. 65-69; Richards, 1987, Chs. 6-7; on social Darwinism in particular see Bowler, 1989 (1983), pp. 285-291, and Bowler, 1990, Ch. 10.

40 Hamilton, 1986, pp. 11-12, 256-258. Darwin himself experienced something akin to a regression to a stage of a lower aesthetic sense when he lost his capacity to enjoy Shakespeare and poetry and replaced them by easily digestible novels (see Levine, 2003, pp. 46-47).

41 Stevenson, 1981 (1886), p. 87.

Jekyll, in contrast, yearns for the approval and fears the disapproval of his peers. The Jekyll-Hyde problem seems like an aggravation of the condition of ordinary civilized man, still vessel of selfish animal instincts imperfectly controlled by a higher moral sense: "But I had voluntarily stripped myself of all those balancing instincts by which even the worst of us continues to walk with some degree of steadiness among temptations; and in my case, to be tempted, however slightly, was to fall."[42] In accordance with an evolutionary understanding, the potion Jekyll takes in the attempt to bring about a complete dissociation of the two natures, produces a Hyde-existence rid of Jekyll, but there is no Jekyll free of Hyde.[43] Jekyll is an inherently composite being, and the more he gives in to Hyde, the less there is left of the non-Hyde part of Jekyll's body as well as mind.

Norris's tragic hero in *Vandover and the Brute* (1895, 1914), too, fails in his effort to live up to the moral standard. Like London, Norris held a mix of 'Darwinian' and 'Spencerian' ideas that made him agree with the anthropological doctrine that without selection, effort, and struggle there would be stagnation or even regression. Western civilization was seen as threatened by growing subcultures that doted on comfort. Whether it was the undemanding environment in which 'the savages' stagnated or the 'lazy and effeminate' fin-de-siècle dandy cultures, the detrimental effect on mind and body was perceived as analogous.[44] Norris's Vandover, when choosing the company of prostitutes, freaks, gamblers and alcoholics over that of respectable society, automatically adapts to his new surroundings.

Rather than finding himself under different selective pressures, the atmosphere of vice is portrayed as a world bare of struggle, where men and women self-indulgently succumb to their animal instincts, giving in to regression: "His [Vandover's] intellectual parts dropped away one by one, leaving only the instincts, the blind, unreasoning impulses of the animal."[45] It is only when re-entering the productive world of business and industry that Vandover finds himself subjected to a fierce struggle demanding an effort that he is increasingly unable to live up to. The habits acquired in the late hours of the night have reduced him to the brute and have finally brought him beyond the reach of those checking agents, the attitude of society, conscience, and remorse. Through the figure of Vandover's friend, Charlie Geary, however, the reader learns that for Norris our atavistic sides were not entirely negative. The rudiments of the brute within were necessary to make one's way in the struggle for survival and success, albeit under the guidance of an intellect sharpened in competition.

But Vandover succumbs, gliding into madness and idiocy, and in the end like Jekyll literally reverts to the animal. He falls to the floor on all fours and in any other way turns into a wolf – his physical and psychic reversion are always in step. Even Vandover's environment seems to regress

42 Stevenson, 1981 (1886), p. 93.
43 E.g. Stevenson, 1981 (1886), p. 85.
44 For Spencer, for example, women, especially those of inferior rank, 'savages', and primitives possessed lower intellectual faculties due to limited experiences in an impoverished environment. However, their faculties were also physically underdeveloped as a consequence of continued under-use over many generations. On the basis of the mechanism of the inheritance of acquired characteristics, the stage of an individual's or group's mental (including intellect as well as emotions) and anatomical stage were at corresponding levels (e.g. Spencer, 1895 (1855), Vol. II, Part IX, Ch. 3). The related argument that the cessation of the cause for a certain adaptation leads to the regression of the organ or individual to the state before the cause came into effect can be found in Spencer, 1897 (1866), Vol. I, pp. 199-200.
45 Norris, 1986 (1914), p. 228.

to primeval times: "The room was small, and at some long-forgotten, almost prehistoric period had been covered with a yellowish paper, stamped with a huge pattern of flowers that looked like the flora of carboniferous strata, a pattern repeated to infinity wherever the eye turned."[46] The monotonous repetitiveness of the wallpaper seems to mirror the infinite repetition of phylogeny in each individual's ontogeny and the associated struggle against reversion.

As a last possible literary tool of London's trade, I will turn to a story by O. Henry of the title *The Atavism of John Tom Little Bear*, which was published July 1903 in the same serial in which London published *Before Adam*. In it, one encounters a more deterministic kind of atavism. In nineteenth-century evolutionism, the environment was still seen as formative for the development of a human race, but the strong adaptationism of eighteenth-century environmentalism had given way to a less flexible notion of racial variety. The differences created in the evolutionary long-run were perceived as having become hard-wired.[47] Similarly, the narrator in the story's frame claims that the 'lower races' are prone to regress to 'their primitive state' despite all attempts at civilization. Like Red-Eye, the tropical man "[wi]ll be happiest in his own way."[48] To convince his guest of the futility of education in the case of 'the savage', the narrator tells the story of the educated Cherokee John Tom Little Bear, who had apparently completely assimilated white American culture, but who in a moment of crisis relapsed to his original state of primitive Indian.

When a boy and his mother, for both of whom Little Bear deeply cares, are threatened, Little Bear, aided by the brutalizing force of liquor, regresses to 'the subhuman ways of the savage', becomes criminal, by murdering the dangerous man and taking his scalp as a trophy. At this turning-point, this atavistic moment, he re-assumes the behavior as well as looks of the Cherokee. He even looses his polished English and reverts to the Creole. When the narrator and Little Bear wake up the following morning, the narrator is happy to see "the nineteenth century" in the Indian's eyes again. Although firewater may have played its part, Little Bear adds: "Combined […] with the interesting little physiological shake-up known as reversion to type. I remember now."[49]

O. Henry's deeply cynical farce renders it dramatically obvious how the fall back to a lower stage of development was perceived as a fall back in time. As indicated in this last quote, it was therefore associated with the process of remembering, just as progress was associated with the process of learning. This leads on to London's last atavism in *Before Adam*, the psychological peculiarity of the narrator.

4. Tools from psychology: Dreams as psychological regression to racial memory

In "A Biographical Sketch of an Infant" (1877), Darwin introduced the notion of a phylogenetic memory on the basis of a recapitulationist interpretation of mental development. At the experience that his son was afraid of the animals of prey at the zoo without ever having had the chance to learn that fear, Darwin speculated: "May we not suspect that the vague but very real fears of children, which are quite independent of experience, are the inherited effects of real dangers and abject superstitions during ancient savage times?"[50] The notion of traces of experiences

[46] Norris, 1986 (1914), pp. 234-235.
[47] Stocking, 1982, pp. 42-68, 110-132; see also footnote 44.
[48] O'Henry, 1903.
[49] O'Henry, 1903.

transferred from a prehistoric past was central for the structure of *Before Adam*, and London described an almost identical instant in the childhood of the narrator, when he is taken by his father to the circus and instinctively fears the lion, even though London took the concept one step further from instinct-like and unconscious memory to conscious remembrance:

> Nevertheless, it was with fear and trembling, and with much encouragement on his [the father's] part, that I at last approached the lion's cage. Ah, I knew him on the instant. The beast! The terrible one! And on my inner vision flashed the memories of my dreams, – the midday sun shining on tall grass, the wild bull grazing quietly, the sudden parting of the grass before the swift rush of the tawny one, his leap to the bull's back […].[51]

In *Before Adam*, more has been handed down to the narrator than an irrational fear of predators. He has access to a memory that in the average person remains hidden in the unconscious – the race memory; it provides the view back through time into our Pleistocene ancestors' lives. Through his dreams, the narrator, who carries "an excessive freightage of memories", who is more "atavistic than other strains", establishes continuity between the individual conscious and a racial unconscious. The notion of instincts as unconscious memory was present in both Darwin and Spencer,[52] and London's explanation of racial memory seems an amalgam of the current biological and psychological theories:

> Now a fall [of our arboreal prehistoric ancestors from the trees], averted in such fashion [by clutching branches], was productive of shock. Such shock was productive of molecular changes in the cerebral cells. These molecular changes were transmitted to the cerebral cells of progeny, became, in short, racial memories […] There is nothing strange in this [the heritability of memory], any more than there is anything strange in an instinct. An instinct is merely a habit that is stamped into the stuff of our heredity, that is all.[53]

The way in which London worked with instinct theory, racial memory, and dreams brings to mind the development of these notions in Sigmund Freud (1856-1939). Indeed, London, who had a great interest in psychology, processed texts by Freud in his laboratory.[54]

In *Totem and Taboo* (1913), the way in which individual child experiences might produce neurosis in the adult, was seen as analogous to the fact that experiences accumulated in the childhood of the race, in its primitive state, might haunt a race in its mature, civilized state. Freud meant to bridge the gap between the non-analytic folk-psychology of Wilhelm Wundt (1832-

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50 Darwin, 1877, p. 288. Spencer, 1895 (1855), e.g. Vol. I, pp. 461-462, too, conceptualized mental evolution as analogous to mental development, which allowed him to use the minds of 'savages' and children as evidence of the evolution of the civilized mind from animal mind.

51 London, 2000 (1906-1907), pp. 7-8.

52 See particularly Spencer, 1895 (1855), Vol. I, p. 452, where the transformation of conscious memory into unconscious, organic, and heritable memory is likened to the process of learning where, too, the effort gets less with each repetition, until the habit becomes automatic and unconscious. The heredity-memory analogy guided many evolutionary theories of mind of the last quarter of the nineteenth century (see particularly Samuel Butler, e.g. *Unconscious Memory* 1880; but also Karl Ewald Hering, "Über Gedächtnis…" 1870 [trans. in Butler]; Richard Semon, *Die Mneme* 1904; Edward Dinker Cope, *The Primary Factors of Organic Evolution* 1896).

53 London, 2000 (1906-1907), p. 14.

54 Hamilton, 1986, pp. 129-130.

1920) and the psychoanalysis of the Zurich school.[55] In the small book which contained four articles first published in Freud's *Imago* (1912/3), one finds ample expression of the recapitulationist theory and its tenet that 'primitive human races' represent frozen ancestral states of western civilized man. 'Savages' were prehistoric humans still living amongst us. For psychology, they were of particular interest as illustrations of an earlier stage of mental evolution. Since the neurotic was a kind of regression or atavism, his or her psychology was expected to show similarities to the psychology of 'the savage'.[56]

Freud assumed that the Darwinian primeval horde had really existed, and that actual events had led to the initial acquisition of the Oedipus complex that then became part of our phylogenetic heritage.[57] In the prehistorically reified Oedipus complex, Freud saw the explanation for all neuroses.[58] Without the assumption of a collective psyche that would guarantee continuity in the emotional life of humans, that could bridge the gaps created in the stream of souls through the death of individuals, folk-psychology could not exist.[59] Most importantly in this context, Freud not only thought that the individual's memory spanned beyond his or her life, but saw dreams as the means through which experiences that reached back into prehistory were brought to the surface. Dreams represented regressions to the earliest conditions of the dreamer, a revival of his infancy. Beyond that, the dream might lead to the mental infancy of the race.[60]

Even more than Freud's, Carl Jung's (1875-1961) psychology meant a revelation to the London who was approaching the end of his life. In fact, London read *Psychology of the Unconscious* (1916) in his last year.[61] Here, Jung presented myths as the collective dreams of young humanity, resurfacing in the dream of the modern individual, which was a form of historical regression to the infantile stage of the human race. In parallel to recapitulation theory based on evidence from comparative anatomy and embryology, Jung analogized the way of thinking of 'the lower human races', to the myths of the ancients, to the fantasies of children, and to the dream of the modern civilized adult: "[…] ontogenesis corresponds in psychology to phylogenesis."[62] This turned the dream into a re-echo of the ancient and prehistoric.[63]

In the earliest blossoms of psychoanalysis, London finally found a solution to a paradox that had troubled his life as well as his literary work: How were the individual (the hero, the superman,

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55 Freud, 1913, p. 5.
56 Freud, 1913, p. 7. In *Totem and Taboo*, Freud already described the neurotic constitution as atavistic.
57 Freud, 1913, pp. 158-161. On Darwin's influence on Freud in general see Ritvo, 1990.
58 Freud, 1913, p. 174.
59 Freud, 1913, pp. 175-176. On Freud's concept of racial memory see also Heyman, 1977; Slavet, In progress; Stewart, 1976.
60 E.g. Freud, 1991 (1900), pp. 539-540. Note that in *Studies on Hysteria* (1895), Freud theorized hysteric traits as rudimentary organs; in *Before Adam*, on the other hand, the narrator, whose atavistic psychology gives him conscious access to the race memory, is described as hysterical (London, 2000 (1906-1907), p. 9). Not only in his developmental conception of mind, Freud had other precedents than Spencer, but also his dream theory, that is the notion of dreams as access to primeval mental states of humankind, had been anticipated by such figures as the neurologist John Hughlings Jackson (1835-1911), the psychiatrist James Sully (e.g. "The Dream as a Revelation," *Fortnightly* 59 (1893), pp. 354-365), and Havelock Ellis (1859-1939). This was true for Britain as well for the U.S. and France, where the notions of mental regression, dissolution, and involution were taken on before 1900 (Sulloway, 1979, pp. 257-275, 321-327).
61 Jung, 2002 (1916). See also McClintock, 1970.
62 Jung, 2002 (1916), p. 28.

the adventurer, the colonizer) and the social (altruism, universal love, brotherhood) to be reconciled? In his literary atavisms such as Red-Eye, London problematized these opposing demands, which were linked to the animal and moral aspects of human nature.[64] Jung emphasized that it was this dual human nature that lay at the bottom of pathological as well as non-pathological psychological problems. Jung saw the process of civilization in a progressive submission and domestication of the animal in humans, which could not be carried out without rebellion from the part of the freedom-loving vestiges of the primordial soul. The neurotic therefore experienced an aggravated condition of a universal human conflict; being at odds with oneself was seen as characteristic of the cultured human being *(Kulturmensch)*.[65]

The ways in which the novels by Stevenson and Norris foreshadowed the explanation the new psychoanalysis provided for the inner human struggle was thus made strikingly obvious by Jung, who defined neurosis as the symptom of an unsuccessful attempt to synthesize the opposing demands of the conscious moral ideal and the according to contemporary opinion immoral ideal of the unconscious.[66] London's narrator in *Before Adam* might be seen as approaching the psychoanalytic understanding particularly close, since for Jung and Freud the unfulfilled wish of the unconscious was of infantile, if not animal or prehistoric origin; it had been born in childhood or beyond and thus no longer fit the contemporary adult moral world.

In *Before Adam*, one also encounters the struggle of the individual to distinguish the self from the other, which for Jung was synonymous with the difference between personal (un-)conscious and collective unconscious, and to leverage his or her dual nature. According to Jung, the recognition of the psychic reality of the other within the self would allow to use the energy before bound up in the confusion of the two for their synthesis.[67] However, failure of recognition opens up the possibility of interpreting the notion of atavism in psychoanalytic terms as a projected archetype. The atavism would then not so much represent an aspect of those who are thus called, but rather the repressed irrationality of society at large that in a powerful resurfacing is projected on those that are feared.[68]

In *Before Adam* London described the dissociation into a wake-a-day personality with a memory of the experiences of the individual and a dream personality with a memory of past-day

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63 Jung, 1971 (1916), pp. 71, 78-79. This interpretation of dreams London met with in Freud, Jung, and also Friedrich Nietzsche (1844-1900) (e.g. 1879. *Human, All Too Human: A Book for Free Spirits*, trans. Marion Faber with Stephen Lehmann. Lincoln: University of Nebraska Press, 1984, e.g. Vol. II, p. 27, where Nietzsche explained that in the dream an atavistic relic of humanity manifested itself, since the dream represented the way of thinking of prehistoric man).

64 See also Mills, 1955. Note that this tension can be seen within non-atavistic individuals, too. Lop-Ear, for example, stays with the wounded Big-Tooth even though this is dangerous and "he is anxious to be gone", and the narrator takes this as "a foreshadowing of the altruism and comradeship that have helped make man the mightiest of the animals". He "often meditates upon this scene – the two of [them], half-grown cubs, in the childhood of the race, and the one mastering his fear, beating down his selfish impulse of light, in order to stand by and succor the other" (London, 2000 (1906-1907), p. 91).

65 Jung, 1971 (1916), p. 22.

66 Jung, 1971 (1916), p. 23.

67 Jung, 1971 (1916), pp. 74-75.

68 Jung, 1971 (1916), p. 97, explained for example the devil in this way, as an avatar of the shadow archetype, that is of the dangerous aspect of the unrecognized dark half of the human being. Besides the devil, Jung identified the demon as another main archetype, which often appears in the guise of the medicine man, old, dark-skinned and of the mongoloid type.

race experiences.[69] In this sense as in Freud's and Jung's, the racial memory really was the memory of an other, a memory that may lead back to the very beginning: "For Big-Tooth also had an other-self, and when he slept that other-self dreamed back into the past, back to the winged reptiles and the clash and the onset of dragons, and beyond that to the scurrying, rodent-like life of the tiny mammals, and far remoter still, the shore-slime of the primeval sea."[70] In Jekyll and Hyde, too, Hyde first exists in the daydreams of Jekyll, and Vandover at one point, close to hysteria, seems to gain a glimpse into the memory of all times – both Hyde, (homophone of *hide*,) and the wolf into which Vandover turns, are the prehistoric other.[71]

The dual-characters Jekyll-Hyde, Vandover-Brute, and Narrator-Big-Tooth fail at a synthesis; through the attempt of suppression or persistent segregation, they constantly feed and are finally swallowed by the residues of an evolutionary past. The state of dissociation between individual and collective psyche is maintained if not sharpened. Thus, as suggested by Jungian psychology, the modern self continues to find itself opposite a kind of 'Negro culture' (*Negerkultur*), opposite a primitive state. This is in Jung's appraisal the general state of his culture – a thin crust of civilization covering a dark-skinned beast (*eine dunkelhäutige Bestie*).[72] In the end, the atavistic sides of the literary personalities win, and the regression in the psychoanalytic sense is complete; at this point, one may recognize the condition of the neurotic who is unable to see the archetypes of the unconscious as psychic realities and instead takes them for concrete realities. In other words, the individual identifies with aspects of the common prehistoric past of humankind.[73]

This is where the dream comes in. Jung ascribed Freud the primacy of the insight that the analysis of dreams was the most important method for unraveling the unconscious.[74] London's narrator calls himself an "atavistic nightmare", which links the notion of atavism with that of dreams as windows into prehistory – which open particularly wide for him, because his psyche or his brain is atavistic.[75] In psychoanalysis, the dream, the myth, and other manifestations of the other in the self gained remedial impact in the healing process of the split human being. The wordless events that might be awakened through a regression into pre-infantile time have their origin in the lives, sufferings, and joys of our ancestors, and they must be idiosyncratically re-embodied by the individual. Due to their inherent contradiction to the conscious, they cannot, however, be translated immediately – the dream may function as mediator between the reality of the unconscious and that of the conscious.[76] Through the interpretation of dreams, an intense dialogue between unconscious and conscious is initiated. The analysis of dreams fulfils a transcendental function that might enable a personality to realize his or her original potential for wholeness, completeness, and perfection (*Individuationsprozess*).[77]

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69 London, 2000 (1906-1907), p. 15.
70 London, 2000 (1906-1907), p. 139.
71 Norris, 1986 (1914), p. 162.
72 Jung, 1971 (1916) pp. 97-98, see also pp. 74-75.
73 Jung, 1971 (1916), p. 97. Jung thought of archetypes as having lives of their own (*Partialseelen*, pp. 67-68).
74 Jung, 1971 (1916), p. 24. On Jung's reception of Freud see also Jung, 1972 (1906-1916).
75 London, 2000 (1906-1907), "this atavistic brain of mine", p. 21, also p. 20.
76 Jung, 1971 (1916), pp. 81, 103.
77 Jung, 1971 (1916), pp. 99, 110-111.

In *Before Adam* as well as in other writings of London and his contemporaries such as Norris and Stevenson, the problems associated with the duality of human nature were expounded, and ways were sought to overcome opposing demands in the authors' own lives.[78] The potion, alcohol, madness appear as unsuccessful remedies. In the dream, however, a solution was foreshadowed that would find full expression in Freud's and Jung's theories. London immediately recognized the new psychology as the means for synthesizing the racial heritage with the modern self he had been searching for. In the child, the criminal, the insane, the primitive, 'the savage', and the atavistic reversion to such states, the selfish animal instincts were set loose. But the same ruthless instincts drove the average Western male on to conquer ever new coasts – to return to London's metaphor.

However, in civilized man, our primordial nature was in conflict with the higher social and moral instincts. In the perfect case, the constructive sides of our animal origin were brought to use, while the destructive aspects were subdued by the moral sense. London ascribed dreams a synthesizing function in the individual's attempt to gain such a balance. They were atavistic devices in the understanding of the individual as part of the community. Like London's books, they were memory aids; they enabled an understanding of self as part of the prehistory of the race.

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78 On a possible influence of Jung on the earlier fiction of London, such as *The Call of the Wild* (1903), *Wild Fang* (1906), and *Before Adam*, see Crow, 1966; on such an influence in the late South Pacific fiction see Reesman, 1988; on Jungian psychology in "The Red One" (1918) see Berkove, 1966. West, 1998, identifies several unresolved contradictions against which Norris struggled – such as effete east and rough west, artist and businessman, romance and realism, nineteenth and twentieth century, morality and strength, egalitarianism and racial suprematism, communism and capitalism, sensualism and intellectualism, primitive and civilized, determinism and freedom – and connects these to the unconscious/conscious dualism. In fact, both Norris and London suffered from similar paradoxes that marked their generation as a whole (on paradoxical stances in the racial question see for example Furer, 1966).

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Race and Kinship in Anthropology: Morgan and Boas

Staffan Müller–Wille

Introduction

Late nineteenth–century anthropological research was to a large extent preoccupied with questions of inheritance, but it was so with regard to two sets of phenomena. On the one hand, cultural and physical differences among humans were subjected to description and comparison. Inheritance was of interest here, as the present distribution of the varieties of mankind was interpreted as bearing some relation to the stages and bifurcations through which human evolution had occurred in the past. On the other hand, societies other than contemporary European ones were scrutinized for the institutions that made them work. Classification of kinship and organization into clans, moieties, castes, etc., in short: the structures along which names, social status, and property were inherited raised the interest of anthropologists here for their relation to the temporal stability of the social order.

From historical literature on late nineteenth–century anthropology one could get the impression that it was the first aspect, that of racial descent, which dominated research agendas. Yet some of the most influential works of the period were dedicated to what we today would call questions of social anthropology: Johann Jakob Bachofen (1815–1887), Swiss lawyer and philologist, argued in *Das Mutterrecht* (1861) for a primeval stage of promiscuity, followed by gynæcocracy, which again, though only in select cultures, yielded to patriarchy. Henry James Sumner Maine (1882–1888) directed attention to the complex relations between kinship and territorial bonds in his *Ancient Law* (1861), and John F. McLennan (1827–1881) coined the analytical categories "exogamy" and "endogamy" in his first major essay *Primitive Marriage* (1865). Like Bachofen, both Maine and McLennan were lawyers. Edward B. Tylor in his *Researches into the Early History of Mankind and the Development of Civilization* (1865), and Henry Lubbock, in his *The Origin of Civilisation and the Primitive Condition of Man* (1870) laid the foundations for understanding the interdependence of cross–cousin marriage and exogamy, mother–in–law avoidance and matrilocal residence. Finally, Lewis H. Morgan, again a lawyer by training, laid bare the intricacies of kinship terminologies world wide in his *Systems of Consanguinity and Affinity of the Human Family* (1871) and demonstrated their correlation with forms of marriage and rules of descent, among other things drawing the distinction between descriptive and classificatory kinship systems. Through their reception by Charles Darwin, in his *The Descent of Man* (1871),[1] as well as by Friedrich Engels in *Der Ursprung der Familie, des Privateigentums, und des Staates* (1884),[2] all these works fed into the main–stream ideologies of late nineteenth–century, no less so than evolutionary anthropology did.

Was there a common ground for this twofold preoccupation with inheritance? At first sight it may well seem so: Descent, affinity, and genealogy seem to afford a common, and what is more,

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1 Kuper (1997).
2 Krader (1973): ch. 2.

universally shared metaphorical framework for the study of both racial descent and kinship. And indeed, as George Stocking has pointed out: Before the twentieth century, "'[b]lood' —and by extension 'race'— included numerous elements that we today would call cultural; there was not a clear line between cultural and physical elements or between social and biological heredity."[3] Modern European racial discourse, as argued by Renato Mazzolini, had itself grown out of the extension of a caste system of European making with corresponding interdictions and regulations regarding marriage and inheritance.[4] Eugenicists and racial hygienists relied on what may clearly be addressed as rules of exogamy (avoidance of incest) and endogamy (segregation by race and class, exclusion of the 'unfit') in their programs for social reform.[5] The study of race and kinship seem to be nothing but the two sides of one and the same coin, a widespread, and deeply rooted fear of the consequences of promiscuity and incest, and a corresponding obsession with the rationalization of even the most intimate human relations.

As soon as one tries to move beyond analogies, the relation of kinship and racial descent becomes curiously unclear however. The main reason for this is, that the categories used to analyze kinship and racial descent respectively do not map onto each other. Racial categories, in the nineteenth century at least, obeyed the logic of the Linnaean hierarchy, constituting a system of mutually exclusive classes contained within more extensive and again mutually exclusive classes. Ideally, an explanation of their formation would lead back to one or several singular origins. The history of racial descent is one of progressive differentiation, usually represented as the growth of a tree-like structure. Kinship, on the other hand, employs categories that overlap systematically: one individual can be a daughter, a mother, an aunt, a cousin at once, and these categories do not form a hierarchy of nested classes. Their explanation, in the case of humans at least, does not lead back to singular origins, but to bonds of marriage. Reducing kinship to a singular "ancestor" is the task of genealogy, quite a different matter, and one that systematically has to discard certain kin relations. Finally, the history of kinship is one of alliances and separations, of a continually woven 'fabric […] in which warp and filling yarn correspond to localities and tribes', as Claude Lévi–Strauss formulated it.[6] Darwin, famously, looked for sexual selection or "long-continued intercrossing" as a means to explain the "protean or polymorphic" nature of mankind.[7]

My aim in this paper is largely descriptive. I will look at the way in which the discourses of racial descent and kinship related to each other in a selection of texts, with particular emphasis on Morgan's *Systems of Consanguinity and Affinity of the Human Family* (1871) and Franz Boas's (1858–1942) anthropological papers from the late nineteenth century. In particular, I will be interested in how the analysis of kinship, establishing definite relations among individuals, was used by anthropologists of the late nineteenth century as a tool to reach a complete analysis of the varieties of mankind – the "races", "families", or "nations" constituting mankind.

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3 Stocking (1994): p. 6.
4 Mazzolini (in press).
5 See, e.g.: Richardson (2002).
6 Lévi–Strauss (1962) (cited from German edition, Frankfurt a. M. 1969: p. 97).
7 Darwin (1871): p. 550; cf. Endersby (2003).

Morgan and the analysis of kinship

When Lewis Henry Morgan carried out his first interviews with Iroquois informants in Albany 1844, his immediate motive for understanding the workings of their social life was a curious one: He had joined a secret fraternal organization, the Grand Order of Iroquois, and wanted to duplicate the structure of Iroquois society in this organization. After the renewal of his ethnographic interests in 1857, he began to collect more extensively, by setting up a circular containing a recent paper on the 'Laws of Descent of the Iroquois' and a set of questions regarding kinship and clan organization, in particular a long list of questions asking for the counterpart of kin terms to the fifth collateral degree in other languages. Morgan had set up this list of questions or "schedule", as he also called it, with "considerable labor" to be "sufficiently full to describe every known relationship, and yet arranged upon such a method as to be simple and intelligible."[8]

Morgan called his schedule a "new instrument in ethnology."[9] According to the introductory chapter to *Systems of Consanguinity and Affinity,* he first sent it out, in printed form, to "the several Indian missions in the United Strates, to the commanders of the several military posts in the Indian country, and to the government Indian agents." Unsatisfied with the answers he thus received –one district officer had answered to Morgan's questionnaire of the "customs and manners" of natives: "Manners none, customs beastly"–[10] Morgan began to undertake "annual explorations among the Indian nations" himself to gather kinship terms. To get at similar information outside of North America, Morgan addressed both the Smithsonian Institution and the U.S. government for assistance. Assistance was granted, and Morgan could rely on a worldwide network of diplomatic staff, missionaries, and scientists to procure relevant data for him.[11]

What the questions contained in the schedule must have looked like – a copy is preserved in the Smithsonian collection,[12] but I have not had a chance to have a look at it– can be derived from the tabular presentation of results in Morgan's *Systems.* The data is presented for the "Seneca–Iroquois" and the "Tamil People of South Asia", both supposedly representative of what Morgan called the "classificatory system of relationship", i. e. a system that classifies collateral cousins of various degrees together with siblings under one and the same term. The respective Iroquois and Tamil kin terms are tabulated against their translations into English and a long list of terms headed "Description of persons." Despite its affinities with what Morgan called, in a sweeping generalization, the "descriptive systems" of relationship among the Aryan and Semitic languages, which address each relative by a definite, mostly composite term, the latter "descriptions of persons" do not stem from any natural language. Morgan himself called them "numerical in character", "resting upon an ordinance of nature," and both "universal and unchangeable."[13] The principle they build upon is simple: each possible kin relation between an assumed "ego" and another person, up to the fifth collateral degree, is expressed in a combination of primitive terms:

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8 Tooker (1992).
9 Morgan (1871): p.9.
10 Fox (1877): p. xxxii.
11 Tooker (1992): p. 9.
12 Ibid.
13 Morgan (1877): p. 11.

"mother", "father" "son", "daughter", "sister", "brother", and, for affine relatives, "husband" and "wife". The list of terms under "Description of person" reaches from simple terms as "my son" to complex ones like "my mother's mother's mother's brother's son's son's son." All in all, the table lists 237 terms like that.

What is the purpose of this exercise? The introductory chapter to Morgan's *Systems of Consanguinty and Affinity* contains the following discussion of the difficulties Morgan encountered when asking correspondents to answer his questionnaire:

> A large number of schedules, when returned, where found to be imperfectly filled out. Misapprehension of the nature and object of the investigation was the principal cause. The most usual form of mistake was the translation of the questions into the native language which simply reproduced the questions and left them unanswered. […]. As our own system is descriptive essentially, a correct answer to most of the questions would describe a person very much in the form of the question itself, if the system of the nation was descriptive. But, on the contrary, if it was classificatory, such answers would not only be incorrect in fact, but fail to show the system. […]. Every system of relationship is intrinsically difficult until it has been carefully studied. […]. It is easy, therefore, to perceive that when a person was requested to work out, in detail, the system of a foreign people he would find it necessary, in the first instance, to master his own, and after that to meet and overcome another, and perhaps, radically different form.[14]

These observations are highly revealing. They demonstrate that the terms contained under the rubrique "Description of the Persons" are not merely descriptive, but in fact provide a complete analysis of kinship. They break down kinship into each and every possible kin relation, thus serving the translation of terms between different systems by defining their respective extensions. Without this device, the systems of "foreign people" would remain largely opaque to their observers, and, vice versa, informants would be unable to give "correct" answers to the questions posed to them. A peculiar property of the schedule betrays its analytic character: It is, in Morgan's words "necessarily self-corrective […], since the position of Ego and his or her correlative person is reversed in different questions."

Morgan himself, it is true, systematically blurred the distinction between the schedule of his own making and the "descriptive systems" he saw at work in the Aryan and Semitic language families. His contemporary James McLennan saw that already, and in *Ancient Society* Morgan felt himself forced too meet McLennan's criticisms in a 12 page long "note".[15] But analyticity remains a decisive character of his schedule, one that distinguishes it from kinship terminologies used in everyday contexts.[16] It betrays the extent to which his concerns were not philological in the first place. The preface to *Systems* refers to "philology", but as a mere "instrument for the classification of nations into families." And adds, that "[i]t was with special reference to the bearing which the systems of consanguinity and affinity of the several families of mankind might have upon this vital question, that the research, the results of which are ascertained in this volume, was undertaken." [17]

[14] Ibid.: p. 6.
[15] Morgan (1877): pp. 509–521.
[16] Tooker (1992, p. 9) emphasizes that there is no vernacular kinship terminology that does not provide the possibility to describe each relative by terms as the ones employed in Morgan's schedule and adds: "Morgan's schedule rests on this fact."

Systems of consanguinity, Morgan believed, were particularly well–suited for the task of classifying humankind: "The family relationships are as ancient as the family. They exist in virtue of the law of derivation, which is expressed by the perpetuation of the species through the marriage relation. A system of consanguinity, which is founded upon a community of blood, is but the formal expression and recognition of these relationships."[18] And in his 1877 *Ancient Society* he should fomulate: "[Modern institutions] have had a lineal descent through the ages, with the streams of the blood, as well as a logical development."[19] To Morgan the analysis of kinship reveals, one might say, the organization of mankind, and differences in that organization immediately reflect the different stages and branches of its evolution.

Boas and the statistics of culture

George Stocking has assigned Boas the role of a founding father of the "modern anthropological culture concept" characterized by "historicity, plurality, behavioral determinism, integration, and relativism."[20] On the other hand, Stocking has portrayed Boas's work in physical anthropology as instrumental in the "passing of a romantic conception of race – of the ideas of racial 'essence,' of racial 'genius,' of racial 'soul,' of race as a supraindividual organic identity". In particular it was Boas's statistical approach that was, as Stocking put it, "subversive of traditional racial assumptions."[21] Boas, as is probably too little known, was an ardent practitioner of physical anthropology and biometry, designing instruments for craniometric measurements,[22] carrying out large–scale anthropometric studies on native populations of North–America,[23] and creating mathematical tools for the study of correlation.[24] In the following, I want to argue that Boas advocated a similar statistical approach in his studies of primitive culture as well, and that kinship was integral to that approach.

In 1887, Boas became involved in a debate about museum displays.[25] Otis Tufton Mason (1838–1908), curator of ethnology at the Smithsonian institution, had suggested to arrange ethnological displays at the United States National Museum according to a classification of the objects displayed: exemplars of different kinds of artifacts were arranged in series, each representing a stage in the evolution of its kind; the rationale on which this presentation rested was borrowed from natural history. As Boas quoted Mason:

> [Human inventions] may be divided into families, genera, and species. They may be studied in their several ontogenies (that is we may watch the unfolding of each individual thing from its raw material to its finished production). They may be regarded as the products of specific evolution out of natural objects serving human wants and up to the most delicate machine performing the same function. They may be modified by their relationship, one to another,

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17 Morgan (1877): p. v–vi.
18 Ibid.: p. 10.
19 Ibid: p. 4.
20 Stocking (1983a): p. 230.
21 Stocking (1983b): pp. 192–194.
22 Boas (1890).
23 Boas (1891b).
24 Boas (1894).
25 Jacknis (1985); Jenkins (1994).

> in sets, outfits, apparatus, just as the insect and flower are co–ordinately transformed. They observe the law of change under environment and geographical distribution.[26]

The alternative Boas proposed was to arrange collections "according to tribes, in order to teach the peculiar style of each group." The reasons he adduced for this position were epistemological:

> In regarding the technological phenomenon as a biological specimen, and trying to classify it, [Mason] introduces the rigid abstractions species, genus, and family into ethnology, the true meaning of which it took so long to understand. It is only since the development of the evolutional [sic] theory that it became clear that the object of study is the individual, not abstractions from the individual under observation. We have to study each ethnological specimen individually in its history and in its medium […]. Our objection to Mason's idea is, that classification is not explanation.[27]

This seems to be a strange reasoning: First of all, "studying each ethnological specimen individually in its history and in its medium" would, taken literally, be an endless task, and both Mason as well as other participants in the debate pointed out the practical difficulties that an arrangement by tribes would imply.[28] Secondly, an arrangement according to tribes seems to involve as much classification as that proposed by Mason, namely a classification by "tribes." Also this criticism was raised in the debate,[29] and the point has only recently been reiterated by a post–modern epigone.[30] To this, Boas only had a short, categorical reply: "Such groups [i.e. tribes, and groups of tribes] are not at all intended to be classifications."[31]

Boas's studies of native myths along the North–Pacific coast can serve as an example to elucidate what he had in mind with this strange assertion. In these studies, Boas broke down the myths into constituent "elements" and recorded their distribution within a group of geographically contiguous "tribes." "We can in this manner," as Boas explained, "trace what we might call a dwindling down of an elaborate cyclus of myths to mere adventures, or even to incidents of adventures, and we can follow the process step by step."[32] In more detail, he described this method as follows:

> If we have a full collection of the tales and myths of all the tribes of a certain region, and then tabulate the number of incidents which all the collections from each tribe have in common with any selected tribe, the number of common incidents will be larger the more intimate the relation of the two tribes and the nearer they live together. This is what we observe in a tabulation of the material collected at the North Pacific Coast. On the whole, the nearer the people, the greater the number of common elements; the farther apart, the less the number.[33]

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26 Quoted according to Franz Boas (1887b): p. 485. Boas does not give a reference, and I have not been able to identify his source.
27 Ibid.
28 Dall (1887): p. 587; Powell (1887).
29 Powell (1887).
30 Jenkins (1994).
31 Boas (1887a): p.614.
32 Boas (1896a): p. 2. This paper is a summary of Boas (1895a).
33 Ibid.: p. 3.

It should be noted, that it is the unequal distribution of elements of myths that defines them as their constituent elements in the first place. Such a "statistical inquiry", as Boas called it,[34] rested on a "fundamental condition", which "differentiates our method from other investigators […], who see a proof of dissemination or even blood relationship in each similarity that is found between a certain tribe and any other tribe of the globe" – namely, that the material, on which it was based, was "collected in contiguous areas."[35] This contiguity was not necessarily, Boas emphasized, a geographical one, and it is here were marriage, kinship, and social structure enter the picture. "The social customs of the Kwakiutl," the ethnic group most intensely studied by Boas during several field trips, are, as he maintains, "based entirely upon the division into clans and the ranking of each individual is the higher –at least to a certain extent– the more important the legend of the clan." Moreover, "the customs of the tribe are such that by means of a marriage the young husband acquires the clan legends of his wife, and the warrior who slays an enemy those of the person whom he has slain. By this means a large number of traditions of the neighboring tribes have been incorporated in the mythology of the Kwakiutl."[36]

It is by relating the distribution of mythical elements to a space whose contiguity can be ascertained in terms of social relations among individuals –alliances as well as antagonisms– that Boas wants to circumvent the pitfalls of analogical reasoning. In the analysis of these relations, Boas clearly relied on the categories that others before him, most prominently Morgan, had developed to account for social structure in terms of kinship. I cannot discuss this in detail here, but let me state, that the clan system that Boas detected among the Kwakiutl was actually more complex than he described it in the quote I gave you above: through marriage it was not the husband personally who acquired the clan status of his wife, but he acquired it "for his son."[37] Ironically, the grand picture that Boas came up with on the basis of such studies was strongly opposed to that of evolutionists like Morgan:

> A great many [...] important legends prove to be of foreign origin, being grafted upon mythologies of various tribes. This being the case, I draw the conclusion that the mythologies of the various tribes as we can find them now are not organic growths, but have gradually developed and obtained their present form by accretion of foreign material. Much of this material must have been adopted ready–made […]. We are, therefore, led to the conclusion that from mythologies in their present form it is impossible to derive the conclusion that they are mythological explanations of phenomena of nature […], but that many of them, at the place where we find them now, never had such a meaning. If we acknowledge this conclusion as correct, we must […] admit that, also, explanations given by the Indians themselves are often secondary, and do not reflect the true origin of the myths.[38]

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[34] Ibid.
[35] Ibid.: p. 6.
[36] Ibid.: p. 8–9.
[37] See Boas (1895b): pp. 334–335.
[38] Ibid.: p. 5.

Conclusion

Boas rejection of an "organic growth" of culture, and the model of a "complex growth"[39] by accretion which he advocated instead reveals that his famous environmentalism was far from being Lamarckian in character. The elements of culture are transmitted without bearing the mark of the context in which they originated, and their combination is a matter of historical accident, not of influence or the weight of tradition. The similarities that this model exhibits with respect to the distinction that biologists and biometricians began to draw between the transmission of germinal elements and the development of somatic characters is remarkable. In 1901 Boas wrote: "[… T]he development of *culture* must not be confounded with the development of *mind*. Culture […] shows the cumulative effects of the activities of many minds. But it is not an expression of the organization of the minds constituting the community."[40] And when Johannsen canonized the distinction of genotype and phenotype in 1911, Boas whole–heartedly endorsed it.[41] Boas cultural relativism did not develop in opposition to rival claims from physical anthropologists in the explanation of cultural phenomena. His ambition was to base the whole of anthropology – somatology, ethnology, and linguistics alike– on an inductive basis that proceeded not by analogies but by connecting individual phenomena with the help of statistical procedures.[42]

Is there something to be said about the motivation to do this? George Stocking has identified the central concern of Boas, related to the *Kulturkampf* of his native country, with the "search for 'the psychological origin of the implicit belief in the authority of tradition.'"[43] For Morgan, tradition had been largely inescapable: "[T]he primary institutions of a people are necessarily permanent from age to age […]. It is only by the entire and absolute transmutation of a race from the hunter to the civilized condition, that such institutions can be eradicated." Likewise Boas admitted, that "[e]ach individual must be influenced to a greater or less extent by the mass of traditional material present in his mind." But to Boas this influence was not inescapable: "There is an undoubted tendency in the advance of civilization to eliminate traditional elements, and to gain a clearer and clearer insight into the hypothetical basis of our reasoning." The method to achieve this was "to carry the analysis of any given phenomenon to completion." I have tried to show that the analysis of kinship, as instituted by Morgan, served this aim, and that it actually succeeded in revealing and overcoming cultural preconceptions. And it is telling, in this respect, that Morgan took "descriptive" systems of kinship terminology to be the hallmark of "civic" societies as opposed to "gentile" ones, that were "founded upon relations purely personal."[44]

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[39] Boas (1891a): p. 20.
[40] Boas (1901).
[41] Boas (1911).
[42] Boas (1896b).
[43] Stocking (1992): p. 97.
[44] Morgan (1877): pp. 393–395.

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