HUMAN HEREDITY

1750-1870

The Construction of a Scientific Domain

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Human Heredity (1750-1860)

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Abstract

This thesis makes a case for the (human) origin of the idea of *heredity* as an explanatory cause in the biological sciences. Focusing mainly on the French and British milieux in the period given (1750-1870), it shows how a transition from a medical to a general (biological) conception of hereditary transmission came about through a process of reification of the metaphor (from the legal sense of 'heredity'), a de-pathologization of its reference and a generalization of its main features. The consequence was that the possibility of an independent domain of research concentrated on the 'hereditary cause' became a distinct one both for French and British authors sometime during the early 19th century. Human heredity was at the forefront of this structuration of a new domain. The conceptual and evidential moves that medical men had made during their disputes over hereditary diseases fed directly into the wider, biological domain in a way that has not been recognized up to now.

The thesis analyzes several of the features that Heredity, as a concept, received from its medical (pathological) origins. The most notable ones include the introduction of latent causality in order to account for irregularity and indeterminacy of effects. This mediate approach to causality, in which appeals to concepts such as pre-disposition were common, helped to make sense of the patterns of reoccurrence that had been for a long time associated with the hereditary: atavism, homochrony and so forth.

Also discussed in the thesis is the transition from the medical tradition of inferring causal connection from cases (stories, anecdotes) to the practice of organizing positive and negative cases in statistical fashion. The dispute surrounding the validity of such methods was particularly important in the domain of Heredity, since it led to Francis Galton's breakthroughs in statistical reasoning. The relations between Galton's work and that of previous hereditarians are therefore also examined.

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Introduction

> Quel monstre est-ce que cette goutte de semence, de quoi nous sommes produits, portes en soi les impressions, non de la forme corporelle seulement mais des pensements et des inclinations de nos pères? Cette goutte d'eau, oû loge-t-elle ce nombre infini des formes? et comme portent elles ces ces ressemblances d'un progrès si temeraire et si déréglé que l'ariere-fils répondra à son bisaïeul, le neveau à l'oncle?
>
> Montaigne, 1580, *Essais*, Livre II, 37, p.540

> By revealing to us the absolute mechanism of all action, and so freeing us from the self-imposed and trammeling burden of moral responsibility, the scientific principle of Heredity has become, as it were, the warrant for the contemplative life.
>
> It has shown us that we are never less free than when we try to act. It has hemmed round with the nets of the hunter, and written upon the wall the prophecy of our doom. We may not watch it for it is within us. We may not see it save in a mirror that mirrors the soul. It is Nemesis without her mask. It is the last of the Fates, and most terrible. It is the only one of the Gods whose real name we know.
>
> Oscar Wilde, 1891

The arch that covers the distance between the two quotations above defines the space over which the present work makes its transit. From the amazement of the observer who perceives a capricious pattern of reoccurrences of physical and psychological peculiarities within a family and begins to ask for an explanation, to the arrogant nominalism of he who thinks that he has the answer, and it answers much more than was originally asked.

Biological Heredity began by being Human Heredity. That is the first of many historical facts that has to be taken on board before understanding the links between Wilde's answers and Montaigne's questions. And Human Heredity began with the perception that genealogical relationships between persons implied more than social bonds: there was also physical rapport manifested by the looks and by health. The inference that something must be passed on from parents to offspring in the act of generation that accounts for the perceived similarities is direct enough; not so the analogy that was found suitable for it: as land, craft, wealth and title were inherited, so could body and mind (or some of their features) be inherited too.

But Human Heredity, in the biological sense, was for many centuries only a metaphor. The noun itself (heredity) did not have the causal meaning that has come to characterize it. Since antiquity till the 18th century, the adjective "hereditary" was the one employed when a given trait was found to characterize a family or another genealogical group. And then, it occurred with much more frequency when anomalies, moral or physical were the subject. Hereditary gout or hereditary depravities were more common formulas than their positive counterparts. Montaigne, for instance, wrote his piece to describe how he among all his brothers was unlucky enough to have inherited the bladder stone that plagued his father's life. Beside the mystery of the transmission he refers to above, he found mysterious the fact that his father's stone appeared well after he was born, and his own stone appeared more or less at the same age his father had when it happened to him. What kind of funny influence could hide itself like that and then know somehow when to show its ugly

face?

For Montaigne the hereditary elements of body and mind, were always accidental. They only affected the kind of superficial features that individualized humans but did not change them nor touched their essence. Many of his contemporaries, and successors were rather sceptical about the whole issue of hereditary transmission, preferring to believe that external factors were to blame for stones as well as all other accidental similarities between parents and children.

For Wilde, or it would be better to say for the age Wilde was being ironical about, Heredity had become *essential*, it had taken over the core. Human (biological) heredity had captured the imagination of the 19th-century Europeans. The infection began, as I will show, among the medical men. Their old concept of "hereditary disease", which had kept the transmission metaphor alive, developed into the powerful explanation tool of naturalists, physiologists and, on another path, of psychiatrists, social reformers and novelists of doom.

Some people perhaps would not agree that Heredity as a biological concept in the 19th century is conceptually the same thing, or has any relevant connection with the Heredity that Wilde is above making reference to, i.e. that of psychiatrists, eugenists and social Darwinists.[1] But they shared more structure than is obvious, and this, apart from the contemporary influences that they exerted over each other, can be explained in part due to their common medical origin.

As a biological explanatory concept, Heredity received a major boost when it became essential to understand its workings in order to accept or challenge the logic of change established by Darwin's long argument. But by then it already had a place in the century's intellectual and political life. Darwin, and for that matter all his contemporaries who paid attention to hereditary transmission, such as Spencer or Galton, found the concept of Heredity with more structure than has been recognized. Some of the claims by historians to "originality" in their ideas can be challenged by understanding where the influence they received came from. In other words, by bridging the barrier that historians have built between the non-biological, medical and social uses of Heredity in the 19th century, and its scientific, biological uses, a different panorama of how the concept of Heredity in biology got to be "constructed" emerges.

Several authors have at different moments pointed towards the medical community of the 18th and early 19th centuries as responsible for an early stage of what whig historians call the "Prehistory of Genetics". But only recently, with the works of some less prejudiced researchers, has the substance of the conceptual relationship between the two areas begun to be unearthed.[2] It is the aspiration of this work to contribute to filling that gap. I have tried to follow the avatars of a metaphor that ancient and then Renaissance physicians were the first to convert into an explanatory tool. The communication of disease, or of a tendency to develop it, from parents to children, which they saw to be a fact, demanded causal hypotheses, and

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[1]I emphasize the internal conceptual relation because much has been written about the external mutual interaction between hereditary ideas and the social interests of their holders, in these two spheres. Social historians, such as Cooter (1984), Desmond (1989) and Mackenzie(1981), have chartered that side with excellent results, but they leave us starved for credible conceptual analysis.

[2]See R. Olby (1987), and "Constitutional and Hereditary Disorders" (in print); and V. Hilts (1982).

they promptly began to produce them. These, by their nature had to interact with the ideas from generation theorists, and in relation to them they took always a secondary role. The hereditary transmission of “accidents” was for a long time simply an empirical testing field for generation hypotheses. But in the hands of physicians, after the 17th century, it developed into a filed of inquiry of its own.

The prevailing generation theories of preexistence produced, during the 17th and 18th centuries some degree of scepticism in relation to hereditary transmission of peculiar characters. But the *a priori* reasonings in which such scepticism was based was not of the liking of many 18th century medics, who saw the confusions and baroquisms of generation theories less reliable than the empirical evidence collected or heredity of both good and evil aspects of individuals’ constitutions. At the same time, the previously prevailing view of hereditary transmission of disease through generation, that of humoral taints, was being progressively discarded.

The combination of these factors led some physicians to attempt different physiological accounts of hereditary transmission, based on what they called “solid to solid” communication. Eventually, this step was to prove crucial for a unification of hereditary speculations in different fields under one theoretical frame, thus bringing together what had been until then only an analogical relationship between hereditary phenomena among humans and those among other animals and plants. In this way, the longer history of analysis and clarifications of how the hereditary cause worked was applied to other fields, first of human and then of general interest. The study of the origin of human varieties is perhaps the first example, and the specialized study of hereditary transmission of mental illness the most notorious, before Darwinism took over.

Some sectors of the medical community in France, at the end of the 18th and the beginning of the 19th century, focused their attention on hereditary transmission. They were followed shortly after by physiologists and naturalists (usually with medical formation), and later joined by alienists (psychiatrists), psychologists and the like, in the pursuit of an acceptable explication of the new concept they were all finding so useful: Heredity.

The advent of Mendelian genetics, and their eventual marriage to Darwinism in the course of this century, together with the obnoxious consequences of human hereditarianism, have dramatically distorted the way we read events and theories related to the 18th 19th century hereditary discussions, especially in the human case. The effort to by-pass such distortions, however, must be made in order to genuinely understand how, for instance, an infuriatingly bigoted book like Galton’s *Hereditary Genius* (1869) was at the same time a major development in the history of science. Without such efforts we will never achieve a satisfactory account of how our modern obsession with heredity and, later, genetics came about, as we will be always putting our prejudiced cart in from of the horses of actual historical understanding. This dissertation, I believe, has made such an effort. Its success id for the reader to judge.

Chapter 1

The Hereditary: from metaphor to cause. A reification story.

1.1 From adjective to noun

The term “heredity” was brought into the English language to match the French use of “hérédité”. This happened between 1860 and 1870, when a noun was needed to refer to the maturing domain of scientific enquiry that had come to crystallize around the set of phenomena that were previously clustered around the adjective “hereditary”. French physicians had been using “hérédité” in such a specialized sense for several decades, and “heredity” seemed a good option (instead of “inheritance” or “heritage”, for instance) because it could be both directly related to the widespread French noun and to the adjective “hereditary”, which had been long in use in a similar sense, originally brought into English medical parlance by physicians translating from the Latin adjective *haereditariis.*

The adjective “hereditary”, in the natural sciences, is an ancient borrowing from the legal and social, based on the straightforward analogy between handing down property or titles to descendants and transmitting physical or moral qualities to them. Although in everyday language the metaphorical (analogical) use of the adjective has been common and widespread in most Western languages for centuries,[1] the first consistent technical borrowing of the adjective into natural science was done by physicians when they categorized a set of diseases as “hereditary”.

There is within the Hippocratic-Galenic tradition a persistent and pervasive attention to the fact that disease, or a disposition (propensity) to it, can be causally transmitted from parents to their offspring.

The old coinage “hereditary disease” (Greek = *Nσοιχληρομ iotaχαι*, Latin = *haereditarii morbi*) has been in use in several European languages, in and outside specialized contexts, at least since Hippocrates and Aristotle. But it has only been partnered with nouns (“heredity”,“inheritance”, “heritage”) for over a century. The Oxford English Dictionary provides a good evidence of this. While it quotes 16th,

[1]Any sort of similarities or coincidences between parents and children, in physical appearance, abilities or disabilities, patterns of behaviour, etc., could be said by the ancients to be “hereditary”, without further explanation, both in ancient Greece and Rome. See David B., (1971) *La pré-histoire de la Génetique*; and Zirkle C.(1946) “The early history of the inheritance of acquired characters”. An insightful “deconstruction” of the concept of heredity as based on a metaphor was done by J.A Thomson (1908).

17th and 18th century uses of "hereditary" in relation to diseases,[2] the first biological use of "heredity", or "inheritance", it gives come from the 1860's.

The French noun "hérédité" was the first to establish itself as a strong, explanatory scientific term. Championed by a whole generation of French physicians who had decided that "the hereditary" could and should play more than a secondary role in the understanding of mankind's past and present conditions, and in the shaping of the future one. After 1830, "hérédité" stormed their writings and became the emblem of their new, brash and post-enlightened and post-revolutionary approach.

In his autobiography Francis Galton tries to pass himself off as responsible for the introduction of "heredity" into English. He mentions having been criticized by some of his contemporaries for coining the gallicism "Heredity" during the late 1860's.[3] R.S. Cowan has argued that this choice of a neologism by Galton signals an intention of putting some distance between his research project and previous, "pre-scientific" ones, including French medical hereditarianism.[4] The fact is that both Spencer and Darwin used the word several years earlier[5] and Galton almost certainly took it from them. They in turn had been driven to its use by exposure to French authors, principally, I believe, Prosper Lucas.[6] Contrary to what Cowan tries to show, the use of "heredity" instead of "inheritance" was not at first of any theoretical importance, and both terms could have been, and were, basically interchangeable. What was important in the 1860's was to have a noun were there was none before.

More recently F. Churchill has described how in Germany, after 1880, the need for a noun that emphasized the focus on hereditary transmission mechanisms made theoreticians use "Vererbung" instead of "Erbrecht" or "Erblichkeit".[7] He also indicates, with some surprise, that in German dictionaries none of these nouns appear to have received any attention, in their biological sense, before the last decades of the 19th century. But this is far from being exceptional, because only in France did biological "hérédité" become a focus of general attention early on in the century. Against what Churchill states, British cyclopaedias and dictionaries, both medical and general, did not have entries under "heredity" or "inheritance" until the very last decades of the 19th century, when almost all of them where written by the Scottish

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[2]Morley, 1597: "The fault which like unto a hereditaire (gallicism?) lepresie in a mans bodie is incurable"; *Misaurus*, 1699: "I have heard you confess that yours is a hereditary gout". OED, 1989, 2nd. Ed., vol. VII, p. 544. The medical origin of heredity as a biological concept was perceptively noticed by the historian Emanuel Ràdl, who wrote that "Before Darwin's time", biologists left the work on the problems of hereditary transmission "to their medical friends". See Ràdl, 1930, p.242.

[3]"It seems hardly credible, Galton wrote in 1908, that the word heredity was then [in 1869] considered fanciful and unusual. I was chaffed by a cultured friend for adopting it from the French". See *Memories of my Life,* p.288.

[4]R.S. Cowan, (1972), "Francis Galton's contribution to Genetics". See also her thesis (1969) *Sir Francis Galton and the study of Heredity in the 19th Century.*

[5]See "Heredity" in OED, 1989,vol.VII, and in *The Century Dictionary,* vol.III, 1889.

[6]The marginalia of Darwin's copy of Lucas' *Traité de l'Hérédité Naturelle*,(1847-50) (U.L.,Cambridge) are full of occurrences of "heredity", instead "inheritance" which he used in his writings.

[7]F.Churchill (1987), "From Heredity Theory to Vererbung", p.338.

physician J.A. Thomson.[8] The first time the Encyclopedia Britannica commissioned an article on "heredity" was very late: 1911, by Peter Chalmers Mitchell.[9]

Such situations in Germany and Britain contrast sharply with the situation in France, where after 1830 no medical dictionary, and after 1850 almost no general one, failed to include an entry under "hérédité". French medical men were the first to adopt and popularize the noun in its specialized sense. "Hérédité", with its ontological and causal implications, spread from the medical to broader circles through the increasing weight it received as an explanatory resource in the technical, programmatic and propagandistic texts of the post-revolutionary waves of French physicians. Alienists, criminologists, hygienists, and other socially oriented branches of the medical profession found the shift from an adjectival approach to the hereditary to a substantive one a very attractive move.[10]

The popularity of the subject of hereditary diseases grew steadily among French medical students after around 1815, as can be seen by the increasing number of final dissertations on the subject in both Paris and Montpellier. But the crucial moment was signaled, I believe, when after 1830 such dissertations, ever on the increase, switched their adjectival formulations in their titles (and treatments) from variations of "les maladies héréditaires" to a substantive "L'Hérédité dans les maladies".[11]

This change from adjective to noun points to a change from analogy (or metaphor) to a direct, ontological commitment to the reference of the concept. In other words, a reification process that perhaps began many centuries earlier (with the Greek medics' adoption of "hereditary") came with such shift to a conclusion.

To describe the major features of such reification process, from "the hereditary" as a metaphor to "heredity" as an explanatory biological concept that implies as particular kind of independent causation (mechanism, force), is the aim of the present chapter.

1.2 "The hereditary", an external boundary for generation theories.

The empirical set of facts that from antiquity were considered as belonging to "the hereditary" can be assorted in three categories: the resemblances between parents and offspring that give a "family air"; the strange combinations of characteristics that are the product of hybridizations; and the familial pattern of occurrence that certain deformities and diseases adopt. For the sake of the argument, but following closely an association made by many antique and post-Renaissance authors, I will call them collectively as "the hereditary" (but without the quote marks).

The close associations among these sets of phenomena were recognized and handed down within the Hippocratic and Aristotelian traditions. The hereditary was eventually identified with all the characteristics of the constitution or temperament of the body that were in some way or other affected by the parent's constitution:

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[8]From 1885 to 1900, Thomson contributed articles under the head "Heredity" for *Chambers, Blackie's, Nelson's* Cyclopaedias, and the *Encyclopaedia Medica.*

[9]11th edition, vol. XIII, pp.350-354

[10]For a recent sociological analysis of this phenomena in post-revolutionary French psychiatry see D. Pick (1989) and I. Dowbiggin (1991).

[11]See below, appendix 1.

characteristics that existed, actually or potentially, in the new being since its first rudiments. The transmission, or at least the causal relation between parents' and offspring's bodily features, and its consequences was presupposed. The hereditary was thus not limited to the pathological, although the adjective does appear more frequently related to disease or deformity.

The curiosities and vagaries of family resemblance were perhaps the first natural object of the analogy we are dealing with. Resemblance, with its surprising details, its astonishing recurrence in members of a family of like tones of voice, gestures or bodily movements, was always an object of attention, perplexity and speculation. In almost any cultural tradition it is possible to find some kind or other of wisdom as a product of the observation of the patterns of similarity between the generations and within familial groups.[12]

Dissimilarity is the contrasting and also striking companion to such patterns. The detailed and sometimes stubborn manner with which some often irrelevant features are sometimes preserved through several generations, contrasts acutely with fact that only some, but not all, the descendants inherit them and in a somehow haphazard way. The close observation of patterns of resemblance and dissimilarities within families and broader genealogical groups produced then, as always, paradoxical views. According to the Greek philosophical essentialism, the hereditary was not an easy target to assimilate. The capriciousness and irregularities of family resemblance could not easily be accommodated to, for instance, Aristotelian typologies of characters and causes. That sometimes secondary ("accidental") features were at least as persistent and predictable in their genealogical behaviour as those characters that were considered as more essential was bad enough. But that undesirable deformities, defects, illnesses, etc. seemed often to follow the same paths and patterns, mystified the natural philosopher (Aristotle) and the physician (Hippocrates) alike.

Their attempts at providing coherent physical and metaphysical accounts of the human (and animal) existence faced both authors with the facts of the hereditary, especially when describing their views on "generation". For them, as for the 18th century theorists long after them, the hereditary —the facts of resemblance, hybridization and familial diseases— constituted a part of the phenomena they were supposed to "save" with their theoretical elaborations.

A puzzling related phenomenon was the existence of relatively stable subgroups within the boundaries of a species; and in the particular case of humans, the hereditary physical and moral characteristics that made —and preserved— the differences between human groups, (nations, races) and how they could be "mixed" in the individuals product of their interbreeding.

Basically, under what Ernst Mayr (1982) has described as an essentialist view of biological species, the homogeneity of the genealogical groups through the generations is to be assumed and all the inner (specific, group, familial) irregularity, variation and diversification becomes a surprising irregularity in need of explanation. In that sense, most features of a given organism should in principle resemble closely those of its parents, and any dissimilarity should have some kind of check or other. Such a situation, from the beginning of times, would preclude deviation or degeneration of type in its Aristotelian sense.[13]

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[12]See B. David, 1971, pp.12-19.

[13]For Aristotle's view of degeneration see Buffon's exposition, in Lanessan's edition of the *Oeuvres*, tome 4, pp.469-504.

Variation, and not similarity is the oddity then. As Henry Holland, the British early Victorian physician put it

> While we find cause for wonder at the transmission of resemblances from parent to offspring, we must admit the wonder to be equal that there should ever be deviation from this likeness, and that such deviation should be so little governed by any apparent rule or law. The one case is in reality as great a miracle to our understanding as the other.[14]

Charles Darwin marked with double lines this particular passage in his 1855 copy of Holland's book (though not on the 1839 one) and later rephrased it in his chapter on inheritance in his *Variations of Animals and Plants* (1868), with a curiously shifted emphasis. While for the physician homogeneity and variation in hereditary transmission constituted equally wonderful mysteries, for Darwin (as for Aristotle) "the real subject of surprise is ...not that a character should be inherited, but that any should ever fail to be inherited".[15]

The makers of systems have always found irregularity and unpredictability a pain. At least since Empedocles' time, for anybody in the business of fashioning an account of human (and animal) generation, the paradoxes of the hereditary were a serious stumbling block. Aristotle's view of the male seed as the only causal contributor to the shape (form) of the body of the offspring had to find ways to by-pass the empirical evidence of female transmitted characteristics, such as resemblance to mothers, hybridization, etcetera.[16] The most convincing account of the irregular mixtures of resemblances to both parents was given by the views of generation that Boylan calls "dual seed theories".[17] Some kind or other of them were maintained, in their various fashions, by among others Empedocles, Democritus, Pythagoras and Epicurus. But by far the most influential version of a dual seed theory was the one put forward by Hippocrates and re-shaped centuries later by Galen. It became the standard view of generation for all medical men.

Since offspring of inter and extra typical crossings can, and often do, resemble both male and female parents; since any kind of characteristic (vague as a family air or precise as an extra finger, essential or accidental) could apparently be hereditarily transmitted; and since there seemed to be a sort of combinatorial, or at least commingling, for the reassortment of the parts, properties and characteristics of the parents in the production of each new organism, the hypothesis that came to be known as "pangenesis" was the natural complement to the dual seed (or double semen).[18] Both seeds are thought, under such view, to be the product of a separation, in the gonads, of parts or particles coming from all over the body, in such a way that each and every part is represented in it. Conception was then thought to be a getting

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[14]"On Hereditary Disease" in *Medical Notes and Reflections*, 1839, p.14

[15]"Inheritance", Chapter XII of *Variation...*, (1868) vol.II, p.2

[16]Aristotle's difficulties with the hereditary have been described by Morsink (1979), Boylan (1984) and by Jacques Roger (1963), pp.81-91.

[17]M. Boylan (1984). Others call these hypotheses double semen, double seminal, or double semence.

[18]Democritus (in David's French translation): "La sémence...est constitué de tous les éléments du corps et surtout des principales parties (os, chaire, veines). Les parties du foetus communes aux deux sexes proviennent aussi bien du père que de la mère: la liquide sèminal répandu par le deux sexes est élaboré dans les testicules chez les mâles et des organes analogues chez les femelles.

together of the two seeds, and a process of bargaining between the male and the female parts decided in what kind of a combination or mixture the offspring would result.[19]

In a great measure, it was, I believe, for giving weight to the hereditary in the shaping of their views on generation, that the double seminal views were favoured amongst physicians. Instead of dismissing the evidence of hereditary transmission as insignificant due to its accidental character, they chose to privilege the day to day facts they faced during their practice. Among them, the recurrent familial patterns of certain diseases.

Though philosophically and physiologically dubious, the leap of postulating the existence of a female semen (analogous to the male one and equally liberated during intercourse) was seen by medics as completely justified on account of the failure of all other views of generation to clarify the hereditary. This argument was to reappear in the 18th century in Maupertuis' *Vénus Physique* (1745).[20]

Given the strength of the Hippocratic-Galenic tradition in Western medicine, the double seminal account of generation, with its small but relevant "hereditary" empirical support, came to have a deep and lasting influence on Western science. All the way up to the end of the 18th century, parallel to the discussions on preformation and epigenesis, medical men maintained a relatively independent approach to generation and the hereditary, only touching the mainstream discussion in isolated cases, through medics turned philosophers, as with Haller.

The Hippocratical solid-humoral physiology, with its conception of the body's properties and dispositions based on the theory of temperaments (or constitutions), provided the frame for such long-standing tradition. Within it, humoral or humoral-solid causal "mechanisms" were possible as bearers of the hereditary influence through the generations. Temperaments themselves were considered to have a strong "hereditary" character, as a product of a mixture of the initial humors, or semens. The unstableness of the hereditary, its irregularity, was easily relatable to the character of the influences: fluid, soluble, miscible, etcetera.

The experience of family resemblance and family disease was on other hand common to medical men, who often treated members of several generations of the same family and experienced first hand the most striking cases of hereditary transmission.[21] This complementary situation —of a relatively uncomplicated

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[19]See Boylan (1984), David(1971), Zirckle (1936).

[20]The search for the female semen occupied the speculations and anatomical research of many workers up to Buffon and Needham. The hereditary as a a serious difficulty of most non dual views of generation was stressed by all important 18th century writers ,i.e. Diderot, Maupertuis, Buffon, Haller, Bonnet. See Roger (1963).

[21]The importance of their observations in this area was enhanced when they had to deal with nobility, and Royal families (as the most prominent of the profession tended to). Discussions, for instance about the suitability of certain marriages etc. had for a long time been related to medical opinions about the hereditary character of certain good or evil qualities, and in many cases they could become matters of State. An example is Lyonnet's (1643) *Treatise on Hereditary Disease*, which was mainly written to appease the King Louis XIII. Other royal physicians interested in hereditary diseases were: Jean Fernel (1497-1558), doctor of Diane de Poitiers and Henri II; André du Laurens (1550-1609) physician of Henri IV; Luis Mercado

description of transmission and very striking cases, both in authoritative literature and everyday experience— gave theoretically minded physicians a strong resilience to withstand criticism from theologians, philosophers, and natural historians who from different perspectives saw their double seminal account of generation as flawed.

In acute contrast with most physicians' attitudes, the makers of systems did not as a rule pay too much attention to the facts of the hereditary. Due to their direct relevance to the discussions and theories of generation, they tended over the centuries (from Aristotle to Charles Bonnet) to find the evidence for hereditary transmission as a hurdle. The clean, *a priori*, schemes of how Nature (or the Creator) ought to proceed were usually at odds with any focused attention on the behaviour of particular characteristics over the generations, such as hereditary disease, resemblance, etc. The real, and apparent absurdities and contradictions of many of the empirical hereditary claims (like the chimeric crossings of very distant species) made more or less easy their neglect by some authors, and philosophical grounds (such as the incompleteness or impossibility of certain kind of knowledge) contributed to the isolation of the hereditary as an area either immature or irrelevant.

It was not until the 18th century brought to head the strongest discussions around the theories of generation that the role of the hereditary began to be emphasized. There had been since the first years of the 17th century, for some reason, a reawakening of the interest among medical men in the issue of hereditary diseases, and several authors, important and marginal, had published treatises on the subject.[22] The evidence collected in them, and the other facts belonging to the hereditary, were recognized early in the 18th century by several authors as damaging for the prevailing preformationist orthodoxies. Hereditary transmission of characteristics, i.e. a sort of causal link between the bodily properties of different —though related by parentage— organisms, was in principle repulsive to the idea of preexistence, and, although in a lesser degree, that of preformation. The fact that most hereditary observations pointed towards a contribution from both parents made for a stronger threat.

Chambers' 1738 dictionary mentions, in its entry for "Generation", that Sir John Floyer "starts a difficulty, which seems to press equally against each system (ovism & animalculism), taken singly". Floyer's difficulty is the fact that mules (which he classifies as monsters) partake of the characteristics of both horse and ass, and that the defenders of both systems artificially choose the characters that favour their view as important for the determination of the origin of the foetus, making secondary the characters conveyed by the sex opposite to the one they favour.[23]

When Diderot was preparing in the 1750's his *Élemens de Physiologie*, he decided to assign a special weight to the hereditary in the evaluation of the several systems of generation he intended to describe there. The difficulty that preformationist views had in dealing with "maladies héréditaires; ressemblance des parens; mules et mulets qui engendrent"[24] was particularly highlighted by him in those notes. He was probably following here the trails of both Maupertuis and

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(Mercatus, 1513-1599), doctor for both Felipe II and Felipe III; in England Henry Holland (1788-1873) was the physician of Queen Victoria for some time.

[22]See Bibliography on Hereditary Disease in the appendix 1.

[23]See "Generation" in *Chambers Dictionary* (1738), vol I, 2nd. edition.

[24]See "Génération", chap. XXIV in Diderot's *Élemens de Physiologie*, 1964, ed. critique by Jean Mayer, pp.182-185.

Buffon, who famously had, a few years earlier, used resemblance to both parents, transmission of polydactily, mules, and other hereditary cases as evidence against preformation, and for a new kind of double seminal, successionist system of generation based on some organizational natural principle.[25]

Diderot, in his adjudicator's stance, knew well that even if the double seminal views could account with more ease for the hereditary, they had serious problems of their own when facing actual, anatomical observations, and detailed physiological questioning. He writes, for instance "Dans ce sisteme placenta, et envelopes impossibles a expliquer". This is the kind of criticism that both Haller and Bonnet would forcefully make, just a few years later, against double seminal, successionist (epigenetic) positions.

What is important to point out now is the different character of the empirical facts that posed problems for the competing approaches to generation. While detailed observation of the organs of generation, and of the development of the embryo, backed strongly the preformationist (specially the ovist) position, dual seminal accounts were favoured by what may be called "genealogical" observations: that is, the observation of the patterns of similitude and difference in organisms with a genealogical relation. While the first kind of observation implies a focus on the individual, its origin and its characteristics, the second one implies a higher level, comparative perspective.

The latter kind of observation is the basis, of course, of the claims for the existence of a hereditary relationship between different organisms, and/or between their characteristics. It presupposes that the focus of attention be a more or less well defined characteristic of which similitude or dissimilitude could be claimed between two related individuals. What kind of characteristic could be a candidate for genealogical observation could vary widely from very general and vague resemblances of shape, form, or aspect, to precise characters like an extra digit, a big mole on the cheek or a crooked nose, and on the pathological side from general tendencies to unhealthiness to precise ailments that develop in the same manner and age in related individuals.[26] The genealogical approach to evidence and observation opens up the possibility of setting exterior limits to physiological speculation, in

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[25]Maupertuis, *Vénus Physique*, 1744; Buffon, *Histoire Naturelle*, 1749. See also Hoffheimer (1982). I use "successionist" following Antoine Louis' term when referring to the belief that each new organism was organized anew at each conception, and thus creation is successive and not simultaneous, as in pre-existence. A common usage at the time to refer to this view was "epigenesis", but several historians have argued that such use is confusing as it does not distinguish between instantaneous organization and developmental (epigenesis in its modern sense) hypotheses. Successionist includes both the latter and opposes only pre-existence. See Bowler (1971), Gasking (1967), and Roger (1963) for elucidations of the different 18th century generation theories.

[26]By the beginning of the 20th century, after the works of Darwin, Galton, Weismann and Mendel, among many others, the idea of a hereditary character acquired a different sense, related to what we call Genetics. Its previous, mainly physiological sense is lost to most modern thinkers, who have to avoid an anachronistic (genetic-like) approach when referring to hereditary transmission in pre-Darwinian times. This point has been very forcefully made by Berthélemy-Madaule with respect to Lamarck and the transmission of acquired characters. See her 1982 text, ch.4, p.72.

contrast with the interior limits set by dissection and microscopy. The gathering of convincing cases of hereditary transmission of a wide range of different characteristics, and the progressive closing of alternative avenues of dealing with them (such as their ascription to chance, or their sheer irrelevance), was one of the central themes of the 18th century debates around generation. Bonnet's very complex and sophisticated ovism, in which many elements of the dual seminal views are incorporated, is in a sense a product of the strains put onto it by the external, hereditary, evidence.[27]

When Maupertuis decided, in the first anonymous version of his *Vénus Physique*,[28] to chose the hereditary as his main weapon against preformation and preexistence, he was not making the breakthrough that many historians of genetics have suggested he did.[29] The double seminal view of generation, as transmitted by many generations of physicians, implied from its beginnings a strong reliance on the hereditary.

One of Maupertuis' main contributions can be said to have been his restating of the dual seminal hypothesis along the lines of a mechanistic approach (which he, like Buffon shortly after, claimed as Newtonian), in which he tried to imagine (visualize) a way in which particles from the two semens could both be mixed and interact in such a way as to produce organization, differentiation, etc. This was also his weak point, as the strongest attacks to his position (by Haller, for instance) were directed to it

With hindsight, however, what seems most important and surprising, in his compact and clear argumentation in the *Vénus Physique*, is the restructuring and hardening of the external bounds that the genealogical (hereditary) approach to observation made to preformationism through the careful analysis of its causal logic. A simple probabilistic argument (which was once erroneously compared to Mendel's)[30] with the very well chosen example of polydactily of the Ruhe family in Berlin,[31] strengthens enormously the case of the double seminal view. He closed for its opponents some traditional evasive routes, like "chance" coincidences, or the vagueness of the inherited characters. To defend exclusive maternal or paternal influences over the embryo's formation became more difficult after that. In short, by

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[27]For Bonnet's theory of generation see his *Considérations sur les corps organisées*, Bowler (1973) and Roger, 1963, Ch.IV, p.712.

[28]Originally, and revealingly, called *Dissertation physique à l'occasion du Nègre-Blanche,*(1744) and motivated by a desire to explain the existence of albino individuals among human blacks.

[29]Maupertuis has been anachronistically taken to be another mythical precursor by many authors, since Hervé's reappraisal of his work his "Maupertuis génétiste" (1911), and Bentley Glass "Maupertuis —pioneer of Genetics and Evolution"(1959). I. Sandler has written a thesis showing what should have been, but wasn't, obvious: that Maupertuis was no geneticist, but a sharp 18th century thinker. See her (1979) thesis, and (1983) paper.

[30]See Sandler (1983) and chapter 6 below.

[31]Maupertuis took the idea from Réamur, who had argued that observations on the hereditary transmission of some anomalous characters could help tip the balance in favour of one or another of the preformationist positions. See his *L'Art de faire éclore...*, tome II, p.335-336. Réamur himself later did a very similar research on transmission of polydactily within a family.

focusing on a very distinct and unmistakable character, whose inheritance was seen as improbable (as a monstrosity), and proving its transmissibility by both the paternal and the maternal routes, Maupertuis tidied up the act of the (mainly physicians) defenders of the double seminal view, who had for many years used similar arguments around shadier similarities, using selected case stories either factually open to doubt, or inconclusive in their causal claims. Causal hereditary links never were easy to prove, but they became more so after Maupertuis (see chapter 6).

In the end, however, Maupertuis was not (could not be) interested in postulating a law of heredity or developing a theory of it. Neither, for that matter, were Buffon, Bonnet, or Haller. For them the hereditary was in a sense the same as it had been for Aristotle: a lateral set of facts that had more or less bearing in the generation of organisms. Up to and until the end of the 18th century the first formation of a living being was the real mystery, the source of awe and the target of explanatory speculation; the hereditary was a secondary collection of occurrences that would eventually be explained away by an adequate view of the fundamentals of life. As Jacques Roger wrote regarding the theoretical tasks of 18th century French naturalists:

> la science de l'époque ne se préoccupait pas vraiment des questions d'hérédité et d'hybridation...Le grand problème a ses yeux était la formation de l'être vivant, considéré comme un individu isolé, sans rapports avec les individus de même espèce qui l'avaient précédé et engendré.[32]

Heredity, it must be added, was not a possible question. Even to start to pose the problem of heredity as a target for autonomous theorizing, an independent, or at least, partially isolated field of regularities has to be recognized. To be conceivable, the idea of a law or a force of heredity requires the stabilization of a domain, the structuring of a differentiated set of facts and the presumption of an exclusive causal connection between them. The hereditary until the very last years of the 18th century was not such a domain. It preserved much of its basic analogical (non-explanatory) origin, and —notwithstanding Maupertuis' or Buffon's clarifications— did not suggest to most writers the necessity of postulating an autonomous set of laws or forces for it.

The exception, again, was to be found amongst medical men. It was in their ranks that the major distinctions were made that began to give shape and structure to the hereditary, and to push it towards forming an area of independent scientific enquiry.

1.3 1600-1800, Medical Men and the Hereditary. An overview

In their conceptual quest to make sense of the idea of an hereditary disease, and restrict its boundaries as clearly as possible, Hippocratic-Galenic physicians were forced, long before other naturalists, to focus on genealogical patterns of character transmission.[33] A main objective for them was to have criteria to distinguish those

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[32] J. Roger, 1963, p.388

[33] There is a problem, that will be treated extensively in next chapter, in the equating of normal characters and pathological ones, like a disease. The allegation that what is hereditarily transmitted is not the disease itself but a constitutional disposition to it,

diseases that could properly be called hereditary from those which, sometimes adopting similar patterns of occurrence, were not transmitted in the act of generation and had to be classified apart. As a consequence, observation of the characteristics, and development of candidate diseases, became increasingly crucial to the discussion. The timing and duration of their attacks, for instance, were seen as important telling factors concerning the origin and ultimate cause of the disease. One that would have been acquired at the moment of conception by parental influence (the main criteria for its hereditariness for most authors)[34] would typically be a constitutional, chronic disease, and manifest itself at the same age, more or less, in the offspring as it had on the affected parent. The more careful authors tried to discuss and eliminate the other possible, non-hereditary, causal influences.

In the process of clarifying what the adjective hereditary meant to them, the medical men began to give structure and causal meaning to what had been until then a purely descriptive term. This process seems to have gathered momentum after 1600, when medical physiologists were trying to adjust their traditional Hippocratical (humoral-solid) views of disease to the less dogmatic and more empirically oriented environment of the post-Renaissance.

As a base for the discussions on hereditary disease, the collection of relevant ancient and modern evidence, registered in the form of more or less trustworthy cases in the literature, was increased enormously during those years (1600-1800), and it eventually became, in itself a very powerful tool against the sceptics, who from different quarters tended to dismiss the hereditary claims as delusions or impossibilities.[35]

Besides the evidence gathering activity, those medics interested in hereditary transmission, as mentioned already, embarked on the production of theoretical distinctions based on such evidence, distinctions that began to give the subject a more sophisticated profile. Their need to locate the possible causal routes to disease made them realize that a simple external pattern, familial, group, regional, or other, was not enough for claiming an hereditary cause for a disease. In other words, their need to establish clear criteria for recognizing the hereditary diseases (which they identified with constitutional ones) from those “acquired” after conception (both congenital, and post-natal), forced them to focus on peculiarities of original source, chronology of appearance and recurrences, and permanence (or chronicity). The consequence of this was that by the end of the 18th century there were circulating among medical men, mainly but not exclusively in France, a series of quite sophisticated distinctions, and definitions of what is meant for a disease, and a character in general, to be hereditary, than were usual in other circles.

The roots of the criteria developed by 18th century physicians in their attempts to

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made clearly by physicians since the 17th century, however made a long part of the way towards the unification of pathological and non-pathological hereditary transmission.

[34]I will describe in more detail medical authors and their views on hereditary disease, from 1600 to 1800, in the following chapter.

[35]In the mid-18th century several important medical authors were still very sceptical about the real possibility of there being any causal communication (through the seminal fluids or other) between the temperament or constitution of parents and that of the children. Antoine Louis articulated very lucidly these doubts in his 1748 essay *Des Maladies Héréditaires.* See following chapter.

define the hereditary can be traced back to the writings of Hippocrates and Aristotle, but became increasingly stringent and logical in order to exclude the non-hereditary. The more subtle and careful thinkers, amongst medics, wanted to claim as properly hereditary only those constitutional characters and diseases that, coming from any one of the parents, established themselves as part of the organic constitution (solid and/or humoral, this was a matter of dispute) before the first solidifications of the seminal humors in the womb gave rise to the new individual in all its complexity. For other authors, other sources of parental humoral influences, beside the seminal ones, had to be considered within the realm of the hereditary: the maternal ones via the placenta, and for the more inclusive or least discriminatory of them, also the influence of lactation on the newborn's constitution. Non-humoral sources of influence were also discussed, like the emotions and imagination of the mother.

Discussions between those exclusivists (who wanted hereditary to apply solely to a causally restricted class of disease) and the inclusivists (who wanted the term applied in a general way to all familial illnesses) were of course not easily brought to a clear-cut edge, dependent as they were on the unstable and oscillating views that physiologists were producing at such a time, when alchemy was on the wane and the new chemistry still tentative and insecure. On the other hand, the acceptability of non-material influences, or at least non solid-humoral ones (like vapours), was always linked to wider philosophies of life. However, resorting to careful genealogical and etiological observation was always open, and was used in order to try and establish the route that a given character, or disease, could have taken to go from one or another parent to the offspring, and to establish the moment it became part of the latter's constitution or temperament.

The latency of hereditary causation, for instance, posed a crucial philosophical, and physiological problem. It had always been a source of mystification and confusion, at least since Aristotle described with surprise the atavistic "jumping" of a generation in the descendants of a mating of a white woman with a black man. Latency, that is, the existence in the organism of a "causal element" without any manifestation of its effects, was seen not only in atavism (which came later to be called "reversion") but also in more common hereditary occurrences, like the bodily changes —such as dentition, puberty, balding or certain kind of blindness— which always are triggered with the arrival of certain age (homochrony is the technical name introduced later by Haeckel) before which they remain hidden. The existence of latent causes and some kind of timing that controls, for instance, the triggering of second dentition, was thus used to justify the belief in latent causal elements, both for certain normal characters and for dispositions to certain diseases. The idea that in some individuals such causal elements remained hidden all their life, and that they could however pass them on to their descendants, accounted for atavism.

Similar philosophical and physiological subtleties of causation were usually a main component in the works of the more reflective amongst 17th and 18th century physicians, and some of them were particularly sharp when addressing hereditary transmission, where elements like irregularity or uncertainty of effect, and the talk of potencies, predispositions, indirect or multiple causation, gave ample space for both argument and sceptical denials, all of which asked for better and clearer definitions.

At the same time, the often striking and convincing accumulation of cases of hereditary transmission of disease, which physicians tended, sooner or later, to witness themselves, provided the certainty that theoretical considerations did not. The difficulties of imagining, and developing a convincing account (with all the

theological, philosophical and physiological obstacles) had thus as a counterpart the conviction that individual cases, both quoted by authorities in the literature and witnessed personally, gave to most of them. Accident was not taken seriously by most of them as an alternative to some kind or other of causal link, no matter how "accidental" some of the hereditary characters were.

All in all, the hereditary never ceased to be for 18th century physicians, as for other naturalists, a puzzling collection of empirical facts. Their attempts at constraining it within a rational, coherent framework made them conscious of the advantages of collecting and organizing genealogical evidence, and in the somehow independent status that it had in relation to anatomical, physiological and philosophical considerations that guided the very difficult discussions around generation. For some of them, the uncontroversiality they felt the facts of hereditary transmission of disease had, even allowed for a relative by-passing of any generation theory, and for a sort of wait on the sideline to see whatever, if anything, came out of them; only asking, of course, that the physiologists' theories be coherent with their views of the routes of transmission; in other words, with their double seminal (or at least double causal) conclusions.

This sort of parallel and obscure existence of the debate on the hereditary within the medical community lasted until the first few decades of the 19th century, when a change of circumstances in post-Revolutionary France brought it to light. Due to a complex social, political and professional reorganization of the French medical profession, that has recently been analyzed by several authors,[36] the hereditary emerged as a powerful explanatory tool that gained increasing notoriety outside the medical circles. It did not emerge however without a structure. The groundwork, so to speak, that had been done by physicians during the previous decades gave the idea of hereditary transmission, as used by early and mid 19th century medics, a complex, causal, explanatory structure.

It was then, in post-Revolutionary France, that hérédité, as a technical noun, began to occur with increasing frequency. It became a synthetic and inevitably reifying concept that eliminated the need to use the adjectival form constantly, and reflected the importance that the subject was acquiring in the period. Its implication of the existence of a particular, independent, causal mechanism or force responsible for the hereditary phenomena was taken on board as a reality by some of the French medical men, who started, in the Montpellerian tradition, writing about laws and forces of hérédité.[37] The subgroup among French physicians that perhaps was more interested in promoting hérédité as self-sufficient explanatory resource seems to have been the mental doctors, followers of the famous Pinel and Esquirol, that called themselves alienists. The multisided appeal that it had for them has been thoroughly described by Dowbiggin (1991). They took hérédité as a bottom line for their analysis of mental illness in early 19th century France and began reorganizing the enormous body of evidence, that had been collected over the centuries, in favour of what we now describe as an hereditarian approach to human society.

Eventually, it was among their ranks that the first fully fledged, independent and considerably self-contained theory of heredity was produced: Prosper Lucas'

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[36]See particularly Daniel Pick(1989); and Ian Dowbiggin (1991).

[37]For an excellent account of the Montpellerian school of vitalist theorizing see J.Roger, (1963) Second Part, Chapter I., p.163 . See also M. Staum's *Cabanis*, (1980), Duchesneau (1982), Guyenot (1941).

influential *Traité de l'Hérédité Naturelle* (1847-50).[38] This extensive work synthesizes and evaluates the immense number of recorded facts and discussions that so many decades of interest in the hereditary by physicians and naturalists had produced. In its two volumes Lucas managed to accommodate, rather indiscriminately for some tastes, an enormous amount of the confusing and contradictory evidence that was dispersed in books, and to provide at the same time a rationalistic scheme in which to make sense of it. Based on a Montpellerian (vitalist) approach to his theme, Lucas concludes that only the postulation of two opposing forces, a conservationist one (hérédité) and a variational one (innéité), acting in different moments and combinations, can produce an understanding of the irregularities of resemblance on the one hand, and the striking cases of capricious reappearance or loss of characteristics between the generations (mediate or immediate) of a family. By the time Lucas published his work, the process of reification of heredity that I have summarized above was complete. The metaphorical character of the hereditary had acquired an ontology in itself, and this was reflected in the coinage of a noun. Very few people were sceptical of the reality of heredity, and the mental phenomena, which alienists and phrenologists claimed to be knowledgeable about, were considered by many physicians and naturalists, to be well within its causal reach.

Heredity was first and foremost linked to preoccupations concerning human beings, as a biological species. The implications of any view of hereditary transmission of normal and pathological qualities for the understanding of humanity was always a main issue when the subject was treated. The unavoidable links between genealogical transmission of physical and mental characters and the structure of societies in familial, regional and national and racial groupings, with differing sets of characters between them, were made from the very start. In the 19th century they were certainly the issues which pushed the subject to the forefront of many scientific and social disputes.

1.4 What the dictionaries say

The story of the reification of biological heredity that I sketched above, can be verified, so to speak, by tracing the history of the terms "hereditary" and "heredity", in their biological sense, in European medical and general dictionaries and encyclopedias. By following both the sequence of their appearances, and the way in which their definition was successively gaining in importance and complexity, we can have a very reliable and clear overview of the structuring of the hereditary I have talked about above. In its first appearance in a French medical dictionary, early in the 19th century, the noun hérédité was already carrying a heavy luggage of definitions, and re-definitions. This we shall try to show now.

The reification process, as was stressed, took place mainly in the restricted context of human hereditary transmission of very striking constitutional (or bodily) qualities: family resemblance, particular malformations, marks, etcetera. But it was the tendency to inherit certain diseases that first took the adjective "hereditary" to the dictionaries. The pathological sense was the first to acquire a technical status, in the phrase "hereditary disease", and the pathological connotations, we shall see, were

[38]The semi-complete title is *Traité philosophique et psychologique de l'Hérédité Naturelle dans les états de santé et de maladie dans le système nerveux...*

always central to human heredity.

The revival of interest in hereditary disease at the beginning of the 17th century produced several treatises in which the Latin formula "haereditarii morbi", or a similar variant, was employed in the title.[39] The medical dictionaries of the period reflected what seems to be a slow increase in the use of the phrase by the medical profession a few decades later. In Britain, Stephen Blancard's *Physical Dictionary* only incorporated an entry under *Haereditarii Morbi* in its 5th edition, in 1708,[40] where they were defined simply as those "which the parents had, and have already seiz'd the children, as the Gout, Consumption, and Stone."[41] This definition is basically descriptive, giving only the familial pattern as criteria for the hereditary, and fails to use any of the distinctions, causal or other, that several 17th century authors had developed (especially the Irish physician De Meara). The examples are the only substantial part of such definition, as they emphasize the constitutional, chronic character of the hereditary diseases.

A slightly more detailed definition is to be found in John Quincy's (1736) *New Medicinal Dictionary* : "Hereditary Disease is such as is transmitted from the Parents in the first Rudiments of the Foetus, which is the origin of many Chronik cases".[42]

Two years later, in 1738, a British general dictionary follows suit. In Chambers' celebrated *Cyclopedia*, after the definition of "hereditary" in its first, legal, non-metaphorical sense,[43] is added the following:

> hereditary is also applied figuratively to good or evil qualities, habitudes, etc., capable of being transmitted, by blood, from father to son. The gout, king's-evil, madness, etc., are *hereditary* diseases, i.e. are transmitted from the parents in the stamen or first rudiments of the foetus. And such, probably is the origin of numerous other chronic diseases[44]

As can be seen, some important restrictions are displayed both by Quincy and Chambers to the sense of "hereditary disease". Their definitions, in emphasizing that it must be a constitutional disease whose causal element is already present in the very first formation (stamen, rudiments) of the embryo, eliminate from the set many diseases that, adopting a familial pattern, were sometimes called hereditary, but which

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[39]Ludovico Mercatus, 1594; Dermutius de Meara, 1619; de Bourges, 1621; Cörnerus, 1627; Janus, 1627; Crüger, 1636; Lyonnet, 1643. For a complete list, see appendix 1.

[40]The 1st edition being from 1684. Stephen Blancard, *A Physical Dictionary, in which all the terms relating either Anatomy, Chirurgery, Pharmacy, or Chymistry are very accurately explain'd, London, printed by J.D.*

[41]These three diseases were typical of the sort more commonly believed to be hereditary. These were diseases that depended on flaws in the Temperament or Constitution of the individual, that is on some kind of fundamental defect (or "taint") in the organization of the body.

[42]John Quincy, 1736, *Lexicon Physico-Medicum.*

[43]"something appropriated to a family, or belonging thereto by right of succession, from heir to heir".

[44]Chamber's (1738) *Cyclopedia*, or Universal Dictionary of Arts and Sciences, vol. I, 2nd ed.; Chronic and Constitutional Diseases were seen as being basically the same. As product of organizational (temperamental) imbalances or defects, they are quite more difficult to eradicate, though less dramatic, than Acute or Acquired (invasive) diseases, which are sudden and relatively superficial.

were known to be contracted from the parents after conception, either in the womb or through the maternal milk. This approach to classifying disease was further extended by Chambers when, in another article of his dictionary, he writes that according to their cause (constitutional, chronic) diseases can be divided into "hereditary, connate or acquired".[45] The first class refers to pathological influence carried in the male or female semen (or seed) which exists at a time previous or simultaneous to conception and is incorporated into the child's constitution. The second one to prenatal constitutional alterations due to morbific elements (humors) transmitted via the mother's blood through the placenta and which affects the still not "solidified" structures of the embryo. The last one refers to any *post-partum* influence that could exert dramatic constitutional alterations.[46]

This emphasis on differentiating the (causal) routes of transmission in such a clear-cut manner, based on mainly "external" evidence, such as timing of outbreak, kind of affection and patterns of re-occurrence, did not however convince many medical men. The physiological theories in place (with their humoral-solid bases), did not help restrict the possible causal routes, and the big, crucial problem of determining how and when "the first rudiments or stamen" of the embryo was actually formed (the prestigious and overwhelming problem of generation). This gave ample room for discussion and dissent between different positions. Perhaps the constitutional disease that received most attention for it's hereditary pattern was scrofula.[47] Some medical dictionaries reserved their discussions on the hereditary to the entry for this illness. This is the case in R. James' *A Medicinal Dictionary* (1743-45),[48] from which I will quote at large in order to show the intricacies of the issue of hereditary transmission under the early 18th century medical viewpoint. James' dictionary begins by locating"Scrofula" as a candidate for being an authentic hereditary disease, as it exhibits the characteristic familial pattern, and as it always manifests itself at roughly the same time:

> so this disease seems owing to a hot, sharp Humor propagated a Semina from the Parent, in the first formation, discovering itself at an

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[45]Classification which coincides with de Meara's in his *Pathologia Haereditaria*, 1619, who I believe could have been the source for Chambers' articles on the subject. See Chambers' entry under "Disease", vol. I.

[46]The communication of disease through the milk of the mother, or of a nurse, was called hereditary by many authors. Although at least since Lyonnet's piece (1643) that this use was criticized, it remained a much debated theme until the 19th century (see Lomax, 1977,79). Some authors considered this route to be akin to the connate influences. Some others put them on the same level as the acquired influences, just like any other nutritional action. Among the acquired influences, climatic ones were often highlighted, and even competed with the hereditary, in some works, as the explanation of familial pattern.

[47]This disease, popularly known as king's evil, was shown to be a tubercular infection of the lymphatic system only in the 1860's. It was a mythical disease at least since the middle ages, when it was thought the touch of the hand of some Kings could cure it.

[48]R. James, 1743-45, *A Medicinal Dictionary*, London, T. Osborne. 3 vols. This work was a very thorough compilation of many previous Foreign and British medical dictionaries. The translation of this dictionary into French provided Diderot with a very ample medical knowledge, and it is considered one of the chief influences on his *Encyclopedie*."Scrophula" is on volume 3.

age, when Glands are fitted for its reception, and disappearing when the digestive powers have arrived to their greatest strength.

The author is aware of the scepticism that this kind of assertions can generate, so he sets himself to acknowledge the difficulty of the hereditary claim, and tries to justify it for the case of scrofula:

> That such a Humor can be derived from the Parent, is granted perhaps in more instances than were it is really so, and is likely to be yielded by many more on the score of a vulgar opinion, than for any true Notions of the manner how such a thing is possible: It may be therefore necessary to form some rational Conceptions hereof in order to judge what Disorders spring from such an origin, and which not; because *without some rules to determine by, Cases may be confounded and mistaken from some Resemblance in their appearance, which flow from very different causes.* (my italics)

After this, the author proceeds to give a detailed physiological description of how certain constitutional diseases, like scrofula, might be transmitted from parents to offspring, and come to become part of the fabric (or constitution) of the body and manifest its evil effects at a certain point in the development of the organism. He writes also about how sometimes, when internal physiological conditions change, the hereditary cause can later reduce its malignity.

In the manner of several authors of the time, James (or his contributor) supposes that hereditary morbific causes could be salts that can both flow diluted in the humors (in blood for instance) or crystallize or incorporate into the solid parts of the body in some way. A revealing aspect of James' discussion of the hereditary is that he starts this part of his analysis by denying that recent microscopic observations, and their concomitant speculations on generation, have relevance to his argument:

> To this purpose then I cannot see what we have to do with the Philosophy of the Microscope, so far as it asserts the Semen to be animated before Generation, because it seems not in any manner to affect the Matter under Inquiry,[49] but so far as we got any knowledge of the sensible and manifest properties of the small Portion of Matter from whence we boast the Production of the finest Machines in the Creation, it appears to consist of a very subtle, active salt, floating in a soft, balsamic Vehicle, whereas, therefore, we conceive what consequences to the Oeconomy already formed, may flow from an Excess or Defect in the more active Principle of such Composition; so may we by Parity of Reason, conjecture, what must the result of every Deviation from the natural Standard in the same Principle before its Animation in the Matrix. When the Principle abounds...in the masculine Semen[50] it

[49]This statement shows very clearly the independence and the limitation of the humoral-solid physiology. It would partially separate its speculation from the actual discussions on generation, but at the same time any accuracy they could obtain in their classification of diseases according to causal routes lost materiality under the excessive reliance on many unobservable physiological occurrences.

[50]Most post-Renaissance physicians, influenced by Aristotle, saw the male contribution to the offspring's constitution as either determinant, or the strongest influence. they did not however deny the female contribution, following in this Hippocrates and Galen, and their two seeds.

> will...carry with it the same Qualities into the impregnated ovum; and without some uncommon Interruption, or Cotemperature from opposite qualities, will encrease in the growing foetus, Proportion to its enlargement, and make a part of that Constitution to which it gave being, with the same Affections and Properties as it stood possessed of in the generating Semen

As with some family resemblances, the hereditary (seminal) hypothesis of disease transmission has to account for the phenomenon of latency, that is the existence in a body of a given causal factor without its concomitant effect (the resemblance, the disease). James' humoral-solid account tackled the issue:

> ...it will be no difficult thing to imagine what a condition the Offspring of such a Parent must be in, and how sooner or later, in one or another part,[51] this primitive matter may shew itself in a very troublesome if not a very mischievous manner, as the circumstances of life and strength of the Constitution encourage or obstruct its Exertion, and the peculiar Configuration of the Glands favour or resist its accumulation and lodgement[52]

The particular case of Scrofula (which manifests itself, according to James, very soon after birth and lasts until after adolescence, when the "state of manhood" is reached), is explained by him in saying that before birth, and for the few following weeks, the morbific cause

> is not in quantity enough to be discernible or (is) hindered from Exertion by the laxity of the parts and viscidity of humors, which is always more or less the case of very young children; but when the parts have got some degree of firmness, and have digested away the tough humors, this hot, sharp matter becomes sensible to the fine strainers and membranes as it passes in course of circulation, and at last fixes upon them so as to occasion pain, inflammation, swelling and running sores

The aggressive salts then stay in place, swelling the glands and debilitating the constitution, until the latter "takes another turn and arrives at its utmost vigour" when the "digestive powers" manage to soften and detach the evil salts from the solids of the body and send them in solution through the circulatory system to the adequate excretory channels, among them —unfortunately— the seminal one completing the "cycle" of transmission when its own semen carries it to the following generation at conception.

James' description of the hereditary transmission of scrofula concludes by pointing out that this "mechanism" is "not only out of question from common

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[51]It was thought by some that the same taint (causal influence), if situated in different parts of the body, can give rise to different diseases. When this opinion is taken to the extreme all hereditary diseases are said to be the consequence only one taint that manifests itself with different symptom in the different organs.

[52]This could be taken as a relatively early statement that what really is inherited is not the disease itself but a predisposition to it. Additional, triggering (efficient) causes would be necessary, under the later more elaborated view, for the disease to be developed.

Experience,[53] but the general manner of it may in some measure be conceived from Hints and the nature of the generation matter".

The above description of the causal route of hereditary scrofula, James adds, can also help in the visualizing

> in what circumstances a Person may fall into this Distemper without having it to charge upon Parents, or the Milk of a tainted Nurse, which also may possibly happen tho' it is believed very rarely.[54]

Contagion, it is implied, can be produced by the tainted humors, excreted from the ill and recovering person, finding a route into a healthy and immature's person circulation. This person would thus "acquire" the disease and could itself transmit it to its offspring.[55] The moment and fashion of such acquisition, as we saw, determine the strength of hold that the morbific cause would have, and the curability. A hereditary contagion would be stronger from a connate one, and this one stronger than a acquired (post-natal) one. Some authors held that the longer a taint had been within a family (the more generations of it had plagued) the stronger hold it would have. Some others held that there was a natural weakening of the morbidity from generation to generation until the effect became negligible. Other authors held an intermediate view, using the analogy to a life cycle. They held that a disease in a family would first grow from generation to generation, mature and eventually begin to decline, until it died away. I mention this now as an example of the kind of speculative distinctions medical men preoccupied with transmission of diseases within families were trying to make, using the evidence available and shaping it up in different fashions to try to back their points.

The categories of disease some physicians saw as reflecting the true causal workings of hereditary transmission, and the physiological and genealogical evidence they drew upon for support were not, however, precise enough to convince the sceptics. With Generation Theories muddled up in the deep confusion of arguments between the dual seminal position of successionist (anti-preformationist) mechanicists, like Buffon or Maupertuis, and the reformed ovists like Haller and Bonnet, it is not surprising that not many people outside the restricted schools of the physicians appreciated their apparently backward-looking discussions on the hereditary.

Both James' *Medicinal Dictionary* and *Chambers' Dictionary* have been recognized as important influences in Diderot's conception of the *Encyclopédie.*[56] Due to Diderot's own personal interests, the *Encyclopédie* exhibited a very profound and overarching interest in all matters medical. The topic of hereditary diseases was one of them.

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[53]He is alluding here to the growing accumulation, in medical literature, of cases that give testimony of hereditary transmission of certain constitutional diseases, and to the everyday experience of seeing them coming down in families.

[54]"Scrofula", James' *Dictionary.*

[55]This view, of course, presuppose the "inheritance of acquired characters", but it is uninteresting since all 18th century views of the hereditary presuppose it. Which is what make so misguided any interest in finding "pioneers" of what we now call Lamarckism.

[56]See for this J. Mayer (1964) "Introduction" to Diderot's *Élemens de Physiologie*, and F.A. Kafker (1981) *Notable Encyclopedias...*

Given that France had been only a few years before the scene of what was perhaps the most important sceptic attack on the very idea of an hereditary disease, and adding to this that the author of the piece was the famous surgeon Antoine Louis,[57] a man who became a constant and important collaborator of the *Encyclopédie*, it is somehow surprising that the entry for the subject (in volume VII, 1760?) came out strongly in favour of the hereditary. Almost certainly penned by Diderot himself,[58] it drew its material and arguments from previous dictionaries and, more interestingly, from the rather obscure, pro-hereditarian and acute analyses of the subject published by Stahl, Zeller and, specially, De Meara.[59]

The adjective "héréditaire" was given only its medical definition in the Encyclopédie. The first thing the encyclopedist points out is the contingent character of the adscription, given that it depends more upon the route of contagion and not upon an essential quality of the causal influence. According to him, a disease is hereditary if its cause (vice) is contracted due to the quality of the seminal liquid and of maternal humors, that are joined to form the embryo and to give it the principle of life.[60]

The analogy chosen by the encyclopedist to illustrate the kind of causal pattern he has in mind qualifies however such contingency, making it seem quite important. He choses the physiological and anatomical (constitutional) changes that adolescence sparks in the male and female body as an adequate simile.

> Tous les hommes mâles ont acquis dans le corps de leur mere la disposition a ce que la barbe leur croisse à l'âge de la puberté, & les femelles a ce qu'elles deviennent sujets au flux menstruel: cette disposition peut donc être regardée comme héréditaire, en tant qu'elle est transmisse des peres & meres aux enfans; il en est de même de certaines familles éprouvent tous qu'ils deviennent sujets à certain âge; telle sont par exemple, l'epilepsie, la goutte...

By choosing this simile, I believe, Diderot is asserting his belief in the transmissibility of latent constitutional causal influences of some kind: that is, of material elements that can transform the bodily organization at a given period of the life span of an individual. The mystery and the solution to both the dramatic changes at puberty and the appearance of the same disease at the same time in parent and offspring are, in his mind, strongly bound together. Furthermore, he writes, the feasibility of destroying the disposition to develop a disease that has already been inherited is as meagre as that of destroying the disposition "qui fait croître la barbe à un jeune homme qui est en bonne santé".

The strong hold that hereditary elements have on an individual's constitution derives from the fact that they are there since the first instant of formation of the new being (the stamen, or rudiments), and the contingency of the seminal route for a morbific element does not diminish such strength. The Encyclopedist thus highlights the importance of making a distinction between the disposition to disease that are

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[57]A. Louis (1749). See appendix 1.

[58]For a discussion on Diderot's medical knowledge, see J. Mayer, op.cit. For the medical contributors to the *Encyclopédie*, see F. A. Kafker and S. L. Kafker (1988); and H. Zeiler (1934) *Les Collaborateurs Médicaux de l'Encyclopédie.*

[59]See bibliographical details in appendix 1.

[60]See "Héréditaire" in tome VII, p.156, of Diderot and D'Alambert's *Encyclopédie, Dictionnaire Raissonée des Sciences, des Arts, et des Matiéres.*

acquired at conception (truly hereditary) and after it.

> On doit distinguer les maladies héréditaires de celles que les Pathologistes appellent connées, morbi connati, c'est-a-dire que le foetus a contractées accidentellement dans le ventre de sa mere, que l'ont apporte en naissant, conséquent sans qu'elles soient l'effet d'un vice de la santé des parens, antérieur à la conception, transmis aux enfans, comme dans le cas des maladies héréditaires.[61]

After the *Encyclopédie*, the adjective "héréditaire" in its technical sense became a recurrent entry in French general and medical dictionaries. Simultaneously, in many of the discussions around generation theories the awareness of the challenge posed by the mixed hereditary influence made sure that the hereditary phenomena (resemblance, hybridization and hereditary disease) were thoroughly discussed. As R. Rey has recently pointed out, in most dictionaries of the 18th century, it was under the entry of "generation" that the facts of heredity were discussed.[62] The difference in the interest between generation theorists and those physicians who were worried about the hereditary character of certain disease made their approaches to the subject quite different.

The system makers (Haller, Bonnet, Needham, Buffon) were after the whole organism, that is, after an account of how it came to be, either produced anew or unfolded from the egg. They tended then to privilege their theoretical schemes and to consider the problem of transmission of characteristics between generations as relatively secondary, helpful or unhelpful for their views, but not determinant.

Physicians, less ambitious, focused particularly on morbific causes and their possible routes. Transmission of disease from person to person was their issue, and the existence, or not, of an hereditary route was at the centre of their discussion. The fact that they could see analogies between the behaviour of normal, apparently inherited, characters and that of their candidate hereditary diseases reinforced their belief in such a route. Part of their effort was then concentrated on gathering the factual evidence for transmission, and another part on producing more or less convincing causal (physiological) accounts for the transmission.

The latter part of their works was the least convincing. It was often in conflict with knowledge and ideas in other fields: chemistry and physiology, besides generation disputes. But there was conflict also within the French medical community. For instance, some favoured a distinction between normal and pathological transmission, while others believed in the existence of a unique sort of hereditary transmission that included both kinds.[63]

In the 18th century, then, the theme of hereditary transmission of disease was perhaps more alive in France than anywhere else. An important stimulus to their thought was, I believe, Antoine Louis', already mentioned, intelligent and highly sceptical small essay. The challenge made them focus their attention on the weak points of their views, and the quest to gather and organize the evidence, from literature and their own practice. No convincing transmission mechanism was

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[61]"Héréditaire", *L'Encyclopédie*, vol.VII, p.156-57

[62]"destinées —she writes— à confirmer ou à invalider les grandes thèses en présence sur les mecanismes de la reproduction" . R. Rey (1989), p. 16.

[63]The dispute between humoralists and solidists had this as its theme, see following chapter.

however described. This made the Royal Academy of Medicine, late in the century (1788), call for competition essays amongst physicians on the subject of how are hereditary diseases transmitted. As we shall see in the following chapter, this competition revitalized the arguments around the subject and was crucial for it "being in the air" in the heated post-Revolutionary times, when it was taken up by higher order, socio-political forces.

The social and political importance of several constitutional diseases that had for a long time been classified as hereditary (v.gr. scrofula, syphilis, madness) seems to have grown with the urban concentrations and the industrial revolution. On the other hand, such "fatalistic" explanation of them has always tended to acquire some importance in non-medical thought. Sempiternal ideological views about the purity of blood, of breed, etcetera, within races, or nations, or within regional or familial groups, were always easily nourished by any kind of consideration of transmissibility of ills through family lines.

For much of the 18th century scepticism about hereditary claims, and a strong commitment to climatic and other external forces as a source of differences between humans, maintained the hereditary in the background, with regard to explaining human physical and cultural diversity. The situation changed in post-Revolutionary France. Several authors have tried to explain this apparently paradoxical shift.[64] The case is that the hereditary, as an explanation for a varied number of phenomena, began to receive more attention, especially as a way of accounting for many social ills: madness, syphilis, scrofula, tuberculosis.[65] The previous efforts of 18th century physicians in clarifying the causal structure of hereditary transmission was put into use by the brash and enterprising early 19th century generation of French physicians.

This phenomenon was also registered in the dictionaries (they are a very reliable source of traces of conceptual shifts, I believe). Post-Revolutionary France saw a flourish of Encyclopedist activity. Like other bodies of savants the physicians busied themselves producing compilations of their knowledge, in all of which "héréditaire" first, and "hérédité" after 1830, appeared heads of entries. In 1798, as part of the volume VII of the *Dictionnaire de Médecine*, that was part itself of the enormous project of the *Encyclopedie Méthodique*,[66] "héréditaire (maladie)" received an unusually long and detailed treatment. Written by Jean-François Pagès, this deep and meticulous essay had been, in a first version, a prize-winning final dissertation at Montpellier in 1788.[67] A revised version of that essay received in 1790 an honorary mention in the second round of the Royal Society of Medicine's competition I have mentioned above.[68]

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[64]See I. Dowbiggin (1991), D. Pick (1989). and R. Nye (1984), and C.E. Rosenberg (1976).

[65]See accounts of this by Lomax (1977,79) and Ackerknecht (1965,67).

[66]After the *Encyclopédie* this was the most ambitious French compilation of human knowledge. Begun before the Revolution, it was completed over several decades and with very irregular publishing schedules. It's medical part was edited in its first seven volumes (until its first interruption in 1798) by the famous secretary of the Royal Society of Medicine, Vicq D'Azyr.

[67]See bibliography in appendix 1 for details on Pagès and his work on hereditary disease.

[68]The prize was not given in the first round (1788) and only with difficulties in the second round it was awarded to a French expatriate in Bonn, doctor Joseph-Claude

Oddly for such a young author, Pagès' essay shows a more profound analysis of the issue than anything that had been published before. Only when it is compared with other essays, published or not, of the same competition, can one see that he was not alone, in the 1780's, in making the subtle and imaginative distinctions that are found in his text, the most important of which must be the clearly argued distinction between inheriting the disease itself and inheriting only a disposition (a propensity) to it.[69] Another is the further clarification of what it is to be connate (maladies connés) in contradistinction to what hereditary means. A further crucial position that Pagès takes is related to what has been called the transmission problem, in the context of the debate between humoral and solid causes in medicine. Pagès is adamant that hereditary transmission never should refer to humoral causes, but only to solid related, constitutional ones. Favouring the latter as the only truly hereditary, he reduces ad absurdum the former ones by demanding that only those diseases that occur in the very same organs and at the same period of life in parents and offspring are strictly to be considered hereditary. This would require, he argues, the existence of a different humoral vice (or taint) for each individual disease, which would make for an absurd proliferation of "vices" or humoral causes. These would on the other hand have to be specific for each organ or part of the body, which is implausible. How, he then adds, can a "virus" be lodged in a body without producing its evil effects. Only the existence in the body, since conception, of a constitutional (solid) defect, that is later made evident at a certain point in life by the triggering of a new stage, can explain this. In other words, only a disposition is what this inherited defect gives; not the disease itself.[70]

The latency of hereditary phenomena in general, and atavism in particular, were also claimed by Pagès as more easily explained by his solid-constitutional view of the hereditary. Given that it is dispositions, and not the diseases themselves (or a malefic humoral cause) that are communicated in the constitution, the fact that in a given generation the effect (the disease) is not produced can be understood as an absence of the triggering "causes occasionelles". This however does not preclude the possibility that a healthy person, who carries the disposition in his internal organization, can in turn transmit it to his descendants, any one of which can develop the disease if the triggering occasional causes concur.

As I said, the clarifications, and restrictions that Pagès makes to the concepts of a constitutional disease and its possible hereditary character seem, on their own, to add

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Rougemont. This was in 1790, a few months before the Society was dissolved by the Revolutionary Council. Pagès and Rougemont's essays both are missing from the archives, now kept in the National Academy of Medicine , Paris. Fortunately, both were published, Pagès' in the Vicq D'Azyr's Dictionnaire, and Rougemont's in a German translation, in Franckfurt, in 1794. See appendix 1 for bibliography.

[69]A distinction which, as I said, had been obscurely implied by previous authors. In Britain, it appears, it had been made also, at more or less the same time, by John Hunter. But the British surgeon never clarified it in writing and we have to take the word of his disciple John Adams for it. Adams used the idea to develop his views in his well known book on Hereditary Diseases of 1814. I copy from Pagès essay: "On apelle maladie héréditaire une maladie qui reconnoît pour cause une disposition particulière du corps à en être attaqué; dispositions que les parens qui ont été sujets à cette maladie, transmettent a leurs enfans par le moyens de la génération."

[70]Pagés, *Dict. de Médicine*, 1798, pp. 162-163.

up to a surprising individual effort, but in fact they acquire their proper dimensions when set in the context of the many other essays sent to the same competition,[71] whose manuscripts are kept in the Library of the National Academy of Medicine. That the advancing of those kind of distinctions was specifically what the setters of the Essay question were looking for can be seen in their reports and evaluations of the results, both in manuscripts and in the Memoires de la Societé Royale.[72] The other two prize essays made similar attempts, especially Alexis Pujol, physician from Castres, whose very long dissertation, when it was published some years later (1802) became one of the most powerful statements of French medical hereditarianism in the early 19th century, although he never gave up humoral causation as an outstanding part of the hereditary influences.[73]

Other French medical dictionaries of the early 19th century gave a preponderant position to essays under the adjective "héréditaire". Antoine Petit's 40 page *Essai sur les Maladies Héréditaires* (1817) was incorporated with slight changes into the voluminous *Dictionnaire des Sciences Médicales*.[74] Petit's work followed closely Pagès in his desire to define as clearly as possible the hereditary in terms of the causal transmission route, the moment at which the disposition to disease becomes established in the bodily constitution, and the need for additional causes to trigger it.[75]

In the shorter *Dictionnaire Abrégé des Sciences Médicales* (1823) space was found for five packed pages of analysis of "hereditary diseases" written by an anonymous author who reacted against the strict solidism of previous dictionary entries. [76] The increasing Dictionary presence of the hereditary in dictionaries was, of course, a symptom of a broader phenomenon that was happening in the French medical community. For instance, a growing number of medical student's theses and similar dissertations, both in Paris and Montpellier, were being focused on the general question of hereditary diseases, or on hereditary explanations of the sources of particular diseases, such as insanity or scrofula. During the 1820's such theses and dissertations would typically refer in their title to "maladies héréditaires". But around 1834 they switched to the formula l'Hérédité dans les maladies.[77] It was at this point that, I believe, heredity had completed, within the French medical community, its transition from a metaphor to thing, from an analogy to an independent and self-sufficient cause.

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[71]Only two more of them were published to my knowledge. The other honorary mention by Alexis Pujol, and the price winning essay by Rougemont.

[72]*Histoire et Memoires de la Societé Royale de Médecine* 1786, 1787, 1788 vol.IX p.17, 18 and X, XI and "minutes d'examen de memoires". Bibl. de L'Academie National de Médecine. Archive de L'ancienne Societé Royale de Medécine 181-23-1,5. See appendix 1 for details.

[73]"Essai sur les Maladies Héréditaires" in vol. 2 of Alexis Pujol (1823) *Oeuvres de Médecine Practique*. Only Pagès' piece Antoine Portal's (1814) book on Maladies Héréditaires, seems to have been more influential.

[74]A.Petit (1817) *Essai sur les Maladies Héréditaires, Paris, Chez Gabon.*

[75]See *Dictionnaire des Sciences Médicales* (1817) vol. 19 pp.58-86. This Dictionary had 52 volumes.

[76]"Hereditaire" (1823) Dictionnaire Abregé des Sciences Médicales, vol.9, pp 45-49.

[77]The first work that shows this change was the excellent dissertation by D.A Lereboullet (1834), a disciple of Fodéré at Strasbourg. See appendix 1 for evidence of the shift.

This was also registered by a switch in French Dictionary and Encyclopedia entries, from the adjective "héréditaire" to the emphatic, strong sounding noun "Hérédité".[78] A clear evidence of this is found in the French translation of J. Forbes (1833) *Cyclopaedia of Practical Medicine*. The entry this influential dictionary dedicates to the theme was written by Joseph Brown, who followed closely the lead that in Britain had been given by the writings of Joseph Adams and J.C. Prichard. Significantly, the cumbersome phrase entry "Hereditary Transmission of Disease" is straightforwardly delivered by the French translator as "Hérédité".[79]

Hérédité, as used by medics (that is, as a synonymous with "hereditary transmission of disease) began to overflow the limits of their speciality. Its pathological connotations became increasingly common in wider sectors of society, and began to "interact" so to speak with the worries and ambitions of social thinkers and reformers.

At the same time, physicians began to recognize the unavoidable relevance of knowledge and observation of hereditary transmission among animals (both of normal and pathological characters) for the validity of their claims for pathological heredity in humans. By the 1840s it became obvious that what was needed to be done was to unify the physiological and pathological knowledge into a joint account of "positive", i.e. normal transmission of characters. The privileged status that medics were giving to hérédité as an explanatory tool, they realized, had to be backed by a fairly well organized collection of "normal" physiological, zoological, and botanical and embryological facts, and better theories than were available. The unification of pathological and normal hereditary transmission under a simple scheme had been by the already advanced considerably by J.C. Prichard, in Britain (see chapter 3).

On the other hand, a question raised by the Montpellier physician Lordat, in a 1842 essay "Les Lois d'hérédité physiologique, sont elles les memes chez les bêtes et chez l'homme? "[80] was a question in many of his colleagues minds, and that had begun to permeate the work of scientists and social reformers.

At that stage, the perception of the existence of such a thing as the Laws of Heredity was not one shared outside France, with the exception of a few British authors. In quotations and translations of books and articles the French Hérédité was transformed into English adjectival forms, like Hereditary Disposition or Hereditary Transmission.[81] At the same time, for instance, in Todd's *Encyclopedia of Anatomy and Physiology*, 1839 a satisfactory rendering of recent French, German and British work on the subject by Dr. Allen Thomson was hidden, in the 18th century fashion, in an article on "generation", and under the heading "Influence exerted by parents on the qualities of their offspring in generations", and referred to in the general index as

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[78]For French physicians of the first post-revolutionary generation, such as Lereboullet, Piorry, the reality of hereditary transmission was not even an issue, it was a given. Among alienists of Esquirol's school it was also common. Naturalists and physiologists also joined in the usage of hérédité. The work of Girou de Buzareingues and the translation of Burdach's Physiological works into French also gave hereditary explanations a boost. See chapter 5.

[79]"Hérédité" in *Encyclopedie Médicale Anglaise 1836.*

[80]Lordat, 1842, Montpellier.

[81]J. Esquirol's proposed cause for Manie (Hérédité) was translated into English as "Hereditary Disposition" somehow diminishing the strength of the French author's statement.

"hereditary qualities; mental and physical phenomena of (their) transmission from parents to offspring.[82] The measure in which early 19th century "hereditarianism" was overwhelmingly a French physician's "craze" can be seen in the imbalance that exhaustive bibliographical research produces. The evidence for this can be seen in appendix 1.

Before 1870 very few medical or other British treatises had "heredity" in their titles. There was no equivalent proliferation of medical theses focused on the hereditary transmission of disease (or any other physical or mental character) in British medical institutions. After the "pioneering" work of Joseph Adams and J.C. Prichard and William Lawrence on the matter, only a few medics followed, and those mainly interested in the already sidelined phrenological studies. What was more relevant in those years in Britain was a small but significant current of scepticism around hereditary explanations of constitutional diseases, stemming basically from the ideas of the 18th century physician Wiliam Cadogan, and represented by authors like Henning and Phillips (see chapter 4).

A German immigrant, Julius Henry Steinau published in 1843 one of the few known treatises in Britain on hereditary transmission of disease. A work that originally had been written and published in Germany and it seems to have had no local repercussions.[83] Sir Henry Holland was, to my knowledge, about the only physician of prestige to have dedicated attention to the subject and in so doing directed Darwin's attention to it.[84]

It was not until Spencer, Lewes, Darwin and Galton emphasized the centrality of the hereditary, the second half of the century, that hereditarianism finally made the leap to Britain. It was not pathological hereditarianism which took hold here, but a more general, theoretical approach. But it had nevertheless its structural origin in the efforts of classification and analysis that French physicians had made concerning the problem of hereditary transmission.

All the general phenomena associated with the hereditary that physicians had recognized and tried to account for in their treatments of the pathological were later seen as important also for an understanding of the normal. By highlighting of the irregular behaviour of character transmission (similarity vs. dissimilarity), the latency of causes, atavistic regression, homochrony, etcetera, physicians provided a scheme upon which other naturalists could incorporate their questions and evidence. Hybridization studies by botanists and breeders; the advancing edges of embryology, cytology, physiology, etc.; and the appearance of the Darwinian argument, all made French physicians causal views of heredity seem very soon outmoded and untenable, but it seems undeniable to me that the very idea of the existence of such a thing as heredity, and its general laws we owe to them.

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[82]Allen Thomson, 1839. "Generation" a Todd's *Encyclopedia of Anatomy and Physiology*, vol. 2, pp. 470-480.

[83]Julius Henry Steinau (1843) *Pathological and Philosophical Essay on Hereditary Diseases*, London, Marshall & Co.

[84]Sir James Paget was an exception, and wrote a piece on hereditary cancer (1857, see appendix 1). Darwin owned two editions 1839 and 1855 of Holland's *Medical Notes and Reflections*, and in both it is only the Chapters "On Hereditary Disease' which are annotated. He seems to have been particularly interested in evidence of homochrony and atavism.

Chapter 2

Les Maladies Héréditaires; 18th Century Disputes in France

> This one is mad, we say; his father was too and his children will also be mad, it runs in their family, its an hereditary disease! Are diseases then inherited like property? Yes, no doubt about it; a father will leave behind to their children the whole lot: his land, his post, his house, his money and the gout (...), it is a patrimony impossible to dilapidate; it has to be passed in direct line up to the very last generation, and all the progeny of this vitiated source will receive, with the principle of life, that of the gout.[1]

These rhetorics —more akin to the darker sides of 19th century hereditarianism than to the Lumières— were employed in 1750 by J. de la Porte, a French literary critic and editor, to describe the frame of mind of those who took hereditary transmission of certain diseases as self evident. They, he reckoned, constituted a majority among lay persons, and nearly the totality among medical men.

In the article in question[2] de la Porte, as editor of *Observations sur la Litterature Moderne*, had given a recent small booklet, by a novice author, on the rather specialized and relatively marginal subject of hereditary disease, the rare privilege of being reviewed, and commended, alongside recent publications from mainstream French literary and intellectual authors such as Voltaire, Maupertuis or Montesquieu. Apart from pointing out the unusual clarity of ideas and elegance of exposition of the young author, Antoine Louis (1723-1792),[3] the reviewer justifies this inclusion by

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[1]The French original: "Un tel est fou, dit-on quelque fois; son pere l'étoit, ses enfans le seront, cela vient de famille, c'est une maladie héréditaire! On hérite donc des maladies comme des biens? Oui sans doute; & un pere laissera à ses enfans tout a la fois sa terre, sa charge, sa maison, son argent & la goute (...) c'est un fond qu'il ne leur est pas possible d'aliéner; il doit passer en ligne directe jusqu'à la derniere génération, et tous les rejettons que produira cette souche vitiée, recevront d'elle, avec principe de la vie, celui de la goute."

[2]J. de la Porte, "Dissertation sur les Maladies Héréditaires par M.Louis", (1750), *Observations sur la Litterature Moderne.* See appendix 1.

[3]Antoine Louis, (1723-1792), was soon to become an important contributor, mainly in surgical matters, to Diderot's *Encyclopédie.* Later in life he became permanent (and polemical) secretary of the Royal Academy of Surgery (1764-92), and among many other things was the co-designer of the infamous "guillotine", which for some time was known as the "petite louison". Biobibliographical information on him is summarized in "Louis Antoine", in F.and S.Kafker (1988) *The Encyclopedists as individuals.* See also "Eloge de Antoine Louis" by P. Sue (1793), in Antoine Louis

emphasizing the importance of the subject for wider spheres than solely the medical one, given that knowledge, or ignorance, on it could affect not only individual families, but whole nations and for great spans of time.

But perhaps the most attractive feature of Louis' small dissertation was that it argued, against the tide of opinion questioning the very existence of hereditary diseases, and boldly calling into question centuries of assumptions and presuppositions, and of accumulated statements of fact, very dear to the medical profession. As Louis in fact challenged the reality of the transmission of any individual (non-essential) characteristic from parents to children, his scepticism extended to the whole domain of what we have been calling the hereditary, i.e. family resemblance, hybridization, and transmission of deformities (or monstrosities) within families.

All this went against what most of the 18th century medics considered to be very well established facts, strongly backed by most authorities within the dominant Hippocratic-Galenic tradition in medicine, and by their own day to day practice. It can also be said that, as part of their overall theoretical approach to physiology and disease, medics had very high stakes invested in the reality of such phenomena, and they did not take lightly any such challenge.

Alexis Pujol (1739-1804), a physician at Castres, wrote some time later that Louis' essay had been more popular among amateurs than among professional medics. The latter had found his points witty but unconvincing, and —as he puts it— had ignored him and carried on with the business of unriddling the very complex affair of hereditary influence in disease, convinced (as they should be given the immense power of the accumulated evidence) of the reality of the phenomenon.[4]

The truth, if we are to believe the statements of several other late 18th century French physicians,[5] is that the inclusion of Louis' dissertation in a widely read and discussed journal did put some pressure on the French medical community to produce a clearer account of hereditary transmission. Pujol himself ends up recognizing such pressure when he affirms that when, in 1787, the French Royal Society of Medecine decided to open up an essay competition on the theme of hereditary diseases (which had prompted the production of his own essay), it did so because it regarded as "douteuse et problematique l'existence de ces maladies", as can be inferred by the wording of the essay questions.[6] As he writes,

> Il est donc clair que la Société n'avait en vue que de réunir de grandes preuves contre les assertions hassardées autrefois par M.Louis.[7]

Louis' essay itself had been written in response to an essay competition forty

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(1859) *Eloges...1750-1792*; and H. Zeiler (1934) *Les collaborateurs médicaux de l'Encyclopédie*, Paris, L. Rodateri.

[4]A. Pujol, "Essai sur les Maladies Héréditaires", see section 2.4 below and appendix 1.

[5]Prominent among them, the historian of medicine and physician at Montpellier, Pierre-Joseph Amoreux, who in an unpublished essay (see appendix 1) recognizes —with the author of the review— the persuasiveness of Louis' arguments. This effect of Louis arguments lasted well into the 19th century when different readers, like Prosper Lucas or Charles Elam believed it necessary to address them.

[6]See D. below, and appendix 1.

[7]A.Pujol, (1823), p.214

years earlier. To the details of the first essay competition, called by the Dijon Academy, and the argument of Louis' sceptical essay, I now turn.

2.1 Antoine Louis' sceptical challenge

In 1748, only a couple of years before Rousseau's first polemical participation in an Academy of Dijon competition,[8] another much less known dispute had its origin in a similar event. In one of the first essay competitions with a medical theme in the history of the Academy,[9] an essay question was chosen that, though being a particular (and traditional) medical issue, was at the crux of several debates, then in progress, in physiology, and which was seen by some as having important consequences for the understanding and treatment of some of the most dramatic diseases of the time. The question was "comment se fait la transmission des maladies héréditaires? " and the polemical contribution, written by Antoine Louis, was, in contrast with Rousseau's case, not even mentioned by the judges in their final assessment, perhaps because his essay did not try to answer the question.[10] Instead, in what Pujol later called "la dernière insurrection qui s'est faite contre la transmission héréditaire des maladies"[11] Louis set himself up to challenge the basic assumption on which the essay question depended, i.e. the reality of such transmission.

To justify his attitude, Louis gave the example of Bernouilli's prize winning essay of 1724 for Academy of Sciences of Paris, in which instead of answering the question set by the academicians, he showed that the phenomena they wanted explained (the transmission of movement between rigid bodies) never take place because such bodies' existence is impossible. Louis offered to do the same for his subject, though he acknowledged that a question of medicine, related to practice and empirical evidence, is not exactly in the same case as the physical problem he quotes. He believes, however, that to inspire legitimate doubts over the question proposed is as valid as trying to answer it.

The judges of the competition, to whom Louis addressed all the clever, sceptical doubts of his essay —in a rhetorical, as well as rational attempt to switch their opinions— obviously were not convinced, and gave the prize to Chambon, a physician lecturer at Montpellier, and two special mentions to provincial medical men: Guillaume Rey of Chaumont (in Lyonnois) and Gravier of Parray (in

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[8]See Tisserand (1950) *Les concurrents de J. J. Rousseau a l'Academie de Dijon.* Also J. Roger (1971)"Introduction" to Rousseau's *Discours sur l'origine et les fondements de l'inégalité parmi les hommes*, Flammarion.

[9]The first essay prize ever of the Dijon Academy was set in August 1741, and the theme was a problem in Physics, after which the prize was alternated yearly between Moral, Medical and Physical questions. See for details on this "Histoire de l'Academie de Dijon" in Mémoires de l'Academie de Dijon, Tome I, 1759. See also R. Ruffey (1909) *Histoire Secrete de l'Academie de Dijon*, Paris.

[10]Louis was then a relatively unknown, very ambitious, 25-year-old, military surgeon. Pujol wrote about his motivations: "fort jeune encore (il) avait besoin de se faire un nom par quelque crit éclatant, s'amusa a fronder le programme de Dijon, par une Dissertation très-ingeneuse, qui fut de bruit, et tira tout-à-coup son son auteur de la foule des crivains". op.cit. pp.212-213.

[11]A.Pujol,op.cit. p.212.

Charolois). All three of them participated with "positive" contributions.[12]

The young Louis strongly felt that justice had not been done, and decided to publish his essay the following year, with a challenge to the winners to publish theirs, and to the judges to confront what he felt to be his insurmountable argument against the very existence of hereditary disease.[13]

Do the "maladies héréditaires" really exist? Louis asks. And immediately questions the automatic "yes" that all physicians of his time tended to give. He writes:

> Il ne presque parlé des maladies héréditaires que par simple dénomination, dans la division générale des causes des maladies: peu d'Auteurs insistent sur la cause héréditaire dans les details pathologiques. Cette cause seroit-elle un être de raison, un vice imaginaire dont on ne parle que par habitude & sans conoissances positives?

All authors, including the other competitors for the Dijon prize, Louis blatantly affirms, go straight on to answer how disease is communicated from parents to offspring, without second thoughts about the reality of the phenomenon. The discord between them is about particular routes and media of transmission, and is based on their allegiance to this or that school or practical tradition in medicine. It seems indeed strange to him that the existence of hereditary disease, in itself, has been

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[12]Probably one of the principal judges of the competition was Lecat, a surgeon with whom Louis had had a heated priority dispute only a couple of years earlier. I don't know if this could have affected the decision, because the work must have been submitted anonymously. Lecat is also known to have opposed the granting of the prize on moral to Rousseau in 1750. See "Histoire de l'Académie de Dijon", 1759. There is a possibility however that the prize was not judged fairly at all. Ruffey, in his *Histoire Secrete de l'Academie Dijon* (which remained unpublished until 1909) made very severe accusations concerning the adjudication of the Dijon prizes during the period in question (1741 and onwards), especially those of Medicine. I copy from his text:"Dans l'une des sçéances publiques on distribuait chaque année un prix de la valeur de trois cents livres...pendant quinze années [1841-1856] presque tous les prix, surtout ceux de médecine et de physique, furent donnés par faveur et par intrigue à des gens qui prêtaient leur à un médecin de l'Academie auquel ils abandonnaint la valeur du prix...". Ruffey, himself a member of the Academy in those years, identifies the perpetrator of the mischief as M. Fournier de Languedoc, who apparently was caught *in fraganti* in one of schemes. Unfortunately Ruffey omits the details of the prizes that were bent, and I ignore if the documents still exist in Dijon.

[13]Antoine Louis (1749), Dissertation sur la question ...comment se fait la transmission des Maladies héréditaires? (see appendix 1). About Louis' own attitude towards the truth of his pronouncements there is some discussion. In his Introduction to the published essay, by the wording and the tone of it, he seems very strongly commited to it. Most medics however believed he had been cynical in his will to ignore the most obvious facts of the hereditary. Amoreux thought that Louis had softened his position later in life, and de la Porte, who seems to have had personal links with him at the time, wrote: "M.Louis ne regarde pas ce raissonemment comme invincible, & l'on voit bien ce n'est que l'envie d'avoir de plus eclaircissemens sur cette question importante de la Médecine." Among the winners of the contest, only Rey published his competition piece (1749), and the "silence" of the winner probably confirms Ruffey's accusations (see footnote 12).

admitted by all schools and at all times.[14]

This attitude of physicians of trying to explain something that nobody has bothered to prove the existence of, is compared by Louis to the discussions of those savants of the old days who tried to explain why underground sites were hot in the Winter and cold in the Summer without never taking the trouble to verify such a statement.[15] I have tried to discover, he writes, the motives that have persuaded so many generations of physicians and authors of the existence of such a kind of "transmission morbifique".

> Je n'ai apperçu sur ce point que des allégations vagues, qu'une tradition reçue aveuglement & transmise de siècle en siècle, sous l'autorité des quelques faits particuliers, dont les différentes circonstances paroissent n'avoir point été assez exactement observées.[16]

Authors, Louis argues, blind themselves when they can hold in their imagination an idea that seems to link everything together by supposed causes, and cease to see all the inconsistencies that surround the matter. He believes that to hold theories so dearly as most physicians do is quite a dangerous attitude, one that usually impedes the acquisition of real, empirical, detailed knowledge. The error is to believe that one can avoid burdening the memory by having general recipes for all circumstances; that one needs only to "have the thread" in order to master "all the ideas".[17]

To answer the basic question of the existence of hereditary diseases, Louis wants first to establish what are we to understand by the adjective hereditary. He refuses to use the adjective only on the basis of a familial pattern of occurrence.[18] An example of this common mistake, he believes, is saying that venereal diseases (the communication of a "virus véneriene") are hereditary, just because the baby is infected at birth, and the mother is infected too. Given the mutual communication of "liqueurs" between mother and embryo, the vices of the mother's humors will necessarily influence the child's health. Furthermore, this kind of communication of disease is no different to the one effected through the milk during lactation, either by the mother or by a nurse, neither of which should be called hereditary.[19] These must

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[14]A.Louis, op.cit., pp.7-8.

[15]He quotes for this Fontenelle's *Histoire des Oracles*.

[16]A.Louis, op.cit., pp.9-10.

[17]A point must be made here concerning the possible motives of someone in the position of Louis. As a surgeon, he had an interest, at the period, in making his profession as reputable as that of the other medical men, which it was not, yet. During his lifetime he struggled and achieved, together with other surgeons, a comparable status for his branch of the profession, but struggles and divisions always existed between surgery and general medicine. Many of his writings were aimed at cutting down the theoretical general claims of other physicians and at forwarding the more empirical, detailed approach of surgery. To accuse thus most physicians of living in a confused world of theory laden, facile and subjective explanations, was harsh but in tune with this general aim.

[18]"Je ne donnairai point —he writes—, avec quelques Auteurs, ce nom à certaines Maladies que les enfans apportent en naisant & dont les parens son actuellement attaqués" op.cit. p.12

[19]A. Louis, op.cit., p.13-14. John Hunter, famously proved that contagion during birth was the reason of infantile syphilis. He also differentiated this communication of disease from the hereditary one. See chapter 3.

certainly be considered, he writes, among the contagious diseases, whose routes of contagion are not exclusively familial ones. Surely, Louis concludes, if we are to use a special category for hereditary transmission it must be backed by the existence of an autonomous and independent route of communication, which is carried on to the future embryo by the seed or germ itself through which life is communicated.[20] The burden of proof is then on those who believe such a thing.

Louis then asks himself if what some physicians call the succession of disease in families (as different from the acquisition of them) does really have an independent, particular route. To answer this he first explains that the most careful authors do not think that it is the disease itself which is transmitted to children but a disposition to it, and such disposition is, in any case, the proper target of the adjective "héréditaire"

> ensorte que les parens peuvent l'avoir reçue de leurs ayeux & les transmettre à leur posterité sans avoir eux-mêmes jamais été attaqués de la maladie que cette disposition pouvoit produire; parce que leur tempérament particulier, & les différens usages qu'ils ont faits de choses non naturelles on peu changer cette mauvaise disposition.[21]

With this move Louis gives centre stage to what had been, up to then, considered by several authors the main mystery of general (not only pathological) hereditary transmission of physical and moral characteristics (the sum of which was the constitution, or temperament of the individual), and at the same time the most important criteria for the recognition of the phenomena: atavistic regressions, or generation jumps.

The existence of hidden (latent) morbific causes, the sort that could account for the atavistic pattern, was then put under scrutiny by Louis. Under such a view of the hereditary, he writes,

> le vice héréditaire, s'il y en a, doit se trouver dans le germe antérieurement à sa fécondation; &...differents causes extérieures dont les modifications peuvent être infiniment variées, pourroient substituer la succession, & ne la transmettre, par ex., qu'à la centiéme génération.[22]

Louis, in some way, tries to turn the argument around. If such hidden causes will

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[20]Mistrust about any solely humoral physiological explanation of constitutional disease was on the rise in Louis' days, and being a surgeon made him sympathetic to the solidist account. Preexistence, the view of generation which had dominated the first half of his century also was biased toward solidism, and was interested in diminishing the relevance of hereditary phenomena, as the cases of both Louis and Haller show. See below, footnote 50.

[21] A.Louis, op.cit., p.18."Les choses non naturelles sont six —writes Louis—: l'Air, les Alimens, le Travail & le Repos, Le Somneil & la Veille, les Excrétions retenues ou évacuées, & les Passions de l'Ame"(footnote, page 18). Louis is of course following here the old Hippocratic distinction between natural and non natural influences on health; but in this context, seeing the "non natural" (external) ones considered as triggering of, or complementary to, hereditary dispositions, one is tempted to relate the distinction with the much later one between nature and nurture; temptation which perhaps should be resisted.

[22]In referring to the pre-conception state of the embryo as "germ", Louis is assuming a preformationist view of generation. He later pretends, however, that his argument worked both under the premises of both preexistence and succession.

only take effect with the concurrence of external causes, the disposition could properly be known to have been in place only when the disease develops, so one can never be sure that it has a hereditary origin. It cannot be said either that a general tendency is or was shared by a whole family lineage, unless it can be proved that similar external conditions cannot have been the cause of similar patterns of ill health, which it seems to him impossible to do. Given that external factors obviously exist, are extremely numerous and complex, and so obviously play a much stronger causal influence than any supposed internal disposition, Louis argues, there seems to be no reason to make use of the latter anyway. Hidden causes seem to him a dubious approach in principle. A further reason for scepticism Louis finds in the actual embodiment that any hidden, constitutional influence (cause) could take in order to exist in the germ before impregnation. A perfect knowledge on this question would require a much better grasp of what occurs during conception, and what the word generation really means. In his lifetime, and for some time after, Louis adds, those things are bound to remain a "mystère impenetrable".

Louis however does incorporate some general considerations about generation to back his sceptical approach to the hereditary. Basically, what he tries to argue is that no particular (individual) characteristic can be communicated by parents to the first rudiment of the embryo, and that all the so called hereditary phenomena are caused by external actions; the idea of a transmission or communication of the disposition to a given disease (or any other accident or particularity) being simply an illusion originated by the fixation of minds in the deceiving familial pattern of resemblances and reccurrences.

In order to develop this part of his argument, Louis considers the two basic alternatives within the theories of generation: Individual germs are either formed one after the other one, in epigenetic succession, or they are all formed simultaneously. In the former case "le germ de fils doit sa formation à la vertu productrice de son pere", while in the latter "le premiere homme contenoit tous que sont sorti de lui".[23]

In preformed germs all constitutional alterations (that could predispose to disease) are necessarily posterior to the first formation (they cannot be attributed to the Creator), so the issue of hereditary transmission, as he defined it before, doesn't even make sense.

In successive generations, Louis argues, any conceivable transmission is made by a restricted portion (the generative one) of the parent's organisation, so there is no way a grandparent could actually and particularly affect the organization of the grandchild.[24] Louis dispells with this point what for many medics was one of the main peculiarities of the hereditary influences, their latency, or capacity to remain hidden in the individuals' constitution for some time, or through several generations. In any case, Louis writes, to conclude this part,

> les desordres de l'oeconomie animale doivent s'acquerir particuliérement par chaque homme: toutes les maladies seront individuelles puis qu'elles doivent être postérieures a la formation des

[23]It seems clear that though worded in male oriented terms, this argument applies to both ovism and animalculist preformation, and to hypotheses of male and-or female seminal fluids.

[24]Louis is here ignoring the traditional argument that has come to be known as "pangenesis", that is, that all the parts of a parent's organization contribute with causal (or material) elements for the constitution of the offspring.

germes qui n'ont reçu aucune altération dans leur principe.

What is then at the root of Louis scepticism is his strongly held belief that only general, non-individual characteristics are acquired by the new being through the act of generation, and all the contingencies of individual differences (and similarities) are a product of the interaction of this "essential" germ —preformed or not— with its environment, starting with the maternal nutrition during pregnancy. All idiosyncrasies —which according to Hippocratic medicine, add themselves up to constitute an individual's temperament— are pushed, by Louis' argument, outside of the possible reach of the hereditary.

The belief passed down by the medical tradition in a hereditary communication of temperament or constitution was, of course, at the base of most medical men unquestioned acceptance of hereditary transmission of certain (constitutional) diseases, and it was in consequence the target of Louis' most skillful and rhetorical —and most heretic— paragraph in his dissertation:

> Le tempérament des enfans qui naissent d'un même pere, & d'une même mere est presque toujours différent; les uns son bilieux, les autres sanguins; les uns son guais, les autres sérieux, pésans: ces différences d'humeur, de caractere et d'inclination dans les freres et soeurs, sont des suites de la différence des tempéramens; et elle depend peut-être moins de la constitution primitive ou radicale, qui paroît devoir être la même dans tous les enfans; que d'une disposition acquise par la combinaison infiniment variée de toutes les choses extérieures.[25]

Among the exterior influences Louis mentions the weather at birth, the suffering during birth, the amount of blood in vessels at birth, the quality of the nurse's milk, the thickness of air that was breathed during the first hours, etc. ("on ne finiroit à faire l'enumeration"). No wonder, he writes, that their are different temperaments within the same family.

Like most physicians of his time, Louis believes it is in the individual's temperament that resides the source and beginning of all illness, because it makes the person more or less susceptible to the effects that morbific causes can produce. Diversity of temperament is responsible for the differences of individual reaction to contact with such causes.[26] If such "diversité des tempéramens n'est point héréditaire —Louis asks— comment les maladies qui en sont les suites pourroient-elles se transmettre par les parens? "[27]

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[25]A. Louis, op.cit. p.35. This part of Louis exposition was obviously shocking to many orthodox physicians within the hippocratic-galenic tradition. Most of Louis' critics, a few decades later concentrated their attack on this fundamental assumption of temperaments as secondary and accidental, as I said these paragraphs preserved a lot of their provocative power for several decades.

[26]The humoral-solid physiological explanation Louis gives for this diversity goes as follows: "l'action des fibres plus ou moins forte & vigoureuse, faonne & modifie différemment les humeurs de notre corps; ces humeurs agissent suivant leur quantité sur les solides dans lesquels elles sont contenues, & elles en determinent diversement les actions: de-là viennent les complexions particulieres qui mettent tant de différence entre les hommes, tant par rapport aux dispositions du corps qu'aux caracteres de l'esprit".

[27]A. Louis, op.cit. p.37.

Louis admits that there are several diseases (like gout, stone and pthisis) that adopt a striking familial pattern of occurrence, and he understands the spontaneous imaginative movement of many simple minds in inventing a communicative link to transport them from parents to their offspring. But, he says, all those cases can be more accurately described and explained by external causes. He chooses as an example the well known example of Montaigne's bladder stone. The French essayist shared the infirmity with his own father and used the experience to raise his precisely worded question about the power of nature to achieve hereditary transmission of such complex things through something as simple as a drop of semen.[28] Louis is disappointed, he says, with Montaigne, because having seen the difficulty, the near impossibility of such transmission through a sign in something as amorphous as semen, he chose to believe in a quasi miracle. The fact that only he (Montaigne) among many brothers received the legacy, and that the communication occurred 25 years before his father realised he had the stone, should have put him off such an explanation, Louis believes. It is much more natural, he says, to imagine that the same combination of external influences, diet, habits, etc. was acting upon both father and son, who also shared a disposition to the disease for the same or different external reasons connected to their very first moments of existence. Cases of Gout and Phthisis are similarly explained away by Louis as non hereditary.

Two factors are repeatedly used by him in these examples to point out the absurdity of an hereditary hypothesis: the irregularity of transmission (some children are affected by same diseases as parents and most are not) and the length of time that elapses since the postulated causal communication and the actual development of the diseases.

In his final attempt, Louis tries to invalidate the two possible physiological, non-external routes of disease communication between parents and offspring. Not surprisingly, the two kinds hereditary causes of disposition to disease that Louis can imagine are on the one hand the humoral, and on the other the "solid" ones.

Humoral vices are discarded by him for several reasons. It seems unlikely that they would not destroy such a fragile thing as a germ. Beside that, one inherited morbific humor would conceivably produce a whole variety of different diseases in several parts of the body at several times and circumstances, so the pattern of the same and only disease in the same family claimed by some hereditarians would seem unlikely.[29] To add to the confusion, many different humoral influences could conceivably produce the same kind of symptoms and effects. Such unruly set of possibilities, Louis adds, is so confusing that one would have to use freely some "privilége de deviner, pour assurer qu'une telle maladie est ou n'est point héréditaire".[30]

Considering the solid communication of disposition to disease, Louis finds it

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[28]See Montaigne's quote at Introduction, above. See also Corcos, (1973), "Montaigne's insight in questioning heredity".

[29]This point was actually made "positively" by several humoralists before and after Louis essay, in order to try and reduce to one, or a few causal (morbific) humors all hereditary diseases. The climatic moment of this position seems to have been the defense of it made by such an important a physician as Antoine Portal, early in the 19th century, in his (1808) Considerations sur la Nature et le Traitement des Maladies de Famille et des Maladies Héréditaires.

[30]A. Louis, op.cit., p.50.

difficult to believe in any latency whatsoever. Any "hereditary" disposition based on malformation of solid parts, he writes, would be manifested immediately. Could an organ, he rhetorically asks, work well during 50 years if it is badly built?

Worst of all for any solidist defense of the hereditary, Louis continues, there is no way to really picture, or visualize, the transmission between solid, organized. parts. Furthermore, there are very many cases, well known and authenticated, of patently defective individuals (with difformities or mutilations of solid parts) that had perfectly normal and healthy children (blind parents with sighted children, hunchbacks with normal ones, etc.) and they seem to be the rule rather than the exception. If the constitution of the solid parts of the parent really did affect hereditarily, as a rule, those of the child, how is one explain such constant failures of the influence. And in any case, there is no easily conceivable way through which a physical flaw could affect the germ.

> Ainsi, je pense —Antoine Louis concludes— que quand on sauroit par révélation qu'il y a véritablement des causes héréditaires de maladies, il n'y auroit point de connoissance plus stérile, suivant ce que nous venons de dire sur la production d'une maladie par des causes différentes, & sur le déguisement d'une cause sous différens effets; pernicieuse fécondité, dont nous ignorons entièrement les bornes.[31]

His work, Louis adds in a note to the judges for the prize, is no doubt relevant to the question raised by the Academy, so it should not be excluded; moreover if his reasons should be considered of any weight, all the other, positive, works are invalid. The judges as we said were not convinced, or they had, as Ruffey suggests, made up their minds beforehand.

2.2 The antecedents to Louis' revolt

When publishing his essay, Louis incorporated as an appendix a long commentary on Robert Lyonnet's *Brevis Dissertatio de Morbis Haereditariis* (1647).[32] This text, Louis claims mistakenly, was the only previous one, in modern times, which had the subject of hereditary disease as its only topic. He also claims to have been surprised by the coincidences between his arguments and those of Lyonnet, who is also is sceptical about the humoralists claims about hereditary transmission, and who would only accept a solidist cause as truely hereditary: that is to say, that only solid parts (organs, tissues) can both be the seat and the cause of a hereditary disease, and its transmission to a descendant has to be seen as a solid to solid relationship. If to this premise, one adds the fact (that Lyonnet ignored) that there is no conceivable solidist causal link between the parents' body frame and their offspring's, Louis argues, the conclusion follows that there is no hereditary transmission.

There seems effectively to have been among European physicians a previous revolt against hereditary transmission of disease very early in the 17th century which shared with Louis' the mistrust of the excesses of 16th century humoralists and their uncritical resource to morbific vices. Alexis Pujol gives an account of this episode,

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[31] A. Louis, op.cit., pp.51-53.

[32] Lyonnet was the physician of the king Louis XIII. His dissertation on hereditary disease was written, apparently, to "tranquilizer l'esprit" of the queen, with regards to the many diseases of their son, the future Louis XIV. It was published in Paris, with 87 pages in 4o.

which he took from a testimony by an outraged François Ranchin. At the time (early 17th century), Ranchin "chancelier" at the medical school of Montpellier. Some medical men had gone to the extreme, Ranchin wrote, of denying "absolument" that any disease could be transferred hereditarily. Ranchin was so persuaded that those medical men were mistaken, Pujol adds, that he was prepared to maintain that they were defending such view against their own inner conscience, with perverted motivations such as the search of fame and notoriety, and not in a disinterested pursuit of truth.[33]

The revolt that worried Ranchin so much must have been related to the speculative excesses of a previous generation, especially I believe those of the famous French physician, Jean Fernel (1497-1558) who perhaps was the most influential writer on physiological and pathological themes of his age.[34] Among the many themes he worked on, Fernel made an attempt to explicate Galen's rather complicated effort of reconciling Hippocratic (dual seminal) and Aristotelian approaches to Generation.[35] Like Galen, he accepts the necessity of granting to Hippocrates the existence of a female semen, as the best way to cope with the contradictory facts of the hereditary, and he follows him also in the belief that the two seminal contributions, which must be mixed together before the new being emerges, need an extra, Aristotelian organizing influence. As David (1971) and Roger (1963) have described, strict Aristotelian philosophers always opposed any formal, organizing causal contribution from females in generation, and in doing so challenged the mainstream, dual seminal, medical tradition.

In his reconciliatory effort, Fernel inclines himself toward a non-material (spiritual) formative cause (or "virtue"). He then goes on to separate the explanations of the hereditary facts that previous authors had kept together. The positive ones (resemblances to father and mother) he explains through the spiritual cause, which he links to the mother's imagination. For the negative ones (hereditary disease and deformities) he gives a purely material cause, linking them to influences carried in the seminal contributions.[36] Following previous authors, like Paracelsus, Fernel seems interested in distinguishing between diseases that are acquired from a parent ("natural") and those acquired by external ("non natural") causes. The natural, pre-birth diseases he divides into hereditary (contracted through the semen) and connate (contracted in the uterus after conception, through the mother's blood for instance). In what was to be seen by many critics as an exaggeration, he postulated that all the diseases that a parent had, at the time of conception of the child, were candidates to be transmitted to it.

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[33]A. Pujol, op.cit., p.238.

[34]J. Fernel, 1655, *les 7 livres de Physiologie*, (translated from Latin), Paris, 775 p. Fernel's complete medical writings had 97 editions from 1554 to 1680, in Latin and different European languages. For an evaluation of his work see J. Roger (1964) *Jean Fernel et les Problèmes de la Médecine de la Renaissance*, Conférences du Palais de la Découverte, Série D, 70, Université de Paris; and L. Figard (1903), *Un Médecin Philosophe au XVIe siècle, étude sur Jean Fernel*, Slatkine Reprints, 1970, Genève.

[35]For an analysis of Galen's attempt see M. Boylan (1986) "Galen's conception theory" ; for Fernel's view on generation see chapter VII of Figard's book, op.cit, pp.191-219.

[36]For details on this see Chapter VI, "La Renaissance", in B. David (1971) *la Préhistoire de la Génétique*, op.cit., p.61-77.

> ...quelque mal que le père soit atteint quand il engendre, il le transfert à l'enfant par l'entremise de la semence; parce que la semence, étant derivée de tout le corps, elle contient en soi la vertu tant de la maladie que de la cause d'icelle. C'est pourquoi les vieillards et les valétudinaires font des enfants imbéciles: les graveleux, gotteux, épileptiques, laissent à leur race une constitution vicieuse par laquelle ils encourent enfin semblables maladies que pour ce sujet on appelle "héréditaires" de façon que les enfants succèdant aux pères ne sont pas moins héritiers de leurs maladies que de leurs biens, voire même le sang maternel, lequel sert de premier aliment à l'enfant pendant qu'il est encore au ventre de la mère est aussi cause du tempérament et de la constitution, et imprime pareillement ses vices au corps de l'enfant mais non pas si fort que le fait la semence.[37]

Other important 16th century medics, like Ambroise Paré (1510-1590) and André de Laurens (1550-1609) wrote about hereditary disease in much the same terms as Fernel. They shared with him a reliance on "virtues" and "faculties" (good or evil) as means of explanation, and as a way round (in the particular case of the hereditary phenomena) from the Aristotelian criticism that resemblance could not attributed only to material causal communication between parents and offspring due to the fact that it extended beyond the physical, to immaterial aspects of a person such as voice, movements and gestures. Paré for instance, insisted that semen does not come from the solid parts at all, but that it comes exclusively from the blood, from which the "ideas" of each of the parts of the body, and of other elements of the individual, are gathered into the formative virtue. It was this kind of increasingly immaterial explanation of the "humoralists" that exasperated the following generation. But contrary to what several historians have written about their stagnant age, in the subject of hereditary disease they all contributed clarifications that proved to be useful.[38]

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[37] J. Fernel, *Pathologie*, Livre I, Chapitre XI, quoted by B.David, op.cit. p.67.

> Since those relatively early times, this kind of association between inheritance and illness was accompanied by some kind or other of what we now would call an eugenic proposal. Fernel, for instance wrote at the end of this chapter:"Ces commencements de notre être nous importent donc beaucoup et ce ne sont pas peu fortunés qui ont une bonne naissance partant ce serait un grand bien pour la race des hommes s'il n'y avait que ceux desquels se portent bien et sont parfaitement sains qui s'employassent a faire des enfants, car si les laboureurs savent choisir le meilleur grain pour ensemencer leur terre ayant expérimenté que d'une semence flasque et gâtée on ne peut espérer qu'une chétive moisson, combien plus exactement cela se devrait-il practiquer pour la propagation de notre espèce".(Ch.XII)
>
> A partial translation of this passage to English can be found in Robert Burton's (1621) *Anatomy of Melancholy*, were a similar view is forwarded. (M. Dent's edition, p.215). Burton's exposition of the external (non natural) and internal (natural, including hereditary) causal combinations for the ocurrence of disease is an excellent synthesis of the 16th century view.

[38] Paré, for instance, emphasized the fact that a double seminal view of hereditary transmission of temperament provided a good explanation of generation jump of

Scepticism arrived, however, as we have seen, and the 17th century began with an increased attention of medical authors on the hereditary transmission of disease, per se, and not so much as a evidential weapon for their arguments against the strict peripatetic philosophers, and their one sided view of generation. The historian Amoreux describes it as a sudden outburst of publications of treatises with "haereditarii morbi" (or a variant of that formula) in their title; a cursory look at the appendix 1 (below) will show how justified he was in using the expression. Where all previous authors had treated the subject in their more general discourses on generation or pathology, from the early 17th century onwards special volumes were dedicated to hereditary disease. Contrary to what Louis wrote, Lyonnet's 1643 treatise was not an exception; there were various other authors who reacted against the extremes of humoralists and spiritualists, and their appeal to morbific virtues and faculties, and tried to develop mechanicist (solidist) alternatives for the phenomena of hereditary transmission.[39] Workable (picturable) transmission mechanisms based on solidistic causes (or in a combination of them with humoral ones as in iatrochemical hypotheses) were proposed in them and their influence lasted until well into the 18th century. Many of these treatises, for instance, were consulted in the 1750's by the encyclopedist (most probably Diderot) responsible for the entry under "Héréditaire (Maladies)" and commended as very useful by him.[40] Perhaps the most influential and clear of them was written by the Irish clergyman Dermutius de Meara, who synthetised most of the clarifications gained by 16th century authors and managed to develop a very convincing argument in his (1619) *Pathologia Haereditaria*.[41]

One of the main intentions of de Meara's treatise was to attack Fernel's view that all diseases are, or can be, hereditary and to return to the ancients' position that only those diseases that depend on defects of the organised solid parts (organs and tissues) are communicable in a hereditary way (through the semen).[42] No degenerative

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characters (atavism); specially of disease, given that a bad temperated element of one of the parents could be compensated by a good one of the other, and still be transmitted to a following generation in which,if not compensated in the same way, it would show its presence. Paré writes:"la semence fuit la complexion et tempérament de celui qui engendre...en sorte qu'un homme et un femme bien tempérés produiront une semence bien complexionée, alors qu'au contraire s'ils sont intemperés, produiront une semence mal complexionée et non propre pour engendrer un enfaint sain.....la bonté de la semence de la femme et température de la matrice, corrigeant l'intempérance de la semence virile, tout ainsi que celle de l'homme peut corriger celle de la femme." quoted from B.David, op.cit. p.70.

[39]After 1594 and before Louis' essay, at least three dozen dissertations on the theme had been published in Europe. Several of them written by influential authors. Mercatus (1594), De Meara (1619), Lyonnet (1647), Hoffman (1699) and Stahl (1706) among the most important of them.See appendix 1.

[40]The Encyclopedist (Diderot) mentions de Meara, Zeller and Stahl.

[41]D. de Meara, 1619, Pathologia Haereditaria. I will quote from the fragments translated into French by Bernard David, op.cit., ch.VI, pp.79-92.

[42]Lonie (1981) discusses the "solidist" origin of hippocratical medical writer's first views of hereditary transmission of disease, who took from Democritus a general (pangenetic) theory based on the body tissues (solids)and adapted it to their humoral theory. "But even in Democritus, molecular structures from the tissues must have been conveyed in some way, presumably in a fluid...the tissue interpretation and the

disease part (like a tumour or an ulcer) nor any disease dependant on mobile humors (like catarrh, feber, asthme) was, according to him hereditary, because

> dépendant justement d'humeurs fluctuantes et dépourvues de caractères fixes elles n'ont pas le pouvoir de donner leur empreinte à la semence[43]

Such fixity, de Meara believes, is only possessed by those morbid influences that can actually insert its roots in the solid parts of the body. Taking on board the proposals of a Paracelsian French medic Joseph du Chesne (1521 or 1546? - 1609), de Meara proposes a iathrochemical explanation, based on two salts, sulfur and mercury, whose presence at critical times in the tissues predispose the individual possessing them to certain diseases. I quote:

> Une maladie héréditaire est une maladie qui, lorsqu'elle a atteint un des parents et que sa racine s'est attachée d'une façon stable à l'une des parties solides quelconques de ce parent, descend, par une sorte de droit héréditaire, dans les héritiers[44]

By the word "parent", de Meara later clarifies, he does not mean only the immediate couple that conceived the individual, but more distant ones too, many of them of remote generations, as the root of the diseases can pass down through one or several generations without necessarily showing itself through the signs of the illness. Any constitutional disease that does not come in the semen (male or female), de Meara wrote, must be considered accidental, although most of them have to act in utero, while there is still some fluidity, or indefinition in the individual's constitution. A typical accidental influence is exerted by the nutrients the body receives from the mother's during gestation. These other influences are never however as strongly attached to the solid parts of the body as the roots of hereditary disease.

A theme that de Meara treates with some depth is atavistic transmission. He accepts the Aristotelian stance that there are, a priori, grounds for doubting the proposition that diseases, or any other characteristic, can be transmitted from grandparents, or any previous generation, to the newly born without it having been possessed by at least one of the parents. First, it seems impossible that a causal agent could act without direct contact with the subject receiving the action. And second, to be able to transmit anything from a first party to a third one, the intermediary must at some point have it itself. If it is a disease, then no one who is not afflicted by it, the Aristotelian argument would go, can transmit it.

Evidence shows, de Meara retorts, that patterns of occurrence of disease point towards the existence of such strangely behaved (latent) causes, but only a vulgar empiricist would feel satisfied with "raw experience". The medic, as the philosopher, should research the causes. The fact that both parents provide active elements in their semen, adds de Meara (following Paré), allows for a balancing effect, when the influence of one healthy parent abolishes the defects of the unhealthy one, or diminishes in such a way as to make it imperceptible. Impurity however is very rarely suppressed by the mixture of parental semen, and can still form part of the semen of the offspring, as this can be said to be an extract (or representation) of the man. Once

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humoral interpretation are by no means mutually exclusive". "Commentary on On Generation" in The Hippocratic Treatises, p.116.

[43]from David, op.cit.,p.83.

[44]from David, op.cit.,p.79.

transmitted to the following generation the impurity (a salt, for instance) can produce the disease even if the parent did not develop it. In all the participants, both ill and "healthy", the root of the disease is fixed. Such fixing of the root in the solid parts of the body is not however synonymous with having the disease. The prohibition of Aristotelian philosophers is thus bypassed, he believes, because although all causes act by contact, it can be said that this contact needs not be immediate, and can be mediated. A disease, it can be said, can be transmitted by the grandparent to a grandchild before it is born, in potency. There is something in the constitution of the intermediary parent that resists the expression of such potency, namely the healthy constitution of the other grandparent. There are situations, he adds, when the same cause can produce different effects in the children and in the grandchildren. The hereditary influence can either be resisted or not. Atavistic reappearance of a hereditary disease in a descendant, after having been absent in the family for one or several generations, ceases under this description to be a mystery.

De Meara concludes his discussion on hereditary diseases arguing that the curability of such diseases is related to the strength with which their roots are fixed to the solid parts of the body. They are in general more difficult to cure than non-hereditary ones. The strength of the hold is proportionate, he seems to be saying, to the level of impurity at the moment of the mixture of semen in fecondation. Good marriages (with healthy consorts) improve the situation by diluting the impurity, making the root easier to be taken out by chemical media. The latter, he affirms, following his Paracelsian inclination, must be specific solvents capable of washing the salt in question away. He also refers to an independent cycle that, he believes, hereditary diseases have that makes them increase first and then decrease their intensity, within a family, as they are passed through the generations.[45]

De Meara's account of hereditary transmission was based on solid causation of a kind. He relied heavily on Paracelsian (iatrochemical) physiology, and in that aspect it had a relatively short life span,[46] but his criticism of humoralist excesses, and his argument for a latency of hereditary causes brought closer the solidist dispositional account that characterized the most important medical authors of the late 18th century. The idea that it is not the disease itself but a disposition to it what is transmitted was a development of the use of Aristotelian potencies that previous authors (Fernel for instance) had made. A dispositional cause can remain latent, and is best pictured as a defect (or a pernicious element) in the solid parts of the constitution, that makes the individual prone to react to triggering external factors.

The other significant clarification that de Meara carefully stressed, and which proved important for the 18th century debate, was the limitation of the hereditary (or as he sometimes called it: the natural) to whatever cause or influence is acquired by the offspring's constitution at the moment of its first conception, and is brought there by one (or both) parent's seminal juice. Leaving strictly aside whatever influence came to bear on the individual's constitution after it acquired its definite (solid) structure. Familial illnesses could thus be distinguished between those with a properly hereditary cause (which were stubborn and remained within a branch for generations), and those which the children also carried at birth, but were acquired through

[45]See De Meara, in David, op.cit.pp.89-92.

[46]Although some physicians were still refering to his account of the root of hereditary disease well into the 18th century. See for instance the description of transmission of scrofula given in James', 1743, *A Medicinal Dictionary*, in chapter 1.

alternative routes, like mother's blood (the foetus' nutrient) or even mother's imagination (strong impressions, frights, etcetera.).

It is important to add here that many 17th century physicians did grant importance to other evidence beside the existing body of facts described by the ancients in the received literature. Their efforts at refining the concept of hereditary disease, as both Roger (1963) and David (1971) have shown, was accompanied by an addition of their own observations. which could only be case studies of patterns of recurrence of certain diseases within families close to them. The irregularities and whims of character occurence within a family mystified them as much as they had all previous generations, but their only tools, so to speak, were external observation of patterns, repetitions similarities, etc., captured in the form of narratives and cases; and the speculation about internal, physiological occurrences base on the inherited and slow moving Hippocratic conception of the body. The choice, for instance, of the diseases they saw as hereditary might seem odd to us (gout, melancholia, epilepsy, stone, scrofula, phthisis, etc). Such a list, it must be understood, stems from the widely accepted idea that deep, constitutional failings were the origin of all stubborn, age related, apparently un-caused and idiosyncratic illnesses. To this we must add the related idea that the individual's temperament (his-her particular combination of the four basic humors) was always the main determinant of whatever reaction it had to an external influence (climatic, or other).

The whole solidist critique of humoralists was only one of emphasis, as both groups considered the humoral-solid physiology as starting points. It is interesting however to point out that the gradual shift of language, from temperament to constitution (which are in a sense synonymous) seems to show a shift from humoralist to solidist explanations. The mechanical spirit of the age —Roger has argued— seemed to increasingly abhor the loose, excessively metaphorical language of temperaments, preferring the more precise, picturable one of constitutions, organization and solid interactions.

De Meara, as I said, tried to develop a workable picture of solid to solid transmission of disease, or its cause, through the seminal fluid. He was part of a wave[47] of discontent with the previous century' speculative proliferation of hidden faculties and immaterial causes. Roger's book (1963) has shown how this reaction coincided with the growth of Cartesian mechanicism that had as a consequence the adoption of pre-existence as basically the most tenable view of the origin of organisation and complexity in living organisms.[48]

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[47]According to Louis, R. Lyonnet, in his (1647) *Brevis Dissertatio de Morbis Haereditariis*, saw clearly the problem, was very emphatic about his rejection of the hereditary character of most humoral diseases, and wanted to limit the hereditary cause to those communicated through the father's semen (He called them thus *morbum seminarum*), as the semen was the authentique origin of the solid parts. Lyonnet's further explanation that the seminal spirit can receive alterations from the solid parts and communicate them to the following generation was dismissed by Louis as a regression to previous time's "qualités occultes". A. Louis, op.cit., pp.55-71.

[48]See specially Chapter III "A la recherche des idées claires" of this excellent book.

2.3 Pre-existence and the hereditary

By 1748, when young Louis took hereditary transmission of disease to task, the pre-existence of the germ had been for many decades the mainstream view of generation. A considerable number of medics, however, remained faithful to the ancient Hippocratic and Galenic "dual seminal" view.[49] Although, as I described above, Louis tried to make his general analysis applicable to both positions, the language and emphasis he makes give away his preference for pre-existence. Both Maupertuis and Buffon had recently shown that the messiness of the hereditary phenomena was one of the principal empirical obstacles in the way of pre-existence. Louis seems to have had this clear. So did Albert Haller (1708-1777), who shortly after him, when criticizing Buffon's double seminal view of generation and his use of hereditary resemblances as an empirical justification for it, produced a remarkably similar argument to Louis'.[50] A courageous argument based on denying the reality of such phenomena (the resemblances between parents' and childrens' physical constitutions) and on undermining the evidence in its favour.

Both Haller and Louis seem to have viewed, at that stage of their respective careers, the widespread belief in hereditary transmission of details of temperament, resemblance, and malformations as a pernicious prejudice that had to be checked. In the case of Louis, the ubiquity of variation was his evidence and the multifarious influence of secondary, external causes his theoretical resource. Under his view the original germ, pre-existent or not, was acted upon by innumerable non-natural (external) things that could produce in it different sets of secondary, accidental qualities.[51] Only the more essential qualities and organization were given by the

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[49]As evidence for this see the dictionary entries in previous chapter. They are evidence of what Louis considered to be a sort of drowsiness and mental inertia among the majority amongst the medics (most of them provincial and backwards looking) which, it can be argued, his essay aimed to challenge and upset. What he saw as an irreflexive clinging to the hippocratic-galenic tradition in general, and to its views on hereditary communication of the idiosyncrasies of temperament (among them some diseases, or a disposition to them) was however seen by those physicians as uncontroversially true, as they received verification in their personal experience, in their practice, with different members of the same families, from several generations.

[50]Haller decided to counter Buffon's Maupertuisian use of resemblance to both parents as an empirical fact which preformation couldn't explain (and which needs a dual seminal, successionist explanation) for that, he "prefer(ed) to deny to deny to Mr. Buffon that offspring resemble their parents. If I prove this point, the offspring are no longer images of their parents, and the remainder of the edifice will collapse upon itself. We leave aside that for any case in which resemblance to a parent can be adduced there always are a greater number of cases in which the offspring has acquired neither the traits nor the likeness of any of them. My thoughts go still further. There is no man who is similar to another in the internal structure of his body, and as consequence no child is similar to its parents." (adapted from Sloan's translation, Lyon and Sloan, eds. 1981). This last theme, of internal resemblance, was later to acquire a high profile in the discussions around the hereditary, as more observations were gathered.

[51]"les variations —writes Louis— décident donc rien en faveur de la question des maladies héréditaires, puis qu'elles ne vienent point d'un principe interne et de dispositions inhérentes et immuables; mais qu'elles dependant uniquement des chose

internal (germinal) route. No deviation or peculiarity that distinguished any family or group could pass through such route, and so they could not be called, in any proper sense, hereditary.

Louis made use of Boerhaave's well established physiology of fibres (as the solid elements of the body) and humors to bring home his point about the secondary role of the latter:

> l'action des fibres plus ou moins forte & vigoureuse façonne & modifie différemment les humeurs de notre corps; ces humeurs agissent suivant leur quantité sur les solides dans lesquels elles sont contenues, & elles en determinent diversement les actions: de-là viennent les complexions particulieres qui mettent tant de différence entre les hommes, tant par rapport aux dispositions du corp qu'aux caracteres de l'esprit.[52]

So, in Louis' mind, morbific humors of any kind could only have superficial, eradicable influences. The obvious weakness of his position was —as his critics insisted— his stubborn dismissal, as coincidences or tales, of all the striking cases of hereditary transmission of disease or malformation that impressed most other medical men.

But pre-existence allowed at least another approach to the hereditary that was, paradoxically, based on humoral causes. The judges of the 1748 Dijon contest were not apparently disinclined towards such a view, as they gave a special mention to G. Rey, a provincial medical man,[53] for detailing it. His basic argument is that the germ is in a state of emboîtement (in the mother) previous to its contact with the humors in the semen during fecundation, after which —Rey writes— there is "a development des fluides et des solides". It is this development undergone by the undifferentiated (though organised) germ that, according to Rey, made possible hereditary transmission of resemblance in general, and of disease in particular.

The two suppositions that Rey puts forward as reasonable candidates for proof are 1)that the germ interacts at fecundation with seminal fluid from both paternal and maternal origin, and 2)that "les deux fluides masculins & féminins, qui en decoulent, sont impregnés de toutes les humeurs particulieres du pere & de la mere".[54] Both hereditary resemblance and disease, Rey argues, are a consequence of the transformations that these humors induce in the solid parts while they develop. Only a

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non-naturelles qui sont toutes extérieures...Les hommes sont soumis a cette regle generale comme les plantes et les animaux leur caractère & leur tempérament dependent d'une infinie de choses extérieures qui peuvent être variées a l'infini: c'est une verité reconnue en médicine". Op.cit. pp.74-75.

[52]A. Louis, pp. 36-37. Towards the end of the 17th century, Boerhaave, who was one of the most influential physician and physiologist for the first half of the 18th century, developed an alternative solidist description of the body and, among many other things, wrote about the hereditary transmission of constitutional particularities within his physiology of fibres. See for the latter G.A. Lindeboom, (1970) "Boerhaave's concept of the basic structure of the body".

[53]From Chaumont, close to Lyon. G. Rey was the only author among the winners of the contest to respond to Louis' challenge by publishing, immediately after him, his dissertation: *Sur la transmission des Maladies héréditaires*, qui a balancé le Prix de l'Académie de Dijon en 1748, Paris, 1749.

[54]G. Rey, op.cit., p.10.

liquid (fluid) cause, Rey adds, can account for the mixing of characters (of both parents) in hereditary transmission, and previous authors have been wrong in trying to deny this. Liquids can permeate through all the bodily parts and affect the developing embryo in a pretty discriminate way because they can find their proper place of action through "affinities". There are "general" humors (lymph) which affect general parts, and particular humors which act upon specific organs.

Rey also argued that strange, or not completely compatible, liquids can affect and distort a developing part, Rey writes, because there can be partial affinities to them. Both the existence of "mulatres" and that of hereditary diseases can be explained, he claims, by this incomplete or distorted action of some fluids.[55] Either having the seed of a disease, or because they had the influence of a different variety or species, these fluids could thus affect and distort the general constitution, or a specific organ or part, depending on the kind of humor, general or specific. "Métissage" and the transmission of certain constitutional diseases (like scrofula or scorbut) are both due to the action of the "general lymph", whereas other, localized ailments are given by tainted particular one.

Two further crucial factual characteristics of the hereditary phenomena were dealt with by Rey with his humoral theory. One was the "irregularity" of transmission, and impredictability of outcome of crossings, which he explained, as the ancients had, by the fluidity and incompleteness of the mixtures, and the different power exerted by the lymphs of the mixture i.e male, female and the own germ's lymph.

The other characteristic he explained was atavistic transmission. Claiming that the only way to make sense of the "jump" of characteristics from several generations behind (avoiding the unphilosophical appeal to indirect causation or to latent causes) was by the existence of the germ inside the ancestors' body. Thus atavism is evidence for emboîtement. Fluidity also explains the way that this communication, for instance from grandparent to grandchild, is effected. The idea is that humoral "vice" can diffuse itself from the grandparent's body into the nested grandchild's germ, affecting it without necessarily affecting the own child (the parent), whose tissues can, in some cases, be passed through as a filter is without receiving the damaging seed.[56] The closer the nested germ is to be born (i.e. the less germ boundaries the morbid humor has to filter through) the higher is the chance of infection.

By "solving" at one stroke the problem of the origin of the solid parts of the body, the defenders of pre-existence left open the door for a humoralist revival. The hereditary influence of the seminal fluid can still be accepted under an ovistic theory, with the further advantage that Rey did not use, of avoiding at the same time the problem of the female semen that plagued the double seminal accounts.[57]

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[55]"il paroit - —Rey writes— que les corps parfaitement homogénes sont parfaitement miscibles entr'eux, & que les autres refusent plus ou moins de s'unir & de se marier ensemble, suivant le degré plus ou moins grand de vleur hétérogeneité, ou plutôt de leur improportion".Op. cit., p.16.

[56]Rey recognized that under ovism this only gives a straightforward explanation of maternal line atavism, but did not extend his argument in this direction.

[57]This logical possibility was seen by nobody as clearly as by Charles Bonnet, whose highly sophisticated view of generation depended on his attempt to explain the hereditary transformations that the male's semen induces on the pre-existent germ in the case of mules. He felt sure that all resemblances could be accounted for once this, the most striking of cases, was understood. Concerning hereditary disease, Bonnet felt

It can be said, I believe, that with regard to the hereditary, the conundrum in which the medical community was entangled in when Louis and Rey wrote their pieces (and which became more acute after them) had to do both with the demise of ancient humoral physiology, and the strength of the arguments for pre-existence and against successive generation. As we have seen, humoral physiology and the Hippocratic-Galenic double seminal view of (successive) generation had, so to speak, the facts of hereditary recurrences on their side. Both irregularity and resemblance to both parents and to ancestors, etc., fell with certain ease under their explanatory wing, while the rival theories had to stress themselves, or simply ignore as irrelevant those same facts. The problem was that all appeal to proliferating humors, though still popular among many medics, was seen as completely regressive by those trying to leave behind the retarding weight of the ancients. Particularly questionable was the idea of humors which could have discriminative powers in order to act selectively and subtly, thus giving all the nuanced effects that were found in the hereditary.[58] "Solidism", with its more down to earth view of causation and individuality, was seen by most as the best alternative, one that was favoured, by the rising profession of the surgeons.

If constitution (or temperament) was to be basically understood as dependent on the solid parts (as a whole or as a set of separate organs), then disposition to disease was to be dependent on the organization or structure of the whole bodily frame, or of particular tissues or organs. So much was clear, but the problem remained of how to account physiologically for their "hereditary" transmission, whose reality very few medics were really willing to deny.

At the same time, the debates around generation (after Haller's and Bonnet's strong attacks on the mid 18th century wave of "successionists" and "epigenists" like Maupertuis, Buffon and Needham)[59] seem to have entered into a sort of impasse or

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even more confident of explaining them, as he wrote:"Les maladies héréditaires souffrent moins de difficultés. On conçoit facilment que des sucs viciés doivent altérer la constitution de germe. Et si les mêmes parties qui sont affectés dans le pere ou dans la mere, cela vient de la conformité de ces parties qui les rend susceptibles des mêmes alterations"(p.32) See *Considérations sur les Corps Organisés*, tome 3, Oeuvres d'Histoire Naturelle et de Philosophie, 1779, Chapters III and V of part 1, and Chapter VII of part 2.

[58]"On sait aussi —wrote Pujol concerning this point— qu'il est bien des Médécins modernes du plus grand nom, qui rejettent de la Médecine tout ce qu'on nomme causes humorales des maladies, comme êtres phantastiques et absolument imaginaires; pretendant que tous nous maux sans exception on pour cause nécessaire quelque vice des solides, ou ce qui revient au même, quelque vice organique". op. cit., p.228.

[59]There is as I said above some confusion around how to call the position of those who opposed pre-existence. Bowler (1971), following Roger (1963), has suggested that position's like the ones held by Maupertuis and Buffon should not be called epigenetist (though they were called so at their time) because they did not advocate a progressive development and appearance of the parts, but a sudden organization of a the new organism from the mixture of semens. Louis' division between pre-existence and "succession" perhaps helps, in leaving epigenesis free to describe developmental views, such as Wolff's. The problem with all this, to my mind, is that with it we are

stalemate. Most authors of the next generation who could not see how to decide between the very coherent and complex arguments developed by the opposing factions with the available evidence.[60] Some medics felt very uneasy about their appeals to hereditary transmission of constitution (or temperament), because as a very basic supposition in most of their approaches to individuals with idiosyncrasies both of disease and of reaction to treatment it did not however have a solid enough physical or physiological base. Beside that, the rhetorics of case collecting and storytelling (very common in the medical tradition, and used to establish and disseminate the belief in hereditary disease) were increasingly coming under attack by both statistically oriented, materialistic and mechanically minded authors of the late 18th century.[61]

As Louis wrote: the category of hereditary disease was very common in informal parlance among medics but was, in the 18th century, normally not considered in the classifications of disease.[62] Either because the origin of the disposition (or diathesis) wasn't considered crucial for its cure, or in the other extreme, because hereditary diseases (or the dispositions) were considered incurable. The fact is that by the 1780's the phrase itself "hereditary disease", so pervasive among medics, was becoming increasingly difficult to use as self-explanatorily as before. The hidden humoralists associations it conveyed and the absence of a proper (solidistic or other) account of transmission increasingly worried the most theoretically minded physicians. In France, Louis' challenge had basically remained unconfronted. In Britain, as we shall see, similar sceptical arguments had began to appear.[63] It was under this situation that a second essay competition was organized amongst French physicians with the subject matter of hereditary disease. This time by the Parisian Société Royale de Médecine between 1787 and 1790, as close to the edge as it unknowingly was.

2.4 Maladies Héréditaires and The French Royal

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anachronistically imposing over the 18th century authors our own views of what a developmental theory is.

[60]See for this E. Gasking (1967), *Investigations into Generation 1651-1828.*

[61]What makes unique the discussions that physicians had in late 18th century France around the subject of hereditary disease, is that they reversed the terms, so to speak, on which the debates on generation had been carried. The justification of their belief in hereditary transmission of dispositions and idiosyncracies was their aim, and the discussions over generation were a mere background, which they conformed to but did not feel they had to slavishly follow. They concentrated their efforts in producing viable transmission hypothesis with their physiological theories and the growing restrictions that anatomy, chemistry and the accumulation of observations were setting them.

[62]Neither Ph. Pinel nor Cullen, the French and British classifiers of disease included hereditary as an important nosological category, although they both strongly relied on ther concept at some point in explaining madness (Pinel) and gout (Cullen). This of course can also be due to the equivalence for them of constitutional and hereditary. Or the belief that constitutional diseases tend to be hereditary.

[63]The British physician William Cadogan (1711-1797) published in 1771 a forceful criticism of hereditary explanation of gout and other constitutional diseases. See chapter 3 and appendix 1.

Society of Medicine (1788-90)

I have tried to show above how Antoine Louis' small 1748 essay on "maladies héréditaires", helped by his posterior acquisition of prestige and power, gave to the subject a unique status in pre-Revolutionary 18th century France. Beside the purely conceptual reasons (which as we saw were not few) many medical men had other motives to meet his, and other sceptics' arguments. Perceived as coming from the controversial permanent secretary of the Académie Royale de Chirurgery, the arguments must have acquired a particular poignancy, especially within the Academie's rival, the Société Royale de Médecine. Eventually, this institution promoted the search for a solution to Louis' challenges in two successive competitions (1788 and 1790).[64]

As said above, the tone in which the questions were set made it clear that the Society did expect the competitors to face Louis' challenge head on, and did not want them to fall back on the old, received, presuppositions and unspoken assumptions about the obviousness of the phenomena of hereditary transmission of disease, and on the same less than rigourous, unverifiable explanations (humoral, vitalistic or other) that usually accompanied them. The questions set for answer by the Society on the public session of February 27, 1787, with a prize of "600 livres" were: Déterminer 1o. s'il existe des maladies vraiment héréditaires, et quelles elles sont: 2o. s'il est au pouvoir de la médecine d'en empêcher le développement, ou de les guérir après qu'elles se sont déclarées.[65]

According to the judges' annotations, there were 13 dissertations sent to the first contest. 12 of them survive in the archives of the Academie de Médecine (Paris). The length and quality of them vary very much, but there are several worth looking into, as they were carefully researched and forcefully argued.(See appendix 1 for details).

After what seems to have been difficult negotiations (the evaluations of the judges that remain on paper[66] show that they favoured different candidates for the prize and that they evaluated with markedly different criteria) the jury decided not to grant the prize, but to re-open the contest with the same questions but raising the prize money to 800 livres. The jury declared itself not satisfied with the results, but three dissertations were singled out as valuable. One, written in Latin in an elegant aphoristic style by the Viennese doctor Michel-Raphaël de Gellei, was given a "prix d'encouragement" of 100 livres, as the only one which had understood "le sens du programme", although in many instances his responses seemed inadequate or incomplete. Of the other two dissertations mentioned in the report of the Society as

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[64]Who was directly responsible for the choice of subject I have not been able to discover, but it seems to me likely that Vicq D'Azyr, the brilliant and theoretically minded permanent secretary of the Society, had some influence in the decision, as he later published in the section he was coordinating of the Encyclopedie Méthodique one of the most analytical and clarifying essays (submitted by Pagès). Another senior member, Thouret could have had some influence. He was the most influential among the adjudicators of the prize.

[65]See Histoire et Memoires de la Société Royale de Médecine, vol. IX, 1786-87, p.17.

[66]Minutes d'examen de memoires sur maladies héréditaires, 181-23-1,5, Archives de L'Ancienne Société Royale de Médecine, in Library of the National Academy of Medicine, Paris. All documents described with similar codes (v.gr: 154-9-4) refer to the same archive. See appendix 1 for a list.

having some "well presented details" one is missing from the archives, and the other one is the first version of Alexis Pujol's essay on the subject.[67]

In the summing up, the report detailed further the discontent with the work sent

> La plupart des concurrens ont supposé plutôt qu'ils n'ont prouvé l'existence de maladies héréditaires; ils n'en ont pas assez exactement déterminé la nature. Il s'agit de savoir si quelques vices morbifiques se transmettent réellement & individuellement des père aux enfans, ou si les maladies qu'on appelle héréditaires, ne sont pas plutôt une suite de la conformation des organes, qui, dans les pères et dans les enfans, doivent être, à raison de leur structure, sujets aux mêmes affections. C'est sur l'existence & la nature de ces maladies qu'il faut sur-tout porter ses recherches.[68]

The Society was here rephrasing Antoine Louis' old question. If, on the one hand, diseases that "seem" hereditary are only due to common defect of conformation of the solid parts, and there is no clear way in which the individual conformation of the parents body can have a direct causal effect on the conformation of the embryo; and if, on the other hand, hereditary transmission of disease through humoral vices cannot be said to have any particular route or character that would justify the separation of it into a different category; then there is no real sense in using the old analogy of inheritance.

The first set of comissioners thought that there was not enough scepticism in the essays and-or there was not any clear picture of how transmission can really (not speculatively) be accounted for. They wanted the contenders to fight Louis in his own terms. This impression is certainly reinforced by the fact that de Gellei's essay was praised in one of the judges' personal notes (Thouret; who was, by the look of it, the dominant judge) for beginning by throwing doubts on the existence of hereditary disease and then building up his way into proving there existence by refuting the doubts. On the other hand, that same judge criticized several of the dissertations (including the missing one coded as F) for admitting too readily the fact of their existence. At the same time he praises in "F" the style and manner of the rest of its argument.[69]

Several of the contenders of the second round (some of them having rewritten their first version) began their arguments by showing certain surprise, and some outrage at the Society's demand for further proof of the existence of hereditary transmission of disease, claiming that the overwhelming number of indubitable cases gathered in the literature —and seen every day— was enough to take any reasonable person close to certainty (which was exactly what some judges did not want to hear). They complained of undue bias. One of them later wrote a personal account of how he saw the procedure of this contest and went as far as accusing the judges of having reversed a previously made decision that favoured him for ideological (anti-religious)

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[67]The report only gives the epigraphs under which they were submitted. The missing dissertation had one from Voltaire with a sceptical air: "Il ne suffit pas qu'un système soit possible pour mériter d'etre cru". Pujol chose a classical Hipocratical dictum:"Semen ab omnibus partibus prodit, à sanis sanum, à morbis morbosum". See *Histoire de la Société Royale de Médecine* , op. cit. p.18. Pujol's first dissertation is number 200.2.9 of the archives of the old Royal Society of Medicine.

[68]*Histoire de la Société Royale de Médecine*, op. cit. p.18.

[69]See "Examen des mémoires...", op.cit., especially 181-23-1.

reasons.[70] The fact is that many of them were surprised that good, scholarly well informed and well argued dissertations did not manage to convince the judges. Amoreux (whose first dissertation had been the favourite candidate for the prize of at least one judge) wrote that he did not see how anybody could satisfy the Society with more; having quoted almost every important author, ancient and modern, on the subject, and having mentioned a fair number of reliable cases, having argued fairly and clearly about causation with the prevailing physiological knowledge, what else could anyone do? he asked; then proceeded to re-write his piece adding detail to every one of its parts, especially to the already outstanding bibliographical research.

Apart from the rightful claim that most authors had been somehow begging the question in their 1788 entries, it can be said, I believe, that the many considerable analytic virtues of some of the essays submitted to contest were ignored in the first set of judges' reading. Several authors displayed a striking clarity about the distinction between congenital and connate causes (which was later praised by the second group of commissioned judges), emphasis on the need for clear-cut observational criteria to distinguish the hereditary from other influences (like homochrony, specificity, curability, etc.) and about the inevitable relationship between "normal" and pathological heredity that solidism (but not humoralism) presupposed. Perhaps a reason why the first set of judges were not impressed is that these ideas were more common amongst French 18th century medical men than it appears at first sight. The judges' reluctance did however provoke a sharpening of the arguments presented for the re-run of the competition.

In 1790 a new set of judges, and a new and more carefully argued set of pieces produced a different result. At least four dissertations of very high quality, although not agreeing in all aspects, synthetized in a strong and authoritative way why a physician of the late 18th century could and should defend the principle of hereditary transmission of disease (or better still, of the disposition to it) regardless of what some theoreticians could sceptically say or write. These were those dissertations written by Amoreux, Pujol, Pagès and Rougemont.[71]

Although again not completely satisfied, the Royal Society commisioners decided to grant the prize to Joseph-Claude Rougemont's (1756-1818) contribution.[72] Under a "eugenic" epigraph from Fernel (*Maxima orts nostri vis, nec parum felices benè nati*), Rougemont, according to these new judges, treated the question under all its

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[70]See A. Pujol's "Notices et éclaircissemens préliminares sur cet ouvrage", op.cit., pp.211-236.

[71]Other dissertations could be added to this list. For instance those of Ladevere (119-30-5) and Girard (119-33-A) were excellent in some aspects but were less well rounded. Both Pujol's and Pagès' works were published around the turn of the century, as we have seen above.

[72]Unfortunately the manuscript of Rougemont's prize winning essay is missing from the archive, so I could not profit from it for the present work. For some reason Rougemont's text was not published in France. As he was Professor of Anatomy and Surgery at Bonn University at the time, his work on hereditary disease was published in Franckfurt, in 1794, in a German translation, and according to Steinau (1836), was a classic on the subject in early 19th century Germany. For Bio-bibliographical details on Rougemont see Dezeimeris et. al. (1828-1839), vol. 4, pp.24-25. In the same work information can also be found on Alexis Pujol (vol.3, pp.764-65), and Pierre-Joseph Amoreux (vol.1,p.111).

aspects, and made an "exacte & sévère analyze de tous les écrits & de tous les faits qui ont quelque relation avec le problême proposé". He carefully distinguished hereditary diseases from those that the child can contract within the mother's womb or during birth. Some shortcomings in method, they add, are balanced by the clarity he brings to the whole subject. An "accesit" prize was given to Pierre-Joseph Amoreux, whose great historical erudition is highlighted (the essay by Girard (119-33-A) was also very good on this aspect), but who failed on the "prophilactique & curatif" aspect. Both Alexis Pujol (1739-1804) from Castres and Jean-François Pagès from Alais received honorary mentions. Among their many merits, the Society again chose to praise their clear distinction between congenital and connate diseases.[73]

2.5 Responses to the Sceptic

As I said, the commissioners of the Royal Society of Medicine were not yet totally satisfied with the second, improved, round of essays. But they gave the prize anyway, because it already had been remitted once. The question —the comissioners stated— was not yet solved. Far from it, they wrote, there is a need of "nouveaux éclaircissemens" that medical men would need to apply their zeal to.

> Dans ce genre —they added— les observations isolées considerées séparément, ne peuvent avoir qu'un degré d'utilité très-borné. Ce ne sera qu'en les réunissant & en les comparant, qu'on pourra leur donner de la valeur.[74]

This invitation to surpass the narrative, case-quoting method that - ----according to some authors— had plagued the subject for too long and gather the evidence in a more cummulative fashion, was a clear recognition that the consensus amongst wider

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[73]This distinction was made by many authors in slightly different ways. The meaning of this vocabulary is sometimes confusing, and as Pujol pointed out (see op.cit. p.231) even the Societies' commissaires seem not to have had the distinction clear. Traditionally, as we have seen above, the connate diseases are those acquired after fecundation by contact in the mother's womb with her humors (through her blood). Some authors include among these those diseases and defects acquired by mechanical influences during pregnancy (blows, etc.), and others believe that mother's spirit, imagination or states of mind can also exert some kind of influence and produce connate peculiarities. Under old style humoralism many authors did not see any reason why not to include the influence exerted in the newly born child's constitution by yhe mother or the nurse's milk. Congenital diseases on the other hand are those believed to have been transmitted to the offspring at the moment of its first formation, via the parent's seminal contributions (fluid or solid). Under a solidistic perspective this was the only kind of truely hereditary transmission. Humoralists were more divided around this matter, some wanting also to restrict the hereditary to influences at the first formation, while others saw no reason for this restriction, considering any humoral cause communicated by any of the parents before the constitution of the infant becomes completely fixed (i.e before the end of the first year) as worthy of the hereditary title. Although old, then, the distinction between congenital and connate was difficult to determine, the best participants in the 1790 contest can be said to have brought to it a renewed clarity.

[74]*Histoire et Memoires de la Société Royale de Médécine*, vol. IX, 1787-88, pp.x-xi, Paris.

sectors of the medical community was changing, away from their old, case-based and bookish inductive strategy, towards a more "statistical" approach. In several competition essays, authors had acknowledged that Louis had a point when he questioned the use of tales and anecdotes to justify belief in the hereditary, and was right when he wrote that "les principes qui forment la vraie Thèorie de la médecine ne s'aquierent que par des recherches penibles, et des travaux longs et difficiles".[75]

However, Louis' methodological point was not the focus of so much attention in most competitors' works (as it was to become for hereditarians of the following century). Other aspects of his challenge took center stage. Four of the issues raised by him were recurrently and vehemently taken up by the more acute participants; I will briefly describe how these issues were tackled by the more lucid competitors.[76]

The challenge to the Parent-Offspring Resemblance of Constitution (or Temperament) "Theory"

This was Louis' most dramatic challenge to 18th century medical common sense. Hereditary communication of at least some of the components of an individual's Temperament (or Constitution) was unquestioned by the whole medical tradition. Under humoralism all resemblances (moral, physical, pathological) within a family (or other genealogical group) can be attributed to the similarities in the proportions of the different humors. With the rise of mechanistic solidism in the 17th century, family (and group) resemblance became linked with the explanation of the origin of solid parts, and their posterior malleability under the action of humors. Alternative generation theories have different bearing on this issue. Pre-existence, as we saw in Louis' and Haller's cases, could provide a base for the radical questioning of the reality of any causal link between parents' and offspring's organizations, i.e. of their resemblances. Constitution (solid organization) is something that comes with the pre-existent germ, and any predisposition to disease (or diathesis) is either already there, or is acquired, but not inherited; unless you open your criteria and include humoral causes into the hereditary, in which case the problem exists of separating those causes from other, external humoral influences. As shown above, this is the position adopted by Rey in 1748, and also —with variants— by many of the 1788 and 1790 competitors that were not prepared to abandon humoral hereditary influences.[77] The

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[75]A. Louis, op.cit., p.4. In this point, Amoreux, Ladavere, Pujol, among others, did try to justify their inductive procedures, and as I said felt somehow betrayed by the Royal Society's siding with Louis in this issue. They believed that carefully selected cases, accompanied by sound physiological and pathological knowledge, did provide a good base for making general statements about hereditary transmission. The adequate selection of old authorities, and specially the experience that many years of practice with sometimes different members of the same family gave to the best observers amongst medical men, clearly provided them with a sound basis for belief. On the other hand, sheer accumulation of cases never would compensate for the combination of a well trained eye with a well read mind that good, old style physicians had.

[76]A brief description of the structure of each essay submitted for the 1788 and 1790 competitions can be found in appendix 1.

[77]A reason being that so some of the traditionally hereditary diseases seemed to be of a humoral nature (like scrofula). On the other hand (and under the assumption that lodged in different organs, one morbid humor would produce different diseases) the "heritability" of humoral causes among the hereditary provided an economy of

pre-existentialists among them (especially Besuchet (200-d-2, 3)) used Bonnet's ideas about the influence of seminal humors in the transformation of the germ during its development, which as I said were similar to Rey's.

The strongest solidist in the 1790 competition was Pagès. Where others (Pujol, Amoreux, etc.) were prepared to accept that certain hereditary diseases had as their main cause a morbific humor ("levain", "virus"), Pagès was inflexible in his refusal to grant that point. His solidism he related also to the disputes over generation. Although he denied that generation theories should have a primal position in the discussion about hereditary disease,[78] , he argued that dual seminal views (like Hippocrates' and Buffon's) made it easier to account for hereditary transmission of constitutional traits, both from father and mother, and from ancestors; and would also help draw a line between real hereditary influences (internal: incorporated into solids at first formation) and secondary (humoral, external) ones. In his view, only a small set of specifically transmitted (through generation, in semens) constitutional predispositions to certain diseases deserve the adjective hereditary.[79] The obscurantist and abusive practice of many previous, and contemporary, physicians in applying the adjective to any —and every— disease could be checked, Pagès believed, with clearer external criteria, derived from a clear definition. The principal criteria had to be the time of appearance of the disease.

> Un caractère essentiel —he wrote— des dispositions héréditaires c'est d'observer pour leur développement, dans les enfans, la même époque, le même age que chez les parens[80]

"Homochrony", as this came to be known after Haeckel, was also stressed as a crucial element of the hereditary by other competitors. Pujol and Amoreux used it to separate humoral hereditary influences from non-hereditary ones. The transmission of the influence would occur through the semen (or the mother's blood) and it would then either affect the constitution during the first formation or remain in the body without effect until at a later, determined stage, it would produce its nocive symptoms. As Amoreux wrote, hereditary diseases are usually not carried by the children at birth but are "developed" at a certain age because

> transmissible au moment de la formation, par un hétérogene mélé a la

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causes, given that the persistence in one family of only one kind of morbid humor could account for different affections in different individuals and generations. Very many 18th-century medics seem to have believed this.

[78]"Je croi —he wrote— que la nature des maladies héréditaires, loin de recevoir quelque lumière de la part des hypothèses de la génération, doit au contraire leur fournir des preuves, & que si on parvenoit à la connoître clairement, cela répandroit beaucoup de jour sur le mystère de la génération". Pagès, op.cit., p.162.

[79]They are "epilepsie, hemoptysie, pthysie, manie, melancholie, hysteria, hypochondria, & apoplexie". I believe that Pagès' stern criteria, and strong solidism appealed to Vicq D'Azyr more than the other authors' "eclectic" positions, and that is why, although not given the prize by the Royal Society, it was this essay that he included in the Dictionnaire de Médécine of the Encyclopèdie Méthodique.

[80]Pagès,op.cit., p.160. This author explains homochrony based on a peculiar physilogical theory. He sustains that each organ of the body has a certain period, in the individual's life, at which it exerts its main influence. It is when it is "switched on" when the period arrives that the organ weaknesses and latent predispositions are revealed, in the form of ailments or disease. See p.163.

> semence prolifique, le principe morbifique s'entre, pour ainsi dire, sur le germe seminal au moment de la formation, et ce principe plus ou moins fortement, se modifie et s'altere pendant l'acroissement du foetus, de l'enfant et de l'adulte, et donne lieu ou à un mauvaise tempérament ou à une maladie, ou enfin à une simple disposition[81]

The other basis of Louis' scepticism concerning resemblance of general constitution was the uncontrolable proliferation of causes (external and internal) acting at the moment of the first formation. To counter this argument, Amoreux first accepts that variation among children of same family is a striking reality, and that it is due to the varying influences at time of conception. However, he adds, the weight each kind of cause has in shaping the individual's temperament is not equal. Primary, humoral and solid, hereditary causes far outweighting the secondary, environmental ones. It seems undeniable to him, as to many others, that peculiarities of temperament and constitution run in families. As Pujol wrote

> ...s'il est vrai que la couleur de la peau soit héréditaire parmi les hommes, comment les tempéramens ne le seraient-ils pas, eux dont cette couleur est ordinairement le signe et même l'effet? ...on ne conçoit comment cet habile homme [Louis] a pu se determiner à nier un fait si notoire et si général. ...la propagation des tempéramens, par voie de succesion et d'héritage est un de ces faits généraux dont il est aisé de constater la realité, dès qu'on veut examiner curieusement et en détail les differentes familles dans la réunion qui composent les grandes cités.[82]

This curiosity however was not to remain on the superficial qualities of colour, height, weight, and form of the body, but to be extended to internal constitution of tissues and organs. Surgery provided, in the late 18th century, an new observational window that few of the contenders failed to mention. Resemblance within families could be traced to the minor details of inner configuration, opening a window to a multitude of other facts that increased both the number and the evidential strength of hereditary claims. Specially when peculiar hidden defects began to turn up in autopsies. As Amoreux wrote

> Les anatomistes se sont quelquefois apperçus d'une conformité de structure ou d'organisation defectueuse en explorant les cadavres de plusieurs sujets de quelques familles; et les exemples seroient sans doute plus frequents si ont suivoit avec plus d'attention ces sortes de recherches[83]

"Normal" resemblance within families had since Hippocrates been used, in an analogical argument,[84] to justify the belief in "pathological" resemblance. The rise of solidism —and surgery— tightened this analogical move amongst physicians. The emphasis on personal observation (and less reliance on ancient reports) and the attention to structural detail, reinforced the medical men's confidence in the reality of

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[81]Amoreux, 1790, pp.17-18.

[82]Pujol, op.cit.,p.248.

[83]Amoreux, op.cit., p.15. This confronts Haller's denial of internal resemblance.

[84]The importance of this analogical argument cannot be exaggerated fo the history of hereditarianism. As Canguilhem has shown, (1972) the move from normality to pathology was not a natural one until well into the 19th century, so it was I believe an anomaly of the hereditary. Blumenbach for instance reacted strongly against this kind of argument before adopting them himself, and popularised them.

hereditary transmission of individual (idiosynchratic) constitutional characters in general, and of the predispositions to diseases that they could entail. Whatever the way the first "rudiment" or embryo came to be formed, they were convinced, there had to be a causal mechanism responsible for the impression on it of (some of) both of it's parents particular constitutional characters. For the want of a better model (which generation theories did not provide) Pujol described this by a metaphor

> la même main qui calque si scrupuleusement la physionomie du fils sur celles du père et de la mère, doit passer aux ressemblances intérieures, et rendre avec la même exactitude, organe pour organe, viscère pour viscère.[85]

This hand, this mechanism whose basic "external" manifestations late 18th century medical men were trying define, was to become, some years later, l'Hérédité; Heredity, with a capital H. The idea of such a general (unified) mechanism for hereditary transmission of both "normal" and "pathological" features was facilitated then by the strenghtening of solidism. Humoral morbific causes would always maintain a connotation of a poison: alien, external disruptive influences (somehow more easily eradicated). On the other hand, the solid-solid communication (through a "normal" mechanism) of conformational flaws, provided with a perfect frame for the analogical reasoning described above. A frame that somehow could encompass naturally all the biological phenomena that came under tha aegis of the adjective "hereditary", normal or deviant. From family resemblance to hybridization; from transmission of physical deformity to hereditary disease. This strenghtening of the analogical domain, by unifying it under one kind of causal mechanism of transmission (and not a diversity) opened, as we shall see, the door to a further step: from visible resemblances to invisible ones.[86]

The challenge to the causal-physiological resource to humoral causes, and their confused and malleable non-specificity, or proteism.

Humoral causes for hereditary diseases were defended by several of the contenders. Pujol, for one, was specially annoyed that the Royal Society preferred to stand by Louis' speculative denials. He decided to write for the second (1790) contest a whole new chapter trying to prove this. He started by arguing that

> la transmission des vices humoraux et virulens par voie d'héritage ne sera jamais prouvée complétement par des raisons speculatives[87]

Due to the close interdependence between humors and solids, he claimed, it is never easy to pin-point the original bearer of a morbific cause. Ill humors affect solid parts as much as diseased solid parts alter the fluid parts' normal composition.[88] To restrict the hereditary to solid-solid transmission is, according to him, to blind oneself to empirical evidence, both physiological and pathological. Different tainted humors

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[85]Pujol, op.cit.,p.244.

[86]Which was to prove crucial for the leap from physical inheritance to moral inheritance.

[87]Pujol,op.cit.p.228.

[88]Amoreux:"Un virus transmisse une organisation vicieuse derangeront bientot l'harmonie qui doit regner entre les parties solides et les fluides; de la des maladies organiques et des maladies humorales" op.cit., p.11.

can act over solid structures at different times, it is sufficient to show that some of them have acted on the conformation of the germ before or at the moment of its fecundation, or that it was incorporated to its bodily fluids at that moment by forming part of the semen, to call its effect hereditary. Many observations point in that direction, he continues. The fact that some of these "levains" can also be communicated non hereditarily is no reason to deny this. Amoreux adds to this that strict solidism is untenable because many real hereditary diseases cannot be unambiguously classified as either humoral or solid.

The capacity that humoral vices have to produce many different kinds of effect on different organs at different times (their so called proteism), that was given by both Louis and Pagès as a main reason for discarding them as bearers of real hereditary influences, was seen by Pujol and Amoreux as a further reason for keeping them within the field. Following the "transformations" of humoral hereditary diseases from generation to generation within a family would eventually lead to a reduction of hereditary influences to a small set of that could account for the diversity observed.[89] Hereditary causes then are not only reduced in number but they gain in "extension". Dispositions to different diseases need not have each a particular humoral cause, but perhaps only a particular effect over an organ or tissue. All hereditary diseases might still depend on constitutional flaws, but they would need a tainted humor as a vehicle for inespecific transmission. Antoine Portal took this position to an extreme when he wanted all hereditary diseases to be caused by one proteic vice, the scrofular.

Contrary to the claims of both Pujol and Amoreux, their dual causal approach to the hereditary left too much room for all kinds of diseases to be considered, one way or another, as hereditary. This proliferative and unbounded character (as both author's rich classifications show)[90] was precisely what some judges were against, and what was at the root of Royal Society's formula "Maladies vraiement héréditaires". That is why they lost the competition.

The curability of some hereditary diseases was also at the root of the defense of humoral causes for them. It was widely believed amongst 18th century physicians that humors could more easily be affected by medical treatments than solids. The former could be affected by both nutritional, chemical, and some therapeutical means (like blood-letting) whereas most of the latter were incurable, and only the symptoms could be ameliorated.

But I believe, that the disagreement, in the end, between the sides in this humoral-solidist dispute was not so much about what kind of physiological interactions the different constituents of the body might have between them, but about a way to classify them that privileged some as authentically capable of hereditary transmission, and left the rest of them in another category. The obscurities surrounding generation, and the impossibility to trace the details of physiological interactions forced the

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[89]Contrary to Pagès, both these authors accepted as hereditary a whole range of very different diseases, most of which adopted familial patterns. The idea to follow this transformations in time was taken from some ancient authors, and was retaken later on by several 19th century hereditarians and advocates of degeneration. See for this Pick (1989) and Dowbiggin (1991).

[90]Amoreux considers a list of 31 different kinds of diseases, and most of them he grant "heritability". Pujol mentions at some point the existence of an "échelle d'hérédité" of diseases, in which all known ailments can be accomodated according to their feasibility of being inherited.

decision of how to define the hereditary towards external evidence, which could not in the end tip the balance one way or the other, but only draw some limits or boundaries. It was as we have seen in the clarification of these, with regards to pathological evidence, where 18th century French physicians made their mark.

The challenge to the appeal to hidden and indirect causation, and latency of hereditary influences

Homochrony, we saw above, was the clearest of "external" criteria for the hereditary. But it was only one manifestation of the main characteristic that hereditary causes had been seen to possess: latency, or the ability to hide for some time in a healthy body without any sign or symptom. Atavism, of course, was the other important manifestation of this property linked to hereditary transmission. In their attempt to establish the category of "maladies vraiement héréditaires" most competitors for the Royal Society's prize realized they needed to give some attention to the special status of hereditary causation.

Louis had discarded as absurd any indirect or mute causation. Specially he did not conceive of a morbid cause (be it constitutional or humoral) that could remain quiet for an entire lifetime (or several) and only manifest itself at a future generation. The transmission of predispositions not of actual diseases, as he himself could see, was the answer. Derived, as we said above, from Aristotelian potencies, the idea of a latent cause was in need of a credible description that gave it some substance in order to transcend the hypothetical status. Most of them seemed confident that through a distinction between kinds of causes the problem could be solved. Fernel had already provided the basic frame: that a hereditary (predisposing) cause needed to be supplemented by a triggering, efficient cause, whose absence would leave the former one mute. What most competitors tried then to do was to flesh up the scheme based on their physiological biases. Materialist approaches to causation were favoured by most of them, both humoralists and solidists.[91]

Under a humoral account, latency is best explained by the permanence in the body of the tainted fluid (vice, levain) for an indefinite time without acting noxiously in any of its parts (this gives a sort of predisposition). It is only when at some stage of the development of the solid parts, that one (or several) of them becomes vulnerable to the ill action and that the disease develops. Other triggering causes can be external ones (emotional, physical, climatic, etc.)[92] According to Pujol, this explanation of latency was first forwarded by Gaubius.[93]

The solidist explanation of latency we have described earlier. An inherited conformational mark gives the predisposition, and a complementary cause (developmental or external) triggers the disease.

In any case, secondary, triggering causes were given a quite important role in the

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[91]Amoreux, for instance, stressed that an important thing about both humoral and solidist causes is that they avoid all plastic, immaterial efficient forces, archées, etc., which he considers "empty" notions:"cette substitution des mots —he wrote— n'a jamais donné une idée plus claire de la chose". op.cit., p.13.

[92]"Les causes accesoires decident plus promptement le principe des maladies à se developper...tel es l'abus ou l'erreur dans les choses dites non naturelles". Amoreux, op.cit.,p.11.

[93]Pujol,op.cit., p.326.

manifestation of the hereditary. They had however a non decisive role, as the variability of reaction within groups and families proved

> Tout étant egal (wrote Amoreux) les causes secondes agiront de même sur des sujets egalement disposés, elles agiront différement sur des sujets différement disposé, ce qui explique pour quoi dans une famille tous les enfants ne sont pas toujours atteints de la maladie de leur peres.[94]

Authors could shed doubts over the possibility that either morbid humors or constitutional defects could really remain hidden in a healthy individual for long periods of time. The idea of a predisposition itself, and its being hereditarily transmitted, could also be attacked from a high, a prioristic ground. But to most 18th century French medical men these ideas provided an excellent resource for picturing the most irregular and untamable of their empirical set of observations.

In this case too, the analogy with the normal helped reinforce their belief. Homochronic phenomena (like dentition and puberty) on the one hand, and latent hereditary transmissions (like family male boldness received from the mother) on the other, could respond to an analogous kind of latent causation as the one they were advocating.

The general challenge to the need of a special category for Hereditary Diseases

"Les Maladies Vraiment Héréditaires", as the French Royal Society of Medicine declared in 1791, a few months before its dissolution, was a category still in search of a precise definition. The stinging effect of Antoine Louis' arguments, together with the perceived stalemate in generation studies, had made it evident to the best prepared members of the French medical community, that if they were to preserve their cherished belief in hereditary transmission of all those mysterious diseases that seemed somehow to burst out spontaneously in some individuals within some families, they had to give ever more clear-cut characterizations of their main features, of their etiology and development. Chronic, constitutional diseases that once declared seemed impossible to eradicate, like gout, epilepsie, apoplexy, mania, or tuberculosis, were very difficult to account for except by a deep routed predisposition in the body. The category of a hereditary disease was one needed by the medical community, in part to account for its failure to prevent or cure these set of maladies.[95]

The discussion then had to be focused not so much on the reality of hereditary transmission but on the conformation of the theoretical space that was to describe them. To include more phenomena than those truly necessary, in other words, to have a category of hereditary disease so loosely defined as to have room for all diseases with some familiar pattern was absurd. Restrictive conditions based on the communication mechanism or route were needed, but the restrictions themselves had to be checked so that the category would not become impossible, as Louis had tried to argue it was.

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[94] Amoreux, op.cit., p.12.

[95] As Ackerknecht has rather sternly put it: "heredity has always been the facile explaining the inexplicable" (1965), p.63. For an excellent recent exposition of the role of predisposing causes in the medical debates of early 19th century see Hamlin (1992).

Pujol and Amoreux, the more enthusiastic defenders of wide range inclusion, saw the creation of some kind of gradient, or set of subclassifications based on kinds of causes and intensity of their effects, as the solution to the riddle. Amoreux proposed for instance four orders of truly hereditary diseases, although he did not want to clarify this, the first two are basically humoral, while the second two are solidist in cause.

1. Those that are transmitted specifically, without a change of nature.
2. Those (inespecific) that have an hereditary origin but change their nature.
3. Essential bodily dispositions.
4. Bodily deformities that are transmitted and are indelible.

Falsely hereditary diseases he considered all those products of accidental occurrences during pregnancy, non transmissible bodily defects, like “taches”, and all those opportunistic diseases that emerge from a weak constitution but that are not determined in any way.[96] This left still too much room for the stern Royal Society judges, and only the prudence of Rougemont and Pagès seems to have satisfied them here. Their strong restriction of the category of the hereditary diseases, only the most obviously constitutional and chronic ones, leaves however a residual problem of how to account for the widespread occurrence of familial patterns that are not easily accountable by external contagions. The discussion around transmission through nourishment pre- and post-partum (through mother’s blood and the nurse’s milk) falls into this unstable domain. Again, the lack of a clear physiological description precludes the closure of the debate.The assault on the hereditary by those wishing to account for all its target phenomena through external causation was still an open possibility, but the set of distinctions (congenital-connate) external observational criteria (homochrony) and causal analysis (latency) that most French physicians had agreed upon by the end of the 18th century seems to have given them a strong enough base for belief in a kind of independently describable system of transmission of physical peculiarities from parents to offspring that, in its de-pathologized way, could be synthetically referred to as Heredity.

[96]See Amoreux, op. cit., p.27.

Chapter 3

Of Taints and Crystals. British late 18th-century views of Hereditary Disease

3.1 Erasmus Darwin, a Prelude

> The clime unkind, or noxious food instills
> To embryon nerves hereditary ills;
> The feeble births acquired diseases chase
> Till death extinguish the degenerate race.
> (...)
> E'en where unmixed the breed, in sexual tribes
> Parental taints the nascent babe imbibes;
> Eternal war the Gout and Mania wage
> With fierce unchek'd hereditary rage;
> Sad Beauty's form foul Scrofula surrounds
> With bones distorted, and putrescent wounds;
> And, fell Consumption! thy unerring dart
> Wets its broad wing in Youth's reluctant heart.[1]

Rather contorted and cacophonous, these couplets from Erasmus Darwin's posthumous *The Temple of Nature* (1803) reflect the poet-physician's view of how hereditary diseases work as a negative trend against the general current of improvement and perfection on which life is embarked. The typical hereditary diseases, gout, mania, scrofula and consumption, which were among the most feared by Europeans of the time, are given in these verses a will of their own, as if their purpose were to invade and take root in the generational movement of the families, or nations in order to destroy them. This kind of fears were never too far behind in the minds of all the medical and natural historian attempts to understand the mysteries of resemblances: between parents and offspring, or between different members of the same families or groups, nations or races. The two main questions that made this maintenance of resemblances a crucial phenomena were then the worrying existence of family linked diseases, and the physical and "spiritual" differences perceived among the various human groups. Resemblances include thus both the normal and the

[1] E. Darwin, *The Temple of Nature*, 1803, Canto II,IV, 163-166, 177-184.

pathological, and keep both particular and general features through the generations within family lines.

The causes responsible for this persistence of peculiar resemblances were, since antiquity, attributed to different kinds of causes that can generally be divided between external and internal with regards to the original constitution of the body. A division that followed the same lines of the Hippocratic-Galenic distinction between the natural and the non-natural things. The natural things (causes) being those that had their roots in the original qualities of the body; what the medics called the constitution, or the temperament. The non-natural things were, on the other hand, those that acted upon the original body. Linked with the situations and events, both physical and emotional, that affected the body and could alter its health, like climate, nutrition, habits, emotions.

The relationship between the parents constitutions' and that of their offspring seemed undeniable to most observers. Obvious in its effects, its deep causes were however shrouded by the ignorance of the physiological details of reproduction. The substitute for this ignorance, as we have been showing in the previous chapters, was the metaphoric allusion to inheritance.

E. Darwin makes use of the power of the metaphor, stressing the negative, apocaliptic undertones it bore in his time. By its continuous association with the deviant, during most of the 18th century, the entire field of what I have been calling "the hereditary" was, on the whole, charged with negative connotations. It evoked pathology, deviations from the type, degeneration in the Buffonian sense.

As shown by the case of the 18th century French disputes we described above, such negative associations were mainly promoted by medical men. However, a movement towards turning around the meaning of "the hereditary", and giving it a set of positive connotations can be perceived in the writings of some late 18th century authors, and more clearly, during the first half of the 19th century. Although pathological inheritance never ceased to be a source of worry and speculation, and many of the best documented cases of hereditary transmission of character remained to be of illnesses or a tendency to them, a shift in emphasis ocurred, towards the creation of a unified field of inquiry with the laws of character transmission, both normal and pathological, as its main objective. The late 18th century attempts at explaining the origin of human varieties as the fixation of hereditary diseases that altered dramatically the complexion of non-white, non European races, provoked, I believe, such a reaction of de-pathologization of the field. This chapter and the following one are an attempt to charter these changes within the British milieu, and how they affected both the pathological and the normal approach to human hereditary transmission up to the 1850's.

In the fragment quoted above, E. Darwin was not only exploiting the metaphorical powers of the hereditary, but also its associations with the maintenance of variations within given genealogical lines; wether families, tribes, or nations. Fortunately, Darwin did not limit the exposition of his ideas to the poetical and metaphorical. He wrote for one of the additional notes to this long and tedious poem,[2] a short essay

[2]Although Erasmus Darwin (1731-1802) built himself a considerable reputation as a poet in his own time, it did not last long, and was demolished by critics and satyrists of the following generation, for his uninspiring and formalistic natural philosophical tirades, the most famous of which is of course his *Zoonomia*. Some authors maintain

explicating these few verses.[3] . "All the families —he opens it by saying— both of plants and animals appear in a state of perpetual improvement or degeneracy, it becomes a subject of importance to detect the causes of these mutations". Degeneracy (or negative variation) Darwin identifies with "hereditary disease". External influences ("the clime unkind or noxious food") disrupt the normal conformation of the plant or animal, and once in it, such disease tends to pass (through generation) to the subsequent generations using the internal, self-replicating habits that constitute "the hereditary". Symmetrically, we learn in other parts of Erasmus Darwin's oeuvre, positive variations are incorporated and subsist into the future transforming and perfecting the types of individuals, families, groups or species.

Leaving aside the importance of such transformism in Erasmus Darwin as an antecedent for his grandson's work, it is necessary to pay attention to his keen interest in the hereditary,[4] conceived as a conservative influence that affects both abnormal and normal constitutional characteristics.

Sex, according to E. Darwin, is the only remedy against the eventual destruction that degeneracies bring to any genealogical line once they have set root on it. Organisms without a sexual mode of reproduction are therefore more prone to hereditarily perpetuate and aggravate any induced flaw, and become extinct.[5]

It is, he writes, the

> greater similitude of the progeny to the parent in solitary reproduction [that] must certainly make them more liable to hereditary

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that he was nevertheless influential in the formation of greater poets, like Coleridge. See D. King-Hele (1968).

[3]"Hereditary Diseases", additional note XI, in the *Temple of Nature, or the Origin of Society*.

[4]It is important not to fall into the easy assumption that he, or any of his contemporaries had a view of Heredity as a mechanism or force of transmission of characters. I insist then in referring, somehow cumbersomely, to the set of phenomena as "the hereditary" because the adjectival use is closer to the epoch's conception. The same reason I believe has to be given to dey all the allegations about the importance of "the inheritance of acquired characters" for all these authors.

[5]His main example is horticultural:

> "Where no sex with glands nutritious feeds,
> Nursed in her womb the solitary breeds;
> No mother's care their early steps directs,
> (...)
> So grafted trees with shadowy summit rise,
> Spread their fair blossoms and perfume the skies;
> Till canker taints the vegetable blood,
> Mines round the bark, and feeds upon the wood.
> So years successive, from perennial roots
> The wire or bulb with lessen'd vigour shoots;
> Till curled leaves, or barren flowers betray
> A waning lineage, verging to decay;
> Or till, amended by connubial powers,
> Rise seedling progenies from sexual powers."

Temple ..., Canto IV, 157-159, 167-176.

> diseases; if such have been acquired by the parent from unfriendly climate or bad nourishment, or accidental injury[6]

The old idea that with a change of place and the passing of time degeneracy can be prevented does not persuade the farmers any more, Darwin writes, only the bettering of the seed does.[7] By favouring the fall of "the dust" of one kind into the flower of another "the new seeds or plants may be bettered, like the marriages of families into different families". The external influences, who have the power to provoke the degenerative variations do not seem capable of checking them. It is only through the balancing effects of alternative hereditary influences that this can be done. And the analogy is then possible, if "isolated" reproduction is noxious for asexual organisms, it has to be too for in-breeding groups:

> As the sexual progeny of vegetables are thus less liable to hereditary diseases than the solitary progenies; so it is reasonable to conclude, that the sexual progenies of animals may be less liable to hereditary diseases, if the marriages are into different families than into the same family; this has long been supposed to be true by those who breed animals for sale; since if the male and female be of different temperaments, as these are extremes of the animal system, they may counteract each other; and certainly where both parents are of families which are afflicted with the same hereditary disease, it is more likely to descend to their posterity.[8]

The use of the adjective "hereditary" did not carry in Darwin, nor in many of his contemporaries, a strong explanatory weight. It was basically a metaphoric, descriptive term, aimed at describing a pattern of disease communication, but not as a particular etiological type. Medical tradition had been using the descriptive formula "hereditary disease" for centuries,[9] but the adjective was rarely used in "positive" contexts within what we would call a biological frame. Other words were used usually to refer to the transmission through generation of neutral or advantageous characters (or variations), perhaps because hereditary carried, through its centuries of association with disease, a negative connotation.

"Hereditary" however had also strong "moral" connotations. Good or bad human qualities were said to run in families, groups, nations, and the metaphor of heredity was used in theological, ethical and social "programmatic" disputes with some regularity.[10] Despite Darwin's declared intention of giving in his work and notes an

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[6] *Temple* ..., Ad.note XI, p.43.

[7] See for this James Anderson's, 1799, "An enquiry into the nature of varieties". In this very illuminating analysis, Anderson insists that only by localizing and painstakingly reproducing and augmenting by selective breeding the desirable hereditary properties of domestic animals, can the objective of improving the breeds be obtained. This essay is very interesting also for the hierarchical classification Anderson makes of the different kinds of characters (or their variations) with respect to their transmissibility and their fixability within a breed.

[8] *Temple* ..., p.44

[9] As we saw in chapter 2, the dictionaries registered the medical use of "hereditary" at least two centuries before any "neutral" biological use.

[10] See for instance, "On the Hereditary Genius of Nations" in Reverend John Adam's book *Curious Thoughts on the History of Man*, 1789, London, pp.226-243., and W. Coggan's "Letters to William Wilberforce on the doctrine of Hereditary Depravity", London, 1799.

objective account of Nature's ways, it is evident from the tone (and subtitle) of the poem that it is humanity (the moral side of it) which really is at the front of his attention. The hereditary metaphor he uses fully charged with those associations; as in the passage above, in which Darwin qualifies "ills" and "rage". The poet (and prophet) in him eclipses the naturalist, as he exploits the metaphorical strength of the term. Plants and animals becoming a mere mirror for his "apocaliptic" concerns: Families (or genealogies) made peculiar by transmissible physical and moral characters, ill or good, irrupting into them from the outside by some kind or other of influence (curses or blessings) provide a powerful storyline that has been repeatedly used in traditional literature. Darwin, the physician and naturalist, as some before and many after him, informs his ethical narratives by drawing analogies from other organisms, plants and animals, wild or domesticated. But the facts about human hereditary disease, with its long tradition within Hippocratic-Galenic medicine provided for him the central point of reference for the metaphor. Even in this non-scientific work, the structure that a medic gave to the emerging concept of heredity determined the shape it would have in the other, non-pathological realms, and not the other way round. The idea of variation itself was highly "pathologized" in most medical minds. For instance, hereditary pathologies were seen as the source of variation between races within the human species. Degeneration, in its Buffonian sense, was associated to deviation from the healthy original stocks. The idea that these distortions of human (and animal) constitution could start by an accidental individual occurrences that somehow managed to root themselves into the constitution and use the generation process to be passed from parents to offspring both fascinated and mystified late 18th century thinkers. Especially for its consequence that whole genealogical lines (families, groups, nations, races) could be in the end marked (tainted) by such accidents. Disease and variation were thus never wholly separated in the mind of Hippocratic-Galenic medics, and the late 18th century saw a surge in speculation about their links. The analogical move, which had always been tempting was however made in the direction of disease; bringing variation to the camp of pathology rather than the other way round. Variation and constitutional disease were both degenerations (as Aristotle and Buffon had wanted) liable to become hereditary. E. Darwin was an example of this. Though having in his general scheme room for both improving and degenerative variations, he was more fascinated by the evil side of the hereditary.

The French physician and historian of medicine, Pierre-Joseph Amoreux (writing in the late 18th century) explained that two parallel developments seem to have been responsible for a surge in interest amongst medical men in hereditary transmission of disease after the 17th century. First the growth of urban populations in Europe, and the European explorations and discoveries of increasingly remote places . Through these events, he argues, European physicians were brought into contact with, on the one hand, a much wider variety of human races, and with their peculiar endemic (regional) maladies; and on the other hand, with a collection of constitutional ailments that different classes, racial groups, or families developed with higher frequency than others in urban concentrations. Hereditary transmission seems, at some point, to have become the most economical way of describing first, and of explaining later, the very complex patterns of occurrence of certain urban diseases.[11]

[11]This extended into the 19th century disputes around contagionism and anticontagionism, as C. Hamlin (1992) has recently shown.

Although France, as we saw, was certainly the birthplace of modern medical hereditarianism, Erasmus Darwin was not alone in the British Isles, at the turn of the century, in his concern for hereditary disease, and its ramifications. Not as prominent as in France, but there certainly was in 18th century Britain an equivalent discussion among medical men about hereditary transmission of disease. Separated into similar camps as in France (which we can loosely call humoralists and solidists) some of the most eminent physicians of both England and Scotland took one or the other side in the matter.

Some issues that (perhaps through the distorting windows of hindsight) we consider of an utmost relevance for the forging of our idea of biological inheritance, were being considered by them in the more limited area of transmission of disease. As in France, questions about the kinds of causation, routes of transmission, and explanatory reach of the hereditary were carefully and skillfully analysed by the few British medical men busied with pathological inheritance.

As we argued above, ever since Hippocrates and Aristotle, the facts of normal familial resemblance were used both to throw some analogical light over the familial pattern of abnormal events (deformities, disease) and to justify a belief in their transmission. The late 18th century brought both in France and Britain what could be described as a reversal of the analogy. The pathological side of the hereditary began to illuminate the non-pathological one, to transpose towards it its causal structure and evidential procedures. Such reversal is a fact that has not sufficiently been recognized. Its outcome was, as I will try to show, that early and middle 19th century physicians developed a more analytic and structured concept of hereditary transmission that was neutral with regards of the normality or abnormality of the characters transmitted. The same kind of argument could finally be made for all the empirical sets of facts that since ancient times were regarded as belonging to the hereditary. The increasing awareness amongst some thinkers that there were some underlying common causes that could in the end make sense of the very confusing and irregular set of facts, independently of what view of generation was upheld. The movement towards the elimination of the pejorative ring that its association with diseased conditions had given to the hereditary thus accelerated in the early years of the 19th century. In France, writers began for instance using “hérédité” as a general concept that had to be then qualified, according to the characters under consideration as “normal” and “pathologique”[12] . In Britain, which is the case I will describe in the following chapter, what I want to call a “de-pathologization” of heredity was finally achieved in the work of the Scottish physician James Cowles Prichard, and then detailed and popularized by William Lawrence. Several people have reasonably argued that the work of these two writers was a necessary step towards our modern view of heredity.[13] But the link between their approaches and contemporary discussions around hereditary disease, and the input that such link made to the way they thought about hereditary transmission, have not been analysed before.

Before doing it, I will in this chapter move back again a few years to describe the sources from where Prichard took his cues.

3.2 Solidists vs Humoralists; Hereditarians vs

[12]See chapter 1.

[13]On Prichard’s work see Odom (1970), Stocking (1973), Zirkle (1946); on Lawrence see K.D.Wells (1971).

Sceptics

The dispute between 18th century French medics, that I described in the previous chapter, was about the existence of a valid category of diseases under the head of "hereditary" and the boundaries it should have, if accepted. At the root of the dispute was the struggle between humoralism and solidism in pathology. If Humors dominated the scene, there existed plenty of room for external influences to affect dramatically the essential aspects of bodily functions. Fluids could come and go from the interior of the body to its exterior, could mix, circulate, be exchanged between individuals, cause different effects in different parts of the body or in different persons. The quality of the mother's blood, the nurse's milk, the food, the drinking liquids, even the air breathed, acquired a great importance for the individuals constitutional (or temperamental) state on which his or her health depended. Under this vein, Erasmus Darwin wrote that

> The hereditary diseases of this country have many of them been the consequence of drinking much fermented or spirituous liquor; as the gout always, most kinds of dropsy, and, I believe, epilepsy and insanity. But another material, which is liable to produce diseases in its immoderate use, I believe to be common salt, the sea-scurvy is evidently caused by it in long voyages; and I suspect the scrofula, and consumption to arise in the young progeny from the debility of the lymphatic and venous absorption produced in the parent by this in nutritious fossile.[14]

Such susceptibility of temperaments (or constitutions) to aggressive external materials, paired with the idea of a strong direct (hereditary) influence of parents' temperaments in those of their offspring leads then to Darwin's picture of unstable (progressive or regressive) genealogical lines in which potentially hereditary variations inflicted by any sort of cause are the rule rather than the exception. It is important that under this view the transmission of particular diseases from parents to offspring is not essentially different from that of other (externally transmitted) diseases. It is only the route that those influences take from one individual to the next one (through generation) that makes them peculiar, and that generates the familial pattern they are found under.[15]

Humoralists agreed with solidists that what physicians called the "general frame" of the offspring's body was undoubtedly influenced, in the offspring, by the parents' in what we could describe as an unlocalized fashion. They preferred to refer to this as the influence of parent's temperament in the child's, emphasizing the old idea of an ultimate dependence of everything on the balance between the four fundamental humors. In the view of some of them, besides the particular inheritance through generation of some taint or other, the communication of a weak or unbalanced general temperament gave an hereditary, but unspecific propensity to disease. As we have seen in the French disputes (chapter 2) such proteism and unspecificity of the action displeased many authors, who considered idle (or damaging for the cause) to include this vague and general notion in the restricted domain of the hereditary diseases.

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[14] *Temple* ..., p.44

[15] The fact for instance that some humors, or fluids can find their way more easily into the seminal liquid, and/or the germ is, under this view, what makes some diseases more hereditary than others.

A very similar situation can be found amongst solidists, some of which also saw a communication of a "general frame" (which on the whole they preferred to call constitution) as a base for unspecific tendency to disease. Many however chose to particularize more the constitutional defects to bodily systems (lymphatic for scrofula, nervous for insanity, etc.) or even organs.

In the struggle to isolate and define a clear-cut category for the hereditary in relation to disease, the claim for a specific, independent and strong kind of causation was blocked by the fact that very few of the diseases with a familial pattern (which most physicians agreed should count as hereditary) manifested themselves at birth or soon after. All opponents of the hereditary explanation mentioned this. The period between birth and the occurrence of the first symptoms allowed for the intervention of many possible factors.

Defenders of the hereditary, humoralists and solidists, were made continuously aware by their foes of the difficulties of claiming the direct transmission of a disease through generation. Many newborn babies of diseased parents, for instance, are completely healthy and only develop the disease at a later period in life, usually at a fixed one. As many before him, to tackle this possible criticism, E. Darwin adopted the stance that Fernel took when faced with this question:

> A certain tendency to these diseases is certainly hereditary, though perhaps not the diseases themselves.[16]

Other authors had used, as we saw, "predisposition" instead of "tendency". In contrast to some of them, the causal subtleties that this clarification implied seem not to have worried E. Darwin much. This was due, I believe, because for him the maleability of bodily parts was open to infinite kinds of influences.[17] But for those inclined towards an austere solidist pathology, with their more restricted view of the body's capacity for change, there seems to have been a more acute need of separating with some clarity structural predispositions (diathesis)[18] from external accessory causes. For them the hereditary element in a disease, when it existed, had to be of the former type, as it depended on a constitutional flaw of organ or tissue. A defect that was incorporated into the structure of the body at the moment of its first formation. After the "solidification" of the individual's constitution, most solidists believed, no other kind of influence could make such fundamental alterations. External influences had a secondary, accidental role, and were not easily incorporated into the basic,

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[16]op.cit., p.45.

[17]He proceeded, throwing a couple of lines that was to be picked up later by his other famous grandson Francis Galton, to advocate a careful selection of mating partners ("the most beautiful in respect to the body and the most ingenious in respect to the mind") and warns of the hazards of marrying a heiress ("As many families become gradually extinct by hereditary diseases, as by scrofula, consumption, epilepsy, mania") as she is not infrequently the last of a diseased family. Op.cit., p.45.

[18]Diathesis: an old greek word that was defined by Stephen Blancard (1684) as "the natural or praeternatural disposition of the body whereby we are inclined to perform all Natural Actions, ill or well." In modern times it was revived by physicians during the 19th century as a synonym of constitutional predisposition to a particular disease, and was associated with hereditarian explanations. See Olby ("Constitutional and Hereditary Disorders", in print), Ackerknecht (1982), and Hamlin (1992). See also the definition of "diathèse" in *Dictionnaire des Sciences Médicales*, vol.9, by Pariset and Villeneuve, commented below in chapter 5.

hereditary structure of individuals, families or groups.

3.2.1 William Cullen

When writing about gout, one of the most notorious constitutional diseases, William Cullen, the famous Scottish physician, criticized the humoralist idea that there was a "morbific substance" responsible for its transmission.

> most hereditary diseases —he wrote— do not depend upon any morbific matter, but upon a particular conformation of the structure of the body, transmitted from the parent to the offspring[19]

Cullen added that gout was "disease of the whole system [that] depends upon a certain general conformation and state of the body" instead of a particular localized one. In line with his "chemical philosophy" physiological approach,[20] Cullen described this general system as depending on the body's "primary moving powers" intimately linked to the nervous system. So Cullen was following his own agenda.[21]

Odom has argued that Cullen's view on hereditary transmission was a radical break with previous approaches, but that is clearly an exaggeration.[22] For instance, Cullen's conception of the hereditary as affecting only constitutional characters, and his defense of this idea based on the predisposing (latent) causality, were not an invention of his, as Odom has argued; the description I gave above of the French contemporary disputes, I think, show that Cullen had a clearer sense, perhaps, of the importance of individual constitutional idiosyncracies, but he did not distance himself that much from his contemporaries. He shared with them, in any case, a "general frame" view of constitution, and was far from defending a localized, particulate version of it. The view of him as a pioneer who produced "the singlest greatest theoretical clarification of the operations of heredity" seems fanciful, to say the least (see footnote 38). Cullen was very close to other 18th century authors. This can be seen for instance in his treatment of other hereditary diseases, besides gout, for which he accepted a variety of causal influences, including humoral and contagious ones.[23] His view of the strength of the disposition to disease has also to be mentioned ("many healthy persons have the hereditary right and disposition to gout but healthy life avoids it") as it brings him closer to authors that radically opposed giving hereditary explanations to gout or other constitutional (chronic) diseases. Specially to those of the most lucid amongst British opponents to hereditary explanations of constitutional disease: William Cadogan.[24]

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[19]See vol.2, p.121. of W.Cullen,(1784), *The Works of W. C.*, J.Thomson ed., 2 vols., Edinburgh, 1827, W. Blackwood.

[20]A description of Cullen's chemical philosophy can be found in L. S. King (1958).

[21]All this is perfectly in line with what Cullen considered to be the way to explain diseases, based on what he called his "chemical philosophy". Medical intervention, in order to restore the "primary moving powers" to their normal "general" state was possible, and so the hereditary taint could be eliminated from a lineage.

[22]H. Odom (1970) *Groundwork for Darwin. Theories of Heredity and Variation. Great Britain 1790-1820.*

[23]"Hereditary diseases depending upon morbific matter always appear much more early in life". op.cit. p.121.

[24]W. Cadogan (1711-1797), Fellow of the College of Physicians, Director of the Hospital of Foundlings.

3.2.2 William Cadogan

In 1771, some years before Cullen's work on gout, when writing about that same disease, and other chronic diseases, Cadogan opposed in a vehement and articulate style the hereditary explanation. He restated the old argument of the inadequacy of the metaphor of heredity due to indirectness and insufficiency of the causation and the indeterminism of the transmission:

> If [gout] were hereditary, it would be necessarily transmitted from father to son, and no man whose father had it could possibly be free from it: but this is not the case, there are many instances to the contrary; it is therefore not necessarily so; but the father's having it inclines or disposes the son to it. This is the causa proegumena or predisponent of the learned, which itself never produced any effect at all; there must be joined the causa procatarctica, or active efficient cause, that is our own intemperance or mistaken habit of life[25]

To call this predisposition hereditary is a misnomer, Cadogan thought, because it implies a necessity (a fatality) that they do not have. Constitution is not destiny. The power of external factors can override innate tendencies:

> Our parents undoubtedly give us constitutions similar to their own, and, if we live in the same manner as they did, we shall very probably be troubled with the same diseases; but this by no means prove them hereditary: it is what we do ourselves that will either bring them on or keep us free.[26]

No constitutional diseases are hereditary. Only local, focused, acute diseases are, Cadogan thought. Those of "taint, or infection or malformation". Periodicity and latency are also criticized by Cadogan as criteria that establish the hereditary nature of some diseases, as the reality of these phenomena is very difficult to demonstrate, and which usually have alternative explanations.

Cadogan shares with Cullen the view that "constitutional" only refers to the whole body and its systems. What separates them is not a different view of what has to happen for some disease to be hereditary, but a different stress in the importance of predisposing causes. And at the root of this difference is the deep disagreement over the degree of maleabilty (and of dependence) of the bodily structure to external influences. Either at birth (or better still: at the moment of conception) an individual organism's physical and mental qualities (present and future) are pretty much constricted by the constitutional features that conform it, or the environment (or the human will) has the possibility of shaping it out of its non deterministic innate constrictions.

Cadogan, like E. Darwin, does not see in hereditary causes anything that needs special physiological description. The superficiality that he attributes to initial individual constitution shows that he believes in a basic similar essential frame for all humans over which only light (ill or good) modifications can be made. Most of them could be cured or controled. In contrast, for Darwin and Cullen individual variations are not so superficial. They are capable of becoming established within a family or a group by repeated transmissions. Their transmissibility can be of lasting

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[25] W. Cadogan, 1771, *A Dissertation on the Gout and all Chronic Diseases.*
[26] op.cit.,p.8.

consequences for the whole genealogical line.[27] For them, only by counteracting events during reproduction can a constitutional modification be displaced from within a family line (therefore the need for sex, according to Darwin).

As we said, most late 18th century physicians came to conclude, (perhaps tired of the unending disputes around preformationism) that the act of generation, whatever the actual detailed system through which it was accomplished, had to produce as its outcome (after conception) a well organized and individualized embryo (first stamen, in the old language) in which hereditary elements of both parents could (or should) be mixed in some proportion or other. This was imposed by the evidence, they claimed, and not a product of theoretical prejudices.

Undeniable as it seemed to most of them, the tendency to repeat in the offspring whatever peculiarities had crept into the parents' constitution was not seen as applying with the same intensity to all characters. Although the essential characters of the species, and also the general constitutional (or temperamental) combinations that gave their identity to races, nations and family groups, were seen as generally re-produced in some way or other in the offspring (except in "sports" or anomalous variations), the peculiar idiosyncratic characters of individuals were not believed to be equally liable to pass on to the next generations. Criteria for distinguishing between them were however not easy to produce. Amongst French medics, as we have seen, the difference between what was congenital (incorporated through the seminal fluids or in the germ) and what was connate or acquired, became progressively relevant as the disputes around hereditary transmission developed.

It is at this point where it becomes increasingly important if an author is using a humoral or a solidist physiological (and pathological) approach. For the solidist, the causal factors present at the first formation of the individual are given a somehow privileged status with respect to those that act at a posterior moment, because they are crucial for the fundamental set of tendencies that the individual will be born with.[28] For him, the environment, so to speak, will not find all individuals equally predisposed nor equally maleable to its influences. The "solidity" of the individual's constitution, achieved before birth, pushes all other influences to a secondary role, but does not, as will happen at a later date, shut the door completely. For authors like Cullen, although himself not so much a solidist but a "constitutionalist",[29] important "acquired" characteristics can still find their way to the congenital set, but only under special circumstances.

In contrast, for Erasmus Darwin, a somehow over-enthusiastic humoralist, there was no limit to the modifications that could be incorporated into (or washed away from) the genealogical line. The moment and form of the introduction of a variation was not crucial.

The British 18th century author who seems to have made the strongest and more clear-cut distinction between the congenital and the acquired, both in diseases and in

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[27]An important difference is that E. Darwin is open to the modification of many different kinds of characters, and does not limit them to the bounds of the species.

[28]Whatever view of how sexual reproduction (generation) took place, for most medical men it was obvious that important hereditary contributions were made by both parents.

[29]He did not see the solid structure of systems and organs the fundamental physiological causes, but in their chemical interaction with the energetic, nervous system.

non-pathological variation, in order to sift the hereditary from the non-hereditary, was John Hunter. He apparently adopted a dual seminal and solidistic view of conception similar to the one Maupertuis held, and that allowed him to produce a clearer picture of how original constitutional variations are the only hereditary ones. His influence on posterior authors, like Adams and Prichard, was, I believe, crucial in making, during the 19th century, the British approach to the hereditary original and fruitful. I will therefore comment in some depth his views.

3.3 John Hunter, predisposition and hereditary disease

John Hunter (1728-93), the famous Scottish surgeon, is reputed to have read very few books during his eminently practical apprenticeship, and to have ultimately benefited from that, because he could keep his theorizing much closer to the facts than most of his contemporaries.[30] A piece of literature I believe he must have read, or known indirectly about, is Maupertuis' *Vénus Physique*, first published in 1744 as there are several striking similarities between the French author's view of the hereditary, and the origin and nature of individual variation, and the not so well known position that Hunter maintained on those issues. Although he could have taken them from other sources, the crystallization analogical model of solid organization of the embryo, and the "frozen accident" view of variation that it entails, both found in Hunter's work, were most thoroughly defended in the 18th century by Maupertuis.

Although not usually recognized as one of Hunter's many contributions that sparked future nineteenth century developments in different biological disciplines, his reasonings and positions on the issue of hereditary transmission can be said to have been quite influential. If one is to believe his pupil and follower Joseph Adams (1756-1818),[31] Hunter spread among the listeners of his lectures and private conversations a very clear-cut and analytic view on the origin and transmission of the so called hereditary diseases. Hunter made the same distinction as other authors between the constitutional alterations (that gave a predisposition to diseases) which could be transmitted to offspring and the non-constitutional ones, which could not be transmitted in such a way.

According to Adams (whose historical knowledge of the issue was, to say the least, limited) the previously prevailing and erroneous view among medical men was one that branded hereditary all diseases with a familial pattern, and that did not make further causal distinctions. This was, he maintains, the target of Hunter's analysis. He aimed at limiting the loose talk among physicians about transmission of disease from parents to offspring and restrict the use of the adjective hereditary to deep, organizational causes. Hunter's famous work on the transmission of syphilis from mother to child at birth[32] showed for him how other physicians erred when calling

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[30]His very irregular —unintelligible! — writing style, and his ignorance of the classics was constant ammunition for his enemies, according to Adams, Oppenheimer, and other biographers. S.J.Cross has however argued that Hunter "cultivated his un-bookish image deliberately". See footnote 23 in "John Hunter, the Animal Oeconomy..." 1981, *Studies in History of Biology*, p.81.

[31]Most are to references to Adams', 1814, *A treatise on the supposed hereditary properties of diseases*, J.Callow, London.

[32]See his Observations...(1786).

that infection hereditary, as it occurs in not a dissimilar fashion as other contagions, and at a time when the individual's body is already formed. No disease is transmitted as the body is first formed, only tendencies to certain diseases are, through peculiar, deviant, conformations, Hunter believed. And here it is important to stress that not only a "general frame" idea was behind his use of the adjective constitutional, but an idea that had room for localized variation.[33]

Herbert Odom reports not having found evidence for the existence of such views in Hunter's writings, and is rather sceptical about their importance.[34] Adams however gives more than enough evidence that Hunter did give to many audiences clear "hints for others to prosecute Inquiry" on this subject. For instance, an enemy of Hunter's, Dr. Crichton, is quoted by Adams as having castigated him (not altogether unreasonably) for not acknowledging his many precursors:

> That people were disposed to certain diseases from birth, as well as from the operation of accidental causes, was an observation or a mere matter of fact, which was taken notice of by the Greek physicians ... ; but a certain inaccuracy of expression, in regard to predisposition, has introduced itself into the writings of many medical men since the early times, and has induced them to call certain diseases hereditary diseases. This inaccuracy has probably been caused by the constancy with which hereditary disposition operates; but it gave occasion to the late Mr. J. Hunter to ridicule the expression, and to assume the old observations of the Greeks as one of his own discoveries.[35]

"There is no such thing as an hereditary disease though a disposition to disease might be hereditary" was a phrase of John Hunter's that seems to have circulated as a dictum.[36] It actually was pronounced by him in 1781 on the witness box of a murder trial. Sir Theodosius Boughton had collapsed after supper and Captain John Donellan was being accused of having poisoned him. The symptoms of the attack were consistent with the poisoning hypothesis and most other experts called into the witness box were certain that the murder had occurred. Hunter however was not to be made to accept such a conclusion so easily, under cross-examination he embarked in a series of punctillious discriminations, under the assumption that other set of causes could also fit the evidence; hereditary predisposition for apoplexy, for instance.

> *Question:* Where a father has died of apoplexy, is not that understood, in same measure, to be constitutional?
>
> *Hunter:* There is no disease whatever, that becomes constitutional, but what can be given to a child. There is no disease which is acquired that can be given to a child; but whatever is constitutional in the father has the power of giving that to the children; by which means it becomes hereditary. There is no such thing as an hereditary disease, but there is an hereditary disposition for a disease.

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[33]Stephen Cross (1981) has admirably described Hunter's concept of latent "disposition" to disease, as hierarchically structured between general (of the whole constitution), and increasingly particular ones (of systems and organs). See op.cit.,p.49, and footnote 158, p.100.

[34]H. Odom, op.cit., p.124.

[35]Quoted by Adams in, 1817, *Memoirs of the Life and Doctrines of the late John Hunter*, London, Callow, p.60.

[36]See *London Medical Review*, 1808, 211.

Question: Do you call apoplexy constitutional?

Hunter: We see most diseases are constitutional: the small pox is constitutional, though it requires an immediate cause to produce the effects. The venereal disease is hereditary. I conceive apoplexy as much constitutional as any disease whatever.[37]

Without further information, not much can be made out of these obscure declarations concerning Hunter's views on hereditary disease. (No wonder the jury found Hunter's arguments confusing and hanged poor old John Donellan). Adams was adamant that Hunter did have a well thought and developed view of the hereditary that he himself used as a base for his conception. The quotation above does not capture it. Adams, on the other hand, didn't apparently convince many people about the issue, and almost no other author has touched the point of Hunter's view on heredity in general, and of hereditary disease, in particular.

Odom, as I said, hastily[38] dismissed Hunter's importance on this area and decided, erroneously I believe, to privilege the contributions of William Cullen[39] and

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[37]Adams, 1917, p.243-44. See also Forbes T.R., 1980, "John Hunter as an expert witness".

[38]H. Odom, op.cit., p.110

[39]Odom wants to argue that Cullen elaborated an original concept around the word Constitution, which made possible subsequent hereditary inferences. But as we have seen his concept of individual constitution did not differ from other 18th century physicians in France in a general sense, only in the particular embodiment he was giving to it due to his own chemical philosophy. Constitution is for instance, the word that carries all the explanatory burden in the wording of Hunter's declaration above. Given his peculiar physiological (vitalist) elaborations, as Cross has ably shown, few of Hunter's contemporaries understood constitution in exactly this same sense, but again its general aspects are quite recognizable as typical 18th century, and not a visionary view.

Although there is a ring of truth to it, it does seem exaggerated to state, as Odom does, that the inheritance of individual constitution was such a revolutionary idea, and that it was developed by late eighteenth century Scottish physicians. His anachronistic analysis, (very influenced by Ernst Mayr's idea of a transition from "essentialism" to "population" thinking) shows why he is so keen:"What had been lacking in previous treatments was the idea of individual constitution or genotype. But that concept had become quite common in British medical literature by the year 1800...the innovation of the position defined in Britain was an emphasis on the heritability of the constitution, as an explanation of family differences, or idiosyncracies, in proneness to certain diseases, or response to particular medication. This conceptualization was a natural model for heredity in general (p.110)". No doubt this points towards an important link between the analysis of hereditary transmission of diseases and later general theories of heredity, but it lacks historical subtleness. This can be seen not only in the bizarre equation between late eighteenth century "constitution" and our modern idea of genotype, but also, crucially in the way he misrepresents the views of his candidate for the role of innovator. Odom writes:"In discussing hereditary diseases, Cullen identified the predisposing cause with the inherited constitution. Thus the exciting cause could provoke a disease only in those persons who had inherited a predisposition to it. The importance of this distinction was clearly that a clear line was drawn between genetic causes and environmental ones. In the case of

another Scottish physician, James Gregory. Odom's view of these two authors as the initiators of a silent revolution in the field of heredity (via a re-conceptualization of hereditary disease) is, as we said, exaggerated and distorts the facts. It is possible to show that it was John Hunter, following perhaps cues given by Maupertuis, who contributed more to the clarification of the field. This can be done by reading Hunter's declarations quoted above under the light of his broader views on hereditary variation, and monstrosities.

The paradox facing any 18th century physician who wanted to separate the causes of hereditary factors from those of external influences in familial diseases, was that no independent causal route could be imagined that could not at the same time be a route taken by external (climatic or other) sources.

Conception, as described at the time by opposite generation theorists (preformationists and successionists) was either an event in which the fluids contained in the male semen activated the pre-formed solid parts of the ovum into growth and development, its nutrition being provided by the mother's fluids (Haller, Bonnet); or it was an event in which the different elements that were to conform the first rudiment, or embryo, were selected and filtered out of the male and-or female fluids and put into the seminal liquid(s), which were then mixed and organized into the differentiated parts, solid and liquid, by some force or other (Maupertuis, Buffon, Needham, Wolff). What was difficult to conceive, in any case, was solid to solid direct causal influence flowing from the parents towards the offspring, one that was

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gout, for example, Cullen recognized the fact that, if excesses bring on the disease, it is a consequence of heredity. Many of his contemporaries believe the reverse to be true: that it was the excesses which produced the hereditary curse (p.144)"

But, besides the anachronisms, other things are misread here. Odom is attributing to Cullen views that were held later, by Joseph Adams and very probably as we saw, by John Hunter. Cullen, in the work Odom analyses, never discusses hereditary diseases in general. He did not even consider hereditary transmission to be a major nosological category when he devised his very influential taxonomy of diseases *Nosologia Medica*, and in the remaining of his *Works* Cullen did not make nearly as much as Odom suggests of the hereditary character of other traditionally familial illnesses, like scrofula or syphilis. He decided to emphasize gout a a constitutional disease because he wanted to contrast it with rheumatism, which he considered a completely externally caused disease, but more importantly because he considered gout to be a disease of the whole "frame" of the body, which in turn depended on the state of the nervous system. It is this wholeness, indivisibility of constitution which makes Cullen's view difficult to reconcile with Odom's claims. Contrary to what the latter affirms, it is not particularities of constitution that are considered by Cullen to be transmitted but a "general conformation or state". He does not side with those that saw the hereditary as a capable of a particularization, as well as an individualization of the constitution, but instead keeps close to the view that held by many eighteenth century physicians. Among them A. Portal, in France, who, in Odom's phrasing, thought that "hereditary diseases were only an accidental perturbation of the generalized human constitution and that nothing peculiar to the individuals permanent hereditary make up was involved" (Odom, op.cit. p.117. See A. Portal "Considerations of the Nature and treatment of some hereditary or family diseases" *London Medical & Physical Journal*, 21, Dec 1808 - June 1809, pp. 229-239, 281-296)

not mediated by humors, or by a constructive, supra-physical force. The claim that a particular variation in structure of a solid part that gives, for instance, a propensity to develop a gall stone at a certain age can be transmitted from parent to child by any of these ways seems difficult to maintain; specially if the claim is to an exclusive, one to one, part to part, causal relationship. How could a force "remember" the details in such a way, or how could a tainted humor from the semen be targetted as to not affect other solid parts of the pre-formed embryo. This questions gave pasture for many ruminations.

The French surgeon Antoine Louis,[40] as we detailed in the previous chapter, argued that there is no conceivable mechanism, that could account for a direct communication of diseases from parents to children. This argument was mainly based on a preformationist conception of generation, which, as E. Lomax recently wrote, "was difficult to reconcile with the hereditary transmission of disease" because in it "the germ cells, presumably perfect when created, were isolated within each other and so beyond the reach of vices located in the solid and fluids of the body".[41]

But Louis and all other opponents of hereditary transmission were at the same time open to the strong criticism of being blind, or stubborn in front of the powerful array of external evidence for such transmission. The accumulating number of instances of ocurrence of the same diseases (gout, insanity, scrofula, etc.) adopting familial patterns, in different climatic, social, and other environmental circumstances, pointed insistently towards an internal cause. Hereditary transmission, as a descriptive metaphor in search of causal substance, had to be kept alive as an alternative view for a specific subset of maladies with a familial pattern. As shall be shown below, one basic strategy for doing that was to "de-pathologize" the mechanism of transmission in such a way as to strengthen the relation between normal resemblance within families and other genealogical groups, and abnormal, pathological and monstrous, resemblance. The constitution of a unified domain of the hereditary, with unified causation and route of transmission, was then an interest that was shared by pathologists, and those writers in other fields worried with the origin and maintenance of variation within genealogical groups, be it "physical historians of humankind" or breeders of domesticated animals.

The "frozen accident" model, based on the analogy from crystallization, provided an image of how variations could come to be established within a genealogical line without a multifarous and uncontrollable set of causes (external and internal) acting simultaneously. The causes acting at the moment of the first formation of the new organism could be privileged over other influences.

3.3.1 Crystals, monsters and variations

John Hunter, like many other 18th century surgeons, believed that the idea of structural and hereditary family resemblances could easily be extended to internal organization by careful observation, specially of post-mortem dissection. As we said, several French authors, including Portal, emphasized this point. The experience of finding internal peculiar similitudes between relatives during autopsies seems to have been common for Hunter. Here is an example:

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[40] Antoine Louis (1749).

[41] E.Lomax,1977, "Hereditary or acquired disease? Early nineteenth century debates on the cause of infantile scrofula and tuberculosis".

> Sir C.C. had but one testicle that had come out of the abdomen, which was on the left side...his son died...I opened him, and curiosity led me to examine the scrotum, and I found but one testicle there; it was of the right side: the other testicle was in the ring. Was this similarity to the father accidental, or was it hereditary? [42]

Many could easily have formulated the same question, but as Adams correctly argued[43] the Hunterian approach to hereditary transmission allowed for a more subtle understanding of its meaning. The distinction Hunter made between hereditary and accidental characters stressed the existence of original causes acting at the period of the first formation of the individual. He was convinced that one could observe in nature different kinds of variations

> in the individuals of each species varieties are every day produced in colour, shape, size and disposition. Some of these changes are permanent with respect to propagation of the animal, becoming so far a part of its nature as to be continued in the offspring.[44]

A variation in a character (or in a disposition) became hereditary, for Hunter, if it originated in an individual organism at the moment of its first constitution, when the elements that were to form it were getting together, in what I have called a "frozen accident", that became permanent and transmissible in the family line. This kind of alterations is properly called constitutional, and can be of a general or a particular nature.

Hunter did not relate this source of variation to a particular physiological mechanism, and was not certain about the exact moment when such kind of alteration could occur[45] He however used monsters as extreme cases of hereditary variation, and a very striking analogy (crystallization) to make his sense clear. Both elements which he probably took from Maupertuis, but could have taken from another author.

Some variations "are so extraordinary, as with propriety to be denominated as monstrous".[46] These are not different from less dramatic variations, some of which are related to hereditary diseases.[47]

On a special section on "Monsters" in his *Essays and Observations* Hunter wrote:

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[42]John Hunter (1861), *Essays and Observations*, p.247

[43]Adams took Portal's view of hereditary disease as a target in his Treatise..., he found the French author lacking "in that cautious rejection of undefined terms, which distinguishes the true disciple of the Hunterian School", op.cit. p.84.

[44]J. Hunter, "On the colour of pigmentation of the eye in different animals" in *Observations* ..., 1886, p.199.

[45]He wrote "whether this takes at the very first union of the principles of the two parents...or whether it takes its formation from the mother, after the first formation of the embryo, is perhaps not easily determined" idem.,p.200.

[46]*Observations* ...,p.201. Not all extreme variations however were justly called monsters, Hunter believed, because "it is neither necessary nor does it follow that all deviations from the original must be falling off; it appears just the contrary, therefore we may suppose that Nature is improving its works; or at least, has established the principle of improvement in the body as well as in the mind", a footnote in same page...

[47]"Is it more remarkable —wrote Adams— that a diseased disposition should be perpetuated than an actual monstrosity? " op.cit. p.22.

> we call everything that deviates from ...uniformity a "monster", whether [it occur in] crystallization, vegetation, or animalization. There must be some principle for those deviations from the regular course of Nature.[48]
>
> Monsters are not peculiar to animals: they are less so in them, perhaps, than in any species of matter. The vegetable [kingdom] abounds with monsters; and perhaps the uncommon formation of many crystals may be brought within the same species of production, and accounted for upon the same principle, viz. some influence interfering with the established law of regular formation.[49]

But the uniformity with which very peculiar hereditary monstrosities (or diseases, for that matter) reappear in each type of animal, plant or crystal, forbids the thought that this latter influence could be "a matter of mere chance" nor could they be related to environmental circumstances. As Adrian Desmond has remarked, this led Hunter to reason "that the cause must therefore lie in the 'original germ' and that monsters are formed monsters from their very first formation".[50] But this was not a must, and a further distinction could be made:

> Whether the principle of monstrosity be coeval with the first arrangement, or arise in the progress of expansion, is not easily determined in many [instances of monstrosity]; but it is certainly not the case in all; for many take place at a late period, and would seem to be owing to accident, or to some immediate impression; but still there must be a susceptibility for such, which susceptibility must be original.[51]

Crystallization gave him a way to visualize this dual origin of variation, their formation can deviate itself due to "...either a wrong arrangement of the parts of which the crystal is to be composed, or a defect in the formation, from the first setting out being wrong, and [the formation] going on in the same [wrong] line".[52]

On chapter seventeen of his *Vénus Physique*, Maupertuis proposed his "Hypotheses on the Formation of the Fetus". He sets his view against Harvey's by suggesting "a better analogy than what takes place in the brain".[53]

> When silver, spirits of niter, mercury and water are mixed together, the various particles of these substances arrange themselves in a pattern so similar to a tree, that the result is called *Arbor Dianae* [arborescent silver]. Since the discovery of this admirable "vegetation" many others have been found...Although these seem less highly organized than the bodies of most animals, might they not depend on the same mechanisms

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[48]J. Hunter, *Essays and Observations*, p.239.
[49]idem.p.240
[50]Adrian Desmond,(1989) *The Politics of Evolution*, p.348-49. See also J.Hunter (1840)*Observations* ..., 2nd. edition, with notes by Richard Owen, p.25-26. For general historical view of the relations of monsters to generation and heredity theories, see Jean-Louis Fischer, 1986, *De la genèse fabuleuse...des monstres.*
[51]J.Hunter, Essays...,p.240.
[52]idem.,p.240-241
[53]Harvey had suggested that the formation of the foetus could be driven by an external shaping force in a similar way as the spirit shapes the contents of thought in the brain.

and on similar laws? [54]

Maupertuis explores the analogy proposing the existence of a cohesive force that link particular particles, contributed by both the male and the female and destined to form the different parts of a body. Like in the crystals he bases his analogy on, imperfections can occur.

> If each particle is united with those meant to be its neighbors, and to no others, the child is born perfect. If, on the contrary, some parts find themselves too far or of unsuitable shapes or too weak for close union with the precise particles, then *a monster by default* is born. But if some superfluous parts still find available place for union in spite of the fact that the acceptable number is already complete, then there is *a monster by excess.*[55]

Maupertuis was writing in a time when preformationism was dominant. The main thrust of his book was to provide such view a definitive blow. "Monsters by excess" had been interpreted by preformationists as being the product of accidental unions of two eggs or two embryos. Maupertuis could carry extra weight for his account by pointing to

> the fact that the superfluous parts are always found in the same area as the normal parts. If a monster has two heads, they are both set on the same neck...[in] men born with extra fingers ...these are always attached either to hands or feet...

If the preformationists were right, he argued,

> how could [the] union occur in such a way as to insure that the remaining parts of the injured [embryo] would become linked to the normal parts of the embryo which had suffered no injury? [56]

The crystallization analogy gave a straightforward answer. And it could in addition account for the fact that some of these deformities became hereditary. Polydactyly was specifically used by Maupertuis in his probabilistic argument for biparental inheritance. An accident at the crucial moment of the first formation could alter the basic constitution of an individual, adding an extra digit. This could be passed on to his offspring until a contrary accident at a similar moment would displace it. John Hunter, several decades later,[57] was thinking along the same lines

> That [the principle of monstrosity] is as early as the first formation, appears from the supernumerary part being almost always placed with the natural or corresponding one; viz. two heads are always on the shoulders...a supernumerary finger or toe is on the hand and foot;[58]

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[54]P.L.M. de Maupertuis, 1966, *The Earthly Venus*, translated from *Vénus Physique* (1745) by S.B.Boas, *The Sources of Science, 29.*

[55]idem.,p.57

[56]Ibidem.

[57]Richard Owen gives some time after 1790 as the probable date of Hunter's reflexions on monters and crystals, as evidence he refers to *phil. trans.* vol.lxxx, 1790, p.296, where the descriptions of some of the monstrosities he describes can be found.

[58]J. Hunter, Essays..., p.243. He later wrote:"I should imagine that monsters were formed monsters at the very first formation for this reason, that all supernumerary parts are joined to their similar parts; for example, a head to a head, &c."

George and Simone Boas point out the fact that Maupertuis, probably in order to avoid giving ammunition to the enemy, fails to relate some exceptions to this rule known to him.[59] Hunter, with his added category of accidents could however add:

> But monsters, in some cases, may be said to be accidental, as the horn growing out of the forehead of the ox or cow.[60]

I believe it is not a coincidence that both these authors used the same analogy and the same kind of examples to clarify their views on how variations could occur in organisms, and be transmitted to their offspring. In other words, I think one should call Hunter's view a Maupertuisian one.

What John Hunter had in mind when he made the hair-splitting distinctions in front of a murder jury can now be understood in relation to his more general views on hereditary variations. We can see by them how he was in a position to differentiate, by way of looking at patterns of inheritance and timing of appearance, between hereditary and accidental variations (peculiarities, diseases, malformations). More to the point, he could also defend with more clarity than other medics a difference between hereditary diseases and hereditary dispositions to disease. As many physicians insisted, direct inheritance of disease (which implies being born with the illness) either was rare, or resulted most of the times in prenatal death. Most familial patterns of recurrence of same or similar diseases could best be described as due to the inheritance of latent causes or dispositions, or what Hunter calls a susceptibility for the case of crystals.

Although Dr. Crichton had a point, as we said, when critizing Hunter's excessive claim for originality, Adams was also on track when he emphasized that Hunterian distinctions made thinking in hereditary transmission a lot easier. Where others had only unified the area of the hereditary by external coincidences, like the pattern of familial recurrences, but had very varied and sometimes inconsistent etiologies and physiological explanations for them, Hunter, based on his analogical approach, could structure a set of clear causal (internal) distinctions that accounted for the pattern in a more subtle way. He could give a meaning to words like "constitution" or "hereditary" that was more conceptually structured . Heredity of individual spontaneous variations (i.e. that occurred during the first formation of the individual) could, under the crystallization analogy, be seen as stable and transmissible. And the timing of the first appearance of a change as a criteria for categorization of the different types of variations and characters.

An important feature of Hunter's view, that later was exploited by Adams and Prichard, was that under his view the distinction, previously based on external evidence, between transmissible "congenital" characters, and non transmissible "connate" or "acquired" ones, was a natural consequence of the model. The mystery of why some peculiarities (diseases, deformities) did seem to get "copied" in the offspring while others did not, could be faced with a clearer mind, and related fruitfully to the patterns of evidence available.

Some caution must be taken in not confusing the above with the different, though related, late 19-th century discussion of Lamarckism. The inheritance or non-inheritance of acquired characters became an increasingly important issue during the course of the nineteenth century. It would, however, be a mistake to try to read too

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[59]See footnote 32, in *The Earthly Venus, p.57.*
[60]J. Hunter, Essays..., p.243.

much into any of these eighteenth century authors' views. The development of modern genetics and evolutionary theories have for many years now distorted our reading of most eighteenth and nineteenth century life scientists' works.[61] I mentioned Odom's hastiness in finding in Cullen a visionary that pre-conceived the phenotype. Hunter has been added to the inexhaustible list of Darwin's precursors . Maupertuis has been found by different authors to have foreseen the works of Darwin or Mendel.[62] Most of this kind of historical interpretations obscure the issues rather than illuminate them.

In the light of what has been discussed above it seems tempting to see in Maupertuis' and Hunter's analogy of heredity and crystallization as an anticipation of the contemporary ideas of replication and mutation. They were certainly not. What can perhaps be said, with hindsight, is that they turned out to be a further step in "reification" of heredity; that long and winding process which transformed, during the 18th and 19th centuries, the metaphoric adjective "hereditary" into an explanatory concept, upon which a relatively independent scientific discipline could be built.

[61]A clear description of this historiographical distorsion has been made by Madeleine Barthélemy-Madaule in her her book *Lamarck, the mythical precursor* (1982), where she wrote: "inheritance of acquired characters was to be sure a necessary support of his theory, but it was not a new thesis. No one either defended it or contested it. It was not even an issue; it simply went without saying. After Weismann took another look at Darwin, the problem of heredity became a real question and the quasi dogma of acquired characteristics came under attack". p.73.

[62]See for instance Quist, G.(1981) "Evolution", in *John Hunter 1728-1793*; and Glass, B.(1959) "Maupertuis. pioneer of Genetics and Evolution" in *Forerunners of Darwin*. See also Sandler (1979, 1983).

Chapter 4

The Hereditary in Britain in the early 19th Century

4.1 Normalizing Heredity: the de-pathologization of hereditary variation

I have in the previous two chapters described the way in which physicians of the 18th century discussed hereditary transmission of disease. How they conceived physical similarities and differences among humans as conforming to patterns that suggested a causal communication between the generations. Reccurrences of some diseases generation after generation in particular, they believed, are due to evil communications. Humoralists saw taints doing the damage that gave or predisposed to the illness. Solidists saw flaws in the organization as responsible. They all shared however a belief that a pattern could be discerned externally of the behavior of hereditary evil causes: These could come from both the mother and the father, or their ancestors. They could remain latent; they could be specific to certain ages of the individuals; they could "jump" generations. They could remain stubbornly in a family for many generations or disappear, sometimes for good. They were irregular and unpredictable. They could not be trusted. They were a menace to individuals, families, groups and nations. But they were knowable. Observation of general patterns of reccurrences could point out the features with which, eventually, it could be possible to distinguish what was genuinely hereditary and what was due to other kind of influences that obscured their manifestation.

The 19th century harvested the fruits of all these previous efforts at understanding hereditary transmission. It saw also the beginning of a change of attitude. Heredity, it came to be realized, could turn out to be more than an obscure medical subject.

In Britain, the first signs of this change can be detected in the first decades of the century in the work of two physicians: Joseph Adams (1756-1818) and J. C. Prichard (1786-1848), both of whom owed, in one way or another, their main insights to John Hunter.

James Cowles Prichard (1786-1867) was the most important figure in the early 19th century British writers on hereditary transmission of physical and moral characters. He developed his ideas while working on the relatively recent area of the origin of human varieties. A highly qualified physician, he was familiar with the discussions around hereditary diseases, and with the relation they had with the questions he was facing. Several authors before him had made the link, but the fact that the hereditary character of some diseases and that of other familial, group or racial resemblances belonged to the same kind of phenomena, to be explained in

similar sorts of ways was by no means accepted by everybody.

During the late 18th century and the early 19th century a tension can be detected, I believe, between different extreme positions: from considering most hereditary variations as pathological and monstrous, and considering them all potentially neutral. The belief that variation is *per se* a pathological phenomenon, and that for instance the origin of varieties and races within humanity had a pathological base was not uncommon. Buffon's ideas about the degeneration of the human and other species due to the climatic conditions in some regions and the persistence within some groups of "stable" altered states was accepted by several physicians, who shifted the emphasis to hereditary causes and declared that different non-European races or groups "laboured" under the burden of chronic physical (and moral) disabilities due to the fixation in their populations of some hereditary disease or other.

The French author Alexis Pujol maintained for instance that dark skinned human races had "la couleur cutanée profondement viciée", and that that similarly, the set of hereditary vices of temperament that account for the differences of the other races from white Europeans are, if not properly a diseased conditions, at least give hereditary predispositions to disease; many of them not mortal diseases, but certainly hereditary and degenerate. Stupidity, laziness and all sort of moral defects of other races could also be hung from this hereditary tree. Pujol was of course not unique in this kind of difamatory speculations, and most of the French hereditarian tradition during the 19th century exploited this dark mental connection between hereditary difference (variation) and degeneration, not only with respect to non-European human groups but also when dealing with marginalized families and groups within their nations.[1]

There was, as I said above, a contrasting trend to regard the tendency to produce hereditary variation within some species as not necessarily evil. Their idea was basically that some non-lethal spontaneous and hereditary variations (in both animals and plants) could be the raw material for the fixation, in isolated or other peculiar conditions, of the striking physical differences that characterize varieties, some of which need not be for the worst. A typical example was albinism, as it was believed that in some remote parts of the world whole tribes of "negre-blanches" lived. Maupertuis, it must be remembered, started off his influential discussion on generation and hereditary transmission as a reflection on albinism;[2] Maupertuis defended the view that this kind of variation can be fixed in sub-specific genealogical groups.

John Hunter, for instance, although imbibed with teratological language and imagery ("White Ethiopians, he wrote, are monstrous in respect to colour"), was also amongst those shrewd enough not to draw a sharp frontier between normal and

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[1]A. Pujol(1739-1804), 1789, "Essai sur les maladies héréditaires", p.269. The pathos of pathological inheritance was also applied by Pujol, and many other French authors to explain degeneration within European societies, using the family, instead of the race, as the genealogical unit of analysis. Such story has received increasing attention by social historians of science; recently I.Dowbiggin (1991) and D. Pick (1989) have produced fairly good accounts of it.

[2]The first version of his *Vénus Physique*, was called "Dissertation sur le Nègre-Blanche", was published anonymously 1743, and was written by him after a friend's request, when they had gone to see a specimen of the sort recently brought to Paris in a exhibition tour.

abnormal variation, as for him one could turn into the other. When he wrote for instance:

> Every species is, perhaps, subject to such [spontaneous] variations; and some of these deviations are so extraordinary as with propriety to be denominated as monstrous.

He immediately called for a footnote where he qualified his statement:

> Perhaps the word, monstrous, is too strong, or not exactly just. It certainly may be laid down as one of the principles of or Laws of Nature, to deviate under certain circumstances. It may also be observed that it is neither necessary, nor does it follow, that all deviations from the original must be falling off: it appears just the contrary, therefore we may suppose that Nature is improving its works; or at least has established the principle of improvement in the body as well as in the mind.[3]

After starting up from a position in which human variation and hereditary disease are emphatically separated as different entities altogether, the German anatomist J.F. Blumenbach (1752-1840) made over the years a relative shift towards a unified description of both pathological and normal variation; accepting that a clear distinction could not really be made between them. To put it in another way, he shifted from a position in which pathological hereditary influence was kept in a different theoretical space from other "normal" variations, into one where there was no essential distinction and both could be considered under the same theoretical frame. He put, in a sense, normal hereditary variation under the pathological aura of the hereditary, which was, with hindsight, a necessary step for the posterior de-pathologization of the field.

As Blumenbach was the strongest of Prichard's influences with regards to human variation and the origin of races, and it was in great measure to his pathologized view that he was reacting when creating his neutral description of the hereditary, it is worth quoting him at some length.

The first time Blumenbach addressed the question of human variety (*De Generis Humani Varietate Nativa*, 1775),[4] he was firmly opposed to considering some physical, hereditary irregular conditions, like albinism, as anything more than

> a disease in the human body, for the most part congenital, exactly like that which I have shown to attack certain animals; it is however different in this, that it plays with the symptoms, and now attacks man lightly, and now severely; in some countries it is rare, in others more frequent and endemic; here it is propagated in families, there it seizes people capriciously and individually. It affects the skin and the eyes at the same time.[5]

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[3]J.Hunter, "On the colour of pigmentation of the eye in different animals" in *Observations* ..., 1886, p.201. This last statement has been taken rather naively to mean that Hunter considered evolution through the improvement of varieties a possibility, and that he was then a precursor of Charles Darwin's theory. This anachronistic argument was made by G. Quist (1981), *John Hunter, 1728-1793*, p.186-187.

[4]Published in translation as "On the Natural Variety of Mankind" in *The Anthropological Treatises of J.F. Blumenbach*, 1865.

[5]Blumenbach,"On the Natural Variety of Mankind",p.133.

At this stage, Blumenbach defends the position that there is no fundamental difference between the communication of albinism between humans and that of other hereditary diseases: i.e. a constitutional flaw that happens to be transmitted. He emphatically states that the origin of human varieties, or any such theme in natural history is completely alien to this kind of phenomena. "The dignity of mankind —he wrote— demanded that these themes should be separated". But the real reason he gives is one of irrelevance. To consider the causes of disease, of any disease, relevant for the subject would be to open an unending listing of all other ailments that have ever distorted the nature of the human body

> The transition from thence to monsters would be easy, and so on to general nosology; and thus the divine study of natural history would run up into a confused and formless mass

Bringing into the fore one of the most extreme examples of those advocating the possible fixation in a human population of spontaneously arising hereditary characters, the case of the porcupine family in 17th century England[6] he insists

> Let us leave therefore unnoticed, for physiologists and pathologists, the black and horny epidermis of the...Englishman and others, and similar peculiar aberrations from the natural condition.[7]

By the time of the third edition of his work, 1795, Blumenbach had shifted his position.[8] No longer were those hereditary illnesses and other noxious variations irrelevant for the origin of varieties, but they were in a sense undistinguishable, except for secondary details. The theme of hereditary diseases now deserved a special consideration, and was used by him to exemplify his analogical argument from animals to humans.

In a section of his new essay called "Hereditary peculiarities of animals from diseased temperament" Blumenbach writes,

> An hereditary disposition to disease would seem at first sight rather to belong to pathology than to the natural history of animals. But when the matter is more carefully looked into, it is plain that in more ways than one it has something to do with those causes of degeneration we are concerned with. For, in the first place, some external qualities of animals, although according to common ideas they are never referred to as truly diseased constitution, still seem to come very nearly to that, for they are for the most part found in conjunction with an unnaturally weak affection. I include among these, for example, the peculiar whiteness of some animals.[9]

Like Buffon, Blumenbach believed in a monogenetic origin of human races, and that degeneration of some sort or other was the cause of human variety. In the account of how this degeneration occurred and was maintained within the genealogical lines they however differed. The stabilization and diffusion of variations in the genealogical group was the other problem, and the old analogy between hereditary resemblance and hereditary disease in families was the obvious candidate. This process, of passing down a deviation from generation to generation, Blumenbach

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[6]Famous case of the 17th century Lambert family; see chapter 6 for details.
[7]"On the Natural Variety of Mankind",p.140.
[8]Also translated in *The Anthropological Treatises* ..., 1865.
[9]"On the Natural Variety of Mankind" 3rd. edition, p.202.

found, could soften out the sharp, noxious edges of the character and "de-pathologize" it into an asymptomatic (though diseased) character

> some genuine diseases —he wrote— when the animal nature has been, as it were, used to them for a long series of generations seem to get sensibly milder and milder and less inconvenient, so that at last they can scarcely be considered more than a diseased affection...

Or further on

> When [some morbific disorders] are propagated by hereditary causes for a long series of generations it shades sensibly away into a sort of second nature and in some species of animals gives rise to peculiar and constant varieties.[10]

When leucaethiopia (albinism) strikes only sporadically a family of animals or humans, it brings with it morbid symptoms that are close to a leprous constitution, says Blumenbach, but when

> it has been established by a sort of hereditary right for many generations, it becomes a second nature, so that in the white variety of rabbits not a vestige remains of the original morbific affection, the existence of which however is determined by the analogy of other animals, which have anomalously white pupils and red eyes.[11]

It cannot be said that Blumenbach has eliminated the negative, degenerative connotations that hereditary variation has associated with it,[12] but by allowing this fading from the pathological into some kind of "normality" (which does not necessarily mean health), he contributes, I believe, to the establishment of a single causal domain that can include both the good and the bad. One finds then that, in Blumenbach's new approach, disease of a constitutional, hereditary kind is not of an essentially different nature from other accidental peculiarities which somehow find their way into the complexion of some persons ("specially in the face, as noses, lips and eye-brows") and then "are universally propagated for few or many generations with less or greater constancy" to form part of the family or national physiognomy. Individual faces, Blumenbach added, however varied and different, fall —all of them— into national categories. And these "national faces" can in turn be classified into racial varieties of faces.[13]

Like some of his contemporaries, Blumenbach was weary of incorporating bolder modifications, like deformities and mutilations, to the kind of those that can "give a commencement to native varieties of animals". He found the evidence insufficient and the irregularities too wild to give a veredict

> I have not at present adopted as my own either the affirmative or the negative of these opinions —he wrote; I would willingly give my suffrage with those on the negative side, if they could explain why

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[10] ibid.,p.259.

[11] ibid.,p.203

[12] Blumenbach did maintain for instance that blackness of the negro depended on a diseased biliar condition that had somehow become stable. He was strongly rebuked on thias point by Prichard, who reused to "believe that all black people labour under an inveterate hereditary jaundice". See *Researches* ...,2nd. ed., p.530.

[13] Blumenbach believes that faces are dependent on skulls, and skulls are the best tool to classify human groups. See ibid. pp.231-239.

> peculiarities of the same sort of conformation, which are first made intentionally or accidentally, cannot in any way be handed down to descendants[14]

What, in other words, makes the difference between these intentional or accidental modifications to the body, and those other "acquired" variations which spontaneously occur in nature, or those brought about by diseased constitutions. How could, for instance, the weather or the nutrition modify the body structure, internally and externally, in a qualitatively different way as these other coarser modifications, so as to make the former candidates for fixation in a genealogical line, and the latter disappear without trace. Once we start obliterating the distinctions some kinds of variations and others, where can we redraw the line so as to exclude some from the reach of the hereditary?

The evidence on both sides was ambiguous, irregular, and a lot of it unreliable. Blumenbach however could not but take sides, partially, by talking of a gradation of "transmissibility" according to the depth that the kind of cause could reach

> Mutations which spring from the mediate causes [of degeneration, like sun on liver or on darkness of skin] ...seem to strike root all deeper, and so to be all the more tenaciously propagated to following generations

But these mediate causes, given the state of physiological knowledge at the time, Blumenbach believed, were hidden

> at such a distance, that it may be impossible even to conjecture what they are, and hence we shall have to refer the enigmatical phenomena of degeneration to them, as to their fountains.[15]

4.2 Prichard's de-pathologization of the hereditary

When describing the problem he wanted to solve, at the beginning of his 1808 Edinburgh medical dissertation Prichard wrote about the issue in terms very similar to Blumenbach

> The reason for these changes, which bring it about that animals beget progeny different from themselves in certain respects, so that ever afterwards the variation must be transmitted in the stock, must remain a mystery, until we have a physiological understanding of the generative faculty, with better grounds by far than today. Since we are absolutely ignorant of the nature of this faculty, how can we hope to dig out its rules and exceptions.[16]

The origin of variation of body structure in animals and humans was to remain a mystery for many decades more. The perception however that there was something really fundamental that distinguished those variations that turned out to have a stronger capacity for remaining within a genealogical line and those that disappeared soon, coupled with the idea that there was no fundamental distinction between

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[14]ibid.,p.204
[15]ibid.,p.206
[16]J. C. Prichard, 1808, *Disputatio Inauguralis*, pp.56-57, as quoted by Odom,op.cit., p.184.

pathological and non pathological hereditary variation, at least at the fundamental causal level, set up the scene for the basic question that Prichard tried to answer, and which somehow differentiated his efforts from other British contemporaries, like Joseph Adams. He focused his attention on the hereditary cause and tried to establish the general features of it from the known patterns. Like Hunter, he believed that only variable internal causal influences present at the first formation of new individuals could make sense of the external patterns, and that these had somehow had to have a different status from other influences that might affect the individual's constitution during her lifetime.

Taking his cue from the distinction forwarded by several previous physicians when writing on hereditary diseases, that of a difference between congenital and connate influences, and also from the Maupertuisian model of variation, Prichard produced a very clear statement aimed at separating external (Buffonian) influences on the body after the first formation, and internal, hereditary ones. Adopting the stance of other medical men, about not getting into the controversy of generation but considering only the evidence of transmission of characters between generations,[17] Prichard built the argument of his first English version (1813) of his essay on the "physical history of man" on a strong anti-Buffonian stance.

> It appears that the principle in the animal oeconomy on which the production of varieties in the race depends is entirely distinct from that which regards the changes produced by external causes on the individual...These classes of phenomena are governed by very different laws. In the former instance certain external powers acting on the parents influence them to produce an offspring possessing some peculiarities of form, colour or organization; and it seems to be the law of nature that whatever characters thus originate, become hereditary and are transmitted to the race perhaps in perpetuity. On the contrary, the changes produced by external causes in the appearance or constitution of the individual, are temporary; and in general, acquired characters are transient, and have no influence on the progeny.[18]

This was in different ways a courageous assertion. To first declare (for rhetorical purposes) ignorance about a given process, and then to try and establish rigid distinctions of this kind put some stress Prichard's credibility, and some of his critics pointed the inconsistency.[19] Several authors have been keen to emphasize the "modernity" of Prichard's defense of a non inheritance of acquired characters based on an analogous distinction between "genotype" and "phenotype",[20] but have failed to appreciate the extent into which he was more than conscious of taking a leap in the dark; and have specially ignored the way in which Prichard himself modulated, and

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[17]This point was nicely made by Odom, when he wrote that "Prichard's professor's held the conviction that the theories of generation had reached a dead end ...and he had to find a new approach". Odom, op.cit, p.184.

[18]J. C. Prichard, 1813, *Researches into the Physical History of Man*, 1st. edition, pp.194-195.

[19]See Brown (1833). A common commentary could be on the line of Boisseau's, when he wrote that the distinction between connate and congenital, that some hereditarian physicians made, was "une pure subtilité...puisque nous ne savons en quoi consiste la conception". In Pujol (1823), pp.434-435.

[20]Odom (1970), Stocking (1973), Zirkle(1946).

qualified this opening tirade in posterior editions of his work, leaving more and more room for a direct influence of the environment in shaping (directing) the variations.[21]

In the context of the disputes about hereditary disease, Prichard's stance can be seen with more clarity. As we have been repeating, the search for a clear definition of the hereditary within the medical community was done with the dispute between solidism and humoralism as the physiological crux.[22] Obviously siding with a solidistic conception of individual constitution, Prichard was attempting at cutting through all the causal confusions that had undermined the works of such great thinkers as Blumenbach, and Haller before him. Both of which for instance when faced with the evidence of anomalous and striking instances of freak "hereditary" acquired variation (like the much repeated case of a bitch that having received a blow on the back while pregnant gave birth to back-damaged puppies) although sceptical, did not want to deny definitely their reality.[23] Prichard was also impatient with the proliferation of far-fetched explanations about the origin of diversity in humans and animals. He was, in the first edition, willing to accept, for instance, that domestication (civilization, in the case of man) had a profound effect on the individual's constitution (he did not explain why) but denied the validity of climatic explanations. Too much has been written, foolishly he thought, about the miasmas of some regions, or the intolerable heat or humidity of others, as eating into the human (and animal) physical constitution of their inhabitants and leaving them eventually in a constant (endemic) diseased state. Pathology, specially humoral pathology, with its proliferation of influences and its open invitation for speculation had to be checked. In the Scottish tradition, Prichard preferred simpler schemes and explanations,[24] Prichard felt that the hereditary was a good place to start imposing some order. The elimination of the pejorative turns of voice (those given by talking of diseased constitutions and degeneration) from his references to the hereditary is one of the main reasons why his texts give an illusion of being more modern, and closer to Charles Darwin, than most. That Prichard was however labouring under the same causal scheme as most of his contemporaries is proved by his increasing qualifications and baroquisms of his second (1826) edition of *Researches*, when he tries to tackle criticisms of those who favoured environmental causes of hereditary variation by incorporating them into his scheme.

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[21]Perhaps in a similar fashion as Charles Darwin, many years after him, Prichard felt compelled at relaxing his stance against "inheritance of acquired characters" when critics piled up evidence that pointed on the opposite direction. The analogy cannot however be pushed too far.

[22]To conceive the individual constitution as basically determined by the unstable behaviour of humoral influences, with their ubiquity in the body and their transportability in and out of it, means opening a large field of interaction between the body and the environment where the latter moves the main causal handles. The "non-natural things", as Hippocratic medics used to call external influences, could shape body and soul, and leave their mark behind in a sort of parasitic, deeply rooted humoral taint, which nevertheless was eradicable. For solidists the individual constitution was given by structure, organization, and it became basically fixed at the moment of the first formation of the individual.

[23]Haller, for instance, reported this kind of cases in his *Similitudo Parentum*, loc. cit.

[24]His work on all areas, from anthropology to psychiatry show this penchant for simple, logical schemes, and his capacity to collect and organize masses of evidence from a wide range of sources.

It was in this second edition of *Researches* where Prichard produced the most impressive, to a modern eye, account of what he wanted to put forward as the "Laws of the Animal Economy in regard to the Hereditary Transmission of Peculiarities of Structure". In this section of his book he begins by describing the problem as one of distinguishing between those "peculiarities of structure" that are liable to be transmitted by parents to their offspring, and those that "terminate with the individual without affecting the race". He answers by stressing that the main criteria is the moment, and way in which a peculiarity is incorporated into the constitution.

> all connate varieties of structure, or peculiarities which are congenital, or which form part of the natural constitutions impressed on an individual from his birth, or rather from the commencement of his organization, whether they happen to descend to him from a long inheritance, or to spring up for the first time in his own person —for this is altogether indifferent— are apt to reappear in his offspring.

His reformulation of this statement, is at the same time vaguer, and for us, more clarifying of his view.

> the organization of the offspring is always modelled according to the type of the original structure of the parent.[25]

Other physicians before him had tried to make causal sense of the distinction between congenital and connate influences, but only those with a clear solidist approach, like Pagès in France, had come close to a similar clear-cut distinction. Prichard did not seem to be aware of previous efforts in the same direction, as he states that the "distinction ...has not been pointed out by any former writer on physiological subjects [and] was first suggested to [him] in conversation ...by Mr. Benjamin Grainger of Derby"[26] Prichard, it can be said, goes one step further than previous authors in that he does not fix his attention only in the causes of disease, but generalizes the hereditary for all peculiarities existent at given moment, and in that he hints at the existence of a unified mechanism of transmission. He not only sets the events at the first formation of the body of the individual as the only truly hereditary, but also sets two frontiers in the temporal sequence, around the moment where actual these hereditary influences can be communicated. Nothing that happens before or after the event of the organizing of the individual can take root in the constitution in such a deep way. The process of organization could be instantaneous or short-lived (as in the Buffonian or Maupertuisian models), or they could take place through a developmental sequence, it was up to generation theorists to discuss that. But from external observations of pattern of transmission of different kind of characters, one could infer that only whatever cause acts there, between those two boundaries, is truly incorporated into the "type" and can ultimately be diffused from the individual to his progeny, affecting eventually the family, the group, the nation, the race.

The forced nature of the position developed by Prichard within the conceptual world he was moving is made clear when focusing on the word "type". The constitution of a child is modelled, he says, on the individual type of the parents. This is taking, as Borie has argued in relation to Prosper Lucas,[27] the typological position to a strained position. Leaving aside the tricky question about whose type, the father's or the mother's, is the child going to be modelled upon, the sheer fact of talking of

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[25] *Researches*, 1826, p.536.
[26] ibid.,p.537
[27] Borie, *Mythologies* ... (1981).

individual type seems paradoxical.[28] The modelling metaphor is clearly making a point of having the new being conformed with the parent's peculiar constitution as source (and not from the species' or the group's). But it is not the particular constitution itself, but it's type (which is as unique) what constitutes the real source. So whatever happens during his life to the actual body of the parent (if it looses a leg or catches malaria), this type, as it was "frozen" at his first formation, will not change, and thus the changes will not influence its offpring's organization. As he puts it

> whatever changes of organization are superinduced by external circumstances and are foreign to the character of structure impressed upon the original stamina, cease with the individual, and have no influence on the race ...this law of hereditary conformation exists with a certain latitude or sphere of variety, but whatever varieties are produced in the race, have the beginning in the original structure of some particular ovum or germ, and not in any qualities superinduced by external causes in the progress of its development.[29]

The problem Prichard did not address in this schematic depiction of hereditary transmission, and that left him vulnerable, is how can this separation between the original and the acquired be physiologically accounted for. Any organizing mechanism (or principle) has to either follow a general pre-established route, or it has to copy an actual particular mould. But how could an "individual type's" action be preserved if the actual constitution it reflects is altered. So, in other words, how can the modelling mechanism tell apart what is original and what is acquired while re-producing an adult's constitution in its offspring.

Prichard's solidist approach, if accepted, could eliminate from the hereditary scene all external (climatic and other) influences acting on the body after its basic organization had been completed (some time after fecundation). But having no idea of how the internal detailed structuring process takes place, the possibility of external influences acting at a period before solidification could not be similarly eliminated.

Prichard was well aware that it was from the medical camp that some of the strongest critics of these speculations would probably emerge, as some of them had more at stake in the discussion. His description of the hereditary ran against the

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[28]Odom, following Mayr has described Prichard's and Lawrence's stress on the individuality of the hereditary constitution as a break with typological thought and a movement towards what they call population thinking (See Odom op.cit., and Mayr (1982). But individuality of constitution (or temperament) and its consequence, idiosyncracies in disease and reaction to treatment, had been in the medical panorama for ages, including in the speculations about the hereditary. The problem that Prichard and his predecessors faced was not the singularity of hereditary variations, but the apparent arbitrariness of it all. The inference, that strikes Odom as particularly modern, from individual variations to fixation of the character on a group (or population), was one that was made repeatedly, since antiquity in relation to the hereditary. The most famous among the ancient cases being the description of a tribe of "macrocefalous" people given by Aristotle, and explained by him with the same scheme. What Mayr describes summarily as "essentialism" or "typological thinking" had room for a quite subtle, hierarchical analysis of character transmission as the example of Prichard and Adams.

[29]*Researches*, 2nd. ed., p.545.

comfortable belief that constant exposure to pernicious external influences (like alcohol or extreme temperatures) would, proportionally to the number of generations exposed to them, eventually become fixed in a genealogical line. The whole range of phenomena investigated by physicians as being based on hereditary predisposition to disease, Prichard argued, instead of being adverse to his broad generalization as many would suppose, will "on closer investigation...appear to confirm it".

> It has been supposed by medical writers, and the notion has been generally received, that any morbid predisposition may be formed in almost any constitution if it be subjected to a certain train of predisponent causes; that what is called gouty diathesis, for example may be acquired by long habits of intemperance, and transmitted to posterity ...it is said that the children of dissolute parents suffer punishment for the vice of their progenitors. If this opinion be correct we have a clear proof of the hereditary nature of acquired states of the constitution.[30]

But it was the meaning itself of "acquisition" that Prichard wanted to call into question. Not every individual that is exposed to a morbid external cause "acquires" the disease; each one responds in a different fashion to the same environmental stimulus. What for some is the triggering cause of disease, remains insignificant for others. This kind of idiosyncratic responses to influences were well known to physicians, and their only explanation, Prichard argued, was to be found not externally but within the individual's own physical frame

> the difference... must be in the natural peculiarities of the constitutions on which they act. These, therefore, are previously fitted by original organization to take on them one form of morbid affection rather than another. It is then clear that the predisposition is laid by natural or congenital structure, in the first instance.[31]

"Acquisition" thus means simply the enacting of a potency that is already there, in the original structure. The exciting causes only serve as revealers of such latent flaws. Within a family several members, or all, can share a given hidden defect; but it can remain unknown for several generations until one of its members is exposed to the triggering influence; in such cases

> the first individual who exposes himself to the morbid causes first betrays the peculiar defect of the race, and is thus erroneously supposed to lay the foundation for it.[32]

Presumably, the event that produced the first constitutional alteration of the line (i.e. the original variation) occurred during the "first formation" of an ancestor who did not develop the disease. Atavism and homochrony, and in general the latency of hereditary causes can be accounted for naturally under this scheme. The problem of hereditary transmission is then circumscribed to a short period of time in the life cycle of humans and other sexually reproducing species; and the causes of variation are also situated there. The latter were of course crucial for Prichard's main goal of explaining the origin of human varieties. It is to this concern that he dedicated the next section of the 1826 edition of *Researches*: "Theories of the Origin of Varieties".

The ignorance of the causes that prompt the appearance of variations at the

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[30] *Researches*, 2nd. ed., p.545.
[31] idem.,p.546.
[32] idem.,p.547.

moment of the first formation leaves room, Prichard acknowledges, for an open ended series of influences, which can at least be reduced by common sense and the knowledge of the main laws of physiology. The action for instance of the mother's impressions or imagination on the forming foetus is not denied by him but is qualified.[33] Quoting Boerhaave, he acknowledges that although the weight of the number of cases collected since antiquity to this effect is not negligible, there seems to be in them "something which does not at all agree with the laws of nature, as they are at present known to us, and yet the facts cannot be denied, unless the laws of Nature were perfectly known to us".[34]

It is in the subsequent section ("Instances of variety in the Breed arising from the operation of external, chiefly local causes") where, as we said above, Prichard began separating himself progressively from his simple explanatory scheme and accepting external (mainly climatic) influences as the cause of stable varieties in the wild. Unique cases of externally induced variation did not concern him so much as the recurrent emergence of similar variations in groups under the same conditions. Domesticity and civilization were, as many authors stated, a main source of variation. And the fixation of some of these variations, in the case of breeding, could be accounted for by selection procedures. The same was not the case for wild species and races. For them, Prichard wrote

> I cannot conceive any other way of accounting for the general appearance of any particular character in the whole race found in a certain situation, but the supposition that the local circumstances have a tendency to call it forth in the breed, or predispose the parents of the stock to produce offspring marked by the character in question.[35]

What to Prichard's mind could (in his first edition and the first part of this second one) explain family, or group resemblance, and even family patterns of reccurrence of disease, suddenly seems to him not good enough to account for geographical variation and racial diversification. The hereditary as the constitutional predisposition had its limits as a source of variation. There is a shift then towards external diversity of causes acting upon a more or less stable set of internal tendencies. The *ceteris paribus* clause is no longer applied to the external, prompting causes, but to the internal reacting ones.

> ...the race is originally the same, and the deviation in it must be attributed to the influence of their circumstances, whatever they may be, which are connected with local situation[36]

Prichard tried of course to maintain certain coherence between the two parts of his exposition. So he was adamant that any external influence, to be effective in producing the variation, had to act upon the embryo during its first formation. And

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[33]As in older days, Prichard wrote, "some modern writers, among whom was Dr. Darwin, [believe] that at the period when organization commences in the ovum, that is at, or soon after the time of conception, the structure of the foetus is capable of undergoing modifications from impressions on the mind or senses of the parent", and such opinion "does not appear altogether improbable", but the constancy and stability of varieties cannot be attributed to these kind of causes, he concludes. See *Researches*, 2nd. ed., p.555.

[34]idem.,p.554.

[35]idem.,p.559.

[36]idem.,p.563.

claimed also that the different "specific diversities" that could stem out of a given genus would probably have to be consequences also of a "modification in the productive causes stamped in it originally". But the strain introduced into his argument by the shift of causal focus, and the dislocation of the hereditary as the basic explanatory resource becomes obvious, even to him, when he starts producing different and incompatible explanations of phenomena he has previously described. The permanence of racial characteristics even in changed climatic circumstances was evidence he had used in the second section to establish his hereditary view. Now he began flirting with the idea of a successive darkening or whitening of populations in changed environments. Where in a previous section he had for instance attributed the "darkening" of the Jewish populations in Ethiopia to the inevitable mingling of bloods through the centuries, in this one he states that it is a matter of successive migratory waves, and that the older stock has had more time to darken itself prompted by the climatic influences. Perhaps being over-tolerant with his inconsistencies, he only brings himself to admit that "there is some difficulty in reconciling with these conclusions the facts alluded to in the second section of this chapter, indicating the permanent transmission of white or white complexion in certain races which have changed their abode from one climate to another";[37] he does not try to face the difficulty.

It seems obvious that at some point between the publication of the first (1813) and the second (1826) editions of his *Researches*, Prichard modified his view of the importance of the external influences in the shaping of the stable peculiarities that characterize different subgroups (races). He still believed it was important to separate hereditary and stable modifications from accidental irrelevant ones, i.e. between those that had a causal "root" in the primary organization of the individual, and those that didn't. But the causal weight of this predisposition seems to have been diminished in his posterior accounts, specially of geographical variation, which he considered somehow more stable and recurrent than what repetition of accidents of constitution and their spread within genealogical line would suffice to account for.

Prichard ended eventually facing up to the fact that there seemed to be different behavior of different kinds of characters within different groups. Human varieties did not have a pathological-hereditary origin, nor a merely environmentally guided one; hereditary variation and environment both had a role to play in their explanation, but no way existed to adjudicate between them. Though in the first edition of *Researches* he forged a clear-cut causal and analytical description of what it could mean to call a character "hereditary", and managed with it to produce unified accounts of most of the phenomena linked to the matter, Prichard seems to have been forced by posterior confrontations with the evidence and arguments of those who defended environmentalist causes, to modify his stance. In the previous decades, it had been the impossibility to accommodate it to their descriptions of how reproduction (generation) comes about that had made the evidence linked to the hereditary quite troublesome for system builders. Prichard's initial attempt at a systematization of the hereditary was similarly vulnerable to more or less well authenticated evidence that strengthened the claim that external, non- hereditary (in his sense) causes were guiding the occurrence of variation. The lack of any plausible physiological account that could substantiate his distinction between the congenital and the "acquired", and explain away the evidence produced by its opponents, made the idea seem too

[37] idem.,p.582.

theoretical and far fetched, and with that some of the possible avenues that the de-pathologization (or "unification") that he achieved within the field were lost. In other words, the field of biological heredity as an autonomous theoretical pursuit, which could seem with hindsight a natural outcome of Prichard's early train of ideas, was still not at that stage a transparent and well defined one. Physiology had always been the clue to the de-pathologization of the hereditary we have been describing. As long as external causal elements are accepted as the basis of hereditary patterns, the separation between pathological and non-pathological actions was inevitable. This is reflected in the use of words like taint, hue, levain, etc., of the humoralist tradition. The alternative solidist view that puts the inheritance of structural peculiarities as the norm gives both favourable and unfavourable characters equal standing in front of a given physiological cause of hereditary transmission. The other ingredient to this de-pathologization is the insistence that a constitutional predisposition and not the disease itself is what is reproduced by the metaphoric copying hand. Such a metaphor in fact captures Prichard's early views appropriately: a wise copying mechanism that can distinguish original structure from superimposed one. But the substantiation of this metaphor in physiological terms was still a very remote possibility. The door of the external shaping influences could not be shut definitively by Prichard, nor by any of his contemporaries.

Many of his readers perceived this weakness. Specially among those medical authors concerned with constitutional (familial) diseases, scepticism grew around the hair splitting lengths that had been arrived at by some defenders of hereditary predisposition as their main etiological determinant.

4.3 Hereditary Disease in early 19th Century England

4.3.1 Joseph Adams' analysis of causes

In 1814, very soon after the publication of the first edition of Prichard's *Researches* (1813), a well known disciple of John Hunter, Joseph Adams, published his essay *A Treatise on the Supposed Hereditary Properties of Diseases*.[38] In it Adams covers a similar terrain and with a remarkably similar position as those chapters of *Researches* touching the origin of hereditary variation. Although it only mentions Prichard's work marginally, Adam's essay can be read as a reaction to its discussion on the hereditary, with the aim of setting the record straight. First by giving due credit to John Hunter as the real source of the clarifications that Prichard produces as his own; the distinction between hereditary and connate, and the stress on dispositional causation. And second by providing an even clearer and more comprehensive analysis based on authentic Hunterian principles. It seems however that he owed more to Prichard than he acknowledges, and in the second edition of his *Researches*, the Scottish author, in return, although obviously borrowing Adam's clearer descriptions, and some examples, paid silence with silence.

As editor of the *London Medical Journal*, Adams considered himself the promoter in Britain of the discussions around Hereditary Disease; he had rekindled the interest

[38]J. Adams, 1814, *A Treatise on the Supposed Hereditary Properties of Diseases*, J. Callow.

in the theme when he published a few years earlier (in 1808-09) the translation of substantial portions of Antoine Portal's essay "Considerations on the Nature and Treatment of of some Hereditary or Family diseases".[39] The extreme humoralism of this work (which he describes as backward, and takes, mistakenly, to be representative of Continental thought about the issue) was used by Adams as a reference point from which to launch his innovative argument. The confusion Portal seems to have had between family and hereditary diseases is pointed out as one generally found in pre-Hunterian literature.[40] A subtler analysis of pattern and causation however dispels the error. Family diseases are those that strike brothers and sisters of a given generation but are not incorporated into the genealogical line via hereditary transmission. Hereditary diseases are those, and only those, that depend on the faculty of parents of communicating constitutional peculiarities to their offspring. The first major source of confusion in Portal, as in most humoralists, is his reluctance to leave out of the picture diseases that are obviously produced by some kind of contagious matter or other. Small-pox or syphillis should never be considered under the same category as gout or scrofula. The latter are constitutional in that they can spring out from a structural variation without contact with external morbific influences. Like Hunter and Prichard, he considers normal and pathological variation one on the same grounds, and tries to produce a unified explanation of their hereditary transmission.[41]

Not all constitutional variations that are the source of disease at some point, Adams argues, are liable to become hereditary. In many cases, of which he mentions some, a tendency is found within families to produce siblings with similar malformations or diseases, but then are not transmitted to future generations. These family patterns seem to have a different etiological basis than the transmissible, hereditary, tendencies, and in order to describe the difference Adams introduces, nodding towards Hunter, the three-fold distinction between carrying, at birth, a connate or congenital disease, a disposition to a disease, or a predisposition to a disease.

Leaving aside those perinatal contagions that produce disease in the child, he considers in the first category those privations and structural peculiarities that are obvious at birth, like congenital deafness or hydrocephalia. Very rarely this kind of peculiarity can be hereditary in the real sense.

The latency of authentically hereditary causes allow for normal births in which the child has a weaker or stronger susceptibility for the disease. The difference

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[39]A. Portal, 1808, "Considerations on the Nature and Treatment of some Hereditary or Family diseases", *London Medical Journal*, vol.21, December 1808 - June 1809. pp. 229-239, 281-296. The pagination of the December issue was wrongly done. It seems to me very probable that when Prichard was writing about the weaknesses of humoralist versions of hereditary transmission, specially their ascription of several diseases in succession to one unique taint, he had Portal's exaggerations in mind.

[40]Portal, in a later edition (1814) of his essay accepted Adam's criticism and attributes it to a having wrongly worded his views, but not to any error of conception.

[41]In the second edition of his essay, only a year later and with barely any change in the text (only an extra appendix on goitre), Adams significantly changed the title of this work in order to generalize its meaning beyond the pathological realm and into a wider domain: *A Philosophical Treatise on the Hereditary Peculiarities of the Human Race*, 1815.

between the hereditary influence that Adams calls a disposition and the one he calls a predisposition is the need, in the latter case, of an extra, exciting cause. Dispositions do not need such supplement; the arriving of the individual at certain moment in his life history is what triggers the onset of the diseases' symptoms. Crucial moments in life, like dentition or adolescence can call forth these developments. Dispositions are in a sense fatal, whereas predispositions can be avoided.

> When the *susceptibility* to an hereditary or family disease is so great as to amount to a *disposition* [it] can be induced without an external causes [and] we can have little hope of preventing it...But when the susceptibility is so slight as to amount only to a *predisposition*, we have rarely any means of discovering it till the disease itself approaches.[42]

Sometimes, what mistakenly is considered as the "same" illness can be introduced by any of the three kinds of causes

> In some families, we see a number of brothers and sisters falling into consumption in succession as they arrive at a certain age. This we may strictly call a *family disposition* to the disease, inasmuch as it is confined to a single generation, and as we discover no external cause to excite it. Another kind of consumption, and the most common in cold climates, is hereditary; but only in *predisposition*, always requiring the influence of climate to induce it, and consequently always to be prevented, and often relieved, by avoiding the exciting cause.[43]

Adams' split of the susceptibility hypothesis into two categories was a very subtle move that was not appreciated altogether by subsequent authors. He could with it encapsulate both the stricter stance of those who saw the homocrony of the occurrence as the criterion for the hereditary, and those who wanted to leave room for exciting, external causes. He gets very close to Prichard's argument when he describes this latter kind of causes as sometimes revealing tendencies that may have been first introduced into the family line several generations before without ever being noted, as nobody had contact with the exciting cause before.[44] He also graduates the importance of the hereditary predisposition to a point where hygienists and moralists, who want to stress the control that humans can have of the activation of the disease through habits and living conditions, can be accommodated. He castigates only extreme humoralists and contagionists, who see no role for the inherited peculiarities as he understands them, and who hide, according to him, in the obscurity of phrases like "hereditary taint".[45]

Adams, as Prichard, has no doubt that constitutional peculiarities of similar sort are at the root of both human varieties and of hereditary disease. He admits that ignorance of the actual physiological mechanism that produces such variations

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[42] *A Treatise* ...,p.21. and p.23.

[43] *A Treatise* ..., p.16. Phenomenologically, we must remember, consumption (tuberculosis) did not behave in an obviously contagious fashion, and both its symptomatology and its pattern of re-occurrence more than justified an hereditarian hypothesis. The same can be said of gout, scrofula, epilepsy and madness. But not of syphillis, for instance.

[44] Compare p.25 in *A Treatise* ... with p. 202 of *Researches* in the 1st edition.

[45] He is particularly harsh with Erasmus Darwin, and he writes that his treatment of the hereditary in *Zoonomia* and the *Temple of Nature* "show the danger of uniting poetry with pathology". See appendix to 2nd edition.

hinders the attainment of a stronger degree of certainty in his inferences, but is adamant that his Hunterian approach is the most coherent and consistent with the known facts of physiology. He specially denies that the original physiological root of hereditary diseases has to be known in order to accept their transmissibility, as some sceptics argued. Why should different standards be applied, he asked, to the acceptance of heritability of other variations, even monstrosities, and that of hereditary disease

> Is it more remarkable that a diseased disposition should be perpetuated than an actual monstrosity? [46]

Transmission of peculiarities is common place, he continued, in very many less complex species, like cattle or vegetables, and they are accepted without waiting for knowledge of their deep causes, and even used for the improvements of breeds. In relation to this, as Odom has pointed out, Adams was convinced that a similar process to the one that created new breeds (i.e. selecting a given peculiarity in several individuals and breeding in and in) was at work in isolated human populations. With more than an allusion to Prichard, Adams pointed out that in the origin of varieties the sifting of ill and well adapted individuals by the climate is a "law which has been too much overlooked". His idea was that by triggering some diseases in some constitutions and not in others the climate would favour the perpetuation of the latter. The similarities of this inference with natural selection has been pointed out by Odom.[47] Adams also put this kind of inference to work in the explanations he gave of the high incidence of some endemic hereditary diseases in isolated (geographically and by prejudice) populations, like goitre among the Cagots.

> secluded from the rest of mankind, the family must constantly intermarry, and any peculiarity or hereditary disposition would, with greater probability, be perpetuated...

In the case of favourable variations, they

> would be preserved with more certainty, because the inclination of each sex would induce alliances with the best favoured, and the most vigourous would live to the age of forming such alliances.[48]

Prichard and Adams set the tone for the views that medical men tended to have in Britain during the following decades of the 19th century. Their solidist analyses share the same basic features that I can now summarize: The limitation of hereditary transmission to only a few constitutional illnesses (i.e. an opposition to using the adjective "hereditary" loosely as a synonymous of a familial pattern); the idea that their source is to be situated at the moment of the first organization of the individual and that they depend basically on peculiarities of structure that do not differ essentially from those that give family resemblances, national or racial variations; the idea that hereditary causes tend to be dispositional, latent and depend on exciting causes with a similar timing (homochronic); the gradation from fatalistic to mild hereditary dispositions. All these features were the ones that helped frame Heredity, in the following decades, when it came to be a central explanatory concept of biological sciences.

Most medical and anthropological authors who dealt with the subject between

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[46] *A Philosophical* ...,p.22.
[47] See Odom, op.cit., p.186.
[48] *A Philosophical* ...,p.13.

1820 and 1850 tended to reproduce these sets of assumptions in one way or another. I will describe briefly some of the most important contributions along that line, but will first introduce the weaker, but eventually quite influential, line of sceptical argument of those opposed to hereditary explanations of disease and of varieties. Their contribution, I must say in advance, was mainly to put pressure on hereditarians by pointing out the many weaknesses of their inductive inferences and theoretical assumptions, which eventually forced them to organize the facts in a different (statistical) way and to refine their causal hypotheses.

4.3.2 Henning, a sceptical challenge

In the same year and publishing house as Adams second edition of his work (1815), George Henning, in a dissertation on the pathology of Scrofula, made the most ably and strongly worded attack on hereditary explanations of disease since Antoine Louis.

> It is a fact —he wrote—, on every account worthy of observation, that gout and mania, scrofula and phthisis, together with epilepsy, the only diseases, I believe, acknowledged to be incurable by means of medicine, are the only ones that have acquired the character of being inheritable. A fact that begets some suspicion, that the medical world has taken sanctuary under this term hereditary, to shelter themselves from the opprobium of not having devised remedies for these obstinate maladies.[49]

To give the impression that these infirmities are so deeply rooted (interwoven) into the constitution (fabric) of the diseased as to be inextricable from it by external intervention is a coarse attempt, he adds, to vindicate the profession, and diminishes rather than enhances its reputation.

Henning sets himself the task of undoing (deconstruct some would say) the strategy of hereditarians, who make the transmission of disease depend on "a *something*, which is transmitted through parents to their progeny". Humoralists believe the "something" to be in "a fomes or taint resident in the fluids". Solidists believe it to be a "peculiarity of structure of the whole body, or of some particular parts of it", and among them some limit the influence to a "praedisposition". All these candidate causes (of scrofula and other hereditary diseases) are very far from being proven, Henning continues, and their only real bases is very weak indeed: the familial pattern of occurrence. The observation that healthy parents procreate healthy children, and diseased ones procreate diseased children is as old as Hippocrates, but the inference to hidden, internal causes of any kind is not justified by any further explanation. The fashion, as he calls it, to account for familial diseases in such a way stems, Henning believes, from a misreading of the rhetorics of Hippocrates an other ancient authors by late Rennaisance medics, like Fernel and, later Duretus. The comparison in some ancient works of family and racial resemblance on the one hand, with familial or group disease or deformity on the other, does not imply an appeal to a common cause, and the label hereditary does not have explanatory value. And neither the Renaissance physicians nor their followers have ever justified their appeal to

[49]G. Henning, 1815, *A critical inquiry into the pathology of scrofula*, J.Callow, London. It is interesting to point out here that some historians have given a very similar explanation for the rise of hereditarian explanations in French medicine (Ackernecht, 1967) and psychiatry (Dowbiggin, 1991).

inheritance by adequate observations

> If these opinions...were the result of accurate observation, and had obtained the concurrent sanction of unprejudiced writers, no reasoning however specious, should convince me of their fallacy, or induce me to controvert them. But since I regard them as not having been originally founded on just observation [but] as still received either from deference or prejudice, I think it necessary to examine the grounds, and some of the modern authorities on which they rest.[50]

Henning first attacks the belief that the set of illnesses considered hereditary are confined to families. Such exclusivity claim is absurd, he points out, specially in the case of scrofula. Individuals from families that have never been affected can develop the disease. A convincing case is that of immigrants from warmer regions where scrofula is unknown, of non white racial origin, and who are afflicted by the disease in Europe.

Addressing the issue of causes, Henning establishes that neither humoral nor solid ones can be appealed to in most cases. The recuperation of health would be completely blocked if the origin of disease were "some particular conformation of part, or of the whole body, some deviation from the usual structure" because "how can that be corrected by ...any means? ". If that were the case and individuals were actually born with the structural defect, how is one to account for the lack of symptoms at birth in most reputedly hereditary diseases. By which sort of device could the body hide the presence of a major flaw. It seems difficult to maintain then that the actual disease is born with us.

The same is true for a humoral hypothesis

> If a fomes or vice in the fluids of the body, and congenital with it be the materia morbi...why does it so generally delay to shew itself during the weakness of infancy; or why does it so often fail to shew itself at all, at any other period of life.[51]

Henning is recirculating here the oldest and strongest argument against hereditary transmission, the unthinkability of latency under the prevailing physiological (materialistic) descriptions of the body. It was precisely against such arguments that all the subtleties of dispositional causation were devised. And these were Henning's next target. Some of the advocates of hereditarian explanations, he writes, are sufficiently sensible to their defects to have

> refined the notion of inheriting disease, into inheriting praedisposition to disease. As if this alteration was anything more than the substitution of one term for another, without obviating any objections.[52]

That a propensity is what is inherited and not the disease itself is a proposition, he argues, that is impossible to prove or disproof. Children can be afflicted by so called hereditary diseases even if their parents or ancestors were not, and on the other hand children from afflicted parents can pass their lives without ever developing the disease. The appeal to exciting causes on the other hand is just as tricky, as many "predisposed" children (i.e. offspring of diseased parents) get into contact with

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[50] *A critical inquiry* ..., p.53.
[51] *A critical inquiry* ..., pp.56-57.
[52] idem.,p.56.

exactly the same situations in life as their parents and do not develop the diseases. The defenders of the predisposition hypothesis make life very easy for themselves in that any way events go they can explain them away, without furthering the understanding of the diseases. The truth is, Henning writes, that

> if either the disease or the praedisposition to disease [were] hereditary, it should be constantly, not occasionally inherited; upon the principle that the operations of nature are for the most part uniform and constant, and that the same cause is usually productive of similar effects...all the children from the same bed should alike inherit and possess the same praedisposition. It must either be hereditary in this full sense, or never can be inherited. There can be no middle course.

The only exception he is prepared to contemplate is the one when a diseased parent is coupled with a healthy one, and the child constitutionally resembles the latter, so the ill tendency could be said to have been eliminated by the healthy parent's influence. But cases that go against the general hypothesis of predisposition are too common to use this single possibility as strong enough evidence.

Henning then proceeds to strengthen his sceptical stance by using the numerical weighting of favourable against unfavourable instances of parents to offspring transmission in some of the most common "hereditary" diseases. In the case of madness he uses the rudimentary statistical comparison done by Dr. Haslam.

> With regard to mania, although the prejudice is so strong and general in favour of its hereditary nature...[such] an acute and ingenious writer (whose opportunities [for observation in] the largest Institution for the reception of insane perhaps in the world, must be very ample) out of twenty nine cases,...[only] in three there were any suspicion of its being a family disease.[53]

The case against hereditary gout, Henning argues following William Cadogan,[54] is even stronger in that the "exceptions" by far outnumber the transmitted cases. To insist that gout is hereditary only because it seems so sometimes, when "if we compute the number of children who have it not, and women who have it not, together with all those active and temperate men who are free from it, though born of gouty parents, the proportion will be found at least one hundred to one against that opinion", Cadogan had written. Henning adds that the proportion would be even smaller if properly measured.[55]

The subtleties, thus, of predispositional arguments, Henning concludes, are nothing but smoke-screens that hide the devastating effects of counter-examples. Statistical tables, he seems to point out, in which favourable and unfavourable cases

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[53]idem.,p.58. He refers to Haslam's *Observations on Insanity*, 1809, 1st edition,pp.95-102. Henning admits that Haslam gives "hereditary predisposition" as as source of madness, but he adds that this author plays down its relevance in all the explanations he provides.

[54]Henning is particularly keen in underplaying Joseph Adam's contentions about John Hunter's importance as the main creator of the predispositional stance, and advocates Cadogan's argument as subtler, though also mistaken. See op.cit. pp.60-66.

[55]See Cadogan, op.cit., p.17-19, and as quoted by Henning, op.cit.,pp.66-67. Henning criticizes Cadogan for not extending his clear view on gout to other so called hereditary diseases.

can be lined up together without the intervening fancies of hereditarians, will eventually demolish their case. It was not however until three decades later that, on another treatise on scrofula, Phillips (1846) picked up the hint from Cadogan and Henning, and developed a statistical argument against hereditary diseases, an argument which was later extended by the historian Buckle against other hereditarian claims.

Perhaps it is not beside the point to finish this exposition on Henning's view against hereditary transmission, by saying that he favoured, for the case of scrofula, a contagionist view of the disease's etiology. Ackerknecht has described how he considers hereditarian explanations as reactionary, and in conflict with the ascendant wave of contagionism in early 19th century France, and although with different intensity, the British case seems to fit his view, though a different interpretation, focusing more on the dichotomy between predispositional and exciting causes than solely on contagion has recently been elaborated by Hamlin.[56]

4.3.3 Prichard's legacy: Hereditary Disease, 1830-1855

For the most part, as I said above, British medical men tended to accept Adams' and Prichard's main contentions about hereditary transmission until well into the 1850's.

The predispositional (latent) cause and some of its consequent signals, like atavism and homochrony, were repeatedly used as the basic criteria for identification of the hereditary in medical works. Among the most important of these were *Cyclopaedia* entries by Joseph Brown (1833) and Allen Thomson (1839); an excellent essay by Henry Holland (1839 and 1855); a "continentally"-influenced full-length essay by the German immigrant Julius Henry Steinau (1843) and Phillips' discussion on scrofula (1846).[57]

Most of these works found some echo in the medical journals and even in some more general periodicals. After Adams's *London Medical Journal* in the earlier part of the century, the *Medico Chirurgical Review* (1820-1824), *The British and Foreign Medical Review* (up to 1848), and the *British and Foreign Medico-chirurgical Review* (into the 1870's) reviewed and commented on most of these works, and published some additional relevant articles on the matter. There was not, as in France, a very big debate around the issue of hereditary disease because most of the commentators considered it too complex and muddled for it to be settled with the contemporary knowledge of physiology and of the laws of generation. But belief in the reality of the phenomena was widespread among the medics. The sceptics like Henning and Phillips were in the minority.

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[56]See E. Ackerknecht, 1967, *Medicine at the Paris Hospital 1794-1848*, p.160, and Hamlin(1992).

[57]J. Brown, 1833, "Hereditary Transmission of Disease" in Forbes' *The Cyclopedia of Practical Medicine*, vol.II, pp.417-419. A. Thomson, 1839, "Hereditary qualities, mental and physical" in Todd's *Cyclopedia of Anatomy and Physiology*, vol.2, pp.470-480. H. Holland, 1839 & 1855, "On Hereditary Disease", chapter II of *Medical Notes and Reflections*. -J. H. Steinau, 1843, *A Pathological and Philosophical Essay on Hereditary Diseases*. -Phillips, 1846, "The hereditary cause" in *On Scrofula*.

Among the most important elements of Prichard's and Adams' influence on all these writers was the identification of hereditary disease as another form of variation, as was described above. The model of a relatively malleable structure of the body that tend to reproduce a subset of its peculiarities in its offspring captured their imagination. The further idea that somehow the reproductive process (which gave rise to the hereditary phenomena) can distinguish between fundamental and acquired variations and only copy the former ones was resisted however by some authors. Brown, for instance, analyzed Prichard's insistence in the non-inheritance of diseased constitutions produced by postnatal (acquired) influences. Perhaps influenced by Henning's (or similar) criticism, he stresses the theoretical, non-observational nature, of Prichard's points regarding disease. Brought into medicine from natural historical considerations, and not natural to the field, the Scottish author's distinctions were indeed questionable on empirical grounds, he stated, as diseases introduced into families manifestly by contagion or climatic influence did persist in them several generations after, even if the exciting causes were no longer present. Brown also alleged that separating well described diseases (like gout or scrofula) into two different subcategories, one hereditary and another not, would be the consequence of such view; distinctions which would be quite artificial as the diseases would be identical in every respect except in their transmissability to offspring. In Prichard's favour he states that if we are to believe that constitutional diseases do in fact behave differently on the face of hereditary transmission, the situation would seem irregular as

> nature would appear to have instituted laws for the transmission of disease on two points, the opposite of those established regarding hereditary varieties in manifest structures.[58]

Adam's distinctions between family and hereditary disease, he adds, points to a possible solution to the conflict. Brown ends his short article advocating openness, and not dogmatism, in the discussion, as the evidence gathered was inconclusive on both sides, and much remained to be disclosed.

It is significant that the equivalence of normal hereditary varieties and pathological ones was no longer questioned for Brown or subsequent authors. They actually took it for granted, which means I believe that the domain of what we now call Biological Heredity was somehow stabilized by Adams' and Prichard's allegations, and the focus of attention was shifted towards regularities or irregularities that could be established from the available evidence. This unified approach is seen perfectly in the detailed treatement that Allen Thomson makes of the subjet in his Todd's *Cyclopedia* article on "Generation", under the double heading of "Influence exerted by parents on the qualities of their offspring" and "Hereditary qualities, the transmission of mental and physical phenomena from parent to offspring".[59] Bringing together, as Prichard had done, observations from Pathology, Natural History, Animal Breeding, Thomson describes the state of knowledge in this still quite irregular field. He follows Prichard also in emphasizing the causal events that act at conception as the really determinant factors for the hereditary. The inconclusiveness of many of the previous generalizations based on generation theories or old fashioned physiology were exposed by him, and at the same time the he selected and tried to organize the best evidence available in the mentioned fields.

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[58] J. Brown, op.cit., p.419.
[59] A. Thomson, op.cit., p.470.

On the "normal" side, Thomson describes the strength of the case for the transmission of different categories of characters: from general constitution (or complexion) to subtle peculiarities of internal and external structure, and some of their effects, like voice or idiosyncratic movements. Significantly, he pays special attention to the transmission of moral characters.[60] On the abnormal transmission, he backs the predispositional account based on constitutional peculiarities being transmitted, and relates them to the transmission of deformities.

As compensating influences to hereditary transmission of variation Thomson mentions, first the differential rate of variation in different groups, and in different situations, and second the tendency of mixed varieties to revert to type. Against Prichard he tries to argue that only special variations, that can constitute the origin of true varieties, can pass in hereditary descent, and be stabilized or fixed within a population, but does not give another criteria for their distinction. He describes latency effects like atavism in transmission of disease or other peculiarities. Like some of his contemporaries, he is somehow sceptical of the case collecting method of proving hereditary transmission, but at the end of his essay he indiscriminately enumerates a fair number of reported cases, from the fanciful to the serious, in an effort to show how profligate and confusing the evidential base for hereditary theorizing still remained. At the same time, in a paragraph reminiscent of Montaigne, he stated the need for detailed knowledge of the physiology of reproduction in order to make sense of the complex, irregular and unstable body of effects that has been accumulated

> It cannot but be a matter of wonder and extreme interest to inquire how, in the unformed germinal spot of the egg of the female at the moment when it receives the vital fecundating influence of the male semen, the disposition to the formation of those minute modifications of structure and function which constitute hereditary resemblances is capable of being retained and transmitted to the future offspring[61]

In trying to be exhaustive and without any theory to defend, Thomson describes ideas and empirical material, old and new, linked with the subject; like the influence of the mother's imagination, or the transitory emotional state of the parents at the moment of conception as a source of variation, etcetera. Although sceptical about their real importance, it is revealing —I believe— of the confused state of the subject that he decided not to exclude this material, some of it evidently outdated.

In 1843, an excellent revision on the subject of hereditary disease was published in London, somehow surprisingly written by an inmigrant German physician, Julius Henry Steinau. Having first appeared in Germany several years earlier and, according to its author, enjoyed a considerable success, an updated translation was prepared by him for English audiences with some urgency. He would have wanted to make a more

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[60]This is a development influenced by the increasing importance that the hereditary explanation acquired in the field of mental alienation, mainly in France, but also in Britain, where Prichard, again, had written the most lucid analysis. The attention to moral inheritance gathered momentum towards the middle of the 19th century until, in France, it overtook the complete field of hereditarian research with books like Prosper Lucas' *Traité de l'Hérédité Naturelle* (1855) and Theodule Ribot's *Hérédité Psychologique* (1872); in England the culminating point was of course Galton's *Hereditary Genius* (1869).

[61]idem.,p.470-474.

extensive research, he confesses, but a situation which he does not describe compelled him to publish soon. One can speculate that recent contributions on the subject, like Henry Holland's, which until recently had not been as popular in England as in the Continent, prompted him to join in sooner, and to leave the hard work for later. He promised a future volume on the subject if the first one was well received, but it seems never to have been materialized, although the first book was favourably reviewed.[62] Steinau's book constitutes an enriching event in the British scenario as it had its basic roots in the ignored Continental literature on the subject. One of its main influences was Joseph-Claude Rougemont's treatise, which, as I said, after having won the 1790 essay prize of the French Royal Society of Medicine was published in a German translation a few years later.[63] This richness of historical information and the complexity that Steinau brought to the discussion were unprecedented in English discussions of the subject. He gives accurate accounts of the whole catalogue of ideas and problems related to the hereditary, and criticizes what he takes to be the generalized dogmatism of most authors. The distinction between the hereditary and the innate, although acceptable in principle he finds difficult to uphold in the face of some evidence. The idea that only dispositions are transmitted and not the diseases themselves, he finds too restrictive. Adams' subtle distinction between disposition and predisposition he finds too subtle (there must always be an exciting cause he argues, however unnoticeable). He accepts, somehow contradictorily, homochrony as a working criterion for dispositional hereditary events.

It is Steinau's eclecticism which I believe made his book a good historical piece, but little convincing as a contribution to the debate. When discussing the deeper causes of hereditary transmission he restates the old quarrel between solidists and humoralists, but does not take side,[64] nor does he dismiss it as a surpassed issue. He states only that "the better physicians of modern times sought the proximate causes of diseases in the solid as well as in the fluid parts of the organism", and this leads him to the suspension of judgement that was becoming so common in his day

> till the physiology of the act of generation, as well as that of the first formation of the embryo, are more rightly understood than at present, no correct opinion can be formed of the first causes of hereditary disease.[65]

Perhaps, in the end, the most influential of all the texts on hereditary disease of the first part of the 19th century in Britain was the essay published by the famous physician Henry Holland in his *Medical Notes and Reflections* in 1839, and in an extended and revised version in 1855. In this essay Holland gives a very well balanced account of Prichard's views, and of successive developments. The renown of its author, and the prudence of his statements, made this exposition more influential than any previous one. Although a part of a miscellaneous volume, Holland's views on the hereditary were widely read and discussed among the medical community.[66] One of its most important readers was Charles Darwin, whose copies

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[62]For instance in the *British & Foreign Medical Review*, vol.XVII, p.247.

[63]See chapter 3, footnote 130.

[64]Although he does favour the solidist point of view that constitutional, chronic diseases, and not acute ones are those liable to become hereditary.

[65]See Steinau, op. cit., p.15; See the rest of his pamphlet for the details of what I have summarized.

[66]See for instance the review on *British & Foreign Medical Review*, vol.VII, p.484.

of both editions are carefully annotated, and only in this section.[67]

Holland begins his essay by recognizing that, for the case of humans, the best working generalizations on "the subject of hereditary temperament and tendency to disease" were achieved by Prichard. With his distinction between original and acquired modifications to the constitution, and their differing capacities of being communicated to future generations, the Scottish author set the ground for the exploration of such previously confused terrain. Although doubts and exceptions persist about the generality of the distinction, it will remain a great piece of theorizing because, Holland writes

> I know not that this principle could have been anticipated, or otherwise derived than from actual observation of facts ...as a general law, it may be deemed highly probable, if not wholly proved; and it is one fruitful of important inferences, both in physiology and in pathology.[68]

The existence of a unique field of research concerning the transmission of characteristics from parents to offspring in humans and, by analogy, in inferior animals and plants, was clearly a perception that Henry Holland had. And in considering the hereditary tendency to disease, he wrote

> whether arising from structure or less obvious cause, it is needful to regard it in connection with, or rather as part and effect of, that great general principle, through which varieties of species have been spread over the globe.[69]

As Prichard had shown, the study of variation within species and that of hereditary transmission were intimately linked. The same principles could be at work in both set of phenomena, and analogies could and should be used to illuminate one with the other, and vice versa. The relationship, that Adams and Prichard had stressed, between the existence of a tendency to reproduce structural peculiarities (and tendency to disease) in the offspring and production of regional varieties by the spreading and fixation of some of these peculiarities in geographically isolated groups, was an example of the fruitfulness of the approach.

Holland was, on the other hand, less pessimistic than many of his contemporaries about the possibilities of having some explanatory progress in the area, even if, as everybody acknowledged, in the end only a discovery of the actual physiological causes impinging in reproduction would eventually solve the fundamental mystery. The study of hereditary variation and disease can nevertheless proceed, he argued, because even in such ignorance we are still

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[67]These can be found in Darwin Library at the University Library, Cambridge. Darwin seems to have been searching for good examples of hereditary transmission of constitutional disease and other variations, and specifically for characters (or diseases) that remained latent either atavisms that could "jump" generations, or homochronic characters, i.e. that tend to appear at the same period of a life cycle.

[68]H.Holland, op.cit., 1st. ed., p.9.

[69]idem., p.11. It is important to note that Holland accepted as a general fact that there were limits beyond which variation could not go, and which constituted the unity and identity of the species. But added that under the state of knowledge in his days, no one was "entitled to deny the followers of Geoffroy St.Hilaire the possibility that it may be otherwise." (op.cit., footnote p.11)

> able to reason upon the effects, and to class them in certain relation to each other, and to the healthy and normal condition of the human frame[70]

Hereditary transmission of resemblance, of general external and internal constitutional features, of peculiarities of conformation, function and tendency to disease, are events that Holland situates beyond reasonable doubt. Instances of them are to be found, he says in many places, in common life, in history and books, but nowhere are they so common as in the practice of medicine. The exactness of modern observation has increased the detail and the persuasiveness of the occurrences, and brought to light a collection of "wonderful instances in which the most minute peculiarities or defects, in structure and function, are transmitted from one generation to another", and in that manner it has reduced the strength of sceptical allegations that want to attribute the similarities to external sources of causation.

In accordance to the general tone of his essays, Holland chose to describe cases of family-related diseases with which he was personally acquainted. He describes in some detail the peculiarities of the defects, functional or structural that he found were transmitted hereditarily, and considered their relative frequency, their tendency to be modified or to disappear between generations, the correlations between different hereditary diseases, and other matters connected to the theme. After considering different organs and functions, Holland summarizes

> Seeking then for the most general expression of facts, we may affirm that no organ or texture of the body is exempt from the chance of being the subject of hereditary disease. Or, in other words, every part is susceptible of deviations from the normal type or natural structure, capable of being conveyed to offspring[71]

Holland includes in this generalization, though with some reserve, the structural malformations that are usually called monstrosities, and in the second and third editions of his work he pays particular attention to the claims that madness and other moral qualities could be hereditary, if based on some structural flaw of the nervous system.[72] He also makes a point of not being too dogmatically solidist in his approach, and in the case of this position's attacks against humoralism he writes

> We can go little further than to say, that the evidence as to the agency of the solids, and the changes they severally undergo, is more distinct and complete; but we are not justified in denying that the blood may also take on morbid conditions, directly transmissible to offspring. ...So close is the mutual connection of the animal solids and the blood, in growth, function and change that it is scarcely possible, to separate them in this inquiry...it might fairly be argued that however difficult to conceive a fluid like the blood, ever in motion and change, being capable of hereditary taint, yet this is not really more difficult to understand than a

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[70]idem., p10.

[71]idem, p22-23.

[72]To his credit one must add that Holland is an exception in that he does not succumb to the easy moralizing attitude that led most medics that dealt with the issue to "eugenical" propositions, based on scare mongering about the decline of families and nations due to proliferation of degenerate types, etc. Although he does state that the study of hereditary transmission surpasses, in its importance, the limits of the medical or purely scientific.

> character or peculiarity conveyed by descent to any part of the solids of the body[73]

Imperfect physiological knowledge blocks then the aspirations of both, rather old fashioned, camps, as distinctions cannot be pursued to the detail. Holland adds in the 1855 edition that, however, "in this, as in other sciences yet unformed" it will only be through better observations and "truly determined" facts that "correct classifications and future laws" will be found.[74]

Such a recent area of knowledge, so full of irregularities and undetermined laws, in close relation to so many striking instances of both positive and negative cases for the few generalizations available, Holland believes, is for the most part still an open-ended field of inquiry. This openness is summarized I believe by the telling, paradoxical commentary which, as we mentioned in a previous chapter, was picked up by Darwin from Holland, in the sense that as much wonder is produced by the transmission of resemblances from parents to offspring, as wonder produces the fact that such communication is never perfect and allows for alls sorts of variations and grades.[75] The phenomenological couple of hereditary transmission and variation, among humans as well as among animals and plants, were in the 1840's and 1850's asking for a theoretical synthesis, of some kind or another. What Henry Holland was hinting at was also perceived by several other authors. The French hereditarians, with their peculiar approach to theory were by then, already producing such a synthesis.

Still alive however, was the sceptical challenge to hereditary transmission of peculiarities of body and mind. Henning's initiative in this direction, as I said, was retaken by Phillips first, and by the historian Buckle later. The main thrust of this questioning was summarized in a long footnote within Buckle's *History of Civilisation* (1857), and consisted in a call for a better, statistical organization of evidence, and wider empirical research of hereditary transmission; together with a curb on loose, old style speculation. This challenge, as R. Olby has shown, was to play an important role for the following (post-Darwin) generation of Hereditarians.[76]

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[73] idem.,p.16.
[74] See Holland, 3rd. edition, op.cit, pp.26-27
[75] idem.,p.24.
[76] See Olby (1985) and his "Galton's response to Buckle".

Chapter 5

Heredity in the Making: pathological, physiological and moral Heredity in post-revolutionary France

We have shown how, at some point in the early decades of the 19th century, French medical men and physiologists began using with increasing frequency the noun "hérédité" as the carrier of a structured set of meanings that defined and unified a general (biological) domain of reference. A field whose elements had previously been loosely connected, to and fro, by analogies from the medical, zoological, horticultural, ethnological, etcetera. The new domain was to become that of general biological heredity.

After 1830, when it was an accepted concept, "hérédité" was usually qualified by different adjectives that aimed, in the French analytical manner, at elucidating oppositions. The most common of these was "physiologique" and "pathologique"; the most influential "physique" and "moral". The first pair was used to mark the division between what we now call normal and pathological, and it served to define the axis through which the structure of the medical concept of hereditary transmission was transported into a general, biological frame. By structure of the concept I mean the cluster of classificatory and explanatory elements that the medical disputes I have analyzed above produced. The distinction between innate and congenital characters, the latent and dispositional nature of causation, etcetera.

Around the second divide were centered the most important discussions of how "l'hérédité" worked, as different physiological ontologies struggled to take over the domain. Being a quite localized development, this process of structuring of the domain of heredity had as its main protagonists French physicians, alienists,[1] physiologists and naturalists of the troubled post-revolutionary decades. With the publication of Prosper Lucas' remarkable *Traité de l'Hérédité Naturelle* (1847-50) the process was somehow concluded. In spite of Lucas' choice of what, to us, were the losing horses of a dualistic physiology and a confused inductive procedure, he managed to organize and structure the realm of hereditary transmission in such a way that most readers, of whatever persuasion in other issues, could profit enormously. A very detailed work of analysis and theorization, based on a very extense bibliographical research, Lucas' oeuvre helped to expose the weaknesses of the medical tradition's approach to the hereditary, but also the great wealth of evidence and understanding that lay in there, hidden to other eyes.

The present chapter will describe the movement of structural elements from

[1] *Aliéniste* was the name given in France to the early psychiatrists. As they dealt with mental alienation, or insanity. The word for insane was *aliéné*.

pathological to physiological heredity, and the input to this provided by some natural historians as they addressed the problem of variation and race. It will also show how the dispute between materialist and dualist physiologies in the problematic field of "moral" heredity (and alienism) was one of the main causes of the growth in importance of the whole issue of biological heredity in the course of the century. Finally, it will describe and criticize Lucas' work in relation to both previous and posterior authors.

5.1 Heredity, old and new

The old medical concepts of temperament, complexion and constitution, which had been used, and refurbished through the different periods according to dominant physiological creeds (Hippocratic, Galenic, Paracelsian, mechanistic) had been for centuries the depositaries of underlying, general potencies and dispositions that could account for both typical and idiosyncratic responses of individual organisms in given situations. By the same token, in all these periods the moral peculiarities of human beings had accordingly been linked or separated from the constitutional or temperamental, mainly on the basis of the coherence between theological and physiological ontologies.[2] Late 18th century medics, when the pendulum was moving towards materialistic approaches, saw again in the medical concept of constitution a good framing device for their attempt at reducing the moral to the physiological. The hereditary provided a link, at first metaphorical and then substantial, between parents' temperaments (or constitutions) and those of their children; that connection, extended over time to whole genealogies, justified the talk of family, or even national characters. Physicians, we saw before, had for many years put to use such link for the explanation of familial patterns of a whole set of etiologically difficult chronic ailments, which were also generically known as "constitutional". Among them insanity, epilepsy and other mental abnormalities were usually included, but not particularly stressed.[3] As has been pointed out, the 19th century brought a new generation of physicians who began to apply in its full strength the idea of a deep dependence of the mental on the body's constitution or physical organization, and at the same time emphasize its hereditary implications. Both positive (or normal) and negative (or pathological) sets of moral qualities[4] were subsumed under this scheme. When hereditary transmission was considered, mental illness gave the most well known examples to support such beliefs, but cases of normal or positive moral traits running in families had also been considered by

[2]Jacques Roger has analyzed this in the French context from the 16th to the 18th century in *Les Sciences de la vie* ..., 1964.

[3]The hereditary character of mental diseases had been a characteristic observed and discussed at least since Hippocratic times. In the Ancient Greek medical corpus it is in discussing epilepsy, the sacred disease (*Morbi sacri*) that some of the clearer passages concerning the hereditary and its relation to generation, is to be found. See for this Lonie (1981) and M.Boylan (1984).

[4]The ancient category of the "moral" included all the particularly human qualities, those that could be described as the emotional, the intellectual, and for some the instinctive. The discussion of hereditary moral qualities, during the 19th century, were split between the mental (or, later, psychological) and the instinctive.

authors from the medical, historical and religious traditions.[5] Both the idea of hereditary degeneration, and that of hereditary improvement of the human stocks (many times hinted at or explicitly mentioned among earlier medical writers on hereditary diseases)[6] acquired a greater relevance and very soon became central to ideologically charged theoretical reformist enterprises like alienism and phrenology.[7] As I said, there have been several successful studies of the expansion of hereditarianism into different areas from the 1840's onwards, however, the dramatic shift in emphasis, during the first few decades of the century, from the hereditary as an important but secondary (predisposing) component of many physical and mental conditions, to heredity as a main (if not the main) cause responsible for them has not been adequately explored.

Such a shift, it seems obvious to me, came with the French Revolution, or to be precise with the Napoleonic reforms. As this is not a sociological investigation I will not get into the details of that aspect of the story, but will point out some of the evidence. The most notorious is the change of emphasis on the hereditary among some authors that produced works both previously and after the Revolution. Three of them were F.E.Fodéré, A.Portal and P.Pinel, all of them very influential in the post-revolutionary reforms. They all mentioned the hereditary cause in early works, but only developed stronger hereditarian stances during the first decade of the 19th century.[8] A main feature of the new position that these reformers adopted was their simultaneous defense of hygienic and hereditarian views concerning some of the most urgent ailments of French urban life. Perceived as based on antithetical explanations of the origin of disease, some authors have seen the strain of compromise in the stance adopted by them.[9] The fact is that all of them managed to justify the apparently contradictory stance, and defend simultaneously strong hereditarian views (with their often fatalistic undertones) and hopeful, hygienist reform programs.

Antoine Portal (1742-1832), we have seen, summarized in 1806 his humoralist, protean, and somehow regressive view of hereditary influences, and rather dishonestly dismissed the importance of the contributions to the subject by contemporary authors, such as Rougemont and Pagès, whose solidism was more in tune with the physiological knowledge of the age.[10] Portal was, with Pujol de

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[5]See for instance the Rev. John Adams' "On the Hereditary Genius of Nations" in his *Curious Thoughts on the History of Man* (1789), or the polemical "Letters to William Wilberforce on the Doctrine of Hereditary Depravity" by a "layman" (Dr. Cogan), 1799, London, J.Johnson.

[6]Clear statements of some proto-eugenic ideas can be found in Pujol de Castres (1808), J. Adams (1814), and Lereboullet (1834).

[7]Here the analogy with the breeders improvement of their stock, or the farmers and horticulturists practice of selecting the seeds were once and again brought into the fore as powerful analogies.

[8]See Fodéré on Goitre (1792), Portal's many works on Apoplexy, Scrofula and other chronic ailments (1781, 1800-1825), and Pinel's pathological and early nosographical works (c.1785).

[9]See Ackerknecht, 1967, p.160.

[10]See Portal's mean assertion that the contributors to the 1790 Royal Academy contest did not advance much the knowledge on the subject.(op.cit.) Castigated by Adams in England, and by Petit in France, Portal's work, together with Pujol's contributed to keep alive the non solidist, protean approach to the hereditary, which

Castres, the last strong defender of an explanation of hereditary disease that was soon to become anathema. The humoral causes had the advantage that they made family diseases seem "curable". Based on postulating the existence of external influences (germs of disease, or taints), that manage to invade the generational process, humoralist heredity can more naturally share the stage with a hygienist position that aims at eliminating such vices from the families of the nation. Solidist hereditarians cannot accommodate their views so easily with hygienic trends, but they have the resource of their distinction between predisposing and exciting causes. They can claim that heredity only predisposes, but exciting causes can be blocked by external intervention and the effect never materializes, remaining latent.

F.E. Fodéré was in his early works closer to a solidist position, but it was in his post-revolutionary oeuvre that he overtly adopted such a view. Possibly influenced by some of the works that resulted from the 1790 Royal Society's competition, Fodéré gave a boost to the constitutional, organizational view of hereditary transmission, and was highly critical of humoralism. Judged by the number of references tha his hereditarian views received later, and the number of his students that adopted them and who became influential in different fields, both his lectures and his writings seem to have played a substantial role in the spreading of what soon became the new medical hereditarianism of the early 19th century. His major contribution, the 6 volumes of a *Traité de Médecine Legale et d'Hygiène Publique*, is carefully oriented to both fulfill the demands of "Codes de l'Empire Français" and to make justice to the medical knowledge and tradition. He carefully divides family diseases (which Portal confuses) between contagious and non contagious ones, and recognizes that the latter, whose existence depend on organizational flaws that predispose different parts of the body to failure, are the most difficult to understand and combat.[11] Fodéré stresses the need for profuse genealogical investigations, as the ones he made on goitre in the Alps,[12] in order to determine how strong is the case for hereditary disposition, instead of only relying on ancient literature and on a few scattered cases. Information extracted from scrofulous, phthisic, insane and other chronically diseased patients (or their relatives) should be organized, preserved and published, he believed, in order to establish the strength of the disposition (or diathesis) and the nature of the exciting causes.

Fodéré was also the first French author (after Pagès) that saw with some clarity how the logic of latency as determined by the division between dispositional and exciting causes, was the way towards a generalized view of heredity. The peculiarities of the hereditary that since antiquity had both mystified numerous authors, and had helped them establish criteria to recognize its actions, stemmed logically, he wrote, from this dual causation. Phenomena like atavism (the skipping of generations by certain characters) and homochrony (the sudden appearance of the same effects at the same age in the different members of the same family) both were due to the necessity of complementary exciting causes. In the first case they failed to concur with the dispositional cause in one or more generations, and in the second case the exciting cause tended to be related to the initiation of a particular period in the development of

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seemed doomed at that stage, but which was later revived by vitalistic, over-enthusiastic hereditarians in both France and Britain.

[11]F.E. Fodéré, 1813, "Des maladies héréditaires", in tome 5 of *Traité* ..., pp.358-382.

[12]F.E. Fodéré (1792), *Essai sur le goitre et le cretinisme,* 290p., Turin. Reedited in Paris, as *Traité de goitre* ..., 1800.

the individual, like childhood, puberty or senescence.

After having left aside the issue in the early part of his career, in his post-revolutionary days, the famous alienist and reformer Phillipe Pinel (1745-1826) adopted a somewhat similar stance as that of Fodéré with regards to hereditary influences.[13] Though a disciple of the Montpellerian vitalists, Pinel had come to accept Cabanis' postulate that all mental phenomena were based upon a physical structure, but his materialism was not as mechanicist as some others. Perhaps due to his interest in the complexities of the deranged mind, Pinel was never a reductionist, and chose to side with Cabanis and the other idéologues whose materialism was not reductionist and that considered "emergent" properties as a possibility.[14] Pinel acknowledged the importance of the fact that mental disease runs in families, but never particularly stressed the hereditary influence. Not until his famous disciple J.E.D.Esquirol began to push in that direction, in the first decades of the new century. This reticence had another root in his strong belief that moral causes (which belonged to the realm of the exciting, not the hereditary) were by far the most important in the onset of mental disease. His reforms to the asylums, and his celebrated introduction of moral treatment for insanity, ran then in opposition to giving heredity too strong a role, and so he left it in most of his writings as a background element.[15] He nevertheless did encourage Esquirol to pursue the statistical analysis of hereditary influences in insanity.

The shift in the emphasis that these three authors made in the late part of their careers towards giving heredity a central role is, I believe, a clear sign of the times. The hereditarian wave gathered momentum, and with the exception of isolated cases of scepticism,[16] the French medical community seems to have been finally overtaken by it by the beginning of the 1830's.

The relative speed with which the conversion of heredity from a marginal "turn of speech" into an important explanatory tool within the biological disciplines happened remains to be better understood. Among the several factors influencing the process a few more can be mentioned, besides the post-revolutionary reforming zeal.

It seems clear that the materialistic traditions stemming from Enlightenment French *idéologues*, like Holbach, Condorcet or Cabanis, played a major role in the medical community's shift towards a redefinition of constitution, and its dispositions; and this in turn forced a revaluation of what was meant by hereditary transmission that favoured the solidist side in the 18th century dispute. The idéologue programme can be summed up by the title of Cabanis' celebrated treatise *Rapports du Physique et*

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[13]"Il serait difficile —he wrote— de ne point admettre une transmission héréditaire de la manie, lorsqu'on remarque, en tous lieux et dans plusieurs générations succesives, quelques-uns des membres de certains familles, atteint de cette maladie" quoted by Semelaigne (1894),p.65.

[14]See the excellent study by M. Staum on *Cabanis* and his group (1980).

[15]See Pinel, *Traité de la Manie*, 1806.

[16]An important sceptical argument was made by Sersiron (1836) who maintained that true hereditary transmission of disease ought to be as "fatal" and deterministic as the hereditary transmission of specific characters, like the shape of the bones or the form of the eyes. Any accidental character can disappear from the genealogical line after a few generations, so it cannot properly be claimed to be affected by the same hereditary cause that maintains the unity of the species. Their transmission is therefore also accidental and not lawfully governed, he claimed.

du Moral de l'Homme,[17] which set the agenda for a whole range of investigations purporting to find the underlying physical and physiological bases to even the most particular human characteristics, those under the domain of the moral. Even if there existed special properties or qualities in mental life, they had to be rooted in physical portions of the body, they believed. How this connection between the two realms was to be understood was not a question over which all *idéologues* agreed.

Several historians have identified these movement as responsible for triggering, in the moral sciences, the impulse towards materialist explanations in the early 19th century, and hereditary explanations filled the requirements.[18] Among the followers of the idéologues there were authors whose main interest was the development of scientific approaches to the human condition, like phrenologists, alienists and some early anthropologists. Perhaps the most important publicity factor in the boost that hereditarianism received in those early decades of the 19th century was its adoption by phrenologists and medical alienists. The case that hereditary transmission of moral characters was popularized in the wave brought about by the new science of "organology" or "phrenology" that crossed France and Britain between 1809 and 1820 has been insightfully argued by Victor Hilts. He describes how both Gall and Spurzheim, using a materialistic stance emanating from the idéologues, underpinned their reductionistic explanatory pretensions by adopting an hereditarian view. Based on the solidist view of human bodily constitution, and the claim that it is strongly influenced by the parents', the phrenologists' case, they believed, was made stronger by the medical tradition's collection of cases in which many kinds of mental characters and dispositions had been proven to run in families.[19] The idéologue style materialistic reductionism was taken to an extreme when Gall and Spurzheim defended the position that mental characteristics were not only based on dispositions given by the person's constitution, but actually these dispositions could be read out of the shape, form and size of the person's head (and with more detail in dissected brains).

But as Hilts correctly points out, the phrenologists did not at first consider heredity as a central tenet in their program. It was only a complementary element given by the medical (solidist) base of the theory. Gall cleverly used the dispositional causation of innate qualities that medics had employed to defend the reality of the hereditary, but was not particularly interested in hereditarian explanations *per se*. Spurzheim was the one who saw the "social" applications of the hereditary postulate, because an interventionist (therapeutic) programme could be based on them. But it was not, I believe, the direct use of the hereditary made by phrenologists, but the reaction they generated amongst alienists and other medical men, that really influenced the destiny of heredity as a generic cause, as they were somehow forced to sophisticate, theoretically and physiologically, the meaning they gave to the phrase "hereditary predisposition". The phrenologist Spurzheim, for instance, started using a simplistic view of character transmission under the vague formula "laws of propagation" (1821) and only in later days he changed it to "laws of hereditary

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[17]In P. J. C. Cabanis (1802), *Oeuvres* ..., pp.105-631.

[18]For recent accounts of the influence of this group of thinkers in the biological scene of the 19th century see M.Staum (1980), *Cabanis*, A. Desmond (1990),*The Politics* ..., pp.42-43. See also J. Schiller's excellent "L'organisation chez les philosophes du siècle des lumières", chapter VI of *La notion d'organisation* ..., 1978.

[19]See Victor L. Hilts,(1982) "Obeying the Laws of Hereditary Descent", 1982.

descent".[20]

In general it can be said that another reason why hereditarianism grew in the early 19th century, is that there was a reaction to 18th century exaggeration of the external influences on the constitution of human and other species' bodies. At the same time, developments in chemistry, anatomy, and physiology, had made external (climatic or other) shaping causes increasingly difficult to maintain, so a need to emphasize internal causes of resemblance and homogeneity between species, races, and other groups, was felt. Besides that, there was growing evidence in favour of a stubborn persistence of type and character within genealogical groups, even after several generations of changed circumstances. Typical examples for anthropologists were those of populations of European origin in tropical lands, or for biologists those of animal and plant species brought from other continents into Europe. As it happened, counterexamples also existed, and so cases could always be picked to back externalist (climatic or other) explanations of both variation and resemblance.

The anthropological work of J.C. Prichard, as we have seen, blended together different, though related empirical phenomena, and he was among the first authors to realize that a kind of general description of the behaviour of inheritable characters through the generations was possible and necessary. In the French scene however, for reasons external as well as internal to science, generalizations about heredity acquired, sooner than in the rest of Europe, an urgent character. The link that the French alienist movement forged with the explanatory power of heredity in the human species was probably the most important cause for this.[21] This link in its turn was only possible due to the great amount of groundwork that the previous generation of French medical men had already done.

5.2 From pathological to physiological heredity

The possibility of the existence of a field of scientific research that focused on the transmission, through physiological media, of general and peculiar characteristics from parents to offspring was, surprisingly, one that only acquired some clarity in the early 19th century. Such a possibility did not emerge suddenly but was shaped in an intellectual environment where the phenomena of hereditary transmission in different species, but particularly in humans, acquired a relevance it never had had before. A clear symptom of this is the increasing number of references to the workings of *l'hérédité* in ever widening contexts. From doctors to naturalists to physiologists to journalists to novelists, the noun increased its currency, and gathered an increasing weight, in the course of the first three decades of the century. The effect of separating from other medical and biological phenomena those of the most striking resemblance between relatives, and accounting for them uniformly as influence of parents' constitution over that of their children, and at the same time reclustering them under such a sonorous and evocative noun, was in the end to create an impression of certainty, precisely the effect that directly experienced evidence has. The recirculation of cases of hereditary transmission of curious details, mentioned in ancient authors,

[20]This is a very similar shift as the one detected in Prichard's oeuvre, were the clear-cut formulation "The laws of hereditary transmission of peculiarities of structure" only appeared in the second edition of *Researches* (1826).

[21]I. Dowbiggin's recent book (1991) *Inheriting Madness* provides an analysis of the role of alienists in the surge of French hereditarianism.

such as Hippocrates and Aristotle; as well as that of all kinds of astonishing cases of hereditary passage of anomalies and curiosities from collections made through the centuries remained a common practice;[22] and their mention in the same breath as the latest results of plant hybridizations or last year's cases from a Parisian asylum, still puzzles many historians.[23] It indicates however, among many things, the extent to which the facts of hereditary transmission, suspiciously regarded in other centuries, were becoming accepted as a given, and how they were helping to draw the boundaries of the domain under construction.

By the 1830's "l'hérédité" as an explanatory resource that needed little or no introduction was common among many medical authors, and an increasing number of other authors outside medicine were making use of it; mainly physiologists and naturalists. It is important to realize that although the communication of peculiarities from parents to offspring through generation had been referred to in different contexts by naturalists and breeders, among others, before the period in question, never before had a concept, like the physicians' hérédité, sought to cluster under it all the similar phenomena associated with it. Hérédité's medical use was thus extended and generalized after the 1820's in the works of medically trained naturalists like Virey, breeders like Girou de Buzareingues or physiologists like Pierre Flourens.[24] Heredity was more and more a common place, and on its way to becoming a common presupposition among French natural scientists several decades before the same phenomenon occurred in other European countries.

It is no wonder then that the French milieu could transform its 18th century medical disputes around "les maladies héréditaires" into a widespread theoretical and ideological preoccupation with the workings of l'hérédité.

To recapitulate: France, in the last decade of the 18th century, and specially early years of the 19th, saw the publication of many more treatises, essays, articles, and dictionary entries on "hereditary diseases" than had ever been published before on the subject. Pagès, Pujol and Rougemont published their competition essays, and they were joined by a succession of very authoritative works, like those Antoine Portal, Antoine Petit, F.E. Fodéré.[25] Hereditary causation was with increasing frequency emphasised in general pathologies and in treatises concerning the main chronic diseases.[26] With some reluctance, but unequivocally, the famous alienist Pinel had

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[22]The tradition of gathering attractive, striking, and curious cases from all sorts of sources and presenting them in the form of collections lasted into the 19th century. Among its practitioners were eminent authors such as Haller, Blumenbach, Meckel. Prosper Lucas, as we shall see below, greatly profited from some of them.

[23]It is of course an old rhetorical resource of physicians the appeal to cases and evidence authenticated by the highest authorities, no matter how ancient and dubious. On the other hand, the failure of some modern historians, like Bowler (1989), of seeing in this a positive historiographical sign has precluded a proper understanding of the importance of human (medical) heredity in the shaping of the general biological domain.

[24]See for instance Virey, *Art de Perfectionner l'Homme*, Paris 1808; Burdach, *Traité de Physiologie*, Paris, 1837, trd. A.L. Jourdan.

[25]See references in Appendix 1. See Fodéré "maladies hereditaires", 1813.

[26]See Fodéré on goitre (1790) and on phthisis (1809), Baumes on phthisis (1805) Portal on apoplexy (1781) and on scrofula (1800), and J. Poilroux, *Nouvelles*

acknowledged the importance of hereditary predisposition in the onset of mental illness, and very soon his followers enthusiastically took to exploring the theme. Among them Esquirol and Fodéré, who promoted the hereditary influence from the back row of minor contributors to madness, to the forefront of one its main predisposing physical causes.[27]

The workings of the hereditary had always been closely related to the medical conceptions of constitution and temperament, or more precisely to the way these influenced or predisposed the individual's body to behave in given manners. As we described above, the transition from humoralism to solidism within medical physiology, during the 17th and 18th centuries, had the explanation of hereditary diseases as one of its battlegrounds, and there were still representatives of both approaches in early 19th century France. This had the consequence of provoking some tension when French medics (and later on alienists) sought to unify their views under the common cause of general (pathological) hérédité.

Besides the nature itself of the hereditary cause, the most important points of disagreement between the two positions were, as we saw before, related to the exclusivity of both the cause, and of the kind of causation. Humoral causes tend to be seen as protean (causing different diseases) and essentially invasive or external. They are described as a taint, a hue, a germ; something removable by the physician's intervention. Solidist causes tend to be localised and exclusive (the child inherits the same predisposition in the same part of her organization), and not essentially different from normal hereditary causes. The physician's intervention is more restricted under such a view, and tends to be seen as limited to the avoidance of the exciting causes that trigger the predisposition. As Lereboullet wrote in his insightful historical introduction to his *De l'hérédité* (1834), for the most part, the existence of taints or the like was increasingly discredited by the beginning of the 19th century, but the humoralist position still found strong supporters. Perhaps the most influential of which, Antoine Portal, (anatomist at the Jardin des Plantes) tried to reduce all hereditary diseases to the action of the scrofulous vice, as he called it.[28] The solidist view of the hereditary, with its emphasis on the idiosyncratic differences communicated simply through the resemblance (or reproduction) of the parents' organizational traits in the offspring, was favoured by most other physicians in the early years of the century. The anatomists had been augmenting for decades the list of internal bodily resemblances (not necessarily pathological) that could be claimed to have an hereditary component, as they were seen to "run in families". This strengthened the case for the solidist cause. It was repeatedly claimed by hopeful solidist hereditarians that soon no detail of the bodily frame (constitution) could be considered out of the aegis of the hereditary cause, and most constitutional diseases

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Recherches sur les Maladies Chroniques (1821). See also Caillot's (1819) *Traité de Pathologie Generale*, tome I, p.478.

[27]See Pinel's article "Aliénation" (1812) in vol I, *Dict. des Sci. Med.*, Fodéré's *Traité du Délire* (1817), and Esquirol's article "Manie" (1820) in vol. XXX, *Dict. des Sci. Med.* ,pp.437-472.

[28]Portal's examples of hereditary proteism (diseases changing into different affections as they jumped through the generations) were later among the main influences of degeneration theorists.

would be found to have their predisposing seat in anatomical details.[29] The dominance of this optimistic approach seems to have been short-lived. Such a reductionistic view of constitution and constitutional predispositions prompted reactions from several quarters, not least of all from physiologists. By the 1820's and 30's a whole range of non anatomical explanations for hereditary dispositions were already common again. As I will argue in the following section, one of the reasons for the mistrust of the reductionist approach was the crudeness of some materialist hereditarians, like the phrenologists, which outraged many physicians (and specially alienists) with their claims that they could "read" mental dispositions from physical features. As part of a "backlash" against anatomically based reductionism and in favour of functional, property-based accounts of constitutional dispositions, not to be revealed by the crude anatomical details, but by the effects it can produce, a new, property-based "emergentist" account of hereditary transmission was defended. The disputes within physiology were more relevant than any other in the determination of how to understand hereditary phenomena, and the alternative options of a structural or a physiological (functional) basis for accounting for constitutional dispositions in general, and for inherited ones in particular was to be a main feature for the rest of the 19th century discussions about heredity.

The outcome was that the general term physiological heredity became accepted as referring to the normal mechanism by which bodily resemblances are transmitted through the generations. Pathological heredity was then to be seen as based on the same principles, but having as object the transmission of deviant particularities that predisposed to disease. This "naturalization" of heredity produced new focus for dispute, as different physiological ontologies began competing for the description of how hereditary transmission comes about, and in which way it has under its control the different kinds of bodily and mental characteristics of individuals, of families, of national or ethnic groups, of races and of the species.[30]

For physiological (biological) heredity to acquire the high profile it attained during the middle decades of the 19th century in France, and later in the rest of Europe, the interests of different groups had to coincide. That biological heredity did not burst out spontaneously, completely formed as an explanatory concept, or as a domain of inquiry, is not often clearly appreciated. Neither did it have a long and featureless pre-history going back to pre-socratic times. Although several early 19th century authors claimed, for rhetorical purposes, that there was an uninterrupted tradition of "normal" hereditary theorizing, going from Hippocrates and Aristotle all the way to Buffon and Haller, that also included a long list of famous medical authors, the truth is that biological heredity as such had never been at the center of any biological (theoretical) enterprise.[31] Only in the medical tradition, with its concern for hereditary diseases, had the fact of transmission and its causal complexities really been seriously addressed. And it was as an overflow from the medical community's preoccupation that the theme began to generate increasing

[29]See for instance Pagès (1796), and Petit (1817) for strong critiques of the humoralist position, and a defense of organic or solidist causes.

[30]Physiologists like Burdach (1823) and Flourens (1848), were among the most influential to take on the task of arguing for a general physiological account of heredity.

[31]For such an interpretation see Pujol (1808), Portal (1813), Lereboullet (1834), Béclere (1845), and specially Lucas (1847-50).

activity and reflection by authors interested in more generalized approaches.

It was, so to speak, a movement in several directions. For instance, in the same way as Prichard in Britain had grasped the possibilities of putting the medical concept of the hereditary to use in the explanation of the origin of human, and other species' varieties, some French naturalists and physiologists were making a similar move. After Lamarck, Virey first and then Flourens (1848) were among the most important early promoters of heredity as a theoretical concept in natural history. Perhaps the failure of several 18th century grand schemes of Natural History, that had tried to imitate Newtonian Physics by postulating the existence of basic forces that could account for the mysteries of life, was one of the reasons why increasing numbers of natural historians and physiologists turned their attention to the rather marginal explorations that a subset of medical men had kept going. As an easily graspable metaphor that physicians had been employing since antiquity, part of the seductive power of the hereditary, was that it seemed a promising and ready-made theoretical device. With its capacity to concentrate the mind on simple external examples of resemblance and reccurrence, at the same time that it left room for accommodating different, even conflicting views on physiology or generation, heredity showed that it could have a life of its own.[32]

Although, as has been described above, the facts of the hereditary[33] were addressed by several of the major 18th century biological theoreticians (like Maupertuis, Buffon, Haller, Bonnet, Wolff, Blumenbach) they did not represent for them but secondary empirical obstacles that their speculations had to save. Early 19th century authors began however to give the idea of hereditary transmission of both generic and individual characters an increasingly central role. Prichard and Lawrence in Great Britain; Meckel, Gall and Burdach in the German speaking world; and in France, Lamarck, Virey, Flourens, Esquirol, and de Buzareingues, [34] Most of these authors had a medical training, and it is more than likely that the main spring of the "hereditarian surge" of the early 19th century took place, before anywhere else, in the classrooms and lecture theatres of the Medical faculties, from Paris to Berlin, from Montpellier to Edinburgh, during the last decade of the 18th and the first two decades of the 19th centuries.

Shortly after the French revolution, and with increasing security, medical men began to privilege the hereditary cause as a main predisposing factor for many important diseases like phthisis, scrofula, gout, etc., and specially, as it was to turn out, for insanity, epilepsy and other nervous disorders.[35] More and more pathological

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[32]From its medical cradle, the rhetorical power of the hereditary was to conquer and dominate for over a century the imagination of wide sectors of the French intelligentsia, and its infecting profligacy was to overspill, through psychology, sociology, literature, hearsay and journalism, into the shapeless ocean of public opinion, and what some French authors call "the collective imaginary". See J. Borie, *Les Mythologies de l'Hérédité*, 1981. Y. Malinas, *Zola et les Hérédités Imaginaires*, 1985.

[33]Which as was said can be divided in three main sets: family resemblance, hereditary disease and re-occurrence of deformities.

[34]All published between 1800-1830.

[35]A convergent account of medical theory developments in the period, with regard to predisposition to disease, was published recently as "Predisposing causes and Public Health in Early 19th-century Medical thought", Hamlin (1992).

dissertations, specially within French medical schools, began to have chapters discussing the hereditary influence, and also an increasing interest was shown to follow the trail of Pagès, Pujol, Rougemont and Portal, Fodéré, etc., in discussing in general the main characteristics of hereditary diseases.[36] As I wrote in chapter 1, at some point the formula "sur les maladies héréditaires" was supplanted by "sur l'hérédité dans les maladies", and this was a sign of a changing attitude towards the phenomena themselves. They were being brought from the backstage of evidential support for this or that generation or pathological theory to the forefront of uncontestable and urgent data about life and humankind. The use of the noun "hérédité" could, for sure, be found occasionally in some medical writers of the late 18th century, like Pujol and Amoreux, Fodéré and Portal; but only as a short formula for hereditary transmission, or as a way to referring to the inherited thing itself, but never as an abstract noun referring a general cause, as began to occur later. With less frequency, naturalists and moralists also employed "hérédité" in pre-revolutionary times, but always with strong metaphorical connotations, and not infrequently associated with degeneration, disease and moral decay.

Only after a couple of decades of the 19th century had passed, "hérédité" began to acquire a life of its own as a generic term. As it began to be used with increasing confidence by French doctors and biologists, it somehow began to loose its purely pathological meaning and to be recognized as the abstract locus of what soon came to be known as normal, physiological heredity: A candidate for biological nomological status. At some point, not only the fact of transmission of characters from parents to offspring was being stressed, but the possibility of having a generalized conceptual scheme that could make sense of the irregular patterns of occcurrence and reccurrence of different kinds of characters, that observation had shown within genealogical lines in humans beings and other species. For it soon became generally perceived that the analogical relationship between hereditary transmission among animals and humans could be further strengthened. The simple fact that there was at the time a strong belief in hereditary transmission of a whole range of characters among other specialists, like animal breeders, gave the human hereditarians a powerful card.[37] A similar reinforcement came from the camp of plant hybridizers, who were showing how subtle intraspecific variations could be manipulated through crossings to produce varieties. The discovery for instance that atavistic and similar hereditary patterns, based on latent or hidden causes, occurred in plant hybridizations, was seen as strongly reinforcing the case for a general approach to the issue.[38]

The first attempts at a general statement on hereditary transmission were thus linked to the growing popularity of the "theme" in the early 19th century, and this popularity can be attributed with confidence to a growing awareness of the transcendence of the extra-scientific implications that belief in strong hereditarian principles in the case of human beings had to social and political issues.

To sum up: The post-revolutionary French medics and medically trained biologists were among the first European thinkers to grasp the possibilities that the existence of physiologically caused, genealogically transmitted peculiarities had for

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[36]This trend has been mentioned by several authors: Lereboullet (1834), Piorry (1841), Lucas (1847), Delasiauve (1862), Grainger-Steward (1864), Semedeni (1960). For a list of the main theses see Appendix 1.

[37]See for this Russell, 1986, *Like engenderin' like*.

[38]For a history of plant hybridization see Olby (1985).

accounting the troubling differences between human beings. For some, differences between races, between nations, between social classes, between families and between individuals could no longer be explained by external shaping factors. Heredity became eventually the candidate to fill that vacuum, and to do so it had to be transformed into a general and regular cause, and its exceptions assimilated under a broad scheme.

Having such a tool in their hands made medical men a more important sector of society, like politicians, lawyers, social scientists, etcetera, would have a need of their expertise to carry out their own endeavours. This fact was clearly perceived by important French medical teachers of the early post-revolutionary period, like Fodéré at Strasbourg and Esquirol at Paris. As a result, the following generation, educated by them, found themselves using with ease both the language of heredity, and its peculiar dispositional view of latent causation.

By the same token, heredity was soon deployed, as a general, a prioris, explanatory tool, in French texts on the origin of human races, and other varieties.[39]

5.3 The Dictionnaire des Sciences Médicales (1812-1820) and the consolidation of *L'Hérédité*

It would not be an exaggeration, I believe, to state that by 1820 in France all medical men, and most physiologists and naturalists considered heredity a crucial issue. Hereditary transmission of a whole range of characters occurred without doubt and what really remained to be solved was the reach, the power and the limitations this phenomenon had in both humans and other species. Particularly crucial for different reasons was to know 1) if some socially damaging diseases, specially mental insanity, were indefinitely preserved within genealogical lines (in this case families), 2) if the racial, national and other group differences between humans could be entirely ascribed to the preservation within genealogical lines of hereditary variations (or degenerations), and 3) if characters that affected the specific type of the living organisms could also be preserved within genealogical lines in such a way as to challenge the age old belief in the immutability of species. It was increasingly believed that these relatively different questions (affecting medical men and alienists, anthropologists and naturalists) could be confronted with a unified analysis of the phenomena: a general theory of hereditary transmission. This was exactly the idea that occurred to several authors during the second decade of the 19th century. A very convincing register of this development can be found in the 60 volumes *Dictionnaire des Sciénces Médicales*, which from the period of 1812 to 1820 captured, in its different entries, this progressive generalization of the hereditary (in the loose metaphorical use) into a nomological approach to biological heredity. If we exaggerate a bit, that dictionary can be described as a kind of forum where the positions of the most important contributors to the domain under construction were given the word in succession.

As late as 1812, for instance, Phillipe Pinel, the grandfather of French alienism did not consider the hereditary cause important enough to deserve a mention in his inaugural paper on "Aliénation", preoccupied as he was in giving his own "moral causation" the leading role. The editors managed to by-pass him however by

[39]See Virey's articles on "Varieties" and "Monsters" in *Dict. Sci. Méd.*

commissioning a further article on the overlapping subject of the "aliéné", to a disciple, Marc, who stressed the central role of a hereditary predisposition to insanity. "Elle établit —he wrote— une des plus fortes présomptions en faveur de la realité de l'aliénation mentale".[40] But it was Esquirol, the crown prince among Pinel's followers (and teacher to the most hereditarian generation the world has seen)[41] who set the record straight with heredity as an influence for mental disease in his 13 articles for the *Dictionnaire*. In both "Folie" and "Manie"[42] he gave advances of what was to become his classic book on *Maladies Mentales* (1838). As is well known, Esquirol was the first to organize in statistical tables the cases of mental insanity, with the intention of sorting out the importance of each causal influence. Heredity he found to be a major "physical" cause, and in certain circumstances a dominant one.[43]

For the entry on "Héréditaire" a recently published essay by Antoine Petit (1817) was included.[44] This piece can be said to have been the most influential analysis published on the subject until the 1840's. It gave the most convincing blow to humoralist heredity up to then. Echos of Petit's precisely worded piece can be found in essays written sixty or seventy years later. In it Petit summarizes what he considers to be the main achievements that medics have attained in the definition of the hereditary cause. Heredity, he asserts, has to be based upon particular states of the constitution communicated to children by parents. These states give an "organic disposition" to re-produce a given effect, for instance a particular disease. He adds that they can be both localized states, or states of the whole *economie*, but he denies that some kind of general qualities of the constitution (like weakness) that establish in the body vague and indefinite tendencies (to disease) are to be seen as similarly hereditary. In heredity a specific, one to one connection must be shown to exist.

Petit praises insightfully the ancient distinction between predisposant and efficient causes as the main analytic resource to deal with the hereditary,[45] summarizes, with more clarity than any previous author, the determinant features of heredity. Latency, homochrony, atavism, all can be accounted for with a proper causal analysis. He upholds the importance of separating clearly congenital and connate influences, and

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[40] *Dict. Sci. Méd.*, tome I, 1812, pp. 311-329

[41] Georget, Moreau de Tours, Baillarger, Morel, among the most notorious. For accounts of Esquirol and his school's work see Ackerknecht (1959), *A Short History of Psychiatry*, chap.VI, pp.37-51. See also Semelaigne, *Les Grandes Aliénistes Françaises*, 1894.

[42] *Dict. Sci. Méd.* ,vol. 16, p.188 (1816) & vol.30, p.437 (1818)

[43] Esquirol for instance believed that among both the English in general and among the French rich people, heredity was undoubtedly an influential cause of insanity. His analysis of how heredity works was very close to the solidist tradition; he spoke of it as a physical, predisposant cause, and believed that homochrony and latency were particular signs of the presence of an hereditary cause. Like Pinel, he was sure of a physical base for human mental states, but was not a fatalist and gave more importance to efficient, moral causes.

[44] Antoine Petit, 1817, Essai sur les Maladies Héréditaires, Paris, Gabon. Reprinted in Dict. Sci. Méd., vol.17, pp.58-86. Petit was a medic from Paris, and member of the Conseil de Salubrité Publique.

[45] "distinction lumineuse...qui repose tout entière sur les faits, sera toujours une des sources fécondes o le médecin habile puisera les notions plus positives". Petit, Dict. Sci. Méd.,vol.17.,p.59.

accepts that only through the process of generation can real hereditary influence be transmitted. He however joins previous authors in condemning attempts to solve the mystery of heredity by an even deeper and more unsolvable mystery of generation. Hypothetical generation systems only confuse the issue. It is far more likely he adds that the proper observation of the patterns and nature of hereditary disease will illuminate the theorizing in generation, than the other way round.[46] Although he is sceptical about the feasibility of any success, Petit leaves to other specialists to decide what the real (intimate) nature of the inherited dispositions is. The good observer however can on occasions find visible, exterior characters that come with the disposition, before its effects are noticeable. Generally, however, this is not the case, and though their is an organic base to hereditary causes, they usually remain hidden (latent) until the time, in the life pattern, comes for their expression. This theme of the hidden cause that exposes itself at a given time was to be retaken by different authors of the *Dictionnaire* of both medical and physiological orientation.

After Petit's solid defense of heredity in disease it is not surprising that the articles of the *Dictionnaire* on all constitutional, chronic diseases give a preeminent role to heredity. The entries on "Scrophules" and "Phthisie", for instance, join vigourously the attack on humoralistic, vice dependent explanations of hereditary transmission,[47] favouring without reserve the view that heredity is to be ascribed to inborn constitutional (organic) peculiarities that predispose to certain effects. There is a wish in several authors of the *Dictionnaire* to make it clear that there is nothing particularly pathological with the route (or mechanism) through which the structural anomalies are communicated from parents to children. Normal physiological processes were responsible for that. Once the constitution acquired a flaw, the natural trend would be to transmit it through generation, as were transmitted all other constitutional features and qualities responsible for general and particular resemblance between parents and offspring. The open end of the discussion (the one Petit shied away from) was however what to (intimately) understand for constitution,[48] and how to describe its causal influence in the life of the organism. Where some medics saw it as a synthetic (cluster) term referring to the sum of the organic parts of the body (organs, tissues, etc,.) and their organization, other saw the term as capturing functional qualities, non reducible, general or particular dispositions. The different attitudes had a root in the tension between material and functional explanations. Between anatomy and physiology. And within physiology itself, between purely materialistic, and dualistic ontologies. "Constitution" was then a term with enough breath to encompass different, and relatively incompatible conceptions of the body, of its organization and function. Heredity, as a derivate concept, had the same quality. "Constitution", with its relative synonymes "temperament" and "complexion", defined a general space whose details, whose

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[46] op.cit. p.63

[47] "Scrophules", by Fournier-Pescay and Begin, *Dict. Sci. Méd.*, vol.50, 1820, pp.278-386."Phthisie", by J.P.Maygrier, vol.42, 1820, pp.15-168.

[48] "Constitution", as one of a triad of theoretical concepts that medics had developed for referring to the assembly of general and particular properties of the body, had an increasing importance, over the other pair, temperament and complexion. Fournier tried to make their differences explicit. "Il existe —he wrote— entre les mots *tempérament, constitution et complexion*, quelle que soit leur synonymie, dans certains cas, des nuances qui permettent pas les employer indistinctement les uns pour les autres."(*Dict. Sci. Méd.*, vol.4, p.158).

actual goings on, had still to be fought over by the proponents of different ontologies. De Montenegre, in his insightful piece on "maladie constitutionelle" provides a striking illustration of this view of the body as a battlefield

> le corps animal peut être considerée comme formé de plusieurs êtres independens, jusqu'à un certain point les uns des les autres, par leur manière d'agir; mais concourans tous à former un résultat général qui est la vie. Il doit nécessairement exister entre ces différens un sorte d'equilibre d'action, ...C'est ainsi que l'n peut concevoir ces dispositions individuelles qui s'étendent au moral comme au physique, et établissent entre tous les hommes une variété infinie.[49]

A constitution could be ascribed general states, or forms of being, that in turn would be responsible for reactions to stimuli, for dispositions, etc. Or it could be ascribed particular states or forms of organization responsible for localised reactions, in a given organ or part. The peculiarity of the constitutional variation could be material, and observable in principle, or it could be only a potentiality rooted in some quality (like irritability) or in a vital force of some kind.

No author doubted that there was a link (a rapport) between the parents' constitution and the new beings they gave rise to. And "hérédité" was there to highlight such a relationship.

As with "constitution", "hérédité" was a frame accepted by all, whose contents where being debated and defined en route. Yes, resemblances, in form and function, in health and illness, in body and mind, had to be somehow rooted in a casual link. But how could this be? Which kind of properties or characters were really communicated? Could only a physical (physiological) heredity account for all resemblances, or was a special, moral (or psychological) kind of hereditary transmission needed to account for the increasing number of statistics and observations of, for instance, insanity running in families?

The differences in focus and depth in the approach of the different sub-groups (general medics, alienists, physiologists, naturalists, anthropologists) involved in clarifying the hereditary cause, show that in the 1810's the domain was still unstable. Naturalists and anthropologists began their struggle to compartmentalize "hérédité" into a rational, a prioristic scheme, in which its boundaries and ways of actions would be clearly defined. The characters it would affect would be chosen from clearly defined sets in a hierarchical classification, and in an all or naught fashion. From specific, through racial, to individual, on one axis; and from physical to moral (or mental) on the other, authors would debate the reality of proposed hereditary transmission. The main problem these generalizations faced was, again, the irregularities, the proliferation of exceptions.

Like Petit, most medics, on the other hand, had strong feelings about not letting general a priori positions from outside impose definitions of constitutional dispositions and heredity without giving enough weight to their accumulated experience. A common reference was to the distortions that the debates over generation of the previous century had produced on the understanding of hereditary transmission of disease: the ill fated humoralist account had, for instance, received a boost in the late 18th century from stubborn preformationists like Bonnet, when it should have been on its way out. The medical evidence of hereditary transmission

[49] *Dict. Sci. Méd.*, vol.6, p.246.

was on the other hand of such a clear-cut nature that it ought to be given precedence over speculation.[50]

For medical men, in short, the specificity of heredity, as a cause was the product of medical attention to the details of transmission of disease within genealogical groups. Other biological authors were arriving on a field already sketched in its main features: "l'hérédité pathologique" should inform "l'hérédité physiologique" before the latter could reciprocate. And so the definition of heredity itself should be based on medical men's assessments of what generally is the case. To which extent, for instance, were only general, unlocalized constitutional dispositions (or characters) inherited; or were also very localized and particular ones (like moles, or bladder stones) transmitted. Such a question was better answered, some medics believed, by observing the patterns of disease communication, given that a disease (or a malformation) was a much clearer sign than other normal resemblances, just like in the case of moral phenomena, it is easier to follow, in a family, a pattern of such a distinctive symptomatology as that of insanity, than it is to follow vaguer, positive qualities, like honesty or strength of will.[51]

It is a sign of the effectiveness with which Petit, Fodéré, Esquirol, and the other contributors to the *Dictionnaire*, made their cases, that many of the matters they discussed around the hereditary were considered as settled by most French medical men after them. Elaborations and complications of their main tennets followed. The schools of medicine of Paris, Montpellier, and Strasbourg were constantly producing theses, both by students and professors (for tenure), dealing with different aspects of hereditary transmission of diseases. Works in which with increasing frequency a nomological attitude towards l'Hérédité was assumed. Lereboullet (1834), Béclere (1845), and specially P.A. Piorry (1840), produced exceptionally well argued expositions of how heredity worked in the communication of disease. But by the time these medics produced their work, the field was ceasing to be a purely medical and pathological one.

Slowly but constantly heredity was getting to be a hotly debated social *and* scientific issue. What medical men and some physiologists had for decades been arguing about the hereditary base of human nature, finally captured the attention of a broad sector of mid-19th century authors, who saw the potential power of their ideas for accounting for the unaccountable: the human soul in its collective and individual dimensions, and its dependence upon the body's constitutional make-up, or organization.

D.A. Lereboullet summarized the general importance of heredity, outside the purely medical realm. He stressed the uniqueness of the human case, of which medics had the privilege of having more experience. Among humans, he wrote "l'organisation nous présente des différences individuelles" based on the innumerable combinations that different constitutions, temperaments, and idyosincracies can produce. Together with the many modifications that external factors (climatic, passions, education) can make they can "rendre raison des nuances infinies que nous

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[50]For a more detailed discussion of the failure of generation theories as a priori basis for an account of the hereditary, see Petit (1817), Lereboullet (1834), and Piorry (1840).

[51]The Bernardian idea that the pathological is a source of illumination of the normal was coming of age during this period, and several of the contributors to the *Dictionnaire* reflect this fact.

observons entre les hommes...sous le rapport de leurs charactère physiques et moraux". But, he added, these subtleties can be further analyzed

> si nous appliquons à l'étude physiologique de l'espèce humaine la méthode des naturalistes, nous pourrons encore distinguer, au milieu de ces nombreuses différences, certains caractères communs, certains types originaux dont plusieurs auront persisté à travers une longue series de siècles. Les points de ressemblance seront plus nombreux entre les individus de d'une nation isolée et et qui n'aura pas contracté d'alliances étrangères. Enfin, si nous portons nos regards sur les membres d'une même famille, nous trouverons entre les enfans et les parens une conformité des plus évidentes: traits du visage, taille, son de la voix, couleur de la peau, constitution, tempérament, habitudes, caractère, moeurs, penchans, tout se ressemble. C'est sous l'influence de cette loi immuable, en vertu de laquelle l'homme donne le jour à des êtres semblables à lui, que l'on voit aussi quelquefois des vices de conformation se transmettre de génération en génération. Ainsi nous héritons de la constitution et du tempérament de nos parens; nous héritons de leurs caractères physiques et moraux; nou héritons de leurs vices de conformation.[52]

The possibility, described by Maupertuis, Adams, Prichard, and Pujol, among others, of having stable, genealogically based, natural human groups under the level of the species (races, varieties), could easily be extended to other "socially useful" categories, like the family, and the nation. Genealogy as the basis for classification, with heredity as the main explanatory concept was profiling itself as a promising approach outside the medical realm. At the same time the vagueness of key working concepts, like disposition, was an open invitation for imaginative theoreticians. "Heredity" was too good a gift for all those interested in explaining (and controlling) humanity, and as hereditary inferences came eventually to be one of the main rhetorical tools that these, often medically based (and biased) schools of thought made use of, they began to receive a much wider hearing, and closer attention began to be put on their rationality and grounding, by defensive thinkers of other persuasions.

"Hereditary transmission" first, and "The Laws of Heredity" later, became too important to be left in the hands of the doctors. The medical input survived however in the structure of the concept, and even in the late 19th century, after Darwin, Galton and Weismann had changed the shape of the domain of biological heredity, the original, pathological connotations of the theme persisted, specially in the French milieu. As late as 1873, under the entry "Hérédité", Larousse's *Grand Dictionnaire Universel du XIXe Siècle* described physiological heredity as mainly a medical concern, although it had to do with the "general tendency of Nature to reproduce in children certain physical and moral characters that occurred in the parents' organization".[53] The main point of describing a physiological (normal) pattern of heredity, it somehow seems, was to contrast it on the one hand with the pathological one, and to subdivide it into two main kinds of transmission according to two distinct

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[52]D.A. Lereboullet, 1834, *De l'hérédité dans les maladies*, thesis for tenure, Strasbourg.

[53]P. Larousse, 1873, *Grand Dictionnaire Universel du XIXe Siècle* tome 9, pp.217-218.

types of characters, physical and moral. “Heredity” had by then become a diverging tree, and each of its branches carried a suitable adjective.[54]

After the 1850’s, much attention was being paid to the “moral”, or later on “psychological” aspect of heredity in humans. Heredity was by then no longer a pathological term with analogical links with normal resemblances, nor a budding candidate concept for nomological status. The branched and qualified “heredity” that French alienists made use of in the second half of the 19th century was a much more complex concept, than it was delivered from the physicians early explorations. After an increasing number of French physiologists, naturalists, ethnologists, and later on, psychologists had incorporated the term into their theoretical resources, “heredity” became charged with a whole range of ontological presuppositions. It ceased to be a simple concept and began to embody more than just a small sector of the medical community’s view of the human body’s original make and dispositions. It’s empirical bases was expanded by its closer linkage to biological phenomena (like the origin of varieties), and its theoretical structure was also thoroughly expanded.

By 1834 D.A. Lereboullet, then a candidate for medical chair at Strasbourg, could confidently assert that the majority of authors understood Hérédité as the transmission of particular (bodily) dispositions that tend to re-produce in children the same characteristics their parents had (resemblances, diseases), at the same age, or in the presence of the same exciting cause. But how to understand the meaning of the clue word “disposition” was going to be a subject of debate. The concept in its medical (pathological) sense, we said above, was closely linked in the early 19th century the idea of “diathesis”.[55] When it was taken over, and generalized by other scientists’ imperatives from the much broader physiological and taxonomical theories made an important input. In the case of “moral” or “mental” heredity, the age old disputed dichotomy between matter and mind played also a major role.

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[54]This trend of multiplying the qualifications of “Hérédité” with a series of different adjectives was inaugurated by Prosper Lucas in his *Traité* ... (1857-50), some of his followers, as so often happen, took it to extremes. See for instance E. Littré’s definition of the word in his *Dictionnaire de la Langue Française*, 1863. It must be a credit to the long tradition of medical analysts of hereditary transmission of disease, that several of the features they insisted as being determinant of the phenomena, like latency of causation and its consequences (atavism, homochrony, etc.), were still considered as crucial by some late 19th century authors, who kept using the same kind of rhetorical devices in their narratives, quoting similar stories to the same effect.

[55]“Diathèse”, wrote Pariset and Villeneuve, is a term that can be synonymous to Galen’s habitus, and other author’s “disposition” and “predisposition”. It refers to the perception that medics have shared since antiquity that “des individus tellement organisés, tellement constitués,...sont constamment sujet à telle maladie plutot qu’a telle autre.” Diathesis, they write later on, “peuvent être originelles ou acquises: les unes dépendent de notre organisation primitive, et nous son le plus ordinairement transmises par nos parens; les diathèses acquises, sont le résultat de l’action ...de tou ce qui peut agir sur notre économie...” *Dict. Sci. Méd.*, vol. IX, pp.248-250. See also R. Olby, “Constitutional Diseases...”,in print, and Ackerknecht (1982) “Diathesis: the word and the concept in medical history”.

5.4 Heredity and the hierarchy of characters

With the emergence of heredity as a biological force, the question of which kind of characters were under its aegis became an increasingly important one. Naturalists, at least since Linneaus, had imposed upon the organisms' characters a classificatory gaze, and found that a hierarchy that reproduced their "difference within similitude" could be established for them. Necessary attributes persist, and are the same within each taxonomic group, while accidental peculiarities can change and are responsible for variety and individuality. Buffon wrote that

> L'empreinte de chaque espèce est un type dont les principaux traits sont gravés en caractères ineffables et permanents à jamais...mais toutes les touches accesoires varient; aucun individu ne ressemble parfaitement à un autre, aucune espèce n'existe sans un grand nombre des variétés[56]

Between individual and species stood the race. A race, to use an anachronistic description, is characterized by a set of correlated variations. The original type, the design, however it is conceived, is responsible for keeping the unity, the integrity of the species. But what keeps the race as a stable entity? is it a similar typological (necessary) "force"? or is is it a contingent set of coordinated influences? The former answer is difficult to adopt, as a proliferation of types and subtypes would end up leaving the concept too thinly spread, and the evidence, by the early 19th century, of mutable varieties and hybridization certainly weakened such position.

What kind of influence(s) were then responsible for the persistence of stable varieties? This question was relevant for both the human and the general biological inquiries. The geographical distribution of most varieties suggested an external, climatic set of causes, which could prompt internal potencies or mould the malleable portions of the constitution. At the same time, ever since Hippocrates had used the example of a tribe of macrocephalic people to illustrate the hereditary transmission of non-essential (accidental) variation, the genealogical explanation of the origin of races (or of stable varieties) had been seen as a possibility by some. That resemblances communicated through the reproductive pathways of the species could certainly produce stable varieties was one of the most influential opinions of Maupertuis. By the late 18th century an increasing number of authors considered such an idea as proven by the selective behavior of breeders and horticulturalists.

As it happens, the hierarchical view of the (physical) characters, that form the constitution, can be combined in different ways with a genealogical (internal cause) approach. James Anderson, the Scottish animal breeder gave a very good example of this, as he developed, without breaching the main ontological assumption of his day, a sophisticated account of why carefully selecting the mating partners actually worked in the creation of new breeds. He adopted a concentric series of hierarchical levels of properties. The species, the race, the family, and the individual, each had its determined characters. Each inferior level can suffer (spontaneous) variations in any direction, but only up to the limits set by the superior level, which constitute its boundary. Racial characters can vary within the specific type, familial characters can vary within the racial type, and individual characters within the familial. By going down through the hierarchy, the breeder can select individual variations within the family, varieties within the race, varieties within the species; and it can eventually stabilize in a new variety, a genealogical line with the desired characteristics.

[56]Buffon, *De la Nature*, Seconde vue, tome VII, p.418.

Anderson's scheme, which denies any influence to Buffonian climatic causes, assumes that variations that do not transgress the boundaries of the hierarchy can be, and are, transmitted hereditarily. And for him, as for Maupertuis and Hunter before, variation is a spontaneous occurrence that gets fixed in the constitution at the first moment of its organization. Crossing different varieties you can obtain new ones, so the number of different varieties could possibly be unlimited; but the number of species is fixed.[57]

Anderson's scheme, as several others divised by his European contemporaries,[58] is an attempt to find a solution to the contradictions between a stable, fixist, hierarchical view of biological groupings, and the instability introduced by the genealogical preservation of peculiarities.

Up until the turn of the 19th century, the hereditary had been linked, as we have repeatedly seen, to the transmission of the deviations to the norm. With the peculiar and striking reccurrences of accidental features within genealogical groups. For most people outside the medical world, the hereditary was associated with a marginal, freak set of occurrences, whose irregularity precluded any idea of law or generality. Individual and specially familiar deviation from the norm (variation) was a pre-requisite for the idea of heredity itself; in other words, it was an unstabilizing agent in a typological world. But during the early years of the 19th century, the de-pathologization of the hereditary cause took place. There were increasing claims that hereditary transmission of the deviant was only a side-effect of normal, regular transmission, and the perception of what the hereditary meant began to change, specially among naturalists and physiologists. From a series of (deviant) facts with a similar pattern of reccurrence, and with possibly related causes, the hereditary was transformed into a general influence, an action, a cause that reproduced the general, stable patterns of each taxonomical group. This is how for some authors, "heredity" came to the forefront of their biological thought, and filled, so speak, the theoretical space that other stabilizing "forces" had occupied in the work of some 18th century naturalists.[59]

A consequence of this movement of heredity into a central position was that a clear-cut opposition between heredity and variation eventually emerged. And much of these naturalists' thoughts and research was dedicated to sort out which kind of characters were under the control of which.[60] Some authors began to identify heredity as the source of the stability of taxonomical groups, arguing that it was the general bodily structure, or organization, rather than particular aspects of it, that was the main object of hereditary transmission.[61] For some, heredity of essential

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[57]James Anderson, 1799, "An Inquiry into the nature of Varieties" in *Recreations in Natural History*, vol. I, pp.49-100. London. T.Bensley.

[58]Pallas, Lamarck, Prichard are some examples.

[59]Buffon's interior mould, Maupertuis' organizing force, Wolff's vegetative force, Blumenbach's *vis essentialis*, or for that matter, the role played by the germ in the preformationist account, See Bowler's insightful essay on Buffon and Bonnet (1973). See also Serres review of F. Jacob's *Logic of Life*, "Las traductions de l'arbre" (1974) in his *La Traduction, Hermes III*, pp.15-41.

[60]The above sketch applies particularly to France. But in England for instance, a similar reflection can be made on the works of Prichard and Lawrence.

[61]The medical distinction between the inheritance of general constitutional dispositions as a whole, and that of particular, separate elements, was mirrored by this

characters was in charge of the preservation of the types, for others heredity of accidental characters gave rise to differentiated families, nations, races... These not entirely inconsistent positions created the polarities and tensions amidst which the domain of heredity was to be explored. When the existence of a normal (non-pathological) hereditary influence ceased to be a disputed issue, the question drifted towards its characterization and powers. When not only accidental variations and diseases were going to be considered hereditary, but heredity was going to be given the leading role in the re-production of the new organization (or constitution), the problem that arose was which characters *are not* transmitted, and why.

Irregularity had always been a main problem for any defense of an hereditary cause. But the inconsistency of transmission, specially of peculiar, accidental features, sharply contrasted with, and was counter-balanced by the occurrence of striking positive cases, whose vivid convincing power was difficult to undermine with rationalistic analysis.

As a conservative influence, heredity was *a priori* linked to specific, and even racial characters, which were seen as the most stable from generation to generation. But observation-wise, it had been the conservation of the whimsical, from family resemblance to monstrosities, that had brought heredity to attention of people in the first place. The observation of much more irregularity and variation in characters that were seen as belonging to subservient levels, such as the national, the familial, and the individual was not an impediment for the use of this pseudo-taxonomical categories for dealing with the subclassification of the human species. This could be achieved in great measure due to the use of a genealogical criteria for the unity of a type. Such criteria promoted the view that the identification of the family with, first the national group, and then the race was *strictu sensu* a reality. Heredity, with its powerful metaphorical appeal, was then seen as the mechanism of preservation of the family's (and the nation's, and the race's) physical and moral heritage or patrimony, which consisted in a correlated set of peculiarities with an organic (constitutional) basis. As Virey wrote in 1821:

> Les traits naturels des familles sont donc un héritage des races...les individus tous émanés du même sein, vivant ensemble des mêmes nourritures, ne formaient qu'un seul corps, prenaient des affections uniformes, des idées et des manières toutes pareilles[62]

As Michel Lèvy wrote "toute famille a son patrimonie organique". Francis Bleynie added a few years later that also "toute peuple et toute race" had their patrimony, which is without doubt "soumis à la loi de l'hérédité".[63] A fair number of authors were attracted by this linkage between sets of hierarchical hereditary characters and the way the human groups varied and were geographically distributed. It provided a frame of discussion for the opposition between polygenists versus the monogenists.[64] The barriers of race, like those of species, could be candidates to

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new dichotomy. For a skillful analysis of the theme See J.Schiller (1978) *La notion d'organisation dans l'histoire de la biologie*, specially chapter X.

[62]in the article "Physiognomie", *Dict. Sci. Méd.*, vol.42, p.206.

[63]M.Lévy, quoted by F. Bleynie in his "Considerations Génerales sur l'Hérédité Physique et l'Hérédité Morale", 1865, p.9

[64]Virey, for instance, who refused to accept that the black and the white races of humans could possibly belong to the same stock, defended the idea that health and perfection were related to purity of race, and breeding isolation. Other authors saw

supply a limit to variation, as much as any other within the hierarchy. On the other hand, as said above, the scheme provided a frame where both materialistic and dualistic physiological ontologies could be accommodated; leaving the substantiation of how the properties and their transmission were to be explained, open for speculation and debate.

The explanatory depth that the idea of hereditary transmission already possessed in the early 19th century, due to the theoretical efforts of physicians, was put to work. That the hereditary cause was capable of remaining hidden (latency), and that it acted through organically based predispositions, whose effects only are made apparent at given moments, and possibly through the concurrence of additional causes, gave it an explanatory malleability that was promptly put to use. That is how, for instance, heredity could both explain the tendency to revert to type (via atavistic regressions) and the tendency to deviate from it, (through the inheritance of variations). Heredity could also account for the sequenced fashion in which different characters appear during development. Virey, for one, craftily mixed this idea with his genealogically based view:

> Les traits sont arrondis, enveloppés dans l'enfant en naissant. A mesure que l'enfant s'accroit en âge les formes se développent, les caractères de famille et ceux de race se gravent principalment à cette époque pour servir de trame première[65]

In short, heredity became a theoretical device that defenders of all positions could call to their support. In the realm of "normal" characters, of natural history and anthropology, both fixists and transformist had uses for it. In the dominion of "deviant" characters (pathology and teratology) both degenerationists and anti-degenerationists claimed its support. Fodéré, in his more general and theoretical contribution to the *Dictionnaire* ("Vie") showed this enthusiasm for linking all the properties of life to heredity.

> il y a pour chaque espèce une forme propre, organique, transmissible par la génération...la vie n'est ainsi qu'un héritage...

Whereas for him, some organic variation, specially of a dramatic kind, as in monstrosities, can break the mould of a species and produce a new one, no degeneration can really become a permanent feature of a group

> Au contraire —he writes—, la propagation des mêmes figures dans chaque race, la ressemblance des petits à leurs parens, l'hérédité même de plusieurs vices de conformation et de maladies organiques, sous l'influence des causes qui les produisent, tout annonce que la nature aspire à conserver ses formes...à rétablir sans cesse l'integrité de ses productions.[66]

Heredity provided then with a sharp lens to focus the parallel discussions (in health and illness) between the proponents of fixity and those of change. But in the French rationalistic style, it needed its opposite to properly account for processes or

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inbreeding and isolation as a source of degeneration, and a solution to it in the crossing between different groups. This discussions tranversed the 19th century (with the loud polemic over consanguinity, etc.) and took graver tones in the first part of our century.

[65]Virey, "Physiognomie", *Dict. Sci. Méd.*, vol.42, p.204.

[66]F.E.Fodéré,(1821),"Vie", *Dict. Sci. Méd.*, vol. 57, pp.434-603.

equilibriums. To know if families degenerate or species are transformed, or if there are built-in mechanisms to impede those trends, it was the interactions between heredity and variation that had to be sorted out. Heredity as a concept (I have been trying to show) was the product of a conceptual reification from a set of related phenomena, a sort of projection to the inner (intimate) workings of the physical organization of the body from a pattern of occurrences. A similar move was made from the 1830's onwards with the concept of biological variation, in order to create a dialectical partner for heredity. The Cuvierian naturalist and physiologist Pierre Flourens wrote in his *Ontologie Naturelle*

> Je trouve, dans l'organisation, deux tendances très-manifestes: 1o. une tendance à varier dans de certaines limites; 2o. une tendance à la transmissibilité, à l'hérédité de ces variations.

These variations, he adds, are spontaneously generated, and do not die with the individual. As they are transmitted from generation to generation they turn from individual into hereditary characters: "et voilá la race formée".[67]

Genealogical criteria for defining taxonomical groups could be compatible with a fixist position like Cuvier's and Flourens', or with a transformist one, like I. Geoffroy-St.Hilaire's. The kind of characters that were believed to be transmitted hereditarily (through generation), was intimately linked to the major preconceptions about bodily structure, organization and the depth (or superficiality) of variations. During the 19th century as both J. Schiller and F. Jacob have shown,[68] there was a "decoupage" of the notion of biological organization that crucially affected the analysis of hereditary transmission. The hierarchy of characters, in its reflection of the taxonomical divisions, began to be perceived as independent elements of a multilayered entity. As I already said, the individual body's first formation began to be perceived as the site over which different (hierarchically divided) feuds took place. The hereditary relationship that a parents' constitution was supposed to have with its offspring was thus fragmented. Heredity was seen as working independently at the species, the racial, and the individual level. The parents' specific characters would influence the childs' specific characters independently of how the parents' individual characters were influencing the children's individual characters. The body's final structure or constitution was then the product of all these actions, and their play of balance and counter-balance.

The contradiction the fixist faced between the tendency to (hereditarily) perpetuate the type, and the tendency to reproduce the individual variations in the following generation could thus in principle be solved. Heredity (and variation) could act with differential strength. The essential structure of the organization repeated itself in all generations with unfailing regularity, (except for monsters and "sports"), while the strength of transmission diminished with the increasingly accidental, and particular characters of race, family and individual, and in these cases variation could occur in higher proportions. Resemblances that were very particular and striking (both normal and deviant) could also be easily accommodated in this rationalistic scheme. This generalized view of hereditary transmission was to become the most accepted one in the post-Cuvierian decades of the French 19th century, and was to attain its highest point in the work of the alienist theoretician Prosper Lucas, which will be the subject of the final section of this chapter. I will before that analise briefly

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[67]P. Flourens, 1863, *Ontologie naturelle*.

[68]Schiller, *La notion ...*, (1978), F. Jacob, *La logique ....*

the other major axis along which Lucas based his analysis; the one that goes from the physical (physiological) to the moral (psychological).

5.5 Heredity and the mind

Mental or, as it later came to be known, "psychological" heredity came in itself to be one of the most important and disputed phenomena of the 19th century, as alienists, phrenologists, hygienists, reformists, eugenicists, and several other interested groups, fell under its contagious spell.[69] It would not be an exaggeration to say that if it hadn't been for the strong preoccupation with the transmission of moral qualities through heredity, this latter kind of biological causation would not have received the level of attention and publicity it eventually got, and some important elucidations would have been delayed. This is specially true for the pre-Darwinian decades of the century. The sociological reasons why the hereditary cause acquired a great importance after the 1840's among the French alienists have been recently explored with success by Ian Dowbiggin and Daniel Pick.[70] Dowbiggin has insisted in the importance of institutional power relationships, and the struggle to establish an autonomous professional domain by the emerging tribe of psychiatrists (A similar argument was used by R. Nye (1984) for criminology). Pick, on the other hand, searches for his explanations in the general field of European social, cultural and political anxieties, linked to the fears of hereditary degeneration and lose of national and racial purity. But neither of them explores with any attention the medico-physiological origins of the concept of heredity itself, and how it was already an important presence in those disciplines in the first four decades of post-revolutionary France. Pick acknowledges this when he writes that

> In the wake of 1848, heredity had hardened into a key term in many aspects of medicine and anthropology. This shift was complex and, certainly, had been taking place for several decades, but 1848 was an important moment of heredity's petrification.[71]

Such a "key term" had come a long way from the early 19th century solidist, simplistically materialist one that appeared in physiognomical and phrenological literature. It had been sophisticated in several ways, as we saw in the previous section. But with regard to the communication of moral characters it suffered more dramatic changes. For what was a relatively short period, the reductionist idea of the *idéologues* thrived in both medical (alienist) and non medical circles. That mental qualities could have a simple, straightforward, organizational (material) base was a

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[69]Many recent historical studies have been guided towards the understanding of why some social issues to which Biological Heredity was related gathered such importance during the 19th century. R.Cooter (1979) and Hilts (1982) on Phrenology; R. Nye (1984) on Criminology; Pick (1989) and Chamberlin (ed., 1985) on Degeneration theories; Rosenberg (1976) and Desmond (1990) on the influence of thought on social sciences and Dowbiggin (1991) on the politics of Insanity. For studies on the relationship between hereditarianism and literature in France see Y.Malinas (1985) and J. Borie (1981). For the same them in Great Britain, P.Morton (1984).

[70]D.Pick, 1989, *Faces of Degeneration,* I. Dowbiggin, (1991) *Inheriting Madness*. See also R. Olby's review of Pick's book (in press).

[71]D. Pick, op.cit., p.23.

claim that was used to challenge the dualistic, spiritual view of the mental after the 18th century. Alienism, as a budding medical discipline, made use of such assumptions very early on, and also of the association of some mental diseases with organic (constitutional) states, that was reinforced by the ancient tradition, in medical literature, of including insanity, epilepsy, and other moral ailments among the hereditary diseases.[72]

As I said, the hope of some early 19th century authors like Pinel, was that the hereditary influence, as physically based disposition, could be relegated to a second plane, leaving the central explanatory, and therapeutic, role to the more complex "moral" (emotional) causes. But the following generation of alienists were not so sure. Leaving aside the sociological reasons for wanting to stress heredity in their explanations of madness, it is true that when the first statistical tables were compiled with the information from the inmates of French asylums, nobody had yet explored in depth heredity as a potential theoretical tool for mental dispositions. The patterns of family related madness seemed to some alienists too striking to be left at the side. It is a pity, Fodéré wrote in 1813, that the great Pinel did not say a word on the subject of heredity in his great treatise on mental alienation, as much light is still to be thrown on the subject. He praises Greding however for having tried to relate some hereditary (familial) conformations of the cranium with some states of alienation.[73]

Paradoxically, only a few years later Fodéré himself was reacting strongly against precisely that sort of crude correlation made by phrenologists between special parts of the brain and some mental qualities, and diseases. His materialism, like that of several of his contemporaries, had a limit, as had his tolerance of such an invasive anatomical pursuit. The complete identification of parts of the anatomy with particular mental states seemed a *reductio ad absurdum* of the *idéologues*' tenets. The strong hereditarian element that phrenologists began to give to their teachings at some point made the critics of their naive materialism rethink their view of hereditary transmission. Doctors that had assumed that a materialist (and solidist) stance with regard to constitutional diseases (including madness), and also in relation to hereditary transmission, would pave the way to simple solutions, suddenly found themselves having to rethink their views. Apart from a few defenders among French alienists, phrenologists were regarded with strong suspicion by most of them.[74] The lack of any progress in the anatomical identification of correlations between brain defects or oddities with insanity brought the realisation that any materialistic program

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[72]Well known examples of this can be found in the Hippocratic treatise on the "Holy Disease" (See *De Morbi Sacri*, Lonie, 1981), or, later, in Burton's *Anatomy of Melancholy* (1651), where the hereditary nature of the deviant mental states are much stressed.

[73]Fodéré, 1813, "Des maladies héréditaires" in vol.5 of *Traité de Médecine Legale* ... He refers to Pinel's *Traité de la Manie*, 1806, and to Greding's *Traité Médico-Philosophique sur l'Alienation Mentale*, Paris. an 11

[74]Dowbiggin tries to give a different impression when he writes that phrenology "was very popular with alienists of the period, including Esquirol, Etienne Georget, Briere de Boismont,..." op.cit, p.24. One only has to look at the entries of the *Dictionnaire*, and also the anecdotes Leuret tells about Gall's reception by Esquirol in la Salpêtrière, and by Pariset in Bicêtre , to perceive the strong scepticism with which phrenology was received among alienists. Related both by Flourens (1861), *De la raison* ..., pp.237-244.

for the reduction of mental phenomena to physical or physiological ones would have to have a more subtle ontology than the simple, structural one proclaimed by early 19th century enthusiasts.

The disenchantment with pathological anatomy that Dowbiggin situates after the fourth decade of the 19th century began many years before. It can be detected in the 1820's when a non structural (but functional) view of hereditary transmission began to be popularized. After Pagès and Petit's solidist medical dictionary entries of the first couple of decades of the century, the anonymous entry for "héréditaire (maladie)" in the 1820's *Dictionnaire Abrégé des Sciences Medicales* strongly criticized some of their main tenets. Accepting that constitutional predispositions are the cause of hereditary disease, this author challenges both the specificity of the transmission, and some of the criteria previous authors had come to defend as the tell-tale signs of hereditary influences, like homochrony. Simpler, more general explanations of how constitutions predispose to disease, he argued, were to be invoked instead.

If we proceed to make the examination of the diseases known as hereditary, we will find in all occasions a simple excess of irritability in a certain organ, a marked predominance of a system or of an apparatus. We will thus witness the collapse of the whole scaffolding of reasonings with which people have tried to establish that the specificity of morbid predispositions and the hereditary transmission of diseases demonstrate the specificity of the latter[75] Not wishing however to be thrown into the same sack as the old style humoralists, the same author immediately qualifies his position by proposing a dual, anatomical-physiological (form-function), description of the hereditary influence, based on what we could call "emergent" physiological properties

> Dire que l'hérédité de telle ou telle maladie dépend de l'excès d'irritabilité d'un organe, ce n'est pas nier l'hérédité de certains modes de structure, car la vitalité est irrémédiablement liée à l'organisation, sans que l'on puisse dire que l'une dépende de l'autre; nous ne reconnaisons pas de modifications de l'excitabilité sans modifications de l'organisme, et point de modifications de celui-ci sans modifications de celle-là; ne s'agit pas toujours des organes, considérés tantôt dans leur structure, tantôt dans leur action? [76]

As J. Schiller has shrewdly analyzed, the notion that biological organization (or constitution) has a series of "emergent" properties (like irritability), which was promoted by physiologists like Bichat, and later Barthez and Bordeu, could still be

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[75]If you think this English phrasing convoluted, try the French:

> Si l'on poursuivait ...l'examen de toutes les maladies réputées héréditaires, on trouverait, pour toutes, une simple irritabilité excessive de tel ou tel organe, une prédominance plus marquées de tel ou tel système, de tel ou tel appareil, et l'on verrait ainsi tomber l'echaufadage de raisonnemens par lesquels on a voulu établir que la spécificité des prédispositions morbifiques et l'hérédité des maladies démontrent la spécificité de ces dernières.

[76] *Dict. Ab. Sci. Méd.*, vol.9, (1823?) pp.45-39.

reconciled with a materialist programme,[77] but at a price; the door was open for vitalistic metaphysics.

Pinel was always sympathetic to Bichat's physiological theories, as they were partially based on some of his pathological discoveries[78] and later other alienists, in the reaction against phrenology, saw in the "dualist" materialism a good alternative. As Dowbiggin has stressed, alienists could not afford to abandon a materialist account of mental illness due to the threat that purely spiritual explanations posed to their medical approach and their claims of exclusivity for their treatment and cure,[79] but a property based anti-reductionism, was acceptable.

Among the first medical authorities to argue for this change of physiological ontology with regards to hereditary transmission was Fodéré. In an explicit attempt to undermine the phrenologist's appropriation of hereditarianism he wrote a long defense of a different interpretation of "hereditary predisposition" to the solidist, localized one. He argued that to make constitutional dispositions reside in specific, concrete structural arrangements of the body is only done by ignoring the way the same arrangement can sometimes have different dispositions, and can be, so to speak, in different states. To make mental qualities depend on concrete parts of the brain, their form, size, etcetera, is certainly an extreme of this same error. Leaving aside the deterministic absurdities that such beliefs would oblige us to accept, he continues, the atomization of mental faculties is simply unacceptable. Higher mental abilities are too elaborate to be situated in single parts, and to be disturbed by simple physical changes. It is in the principles of life, he adds, where the meaning of hereditary predisposition is to be found. It is not in focusing on matter itself, but in the "emergent" properties that this principle is to be understood. The matter that constitutes a body changes continually, he writes, but the properties and dispositions are preserved nevertheless.[80]

This reaction to reductionist materialism in the interpretation of hereditary transmission was not reserved to mental faculties. By 1840, in his very influential treatise *L'Hérédité dans les Maladies*, P.A. Piorry dedicates a whole chapter to normal hereditary transmission of physical characters, in which although he upholds the resistance to old humoralism (with its vices and taints), he reintroduces the idea that protean hereditary principles, which apart from being "emergent" over solid (organic) parts, could well be founded upon, and be communicated in the fluid parts (like blood, or other humors). That this principles could in effect be the cause of several different diseased conditions, according to the organ, or system they affected was then a logical conclusion to this enriched ontology of the hereditary. The possibility of hereditarily (causally) linking very different ailments in members of different generations within the same family, had always been attractive to hereditarian physicians, as it made easier the job of tracing the effects of a morbific

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[77]See Schiller's excellent chapter "Le découpage de l'organisation" in La notion d'organisation.... In it he describes how the split between structure and properties of the organized body created a stressed two tier development of the concept of biological organization, the effect of which lasted during most of the 19th century.

[78]See J.Schiller, op.cit., p.61.

[79]Dowbiggin, op.cit., pp.11-37.

[80]Fodéré, *Traité du Délire*, 1817, pp.121-136. Fodéré identifies his principle of life with Virey's "principe vivifiant" and tries also to account for the success of Pinel's moral treatment with it.

cause through the generations of a family. Such non-specificity raised at the same time the hackles of the sceptics who saw such a procedure as self-indulgent and messy. But though one of the reasons humoralists like Pujol and Portal were relatively discredited was the loose proteism of effects in the causes they had based their case upon, Piorry had no qualms in bringing into play a new generation of protean hereditary causes, under the healthier umbrella of the prevailing physiological dualism.

More resisted among physicians, who saw the kind of confusions and obscurities that unrestricted speculations could drive to, the new hereditary proteism was enthusiastically adopted by most alienists, whose grand overarching schemes of mutating mental diseases and degeneration within families were precisely based on the acceptance of unlocalizable, multifaceted, proliferative hereditary causes.[81]

By the time J. J. Moreau de Tours, B. A. Morel, and other mid 19th century hereditarian alienists wrote their most influential work,[82] physiological (and pathological) heredity had become for them, and for many others, an unquestionable explanatory tool, capriciously adaptable to all evidential patterns, and underpinned by a very thick network of a priorist reasonings based on the prevalent dualist materialism that other French theoreticians, like Flourens, endorsed.

The notion of physiological heredity had been given a basic conceptual structure by physicians in the early years of the century, and the intervention of a couple of generations of interested naturalists, ethnologists, alienists, etc. had stretched its possibilities to the limit. The accumulation of facts, both ancient and of recent (statistical) harvest, was by the 1840's enormous. The number of claims and counter-claims of the hereditary transmission of an increasing number of general and peculiar characters was also growing exponentially in a number of fields.[83] What was this general cause everybody was claiming explained so many things? Was heredity really the first cause of physical and mental qualities in human and other living beings? What was there in common, for instance, between the transmission from parents to offspring of a weak eyesight and that of a brilliant musical talent? Was society right in fearing a proliferation of individuals from affected, ill stock?

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[81]Dowbiggin's analysis of the hereditarian tenets of some alienists, specially of J. J. Moreau de Tours, is very insightful (op.cit., pp.54-75, and 116-143.). He fails however, in my opinion, to acknowledge the extent to which these authors based their positions in the works of previous writers, like Piorry and Lucas, to name just two.

[82]J. J. Moreau de Tours, 1859, *La Psychologie Morbide*; B. A. Morel, 1857, *Traité des Dégénérescences Physiques, Intellectuelles et Morales de l'Espèce Humaine.*

[83]A special case was the scepticism with regards to moral or mental heredity. As Schiller observed, the "dècoupage" of the concept of organization between structure and properties opened the door for the vitalistic metaphysics, of the kind that early 19th century theoreticians had wanted to exclude. Hereditary transmission of mental faculties could not but be challenged in an environment of proliferating causal claims. Montpellier was, of course, the nest from which the major scientific challenges to the alienists' cherished "hérédité moral" came from. The best example is Lordat, who in his "Les Lois de l'Hérédité Physiologique, sont elles les mêmes chez les bètes et chez l'homme? ", 1842, Montpellier, while admitting that physical, structural parts of the body were inherited by both men and beasts, argued that the mental peculiarities of human beings remain out of the reach of physiological heredity, or any other materialistic cause.

Whoever achieved a useful synthesis of that vague and irregular territory would be making an invaluable contribution. That was the task that a relatively obscure, bookish and imaginative alienist, Prosper Lucas set himself to accomplish during the 1840's: To synthesize all the known facts about hereditary transmission, and organize them around a coherent theoretical scheme.

5.6 Prosper Lucas' *Hérédité Naturelle*

Perhaps the best way to begin an analysis of Prosper Lucas' (1805-1885) is the quote from Laplace's *Essai Philosophique sur les Probabilités* he chose to put ahead of his *Traité de l'Hérédité Naturelle*.[84]

> La méthode la plus sûre qui puisse nous guider vers la recherche de la verité, consiste à s'elever par induction des phénomènes aux lois et des lois aux forces.

This empiricist stance describes accurately, if not the result, at least the intention of Lucas' oeuvre. That it is based on an impressive array of facts and evidence there is no doubt. His aim was to uncover the regularities buried behind a jungle of irregularities, of claims and counter-claims, while bringing together and organizing the scattered field of *l'Héredité*. He then proposed to show how those regularities (patterns of occurrence of traits through the generations) could be neatly accounted for by the coordinated interaction of two opposing principles: heredity and inneity (*innéité*: the source of variation). The inference towards the actual existence of these forces completes the movement. The reification of heredity and inneity is then justified by the naturalness with which the evidence falls under their spell. But the truth is that Lucas proceeded the other way round, accepting, *a priori*, the reality of both forces (although he rhetorically denied it), and from there he organized the facts he collected in a rationalistic fashion.

All in all, Lucas performed an outstanding task of fact collecting. There was practically no important source of evidence about hereditary transmission, of whatever age or reliability, that he did not consult.[85] It was, I have said, as the man who sorted out the evidence and put beyond reasonable doubt the importance of heredity in the investigation of the human condition, that Lucas was remembered in the last quarter of the 19th century, and for a few early years of this century. The theoretical scheme around which he organized his factual display has largely been ignored, but it was crucial, I believe to both the enterprise and the strength of the evidence he collected.

Historians have on the whole disdained the theoretical aspect of Lucas' work, and only recently has it begun to receive some attention.[86] The influence that his work

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[84]The complete title is *Traité Philosophique et Physiologique de l'Hérédité Naturelle dans les états de santé et de maladie du système nerveux*

[85]Several previous authors had collected long lists of relevant cases of curious or striking hereditary transmissions (Haller, Blumenbach, Meckel). The main sources of cases were to be found undoubtedly in the medical literature. Although Naturalists had also collected striking cases of hereditary resemblances. Relevant works by breeders, historians, and lawyers were also revised by Lucas.

[86]Recently F.Churchill (1987) and B.Balan (1989) have dedicated some attetion to Lucas' work on heredity. The former only in a limited way, as he choses only to see the aspects of Lucas that are directly relevant to Darwin's pangenesis theory. The

exerted went beyond the closed French milieu (where he strongly influenced a wide spectrum of other areas in the likes of Moreau de Tours, Morel, Ribot, Tarde, Zola), and touched Spencer, Darwin, and Galton in England, and the Swiss botanist Alphonse de Candolle. His grandiose vitalistic scheme that wanted to root the forces of heredity and inneity in the springs of life themselves (creation and innovation) was not in tune with the developments in the biological sciences of the latter part of its century, and could be said to be among the last of the great 18th century-style Newtonian biological projects. This outmodedness, and his exhaustive use of the "striking case" method of induction, so popular in the medical hereditarian literature but vilified soon after, condemned his analytical skills to a very short-lived success. Only the first generation of his readers could appreciate the sense of order he brought to the field of heredity. Of not less importance, from a sociological perspective, is the clear analysis he produced of the deep links between the attention that human biological heredity received in his age and the worries and aspirations of his contemporaries.

A medical man, and an alienist himself, Lucas had no doubt at all of the reality of the "force" of heredity. He was also keenly aware of the actuality and relevance of its effects for the social and political discussions of the post-revolutionary 19th century. The forces of conservation and those of change, that struggled for political dominance, were projections of the deeper biological principles he was trying to reveal. As a conservative himself, he felt that the preservation of the essential fabric of society was to be attained by the healthy conservation of its elements: the national and familial (genealogical) groups, and within them the individual. Hereditary transmission of power, of property and even of crafts, were seen by him as "naturally" justified by the dominion physiological heredity exerted over a whole range of the hierarchical characters that constituted the individual. With Girou de Buzareingues, Lucas believes "il n'y a rien, dans l'animal qui ne puisse se transmettre par génération".[87] But Lucas takes the pain to back this statement with examples of all the kinds of characters he conceives along the two axis: the hierarchical axes of taxonomy, and the dualistic axis that divides the physical from the mental.

All the way from the basic organization that made an individual belong to the human species, to the most indifferent and accidental of individual characters, heredity, he tried to show, had some bearing. In other words, a child would always have a greater possibility of resembling any one of his parents (or ancestors) in any given character, peculiar or not, physical or mental, than he would of resembling someone else.

As always, the problem with any claim to lawfulness in hereditary transmission was the plague of inconsistency. For any case (however striking) of re-occurrence of a given character within a genealogical line, an indefinite number of failures could be pointed to. This was particularly true of inessential, individual variations.

To face this Lucas took what was perhaps his boldest step, the coinage of the term "innéité" to refer to a force that was to be paired with "hérédité" to produce the observed phenomenology of character occurrence.[88] Innéité was conceived as a way

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latter's piece is an unfortunate and anachronistic complaint against Lucas for not having foreseen that the positive science of genetics was just round the corner.

[87]quoted by Lucas, op.cit., vol.1, p.605.

[88]In this, as in other matters, Lucas followed the German physiologist Burdach, whose *Traité de Physiologie* (1837) was the book he most often quoted. Like

to tame the spontaneity and unpredictability of variation, specially of congenital variation, (i.e. of deviations from the type capable of being inherited because they had been incorporated to the individual's intimate constitution; the one he acquired at his first formation.). Lucas relates this force to the capacity for innovation that the order of nature shows, and on the base of this F.Churchill has, mistakenly I believe, associated it with a directional, adaptive force. But Lucas only stresses the capacity for producing change (be it good or bad) at sub-specific levels. Innéité is, so to speak, a tendency to modify the parent's original (individual) type which is serving as model for the offspring. For Burdach, who is the inspiration of Lucas' concept of inneity, such tendency for variation is due to a search for a realization of the multiple modes of existence that a given type possesses in potency.[89]

A way to describe Lucas' model of how heredity and inneity work is to focus on the moment of the first organization of the individual. For Lucas what occurs at that moment is a multilayered compromise between many influences. Resemblances are promoted by heredity. dissimilarities by inneity. The hierarchical relations among the characters allows for an independent bargaining process at each level. Each character, within each level, can be influenced by heredity or by inneity. If heredity prevails, the options of resemblance are open: the mother or the father have the strongest potential influence (resemblances can be complete or partial), but behind them, so to speak, are the possible resemblances to more distant ancestors, whose influence survives in a latent form (thus the existence atavistic reccurrences). If inneity wins the character adopts an unmodelled state. At the species level only heredity is active, inneity cannot affect specific characters, so transformation of a species into another one is blocked.

A further complexity to Lucas' model has to be mentioned at this point. The bargaining process I have described (what Lucas would call the rapports) does not take place between the parents' or ancestors' actual characters and the organization of the new being, but between what Lucas calls the types and the new being. For reasons that will be clear later, Lucas makes an ontological separation between (hierarchical) types and their actual embodiment in the new individual. The types are the real bearers of the hereditary force, and they are the ones which strive to make the elements of the new organism resemble (or embody) their structure and qualities. The species' type, in Lucas' Cuvierian view, only determines, dictatorially, the general aspects of organization, leaving different ranges of possible variation in the inferior types, that go down to the individual type, which is a particularization of all the subtypes it is embedded in. As Jean Borie accurately pointed out,[90] the postulation of the existence of an individual type seems a paradoxical statement, as type implies a collective, a series of possible instantiations of the abstracted, and that clashes with individuality. But it is not a futile element in Lucas' scheme. As it is the individual

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Burdach, Lucas adopted the strategy of trying to solve conflictive issues by the imposition of a new categorical divide above them. Though sometimes it opened new possibilities, it eventually lead him to to surcharge his system with oversubtle and hollow distinctions, and loose sight of his initial intention of having a strong empirical grounding.

[89]See Burdach, *Traité de Physiologie* (1837), vol. II, p.245, quoted by Lucas, vol.1, p.179.

[90]See J. Borie, (1981).

type which, by the genealogical connection,[91] is the origin of the familial type, and through it of the national, racial, or any other collective type that one can discern under the level of species. It is only the species and the individual that has a definite status. The other sub-types are, so to speak, derivative. It is the conflict between the individual and the species that produces the new organism. Each parent's individual type has the representation, so to speak, of the interests of all its ancestors, and these interests can be related is by the degree of resemblance to wider and wider genealogical groups: the family type, the national type, the racial type. They all had stakes in each and every conception. The more distant the mating partners, the stronger the clash. Crossings between species, as they involved the untouchable, were therefore doomed to infertility. Heredity, in a word, is the procedure by which the past generations influence the present ones. Although the species' type is strictly ahistorical, the individual type, by freezing the explorations of inneity, incorportes a historical dimension. Inneity, in this scheme, pushes the individual's constitutions to explore the possibilities that the types have. Heredity tends to re-produce the results of such explorations.

The separation between types and actual constitutions serves the purpose of allowing a multilayered causation. The continuity, so to speak, of group characters in the individual instantiations. It allows the space for a direct causal link (rapport) between an individual's original makeup and that of its offspring, leaving aside the vagueries of its actual life story; which is a move precluded to other authors like Prichard, who could not find how to separate the individual's initial (congenital) constitution's contribution to its offspring's hereditary makeup, and that of its actual adult constitution. Lucas separation gives the latency of transmission, the dispositional causation, atavistic re-occurrences, homochrony, and all the earmarks of heredity a deeper, more fundamental meaning. The gap between hereditary disposition and actual occurrence was the same one between the type's causal input and its actual embodiment. If we wanted to use an anachronistic analogy, we could say that Lucas division between specific type and individual type (with all its intermediaries) is an idealistic ancestor of the modern distinction between a species' genome and an individual's genome. Each instantiation would be, in modern terms a phenotype, in Lucas' termsa constitution. The analogy is however quite fragile, but the complexity of the schemes similar.

The outcome of such elaborate theorizing was that Lucas' empiricist pretensions contradicted this heavy reliance on an idealist and dualist strategy. As in the case of the status of heredity and inneity as forces, Lucas claimed that the individual type was nothing but the collective, inductively arrived concept —which is at odds with the explanatory use he makes of it.

To deal with the problem of mental or moral inheritance Lucas again made use of the German physiologist Burdach's talent for creating dichotomies. In a further version of material dualism, Burdach and Lucas divided the constitution or organization of organisms into two components: the plastic (or material) and the dynamic. Heredity and inneity acted upon both components in a similar fashion. The

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[91]Genealogy, however, was not the only source of resemblance for Lucas. Like Prichard and many others, Lucas believed that a similar external (climatic, for instance) stimuli could trigger parallel variations in the same or different species. Thus the white fur of arctic animals, or the geographical variations in transplanted plants.

plastic referred to all the properties of the constitution that derived from the matter and material structure. The dynamic, although always rooted in the material (i.e. could not exist without it) consisted of the "emergent",[92] vital properties, among them the set of all the mental qualities and dispositions.

Lucas opposed the dualist, vitalistic approach of the Montpellerian school (Virey, Lordat), and firmly defended the inheritance of all mental dispositions, with the possible exception of "genius", which he believed was always a product of (spontaneous) inneity. He however was never very far from a vitalistic ontology as his dynamic properties, like Burdach's, were described as underdetermined by the physical underlying states, and thus in need of a higher level of determination. As with his forces and types, Lucas fell in the nominalistic trap in his division between mind and matter, and made all his rhetorical claims to a purely empiricist base quite untenable.

Lucas use of evidence remains nevertheless memorable. Within his highly rationalistic, branching scheme, he took the medical tradition of proving causal links by carefully selected cases, usually of striking improbability, and with the rhetorical help of a well constructed narrative produced a every convincing effect. Most of his readers were converted to the cause of Heredity. Not so much to that of Inneity, but the role of spontaneous, original (in the sense of congenital) variation was sufficiently well made to impress acute readers, like Darwin and Spencer.[93]

The structure of Lucas' two volume treatise is a reflection of his very complex, multilayered approach to the individual's constitution and the forces that impinge on it. He arrived at that scheme after carefully assimilating both the long medical tradition of dealing with hereditary transmission, and the more recent attempts by physiologists, ethnologists and naturalists, to incorporate those findings into hypotheses. But the field was riddled with contradictions and inconsistencies, as the many sceptics, within and outside the medical community, had repeatedly pointed out. That Lucas meticulously followed his rationalistic scheme, argued his way through (or around) all the objections, and found abundant evidential support for all his claims, has been taken to be a sign of stubbornness and infatuation with his fanciful ideas.[94] It is better understood, I believe, as an admirable synthetic effort that both showed the existence of a valid, unique domain of biological phenomena in need of a general theoretical and experimental effort of comprehension and understanding.

Hereditarianism, as a medical, psychiatric, and social movement in 19th century France was influenced but not created by Lucas, and perhaps his complicated analysis, and effective rhetoric contributed to its long life. The fact, on the other hand, that he failed to convince many biologist or physiologist with his —by then already slightly outmoded— causal explanations, should not make us move his work out of the central role it played. In many other ways Lucas' book determined the profile that the idea of (biological) heredity was going to have in France for the next forty years after its publication. One example is his categorization of the main pathways of hereditary transmission, which is to be found repeated over and over by late 19th

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[92]I'm taking the license again to use this anachronistic term.

[93]Darwin wrote on the margins of his copy of the *Traité* about the reality, and independence of variation.

[94]This is the main position behind B. Balan's "Prosper Lucas" in C. Bénichou,(1989) *L'Ordre des Caractères*, pp-49-71.

century authors were defined in it: direct, indirect, atavistic, and "d'influence" .[95]

As we shall see, the following generation of researchers in heredity, read Lucas' work attentively (as the only general treatise on a subject they had come to regard as crucial), and profited from both his array of evidential support, and the subtlety with which he analyzed hereditary causation.

[95]See the review that E. Littré wrote of Lucas' treatise, which is more like a synthetic exposure of his system, and which was published as a dictionary definition of *Hérédité* in 1853 (in *Dictionnaire d'Anthropologie*, in vol. 42 of Migne's *Nouvelle Encyclopedie Theologique*) and that was to be for many years the base for most authors definition of the term. When in 1903 Yves Delage made a review of all the heredity theories of the late 19th century (*L'hérédité et les grandes problèmes de la biologie*), these divisions established by Lucas still seemed to him the most useful.

Chapter 6

Early Galton; the roots of statistical heredity

Francis Galton (1822-1911) is well known as both the founder of the British Eugenical movement and as an intuitive theoretician that set the basis for the early 20th century's impressive development of statistical tools of analysis of data that revolutionized the natural and the social sciences. Much historical attention has recently been given to the development of his work, from both an externalist and an internalist perspective (to use this outmoded but useful distinction). Although in many senses Galton's case provides the ideal elements for a social explanation of the inner workings of a science in the making (as Heredity was in his time), none of the attempts of exploiting them has, in my opinion, been completely successful, and one reason of the shortcomings is that they have tended to take at face value Galton's (and his followers') own accounts of the origin and place of the work in the history of science. But much of the novelty of the work, especially in its early stages, was exaggerated by them; and the work of many sociologists have been geared to explain a novelty and originality claimed by those interested actors, but they, perhaps unwillingly, ignored several of their intellectual debts.

There are many answers yet to find from the perspective of the conceptual history of biological heredity in order for the social accounts of its development to acquire more depth. Given that the concept of biological heredity was, at the mid-19th century at what could be described as a "crossroads" between medicine, psychiatry, ethnology, natural history, horticulture and animal breeding practices, the number of avenues that lead to the synthetical achievements of Francis Galton have proved to be many; more than what at first sight was imagined. His cousin Darwin's towering figure, on one side and his follower Karl Pearson's idiosyncratic interpretation of his life's work have, I believe, distorted our perception of the field over which Galton laboured.[1]

Recently several previously unacknowledged roots to Galton's work have been uncovered. R. Olby (1985) has shown how, H.T. Buckle, H. Spencer, G.H.Lewes, in the 1850s and 60s had already set many of the parameters that Darwin and Galton were to retake in their considerations of human heredity. V. Hilts (1967) and R. E. Fancher (1983), on the other hand, have shown how ethnological (rather than "eugenical" or "Darwinian") considerations were the triggering element for the early

[1]On the other side, the justified contempt that contemporary writers feel for both the ethical standpoint and the evil consequences the eugenical movement and its interventionist social practices have been anachronistically projected over Galton, and have been allowed to play a bigger role in his motivations than would otherwise seem proportionate.

hereditarian researches of Galton, and how it can be linked to schemes developed in several other authors' works, like Prosper Lucas'.[2] A general, and important connection is still to be made, I believe, between the long hereditarian tradition within medicine, of which Lucas' oeuvre is a crucial moment, and the statistical approach that Galton developed. The present chapter is focused towards bridging that gap by relating the evidential procedures of such tradition, and the original statistical developments by Galton, and the explanations that one and the other approaches provided of irregularity and variation in relation to hereditary transmission.

6.1 Irregularity, variation and the hereditary

Prosper Lucas wrote that Antoine Louis' reasons for denying the very idea of any hereditary (causal) communication of constitutional (organizational) features from parents to their offspring can be in the end reduced to one main objection: the lack of constancy, of universality and of continuity in the succession of characters within a family.[3] This chronic irregularity of all things hereditary was of course a fatal drawback if what the theoretician was after was a univocal, universal and sufficient causal link, and certainly was to be recurrently mentioned by sceptics as the unsurmountable obstacle for any attempt at a general theory of heredity. The French medic Sersiron (1836), we mentioned, considered the stable, always transmissible characters of the species' type as the only ones that deserved the label of hereditary.[4] Individual, idiosyncratic variations, however subtle or dramatic, could always fail to reappear, and could be eliminated from the progeny, and must necessarily be ruled by another, and accidental (i.e. unimportant) set of causes. What Sersiron, and most other sceptics failed to realize is that the very idea of an hereditary transmission among the living organisms originated with the observation of the existence of transmissible peculiarities, or in more modern terms hereditary variations. Herbert Spencer eloquently described this apparent paradox

> the evidence which proves Heredity in its smaller manifestations is the same evidence which proves Variation; since it is only when there occur variations, that the inheritance of anything beyond the structural peculiarities of the species can be proved[5]

It is not difficult to see that all the oldest historical mentions of hereditary transmission, both in humans and other species, are linked to the transmission of striking deviations from type, from Aristotle's macrocephalic tribes to Maupertuis'

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[2]See R. Olby (1985), V.L.Hilts, (1973), R. E.Fancher, "Francis Galton's early ethnography"(1979).

[3]Lucas, 1847, p.312. To be fair, this only addresses one side of Louis' objections, the external side of the empirical facts. The other side, internal, was (as we mentioned) the impossibility of a solid to solid causal link between the parents' organizational features and the offspring's, given the prevailing views of generation in the mid-18th century.

[4]We have seen how Cadogan and Henning in England (Ch.4) and Sersiron (Ch.5) in France opposed the idea of hereditary transmission of constitutional diseases because the abundance of exceptions made the causal connection claimed by hereditarians unlikely, and forced them to a contorted predispositional account which was "even more difficult to prove"(Henning).

[5]H. Spencer, 1864, vol.2, p.258.

nègre blanche. Heredity as an explanation of homogeneity within groups was a late arrival; a 19th century substitution when all other accounts had failed. By the middle of the 19th century however the belief in the strong hold of heredity over the specific, type related structural characters (that Cuvier had so thoroughly analysed) was widespread, while, quoting Spencer again, it was "not universally admitted that non-typical peculiarities are inherited..."

> Some naturalits —Spencer adds— seem to entertain the vague belief that the law of Heredity applies only to main characters of structure, and not to details[6]

Once hereditary transmission had been transformed into a main biological phenomenon, responsible for the stability of the taxonomical building, the irregularities of transmission that medical men, breeders and some naturalists had argued about for decades became a mainstream biological problem. The burden of proof seems to have shifted towards those who saw in hereditary transmission of all constitutional peculiarities a rule rather than an exception. That Charles Darwin was among them turned out eventually to be determinant.

As we have shown repeatedly, the question of what could and could not be transmitted has two main approaches: an external one, in which the ostensive showing of particular facts of transmission provides the reason for a belief that such character is, or can be, under the aegis of heredity. And an internal one, by which from an assumed, or inferred route of transmission (physiological or other) the belief of the reality of a claimed hereditary influence was assessed. For decades, after the 18th-century failures based on generation disputes, the second approach was considered too risky. Many authors made a point of consigning, at least rhetorically, the actual details of heredity to a mysterious area. Robert Olby has turned our attention to the fact that as late as 1883 one could read in the *Encyclopedia Britannica* that

> a mysterious transmission of properties has still to be accounted for and interpreted in terms of the physiological and morphological, the chemical and physical, composition and properties of the germinal matter of parent and offspring.[7]

Spencer, along the same lines, wrote that "a positive explanation of Heredity" was not to be expected given the state of biological knowledge of his day.

> We can look for nothing beyond a simplification of the problem; and a reduction of it to the same category with certain other problems which also admit a hypothetical solution only.[8]

He proceeded, as is well known, to develop a hypothetical model for heredity of his own, and was followed in similar pursuits by both Charles Darwin and Francis Galton.

The first theoretician of general heredity, Prosper Lucas, we have seen, decided that only a combination of opposing forces (with differential hold on the different

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[6]idem. p.239.

[7]quoted in R. Olby, (1980) "Human Genetics, Eugenics and the State", p.2 (of manuscript). Gloria Robinson (1979), following the exhaustive investigation of Yves Delage (1895) described all the different "transmission theories" that were proposed during the second half of the 19th century in order to fill the gap here described.

[8]Spencer, op.cit., p.255.

kinds of characters) could really account for the exuberance of the phenomenology he wanted to cover under his theory. Lucas' a prioristic, rationalist approach was not however very palatable to the more influential among British authors who followed his tracks, who accepted the centrality of heredity but wanted a different approach to it, both in its empirical, fact gathering, external side, and in the internal (physiological) models they were willing to accept as tenable.[9]

Several among the British readers of Lucas' *Traité* took it as a departing point when attempting to establish some order in the field of heredity. Among the most influential were George H. Lewes, Herbert Spencer and Charles Darwin himself. Francis Galton, who was to change definitively the field of heredity with his statistical approach, was probably more influenced by Lucas through these three authors than by a direct reading of him.[10] Lucas had taken from his tradition, the French medical and physiological tradition, both the fact collecting as a base for inductive inferences, and the dubitable, property based, dualist physiology. The object of the former was to establish independently the power of heredity over every different kind of physical or mental character, within his hierarchical view of characters. The latter provided the basis for his rationalist analysis Both left an important mark in the way heredity was studied in the mid-19th century. Attempts at going beyond these unconvincing (medical) approaches lead on the one hand to a more sophisticated use of statistics in the handling of data, and on the other to the search for "transmission" hypotheses more in line with the advances in cytological, embryological and some experimental research.

6.2 From story-telling to statistical tables

When Charles Darwin, in the chapter dedicated to inheritance in his *Variation of Animals* ... (1868) wanted to convince his readers of the pervasive and impressive action of hereditary transmission among living organisms he wrote

> It is hardly possible, within a moderate compass, to impress on the mind of those who have not attended to the subject, the full conviction of the force of inheritance.

The solution he sees, in order to try and remedy this is to

> select a few facts of the kind which, as far as I can judge, have most influenced my own mind.[11]

To produce well selected, striking, authenticated cases of hereditary communication, and to collect them and list them when persuasion about the reality of the phenomena was needed, was an old procedure. Medics and naturalists used it

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[9]Lucas did have a few followers in Britain who were willing to buy the whole ticket of his, dualistic, quasi-vitalist approach to heredity, basically among Medics. Dr. Elam's essay (1869) and Dr. Winn (1869, see appendix 1) are eloquent examples of this.

[10]Galton's copy of Lucas work (in the Galton Lab. University College) seems quite unused, and is not annotated; R. Olby (1985) and V.L.Hilts (1974) have been the historians who recently have more clearly appreciated the influence of authors like Lewes and Spencer in Galton's work, and recognized the debt both of them have with Lucas. Concerning Darwin's use of Lucas' work see Churchill (1987).

[11]Darwin,(1868), vol.2, p.4.

repeatedly during the 17th, 18th and 19th centuries. Darwin felt it was a valid one, and chose, as his first example one of the more striking, and more mentioned cases, that of the English Lambert family of "porcupine men" (which was first reported in the *Philosophical Transactions* in 1731)[12] "whose skin was thickly covered with warty projections which were periodically moulted". Used by many authors before Darwin (Blumenbach, John Hunter, Prichard, John Adams, W. Lawrence, Lucas) the "porcupine" story can be said to be a "standard case" that shows the transmission within a family of a dramatic variation; of a peculiarity that "had appeared only once or twice in the history of the world" in an individual, "but have reappeared in several of the children and grandchildren". Authors could not fail to make the inference that a constitutional alteration like this, transmitted repeatedly within an isolated (inbreeding) family, could easily be the origin of a new variety, a new race, or eventually, for some, a different species. Whatever the origin of these astonishing constitutional transformations, the fact that they could pass through generation into the offspring seemed proven. Other alternative explanations to heredity of the coincidence of such deviations from the type seemed highly implausible, so the case militates for the hereditarian position. There are other typical cases that tend to reappear in the literature, both dealing with human beings and with animals (mostly domestic). As Lucas' research so thoroughly proved, by the mid-19th century there were cases, observed on the main by reliable authors, that could "prove" in this way the (potential) hereditary transmission of many sorts of variations of virtually any kind of character. And as Darwin also wrote, "the evidence of inheritance is more striking when we consider the reappearence of trifling peculiarities",[13] the proliferation of candidate cases for hereditary transmission knew practically no boundaries, as the ancient list of strange occurrences shows (many of which had been updated by recent authorities such as Blumenbach, Haller or Meckel).[14]

The sceptics, on the other hand tended to point to the multiple exceptions, the unreliability of some descriptions, or tried to produce alternative explanations for the recurrences. Some, like the sadly famous Dr. Knox reacted drastically. In reference to another multiply quoted example of dramatic hereditary variation, the Ancona Sheep, he flatly replied

> When I am told that there is a short-legged race of sheep somewhere in America, the product of accident, my reply is simply, I do not believe it.[15]

Depending of the kind of character that a case was supposed to illustrate; depending on how well defined, and how believable it's link to heredity (on transmitted constitutional dispositions) was, the degree of scepticism it would generate would vary. As we have seen before, the case collecting approach left room for all sorts of scepticisms. What turned out to be, the strongest mid-19th century

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[12]Not 1755 as Darwin has it. The reference is: Henry Baker, "A Supplement to the Account of a distempered skin" *Phil. Trans. Roy. Soc.* No.424, vol.49, part 1.5. The family was followed through several generations, and updtes of the pedigree of the mutation were given later by William Lawrence, in his *Lectures*, 1819, pp.449-451, and by James Paget, 1857, in *Medical Times*, p.192.

[13]Darwin, op.cit, p.5

[14]Blumenbach's collection is in his *Biblioteca Medica*, and Haller's in "Similitudo Parentum", chapter IV, vol. I of his *Physiology*.

[15]R. Knox, 1850, *The Races of Man*, p.67.

challenge to hereditary inferences was precisely focused on the unreliability of such procedure. It was raised not by a naturalist or a medic, but by a historian, who was particularly weary of any talk on hereditary transmission of moral or mental characters: H. T. Buckle. Recognized by Darwin, Lewes, Galton and De Candolle as a well articulated (though exaggerated) challenge to normal procedures, and by recent historians (especially Olby and Hilts) as an important impulse for the use of statistical method in hereditary inferences, the following paragraph appeared as a footnote in Buckle's renowned *History of Civilization in England* (1859):

> We often hear of hereditary talents, hereditary vices, and hereditary virtues; but whoever will critically examine the evidence will find that we have no proof of their existence. The way in which they are commonly proved is in the highest degree illogical; the usual course being for writers to collect instances of some mental peculiarity found in a parent and in his child, and then to infer that the peculiarity was bequeated. By this mode of reasoning we might demonstrate any proposition, since in all large fields of inquiry there are a sufficient number of empirical coincidences to make a plausible case in favour of whatever a view a man chooses to advocate. But this is not the way in which truth is discovered; and we ought to inquire not only how many instances there are of hereditary talent, &c, but how many instances there are of such qualities not being hereditary. Until something of this sort is attempted, we can know nothing about the matter inductively; while, until physiology and chemistry are not much advanced, we can know nothing about it deductively[16]

Fascination with exceptional cases, and scepticism about them, or about their use as empirical support for this or that theory, is perhaps one of the central characteristics of the story of the hereditary we have been trying to describe. Many recent historians have condemned that attitude as belonging to a pre-scientific period where midwife tales dominated over sound, observational or experimental empiricism. Darwin himself has been accused of being too credulous. The emergence of statistical analysis as a sound base for inductive inferences (together with the increase in the use of experimental crossings pioneered by hybridists) has been rightly claimed as a major breakthrough that broke the vicious circle of claim and counter-claim.[17] There is however in this appreciation a lack of perception of the extent to which the case quoting, story-telling approach to evidence has a continuity, both in its rhetorical workings and in its probabilistic rationale, with the statistical approach; specially with the early statistics.

Buckle was actually being very partial and unfair when he described as irrational the procedure of case studies, as Darwin poignantly reminded his readers, many of the recurrences in the same family of peculiar characters "cannot be due ...to a coincidence, but must be consequent on the members of the same family inheriting something in common in their constitution". The reason being, as he wrote in *The Origin of Species*, before he knew of Buckle's objections, that "the mere doctrine of

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[16]Buckle, 1857, *Hist. of Civ.*, vol.1, p. 161. Quoted also by Lewes, op.cit.,p.376, and by R. Olby (1985) in an appendix.

[17]See for instance the accounts of Francis Galton's work in R. S. Cowan several articles and thesis, and those chapters dedicated to the subject in Stigler (1987) and Porter (1988).

chances almost compels us to attribute its reappearance to inheritance".[18] Darwin, as I expect to show, was not alone in feeling "compelled" to believe that hereditary transmission rather than external influences or mere accidental recurrences were responsible for the observed patterns,[19] and a part of the seduction of the adequate case, anecdote or story was certainly based on a rational probability assessment; Darwin, I will show below, borrowed from Maupertuis the probabilistic argument that could answer the sceptics.

The other, complementary aspect of the "compulsion" to attribute is the rhetorical role of the stories themselves; their capacity to strike a chord in the imagination, and the preconceptions of the reader. The intimate working of these two elements in the arrangement and presentation of the evidence was what gave the method of induction (so poorly regarded under other lights) the effectiveness it had for centuries, specially in the medical sciences.

Jacques Roger in his work on Fernel described admirably, though somehow too lyrically, the rise of this case- collecting trend among medical men in the 17th century, making a remarkably similar protest as Buckle's of the irrationality of it all

> La fin du siècle...verra la ruine définitive du rationalisme aristotélicien. Contre la logique de la raison, on invoquera l'autorité des faits. Et plus ces faits seront extraordinaires, plus ils seront invraisemblables et déraisonnables, plus ils seront recherchés avec curiosité et acuelli avec intérêt. Les fables les plus extravagantes débitées par le l'Antiquité ou du Moyen Age, histoires de monstres et de prodiges seront soigneusement compilées. Dans cette anarchie de la nature, la raison perd ces droits.[20]

But Roger's condemnation is exaggerated, I believe. His description fails to take into account the surrounding, theoretical general assumptions that justified such tradition in the use of evidence among medics and other scholars of the Renaissance and after.[21] Other French historians, like Foucault (1966) and Jacob (1970), have, in their accounts of the development of biological thought, fallen into the same trap of considering the magical, mystical associations of these fact collecting procedures without balancing them with the rationale that both intuitive probabilistic considerations, and the causal hypothesis (physiological or other) of the period could

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[18]Darwin, *Origin of Species*, 1859, 1st. ed. p.13.

[19]Though elements like the "distinctiveness" of the characters (so they could be clear enough to facilitate their identification), and the reliability and care with which the genealogical research had been undertaken, as well as the elimination of other explanations (like contagions in case of disease) were all important to Darwin, he relied entirely on the accumulation of facts, from all sort of sources, that took the existence of transmission beyond reasonable doubt.

[20]J. Roger, 1964, p. 16.

[21]Some may find somehow extreme the relation I am making between the Renaissance bookish fact-collecting and Darwin's more modern, careful sifting through the facts according to several degrees of reliability and authentication. But what I find is more a continuum between the two, and not a real rupture. A very illustrative "intermediate" character, I believe, is Albert Haller, who at the same time made an apology for bookish, exhaustive collection of data from ancient observers, however "credulous or fantastic", and an independent assessment of their virtues and probable truth.

provide.[22] A similar failure I find in many of the authors who have ventured into what they call the "prehistory of genetics", that is to say, pre-Darwinian hereditary theorizing.[23]

The rhetorical, evidential power of narratives has elicited much interest among historians and philosophers of science recently, specially after some literary scholars began to apply their analytical tools to some scientific discourses, like Darwin's.[24] Literary scholars have fortunately also paid attention, even before historians of science, to the early and mid-19th century hereditarians in France and Britain. They could not fail to detect the powerful rhetorical work that story-telling, and case quoting, emanated from the medical tradition, was playing in their works. In his insightful *Mythologies de l'Hérédité* (1981) Jean Borie, when analysing Lucas' *Traité* correctly describes it as constituting of two well defined parts. The first volume dedicated to the a priori theorizing in which he distinguishes the specific and the individual types, and gives an account of his laws of heredity and inneity (see chap. 5). The second volume, dedicated to the practical side, where proofs, arguments, examples are accumulated. In this part of Lucas work (which is the one Darwin perused more thoroughly), Borie sees the emergence of

> cent anecdotes, exposés des cas, rappels des faits historiques, en un mot, des personnages. C'est même le charme de tous les ouvrages entarsés par les archivistes de l'hérédité et de la déviance d'accumuler pour nous les pièces d'un immense et fabuleux musée: illustrations, atlas, arbres généalogiques, et surtout, exposés de cas, par dizaines. Un exposé de cas est toujours un recit biographique, un petit roman.[25]

Borie is interested in the narrative structure these hereditarian tales provided for 19th century writers like Zola,[26] but he is aware of the inductive role they are playing in the medical men's quest for a general theory. "Le médecin ne s'interesse, dans le cas qu'il étudie à rien d'individuel, il ne retient que les éléments typiques de la situation".[27] This inductive performance has a part of its leverage in the fact that a striking, well knitted narrative will always have a causal backbone that can lead the reader through it in an assured manner. Whatever mechanism drives a story or an anecdote, there has to be causal link between the successive stages. A story, it can be argued, is always a way to exemplify a causal connection, general or not, in such a way as to impress the spectator. The individuality, the details of the cases add life, poignancy to the experience of assimilating the message, but if there is not some kind of cause-effect thread, however fanciful or contorted, in relation between the initial and the final states, the efficacy of the tale diminishes. In the case of hereditary stories, the connection between the parents' constitutional features (usually exceptional) and those of the children is is the narrative backbone: the casual implication is reinforced by the sense that no other account can compete with it (and

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[22]M. Foucault (1966), F. Jacob (1970).

[23]See for instance Bowler, 1989, *The Mendelian Revolution*.

[24]Gillian Beer (1983) is a leading figure in this area. See also Adams (1916), Morton (1984), Borie (1981), Malinas (1985).

[25]J.Borie, 1981, p.87.

[26]A similar study for the British case has been done by Peter Morton, whose splendid 1984 book *The Vital Science* traces the influence of hereditarianism on writers like Thomas Hardy and Samuel Butler.

[27]J.Borie, op.cit.,p.87.

here the probabilities enter). The medical views of the human body, the theories of temperament, or of constitution, however revised and updated with new physiological accounts, however transformed by the new concept of organization, provide the solid ground for the belief in this communication: peculiarities, idiosyncracies, the tree of differences that constitute the individual, are there, on top of all the common, specific, structure, at the outermost level where most of the interactions with the external world take place. Human beings, among which the differences and subtleties of temperament far outweight the importance of the common, easily generizable background, can better be understood by taking those subtleties into account. The constitutional differences that make the individual unique can be transmitted, in some degree, to their offspring: that is the root of family resemblance, of differences between families, and in the end of differences also between groups, social classes, nations and races. A transmission story could always be transformed into a foundation story. Lambert could always be the Adam of an entirely new race. A similar event could have been the origin Aristotle's macrocephali, or for that matter of the Roman family Nasones, or of each of the Highlander family in Scotland. As King Frederick of Prussia's tall guards, all the stories of individual heredity could, and usually did, become stories of groups, blessed or condemned by their physical and moral, biological heritage.[28]

A transmission story not only established then the fact that a peculiar character could be transmitted, but contributed to the overall impression that heredity was the rule, and non heredity the exception, and that it touched all kinds of characters. But the story, it is important to point out, did not work on its own. It needs the underpinning of plausibility: conceivable ways of transmission under the existing physiogical (and ontological) opinions. A clear proof that such is the case can be found in the case of what has come to be known as imaginationism. Which was the hypothesis that a considerable part of the individual's idiosyncracies, family resemblances and other peculiarities like birthmarks were to be explained by the influence of the mother's mental states, ideas, impressions and imagination during pregnancy (especially during the first moments before the stamen, or the embryo became completely formed, organized and individualized). Although masses of cases were collected (by both serious and cranky authors) during at least a couple of centuries the belief in the efficacity of these stories to show the causal connection they were supposed to diminished progressively in parallel with the untenability, in physiological (or ontological) terms of the connection between the mother's ideas and the child's actual constitution. Nevertheless, the power of the array of stories survived longer than the hypothesis, and authors like Haller or Bonnet, without giving any backing to the imaginationist explanation, did however describe the evidence and claim a suspension of judgement. Similar attitudes survived well into the 19th

[28]Like the Lambert case, all the other I mention here are canonical in the tradition of hereditary thought. That Romans used to have some families whose name was based on a hereditary physical peculiarity, like having a big nose, head or whatever; that in Prussia the increase in average height of the population of St. Petersburg was due to a king's whims, that all the inhabitants of certain valleys in the Scottish Highlands share strikingly similar features, and that these can on occasions be traced back to a common ancestor, Lord of the valley, who has the reputation of having been quite promiscuous, were all stories repeated time and again.

century.[29] Imaginationism was only one of the many attempts at explaining the origin of individual variation. It was always to be a problem difficult to cope with, and whose solution as we said became crucial as heredity took center stage. Nevertheless, as Lucas saw clearly, only the reality of the phenomena needed to be granted for genealogy (plus heredity) to substitute static taxonomy in the classification of characters. Lucas' individual type, with its power of affecting, through heredity, the constitution of wider and wider genealogical linked human groups, could be re-interpreted in a non-nominalist fashion. The "decoupaged" (as Schiller called it) or analysed nature of 19th-century concept of biological organization facilitated this move. The individual's constitution could be seen as an assembly of elements, relatively independent in their origin and peculiarities, determined by the interaction of a multiplicity of influences. And among the ones that could no longer be denied was heredity, the conservative influence. Buckle's scepticism was surely misplaced because, as Lewes argued, although one detected more clearly the effects of heredity with deviations and differences, it was its homogenizing role in subspecific groups that really gave it its importance; its capacity to spread a peculiarity within a genealogical group. Without its influence, there was no longer any other way of accounting for many such stabilities. Charging against Buckle, Lewes writes

> It must be admitted that many of the cases collected to prove hereditary transmission have been allowed to pass unchallenged by criticism, and many of them are worthless as evidence; but is Mr Buckle prepared to deny that the tendencies and peculiarities of men depend on their organisations? If he is not prepared to deny this, his scepticism is illogical, since there can be no shadow of doubt that organisations are inherited. He will not say that it is mere coincidence which preserves intact the various "breeds" of animals...Unless parents transmitted to offspring their organisations, their peculiarities and excellencies, their would be no such thing as a breed, or a race. The cur would run the same chance as the best-bred dog of turning out valuable.[30]

Two elements thus coincide here to undermine Buckle's scepticism towards story-telling: one is the strong belief that in the case of humans, and of domesticated breeds, there is a sense, a purpose in the increased variability that is observed: the perfectibility (or its complement the capacity for degeneration) of the genealogical line. The other element, and perhaps in the end the stronger one, is the, conscious or not, application of the doctrine of chances to the use of cases. The only way to explain the familial pattern of distribution of characters within given species, the argument goes, is an uneven distribution of chances due to the causal link provided by heredity between the members of the same genealogical line. The first time this chance analysis was explicitly made was in Maupertuis' well known study of the six-digitism within the Ruhe family in Berlin. In it the French 18th century savant wanted to demonstrate that hereditary transmission of a striking constitutional variation could take both the mother's or the father's route towards the offspring (thus undermining preformationism). Polydactily, having first appeared in a woman of the family (Elizabeth), had reappeared in three generations of Ruhes. Jacob Ruhe was the

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[29]See Haller, "Similitudo Parentum", quoted above, and more recently Thomson's (1835) "Generation", in *Practical Cyclopedia of Medicine.*

[30]G. H. Lewes, 1860, "The qualities we inherit from our parents", chapter 12 of *Physiology of Common Life*, (1859), pp. 376-377.

intermediary between Elizabeth and her grandchildren. Maupertuis stressed then that chance could have been responsible for the first, spontaneous, variation

> But if one wished to regard the continuation of polydactily as an effect of pure chance, it would be necessary to see what the probability is that this accidental variation in a first parent would be repeated in his descendants.

He proceeded to solve this question with some method

> After a search which I have made in a city which has one thousand inhabitants, I have found two men who had this singularity. Let us suppose, which is difficult, that that three other have escaped me; and that in 20,000 one can reckon on one six-digited: the probability that his son or daughter will not be born with polydactily at all is 20,000 to 1; and that his son and his grandson will not be six-digited is 20,000 x 20,000 or 400,000,000 to 1; finally the probability that this singularity will not continue during three generations would be 8,000,000,000,000 to 1; a number so great that the certainty of the best demonstrated things of ohysics does not approach these probabilities[31]

In a recent analysis of Maupertuis views on hereditary transmission, Isabel Sandler points out correctly that he was using, in the above reasoning, the probabilistic principle of coincidence, with an epistemological interpretation of probability.[32] What is interesting from our point of view is that such principle of coincidence means simply that two events, if they are not probabilistically independent, must be, directly or indirectly, causally connected (one cause the other, or share a common cause). Sandler makes a point of insisting that Maupertuis understands chance (or probability) epistemologically, as a reflection of ignorance. But it is the proof of causal connection that really matters for his argument. In the case that Maupertuis chose to explore, the alternative explanation to hereditary transmission is that some external common cause could be causing the reappearance of the same trait (polydactily) in different individuals of the same family, but even if it existed, it seems highly implausible that such cause could really "pick out" the members of the family without them sharing at least an exceptional predisposition or tendency, which in its turn would have to be hereditary.

Maupertuis' analysis makes evident what most believers in the hereditary transmission knew to be true: that their collection of cases, tales and anecdotes of familial patterns of reoccurrence of peculiarities (both normal or pathological) warranted their inferences to hidden causes because there were not many alternative explanations.

Whereas groupal homogeneity (the existence of races and varieties) could believably be explained by external causation,[33] any series of peculiar familiar re-

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[31]Maupertuis, *Lettre XIV*, p.308. The translation is taken from B. Glass, 1959.

[32]In which she rather otiosely proves that Maupertuis was no precursor of Mendel, but provides however an insightful analysis.

[33]Both Prichard and Lucas, for instance, were willing to accept that the triggering of some potential constitutional characters by external stimuli, like climate, could not be eliminated as an explanation for geographical variety for instance, and thus accepted that resemblance was not always due to parentage, and could sometimes be due to common causes.

occurrences could not. The real difference between Maupertuis' and Darwin's (or Galton's) times was that in the 18th century heredity had not yet been generalized, (and de-pathologized), and transformed into a central explanatory force in biology, in such a way as to make it acceptable that the long run consequence of many successive transmissions of peculiarities within families was the emergence of the other stable, genealogically linked, groups known as varieties, races or, in the case of domesticated animals, breeds.

Many posterior authors were very impressed by Maupertuis' simple calculations (not very common in biological problems) and Darwin himself made a similar, copy-cat argument in his *Variation...* , only exaggerating the proportions and leaving the pencil work to someone else:

> If the occurrence of the same unusual character in the child and parent cannot be attributed to both having been exposed to the same unusual conditions, then the following problem is worth consideration, as showing that the result cannot be due, as some authors have supposed, to mere coincidence, but must be consequent of the members of the same family inheriting something in common in their constitution. Let it be assumed that, in a large population, a particular affection occurs on an average in one out of a million, so that the a priori chance that an individual taken at random will be so affected is one in a million. Let the population consist of sixty millions, composed, we will assume, of ten million families, each containing six members. On these data, Professor Stokes has calculated for me that the odds will be no less than 8333 millions to 1 that in the ten million families there will not be even a single family in which one parent and two children will be affected.[34]

Darwin felt very confident that this calculus actually allowed him to take as proof all the evidence he had gathered of cases of hereditary transmission of variation from the medical, agricultural, zoological, botanical, and other sources (provided other causes were eliminated and the cases were well authenticated). The sceptical views of Buckle and the likes could be well ignored. The rationality of using isolated cases as a base for inductions towards causal links, and the establishment of certain patterns of regularity within the irregularity of hereditary links was assured by Maupertuis' example of the probabilistic "principle of coincidence". Of course, as Maupertuis showed, and as Darwin was well aware, in order to prove hereditary transmission by this "external" evidence, two important problems remained for the inference to be solid. One was the elimination of all the other possible causal sources that could compete with heredity, and which could only be achieved by having as homogeneous as possible groups in all other respects (so the prior probabilities assigned by Darwin and Maupertuis would be justified). The remaining problem is the oldest one: the explanation of the exceptions, the roots of difference and inhomogeneity. It is in these two counts that story-telling needs the underpining of the background physiological theories, of the theories of reproduction, and of the more general evaluation of the importance of other, external, influences. But in this "weakness" story-telling is no different from the statistical evidential procedures that superseded it. In fact, these rather than signifying a radical rupture with outmoded, irrational ways of dealing with facts, can be seen as a completely natural development of it. A way of clarifying the way in which an external pattern of recurrences can legitimately be said to justify a

[34]Darwin, vol.2, p.448.

belief in internal transmission. To mark a distance from the uncritical use of fact quoting that had given some medical hereditarian studies a shady reputation was, it must be added, a part of the motivation.

In a sense, when statistical tables began to be used within the realm of hereditary theorizing they were a new way of telling stories, of capturing both the probabilistic logic underlying case quoting, and the rhetorical power of the narrative.

In relation to heredity, statistical tables, it can be said, began as organized collections of transmission stories that progressively lost their individuality and whose narrative detail became gradually abstracted. This is evident, for instance in the way alienists, mainly in France, constructed and handled their first tables (modern authors say they did it "naively"), and also in the way Francis Galton organized his genealogical, biographical data when he decided he wanted to prove the inheritance of talent.

It was not the belief in the inheritance of physical characteristics but that of moral (mental) ones that made Buckle react against hereditarianism (he had of course strong stakes in his environmental explanations). Although French and British alienist had been assembling statistical tables of morbid mental inheritance ever since Esquirol's first attempts in the 1810s, the transmission of positive mental faculties was still based on loose, story-telling, genealogical procedures. Lucas for instance had tried to prove the passing on of mental capacities in the same way he had done it for other characters. By choosing examples: the Bachs, the Racines, the Tassos, the Bernouillis, etc.[35]

When in the 1860s Francis Galton began collecting genealogies he decided he would eliminate the arbitrariness of this case selecting by using lists of talented individuals chosen independently of if they were related or not to other talented persons. If it turned out that they tended to have more talented relatives than what would be expected from the "a priori" probability of the population, then a case could be made for an hereditary link. Similarly, the alienists after Esquirol had considered that if the registration of insanity cases to their establishments were unrelated to the origin of the disease then there shouldn't be any bias towards the hereditary cause in the selection process, and if a high percentage of them turned out to have affected parents or relatives, then the inference was warranted.

Both in Galton's and in the alienists' cases, there were critics, in their own time, who noticed the unwarranted nature of one assumption they made; that of having non-biased samples. Besides, they could not, as it was also pointed out, eliminate other possible explanations of the correlation between eminence, or madness and parentage.[36] But Galton, as the alienists before, was sure that with his procedure, he

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[35]Lucas, *Traité...*, vol. 1, p.582. Lucas castigates Lordat for denying transmission of mental faculties, though he accepts that his cases are worth noticing, but the explanation is that in them inneity prevails and the offspring is either much more or much less talented than the parents. This "explain away all" position was based, it must be noted, not on selective case quoting, but on the interplay of the dialectical pair of apriori forces.

[36]As Olby has acutely pointed out, the reception of Galton's *Hereditary Genius* was more positive than some believe, but not entirely uncritical. Though some of his critics, like H. Merivale, understood very well the logical structure of his attempts, and thus its flaws and shortcomings. See R. Olby's "Galton's response to Buckle" (in

showed that the number of relationships between eminent persons was considerably much higher than would be expected by pure chance, "the over-whelming force of a statistical fact like this renders counter-arguments of no substantial avail".[37]

Again, it is the probabilistic principle of coincidence which justifies the inference. But a "statistical fact" seems more impressive than a simple occurrence. As Hilts has argued, Galton was well aware that some of the loopholes of his methodology left him exposed to obvious criticisms, like not taking enough account of the social and class related motors of the distribution of merit and honour, specially within certain professions which he had chosen as examples when he expanded and detailed his views in *Hereditary Genius*, (1869). To render the probabilistic argument more robust was then the aim of the rest of Galton's life work. This he had to do first by finding ways of showing as little bias in the selection of the samples as possible, and at the same time increase the definition and level of detail with which a given character could be followed through the generations. And second to clarify the kind of causation that could generate both the "higher than expected by chance" link between the characters of parents and offspring, and the irregularities that the field of heredity was plagued by.

The former aspect of his work was perhaps the most successful. This is what Bateson acknowledged when he wrote that it was Galton who had concentrated attention upon "the outward facts of (hereditary) transmission".[38] With the increasing sophistication of statistical analysis that he introduced, as Baillarger had written many years before, it was no longer the fact itself of hereditary transmission of a given character that was at stake, but the details or structure of the relationship. The invention by Galton of statistical correlation and regression, analysed and explained in different fashions by many historians due to their subsequent importance, can be said to have been the conclusion of a long process within the field of hereditary transmission in which the external evidence of certain patterns of re-occurrences was successively challenged by sceptics.[39]

As I said, Sandler correctly demonstrated that Maupertuis' hereditary explorations were not in any sense a prelude to Mendel's inferences. A better case can be made, I believe, for a connection, both historical and epistemological, between the probabilistic bases with which the French illustrée established (externally) a causal connection that any (internal) generation theory had to account for, and the (external) statistical procedures that Francis Galton developed, not only to establish the hereditary connection between given characters in the parents and those in the offspring, but to evaluate the strength of the causal dependency they had. This connection is one of the threads I have been following through this work: the empirical field of the hereditary, chartered by the collection of cases, from medics and naturalists, in which the familial pattern of reappearance of peculiarities was the

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print). In case of the alienists' statistical analysis, the main critics were made by their many environmentalist critics. See Dowbiggin, 1990.

[37]F. Galton, 1865, "Hereditary talent and character" in *Macmillan's Magazine*, June, p.160.

[38]Quoted by R. Olby,(in print) "Human Genetics, Eugenics and the State", p.1

[39]Recent accounts of this have been given by Olby, Mackenzie, Stigler, Porter, Gayon. Cowan's work on this matter, though important, is now outdated, I believe. Baillarger's pioneering use of statistics in Heredity is to be found in "Recherches Statistiques sur L'Hérédité" (1824).

dominant theme. With varying degrees of sophistication, the reliance on these external evidence as a form of by-passing ignorance, or imposing limits to the dominant views in physiology or the generation theories, was championed successively by hereditarian medical men and alienists in France. The rationale of this empiricist attitude was sometimes obscured, but it allowed in the end the establishment of a series of general features that facilitated the posterior analysis of heredity even in the ignorance of internal, physiological, detail. The first step of which, as we have insisted was the predipositional explanation of irregularity of transmission, and of all the other tell-tale features of a latent cause, like atavism and homochrony. Heredity, in a sense, always presented the problem of establishing a causal connection between the presence of the same feature in two individuals, directly or indirectly related. The localization, definition, stabilization of the feature (a disease, a musical talent) is the first problem. The sorting out of the innumerable irregularities, dissimilitudes, etcetera, in order to eliminate all the possible causal challenges to te connection, the second. From story-telling to statistical tables, there is a continuity: the same problem, the same wild variations.

Historians of mathematics may sometimes believe there is a whimsical and contingent side to the fact that it was within this disputed field of biological research that such powerful, fact connecting, explanatory tools (like statistical correlation and regression) were developed. The more the story of Galton's development is explored, and understood, the more there seems to emerge a sort of historical necessity (as much as there can be such a thing).[40]

6.3 Genealogy and the causes of hereditary variation

> None of the laws of hereditary descent connected with the formation of character have yet been generalized; nor is our knowledge much more advanced respecting the theory of temperaments, which still remains the principal obstacle in the way of phrenologists.

This other footnote incorporated by Buckle in the same section as the one above[41] introduces us to the other connection I will want to make between the pre- statistical approach to heredity stemming fom the medical tradition and the approach developed by Francis Galton. It puts the finger on the theoretical problem facing mid-19th century hereditarians. To generalize the law of hereditary descent from the irregular and unreliable relation between parents and offsprings' characteristics needed a stabilizing frame. That is to say, a reliable theoretical resource for describing how the patterns of similitude and variation can be generated. Having only the data of the genealogies and pedigrees that case collecting first, and the elaboration of statistical tables later, gave them they had to produce a coherent account of how an individual's set of characteristics can at the same time be determined by its ancestors' characteristics, and be loose enough to produce the undoubted diversity and individuality. The interplay between groupal homogeneity (to be explained by a

[40]See for instance Stigler (1987). Mackenzie, on the other hand sees the historical "necessity" of a link between these developments and the needs of the field, but dwells mainly on the sociological explanation and the mathematical and logical links. I have stressed the rhetorical and probabilistic roots of the need.

[41]Buckle, op.cit., footnote 11, p.160

strong repetitive trend established by heredity) and individual variations (the origin of races, nations and families) could account for the observed phenomena, many thinkers semmed to agree, but there was a lack of a coalescing, simple description of how all the influences acted upon the organization of each individuals' bodily structure in order to allow for the case by case unpredictability, and the long run constancy that many authors agreed the facts reflected.

A tenable account of how all the different, hierarchical characters took, or lost their place at the moment of conception. Something that could breach the gap between the external evidence provided by patterns and tables and the ever changing descriptions of reproductive physiology, cytology and chemical biology.

Only for very retrograde authors did the medical theory of temperaments still provide such a frame, for as Buckle points out, it could only perform the task at a loosely metaphorical and unanalytic fashion which was no longer of any use in the mid-19th century. That the characteristics of human varieties, races, national groups, etc. could not be defined and described with any level of accuracy by the predominance of this or that humoral combination was understood by most authors during the 19th century, but the descriptive role of the theory survived its explanatory lifetime.

The other medical concept, constitution (with the naturalists' *organization*) had more powerful descriptive resources as it could accommodate more naturally the advances of anatomy and physiology.[42] It had been, as we saw, employed by Lucas and other medical men, in the description of the conflict of causal influences that each individual's structuration implied. The re-production of the father's general frame and set of peculiarities conflicted with the re-production of the mother's frame and set of peculiarities. Rules were needed to understand how this basic fact of sexual reproduction under the empire of a generalized law of heredity took place.

The medical tradition had since long established some general features of this re-production that proved useful when the search for these rules became important. Both the mother and the father were responsible for hereditary influences. Heredity could be a latent cause. Hereditary factors could remain hidden for one or several generations. Hereditary features tended to be associated with given periods in a life cycle. Hereditary influences could be linked with minute and insignificant traits like the shape of an eyebrow or with dramatic peculiarities like a tendency to bleed to death.

These general features were used as a base for further research in the determination of the hereditary cause. It is arguable that for medics since the early 19th century irregularity of hereditary transmission had long ceased to be a mystery due to the conviction that latency could be due to the communication of constitutional predispositions, and not actual traits or features. The idea that what the child originally inherits is not a part of itself but a tendency (or a part of itself that gives it a tendency) brought the metaphorical use of heredity closer to reification by extending the analogical coincidences between the source (inheritance of wealth, etc.) and its biological dominion. Contrary to Thomson's verdict[43] that heredity was misconceived metaphor because it was the body itself, its actual constituents what a child received from the parents, and not an independent set of things, the predispositional view of hereditary causation allowed for a tightening of the efficacity

[42]Both Schiller (1978) and Jacob(1970) have described this influence.
[43]See chapter 1.

of the analogical meaning.

Furthermore, the 1830's initial split between "heredity" meaning the actual part or trait inherited, and "heredity" meaning the general cause characterized by a pattern of behavior, is linked to the concomitant view that a set or packages of dispositions or tendencies could be "received" by the individual in the act itself of acquiring its constitution or organization. This opened the gap I mentioned above: the need for an account of how the hierarchized characters that integrate every individual get to be there. How are the conflicting hereditary dispositions stemming from each parent sorted out? How are the conflicts between general specific tendencies and particular, individual ones resolved? Lucas' solution to the problem was, as we said, to compartmentalize and make room for every option. To reify inneity as a balancing force for heredity, and to situate the origin of these forces in the two basic typological nodes: the species and the individual. Each species becoming a sort of genealogical flow with two concentric but independent streams: a chore one that provides the main structure and that is unaffected by innovation or variation; and an external which pushes out (via inneity) to vary and innovate, and that preserves these variations (through heredity) in such a way as to form stable subspecific groupings. The longer a trait has been within a group the firmer it becomes and the more likely it will be that a member of the genealogical line will receive it. This creates a sort of separation between the sets of characters that the race, the national group and the family impose over the newly forming being. Racial characters are more strongly "pushed" than national, and these more strongly than "familial". Sometimes the father's characters are more strongly pushed than the mother's, and vice versa. But all the subspecific characters are liable to be affected by change. This liability increases of course with the superficiality (or individuality) of the trait.

This typological metaphor that Lucas creates, of the races, the varieties, the nations, the family, and each parent's (male and female) types exerting differential forces (of heredity) over the new individual at the moment of its first formation is a strongly compelling one. It allows for a picturing of both constancy and variation, as it makes individual the product of a compromise, of a resultant of forces. It also has room for accounts of hybridization and of selective breeding. Although its typological, metaphysical assumptions were anathema for the English thinkers view of science, it is demonstrable I believe that it helped to shape how mid- 19th century authors structured their own, hypothetical, accounts of heredity.

As Hilts splendidly showed in his doctoral dissertation,[44] Francis Galton came to heredity through his interest in ethnology, product of his trips and observations in southern Africa. The focus of his initial attention was the set of physical and moral characteristics of the different human races. Like many of his contemporaries he was convinced that most of the differences were rooted in the physical constitution, even the moral and psychological ones. The family and the nation, as sub-racial ethnological categories around which clusters of characteristics could be held together, and relationship between individual genius and national character, were at the forefront of the reflections Galton was making in the pre-hereditarian phase that Hilts has called the "chaotic years" (the early 1860's). Lewes, who Galton read in that period had taken on board very enthusiastically Lucas' construct that made families, nations and racial groupings dependent on a kind of historical sub-types. If we recall

[44]"Francis Galton and Statistics in the Service of Anthropology", part 2, of (1981, originally 1967).

a quote given above:

> Unless parents transmitted to offspring their organisations, their peculiarities and excellencies, their would be no such thing as a breed, or a race. The cur would run the same chance as the best-bred dog of turning out valuable.

To make hereditary transmission the reason why breeds, races, and in the end national and familial types exist is, in a sense, to justify the focus of attention to genealogy and pedigree. Breeders made the move following economical not theoretical gain, and as Spencer and Darwin argued their reliance on pedigrees warrants an independent, strong justification of the reality of the phenomena. As Spencer put it:

> Excluding those inductions that haver been so fully verified as to rank with exact science, there are no inductions so trustworthy as those which have undergone the mercantile test[45]

But in the case of humans there is no mercantile test. Human natural breeding habits generate the family, the nation and, for some, the race. Special individuals are linked to special families, special families to special nations, special nations to special races, and the special white race to the dignity of humankind: so much was obvious to the average Victorian. But more than an analogy from te domesticated beasts was needed to fortify the induction. More than the aprioristic, *petitio principii* of the French. To transform ethnology into an exact science, or to mathematize genealogy could be adequate descriptions of Galton's early moves in the field of Heredity.

A remarkable, candid, paper read to the Anthropological Society of London in the mid 1860s by the genealogist George W. Marshall provides the perfect frame to understand Galton's early research:. "Remarks on Genealogy in conexion with Anthropology". Genealogy, the tracing of the descent of the individuals, and through them of nations, as Marshall defines it, "can be made practically useful for anthropology". The study of the pedigree of individual families is intimately linked to the one investigating the laws of man's origin and progress. "Are not ethnology and genealogy essentially the same? " he eventually asks.

> If they differ, is it not only in this, that the ethnologist studies man by grouping him into different large races; whilst the genealogist seeks to know him more completely by studying him in individual families? The genealogist is, in fact, the architect who builds up the structure of the science of man, stone upon stone, story upon story.[46]

"Statistical table upon statistical table" the early Galton might have happily added. Marshall continues his discourse as if setting the main lines of Galton's future research. The two main aspects of man, the physical and intellectual, can be both tackled by genealogical research. They are so dependent on each other that both aspects have to be studied together. Furthermore, Marshall adds

> A better knowledge of the genealogy of individual families would do much to settle the question as to whether the intellectual development of man is the result of physical refinement. Some people will tell you that

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[45]Spencer, op.cit., p.242.

[46]Marshall was editor of the Victorian periodical *The Genealogical Review*. "Remarks on Genealogy...", in *Memoirs read before the Anthropological Society*, vol.2, 1866, pp.68-73.

> there have been many talented men who have had no pedigrees, and conclude from this that the refinement of the body has therefore no necessary influence on the intellect...[they] assume that God has created all men equal, that one man is as capable of high mental cultivation as another.

If they were right, Marshall adds, "all deductions from the history of our ancestors are useless, and the study of genealogies ...a mere waste of time". Which they obviously are not. Constitution of men affect all their capabilities, physical and mental. There is a definite limit for each individual beyond which no amount of exterior cultivation (exercise or education) would take him. And that limit is fixed by the ancestral influences of race, nation and family. So

> if one man is so constituted that he is capable of higher cultivation than his fellows, then by finding what manner of men his ancestors were, we may be enabled in some measure to discover what modes of life and what kind of alliances will best develope the perfect man.[47]

Important for all peoples is good leadership, and the maintenance of a healthy, natural stock (or breed) of workers and soldiers. Genealogy is a tool that can help insure that the best bred men get to the former position, and that the worst, those who could spoil the averages, are localized and neutralized. In genealogy all this is linked to the family. The importance of families for the nation is put under a limelight. Are noble families a good thing? Do they last long enough, or too long? Is the level of achievement that the members of different generations of them can attain always equal, or does it decline, or grow? Do they deserve their birthrights or is a meritocracy a better way of distributing honour? The 19th century was obsessed with questions like these. Francis Galton believed he could provide more precise answers than anybody had. But for that a better description of the hereditary relationships between ancestors and descendants was needed.

That between men, as between beasts, peculiarities of all kind arise that differentiate them. That these are transmitted to their offspring (they "run in families") and eventually create genealogically linked groupings (nations, races) cannot be doubted, so as Marshall sums up: "the problem to solve is how these differences arise, and what changes they work in different generations".[48] If differences between individuals could be associated to differences between families, and these to differences between nations, and these to differences between races, then things would be simpler (Lucas' cumbersome typology, for instance, would become superfluous). The genealogical approach could help in the task. Though the numbers of our ancestors grows exponentially ("in the space of little more than six centuries every one of us can boast of the astounding number of five hundred and twenty-four thousand two hundred and eighty-eight ancestors"),[49] and thus a localized following of any influence becomes impossible after a few generations, a careful investigation could be made into the number of generations that one has to go back before there is too weak and diluted influence. Marshall, for instance believed that only three generations suffice

> when I speak of a man "having a pedigree", I mean to say that some two or three generations at least of his fathers have been in better

[47](Which is what they are all after, isn't it?).Marshall, op.cit., p. 69-70.
[48]Marshall, op.cit., p.71
[49]In Lower's *Contributions to Literature*, quoted by Marshall, op.cit., p. 70.

> circumstances than the generality of their fellow men. I do not wish to argue that the longest line of refined ancestors will necessarily produce the most refined descendant ...[but] some few generations of refined ancestors become a sort of guarantee that those who can show them are to a certain extent superior to those who cannot. From the desire to prove and assert this superiority comes the very natural love of genealogy.[50]

A love that Galton, being himself a member of a family with some intellectual "pedigree", shared with Marshall. Besides proving that the inductive inferential power of family tales of hereditary transmission of talent could be reinforced by statistical tables, Francis Galton saw a need to re-describe what exactly could be meant by ancestral influences on the individual. That the race, nation, and family were represented in individual constitution was accepted by him as a given. Atavism, variation, latency were, as in Lucas, a consequence of all these presences, but as S. Cowan, Olby, Gayon, have pointed out,[51] he found a way to ascribe them to the same kind of causal influences. Cowan uses a hopelessly anachronistic description of this fact

> what Galton did was to define the territory that genetics could explore by collapsing three entities inheritance, variation and reversion.[52]

Although acceptable under certain light, the formulation is misleading because heredity and atavism were never for very long phenomena that authors considered as independent entities. As I have shown above, atavism (and latency in general) was for many decades a tell-tale feature of the presence of hereditary influence itself.

It was only around the mid-19th century, when heredity was given a central role in biology and the inference was made that what should be expected if it alone ruled was a plain homogeneity and not the observed diversity and variation that atavism was perceived by some as a different kind of varational influence than heredity. The sources of variations became a crucial question. If not an actual force of innate variations, as Lucas wanted, what sort of influences could be acting over the new being's organisation at their first integration, to distance them from their parents frames, towards which heredity would be pushing them. A straightforward approach allowed for an easy answer, linked with heredity, the same diversity of constitution between the couple of parents permits different combinations, thus the same couple can have very different children. Both George Lewes and Herbert Spencer had no doubt that this "genealogical" variation could alone explain a very high percentage of the observed variations, as in plant hybridizations or inter-racial reproduction among humans. Spencer and Lewes add to this the physiological states of parents at the time of conception, a particular cause that Galton found implausible and eliminated from his list.[53] Most authors agreed that internal ("genealogical") sources of variation are however insufficient to explain all the observed diversity, and the residue must be explained by some unknown "spontaneous" variation, the kind Darwin particularly made use of in The Origin of Species.[54]

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[50]Marshall, op.cit., pp.68-69.

[51]S. Cowan (1970), Olby (1985), Gayon (1989).

[52]R.S.Cowan, "Nature and Nurture..."

[53]This was an old view, recycled by Spencer and a few years later by Alphonse de Candolle. See below.

[54]Darwin's list of the sources of variation include: circumstances hostile to particular characters that block their expression (external), conditions of life incessantly

Galton was particularly interested, and eventually successful in giving a better description, a simpler causal structure to internal variation as a product of heredity. Spencer and Lewes had set a broad description of the territory he was to charter with finer strokes

> It is the fact of double parentage, and double inheritance, with an equality in the amount of influence exercised by each parent, which complicates the question, and produces the seeming contradictions.[55]

The amount of influence of each parent is however only ideally equal, in practice, at each conception there is a struggle, and an inequality can arise in the amount of organizational (constitutional) qualities a given child's bodily frame will actually adopt from one or the other parent. "Both parents contributed their elements, but these elements were differently compounded"

> It is to this inequality in the influence of a particular parent that we must attribute the fact that, while certain peculiarities, trifling and even whimsical, are sometimes seen to be transmitted, they are not uniformly transmitted...Whenever we observe rigorous constancy in the transmission of qualities —as in the breeds of animals— the secret is that both parents had more or less these qualities. Whenever we observe inconstancy in the transmission, the secret is that only one parent had the qualities; and inasmuch as both parents transmit their organisations, the double influence determines the product.[56]

But the parents' conflicting heredities can, under the genealogical view, be further analysed, divided between the influences of the very many ancestors that are being channeled through the couple. Hilts showed how Lewes' position was an English critical appropriation of Lucas' scheme, and a similar comment could be made of some aspects of Spencer's. What in Lucas had been a struggle between "types" for representation in the new individual's constitution, in Spencer and Lewes became an influence of sets of characters that could be assorted or reassorted, a sort of combinatorial scheme. Spencer, for instance abandoned any talk of the species, or the races "type" and substituted it by a reference to their "average", and his particulate model of transmission based on physiological units is in line with this view, as is Darwin's pangenesis.[57] As most British, Galton found this line of hypothetical, particulate model making far more promising than the appeal to special forces of the French physiological school, and he himself developed a very complicated model for transmission in which he tried to account for all the phenomenological subtleties of heredity; what came to be known as his "stirp" theory.[58] By postulating a double flow of characters (one patent and another latent) from one undifferentiated, embryonic stage to the next one in the following generation, and using both a urn-

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inducing fresh variability (external), crossing of distant varieties (internal), reversion or atavism (internal).

[55]Lewes, op.cit., p.378

[56]Lewes, op.cit., p.379 and p.384

[57]Published in 1868 as part of *Variation....* In this model Darwin accounts for atavism, variation and other hereditary phenomena by postulating the existence of particles emited by every part of the organization. These are collected in sexual organs, transmitted, mixed in conception with the partner's particles, and then they reproduce the peculiarities of the part they came from in the offspring's organization.

[58]First exposed in his 1872 "On Blood-relationships".

sampling analogy, and a more revealing one based on political representation, Galton made explicit the view that heredity was to be pictured, as we said, as a "struggle for representation" between the elements bequeated to the living generations by their ancestors. Heredity is a genealogical business, its role is to create workable descriptions and concepts of supra-individual levels, of genealogically linked groups. The title of Galton's first exposition of his model, "On Blood-relationship" is explicit about it. As are both the methods and the kinds of lateral questions that Galton addresses in his first two hereditary works, "Hereditary Talent and Character"(1865) and *Hereditary Genius* (1869). The fate of noble families and the irrationality of life peerages, the weakening of the influence hereditary influence after more than three generations, the dangers of marrying heiresses, and of course the advantages and possibilities of promoting a breeding betterment of humans.

Galton was the first author who closely came to recuperate all the analytical complexity of Lucas' genealogical approach. He avoided at the same time the questionable appeals to special forces, and the conceptual baroquisms of Lucas' hierarchized and multilayered typological scheme. His genealogical approach was particulate and representational: the struggle for influencing the constitution of the future generations is transformed into a probabilistic process in which all previous generations participate, but with declining influence, as their contribution to the "stock" of elements transmitted is halved with each generation.

This idea is, in essence what came to be known as Galton's "Ancestral Law of Heredity". Lewes' descriptions of how homogeneous elements explain similitude while crossings between mixed, mongrel individuals generates dissimilitude and reappearance of hidden elements is perfectly accounted for by Galton's scheme. As is the fact that within genealogical groups the oldest, more widespread elements that account for specific, racial, and national characteristics are transmitted with much higher frequency and strength. In fact, Galton managed with his scheme to forward the idea that the reality of the genealogical groups that so obssesed his contemporaries, like breed, race, nation, family and the like, was merely a statistical one. A fact that he came to represent by using Quetelet's "normal" distribution curve as a criterion for having an unmixed population. individuals belonging to the same genealogical group, that is.

As is well known, Galton came eventually to regard the science of heredity as "concerned with Fraternities and large populations rather than with individuals", and with the "statistical resemblance between successive generations".[59] This perception, and his imaginative power led him to introduce, in only a few years, into the field of heredity a set of very powerful mathematical tools for inductive inference that left all the previous, non mathematical, story telling, approaches looking very "pre-historical" indeed. The path that Galton took from his initial set of prejudices and intuitions of the 1860s into their mathematization, and the invention of correlation and regression has been carefully chartered recently in very good books by Hilts (specially good in early Galton) Mackenzie, Olby, Stigler, Porter, and in unpublished research by Olby and Gayon. I can add at this point little to their efforts. I have however tried to show above how Galton's work is much more closely linked to the long tradition of human hereditary theorizing that preceded him, and that mainly, but not exclusively stemmed from the medical field. Galton was in a sense responding to this previous tradition both imitating the structure of some of their schemes and in

[59]See Natural Inheritance.

trying to avoid what he saw as their main flaws and shortcomings. He expressed his feelings perfectly well when reviewing Theodule Ribot's book on Psychological Heredity in which he continues the French alienists tradition of considering Heredity as a conservative force, and uses all of Lucas' dichotomies and story-telling inductive procedures, and, to Galton's displeasure, has the audacity to put them at the same level of rationality and effectiveness as Galton's statistical tables and inferences. Mr. Ribot's book, Galton writes

> takes us back to the prescientific stage of heredity. It again brings to the light old anecdotes of questionable value, and again treats with seriousness hypotheses thar have become obsolete.[60]

Galton in many senses never looked back again, and never acknowledged any link to previous hereditarian pursuits. And while that was an attitude adequate for someone wanting to establish himself as a leading figure in a new field, it should not be believed by historians. There are, I believe, reasons to affirm that his pursuits and those of many of the authors that I have been dealing with in this work, are intimately linked.

[60]F.Galton, 1875, "Ribot on Heredity", p.118. Ribot, it must be said, had also very critical comments to make of the work of Galton. He saw his statistical procedures as a good way of organizing evidence, but found them insufficient as a bases for explanation of heredity without the complement of what he saw as a necessary inference towards the existence of a conservative, selective force. He saw Galton's empiricism as stubborn and limited. Galton on the other hand accused Ribot of plagiarism, as he did not completely acknowledge that the statistical tables implied much more than just piling up facts. But that is exactly how Ribot saw them; as labour saving compilations of hereditary tales. They were obviously speaking different languages by then. See Ribot's Heredity, p. 254.

Chapter 7

Conclusions

In the first ever entry under the heading of "Heredity" for the *Encyclopaedia Britannica* (11th ed.,1910), Peter Chalmers Mitchell described the existence of an age-old empirical research programme focused basically on the gathering and organizing of facts about the transmission of hereditary characters from parents to children. Its principal upholders were physicians interested in hereditary ills. It was on the whole a tradition of research that did not easily fit with the rest of the brand new investigations of heredity that he describes with excitement in other sections of his paper: Mendelism, chromosome theory, and the like.

The aim of such a fact-collecting enterprise had been to establish “the external facts of heredity”. Its results however would eventually become superfluous, Chalmers suggested, when the ultimate truth about biological heredity became known. The extraordinary complexity of the hereditary phenomena would then have been reduced to their underlying physiological processes. Until that happened (and Chalmers thought it was quite close) the old labour of collecting and organizing facts about transmission of specific characters in certain circumstances, and in certain proportions, would still be helpful, as it had been during the many decades of ignorance about the actual physiology of reproduction and inheritance.

To record and assemble the facts about “the actual relation of the characters of the offspring to the characters of the parents and ancestors” had been a prerequisite, Chalmers believed, for breaking out of the confusion of previous times. For a long while even to establish the fact of hereditary transmission itself was a struggle that consumed the efforts of several generations of workers interested in unraveling the mysteries of heredity.

One very paradoxical feature of Chalmers’ article is his mention of Prosper Lucas’ *Traité de l’Herédité Naturelle* (1847-1850) as the definitive work of the fact-gathering tradition on hereditary pursuits. In Chalmers’ view this work had finally taken “the facts of heredity” beyond reasonable doubt.

The paradox is that soon after this comment, very few people would recognize either the book or the author and nobody would grant them any importance in the history of our theories of biological inheritance. Chalmers’ mention of Lucas was perhaps the last time Lucas, and for that matter any of the authors of the medical tradition in question, was seriously considered as part of the scientific realm. Very soon their contributions were written off the history of heredity as a collection of midwives’ tales and fantastic speculations. This thesis has shown why that view, upheld until very recently by most historians of biology, is a impoverishing distortion.

In the six chapters above I have shown how Lucas’ impressive work was “standing on the shoulders” of several generations of physicians, alienists, physiologists, ethnologists, and others, who for different reasons had made hereditary

transmission their theme.

I have shown also how this basically French tradition influenced the development of some aspects of late 19th century hereditary thinking, in particular that of Francis Galton. But most of all, I have described and discussed how medical preoccupations with the category of hereditary disease and its justification, originated the questions about hereditary transmission that helped shape the broad lines of what eventually became our modern concept of biological heredity. Lucas, again, played an important role in bringing this about.

One telling aspect of Chalmers' point of view is that he reduces the whole tradition to a fact-gathering role, ignoring completely the conceptual developments that it incorporated to the understanding of heredity. This attitude reveals the short-sightedness that the ongoing developments in the field of heredity was already producing about the past. There is a sense however in which it *is* the empirical side to the tradition that makes it so unique. Facts of actual transmission were registered, accumulated through the centuries, and were stored in collections, transmitted and retold during the centuries-long search for explanations. They all coalesced around the same metaphor: astonishing facts, troublesome facts, scary facts about reocurrences of features in the same families; resemblances, the whims of crossings, the fatalities of "receiving" at birth a body or a mind predisposed to disease.

I have shown in this work how the originally analogous relationship between these sets of facts strengthened progressively, in the same measure as the hereditary metaphor was transformed into a truly referential term. I have called this process "the reification of heredity", and maintained that its development brought with it the creation of the space now occupied by our theoretical ideas of hereditary transmission.

As an empirical domain of reference, loosely kept together at first through the metaphor of heredity and not through any causal or explanatory function, "the hereditary" (as I have called it) is an historically exceptional case. It always was a domain in which irregularity and exceptions were the rule that made it an especially problematic area for scientific pursuits. I have also described how, from case-collecting to story-telling, from analogical reasoning to the postulation of causal hypothesis, medical men gave shape to the concept of biological heredity in a way that facilitated the later generalizations after the mid-19th century.

I have shown how, in 18th and 19th century France and Britain, medical men's preoccupation with hereditary transmission of diseases and their possible physiological routes, helped enormously in the development of a much more subtle and profound definition of the hereditary cause. Borrowing a simile from the field, these European medics shaped the "first rudiments" of modern heredity by highlighting some of the features of the hereditary that can be inferred from the observation of external patterns of recurrences; in particular, the latency of causation, which itself implies so many of the better-known features found by medics to characterize heredity, like atavism and homochrony.

Perhaps the strongest theme of this thesis is the importance of the shift after the 17th century from one medical idea of the body and its fundamental dispositions, the humoral, to a new idea, solidist, which was based on mechanical and univocal causes. This shift allowed for a new understanding of predisposing and exciting causes which in turn gave a new meaning to the other dialectical pair: hereditary vs acquired traits. I have argued that a consequence of this was to open a possibility of unifying all the

elements of the empirical field of the hereditary under the same causal scheme.

The rise of heredity to prominence in 19th century France, its acquisition of a central role in explaining homogeneity between genealogical (and taxonomical) groups, and the highlighting as a consequence of its opposition with variation, are all treated in the pages above.

It is shown also how, through the work of Prosper Lucas and other authors, this long tradition of human heredity joined the current of other related pursuits in the second half of the 19th century, and contributed to the flourishing of the kind of hereditary theorizing which Whig historians and short-sighted geneticists *can* recognize from this distance.

Appendix 1

De Morbis Haereditariis

A chronological, commented bibliography on the theme of hereditary diseases, 1586-1886

1.1 Before 1600

1. 1586, Fauvelet, *Suntne haereditarii morbi curabiles?* , Paris.
2. 1594, Mercatus Ludovico (Luis Mercado), *De morbis haereditariis liber*, in Opus, 2 tomes, Madrid (Other editions of the two volume work are 1605 in Valladolid and in 1608 in Frankfurt, 669).

   Pierre-Joseph Amoreux (1790, below) asserts that Mercado was the first author to write a Treatise with the particular subject of Hereditary Diseases.

   Robert Burton writes about this piece in the *Anatomy of Melancholy*, 1621, calling it an “excellent tract”, p. 212.

   Mercatus (Mercado) (1513-1599) was a physician in the Spanish court of both Felipe II and Felipe III.

1.2 1600-1700

1. 1619, de Meara Dermutius, *Pathologia haereditaria generalis*, Dublin, 8o. (also London 1665 in 8o, and Amsterdam, 1666 in 12o.)

   Pierre-Joseph Amoreux wrote about this book (in 1790, see below): “ce médecin irlandois eu celui qui a le plus écrit ex profeso des maladies héréditaires mais imbu des principes chimiques il en a abusé en faisant la usage de la doctrine en voyant partout un certain tatué ou sel qu’il veut pour cause materielle specifique”. pp. 3-4 Girard (also 1790, below) was less tactful about it:“On y trouve des faits interesantes, quelques preceptes vagues tirés des anciens, ce tout etensu, défiguré, noyé dans une foule d’erreurs et d’inutilités. L’hypothèse absurde des sels fixes regardés comme principede toutes les maladies de naissance suffit pour donner une idée de la théorie de cet ecrivain”.

   Diderot gives de Meara as a good further reading to his *Encyclopedie* piece on “Héréditaire” (maladie) (1765, below). He writes: “pour un plus grand détail sur tout ce qui regard les maladies considerées comme héréditaires, on peut trouvé beaucoup d’instruction dans le traité qu’a donné sur ce sujet Dermutius de Meara”, p. 574.

   Discussed by Conway Zirkle in “The early history of the inheritance of acquired characters”, 1946, pp. 134-135. A translation into French of substantial parts of this treatise plus a commentary on it can be found in Bernard David, 1971, *La Prehistoire de la Genetique*, pp. 79-92. David gives the following summary: “l’ouvrage consigne toutes les grandes idées alors en cours sur l’hérédité (J.Fernel, A. de Laurens) ...nous montre comme l’auteur a défini, avec beaucoup de clarté et de précision ce qu’est une maladie héréditaire. Il en a souligné les mécanismes: << les maladies sont transmises héréditairement par la semence à condition qu’elles soient stables et fixes car la racine de la maladie héréditaire est attaché à l’une des parties fixes quelconques de l’un ou de l’autre des parents >>. Disciple de Paracelse, il attribue au vice du sel la cause de la maladie héréditaire”. p. 113-114.
2. 1621, de Bourges, *Ergo à femine haereditarii*, Paris.
3. 1627, Crnerus Balthasar, *De morbis haereditariis*, Wittenberg.
4. 1627, Janus (Jani), *Dissertatio de morbis haereditariis*, Viteb.(Wittemburg), in 4o.
5. 1636, Crüger, *Dissertatio de morbo haereditario*, Regium.
6. 1636, Crusea J. Barthol., *Dissertatio de morbis haereditariis*, Regiom.

   Note: this could be the same work as the previous one.
7. 1647, (1643?) Lyonnet Robert, *Brevis Dissertatione de Morbis haereditariis*, Lion, 4o., 87p. (see mention in Haller Bibl. Med. Pr.II, p. 641)

   This was among the better known treatises on Hereditary Disease at the beginning of the 18th century. Antoine Louis (1749, below) made extensive use of it to back his argument that solidism (and so mechanicism) implied the impossibility of hereditary transmission of physical accidents. A good summary of the book is provided by Louis, with all the anecdotes related to the “royal” origin of the Treatise: it seems that Lyonnet wrote to placate the

worries of Louis XIII and his Queen who were upset about the health of the dauphin (later Louis XIV) and wanted to make sure they were not responsible, specially the mother.

Girard (1790), following very closely Louis' rendition, gives his summary of Lyonnet's work:"il pense que la disposition héréditair aux maladies (morbosum seminarum) doit être imprimé dans la semence, parce que c'est de ce principe que sont formée les parties solides du corps, et que les desordres qui affectent ces parties sont immuables (continuo corpus excrelbunt? neus quam curationem recipient). Il tache d'expliquer cette transmission par l'esprit séminal, l'esprit éthéré qui preside à la forme que prend la matière et qui peut en recevoir des alterations.

Pour prouver que que l'esprit vivifiant, l'esprit prolifique peut être altéré par la matière, notre auteur rapporte l'exemple des plantes que dégénerent visiblement, quoique la semence soit la même." Louis here comments that this is not a good example, because such changes in the appearances of vegetables (*espece de dégénération*) according to regions depend on extenal influences. The analogy, Louis adds, reinforces if anything a belief that so called hereditary diseases are also caused by external influences.

8. 1665, Welschius, *Dissertatius de morbis hereditariis in genere*, Lips.
9. 1681, Metzger, *Dissertatio Affectum praeternaturalium haereditariorum theoria*, Tub.
10. 1688, Fischer Joachim Arnaud, *De Morbis haereditariis*, Erf., 4o.
11. 1688, Myller Carolus Josephus, *Phthisis haereditaria cura*, Miscell. Acad. Nat. Curios., Dec.2, A.7
12. 1692, Xarin, *Ergo à quibus vita, ab iisdem morbus*, Paris.
13. 1692, Behrnaver Gottlb., *De morbis archealibus,in 4o*.
14. 1692, Alberti, *Dissertatio de morbis haereditariis*, Erfordii. (also in Jurisp. Med., Tome III, p. 652)
15. 1695, Stein, *Dissertatio de morbis haereditariis*, Ludg.
16. 1695 and 1696, Hannemann Johannes Ludovico, *De insania haereditaria*, Miscell. Acad. Nat. Curios., Dec.3, A.3, p. 73.
17. 1695 and 1696, Adolphi Christian Michael, *Herniae quoque pertinent ad morbos haereditarios*, Ephemer. Acad. Nat. Curios., Dic.3, A.3,
18. 1696, Rohle, *Dissertatio de morbis haereditariis*, Ultraj.
19. 1696, Vogetius, *Dissertatio de morbis haereditariis*, Ludg., Bat.
20. 1699 and 1700, Riedlin Veit, *De Mania Haereditaria*, Miscell. Acad. Nat. Curios., Dic.3, A. 7 & 8, p. 75.
21. 1699(?), Ovelgün Fridericus Rudolphus, *Nyctalopia haereditaria*, Acta Acad. Nat. Curios. Vol. 7, p. 76.
22. 1699, Hoffmann Frideric, *Dissertatio de affectibus haereditarii, illorumque origine*, Hal.

    Also included in Hoffman's Collected Works: Opp., Supplem. ii, I, 1753, Genève, p. 523. And in posterior editions as part of volume VIII. Amoreux: "le principe mechanique ...y fut soutenu; on n'y admet que l'origine organique des maladies héréditaires".

    Girard states that he used observations made and compiled by Zeller, and

provided a theoretical framework for them. (Zeller, undated, see below). He also writes: “un attachement inviolable au système du solidisme, avec cette ridicule manie de vouloir assigner la raison mécanique de tous les phénomenes de la nature, deparent? singuliérement les ouvrages de ce grand homme”.

This work was analysed in detail by Charles Daremberg in his *Histoire des Sciences Médicales*, 1860, vol 2, pp. 949-953. Daremberg writes: “Hoffmann rapporte d’abord les opinions des anciens sur les causes de l’hérédité morbide, et énumere les maladies qui, à leurs yeux, passaient pour héréditaires; puis il propose son explication mécanique, rejetant toute idée d’un être ou d’une activité spirituelle (efficacia spiritualis) qui, se transmettant par la génération, est capable par lui-même de d’agir ou de ne pas agir. Il n’y a que des causes corporelles, physiques, agissant de nécessité et mécaniques; ...Hoffmann reconnaît dans la semence, avec Malpighi et Malebranche, des linéaments, des filets (stamina) qui sont comme les diminutifs, le compendium de l’organisme à venir.” p. 949.

1.3 1700-1800

1. 1701, Christian, *Dissertatio de natura humana in dispositionibus haereditariis*, Basil.
2. 1701, Zwinger Theodori, *Dissertatio de naturae humanae inclinatione & dispositione haereditaria*, Basilea, in 4o.

   Amoreux writes that there are other mentions to hereditary diseases in this author's Pathological works, which can be found among his dissertations collected in Basilea, 1710, in 8o.
3. 1702, de Pré Joannis Friderici, *De morbis archealibus sive haereditariis*, Erf. 4o.
4. 1702, Roberg, *Dissertatio de morbis haereditariis*, Upsal.
5. 1706, Baldinger, *Dissertatio de morum & morborum transplantatione*, N.Mag. VII B. I, a Bergen, Fr.
6. 1706, Stahl Georgi Ernesti, *Dissertatio inauguralis de haereditaria dispositione ad varios affectus*, Hal., 4o. (A resply to Burchard.)

   Amoreux: "Le celebre cathedraticien fit soutenir de plus fort dans cette dissertation son systême sur l'empire de l'âme rationelle sur le corps. Stahl s'est declarée pour l'hérédité des maladies dans plusieurs chapitres de la Pathologie". p. 5. This text was also strongly recommended by Diderot in the *Encyclopedie.*
7. 1710, Rivin(us) Auguste Quirin, *Dissertatio de morbis haereditariis*, in Coll.Diss.Med.,n.35, (Appio) Lips.,4o.
8. 1712, Gibbs, Observations on *Scrophula and other hereditary diseases*, London, in 8o.

   Amoreux:"il y'a beaucoup de speculation de theories dans cette ouvrage", p. 6
9. 1718, Camerarius R.J., *Dissertatio haereditate morborum*, Tub.
10. 1718, Bulffinger? , *De Haereditate Morborum*, Tubinga, in 4o.
11. 1720, Schurig Mart., *Spermatologia*, Franc., p. 192, 4to.
12. 1720, Teutscher(us) Joann Gottl., *Dissertatio de eo, quod morbi chronici plerumque parentibus jure haereditario sint congeniti, vel in juventute acquisiti*, Erf.in 4o.
13. 1723, de St. Aignan, *an Morbus haereditarius arte sanabilus*, Paris.
14. 1727, Meyenberg, *De haemorroidibus haereditarius*, Zal., in 8o.

    Amoreux:"Cette dissertation fut soutenue sous la presidence de Michel Albert, qui tant a ecrit d'aprés les principes de Stahl", p. 6.
15. 1734, Wirth Tobias, *Dissertatio de morbis haereditariis*, Hafniae (or Copenhagen).

    Discussed by Conway Zirkle in "The early history of the idea of inheritance of acquired characters ...", 1946, pp. 105-106.
16. 1742, Schrader J. Henri, *De Haemopthisi haereditaria*, Helmt. 2nd. ed. in 1752.
17. 1749, de Büchner, *Dissertatio de secura morborum haereditarium praeservatione*, Hal.

18. 1749, Louis Antoine, Dissertation envoyée á l'Academie des Sciences de Dijon, pour le prix de l'année 1748, sur la question ...Comment se fait la transmission des maladies héréditaires? , Paris, Delaguete, 12o.

    This dissertation failed to win even a mention by the judges of the competition. Once published, however, its strong scepticism and tight argumentation generated a considerable debate that culminated in setting another essay price by the Royal Society of Medicine in 1788. It certainly received more comment than any other text before it, and was mentioned and criticized until the second half of the 19th century (see chapter 3). The only other essay published from the Dijon competition was Rey's (see below). There is probably a collection of the competition essays in the archives of the Dijon Academy. It would be of interest to recue the ones written by Chambon of Montpellier (winner), and Gravier of Parray (runner-up with Rey of Chaumont).

19. 1749, Rey Guillaume, *Sur la transmission des Maladies Héréditaires, qui a balancé le Prix de l'Academie de Dijon en 1748*. 12o. 24pp.

    Amoreux:"l'auteur soutient le système de l'emboîtement des germes, il est bornés des idées metaphysiques".p. 7. There seems to have been another edition of his work in 1752, in Paris.

20. 1753, Juncker, *Dissertatio de liberis ab haereditate morbosa parentum legitime excludendis*, Hal., 4o.

21. 1753, Wolf(f) Joh. Jacobin, *Dissertatio de morbis haereditariis*, 32p. in 4o., Basilea.

    Amoreux:"l'auteur a cru trop facilement aux maladies héréditaires. Il y a acumulé des observations de toute espèce pour une infinite de maladies reputés héréditaires. [But he arranged them] sous une ordre alphabetique. Cette erudition indigeste, n'est pas propre a jetter du jour sur un pareil sujet."p. 7

22. 1754, de Poletyka, *Dissertatio de morbis haereditariis*, Lugd., Batau, in 4o.

23. 1755, Büttner, *Dissertatio de qualitatibus corporis humani haereditariis*, Goett.

24. 1756, Schwalbe, *Dissertatio de dispositione haereditaria*, Hal.

25. 1757, Morand, *ex Heroibus Heroes*, thèse aux ecoles de Médicine, Paris.

26. 1758, Procopius, *Dissertatio de morbis haereditariis in genere*, Erl.

27. 1760? , Diderot D. (?), "Héréditaire" (maladie) in *L'Encyclopedie. Dict. Rais. des Sci. des Arts et des Matières*, t.VIII, p. 157

28. 1765, Eschenbach, *Pr. de morbis haereditariis*, Rostoch.

29. 1767, Vogel, *Dissertatio de nonullis parentum deliciis, in morbos infantum plerumque degenerantibus*, Goett.

30. 1768, Nolde I.L., *Dissertatio de parentum morbis, in foetum transeuntibus*, Erf. in 4o.

31. 1771, Cadogan W., *A Dissertation on the Gout and other Chronic Diseases*, London, J.Dodsley. He argues against strong hereditary influence, and for an hygienical cure of chronic ailments; an important influence on 19th century sceptics about hereditary transmission.

32. 1771, Bellosti J. Bapt., *Phtysy haereditaria semper precaveri valeat*,

Montpellier, in 4o. 8p.

33. 1774, Reiniger, *Dissertatio de prole, parentum culpas luente*, Lips. (Appio), 4o.
34. 1775, Matthias J. Gott., *Dissertatio sistens generalis(sima) dispositio(nis) haereditaria(e), et morborum in determinatorum therapia*. Hal.
35. 1781, Portal Antoine, *Observations sur les phthisies de naissances*, Paris.
36. 1787, Chavet Henrico, *De Pthisie pulmonalis haereditaria*, Monasteri (Munster) Westphalorum, in 8o., 183p. See article X, 193-239. The Royal Society of Medicine of Paris gave this work an honorary mention in a competition in 1781.
37. 1788, Pagès Jean-François, *Animadversiones qaedam circa morbus haereditarias*, Laureated thèse, Montpellier, in 8o., 44p. This was the first version of the dissertation Pagès presented at the 1790 Royal Society Competition, for which he obtained an honorary mention, and that later Vicq D'Azyr decided to incorporate to the *Encyclopedie Methodique* (1798), see below. Amoreux praised this thesis as much better than any run of-the-mill work by a student.
38. 1788, Wirchmann, *Dissertatio de morbis haereditariis*, Erf.
39. 1793, Davids, Dissertatio affectionibus haereditariis, Leideia (also *Salzb. Med. chir. Zeitung*, 1801, 1 B, p. 410; and in Hollnd, Museum I, N.6
40. 1794, Müller, *Dissertatio de dispositione ad morbos haereditaria*, Goett, 4o.(also *Salzb. medic. chir. Zeitung*, 1798, IV, p. 193)
41. 1794, Rougemont Joseph-Claude, *Abhandlung über die erblichen krankheiten*; translated into German from the French manuscript that was crowned by the Royal Society of Medicine at Paris, in its 1790 competition (see below) (Translator: Friedrich Gerhard Wegeler), Frankfurt, in 8o. Also published in *Salzb. med. chir. Zeitung*, 1794, IV, p. 291; and N. A. D. B., XVII, B., p. 39; and *Tode medic. Journal*, II, B., 2 St., p. 70, A. L. Z., 1975, n.184.
42. 1798, Pagès Jean-François, "Héréditaires (maladies)" in *Dictionnaire de Médecine*, part of the *Encyclopedie Methodique*, vol. VII, an VI de la République, chez Agasse. pp. 160-176.

    This entry is essentially the piece that was honoured in 1790 by the Royal Society of Medicine. It is an excellent defense of solid to solid hereditary transmission, and as a Dictionary piece must have been widely read, preceding Petit's essay (1817) in breaking the ground for a broader view of heredity. It was chosen for the Dictionary by Vicq D'Azyr among the many very competent dissertations for the competition (See below).
43. 1799, (Eschenbach?), *Ueber Familienkrankheiten, besonders die scrofelnartigen*, L, 8, N. A. D. B. B. LI, p. 97
44. 1799, Zettermann, *Dissertatio de morbis haereditariis*, Jen.(and *Salzb. medic. chir. Zeitung*, 1801, IV B, p. 404.)
45. 1800, Henning J.G.F., *Ideen über die Erbkrankheiten*, Zerbst., 8o., N. A. D. B. B. LVIII,p. 321, (pro iis) A.L.Z. 1802, n.249.

1.4 1788-1790

Dissertations submitted for the Royal Society of Medicine's essay competitions on Hereditary Disease of 1788 and 1790

Most of these dissertations remained unpublished and the surviving manuscripts and are kept in the Bibliotèque de L'Académie de Médecine, in Paris, in the Archives of the old Royal Society of Medicine, where I consulted them.

1.4.1 The first contest, 1788

"La Société avoit proposé, dans la séance publique du 27 février 1787, pour sujet d'un prix de la valeur de 600 liv., fondé par le Roi, la question suivante: Déterminer 1o. s'il existe des maladies vraiment héréditaires, & quelles elles sont: 2o. s'il est au pouvoir de la médecine d'en empêcher le développement, ou de les guérir après qu'elles se sont déclarées."(*Hist. et Mém. de la Soc. Roy*., vol.IX, 8, 1786, p. 17-18).

The judges for this first competition were doctors Thouret, Chauseru, l'Abbé Tessier and Crochet, They could not agree on which piece deserved the prize, and eventually concluded that none responded well enough to the question raised. They decided to reopen the contest, with a higher prize (800 liv.) (f.n. See Minute: Examen des memoires sur les Maladies Héréditaires, document 181-23-1,5 in Archives of the Old Royal Society; also *Hist. Soc. Roy.*, vol.IX, pp. 17-18, and see also the personal account that Alexis Pujol wrote of this contest, in his *Oeuvres de médecine practique*, vol.2, pp. 214- 217, see below).

1. Amoreux Pierre-Joseph, "Des Maladies Héréditaires". 75p. First version. (The document is marked in judges' code as: I; in the classification of the Bibliotèque de l'Academie Nationale it is document 200-2-2. I will be giving both of these codes for all documents below).

   Although puzzled by this first essay's failure to convince the judges, Amoreux reworked it considerably for the re-run, adding many "evidences from authority" as well as an increased number of facts collected from all sorts of sources (see below). Amoreux (fils) was born at Baucaire, the son of a known physician. He taught most of his life at Montpellier. Many of his important historical researches remain unpublished, according to Dezeimeris et. al. (1828).
2. Bésuchet, "Sur le Maladies Héréditaires", 59p. (E; 200-2-3). This dissertation is very well informed, and was perhaps the one that backed its positions with theoretical material from the naturalists, like Réamur and Bonnet. B. was from Nozeroy
3. Bruch, "Morborum haereditarium existenten illustrates" 5p., (M; 200-2-4), in Latin.
4. Coffinières, "Memoire sur les Maladies Héréditaires", 75p. (C; 200-2-5). Thouret wrote about this piece:"trés etendu, assez bien fait, contient rien très frappant. Generalités; vices des solides. Projet de conseil de santé pour assortir les marriages. Details vagues. Curabilité des maladies pretendu hereditaire grande". The reference to the Health Council for Marriages could be interesting for a historian of Eugenics, specially since Coffinières bases his

idea on several previous authors. He highlights a 1760 work by Le Camus, on the necessity of improving the human (national) stock, and stopping the malconformed from reproducing their “degenerescences”.

Here is an overview of Coffinières ideas on Hereditary transmission of diseases, which was a fairly standard (half solidist-half humoralist) amongst the authors of that period, but one which the top members of the Royal Society were struggling to displace, in favour of a clearer solidist position (see below, the note on Rougemont’s work): “Les maladies héréditaires existent dans la semence du pere et dans le germe de la mere, et l’un et l’autre peuvent les communiquer a leur fruit. Ces maladies se propagent d’un individu a l’autre par une conformation particuliere des solides, ou par des levains introduits, ou conservés dans les fluides. La variété dans les solides et l’immense division des fluides forment pour leur ensemble et dans leur reunion cette diversité de temperaments, ces manieres d’etre que disposent les uns à la des passions violentes, à des maladies actives, et les autres à des affections langourentes et des maladies chroniques ...le plupart [des m.h.] ont des ages et des epoques qui lui facilitent leur developpment. Les maladies des solides, ainsi que leurs dispositions sont gravées dans l’embrion; celles des fluides etendent plus loin leur domaine, puisqu’une nouvelle depravation des fluides peut être introduite pour la mere et par la nourriciere ...” C. was from Castel Naudary (?) en Languedoc.

5. Gellei Michel-Raphaël, “Morbi haereditarii”, (N; 200-2-6). Written in Latin, in a polished aphoristic style, this dissertation was judged the best of the first lot. It received as encouragement prize a gold medal worth 100 livres. The titles of his sections suggest the line of his argument:

   I. Morbi haereditarii specifici absolute necesarii.

   II.Morbi per accidens haerediatarii, iique.

   A.Hereditarii justa causa materialis.

   B. Haereditarii causa predisposante spirituales.

   The judges wrote about this work: “Le sens su programme y est bien saisi; & quoique, sous plusieurs rapports, les réponses aux questions proposées y soient incomplètes” (*Hist. Soc. Roy*., loc.cit. p. 18). de Gellei was from Vienna.

6. van de Leeuw F.W., “De Morbis Haereditariis”, 33p. (L; 200-2-12 (1 a 2)). A Latin dissertation, with an epigraphe from Antoine Louis’ piece (above). In it, according to Thouret, he refutes successfully Louis’ sceptical arguments but does not address the questions raised by the Royal Society.. Leeuw was from Dordrecht.

7. Loreille, “Sur les Maladies Héréditaires”, (B; 200-2-7). A summing up: “Il y a deux especes de maladies héréditaires. Les unes viennent de la mauvaise constitution des solides, et les autres du vice des humeurs. Les premières sont incurables. Les seconds peuvent se guerir”. L. was from Buy.

8. Munnier, “Dissertation sur les Maladies Héréditaires”, 44p. (A; 200-2-8). Munnier was from Vesou.

9. Pujol Alexis, “Essai sur le Maladies Héréditaires” (H;200-2-9). First version.

   Pujol extended it considerably for the 1790 re-run but refused to eliminate the theological bits, which according to him, were the reason why he did not win.

His overwhelming erudition was however clouded by a strong humoralism (he advocated both kinds of causation), which was already becoming outdated by then. (Considered as “Trés Bon” and “erudite” by judges, was 2nd best and received a mention). Pujol was born at Poujol, near Breziers, in 1739 (Oct.10).

He studied at Toulouse and took his degree in 1762. He lived and worked at Castres, and won several prizes with his essays at Royal Society Competitions. Pujol died the 15 of September, 1804.(Data taken from Dezeimeris, 1828). Pujol’s works were collected as *Oeuvres Médicales* de Pujol, Castres, in 4 vol, 8o., 1802. Boisseau reissued them as *Oeuvres de Médecine Practique* in 2 vols., Paris, Bailliere & Bechet, in 1823. The second version of the “Essai sur les Maladies Héréditaires”, with the added sections on physiology, appeared in both editions with an Introduction. In the 1823 edition Boisseau worded a commentary on this essay, from a hygienecist point of view, criticizing the use of the hereditary cause as an abuse of language: “il me parait que ces mots ‘maladie héréditaire’ sont rarement applicables ...Toutes les fois que l’enfant en venant au monde n’est point affecté de la maladie de son père ou de sa mère, lors même qu’elle se developpe plus tard chez luis on ne peut dire que soit héréditaire. L’aptitude a contracté et seule été héréditaire, les causes qui on agi sur lui en fait le reste”. Further on he adds: “La distinction que Pujol admet entre les maldies congeniti et connati est une pure subtilité ...puisque nous ne savons en que consiste la conception”. He concludes that there is no way to know at which moment of the new being’s life story the influence is exerted, and by which mechanism. (pp. 432-433).

10. Rechou, “Essai sur les Maladies Héréditaires”, 33p. (K; 200-2-10). R. was surgeon at Chevancoup.
11. Rey, “Sur les Maladies Héréditaires”, 51p., (D; 200-2-11). R. was from Cazillac.
12. — (F) This is a missing dissertation in the archives. We have only the judges’ annotations and a mention to the epigraph under which it was presented. It may have been Pagès piece, but it had a different epigraph (“Il suffit pas qu’un systeme soit possible pour meriter d’etre cru”, Voltaire) than Pagès’ published work (1890), which was taken from the archives of the Royal Society for its publication in the *Enc. Méth*. by Vicq d’Azyr. Alternatively, it might have been Rougemont’s text, which cannot be found (as Pagés’) in the collection. It could also have been of course from a third author. In any case, the judges thought it the third best of the lot, and it was granted a mention in 1788. Thouret writes: “Il en admit leur existence (de m.h.) avant trop peu de doutes sur leur realité, ce memoire est une de mieux redigé”(*Arch.Roy.Soc*., 183-23-1)
13. Vigei de Llorens Thomas, “De Morbis Haereditariis”, (G; 119-29 (a-d)). Vigei was from Barcelona. His Latin dissertation was considered outlandish, he proposes a sort of “physiological memory”. He sent a supplement for the re-run.

    The judges’ summary after the first round, in which the prize was reset: “La plupart des concurrens ont supposé plutôt qu’ils n’ont prouvé l’existence des maladies héréditaires; ils n’en ont pas assez exactement deteminé la nature. Il s’agit de savoir si quelques-uns de vices morbifiques se transmettent réellement & individuellement des pères aux enfans, ou si les maladies qu’on

appele hérédiatires, ne sont pas plutôt une suite de la conformation des organes, qui, dans les pères & dans les enfans, doivent être, à raison de leur structure, sujets aux mêmes affections. C'est sur l'existence & la nature de ces maladies qu'il faut sur-tout porter ses recherches.(*Hist.de la Soc. Roy.*, vol.IX, p. 18).

1.4.2 The second contest, 1790

(New judges Caille, Doublet, Laguérene, Mauduyt?)

1. Amoreux Pierre-Joseph, "Des Maladies Héréditaires" (10; 120-3-1). This piece was given the accesit. An outstanding, modified and extended version of the 1788 document. Amoreux shows abundantly the historical depth of the theme, and displays an impressive knowledge of the bibliography. Several of the comments on 17th and 18th century texts above are taken from this important manuscript. This text seems to have been close to winning the contest, and the judges wrote of it that: "L'Auteur de ce Mémoire a développé une érudition très-étendue. La Société auroit desiré de trouver plus de détails dans les traitemens prophylactique & curatif.(*Hist.Soc.Roy.*,vol.9,p. xi)
2. Bidot-Peyret, "Commentarium in questionem ...Morbis Haerediatriis", (119-32). B.-P. was from Nay at Beneamie (?)
3. Coray, "Sur les Maladies Héréditaires" (181-1). C. was from Montpellier
4. Girard, "Recherches philosophiques et medicales sur les maladies héréditaires", 80p., (12; 119-33-A). This is a very exhaustive investigation. Covers the theme from about every possible angle. It also has a very good historical section, with useful comments on previous bibliography, some of which I reproduce above. On previous authors' analysis of hereditary transmission Girard wrote: "La theorie offre des differentes opinions sur la maniere dont ces maux passent des parens a leur posterité, sur les loix de cette propagation, les raisons que tiennent ces germes cachés, les principes qui les developpent, ce qui tient au mystère de la génération a l'analogie des resemblances, aux metamorphoses des âges, aux lois de contagions, aux systemes de fermens".
5. Kreuzner L. N., "Disseratio de Morbis Haereditariis" (6; 119-31). K. was a physician in Vienna.
6. Ladevère, "Essai sur la question proposé ...s'il existent vraiment des Maladies Héréditaires". (119-30-5).
7. Pagès Jean-François, "Les Maladies Héréditaires", manuscript missing from the Archives. Won the first honorary mention, and was published in the Encyclopedie Méthodique.(Above). Pagès was a physician at Alais.
8. Pujol Alexis, "Memoire sur les maladies héréditaires"(9; 120-3-5). Second honorary mention. Extended version of previous submission. Published in Pujol's Works. For biobibliographical data see above.
9. Rougemont Joseph-Claude, "Discourse sur les Maladies Héréditaires". This work obtained the 800 livres prize, given on August 31, 1790. The manuscript is missing from the Archives. It almost certainly was taken to Germany by the author, where he had it translated and published (see above, 1794). The judges of the Royal Society wrote about this piece: "La question est traité sous tous ses rapports dans ce Mémoire, qui contient une exacte & sévère analyse de

tous les écrits & de tous les faits qui ont quelque relation avec le problême proposé. Les maladies héréditaires y sont bien distinguées de celles que l'enfant peut contracter soit dans le sein de la mère, soit au moment de l'accouchement. On auroit pu desirer plus de méthode dans quelques parties de cet ouvrage; mais lorsque l'Auteur mérite ce reproche, il y supplée par de la clarté." (*Histoire de la Société Royale de Médecine*, vol.9, 1787-88 (backdated), pp. x-xi). Rougemont was born in Santo Domingo, in 1756 (Dec. 10), and was educated at Dijon and Paris, where he graduated with a First prize. In 1783 he became the physician of the elector of Cologne and was given the chair of anatomy and surgery at the University of Bonn. He died the 28 May, 1818. He published several works, among which this one is usually highlighted (biographical information from Dezeimeres, 1828).

The Royal Society's closing words on the theme were the following: "Quoique la Société ait cru devoir distribuer ce Prix qui avoit déja été remis, elle est bien éloingnée de croire que la question soit epuisée; elle la regarde au contraire comme ayant besoin encore de nouveaux éclaircissemens qu'elle attend attend du zèle de ses coopérateurs. Dans ce genre, les obsevations isolées considerées séparément, ne peuvent avoir qu'un degré d'utilité très-borné. Ce ne sera qu'en les réunissant & les comparant, qu'on pourra leur donner de la valeur. La Société publiera les noms de ceux qui lui auront communiqué de nouveaux faits, & elle décernera des Prix d'encouragement, à ceux qui lui auront remis les observations les plus importantes." La Société didn't know of course that its days were numbered, and that in only a few more months it would be dissolved. Nor could it know that the themes it had promoted with so much interest would become the seed of a new biological concept, "heredity", one that would dominate biology and, somehow paradoxically, the ideology of the next post-revolutionary century.

1.5 After 1800

Only those works specifically focused on hereditary diseases, including insanity, are included. Primarily France and Britain are covered. There must be many German works that could complete this bibliography. Left out are most of the better known general treatises on hereditary transmission that include the theme of hereditary disease (see general bibliography of the thesis for these). I also leave out many short notes, reports and comments appearing in journals and magazines, which tend to increase exponentially with the popularity of the theme. Hereditary madness received eventually most of the attention, specially in France (This can be seen in the Annales médico-psychologiques after the 1850s. See for that the cumulative index of the Annales, and the notes of Dowbiggin's *Inheriting Madness* (1990)).

1. 1802, Pujol Alexis, "Essai sur les Maladies Héréditaires" in *Oeuvres Médicales*, 4 vol., Castres, in 8o. Second edition edited by F.G. Boisseau, with notes, a biography and additions, as *Oeuvres de Médecine Practique*, 2 vols., Paris, Bailliere & Bechet, 1823. This essay was written for the Royal Society of Medicine competition in 1788-90, see above.
2. 1803, Darwin Erasmus, "Hereditary Diseases", additional note XI to *The Temple of Nature*, London.
3. 1808, Portal Antoine, *Considerations sur la nature et le traitement de quelques maladies héréditaires, ou de famille*. Mem. Inst. Nat. de France, T.8,

Semestre 2, p. 156. (Read at the Institute the 25 January 1808). Later published as book in 1808. Paris, 4o. And in a revised and extended version in 1814. 3eme edition, Paris. Extracts of this work were also published in *Graperon*, Bull. des Sci. Méd., Tome 2, p. 328.

Almost certainly this work prompted Joseph Adams' famous treatise on the subject, in which he tried to claim for John Hunter, and himself, the "modernization" of the concept of hereditary transmission of disease (see below). The English (edited) version of the piece was published in two installments in Adam's *London Medical and Physical Journal*, volume 21, in Dec. 1808 and June 1809, pp. 229-239, and 281-296.

4. 1813, Fodéré Emmanuele François, "Des maladies héréditaires", section II, Chap.II, 3eme partie, tome 5 of *Traité de Médecine Legale et d'Hygiene Publique*, pp. 358-382
5. 1814, Adams Joseph, *A Treatise on the Hereditary Properties of Diseases*, London, J.Callow (a facsimile reproduction of this work was published in Charles Rosenberg's series History of Hereditarian Thought, 1984, Garland Publishing). The second edition of this work, in 1815, with only a few additions as appendixes received a much more ambitious title: A philosophical dissertation on the hereditary peculiarities of the human constitution. A German translation appeared in the *Neu Sammlung auserlesener Abhandlungen* (tome II, p. 503).
6. 1815, Henning George, "On the supposed Hereditary and Constitutional Nature of Scrofula", chapter III, pp. 49-79 of *A critical inquiry into the Pathology of Scrofula*, London, Callow, 8vo.
7. 1817, Petit Antoine, *Essai sur les Maladies Héréditaires*, Paris, Gabon, 88 pp. This essay became very influential when it was incorporated, with slight changes, as the entry under "Héréditaire" in the *Dictionnaire des Sciences Medicales*, vol.20, pp. 58-86. It was, after Pagès', the most forceful defense of a solidist hereditary transmission.
8. 1823, Poilroux J., *Nouvelles recherches sur les maladies chroniques et principalememt sur les affections organiques et les maladies héréditaires*, 8o., Paris.
9. 1823, Anon., "Héréditaire (maladie)", in *Dictionnaire Abregé des Sciences Médicales*, vol.9, Paris, pp. 45-49
10. 1831, Sauvé, *Dissertation sur les maladies héréditaires*, Thèse de doctorat, no. 189, Faculté de Médecine, Paris.
11. 1833, Brown Joseph, "Hereditary Transmission of Disease" in *The Cyclopedia of Practical Medicine* (J.Forbes, editor), vol.2, pp. 417-419
12. 1834, Lereboullet D.A., *De l'hérédité dans les maladies*, Thése de concours pour l'agrégation, Strasbourg, G.Silbermann imp.
13. 1834, Robin, *De l'hérédité dans les Maladies*, Thèse de doctorat, no.226, Faculté de Médecine, Paris.
14. 1835, Lustremant E., *De l'hérédité dans les maladies et des indications qu'elle fournit*. Thése de doctorat, Paris. 10 de Janv. no.5
15. 1836, Sersiron, *De l'hérédité dans les maladies*, Thèse de doctorat, no.339, Faculté de Médecine, Paris.
16. 1838, Ladmirault, *De l'influence de l'hérédité sur la production des maladies*,

Thèse de doctorat, no.189, Faculté de Médecine, Paris.

17. 1845, Béclère Claude, *De l'Hérédité dans les Maladies*, Doctorat Thesis, Faculté de Médecine, Paris, Rignoux imp., 22 pp.
18. 1839, Holland Henry, "On Hereditary Disease", chapter II of *Medical Notes and Reflections*, London, 1st. ed., pp. 9-37. Some important additions were made to the 3rd. edition, 1855, pp. 16-53.
19. 1840, Piorry P.-A., *De l'hérédité dans les maladies*, thèse de concours pour la chaire de pathologie, Faculté de Médecine, Bury imp., Paris. See also Piorry's "Aptitudes, diàtheses, prédispositions constitutionelles à la maladie", chapter III of "De l'etiologie ou de l'etude des causes dans les maladies" in vol.I of his *Traité de Pathologie Iatrique et de Médecine Practique*, Paris, Fourchez, 1841.
20. 1843, Steinau Julius Henry, *Pathological & Philosophical Essay on Hereditary Diseases*, London, Marshall & Co. Partially inspired by Rougemont's work (above), a first version of this dissertation was published in Berlin a few years before and enjoyed, according to its author, a considerable success. The book was favourably reviewed in British journals, and Darwin mentioned it as a good source in his *Variation* ...
21. 1844, Baillarger J., "Recherches statistiques sur l'hérédité de la folie", in *Annales médico- psychologiques*, t.III, pp. 328-339.
22. 1845, Gaussail A.J., *De l'influence de l'hérédité sur la production de la surexcitation nerveuse sur les maladies qui en résultent et des moyens de les guérir*, 8o., Paris.
23. 1845, Gintrac E., "Mémoire sur l'influence de l'hérédité sur la production de la surexcitation nerveuse", de les maladies qui en résultent et des moyens de les guérir. First prize at the Academy of Medicine, in *Mémoires de l'Academie Royale de Médicine*, 11, pp. 193-382, and offprints, Paris.
24. 1846, Phillips Benjamin, "The Hereditary Cause" in chapter IV of *Scrofula*, London, pp. 99-125. This sceptical text was inspired by Henning (above) and was used by Buckle in his attack on hereditary transmission (see chapter 6).
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   Girard (1790) writes about this work: "Les redacteurs de *L'Encyclopedie* assurent qu'on peut consulter utilment cet écrit, je le cherché en vain mais un savant bibliographe m'a certifié que ce qu'il contenoit d'essentiel se trouvoi dans Hoffmann qui se l'est appropié." This dates the text before 1699.
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Appendix 2

Statistics and Human Hereditary Talent, Alphonse de Candolle vs Francis Galton

The study of de Candolle's *Histoire des Sciences et des Savants* (HSS henceforth)[1] has a very substantial tale to tell about the development of what we now know as the nature-nurture debate in the early post-Darwinian decades, and about the incorporation of statistical methods to it.

Within the arc of the two editions of this book (HSS-1, 1873; HSS-2, 1885) a tense struggle can be perceived in the mind of its author that took him from an overtly "nurturist" position with regard to the causes of scientific eminence, to a confused "naturist" one. It is in the companion essays included beside the main text of HSS where most clues to this development can be found, specially in the numerous addenda to the second edition that put much strain into what aspired to be a coherent set of texts. They deal with many subjects, but the most relevant ones are focused in heredity: "Recherches sur l'Hérédité"(1873) and "Nouvelles Recherches sur l'Hérédité"(1885).[2]

The publication of this book prompted a debate, that developed from hostility to friendliness, between its author and Francis Galton, and a necessary move to bring under a proper light de Candolle's views on the causes that tend to bring forth scientific eminence, in these separate editions, is to face them with the double mirror of two of Galton's works, *Hereditary Genius* (HG, 1869) and *English Men of Science, their Nature and Nurture* (EMS, 1874).

The aim and content of the works of these two authors so closely interacted during a few years (1869-1885) that, whatever posterior developments that condition our present day impression,[3] it seems clear that in a way they belonged to the same enterprise: to pull under the new Darwinian umbrella the old tradition of hereditary theorizing focused on humans. In this sense, it is revealing that, in reviewing the

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[1] *Histoire des Sciences et des Savants despuis deux Siècles*, 1st ed. 1873, 2nd ed. 1885, Georg, Genève-Bale. A new edition (1987) Libraire Arthème Fayard.

[2] Two more are "La statistique, procédé regulier d'observation"(HSS-1), and the long study "Sur la part d'influence de l'hérédité, de la variabilité et de la sélection dans le développement de l'espèce humaine" (HSS-1), both of which are more than important to the argument and to the historical evaluation of the central essay of HSS.

[3] Specially the work on Heredity and Statistics of the Galtonian school after *Natural Inheritance (1889)*.

German translation of de Candolle's work,[4] George Sarton picked these two authors and these particular works as the initiation of a new discipline "la gèniologie".[5] Karl Pearson, even when believing de Candolle's views to be off the right track, could not find a better companion to Galton's initial hereditarian studies than de Candolle, and gave center stage to their mutual letters.

2.1 De Candolle

Alphonse de Candolle (1806-1893) was the son of an eminent Swiss botanist[6] and built himself a high scientific reputation in the same field, which led him in 1873 to what, by his own standards, was the highest recognition: to become a foreign associate to the Académie des Sciences de Paris. His most important work is considered to be the *Geographie Botanique Raisonné* (1855),[7] in which, as J. Browne has shown in *The Secular Ark*,[8] he managed to overcome the atheoretical impasse in which the Humboldtian tradition of arithmetical phytogeography was immersed. He did that by allowing the statistical tabulations to become the base for a kind of causal analysis."He showed —writes Browne— naturalists the way in which figures could be used to vindicate or elaborate upon hypotheses designed to explain geographical distribution".[9] This work was an important influence on Darwin.[10]

De Candolle had a lifelong penchant to the application of statistical and geographical analysis to a whole variety of subjects besides botany. The procedure of regionalizing the subject, quantifying and tabulating the elements of each region, comparing the regions and finally sifting through the possible causes to explain the differences, was applied by him, with the pertinent variations, to such varied fields as crime,[11] savings systems,[12] and in his HSS, the production of scientific eminence.

De Candolle believed that most empirical and non-demonstrative (non-

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[4]*Zur Geschichte der Wissenchaften und der Gelehrten...*, Deutsch herausgegeben von Wilhelm Otswald. Leipzig, Akademische Verlagsgesellschaft, 1911.

[5]Sarton, G., *Isis*, I, 1913, pp. 132-33. Within this discipline Sarton certainly situated his first "inconclue" major contribution to Isis ("Comment augmenter le rendement intellectuel de l'humanité? ", I, pp. 219-242, 416-473), and he seems to have conceived of it as a portion of the History of Science that required a high input of biological fact, mainly from Heredity Theories; thus quite close to the Eugenic program.

[6]Agustin Pyramus de Candolle, 1778-1841.

[7]This book is often referred to as a landmark in biogeography.

[8]Browne, J., 1983, *The Secular Ark, Studies in the History of Biogeography*, Yale University Press, Chapter 3.

[9]ibid. p.85

[10]Darwin followed closely de Candolle's botanical work, they wrote to each other (see Baehni, Ch.,"Correspondance de Charles Darwin et d'Alphonse de Candolle", *Gesnerus*, 1955, 12:109-56. According to J-M Drouin, de Candolle is one of "des cinq auteurs les plus souvent cités par Darwin dans l'ensemble de l'ouvrage" (see note 32).

[11]A. de C., 1830, "Statistique. Considerations sur la statistique du dèlits", *Bibl. Univ. des Sci, Bel.-Let. et Arts, I, p.159.*

[12]A. de C., 1836, "Recherches sur l'origine de l'institution des caisses d'epargne", *Bibl. Univ. de Genève, Septembre.*

mathematical) disciplines should benefit from the application of statistics as a method of observation, the condition being that one could "énumerer des faits de même categorie et les comparer, par groupes".[13] The procedure, he wrote, can eliminate many of the most usual reasoning vices but "arrive seulement à des probabilités". It is applicable within a whole range of disciplines, in which he includes his own (natural history) and many of the moral sciences:"Le naturaliste —he writes—, comme l'historien ou le jurisconsulte, est un homme disposé a comparer plusieurs faits, dont aucun n'est absolutement prouvé, et plusieurs arguments, dont aucun n'est absolument rigoureux".[14] He believed, in short, that this demonstrative-probabilistic dichotomy was a better way of classifying the sciences than, for instance, the natural-social one. This similarity of situation, he believed, is what asks for the use of similar tools, and what allows a prudent and careful observer and statistician (like himself) to move across the disciplinary boundaries. Specially targetting, in his case, problems with a structure suited for the statistico-geographical approach.

The boundaries across which de Candolle moved were not, of course, those we would see by transporting back our present disciplinary divisions. Heredity, for instance, was by the mid-nineteenth century a domain that, at least in the French-speaking world, did not have a clear locus in the natural nor to the social (or moral) sciences, but seemed to be multiply rooted: with anthropologists, physiologists, psychologists, physicians, breeders, and naturalists feeding it with very diverse and not quite compatible materials. However strange it might seem to us, the belief that the study of human cases was more likely to inform that of other species than vice versa was not uncommon. To unriddle human heredity was also, for some, the most urgent task, and seemed the easiest to tackle. "...l'homme —de Candolle wrote— est plus connu dans sa marche au travers des siècles que toute autre espèce, et nous avons sur lui des détails que embrassent déjà quelques milliers d'annés. Il jouit des facultés intellectuelles très-étendues."[15]

It is of course the heredity of the latter faculties that attracted a great deal of attention. In a very influential footnote, within his *History of Civilization in England*,[16] H. T. Buckle, who had reasons of his own to dislike the possibility of such inheritance, called for a suspension of any theorizing around the matter until a proper statistical base could be shown to support the hypothesis. I believe there are good reasons to maintain that both de Candolle and Francis Galton were, in their hereditarian studies, taking up the gauntlet thrown down by Buckle.[17]

2.2 Patriotic Science

Although de Candolle claimed that he only resorted to study human cases when it couldn't be avoided ("plus je me suis occupé des sciences sociales...plus il m'est arrivé de préférer la botanique"),[18] it seems plausible that he undertook those studies as a kind of moral duty. For instance, the ambition to establish on "scientific" grounds

[13]A. de Candolle, 1885, (HSS-2),. p.14.
[14]A. de Candolle, 1873 (HSS-1), p.110.
[15]HSS-1, p. 13.
[16]See Buckle, H. T., 1857, Chapter IV, pp. 161-162
[17]This is a point that Robert Olby has made several times. in Olby (1985) and "Galton's response to Buckle".
[18]HSS-1, p. 13.

what he considered to be a major source of importance of Switzerland, and particularly of Genève, and more particularly of French protestant families like his, was, it can be suspected when following the arguments, a main motivation for the research behind HSS. It was a long standing interest. As he wrote in 1873 to Galton,[19] he first got the idea to use the nominations of foreign members of important European Académies as evidential base 40 years before (i.e. circa 1833).[20] De Candolle père was nominated in 1824 to the French Académie, and in 1829 belonged also to the Berlin Academie and the Royal Society of London, so that could have helped him get the idea, which was praised by Galton as providing a very good and objective starting point for statistical analysis.[21]

The regionalizations and the tabular arrangements which de Candolle proceeded to construct, can be seen, in my view, as a natural extension of his usual statistical-geographical approach.[22] He did a similar thing when he studied the history of the "caisses d'epargne de la Suisse"[23] exploiting the legal cantonization of the country to sift his way towards the possible historical causes. That in both cases the "objective" methodology[24] led to parallel conclusions in which Genève stood high over the rest of Switzerland, and over the rest of the world is a question that I shall leave to more malicious minds.

I wish now to look at where de Candolle met with real and unexpected difficulties, during and after the writing of the different texts that he joined to make up the volume of HSS-1. These had to do with the causation analysis he wanted to give of his results. The causes (or pre-efficients, as he called them) that could lead to the apparition of highly talented scientists in particular places at particular times could, broadly speaking, belong to the natural and/or to the cultural realms. Among the former, what came with the physical characteristics of men could or could not be due to heredity. What was not inherited could be a spontaneous novelty or variation (*inneité*, in Prosper Lucas' terms, see notes 52, 56). On the cultural side causes were

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[19]Galton shared the preoccupation to show scientifically the intellectual worth of his family relations.

[20]See letter of 2 Janvier, 1873, from de Candolle to Galton, in Pearson Karl, *Life, Letters, and Labours of Francis Galton* (LL & L), 1924, Cambridge UP,v.II,p.136. Or in G. de Morsier, "Correspondance inédite (sic) entre Alphonse de Candolle (1806-1893) et Francis Galton (1822-1911)", *Gesnerus*, vol. 29, 1972, p.131.

[21]See letter, Galton to de Candolle, of Dec. 27, 1872, in Pearson, LL & L, v.II, p. 135.

[22]J-M Drouin believes that "le méthode géographique et historique que de Candolle applique à la communauté scientifique européene, n'est pas pour lui une imitation de celle qu'il applique aux flores des différentes régions".(ref 32, p. 161) They are, he seems to believe, different instances of a more general methodology. I believe that view to be the one the author (de Candolle) wanted to prevail, but certainly not the one that a detailed analysis shows. The detailed similarities of approach, and the parallels of many unforced decisions are too many.

[23]"Recherches sur l'origine de l'institution des caisses d'epargne".

[24]De Candolle, it must be said, insisted in the unavoidable subjectivity of the decisions that the use of statistics forced on the observer. He believed that only a kind of judicial balancing of evidence by well intentioned and experienced workers would warrant a decent level of objectivity. "L'appréciation des faits, sous le rapport de leur qualité et de leur nombre à côte de l'ensemble des faits anlogues ou contradictoires, joue donc nécessairement un grand rôle dans la méthode". (HSS-2, p.15)

either distributed by nation (like education), group (like religion), or family (like habits and manners).

The analysis required first a proper statistical base. How to chose a significant sample? His first claim to objectivity, as I mentioned, was that evaluation of the scientific quality of the individuals considered was made by informed organs (Academies) on foreign members (which eliminates many subjective components), from three different European capitals. The number of contributions to lists from each country (and region, or city) should be, in principle, proportional to the number of their inhabitants. Any deviations from this proportions would ask for causal explanation. So, for instance, "Si les savants de quelques petits pays sont nombreux sur les deux listes, anglaise et française, a telle époque, ce n'est pas par hasard ni qu'on se fût concerté. Si des pays très peuplés n'ont aucun représentant sur les listes ou n'en ont qu'un petit nombre, les conclusions à déduire sont tout aussi évidentes".[25] Three epochs for three Academies provided enough evidence that the worlds scientific talent was concentrated in Europe, and specifically in France, Britain, Germany, Belgium, Switzerland and Italy. Adjusting for sizes made obvious the obvious. Now the search for the causes.

De Candolle's geographical approach in this study imposed several restrictions over the kind of causes he could sensibly expect to unveil. He obviously shared the Eurocentrist belief that the racial (inheritable) differences gave white people a clear superiority over the remainder of humankind. But the differences he was interested in were within the race; among Europeans. He wanted to explain, for instance, why Spaniards or Irish were so backwards and unscientific, as their share in eminence proved. The list of causes he analysed is the following: heredity, education, religion, family traditions, opinions, institutions and type of government, size of country, language, climate and race.

His preference in the first edition was to give higher explanatory power to what we would call cultural causes. The kind of qualitative differences that he envisaged as accounting for scientific eminence (a free spirit, strong will, hard working habits, etc.) were to his eyes too unspecific to be determinant in the development of particular skills. His kind of analysis, on the other hand, favoured the elucidation of external and long standing causal influences, such as religion, type of government, tradition, etc. Heredity was not an easy cause for him to handle, as no European community of the epoch could claim any sort of racial or even group purity or stability. Besides, his view of how Heredity worked, although not a clear one (as most of his contemporaries'), did not allow for long-term constant causation. The influence of the previous two generations being overwhelmingly stronger, when detailed characters are concerned, than that of the rest of the ancestry.[26] His favourite example, that of the energizing effect that the Huguenot migration had on the intellectual level of the communities that adopted them, was not accounted for appealing to any racial or group inherited characteristic but to their traditional attitudes. It seems thus certain, as Karl Pearson wrote, that de Candolle was somehow taken by surprise during the course of his lengthy (and perhaps delayed) investigation focused on the "environmental" causes, and that "before his work was issued Darwin first and then

[25]HSS-2, in the Fayard's re-edition, 1987, p. 14.

[26]This view was somehow explicated by Galton's Ancestral Heredity Law, to which de Candolle adhered unreservedly, and which he in fact used when arguing with its author. See letter, 2 Janvier 1873. in LL & L, p.136.

Galton flared up on his horizon and the whole aspect of the country he was investigating appeared different",[27] making heredity an unwelcome but unavoidable factor in his study. Pearson adds: "His book when it was written became a compromise between the old aspect and the new."

Probably, in an earlier or uninfluenced version, Heredity could have been left out of the picture, or at least relegated to a second plane. Tradition (in its multiple aspects, national, familiar, etc.) would have emerged unchallenged as the maker of great men, with the exceptions of "geniuses", whose occurrence was unpredictable and uncontrollable anyway.[28]

2.3 The reach of Heredity

It was then in order to avoid a head on collision with Francis Galton, who had tactically rallied to Charles Darwin's support,[29] that Alphonse de Candolle modified the emphatically nurturist position that by training and sentiment he initially preferred. As Karl Pearson noted,[30] the alteration in the subtitle of HSS-2 signals the change in the author's emphasis. While in HSS-1 the subtitle read "Suivie d'autres Études sur des sujets scientifiques en particulier sur la sélection dans l'espèce humaine", the second had "...en particulier sur L'HÉRÉDITÉ ET LA SÉLECTION..." Pearson's implication is that Galton opened de Candolle's eyes to the importance of heredity, but that was not the case. It was rather Darwin's argument for an evolution of species through Natural Selection that made him focus on it. De Candolle had, in his pre-Darwinian and excellent treatise on *Geographie Botanique Raissonné*, come close to an evolutionist conclusion when considering the distribution and diversity of insular floras.[31] What he could not imagine then was a way in which isolated varieties could become stable and begin thus a speciation process. Natural Selection, working over heritable variation, was such a way: "on comprendr —he writes— pourquoi je me suis attacher volontiers à etudié l'hérédité, la diversité dans l'hérédité et la sélection. Je l'ai fait avec un vif dèsir de voir les idées nouvelles appuyer mes anciennes opinions."

In another post-Darwinian work, previous to HSS-1, de Candolle, had considered the influence of selection and hereditary transmission as a factor in the transformation of human societies within historical time and found it to be negligible[32] As J-M.

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[27]In his LL & L, v.II, pag. 149.

[28]This, as other views of de Candolle's concerning heredity and inneité, owe much to Prosper Lucas. See notes 52 and 56.

[29]"I feel the injustice you have done to me strongly, and one reason I did not write earlier was that I might first hear the independent verdict of some scientific man who had read both books. This I have now done, having seen Mr Darwin, whose opinion confirms mine in every particular." Letter of Dec.27, 1872, LL & L, p. 135.

[30]LL & L, p. 145. For an article focusing on the Nature-Nurture debate and Galton and De Candolle's positions, see Fancher, R.E., 1983, *J. Hist. Behav. Sci.* 19, pp. 341-53.

[31]See HSS-1, p.7, p.12, and *Géographie Botanique Raissonné*, pp. 1087-1098.

[32]"Assurément depuis les anciens Hébreux, Grecs et Romains, les hommes de la race blanche ont bien lutté, sois individuellement, soit collectivement. Les plus faibles au point de vue physique ou intellectuel ont tojours eu un désavantage; les plus forts, physiquement et moralement, l'ont tojours emporté; et cependant, soit pour

Drouin[33] points out, these and other facts made de Candolle cautious when applying Darwinian views to nature, specially to human nature.

In the Introduction (later suppressed) to HSS-1, de Candolle claimed that unity within the diversity of such "volume de melanges" had been given by "l'idée de scruter l'importance du noveau principe de la sélection introduit par M. Darwin" for which purpose, he continued, "il faut voir d'abord s'il y a des dissemblances, d'une nature héréditaire entre generations succesives. Je n'ai donc rien négligé de ce qui concerne la question toujours fondamentale de l'hérédité.".[34]

However, De Candolle's position on the causes of scientific eminence, after his first set of investigations, was not a strong hereditarian one.

Faced with Darwin's scheme he had not only been favourable to it but tried to line up his whole work within its scope. Galton's HG however produced strain and discomfort in him. First as an unexpected competitor within the extension of the darwinian enterprise he had decided to undertake, and second as an obstinate and monolithic defender of the cause he was not prepared to deal with and he would rather had left in the background. Their aims methods and results were not the same, and there obviously was room for both their enterprises to succeed (Heredity and Tradition could be at work simultaneously), but the conflict between their explanatory stresses was enough to produce a confrontation. The first signs of it can be found in de Candolle's critical paragraphs,[35] in HSS-1, on Galton's too ready hereditarian explanations. He considered his sample and procedure much more reliable than Galton's. For instance, he did not believe (as he later made clear in his "Nouvelles Recherches..." in HSS-2) that selecting freely, as Galton had done, names of famous men from biographical dictionaries could give anything but a causally muddled sample; as names could end up there for an infinite and uncontrollable number of reasons.

De Candolle was however sufficiently impressed with Galton's own genealogical probabilistic reasoning as to try it on his own, more trustworthy sample. As Galton had it, if in a non-biased selection of eminent men, through several generations, more of them are members of the same families than would be expected in a random distribution, and if (this being the big "if") external influences can be discounted, then the existence of hereditary causal factors seems the explanation. De Candolle tightened up the access to the lists (there were for instance only eight foreign members at the French Académie at any given time) and found that Galton's claims could only be sustained for the case of mathematicians (the Bernoulli family being the archetypical case). This he related to his view of the different kind and number of capacities that are called to use in the two main types of inquisitive disciplines that, as we have seen, he envisaged. For mathematics (as for music), he claimed, there is a limited number of very special faculties that can certainly be inherited, whereas for the observational disciplines, based on many general and wide ranging capacities, the

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l'intelligence, soit pour la beauté physique, la force et la santé, on ne peu pas dire qu'il y ait une différence évidente entre les modernes et les anciens". A. de Candolle, "Étude sur l'espèce a l'occasion d'une revision des Cupulifères", *Archives des Sciences Physiques et Naturelles*, 15, 1862, p. 211-237.

[33]Drouin, Jean-Marc "Botanique et sciences sociales chez Alphonse de Candolle", *Corpus, revue de philosophie*, n.7, 2e trimestre, 1988, pp. 155-163.

[34]HSS-1, p. 1.

[35]HSS-1, pp. 93-94.

selection of an area of speciality is much less guided by heredity. “La faculté d’observer implique un ensemble assez varié de facultés. Ce n’est pas quelque chose de tout spécial, comme les mathématiques”.[36] So, in most cases “l’hérédité...produirait des combinaisons variées, et permettrait à beaucoup de jeunes gens de suivre une carrière ou une autre, une science ou une autre, avec la même probabilité de succès...jurisconsulte, historien, érudit, naturaliste, chimiste, géologue ou médecin”.[37]

A particular example which de Candolle emphasizes, one is tempted to think, in order to sting Galton’s skin (which he managed to) is the exceptional number of Savants in his lists that were sons of protestant priests.[38] This, he claimed, is unaccounted for by a purely hereditarian scheme. The appearance or disappearance of high level scientific achievement in different regions according to important institutional or political changes, while presumably the same hereditary factors persisted in them, was all in all the strongest point in favour of de Candolle’s environmentalist position.

Galton answered with a belligerent review,[39] and with a strong though carefully worded letter, before which he had secured the backing of his cousin Darwin.[40]

Among his disclaimers: that he (Galton) never intended the defense of a strong heredity of particular professions (although some of his chapters in HG clearly suggest the opposite). He criticized de Candolle for being continuously ambiguous about his views on heredity and its reach, and gave several examples where he contradicts himself or, simply, re-stated his (Galton’s) hypothesis: that the influence of race is superior to all others (HSS-1, p. 195), that within races (between families) the variation is sometimes greater than between races (HSS-1, p. 268), that physical structure and mental qualities are connected and if the former is inherited the latter too (HSS-1,p.326) etc. De Candolle’s methodology, only “afford means of disentangling the effect of one out of many groups of *contemporaneous* influences”(my italics), and thus is ideal for answering just one question: “what are the social conditions most likely to produce scientific investigators, irrespective of natural ability, and *a fortiori*, irrespective of theories of heredity? ”(italics on original).[41] “The author —Galton continues— however continually trespasses on heredity questions without, as it appears to me, any basis of fact, as he has collected next to nothing about the relatives of the people upon whom all his statistics are founded”.[42] This is not altogether fair because de Candolle did have, as we saw, a

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[36]HSS-1, p. 109.
[37]HSS-1, p. 107.
[38]Galton not only was strongly anti-clerical but had a chapter of his book dedicated to undermine the religious men’s image.
[39]“On the causes which operate to create Scientific Men” in *Fortnightly Review*, March 1, 1873, LXXV, vol.13, n.5, pp. 345-351.
[40]Darwin’s own reaction to HSS-1 is registered in a letter to de Candolle, of Dec. 11, 1872: “I have hardly ever read anything more original and interesting than your treatment of the causes which favour the development of scientific men. The whole was quite new to me...When I began your essay I was afraid that you were going to attack the principle of inheritance in relation to mind; but I soon found myself fully content to follow and accept your limitations...” in Baehni, Ch., op.cit. See note 10.
[41]F. Galton,“On the causes which operate...”, p. 345.
[42]idem. p 346.

diachronical analysis to back his anti-hereditarian claims.

The purely genealogical method, used in HG, did not convince Galton for much longer, and he finally saw the improvement of having a closed and standarized sample. In 1873, he decided to combine his and de Candolle's approach in the research that produced his *English Men of Science, their Nature and Nurture* (EMS). In the introductory letter to all his subjects ("persons in the United Kingdom who have filled positions of acknowledged rank"): "In the pursuit of an inquiry parallel to that of M. de Candolle, I have been engaged for some time past collecting information on the Antecedents of Eminent Men...The result of my past efforts has clearly impressed upon me the fact that a sufficiency of data cannot be obtained from biographies without extreme labour, if at all; therefore, instead of imperfectly analysing the past, it seems far preferable to deal with contemporary instances...".[43]

De Candolle had got through one point, but other points didn't land. When he answered Galton's first letter he was emphatic on the distrust he had of being self-indulgent in a statistical analysis; even when some evidence has been found favourable to a chosen causal factor (v.gr. heredity) the inquiry must be taken forward until the possible influence of as many causes as can be thought of are untangled. "Il ne m'a pas été difficile de confirmer par des nouveaus faits l'influence de l'hérédité, mais je n'ais jamais perdu de vue les autres causes, et la suite de mes recherches m'a convaincu qu'elles ont en général plus d'importance que l'hérédité proprement dite".[44]

What de Candolle wanted to see in Galton was an "environmental" analysis where the same kind of actors could be seen under different circumstances. Geography and History both could provide such opportunity. He grew impatient when Galton failed to produce such a test with his English Men of Science. Again, he writes , "on se prive de comparer les effets d'institutions sociales différentes ou successives, comme j'ai le pu faire dans le procédé que j'ai employé."[45] It becomes thus paradoxical that in his "Nouvelles Recherches sur l'Hérédité"(HSS-2), de Candolle in turn adopted and tried to perfect the Galtonian diachronic, generational, unbounded approach, where the evidential weight lies on highly subjective appreciations, and found heredity to be stronger than ever.[46]

Another curious give and take sequence can be followed if one focus the changing level of "achievement" of the men these two authors decided to present as basis for their statistical analysis of the inheritance of moral characters, from HG to HSS-1 to EMS to HSS-2. According to de Candolle, the "geniuses" of the first book belonged to many different kinds, and owed their distinction to too many different characteristics.[47] The lists of the foreign members of the Académies, Galton replied, "are specialists, rather than illustrious men, and are therefore somewhat obscure to fame...they have owed more to concentration and the narrowing of their faculties than to a general prodigality of their nature. Such men are more easily affected by

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[43]See Pearson, LL & L, vol. II, p. 177.
[44]See Pearson, LL & L, vol.II, p.137.
[45]HSS-2, Fayard, p. 84.
[46]"I shall be very curious indeed —Galton wrote to him— to see how far my own data will confirm yours in the "Nouvelles Recherches", but doubt much whether they will show the effect of heredity to be so strong"(!) Letter of the 17 Oct. 1884. G. de Morsier, "Correspondance...", p. 541.
[47]LL & L, vol. II, p. 136.

circumstances than the born geniuses about whom I chiefly busied myself". Then in EMS Galton lowered the stakes with a selection of contemporary English "Medallists of the chief scientific societies; the Presidents of the same, now and in former years...members of the Council of the Royal Society".[48] De Candolle considered this to be "une catégorie de savants moins élevée que celle...dont je me suis occupé...Cette limitation permet de mieux scruter les influences de naissance comparées aux autres, puisque l'éducation, les lois, les moeurs, etc., sont peu près semblables".[49] In his "Nouvelles Recherches..." de Candolle takes the critique further: "nous avons tous deux choisi certains individus distingués ou certaines familles, en raison précisément de leur mérite. C'est ne pas une choix impartial pouvant donner des résultats statistiques précis...Quand on raisonne sur des éléments exceptionnels on se prive de beaucoup de ressources que donnerait l'étude de l'ensemble de tous les éléments".[50] He then picks up his new sample by a non-merit related criterion (personal acquaintance) and tries to give a following of the transmission of four categories of characters in families through the three generations that one old and careful observer (himself again) can get to know.

The result of the "Nouvelles Recherches..." is very strange to contemporary eyes, as it exposes many of de Candolle's pre-Mendelian ("weird") views on heredity, and seems a step backwards from his more tasteful previous research. (This of course could have been a major, wrong-headed, reason to supress them from the re-edition). The strong hereditarianism that comes through does not totally contradict de Candolle's previous "environmentalist" position, but owes too much I believe to the desire not to be left on the wrong side of the post-darwinian wave that completely transformed, towards the end of the century, the whole landscape of hereditarian theorizing. With hindsight one can see that he was left on that undesired place, and that Galton, by having had different intuitions and prejudices about what kind of mechanisms where working in hereditary transmission, could stubbornly develop his statistical techniques up to a point where others could take over.

In many aspects, the series HG, HSS-1, EMS, HSS-2 can be understood as a imperfect dialogue of agreements and disagreements. The attempted rapprochement of two authors who shared a commitment to change the evidential procedures of a "pre-scientific" domain, as heredity —in Galton's words— had been until them, through the incorporation of statistical analysis. Who wanted to accommodate their views but found obscure incompatibilities: The different preconceptions on what sort of a thing heredity is, and more crucially, on how to categorize human characteristics, worked underneath their decisions to make the homogeneity they desired impossible.

De Candolle was certainly right when he declared that both him and Galton had managed to face up Buckle's challenge. "Il y a dans nos travaux un progrès relativement au procédé ordinaire de citer des cas isolés favorables à l'hérédité".[51] The different statistical approaches they successively tried to defend and/or attack the hereditarian view of intellectual and moral faculties left however, as de Candolle could see, just as many important choices on the subjective evaluation of the researcher. The selective picking of favourable cases from medical archives, or of anecdotes from biographies, and their use as rhetorical devices to tip the balance in

[48]LL & L, vol. II, p. 177.
[49]HSS-2, Fayard, p. 84.
[50]HSS-2, p. 55.
[51]HSS-2, p. 55.

favour (or against) hereditarian hypothesis, was supplanted by the selective picking of favourable samples where the examples (cases, anecdotes) would thus appear to be unpicked (i.e. objective). The rhetorical function that story-telling played was taken over by the statistical tables in the assessment of probabilities. The continuity between the two approaches was clearly seen by de Candolle when he wrote that statistics "est un travail d'un homme qui cherche à se debrouiller au milieu de faits mal connus, déterminés par des causes elles-mêmes variées et mal connues. Il s'efforce de compter les faits, de les peser, de les classer et de les comparer. Avec une forte tête, il pourrait le faire sans chiffres; mais alors ses éléments de conviction seraient personnels, par conséquent contestables."[52]

The misunderstandings between de Candolle and Galton show, did not disappear. Too much still depended on their beliefs and expectations. Their differing approaches to the categorizations of human characteristics in relation to heredity was, on my opinion, crucial. De Candolle still adhered to a dual view, similar to Prosper Lucas's,[53] in which one set of characteristics determined the belonging to a group (species, race, even family), and a qualitatively different set determined the peculiarities of the individual. This duality allows a clear-cut distinction between what heredity (nature) and what variational influences (nurture among them) respectively affect, and is not easily connected to an overall hereditarian scheme, which he nevertheless ended up doing. Galton on the other hand worked under the idea of a unique kind of hereditarian causation for all human characteristics, from physical to intellectual. In what we would now feel to be a very modern way, he imagined that heredity was due to the existence, continuity and transmission through the generations of "wonderfully varied elements...all latent and competing" within batches. "These batches —he wrote—, and not the persons derived from them, form the principal successive stages in the line of direct descent". In what would deserve the anachronistic apellative of a "bean-bag" fashion, Galton imagined each character determined by one element, and that "each personality originates in a small selection out of a large batch".[54] This underlying unity of causation (and not his statistical analysis) displaced any external (environmental) effect to a secondary role. It also gave him the confidence on the appropriateness of using a continuous distribution function, such as Quetelet's "normal" curve, and of covering with it even the most exceptional (improbable) occurrences, as that of "geniuses".[55]

2.4 Epilogue

The correspondence between them went on for a long time, until de Candolle's death

[52]HSS-2, Fayard, p. 100.
[53]Lucas, P. *Traité de l'Hérédité Naturelle*, Paris, 1853.
[54]See F. Galton "On the causes which operate..." and "On blood-relationship", *Proc. Roy. Soc.*, 20, 1872, pp. 394-402.
[55]The apparently sporadic and unexpected appearances of "geniuses" in families of average intelligence and achievement had been for a long time, within the Hereditarian tradition, a typical example of the non-inherited, spontaneous kind of variation controlled by other type of causes; Lucas called them inneité. Galton saw as his prize to be able to claim even those most exceptional of occurrences as explained by his scheme. In 1892, he regretted having chosen the word "genius", and not the more neutral "ability" in the title of his book. But it seems to me that in 1869 he had concrete reasons for the decision, which later disappeared.

(1893), After the first couple of letters the tone of successive interchanges between them calmed down as they sought to avoid a rupture. De Candolle was a more prestigious scientist and had in a sense less to lose. He however showed more willingness to compromise and one can only speculate about the role Darwin's position (who apparently, and perhaps unfortunately, sided with Galton) played in this. Neither of them would have wished to be judged badly by Darwin.[56]

De Candolle ended up making most concessions as he struggled to articulate a stronger causal role for hereditary factors with his earlier "environmentalist" results. Most of the changes and additions to the HSS-2 seem designed to flatten the anti-hereditarian edges (which in several cases were explicitly anti-Galtonian). He was thus gleeful when he received Galton's letter of approval."Je suis très hereux de penser que vous approuvez mes dernières recherches sur l'hérédité! "[57]

The chain of replies and counter-movements between these two very careful and clever nineteenth century gentlemen-scientists, when closely followed, unveils many details about the struggle in those days to introduce statistical analysis into such a complex field as traditional human Heredity. All sorts of crucial decisions about the kind of evidential samples likely to produce the most objective results, about the kind of characteristics acceptable as stable and heritable through generations, about the type of analytical procedures more adequate to the underlying causal structure of the phenomena, etc., were taken by these and other authors based on their differing backgrounds and expectations. Subsequent developments, like the physiologization of hereditary research on the one hand, and the Mendelian-Biometrician dispute on the other have, so to speak, deformed the space in which we situate these early post-Darwinian researches. Only by not paying excessive attention to what came later, I believe, can we hope to evaluate properly how and why these men conceived and faced their scientific enterprises.

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[56]In all their interchanges they referred to Darwin reverently; de Candolle consulted Galton concerning Darwin's personal characters when preparing HSS-2, and wrote a very laudatory obituary at his death, which he then passed to Galton for judgement. The piece is "Darwin consideré au point de vue des causes de son succès et de l'importance de ses travaux", *Archives des Sciences de la Bibliotèque Universelle*, 3-période, 7, p. 14, 1882.

[57]Galton's letter: Oct. 17, 1884. De Candolle's: Oct. 27, 1884, See LL & L, vol. II, p.207-209.

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