

# The concepts of heredity and degeneration in the work of Jean-Martin Charcot

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## ABSTRACT

Transcripts of the Tuesday Lessons at La Salpêtrière Hospital show that Jean-Martin Charcot often asked his patients about their family history. The information gathered on patients' heredity played also a significant role in the diagnostic reasoning he instructed his students in. Again and again, he included in his teachings the concept of degeneration to suggest an etiology for observed pathologies. This article analyzes the origin of Charcot's knowledge, imparted in the Tuesday Lessons, by examining the theories of heredity and degeneration successively developed by Prosper Lucas (1808–1885) in 1847, Bénédict-Auguste Morel (1809–1873) in 1857, and Jacques-Joseph Moreau de Tours (–1804–1884) in 1859. I will review examples taken from the Tuesday Lessons to illustrate how Charcot assimilated the ideas of these alienists. Two of his students, Charles Féré (1852–1907) and Georges Gilles de la Tourette (1857–1904), known for championing their master's work, went on to publish their own books that developed theories of heredity and degeneration. I will conclude my review, which aims to examine a little known facet of Charcot's work, with a few examples from these authors' writings.

## KEYWORDS

Charcot; Morel; Lucas; Moreau de Tours; Féré; Gilles de la Tourette; heredity; degeneration theory; La Salpêtrière; history of neurology

What we know is the major obstacle to acquiring knowledge of what we don't know.

Claude Bernard (Bernard 1865)

By the middle of the nineteenth century, the anatomoclinical method developed by René Laennec (1781–1826) was fully deployed by Jean-Martin Charcot (1825–1893; see [Figure 1](#)). This experimental method, and that introduced by Claude Bernard (1813–1878), freed medicine—and research on nervous system diseases, in particular—from doctrinal systems it had conveyed, in a fog of speculative philosophies, for centuries. One of the pillars of medical reasoning remained particularly fragile: the concept of heredity. Influenced by the lectures of Auguste Comte (1798–1857) on positive philosophy, Charcot set himself the task of establishing laws governing the pathologies he encountered, through logic and observation, in line with experiments involving anatomical dissections and observations compiled by Giovanni Battista Morgagni (1682–1771) during the eighteenth century (Lellouche 1992; Morgagni 1761). He was an adept of this scientific medicine, as he described it in the introduction to his *Clinical Lectures on Diseases of Old Age* (the original French was published in 1867). His collaboration with Claude Bernard (1813–1878) in his early years bears witness to this scientific mindset (Bernard and Charcot 1851). Whereas Claude Bernard favored physiology over anatomy, Charcot's credo

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**Figure 1.** Unpublished photo of Jean-Martin Charcot by Albert Londe (1858–1917) (OW collection).

was the “supreme jurisdiction of the clinical” over anatomy and physiology. Yet, apparently without realizing it, he remained, in fact, a prisoner of empirical medicine, adhering to the concepts of degeneration and hereditary transmission of nervous system diseases (Charcot 1867). As established by Goetz, Bonduelle, and Gelfand (1995), “Far from Bernardian vivisectional experiments, Charcot’s clinical gaze hearkened back to the great naturalist Cuvier’s noninterventionist observation of nature’s experiment (Georges Cuvier 1769–1832) and beyond that to the first generation of Paris clinicians who sought to follow the precedent of the surgeon’s penetrating but essential observational epistemology” (55). After reviewing the state of

knowledge available to Charcot in these domains, I will bring it to bear on his descriptions of a few neurological diseases to better grasp this facet of his work.

Since time immemorial, we have been aware of the transmission of physical characteristics from parents to their children, and have tried to understand how this happens. Michel de Montaigne (1533–1592) expressed his own interrogations on this subject in 1582: “What is this monster carried within this drop of seed from which we are produced—the impressions not only of bodily form, but also the thoughts and inclinations of our fathers? Where does this drop of water house this infinite number of forms?”

Gregor Mendel (1822–1884), recognized as the founder of genetics, was Charcot’s contemporary, and his experiments on peas date from 1857, the year Charcot failed his first *agrégation* exam (to enter the path to professorship). Yet Charcot was certainly unaware of the publication (Mendel 1865) on what would later be named the laws of Mendel (Genopole 2020). So what knowledge was at his disposal? In the mid-nineteenth century in France, three authors had proposed theories of heredity: Prosper Lucas (1808–1885) in 1847, Bénédict-Auguste Morel (1809–1873) in 1857, and Jacques-Joseph Moreau de Tours (1804–1884) in 1859.

At this time, heredity was considered a legal and institutional concept that governed the transmission of titles and property within families. Certain political events, such as the abolition of the transmission of certain legislative titles (*hérédité de la pairie*) in 1831, and the end of *majorat* arrangements (attribution of most of the patrimony to the eldest male heir in the family) in 1835, may have influenced the thinking of Lucas and Morel. Conservatives and the clergy saw these new developments as destabilizing successional regimes and threatening social continuity between generations; at the same time, alienists were bringing the contingencies and risks of heredity to light. After a law pertaining to the insane was voted on under King Louis-Philippe I in 1838, the medicalization of heredity extended the medical scope to the organization of social life, which was split between counterrevolutionary Catholic traditions and the emergence of a social Catholicism aimed at improving the fate of the poorest citizens (Dowbiggin 1991; Hochmann 2018). As the same time, we cannot minimize the desire of alienists to acquire specific knowledge with which to advise families, and which would have enhanced their standing as well as distinguished them from the multitude of healers competing with them for the treatment of mental illness.

### **Georges-Louis Leclerc, Count of Buffon**

Georges-Louis Leclerc, Count of Buffon (1707–1788) and director of the Royal Garden of Medicinal Plants, was the first author to use the term “degeneration” in his treatise on the variability of human appearance (de Buffon 1749). For him, all men were originally white, but the skin color of some of their descendants changed due to environmental influences, which he interpreted as degeneration.

### **Prosper Lucas and his *Traité philosophique et physiologique de l’hérédité naturelle***

Prosper Lucas, born on November 4, 1808, into a wealthy and influential family in Saint-Brieuc (Brittany), went to Paris to study medicine (no portrait of Lucas exists). He defended a remarkable thesis on August 28, 1833, before a jury presided over by

Gabriel Andral (1797–1876). Entitled *De l'imitation contagieuse ou de la propagation sympathique des névroses et des monomanies* (On contagious imitation or sympathetic propagation of neurosis and monomania; Lucas 1833), it was inspired by Franz-Joseph Gall (1758–1828) and Pierre Jean Georges Cabanis (1757–1808) and could serve as a historical introduction to the discovery of mirror neurons in our brains as well as a study of mass psychology (Figure 2). Lucas was first absorbed by political life, the 1848 Revolution, and his own failures to convince the electorate. His career as an alienist only started in 1864, when he took over at the Bicêtre asylum from Louis-Victor Marcé (1826–1864), who had recently committed suicide (Luauté and Lempérière 2012). His

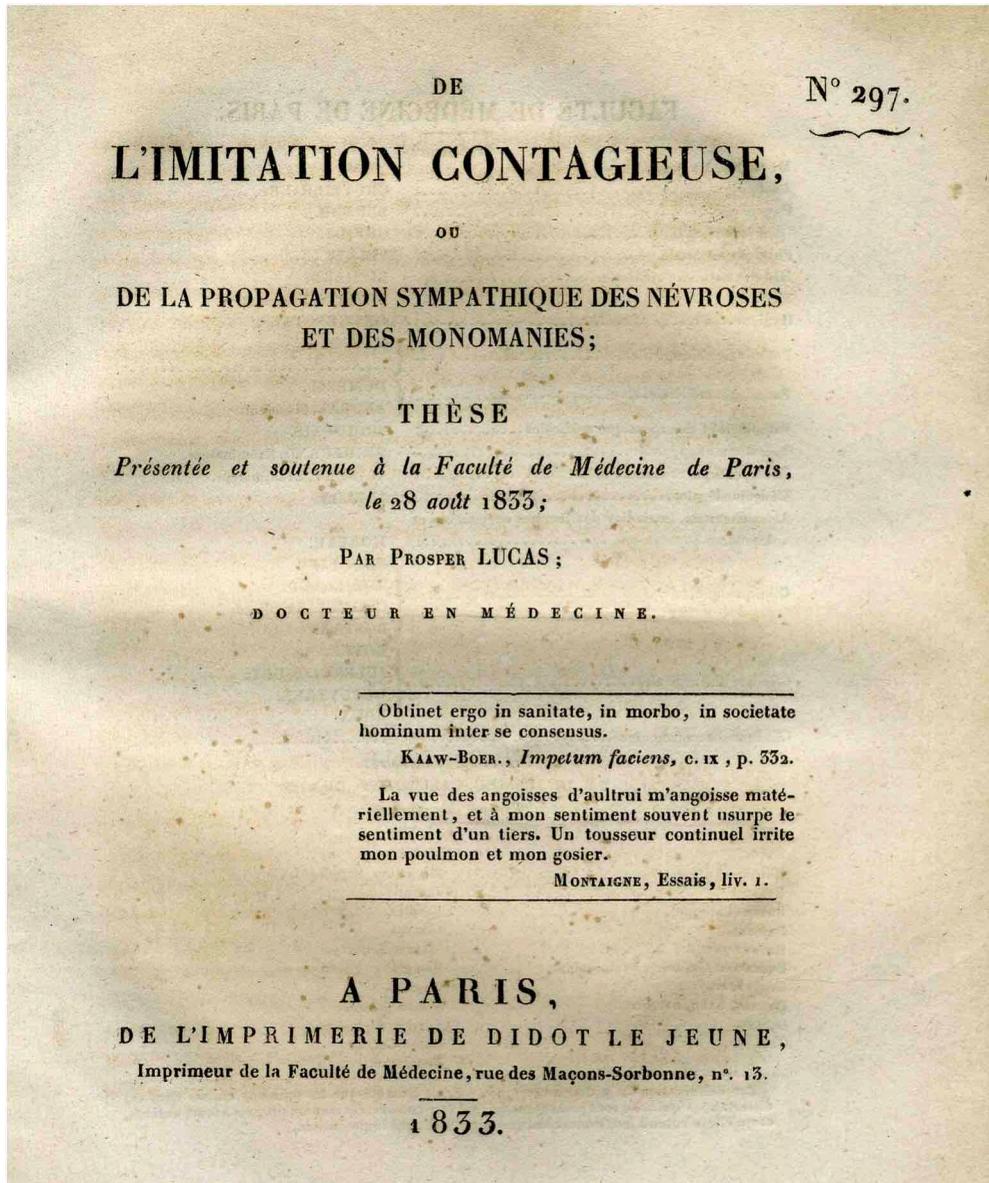


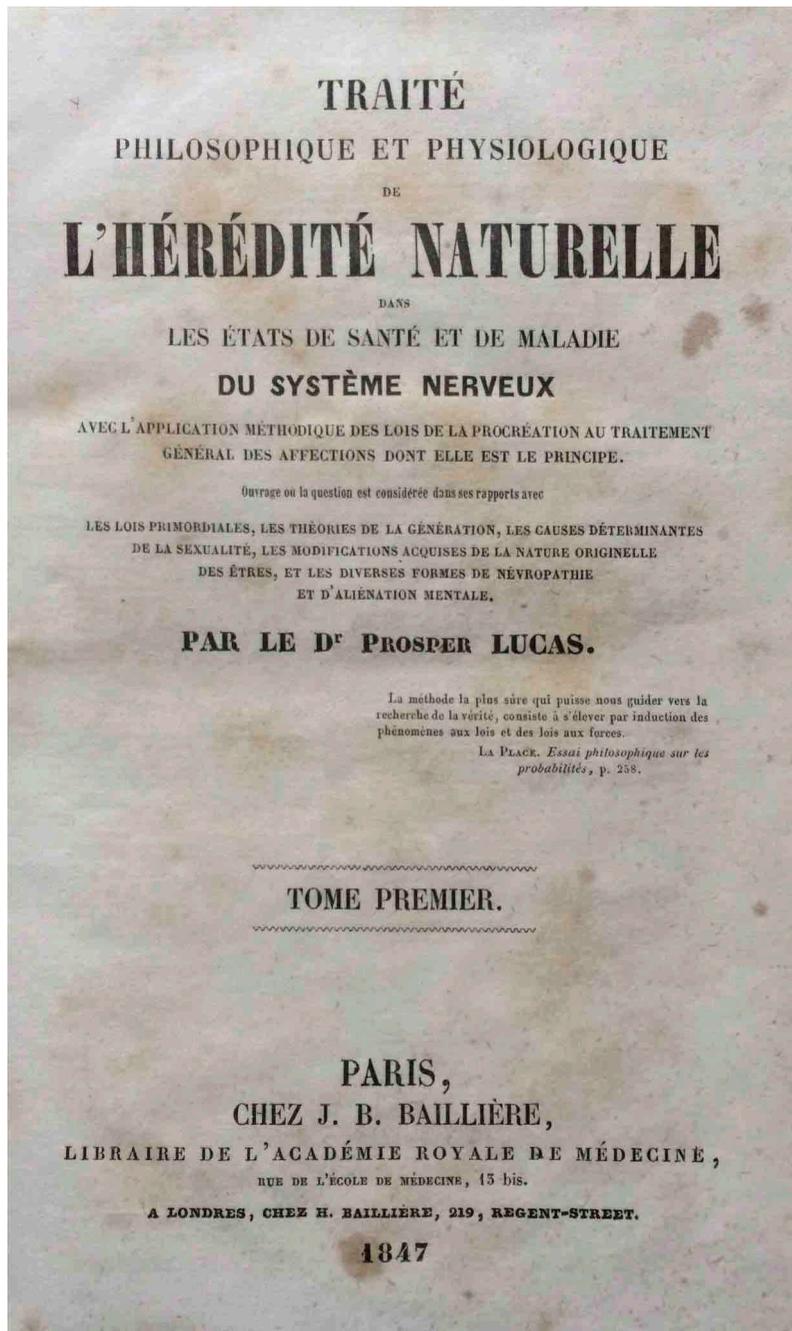
Figure 2. Thesis of Prosper Lucas in 1833 (OW collection).

first resident was Valentin Magnan (1835–1916). On March 31, 1867, Lucas began his position as chief physician of the recently inaugurated Women’s Division of the Clinique de Sainte-Anne asylum, recently inaugurated. With Magnan, Gustave Bouchereau (1835–1900), and Henri Dagonet (1823–1902), he initiated clinical lectures on mental pathology at this asylum in 1872, acting as dean. The next year, his lessons were interrupted by the Prefect of the Seine following a press campaign orchestrated by the newspaper *Le Figaro*. Critics objected to his exhibition of insane patients during his lectures. Although the content of his teachings was not published, the title in the notices for his classes is explicit: “The importance of the science of mental illness and why its study is necessary for physicians and judges, from various viewpoints in modern society.” Retiring in 1879, Lucas left Paris for his property in Mennecy, south of Corbeil-Essonnes (50 kilometers south of Paris), where he died on April 2, 1885 (Trehou 1993).

The publication of his voluminous *Traité philosophique et physiologique de l’hérédité naturelle dans les états de santé et de maladie du système nerveux* (Philosophical and physiological treatise on natural heredity in states of health and nervous system disease), over 1500 pages published in two volumes in 1847 and 1850, not only ensured him lasting fame but also inspired Charles Darwin (1809–1882) and Émile Zola (1840–1902). Darwin read Lucas’s treatise in 1856, as he indicated in his notes, published in 1987 (Barrett 1987), and used several of Lucas’s examples to support his theory, presented in 1868 in *The Variation of Animals and Plants under Domestication* (Darwin 1868), and in 1871 in *The Descent of Man, and Selection in Relation to Sex* (Darwin 1871). What Zola took from this treatise were his deterministic ideas about the human species, which he put into play with the Rougon-Macquart family (Hamon 1983). As for César Lombroso (1836–1909) and Théodule Ribot (1839–1916), they took from this treatise the concept of morbid heredity.

Lucas started by mentioning his predecessors, such Antoine Louis (1723–1792; Louis 1749) and notably the 1828 hybridization experiments of Charles Girou de Buzareingues (1773–1856), a forerunner of Mendel working in the Rouergue region of southern France (Girou de Buzareingues 1828). Lucas’s book, which smacks of scholasticism but reveals his vast erudition, is dense, full of digressions, and hard to read. In summary, Lucas saw procreation as obeying two laws, “innateness, or the law of expression of the diverse, and heredity, or the law of expression of the similar.” Innateness, or “law of invention,” is “what constitutes the originality, imagination, and liberty of life in mediate generation,” which can be understood in terms of trait variability and phenotype. Lucas used the qualifier of invention, which is similar to adaptation in Darwin and would become the idea of mutation. Heredity, or “law of imitation,” represents “what constitutes the repetition and memory of life in the same type of generation,” which he understood as genotype. Lucas saw heredity as “the immutability of the nature of species,” the basis for the fixism in which he believed. In the many examples he cited, he confused genetically determined traits (albinism, for example) and embryogenic defects (harelip, for example). Lucas refuted numerous legends, such as “the action of imagination on coitus” and the role of adultery in malformations, attributed at that time to heredity (Figure 3).

He also added the idea of heredity of similarity and heredity of metamorphosis; in the first case, a disease was transmitted identically, whereas in the second, a nervous disease emerged in the descendant, differentiating him or her from the ascendant. Heredity may be a predisposition, “heredity of the seed”—that is, a latent state that may or may not be revealed at a given time in life, or be a potential and variable disease state. For Lucas, this



**Figure 3.** Cover of Volume 1 of the work by Prosper Lucas (OW collection).

theory manifested itself during mental illness and was mixed together with notions of “return heredity” (or atavism, by which one resembles ancestors rather than parents), heredity of influence (influence of places and climate), and homochronic heredity (correspondence of age of manifestation; see Table 1). His theory attempted to explain situations

**Table 1.** Some definitions of terms used in this article.

Term	Definition
Heredity of similarity or immediate heredity	A disease is transmitted identically.
Heredity of metamorphosis or heredity of transformation	A nervous disease emerges in the descendant, differentiating him or her from the ascendant.
Heredity of predisposition or dissimilar heredity	A latent state that may or may not be revealed at a given time in life, or be a potential and variable disease state.
“Return heredity” or atavism	Resemblance with ancestors rather than parents (transmission of characteristics jumping one or more generations, with possibly a backward jump; origin of the term “returning heredity”).
Heredity of influence	Influence of place and climate.
Homochronic heredity	Correspondence of age of manifestation.

that are no longer considered to relate to genetic transmission: goiter, impulses, passions, propensity for crime, and so forth. In this way, heredity was akin to sociological explanations: “The study and the most infallible expression of what is organic, morbid, and fatal in human nature” (Lucas 1833). This opened the way to medical–legal and anthropological studies of criminality, at the end of the century, by César Lombroso in Italy and Alphonse Bertillon (1853–1914) in France.

Lucas applied these concepts of innateness, heredity of similarity, and heredity of metamorphosis to many pathologies, including convulsions, apoplexy, dementia, chorea, mental illness, hysteria, and hypochondria. And also Charcot referred to Lucas’ work multiple times in his publications. In contrast, Louis Pasteur (1822–1895) and his students destroyed Lucas’s demonstrations of hereditary transmission for diseases such as tetanus, syphilis, meningitis, and tuberculosis. In any case, Lucas’s treatise had considerable influence on alienists and on the work of Morel, in particular.

### **Bénédict-Augustin Morel and his *Traité des dégénérescence physiques, intellectuelles et morales***

Bénédict-Augustin Morel was born on November 22, 1809, in Vienna during the Napoleonic Wars (Figure 4). His father was an army supplier and followed the troops; no information about his mother is available. In 1814, he was sent to a boarding school in Luxembourg. As an adolescent, he was expelled from the seminary, where his tutor had arranged his enrollment, after students rioted in favor of liberal ideas. Morel then moved to Paris and started medical studies while working as a tutor in a rich family. He shared the hardships and poverty students faced at the time with Claude Bernard, who had come to the capital from Lyon. They became close and lasting friends. He defended his thesis on August 6, 1839, before a jury presided over by Jean-Nicolas Marjolin (1780–1850): *Questions sur les diverses branches des sciences médicales* (Questions on the various branches of the medical sciences; Morel 1839). The second question dealt with delirium. He doubted that it was localized in the cortical area of the hemisphere, as his anatomical observations had revealed no lesions. He distinguished between acute delirium, “sympathetic in origin”—that is, linked to a transient general cause, such as fever or alcohol—and the chronic delirium of the insane. Claude Bernard, then a resident at La Salpêtrière, introduced him to Jean-Pierre Falret (1794–1870), who brought him to his department for the insane and helped him enter the French Medical-Psychological Society. As encouraged by Falret, he attempted to show that alienists erred by neglecting the



**Figure 4.** Bénédict-Augustin Morel (1809–1873) (OW collection).

psychological aspect of insanity, instead giving priority to anatomopathological research. Antoine Ritti (1844–1920) reported that the student comradery with Claude Bernard also extended to Charles Lasègue (1816–1883; Ritti 1885). Lasègue took advantage of Morel's fluency in German for his historical research on the doctrines of Stahl and German psychology (Lasègue and Morel 1844), research that would lead to Lasègue's (1846) thesis, defended before a jury presided over by Armand Trousseau (1801–1867) on February 25, 1846 (Falret 1873).

After the 1848 Revolution, Morel was named chief physician of the Mareville asylum near Nancy, in northern France. An adept of nonrestraint, he did away with chains and other brutal measures as well as prison-like cells. Starting in 1851, he began clinical lectures on mental illness. The premises of his thinking on degeneration were laid with his observations of patients suffering from goiter and of cretins at the Rosières aux Salines center, near Nancy, whom he also treated. On May 23, 1856, Morel was named to the Saint-Yon asylum near Rouen, "where he remained the chief physician until his death. There he would develop and complete the studies and works he began in Nancy, and start work on his volume on degeneration in the human species, published in 1857, which was the most significant and important work of his life," according to Jules Falret (1824–1902), speaking at his funeral (Falret 1873). Weakened over several years by diabetes, Morel died on March 30, 1873, while returning from a trip to Le Havre in northwestern France, where he had spoken of Joan of Arc and her hallucinations (Constant-Trocmé 1970; Semelaigne 1930).

On June 26, 1911, the French Medical-Psychological Society awarded the philosopher Georges Genil-Perrin (1882–1964) the *Prix Semelaigne* for a dissertation that would become his doctoral thesis in medicine in 1913: *Histoire des origines de l'évolution de l'idée de dégénérescence en médecine mentale* (History of the origins of the idea of degeneration in the medicine of mental illness; Genil-Perrin 1913). The jury was presided over by Gilbert Ballet (1853–1916). At the beginning of his thesis, Genil-Perrin explained, “The idea of mental degeneration is a poorly defined notion.” For him, this idea was “based on elements borrowed from heredity and the problem of physical and moral relationships” and results from positivism, which makes “psychology into a chapter of biology.” Thereby freed of metaphysics, it became “a vast anthropological-psychiatric concept” (Jennekens 2014). However, Morel’s initial approach was entirely different. What influenced him?

Johannes Burchart, in his thesis presided over by Stahl in 1706 (Burchart 1706), developed the idea of a heredity of predisposition, which became the dissimilar heredity of Philippe Pinel (1745–1826). In his 1801 *Traité médico-philosophique sur l'aliénation mentale* (Medical-philosophical treatise on mental illness), Pinel saw this form of heredity as the origin of madness, an idea that François-Emmanuel Fodéré (1764–1835) carried forward in 1832, followed by Jean-Étienne Esquirol (1772–1840) in 1838. The primitive error of this theory was to mix exact observations with false interpretations, confusing the role played by the transmission of a trait with the influence of social milieus and upbringing in the development of behaviors considered pathological. Nonetheless, it was a theory Morel embraced.

The concept of degeneration developed by Morel was intrinsically linked to his Catholicism, to the doctrine of original sin, and to Adam’s error, which were believed to have caused humanity’s fall. Morel participated in the discussions of a cenacle that also included Laurent Cerise (1807–1869) and Philippe Buchez (1796–1865). Their goal was to promote social medicine, an extension of their shared Catholic faith (Carbonel 2010). Since the Bible and Genesis were at the root of their thinking, Morel, a monogenist and a fixist, conceived of mental pathology as “an unhealthy deviation of a primitive type” (Morel 1857, 2) in which the latter was free of any defects. Deviation occurred, however, because of “the original degradation of human nature,” or the price paid for sin. The paradox of Morel’s thinking, which aimed to be scientific but remained theistic and political, was that it presented degeneration as a biological danger, harming social organization through the hereditary accumulation of transmitted defects, which ultimately and inexorably sterilized the human race. The medicine of mental illness, however, worked “for regeneration and calls on the assistance of all those entrusted with the well-being and the fate of the population, all those who have the means to carry out the improvements that medical science submits to them” (Morel 1857, 6; see Figure 5). Regeneration was seen as the task of public health, based on conservative “moral law,” in which the precepts dictated by Catholicism took precedent—namely, “the natural order of the creator,” centered on traditional family values, “domestic virtues,” and authority. Morel’s reaction to the revolutionary events of 1848 is evident, whereas initially he did actually seek to improve the conditions of workers and the poor.

Influenced by the “zoological philosophy” of Jean-Baptiste de Monet de Lamarck (1744–1829) published in 1809, and *Histoire générale et particulière des anomalies de l'organisation chez l'homme et les animaux* (General and specific history of anomalies in the organization of men

# TRAITÉ DES DÉGÉNÉRESCENCES

PHYSIQUES, INTELLECTUELLES ET MORALES

## DE L'ESPÈCE HUMAINE

ET

DES CAUSES QUI PRODUISENT CES VARIÉTÉS MALADIVES

PAR LE DOCTEUR

**B. A. MOREL**

Médecin en chef de l'Asile des aliénés de Saint-Yon (Seine-Inférieure),  
Ancien médecin en chef de l'Asile de Maréville (Meurthe),  
Lauréat de l'Institut (Académie des sciences),  
Membre correspondant de l'Académie royale de Savoie, de l'Académie royale de  
médecine de Turin,  
Des Sociétés de médecine de Nancy, de Metz, de Gand, de Lyon, etc., etc.

AGCOMPAGNÉ D'UN ATLAS DE XII PLANCHES

A PARIS

CHEZ J. B. BAILLIÈRE

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MADRID, C. BAILLY-BAILLIÈRE, CALLE DEL PRINCIPE, 11

1857.

Figure 5. Cover of B.-A. Morel's most famous work in 1857 (OW collection).

and animals) published in 1832 by Étienne Geoffroy Saint-Hilaire (1772–1844), Morel saw “habits” as the driver of the transformations that were passed on. Unhealthy deviations resulted from the pathogenic conditions of one’s milieu and from repeated automatisms, leading to morphological as well as psychological adaptations. These adaptations were passed on from generation to generation, always toward progressive decadence. Disharmony in the body and dysfunction of the nervous system, both hereditarily and progressively transmissible, defined the inexorable fall—that is, degeneration. To illustrate his ideas, Morel added an atlas of engravings to his text, showing microcephaly and various cranial deformations (Figure 6). Alongside anatomical traits, Morel introduced a new etiological classification of mental illness, which was based on intoxication (alcohol, dietary deficiencies), social milieu (unhealthfulness, poverty, industry), “moral malignity,” acquired (pellagra, scrofula) or inherited (epilepsy) morbid conditions, and so forth.

In sum, two laws governed degeneration: “the law of double fertilisation in terms of physical malignity and moral malignity, and the law of progressivity of degeneration” (Morel 1857, 78). These became “Morel’s laws” for his champions. For example, an alcoholic’s son carries the physical defect of the addicted parent and the moral defect of his dissolute life, making him an irremediable degenerate, capable only of producing even more degenerate offspring. Using the term “heredity of transformation,” Morel generalized the inevitability of disease transmission. In this way, the first generation, in which “a nervous temperament” predominates, saw various neuroses (e.g., epilepsy and hysteria) and cerebral hemorrhages multiply in the next generation. Morel insisted repeatedly on a factor in addition to those mentioned above: the influence of places and climates in the emergence of degeneration. Swamps, cold climates, and humidity were presented as the most harmful, causing fevers and transmissible defects.

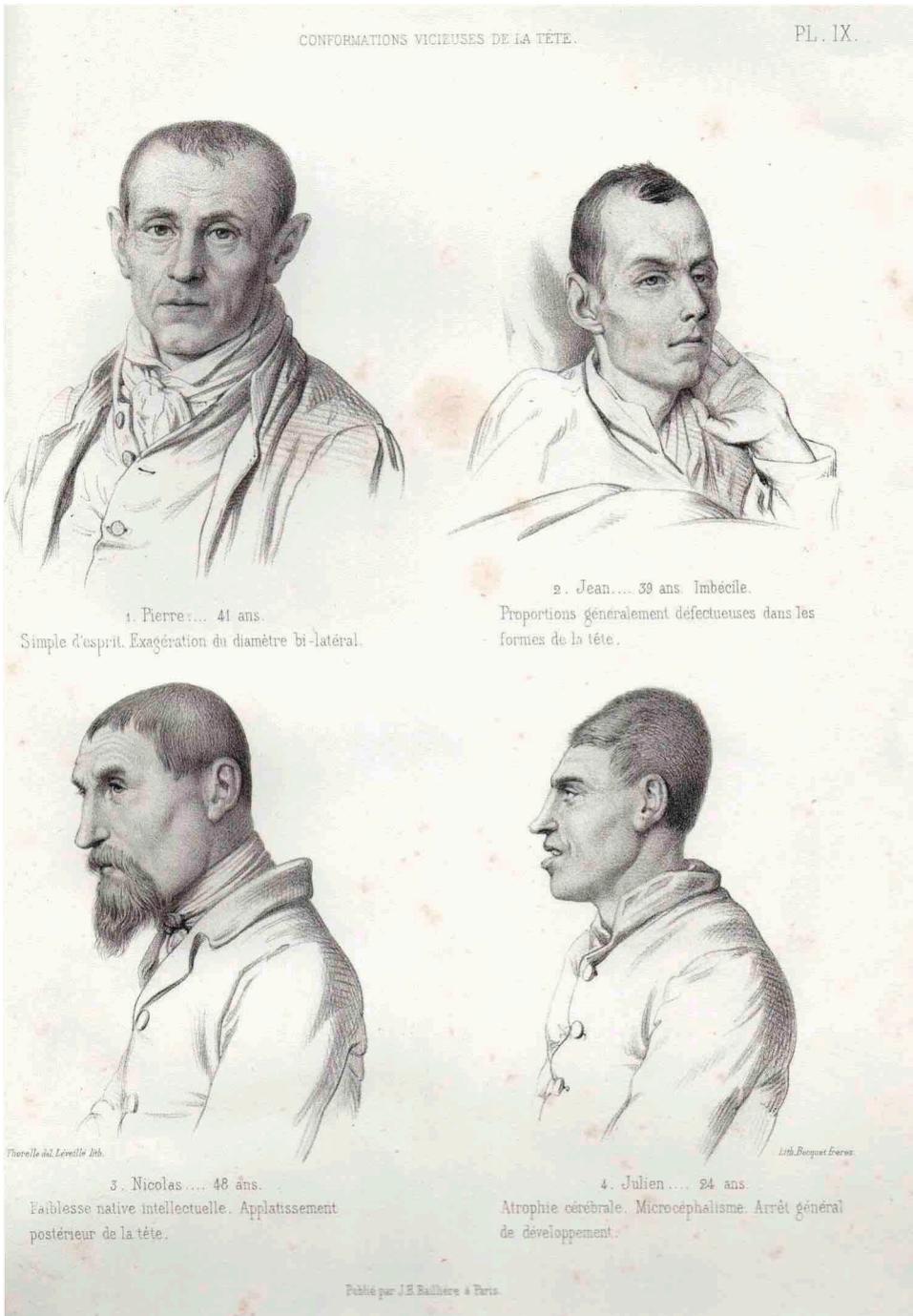
Finally, Morel also included predispositions specific to race, the hierarchy of which was unquestionable for him. This position was ambivalent in that he recommended “racial crossbreeding” to regenerate humanity, influenced as he was by animal rearing and horticulture. Is it enough to “proclaim the need for racial crossbreeding so that humanity’s interests are protected in the future? These interests would, on the contrary, be singularly compromised if moral culture failed to fertilize the vigorous products resulting from crossbreeding and marriage between different social classes” (Morel 1857, 75).

In 1895, Valentin Magnan (1835–1916) and Paul-Maurice Legrain (1860–1939) summed up the concept of degeneration, at a time when nearly all alienists subscribed to it:

Degeneration is the pathological state of the individual that, compared to his most immediate progenitors, is constitutionally less resistant psychophysically and can only partially achieve the biological conditions of the hereditary fight for life. This weakened resistance results in permanent stigmata that are essentially progressive, without intercurrent regeneration, lacking which the species is more or less rapidly annihilated. (Magnan and Legrain 1895, 79)

This concept included social, biological, and psychological data in a single category, providing a facile explanation, lacking any real scientific basis, as to the complex question of the origin of mental disorders. To this day, the etiological question has not been fully resolved.

In particular, Charcot focused on hereditary predisposition as well as dissimilar heredity. For example, a young woman who suffered from hysteria in adolescence and from delirious ideas and mania in adulthood, eventually dying in a state of dementia. A morbid predisposition led to the successive exteriorization of “pathologies of the same kind, manifesting the same



**Figure 6.** Plate IX of B.-A. Morel's atlas: Degeneration of the shape of the skull (OW collection).

pathological aptitude of the degenerate race by various disturbances,” according to Morel. His doctrine, as Charcot understood it, took the form of a vast etiological concept situated at the foundation of all psychiatric and neurological diseases of the nervous system.

## Influence of Jacques Joseph Moreau de Tours

After Alfred Velpeau (1795–1867) and Armand Trousseau (1801–1867), Jacques Joseph Moreau de Tours (1804–1884; [Figure 7](#))—who was the third disciple of Pierre Bretonneau (1778–1862)—a famous physician from the western French city of Tours. A resident under Jean-Étienne Esquirol (1772–1840) for four years at the Charenton asylum, he adopted a scientific approach in his thesis (Moreau 1830) and pursued it throughout his career. He saw the brain, the organ of thought, as being subject to functional disorders, whether they were lesional or not, which led him to consider madness as an organic disease. Heredity, he believed, was the etiology: “Via the path of heredity, and by this path alone, we can work our way back to the psycho-organic source of these phenomena, penetrating and reading, so to speak, the intimate texture of the organ governing their external manifestation” (Moreau de Tours 1859). He disagreed with those for whom heredity only be applied to intellectual phenomena: “Heredity expressly means the seminal transmission, not of moral qualities of one individual to another, but rather nervous or vital forces.” The organization of sensory organs, indispensable for psychic functioning, was transmitted via heredity, as was their own functioning: “Almost equal to sensations, affective passions depend on the organisation; they share its vicissitudes. Like it, they must be transmitted by heredity” (Moreau de Tours 1859, 13). A hereditary predisposition existed; life events would trigger mental pathology. The influence of the environment became crucial as to whether or not insanity emerged. He admitted he could not establish the intimate nature whereby mental states were transmitted, but he held that symptoms may differ between different individuals of the same family: One had tics, the other stuttered, and the third was eccentric and eventually went insane. This notion of predisposition and its corollaries were adopted by Charcot, notably to explain the genesis of hysteria. Climate, morals, nutrition, and many other circumstances might all be causes of deviation from the standard of normality.

## Validation by Jules Dejerine

In his 1886 agrégation thesis, entitled *Hérédité dans les maladies du système nerveux*, Jules Dejerine (1849–1917) stated:



**Figure 7.** Jacques-Joseph Moreau de Tours (1804–1884) (© BIU santé, Paris).

Morel, Lucas, and Moreau de Tours, physicians from our country, were the initiators of this approach. Especially Morel, who formulated and resolved the question for mental illnesses, in several admirable works. He showed how these conditions were interlinked and also linked to the major neuroses by the laws of heredity and degeneration, and inaugurated a fecund research method, carried forward by all contemporary alienists. ... What Morel did for mental illnesses is what clinical medicine is currently doing for the other nervous system diseases. This is the type of research that Mr Charcot has repeatedly stressed in his lessons for several years. (28)

## **Heredity and the concept of degeneration in Charcot's work**

### ***Before 1882: Expression of the histopathological description***

Whereas Charcot merely tried to find correlations between his clinical observations and his anatomical findings at the beginning of his career, the creation of the Chair of the Clinic of Nervous System Diseases in 1882 can be seen as the moment when, in order to teach his students about the diseases he helped identify, he had to research and conceive of an etiology for each one. At the beginning of his career, mostly focused on geriatrics, the predominant explanations were climatic and environmental factors, such as cold temperatures, humidity, and diet. His thesis on chronic rheumatism (polyarthritis, degenerative arthritis) and gout was an example of this.

In the first description of Parkinson's disease in 1861 by Charcot and Alfred Vulpian (1826–1887), degeneration was not mentioned. Rather, emotions were presented as playing a preponderant triggering role, in addition to “the combination of cold temperatures and humidity” (Charcot and Vulpian 1861).

In their *Clinical Lectures on the Diseases of Old Age* (originally published in 1874), the term “degeneration” was only used to qualify the excess fat on cardiac muscle observed in certain gout patients (Charcot and Ball 1874). Similarly, in the first release of *Clinical Lectures on the Diseases of the Nervous System* (originally published in 1872), the word “degeneration” did not appear in the body of the text, and only three times in the footnotes, to describe the changes in appearance of anatomopathological cross-sections of muscular fibers (Charcot and Bourneville 1872).

Charcot's initial uses of the term “degeneration” are only a description of visually observable transformations of tissues examined under the microscope, as caused by pathology. This word did not refer to the etiology of the pathological process. His usage was thus aligned with the definition of the lexicographer Émile Littré (1801–1881) in 1873: “Damage that leads to the transformation of an organ's tissue into an essentially morbid material” (Littré and Robin 1873).

The descriptions of anatomopathological lesions observed by Charcot and Vulpian in cases of locomotor ataxia in 1862 (Charcot and Vulpian 1862) did not employ the word “degeneration” at all. However, in the first description of multiple sclerosis, it is used to distinguish lesions viewed under the microscope:

Sclerosis of the spinal cord, in the strictest sense, must be carefully distinguished from the granular degeneration followed by atrophy that frequently occurs in the same organ due to certain conditions of the brain or the spinal cord itself. This degeneration is sometimes found, over significant lengths, in the anterolateral tracts or the posterior tracts, either on one side, or symmetrically, on both sides at once.” (Charcot 1865, 472)

### ***After 1882, heredity became a predominant etiological entity in neurology***

For Goetz, Bonduelle, and Gelfand the turning point came after the 1881 World Congress of Medicine in London. Supporting his findings with statistics, Wilhelm Erb (1840–1921) demonstrated that more than 90% of cases of locomotor ataxia (tabes dorsalis) were linked to syphilis, as also argued by Alfred Fournier (1832–1914; see Goetz, Bonduelle, and Gelfand 1995) in France. During this congress, Pasteur’s theory on the microbial origin of infections was widely hailed. However, a number of physicians did not accept this theory. Not the least of them was Charcot, who clearly stated his objections during his report on the *Prix Civrieux* awarded by the French Academy of Medicine in 1883 to his students Louis Landouzy (1845–1917) and Ballet, who had examined “research into the causes of locomotor ataxia.” Charcot saw this work as confirming his personal conviction:

For them, the true, essential, and determinant cause of tabes, the cause that dominates, commands, and absorbs all others, and the cause in the absence of which, perhaps, the others are powerless, is hereditary nervous predisposition. ... Tabes is one of the clinical aspects that, when examined in depth, reveals the nervous defect that the patients inherited from their progenitors. ... This view is obviously not new, but it is necessary to highlight it once again, to establish the absolute and rigorous reality based a sufficient number of observations, studied with a high level of clinical expertise and patiently examined from the etiological perspective. (Charcot 1883, 1451)

Here is how Henry Lamy (1864–1909) explained Charcot’s reasoning:

In light of impressive statistics, there is now a tendency to view certain cerebrospinal diseases as being produced by syphilis, whereas the related anatomical lesions do not at any time present the characteristics generally associated with syphilitic lesions. Whatever the merits of this way of thinking, it is not aligned with the scientific method that has guided medicine in our century to its greatest discoveries. It goes without saying that what we know of the damage caused by syphilis in the organs remains quite rudimentary; it may be that, like certain poisons, it acts electively in the nervous centers, on a given differentiated system, initially affecting only the essential element; but this remains only a curious hypothesis, and not a lodestar in the study of how syphilis affects the nervous system. Until the results of bacteriological research have achieved the accuracy that is required today for the study of infectious diseases, only pathological anatomy and clinical medicine can determine, insofar as possible, the relation between syphilis and nervous diseases.” (Lamy 1893, 5)

Charcot would remain convinced until his death that syphilis was only one revealing agent among others, acting through hereditary predispositions. For example, in his Tuesday Lesson on January 10, 1888, he noted: “In all ataxics, I believe there to be something hereditary, but this is especially true in early ataxics.”

On Tuesday, March 13, 1888, Charcot generalized heredity as etiology to all diseases of the nervous system:

On one hand, Friedreich’s ataxia is hardly common ataxia and on the other, there is more than ever good reason to believe that common ataxia is a hereditary disease, in the same way that other members of the neuropathological family are. In addition, Friedreich’s ataxia is not, in the strictest sense of the word, a hereditary disease so much as a family disease, a generational disease, which is not quite the same thing. ... As I mentioned earlier, the etiological characteristics of Friedreich’s ataxia are remarkable, leading some authors to call it hereditary ataxia. But, as I have just told you, real ataxia is also a hereditary disease. This bears repeating, even though I have already mentioned it to you several times. In such cases,

it is not, of course, a matter of homologous heredity, which is very rare, but rather the heredity of transformation, which, as you know, is the rule. (258)

He was speaking of Morel's heredity of transformation, Pinel's dissimilar heredity, and Lucas's heredity of metamorphosis (see [Table 1](#)).

Bearing witness to the "extemporaneous" nature of his consultations, his Tuesday Lessons, all of which were transcribed for 1887–1889, reveal that Charcot focused in particular on his patients' family histories. These were significant in his view, and he believed they frequently provided him with the origin of the disease in question: "The clinician only has an episode to work with, if he wants to limit himself to studying the patient himself, and not take the entire family history into account" (Charcot 1887). Here are a few examples:

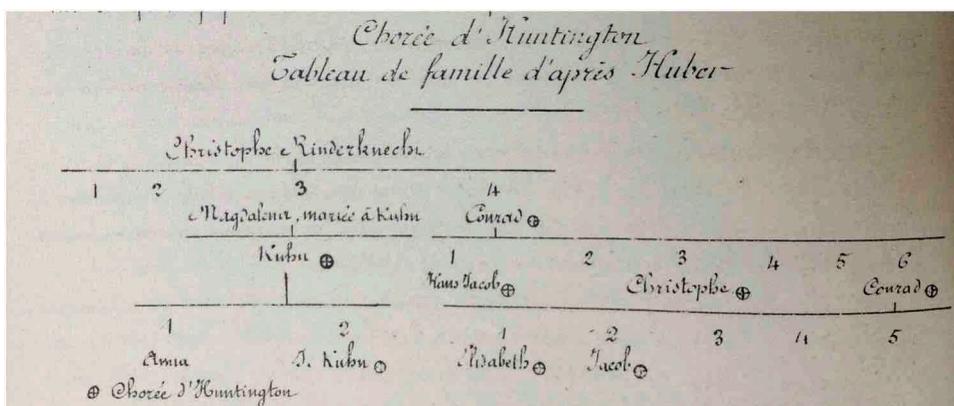
On Tuesday, 17 July 1888, Charcot lectured on chronic Huntington's disease:

Thus Huntington's chorea is like many other nervous system and muscular conditions newly introduced in the nosography, for example: Thomsen disease, Friedreich's ataxia, and pseudo-hypertrophic paralysis described for the first time by Duchenne de Boulogne [see [Figure 8](#)]. All these conditions, as you know, are both similar hereditary diseases and family diseases, while manifesting the heredity of transformation in various ways.

These diseases were thus accurately identified as hereditary, but confusion still surrounded their transmission ([Figure 9](#)):

Similar heredity appears to be one of the major etiological characteristics of the disease, which develops inevitably and is perpetuated from generation to generation, most often in the absence of an identifiable occasional cause. Without apparent reason, it one day emerges and develops, at exactly the most opportune moment, nearly always at around the same age. (Charcot 1887)

Charcot established a parallel with Sydenham's chorea, for which he frequently observed "hereditary of transformation." The existence of a case of epilepsy or hysteria in the family confirmed his idea of heredity of transformation: "It is truly a basic observation in the choreic patient, and you know this is an aetiological fact I often have the occasion to point out in the patients who come for the Tuesday consultation." Charcot's refusal to accept the syphilitic origin of tabes was analogous to his view that chorea and acute rheumatoid



**Figure 8.** Tree drawn by Charcot during his lesson on Tuesday, July 17, 1888 (OW collection).

COTÉ PATERNEL		COTÉ MATERNEL
SŒUR DU PÈRE <i>Scrofule</i>	PÈRE <i>Épileptique</i>	GRAND'MÈRE Plusieurs accès de <i>mélancolie</i> .
COUSIN GERMAIN <i>Épileptique, aliéné</i>	1 <sup>er</sup> FILS <i>Métallophobie</i> Agé de 10 à 12 ans ne pouvait toucher un objet de cuivre, surtout s'il était sale, sans éprouver une sensation de dégoût se traduisant par des crachottements.	MÈRE <i>Tuberculeuse</i>
		2 <sup>e</sup> FILS <i>Kleptomane</i> a. Vers l'âge de 10 à 12 ans, ramassait tous les objets métalliques qu'il pouvait s'approprier et les accumulait dans une cachette. b. Vers l'âge de 60 ans; un accès de <i>mélancolie</i> à l'occasion d'une maladie aiguë.
		II Sa femme issue d'une famille goutteuse est goutteuse elle-même (goutte régulière occupant les gros orteils). Un fils frappé à l'âge de 4 ans d'une <i>paralysie infantile spinale typique</i> .

Figure 9. Page 145 of Volume 2 of the consultations du Mardi, 1889 (OW collection).

arthritis of the joints occurred together simply due to a frequent coincidence, “a certain affinity,” but “not at all because they [were] identical and of the same nature.”

Charcot cited shaking as another example of similar heredity:

This trembling may present, in certain subjects, sporadically; that is, without precedents or concomitants in ascendants or collaterals; but in other instances, it is indeed similar to Huntington's cases; that is, a family disease, strictly speaking. Most often, shaking occurs in childhood and is passed down by similar hereditary. (Charcot 1887, 544; see Table 1)

On Tuesday, February 21, 1888, Charcot questioned a 21-year-old man suffering from tics: “What is more, you know that having tics in a family is not without significance. Tics are a special mark.” What followed was a thorough interrogation on personal and family antecedents. The patient, it turned out, also had a folie du doute characterized by the fear of rabies at the sight of an animal. He had two brothers and a sister with tics: “When there is one ticcer in a family, it is rarely an isolated case.” In one 1886 lesson that was not published until 1893, Charcot discussed laryngeal noises, presented as hysterical and distinct from Gilles de la Tourette disease:

Mr. Gilles de la Tourette ingeniously grouped together sudden involuntary, automatic utterances that are often vulgar or obscene, but are said aloud and intelligibly, under the name of coprolalia. This often affects well-educated people with proper upbringing. ... Take, for example, the case reported by Professor Pitres, of a young lady from Bordeaux, aged

fifteen, with an insane aunt and a ticcer father. She herself was a ticcer who, during her fits, uttered the most filthy language. (Charcot 1893)

In response to a case of peripheral facial palsy, Charcot noted, “we now know this is not a condition where cold temperatures are the cause, but rather a true nervous disease.” For him, the families of these patients revealed cases of “nervous phenomena of another kind, insanity, locomotor ataxia, and so forth.” That is, they revealed heredity of transformation. Charcot made a side note to the effect that Jews would be particularly prone to familial facial palsies.

Once again, Charcot’s thinking was the same for Graves’ disease; he stated it “is often a family disease, passed on by heredity and involving insanity, epilepsy, and so forth. It is in the same category and it also has common links with the arthritic family for the entire series.” In addition: “Alcoholics are not always what they are superficially assumed to be. Alcoholism is sometimes a kind of constitutional vice, a hereditary defect.”

On Tuesday, December 6, 1887, Charcot explained how he saw heredity of transformation: “Nervous diseases are almost never passed on in the same form. You must thus not reason that locomotor ataxia engenders locomotor ataxia, and general paralysis general paralysis. This is not at all true. Heredity proceeds by transformations. A paralytic engenders a hysteric and a hysteric a paralytic.” On Tuesday, July 10, 1888, he summarized the laws of heredity this way:

Take what I proposed to call the neuropathic family. I use this term to distinguish all conditions of the central nervous system and neuromuscular system that are organic or, on the contrary, without any appreciable anatomic lesions, and that are linked by heredity. You are well aware that here we have to distinguish, together with homologous heredity, dissimilar heredity or heredity of transformation, which may be seen much more frequently than the former. (Charcot 1893, 455)

A genetic tree created for the examination of “typical spinal infantile paralysis,” undoubtedly a case of poliomyelitis, showed the range of antecedents, which, to a current-day observer, seem in no way hereditary: epilepsy, phobia, melancholy, tuberculosis, gout (Charcot 1889). A variety of pathologies were thus tied together, leading to a vague concept of dissimilar heredity and thus to a scientific impasse, but one that opened the way to ideological deviations (Pinell 2001).

After 1882, Charcot no longer wrote any articles himself, except in English. He left the task of publishing research results from La Salpêtrière to his students. It is thus interesting to analyze the work of two of his most faithful, prolific, and zealous champions, Charles Féré (1852–1907) and Georges Gilles de la Tourette (1857–1904), both of whom demonstrated unflinching and fervent veneration of Charcot.

### **Charles Féré and the neuropathic family**

Charles Féré was Charcot’s resident in 1881 and defended his thesis before a jury presided over by Charcot on March 18, 1882. The theme of heredity can be found throughout his work and he constantly focused on fighting all forms of degeneration. Féré, like Charcot, did not research the origins of heredity but limited himself to observing the transmission of nervous diseases down through the generations, whatever the biological processes; that is, he was satisfied with the genealogy of the clinical history alone (Gelfand 1987). He championed eugenics, the only way he believed humanity could be saved (Courtin 2007). After an article in *Les Archives de Neurologie* (Féré 1884), his emblematic book was

published in 1884, then reissued and completed in 1894: *La famille névropathique, théorie tératologique de l'hérédité, de la prédisposition morbide et de la dégénérescence* (The neuropathic family, a teratological theory of heredity, morbid predisposition, and degeneration; Féré 1894). Féré coined the term “*famille névropathique*” with which Charcot agreed and used from then on. For Féré, because Lucas, Morel, and Moreau de Tours were only interested in mental disturbances, they “only considered other nervous diseases secondarily and in general.” Taking an approach opposite to Morel’s, Féré made no metaphysical references and adhered to Darwin’s theory of a fight for life that led to selection. He defended the transmissibility of acquired traits, citing hysteria as an example: “Concerning hereditary influence, there is no nervous condition in which it is more clearly manifest than in hysteria.” For Féré, hysteria arose from direct heredity and dissimilar heredity. All pathologies of the nervous system may be associated with hysteria, the “sufficient predisposition,” and the stigmata identified by Charcot externalized this predisposition.

From mental diseases, Féré extended the study of heredity and degeneration to neurological and neuromuscular diseases: for example, tics, functional spasms (neuropathic torticollis, writer’s cramp, i.e., dystonia), shaking, migraine, epilepsy and neurasthenia, and Friedreich’s ataxia. He adopted a view of locomotor ataxia that was identical to Charcot’s. As for amyotrophic lateral sclerosis and multiple sclerosis, he doubted they were hereditary. The final chapters of the 1894 version of his book covered “the teratological stigmata of degeneration.” The “objective signs of degeneration” were as significant as heredity, and the moral and physical causes had the same implication. He also explained: “The traits that are the stigmata of degeneration have characteristics that do not belong to the race and that, passed down in the family in which they have appeared, tend to function in the same way as an accident and remove the family from the race” (317). As examples, he cited variations in skull size and shape, various unusual hair implantations, ear lobe deformations, dental implantation disorders, pectus excavatum, lumbar pilosity, and so forth. It may be relevant here that after the defeat in the 1870 Franco-Prussian War, people in France were haunted by the fear that the decadence of the “French race” would make it impossible to take revenge. Degeneration and decadence were part of the same ideology. Féré did not refer to any of the work by his contemporary botanists, naturalists, or biologists, even though they could have pruned back his theory of disease transmission. During his Tuesday Lessons, Charcot cited Féré’s book multiple times, always with effusive praise, until it was no longer clear who was the teacher and who was the student.

Féré and Charcot shared the same thinking on heredity—that is, a familial predisposition to disease. In this situation, a family carried a susceptibility to neurological disorders that might or might not develop, depending on the environment. If the family member led a quiet and healthy life, he or she could potentially avoid neurological manifestations even when carrying the propensity for disability. On the other hand, the same predisposition would unleash severe disease if the patient’s lifestyle or environment was unhealthy. This topic of “agents provocateurs” (triggering factors) was discussed by Charcot in his November 15, 1887, Tuesday Lesson, during which he stressed the role of trauma (Charcot 1887).

Dejerine wrote in 1886, “Féré deserves credit for being the first to study the question with an overarching approach. His *Famille névropathique* is the most important work published in this field since Morel” (Dejerine 1886).

In 1888, Féré published *Dégénérescence et criminalité, essai physiologique* (Féré 1888) as a follow-up, and in line with the recent books of César Lombroso. The chapters on criminal heredity and anatomical and physiological traits in criminals returned over and over to all the clichés of the time, conditioning both thought and society in ways that would engender the worst aberrations of the twentieth century.

It must be noted that immediately after his stay at La Salpêtrière (1885–1886), Sigmund Freud (1856–1939) adopted the doctrine of the neuropathic family but, when he developed his own theory of hysteria’s origins, he questioned its validity:

It will undoubtedly soon be necessary to review and correct the etiological theories defended by Charcot in his doctrine of the neuropathic family and on which he founded his global understanding of nervous diseases. Charcot so overestimated the etiological role of heredity that no place remained for other neuropathic etiologies. (Freud 1893, 162)

### Georges Gilles de la Tourette and hysteria

For Georges Gilles de la Tourette, his *Traité clinique et thérapeutique de l’hystérie d’après l’enseignement de La Salpêtrière* (Clinical and therapeutic treatise on hysteria based on the teachings at La Salpêtrière), published in three volumes from 1891 to 1895, was his most important work. Although it did not garner lasting fame, it faithfully reflected all Charcot’s lessons and thinking on this pathology: “Mr. Charcot taught that the primordial cause of hysteria was heredity, whether it was similar: hysterical mother, hysterical daughter; or whether it entailed transformation: the progenitor(s) or their descendants suffered from a nervous condition other than hysteria itself. Beside heredity, there are only agents that trigger neurosis” (Gilles de la Tourette 1891–1895, 68).

“The continuity of germinative plasma,” according to August Weismann (1834–1914), explained the inexorability of the transmission of acquired defects and disorders, the ultimate result of which could be none other than degeneration. To list the triggering agents of hysteria, Gilles de la Tourette used elements from the thesis of Georges Guinon (1859–1932), his successor as Charcot’s resident (Guinon 1889). First among these agents were “violent moral emotions,” whose role in the development of hysteria “had always been acknowledged, and their importance became even greater when the mental state of hysterics, characterised by suggestibility, was recognised.” Gilles de la Tourette went on to list the following influences, with relevant observations and discussion: trauma; acute and chronic infectious diseases (tuberculosis); diseases of the genitalia; lead poisoning or addiction to alcohol, tobacco, or morphine; and all nervous diseases (tabes, epilepsy, tic disorder, myopathy).

Gilles de la Tourette reminded his readers that Hippocrates already saw hysteria as a hereditary pathology. To confirm this, he returned to the statistics of Pierre Briquet (1796–1881; Briquet 1859) and those of a Swiss student of Charcot, Émile Batault (1859–1929), whom he himself oversaw during his time as senior resident (Batault 1885). This explains the “law of preponderance in the transmission of traits” whereby “it is not always the healthy individual who predominates, on the contrary; in the large majority of morbid cases, transmission occurs in childhood, even though only one of his two progenitors is sick” (Gilles de la Tourette 1891–1895, 43).

Paul Peugniez (1859–1943), in his thesis before a jury presided over by Charcot on July 24, 1885, stressed the following: “First and foremost, it is the disastrous law of nervous heredity that causes the development of hysteria in youngsters.” He did not fail to underscore such decisive causes as “abuse, frightening experiences, moral emotions, and ceremonies.” Many of these children suffered from what is currently known as attention deficit hyperactivity disorder. Others had authentic epilepsy, or migraine, whereas still others had nascent psychosis.

On November 28, 1888, Grégoire Breitman (1859–1914), a physician from the city of Anyanyiv in Moldavia, defended the first thesis on Gilles de la Tourette syndrome since the seminal article’s publication in 1885, with Charcot presiding over his jury: “Alienists such as Magnan and his students, Legrain and Saury, see in echolalia, coprolalia, and echokinesis an episodic syndrome of *la folie des dégénérés*” (Breitman 1888). Paul-Maurice Legrain (1860–1939) and Honoré Saury (1854–1906), disciples of Valentin Magnan (1835–1916), introduced the concept of *bouffée délirante* (brief psychotic disorder) into the psychiatric nosology; this was after both had done work on *la folie des dégénérés* (insanity of degenerates), with Legrain writing his thesis on the subject and Saury a book.

Breitman, a student alienist at the Vaucluse asylum near Paris, wanted to study the heredity of involuntary imitation symptoms in order to clarify the role of “degeneration” in their genesis, given that:

As the semiology of hereditary degenerates grows more detailed, exact, and manifest, we are relegating the search for the original flaw in the various forms of alienation to a secondary position,” because “the aim is to find out whether, by studying the semiology of each psychic state and by grouping the signs into syndromes, we can, by deduction, demonstrate the manifestly degenerative hereditary origin. (7)

He explained that “under the combined influence of peripheral and central excitations, we will see degenerates, these future candidates for mental alienation, executing a series of bizarre actions, designated as ‘Latah, Jumping, and Myriachit’ by foreign authors.” Breitman showed, based on several observations he had collected, that echolalia, coprolalia, and echokinesis could be seen in various types of mental alienation, especially in deliriums, and concluded that “echolalia, coprolalia, and echokinesis accompanied by tics can make up a perfectly independent morbid syndrome with a determined progression, the tic disease or Gilles de la Tourette disease. Patients with this disease are degenerate” (Walusinski 2019).

## From yesterday to today

With his usual prudence, Charcot first had his students publish his ideas on the hereditary etiology of nervous system diseases before turning them into a doctrine he himself professed, as one can read in the Tuesday Lessons from 1887 onward, when he began to treat patients from the external consultations department. He used genealogical trees to support his demonstration. The belief in transmission of acquired traits was widespread at the time and provided a rather simple response to difficult questions. Degeneration, elevated to the rank of hereditary defect, not only applied to nervous system damage but played a role in explaining individual behaviors, and beyond that social behaviors, as in the books of Emile Zola.

One of the remarkable elements of Charcot's hereditary thinking was the concept of heterogeneous expressions of disease: Various members of the same family could have diseases of the nervous system but different diagnoses—epilepsy, melancholia, infantile spinal paralysis, and so on. At first glance, this type of analysis might be dismissed by modern neurologists as naive, but conceptually, such approaches involving multiple phenotypes with one genotype are highly pertinent today. In 2013, Lesage and colleagues identified disease-causing repeat C9orf72 gene expansions in a subset of patients from a large cohort with different forms of Parkinsonism, extending the clinical phenotype associated with these expansions from degenerative dementias—including Alzheimer's disease and amyotrophic lateral sclerosis—to Parkinsonism (Lesage et al. 2013). Charcot summarized his thought in a limpid manner by this quote: “*Le malade n'est qu'un épisode, l'ennemi c'est la famille*” (the patient is only one episode; the enemy is the family), which provides the key to Charcot's view of all neurological diseases. It also links all areas of pathogenesis—hereditary, toxic, and infectious—allowing nonhereditary triggering factors to play a role in disease manifestation while remaining secondary issues relative to hereditary factors (Charcot 1887).

The swing of the pendulum in scientific discoveries had set genetics in the foreground of etiological research on nervous system diseases but did not validate the vague concepts used by Charcot. Like a nod to the past described above, the discovery of bits of the viral genome in some of our chromosomes could become a potential explanation for so-called prion diseases, illustrating the old meaning of “degenerate”: “To lose the qualities of the lineage.” The meaning of degeneration as conceived by Morel is no longer current, but the expression “neurodegenerative diseases” is frequently used today, referring to structural and metabolic modifications to cells in the nervous system. Charcot obviously could not have suspected these phenomena of disintegration, potentially programmed in the genome. A neuropathologist who studied under Pierre Marie, Ivan Bertrand (1893–1965), entitled one of his works *Les processus de désintégration nerveuse* (Processes of nervous disintegration) in 1923; he probably better expressed the processes under way in these pathologies than if he had employed the word “degeneration,” too often used with different meanings (Jenekens 2014).

As Charcot possessed none of Pasteur's works in his library and made no explicit allusion to microbial theory, he did not realize Pasteur's importance, unlike some of his students, such as Victor Cornil (1837–1908) and Pierre Marie (1853–1940), or his alter ego, Alfred Vulpian (1826–1887).

Why did Charcot so dogmatically believe in hereditary causes, when he was so demanding of experimentally documented data in the rest of his anatomoclinical work? Without posing any real questions, he embraced hereditary etiological explanations where elsewhere he argued against blind acceptance of prior theories or biases, always charting his own path. Several hypotheses are possible: heredity as a seemingly scientific and simple explanation, one that all physicians can test on a daily basis by observing their patients and their patients' families. But like all dogmas, this type of explanation rests on a belief whose apparent validity is based solely on its longevity, dating back to antiquity (Horrobin 1976); the underestimation of Pasteur's discoveries (Gaudillière and Löwy 2014); the adherence to Darwinian concepts, perhaps with the prescience of the future discovery of their biological basis; and the influence of the intellectual milieu in Paris at that time, which focused attention on a young science, anthropology, still marked by racism and the fear of losing the supposed superiority of whites over blacks, and of French people over Germans, given the “vengeful spirit” at the end of the century (Poirier 1983).

Charcot often overlooked the socioeconomic conditions of his patients, focusing instead on familial heredity, which was a way to avoid making outright social or political statements about the causes of disease. Through his adherence to a progressive and anticlerical ideology, Charcot opposed the powerful religious beliefs that existed in his society. Heredity depended on biological laws, yet to be discovered, but they did not need the divine to explain disease (Brais 1993; Lalouette 2002). David Horrobin (1939–2003) has summarized these perspectives on disease inheritance as follows: “The history of science has repeatedly shown that when hypotheses are proposed, it is impossible to predict which will turn out to be revolutionary and which ridiculous. The only safe approach is to let all see the light and to let all be discussed, experimented upon, vindicated or destroyed” (Horrobin 1976).

## Acknowledgments

Many thanks to Jacques Poirier, Hubert Déchy, and the reviewers for their attentive readings and suggestions, and to Anna Fitzgerald for her translation.

## Disclosure statement

No potential conflict of interest was reported by the author.

## Funding

The author received no financial support for the research, authorship, and/or publication of this article.

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