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REVIEW ARTICLE

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The nature of hereditary influences on insanity from research on asylum records in Western Europe in the mid-19th century

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Abstract

This article explores the nature of psychiatric genetics research conducted in asylums in Western Europe in the mid-19th century through an examination of four studies published 1841 to 1864 from Great Britain, France, and Germany. They all utilize asylum records to determine if patients had a hereditary predisposition (HP) to mental illness. A diverse range of topics were investigated, with most attention on whether men or women are more likely to transmit, or are more sensitive to the receipt of, an HP. When significant sex effects were seen, they consistently found women to be more likely to transmit and/or more sensitive to the receipt of an HP. Other questions explored included: (a) the relationship between an HP and recurrence rates; (b) the degree of homogeneity versus heterogeneity of transmission of specific mental illnesses in families; (c) the level of HP among different forms of mental illness; and (d) differences in the proportion of psychiatric patients with an HP as a function of their religion. While the method of assessment of familial/genetic risk was relatively crude, even at this early stage in the history of psychiatric genetics, investigators were asking thoughtful questions about the nature and clinical impact of that risk.

KEYWORDS
asylums, familial transmission, history, psychiatric genetics

> "[T]he study of madness can contribute, more than the study of any other malady, to the improvement of the general history of heredity and to the determination of the laws it follows. The consumptive, the scrofulous, the gouty are dispersed here and there, and only with a substantial amount of effort and time a singular observer would achieve to collect a sufficient number of observations. The lunatics, in contrast, are assembled by hundreds in the asylums …." (Baillarger, 1844) (as quoted by Gausemeier, 2015, p. 168)

One approach to the history of psychiatric genetics is to focus on landmark papers that produced major conceptual or empirical advances in the field. This article takes a different approach. I attempt to characterize the more typical kinds of investigations of the heredity of insanity in mid-19th century Europe. In an effort to be representative (but not exhaustive), four relatively typical studies published from 1841 to 1864 from Great Britain, France, and Germany are investigated. Two are from general asylum reports and two are from published articles focusing on various aspects of the question of heredity. All use, as their primary source of information, records on the family background of hospitalized mentally ill patients' asylums which were the predominant research method at this time (Porter, 2018).

Before proceeding with this review, some background is in order. The 18th and early 19th century in Europe saw a large increase of interest in human heredity particularly from a medical perspective (López-Beltrán, 1992; Ribot, 1875; Rushton, 2009). Of the many disorders that were examined for heritable influences by 19th-century physicians, insanity was one of the most prominent (López Beltrán, 2007; Porter, 2018). Indeed, the leading figures in mid-19th century human

heredity was a French Alienist and author of the then definitive treatise (Lucas, 1847, 1850), later cited extensively by Charles Darwin (Nogu era-Solano & Ruiz-Gutiérrez, 2009).

The concept of heredity in the 19th century was considerably broader than now conceptualized. Inheritance of acquired characteristics was accepted by the large majority of workers in the field, until formally disproven by Weissmann late in the century (Robinson, 1979). Intrauterine effects were far broader than now considered, with psychological traumas to the mother considered as potential sources for a wide variety of physical and mental abnormalities in the child. López-Beltrán succinctly describes this theory:

> 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 idiosyncrasies, 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. (López-Beltrán, 1992, p. 159)

In particular, the understanding of the differential contributions of mothers and fathers to offspring was far less clearly understood than it is today. For example, preformationism was one of several popular embryologic theories from the 17th century onward which postulated that individuals arose from miniature versions of themselves contained either in the egg (Ovism) or the sperm (Spermism) with the other parental influences reflecting only a nonspecific supportive functions (Maienschein, 2017).

These theories, along with "imaginationism," had distinct predictions for the degree to which mothers versus fathers could transmit the risk of insanity to their offspring.

Returning to the four studies we here review, each of them used a common methodology. On the basis of available case records, patients were assigned as having or not having a hereditary predisposition (HP) to mental illness. While not always explicitly stated, typically the criteria for assigned an HP was having at least one relative with some form of mental illness or a related condition. But authors differed on the classes of relatives they considered relevant for the assignment of an HP. Our focus in this review is on the kinds of questions being asked and the answers obtained. Where possible, data are reanalyzed using 2×2 chi-square tests of independence or goodness-of-fit ($df = 1$), methods which were not available at the time of these publications. I present the four reports in historical order. Table 1 summarizes the analyses presented by each author and the outcome of those analyses.

1 | THURMAN (1841)

John Thurman, previously practicing as a local surgeon, was appointed in 1838 as the resident medical officer to the York Asylum, first established in 1792 (Dickinson, 1990). Three years later, he published the 108 page monograph "The Statistics of the Retreat; consisting of A Report and Tables exhibiting the Experience of that Institution of the Insane from its Establishment in 1796 to 1840" (Thurman, 1841). The main text had 51 tables with an additional 6 in the appendix. The Retreat, one of the most famous hospitals in the history of Psychiatry, was begun by the tea-merchant William Tuke to treat the mentally disturbed members of the English Quaker community. This hospital played a key role in popularizing the concept of moral therapy and was often visited by guests who wrote admiringly of the treatment given.

Of the vast amount of data summarized by Thurman in this volume, only a small part deals with heredity with nearly all the information contained in only two tables (11 and 13). Thurman examines a total of 469 patients admitted at least once to the Retreat from 1796 to 1840. Table 11 is entitled "Shewing the numbers of persons in whom a hereditary tendency to insanity was known to exist." Of this total, 153 or 32.6% are known to have what he terms an HP.

Thurman defines what he means by this HP:

> In the above estimate, cases are not considered of an hereditary character, the history of which had only been characterized by the existence of insanity in collateral blood relatives; it being obvious that cases of this description do not necessarily establish any direct hereditary transmission. (Thurman, 1841, p. 19)

He goes on to note that "There were 71 known cases of this kind [of collateral positive family history], and had they been included, the proportion [of patients with a predisposition] would have been raised to about 51 per cent…" (Thurman, 1841, p. 19). Note that the above quote implies that only "insanity" in relatives is qualified for the assignment of an HP.

Thurman does not give a systematic description of how the information on HP was obtained. However, in describing the results, he includes one very informative sentence:

> … many cases not stated to have been hereditary by the relatives and friends have, in this estimate, been considered as such, in consequence of more private information, or of the fact being well known. (Thurman, 1841, p. 19)

This suggests that in addition to reports on insanity in their relatives from the patients and their friends, those keeping the records at the Retreat had access to other information about their family history.

Although he does not comment upon it, Thurman presents his results separately both for males and females and for those whose predisposition comes from the paternal side, maternal side, both, or unknown. We can thus calculate the frequency of an HP in males and females to equal $71/223 = 31.8\%$ and $83/246 = 33.7\%$, respectively, a nonsignificant difference ($x^2 = 0.19$, $df = 1$, $p = .66$). Next, we examine the proportion of patients with an HP that comes from only the maternal and the paternal side. These results are nearly identical

TABLE 1 Topics examined in the four studies reviewed and general results obtained

	Author			
Topic	**Thurman**	**Baillarger**	**Stewart**	**Jung**
Proportion of patients with HP	32.6%		49.6%	31.2%
HP by sex of patient	No difference		Females > males	Females > males
HP from maternal versus paternal side	No difference			
Relationship of HP to other causes	Descriptive data			
HP from mother versus father		Mothers more frequent	No difference	
Risk to offspring from affected father by sex of child		Girls > boys		
Risk to offspring from affected mother by sex of child		Girls > boys		
HP from mothers versus fathers to affected sons		No difference	No difference	No difference
HP from mothers versus fathers to affected daughters		Mother >> fathers	No difference	No difference
Does HP from mother versus father affect more children in the family?		Mother		
Distribution of affected relatives in positive pedigrees			Descriptive data: parents > cibs > avuncular > cousins	
HP among different psychiatric disorders			Highest in dipsomania, melancholy and mania	
Rates of recurrence			HP+ > HP−	HP+ > HP−
Recovery at discharge by HP			No difference	
Degree of homogeneity of familial transmission				Descriptive: relatively high, females > males
HP by religious group				Jewish > Protestant > Catholic

Note: Differences noted when shown to be statistically significant.
Abbreviation: HP, hereditary predisposition.

and respectively, 39/153 and 40/153. We can also examine evidence for sex-specific transmission—that is, do males and females differ in the proportion who have a clear paternal versus maternal predisposition? The answer is no ($x^2 = 0.31$, $df = 1$, $p = .58$).

Finally, Thurman also examines how an HP interrelates with the other both physical and moral (aka psychological) predisposing causes of insanity. Of the 153 predisposed individuals, 72 or 47.1% had no other known risk factor for insanity besides heredity. His list of other risk factors was extensive and included 21 physical causes (e.g., congenital weakness of mind, fever, dyspepsia) and 6 moral causes (e.g., mental disquietude, religious excitement, or ill-regulated temper).

2 | BAILLARGER (1844)

Jules Baillarger (1809–1890) was a major figure in mid-century French alienism who made important contribution to human neuroanatomy (Fulton, 1951), was a cofounder of the leading journal *Annales Médico-Psychologiques (AMP)* in 1843 (Bogousslavsky & Moulin, 2009) and was one of the two alienists to describe "la folie circulaire" ("cyclic insanity") in the early 1850s (Angst & Marneros, 2001). He was appointed as alienist at the famous La Salpêtrière in Paris in 1840 and was working there when he wrote this 11-page article, described as a "Note read at the Academy of Medicine," that appeared in the *AMP* (Bogousslavsky & Moulin, 2009) in 1844, entitled "Recherches statistiques sur l'hérédité de la Folie" (Baillarger, 1844) (Statistical research on the heredity of madness). It began:

> Everyone agrees on the influence of heredity in the production of madness. There is no doctor, among those who have run establishments dedicated to the treatment of the insane, who has not observed a sufficient number of facts to establish his conviction in this regard. (Baillarger, 1844, p. 328)

No details are provided as to how his sample was obtained or other descriptions of them. Rather, Baillarger begins by stating that he seeks to answer three questions about the familial transmission of insanity. First, is madness in the mother more frequently hereditary

than madness in the father? Second, given hereditary madness, does the disease of the mother affect the children in the family more frequently than from the father? Third, is the madness transmitted more often to the same sex children (from mother to daughter and father to son) than to opposite sex children (mother to son and father to daughter)?

The only features of the sample that he describes is that he began with 600 mentally ill patients, presumably from the Salpêtrière, who had an HP, that is had insanity in their families. In 440 cases, a mother or father were insane, in 13 both parents were insane, and in 147 only collateral relatives were affected. He excludes this final group and for this report concentrates on the remaining 453 families.

Regarding his first question, he writes

> Out of 453 lunatics suffering from hereditary madness, the disease had been transmitted by the mother 271 times and by the father 182 - the difference being 89 or about a third. Therefore, the madness of the mother is more frequently hereditary than that of the father by the proportion of one third. (Baillarger, 1844, p. 331)

This is a highly significant difference ($x^2 = 17.49$, $p < .0001$). He addresses his second question by first examining transmission of insanity from the mother:

> Out of 271 families in which the madness had been transmitted by the mother, the disease, at the time when the observations were collected, had manifested in a single child 203 times, in two children 62 time, in three children 5 times and in four children once… The mother's madness had therefore been transmitted to several children 70 times out of 271, i.e. in more than a quarter of the families. (Baillarger, 1844, p. 331)

Then he examines the father, writing

> As for the madness transmitted by the father, I found that out of 182 families, in which the madness came from the father, the disease, at the time the observations were taken, had occurred in only one child 152 times, two children 26 time, and three children four times… The father's madness was therefore transmitted to several children 30 times out of 182, that is to say, in one sixth of cases. (Baillarger, 1844, p. 331)

Transmission to more than one affected child occurred significantly more frequently from affected mothers than fathers ($x^2 = 5.53$, $p = .02$). He concludes that "… the madness of the mother, at the same time that it is more frequently hereditary than that of the father, appears also, all other things being equal, to impact on a larger number of children" (Baillarger, 1844, p. 332).

His third question was more complex. He began by examining sex-specific transmission of insanity from the perspective of the parents.

> Out of 346 children who had inherited insanity from their mother, I found 197 girls and 149 boys. The difference is therefore 48 or a quarter. Out of 215 children to whom the disease had been transmitted by their father, I found 128 boys and 87 girls, the difference being 41 or a third. The madness of the mother itself thus transmits more often to girls than boys in the proportion of a quarter; the madness of the father, by contrast, transmits more often to boys than to girls in the proportion of one third. (Baillarger, 1844, p. 332)

The sex ratio of affected children of affected mothers and affected fathers both differ significantly from chance expectations: $x^2 = 6.66$, $p = .01$ and $x^2 = 7.82$, $p = .005$, respectively. Then, he examined the problem of sex-specific transmission from the perspective of the offspring.

> We find that out of 271 [affected] boys, 146 received the madness from their mother and 125 from their father. The difference is 21 or a sixth. For the girls, the finding is the opposite and much more clear-cut … Out of 274 insane girls, 189 had their mother's madness and only 85 had inherited it from their father. This difference is 104, that is more than half… Hence this conclusion: That boys receive the madness of their father than of their mother but girls, by contrast, inherit, at least twice as often, the madness of their mother than that of their father. (Baillarger, 1844, pp. 332–333)

The ratio of affected mothers to affected fathers of affected sons by Baillarger did not differ from chance expectations: $x^2 = 1.63$, $p = .20$. However, the ratios of affected mothers to affected fathers of affected daughters differed markedly from that expected by chance: $x^2 = 38.48$, $p < .0001$. From his various concluding remarks, these comments are of most interest to us

> By applying the results, I obtained to the prognosis of children born to insane parents, we can arrive at the following conclusions …. the madness of the mother, with regard to heredity, is more serious than that of the father, not only because it is more frequently hereditary, but again because that it is transmitted to more children … [furthermore] the transmission of madness from the mother is more to be feared for girls than for boys while that of the father, by contrast, is to be feared more for boys than for girls…. I think I should add, before I finish, that I am far from claiming that the 600 observations summarized here that I have the

> honor to present to the Academy, are sufficient for the complete solution of the questions that I asked. I only consider them, on the contrary, as the first element of a work which needs to be continued. (Baillarger, 1844, p. 333)

3 | STEWART (1864)

When he wrote this article entitled "On Hereditary Insanity" in 1864 (Stewart, 1864), Dr. Stewart was the Medical Assistant in the Crichton Royal Institution in Dumfries, Scotland. Crichton Royal Hospital had been established with an endowment from Dr James Crichton (1763–1823) who sought to use the funds for "founding and endowing a lunatic asylum in the neighbourhood of Dumfries" in 1833 (Dumfries and Galloway Archives and Local Studies, Wellcome Library, 2021). The institution, originally called Crichton Institution for Lunatics, opened on June 1839 under the direction of Dr Browne, an advocate of moral therapy. It contained 120 patient beds, 54 allocated for private paying patients, and 50 for pauper patients. It gained its royal title in 1840.

After reviewing the prior statistics on the role of hereditary in insanity, Stewart wrote:

> Considering that the records of the cases in the Crichton Royal Institution afforded a good field for such an investigation, I have gone carefully over them, and tabulated the results. The statistics thus obtained embrace the cases admitted since the opening of the Institution, in 1839, to the end of last year, 1863, a period of twenty-four years, and show, in mass, the general history of 901 cases of insanity. The records of the cases were made by Dr. Browne, from 1839 to 1857; by Dr. Dickson, from 1857 to 1859; and by myself, from 1859 to 1863. (Stewart, 1864, p. 51)

Stewart describes the medical record material that he used for this report:

> These records are generally elaborated from answers given to certain queries forwarded to the friends and filled up by the medical adviser of the patient applying for admission. The query respecting hereditary tendency is the following: "Is the patient, or his relative, subject to any hereditary, nervous, or periodical disease, and what? or have they manifested any peculiarity, eccentricity, or prominent propensity, or tendency to crime?" From the answer given to this question, and from any other sources of information he may possess, the reporter makes up a statement on this point. (Stewart, 1864, pp. 51–52)

Multiple sources of information, including likely the patient him or herself, and his friends and relatives, were apparently summarized by the physician who is requesting admission. Stewart outlined a range of questions he seeks to address from this data. In many cases, Stewart compares his results with prior reports most frequently from Esquirol (E. Esquirol, 1838; J. E. D. Esquirol, 1845) and Thurman from the Retreat. First, he examines the proportion of patients with an "HP." He discusses the debate about how to define this measure, in particular which relatives and which diagnostic categories to include. He decides to include both the direct line and collateral relatives out to first-cousins and what he terms "insanity or eccentricity." By these criteria, Stewart found 447 of his 901 patients had a HP (49.6%). If those with unknown heredity are eliminated, that number rises to 63.8%. Of the 447 patients with a positive family history, 181 (40.5%) had only one affected relative.

Second, he examines a question that had not, to his knowledge, been previously addressed: how affected relatives were distributed in the pedigrees of predisposed patients. He divides relatives into five classes and does not comment on how he treats patients with affected relatives in multiple classes. Given that his figures sum to 100%, it seems likely he used a hierarchy. His percentages were: parents 48.1%, sibs 32.0%, avuncular 7.6%, cousins 4.0%, and unknown 8.3%.

Third, he examines the transmission of insanity from fathers versus mothers, noting that mothers "during her pregnancy and lactation might communicate disease to her offspring" (Stewart, 1864, p. 57). He finds that paternal transmission is found in 82 cases or 9.1% and maternal transmission in 68 or 7.5% which is not statistically significant ($x^2 = 1.43$, $df = 1$, $p = .23$). He notes these results do not replicate earlier findings of a preponderance of maternal transmissions.

He then poses a related inquiry "Is the insanity of the mother more dangerous to the females than to the males?" (Stewart, 1864, p. 58). He examines all four sex of parent by sex of offspring pairings and finds that of his male patients, 9.4 and 7.1%, respectively, report having an affected father and mother. This difference is not significant ($x^2 = 1.83$, $df = 1$, $p = .18$). Of his female patients, 8.7 and 8.1% report having an affected father and mother again not significant ($x^2 = 0.07$, $df = 1$, $p = .79$). From these data, we can ask if there was any sex-specific transmission and there was no evidence of such ($x^2 = 0.43$, $df = 1$, $p = .51$).

He then examines the impact of sex in the "receiving" of insanity. Examining his own data and those of his predecessors, he concludes that "… it indisputably shows the greater liability of the female sex to suffer more from hereditary insanity, than the male" (Stewart, 1864, p. 59). However, his own data find only a slight and nonsignificant excess rate of an HP in female versus male cases: 51.0 versus 48.6% ($x^2 = 0.26$, $p = .61$).

Stewart goes on to explore rates of "hereditary cases" among "the different forms of insanity." The results are presented only as percentages and so do not permit statistical analyses. In decreasing order (excluding imbecility) they are: dipsomania (63.4%), melancholy (57.7%), mania (51.0%), moral insanity (50.0%), monomania (49.0%), general paralysis (47.6%), and dementia and fatuity (39.5%).

He then examines the relationship between recurrence and heredity and shows that the proportion of admitted cases who had

recurrent versus a first episodes of illness was significantly higher in the "hereditary cases" 155/439 = 35.3% than in those without an HP (45/240 = 20.0%) ($x^2 = 17.35$, $p = .000031$). His final analysis of interest examines the state of their disorder on discharge. If we subtract those who died in hospital, the rates of recovery in hereditary cases 163/353 = 46.1% is nonsignificantly greater than the recovery rates in cases with no predisposition: 79/187 = 42.2% ($x^2 = 0.76$, $p = .38$).

He concludes his article as follows:

> In passing I have touched on many interesting topics that are yet to be worked out, and there remain over an immense number of points in the history of such cases, which would, if well investigated, enable us better to understand the group of mental diseases with which we have been dealing. These researches are offered to the Profession in the hope that they add something to our knowledge of hereditary insanity. (Stewart, 1864, p. 66)

4 | JUNG (1864)

Paul August Wilhelm Jung (1830–1908) was a German psychiatrist who eventually became director of the Silesian insane sanatorium in Leubus, then in eastern Prussia, and now in western Poland (Wikipedia Contributors, 2018). He received his doctorate in medicine in Berlin in 1853. He worked in 1858/1859 as a doctor in the psychiatric department at the Diakonissenanstalt in Kaiserswerth and from there to being a third doctor to the Provincial Sanatorium for the Insane in Leubus under Moritz Martini. At that time, the Leubus asylum was located in the former Leubus monastery and had 170 beds in the public section and a further 44 in the sanatorium for wealthy sick people. After Martini's resignation, Jung took over the management of the institution in 1873 and served there till 1884.

While working as an assistant physician at Leubus, Jung published a 122-page article entitled "Untersuchungen über Erblichkeit von Seelenstörungen" (Study on the heredity of mental disorders) in the German journal *Zeitschrift für Psychiatrie* in 1864 (Jung, 1864). Like the other reports here reviewed, Jung was working with hospital case records. Jung writes

> This paper aims to provide a statistical summary of the results of my investigations. Excluding relapses and recurrences, it is based on 3606 clinical reports and case studies. This concerns the patients treated over 33 years from the founding of this asylum until the end of 1862, from the public asylum (3284) and the sanatorium (322). They are based … in all cases on official records. (Jung, 1864, p. 535)

Jung goes on to note that his investigations were limited by the material available in the reports. In particular, the information about mental illness in relatives was

> divided into special lists depending on whether the mental disorder was inherited from the father, the mother, the parents, from the closest blood relatives in descending and ascending line, or depending on whether siblings became afflicted with or alongside each other. (Jung, 1864, p. 585)

Jung examined three main psychiatric syndromes: "melancholia, Wahnsinn, dementia and sometimes …Verrücktheit." Melancholia is likely to resemble what we might call more severe cases of major depression (Kendler, 2020a). Dementia would reflect disorientation and poor cognitive functioning but did not carry the chronic deteriorative connotations as the diagnosis used today (Berrios, 1994). Verrücktheit during this time period was roughly equivalent to nonaffective delusional psychosis. Wahnsinn was also a psychotic syndrome but typically had more pronounced mood symptoms.

We are given some details of his approach to defining an HP. He apparently counted both direct heredity by which he means affected parents and indirect heredity under which he includes siblings, grandparents, aunts/uncles, and first cousins. Vaguer references to affected relatives were not considered:

> All uncertain or general indications, such as "mental illness occurs as a family predisposition", all degrees of relationship not noted in detail, such as "a grandparent, a cousin, a side relative were insane", were excluded. (Jung, 1864, p. 586)

He does not provide a clear list of the disorders that qualify to make up an HP. However, in discussing direct heredity, he writes:

> Direct heredity is assumed in all the cases where the father, mother, or both parents were mentally ill or suffered from one of the mentioned brain afflictions, nervous disorders or character peculiarities. (Jung, 1864, p. 585)

This implies that Jung utilized a rather broad definition of affection but, frustratingly, other sections imply that he is dealing with relatively clearly defined forms of mental illness.

Jung examines in considerable detail the specificity of transmission across various relationships. In his first pages, he notes this as a key motivation for this study:

> Elsewhere I have heard the assertion that inherited mental disturbance switches or changes its form, that for example, the child or grandchild becomes ill with a different form of mental disturbance than the father, mother or grandparents suffered from. Focke virtually states that in a family with a tendency to emotional afflictions, if two members become ill, temporally or physically separated, they will only exceptionally or coincidentally be afflicted with the same form of illness. These statements gave me the idea of

> undertaking a comparison between the inherited mental disturbances and those of the ancestor. (Jung, 1864, p. 535)

The extensive analyses that he presented of this topic are sometimes obscure with the main outcome always presented as the percent of relatives having the same diagnosis in various classes of relatives, especially parent–child and siblings, but also more distant pairs of affected relatives. No data were available from which I could assembled 2×2 tables to reanalyze his data to determine if resemblance for individual diagnostic categories in pairs of relatives exceeded chance expectation. To give a sense of these results, I provide two examples, the first being from the section examining men:

> Among the 113 patients whose father had been mentally ill in any way or had suffered from the illnesses related to mental illness, [the sons] displayed the same form of disturbance 42 times in father and son, i.e. 37.17% … Among 53 patients whose father's parents or siblings were mentally ill, 18 had the same form of disturbance, i.e. 33.96%. (Jung, 1864, pp. 591–592)

Here is a parallel passage from his section on women:

> Among 119 cases… where the mothers were mentally ill in any way or suffered from conditions related to mental illness, mother and son showed the same form of mental disorder 52 times, i.e. 43.70%… Further, among 34 patients whose mother's parents or mother's siblings were mentally ill, these and the grandchildren or nephew [were similar] 11 times, i.e. 32.34%. (Jung, 1864, p. 597)

We turn to Jung's conclusions:

> 1) The form of the mental disturbance the father or mother suffered from, is repeated in the form of mental disturbance in the son or daughter in more than half the cases.
> a. If several members of the same family and ancestry become mentally ill, the greater probability indicates that all of them will suffer from the same or a similar form of mental disorder and in the same age period.
> b. If a child has inherited the temperament and character of one of its parents (grandparents, parent's siblings) and if the ancestor becomes mentally ill, he will almost certainly become mentally ill, and very probably with the same form.
> 2) The female part of the family shows a greater tendency to suffer from the same form of the disorder than the male. This appears regardless of their greater tendency to inherit a mental disorder at all.
> 3) Siblings regularly become afflicted – in ¾ of all cases – with the same form. Brothers and brothers more often than sisters and sisters, these again more often than brother and sister. (Jung, 1864, p. 623)

Jung clearly felt that the resemblance seen in parents and offspring were beyond chance expectation and is quoted by historians as advocating for the homogeneity of familial transmission of forms of insanity (Gausemeier, 2015). With respect to HP and sex, in his overall sample, 28.4% of patients had an HP. When examined by sex, he found 31.2% of the female patients (533/1,706) had such a predisposition compared to 26.0% of men (494/1,900), a highly significant difference ($x^2 = 12.1$, $p = .0005$).

He examined the impact of an HP on the maternal and paternal side on illness in male and female patients. Of 605 male patients with an HP, 312 came from the father's side and 293 from the mother's side. For the 629 female patients who were hereditarily predisposed, in 296 and 333 cases, respectively, it came from the fathers or the mother's side. While Jung concludes from his data show "more men with a HP on the father's side [and], more women with a HP on the mother's side," this difference is not significant ($x^2 = 2.5$, $p = .11$).

With respect to religion, Jung reports the proportion of cases with an HP who were Protestant (672/2,195 = 30.6%), Catholic (302/1,239 = 24.4%), and Jewish (36/89 = 40.4%). These rates are significantly different for Protestant versus Catholic ($x^2 = 15.2$, $p = .000098$), Catholic versus Jewish ($x^2 = 11.3$, $p = .000772$), and, marginally, for Protestant versus Jewish ($x^2 = 3.9$, $p = .049$).

Then, he addresses the relationship between HP and course. He gives four possible discharge categories for the patients: recovered, improved, unrecovered, and death. For our analyses, we combined improved (the least frequent outcome) with recovered and excluded cases who died. Examined in the way, the proportion of those recovered + improved equaled 552/894 = 61.7% among those with an HP compared to 1,188/2,085 = 57.0% in those without, a significant difference ($x^2 = 5.9$, $p = .016$).

5 | DISCUSSION

The goal of this article is to give a sampling of one major kind of psychiatric genetics research in mid-19th century Western Europe. We examined four diverse studies in three countries all of which utilized official asylum records containing information about the history of mental illness in relatives. None of these studies were groundbreaking but neither were they unknown. For example, in Koller's important 1895 study utilizing control groups for the first time to investigate the degree of elevation in HP in the mentally ill, a rather detailed review of past studies of rates of HP in asylum patients cites Baillarger and Jung (Koller, 1895). Of the many features of these studies, we highlight three.

First, as evident from Table 1, these investigators were asking a wide-ranging set of questions about the nature of the transmission and the clinical impact of an HP to mental illness. In this historical

period, the field was not simply quantifying the frequency of an HP among the patients admitted to a particular asylum. By far the most common question addressed by these investigators was how transmission of a predisposition to mental illness was influenced by sex—both of the patient and the affected relatives. As noted above, a likely motivation for the importance of this question was the continued uncertainty about whether the role of fathers and mothers in procreation was fundamentally similar or quite different. Alternatively, the basic contribution of the two sexes to the fertilized ovum might be equal but intrauterine effects or those related to nursing could render the mother's influence on risk of insanity much stronger than the father's.

Every one of our four studies examined this question and some consilience was seen in their findings. Two of three studies found that mental illness was more frequently familial in females than in males. The fourth—Baillarger—looked at this separately for affected mothers and fathers and, in both cases, found risk to be greater in daughters than in sons. With some consistency, our investigators found that, given an HP in a family, females were more likely to become affected than males.

Results were less clear when the sex of parents was studied. Three studies examined whether transmission of mental illness was more common from mothers or fathers (or the maternal vs. paternal side of the family) and only one found a significant difference being greater from the mothers. Three studies looked specifically at transmission to both affected sons and daughters and only one of the six comparisons was significant, with mother's transmitting mental illness to daughters more than fathers. Baillarger followed up this line of inquiry in a novel analysis showing that families with ill mothers contained more affected children than families with ill fathers. Overall, across these multiple findings and samples, the consistency of the positive findings is impressive. When a difference was detected, seven out of seven times, hereditary factors coming from or being received by females had more effect than with males.

Second, Thurman, Stewart, and Jung addressed, between them, a range of other interesting questions about the action of heredity in mental illness. Thurman provided descriptive data on how an HP related to other putative causes of insanity—a rich question that continues to be actively investigated today. Stewart asked where affected relatives were most often found in the pedigrees of his ill patients. It is not surprising to us that the first two of his categories, parents and siblings, are the only first-degree relatives who had likely lived through at least part of their risk period for severe mental illness. But any basic theories about the sources of resemblance in relatives, especially Galton's law of Ancestral heredity (Galton, 1889) and Mendel's theories (Olby, 1985) were not known for decades.

Stewart also addressed a question which remains central to the field—the relative frequency of an HP in different psychiatric disorders. Dipsomania (alcoholism) and mania topped his list. Both Stewart and Jung examined the relationship between recurrence and HP. Contrary to the general views in medical genetics of this era that the course of illness was typically more chronic and progressive in "hereditary cases" (Rushton, 2009), they both found a different outcome, that recurrence was more common in patients with than without an HP. Jung, at some length (and obscurity), reviewed the question which was to remain central for both pre- and post-Mendelian psychiatric genetics (Kendler & Klee, 2021; Porter, 2020)—that is, whether the familial transmission of psychiatric illness is largely homogeneous (like producing like) or heterogeneous (Kendler, 2020b). He concludes that his results from his very large patient sample favor the former position. Finally, Jung addresses the impact of religion/ethnicity on the familial nature of psychiatric illness showing significantly higher familiality in Jewish than in Protestant than in his Catholic patients.

Third, it is also appropriate to appreciate the methodological limitations of this research. While the methodology of what we now called pedigree studies—wherein individual assessments and/or records on members of extended families were individually examined—was being pioneered during this time period by Ludvig Dahl in Norway (Porter, 2018, chap. 6), it is likely that nearly all of the data in the asylum records on relatives was indirect, from family informants. That is, all of these results examined here (with one exception noted below) appeared to rely on a single measure of uncertain reliability or validity—the family history of mental illness as recorded in asylum records. Thurman comments in some detail and Jung more briefly on the many methodological questions that would be addressed later in the 19th century on such data, including which relatives should be counted, which disorders should be included, and the problems of the accuracy of the records (Kendler & Klee, 2020a, 2020b). It is clear that our investigators did not all define HP in the same way as Thurman did not accept affected collateral relatives as indicative of an HP in patients while Jung did. Stewart clearly stated that he included, in his HP calculations, relatives who had what we would now call a spectrum disorder ("any peculiarity, eccentricity") an approach not clearly endorsed by any of the other authors. While the other authors appeared to be working largely or entirely from the written hospital record, working largely with the tight Quaker community, Thurman notes he had additional source of information available to him on family history from outside the formal chart material.

The work of these investigators also shared a methodological perspective that dominated most of 19th-century psychiatric genetics—a focus on the ill patient rather than their relatives. Not until the second decade of the 20th century, based on the hope of finding Mendelian ratios, did psychiatrists carefully examine systematic samples of close relatives of the seriously mentally ill (Rudin, 1916). Finally, the concept of a control group was not introduced into psychiatric genetics research until 1895 by Jenny Koller (Kendler & Klee, 2020b). However, this methodological concern applied to little of the findings presented by these researchers who were largely comparing subsets of mentally ill patients divided on the basis of their HP.

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CONFLICT OF INTEREST

The author reports no conflicts of interest.

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