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Ludvig Dahl's psychiatric genetic studies in his 1859 monograph: "Contribution to the knowledge of insanity in Norway"

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Abstract

In 1859, Ludvig Dahl, a Norwegian alienist, wrote a rarely referenced book entitled "Contribution to The Knowledge of Insanity." In it, he describes a highly innovative psychiatric genetics research project with severable notable features. First, while the vast majority of 19th century psychiatric genetic studies were based on asylum hospital records, Dahl did field work to find cases of mental illness in certain defined areas within Norway, using census data, key-informants, record reviews, and personal interviews especially of suspected affected individuals. Second, for the first time in the history of psychiatric genetics, and perhaps more broadly in medical genetics, Dahl studied and graphed extensive pedigrees covering up to seven generations demonstrating a high density of psychiatric illness. Third, he proposed and conducted the first controlled investigation of familial aggregation of insanity. A 126 member 5-generation pedigree that he studied contained 8 individuals with confirmed insanity compared to 16 cases in the remaining 2,974 individuals in the Parish, a relative risk of nearly 12. Dahl also noted the co-segregation within pedigrees of mental handicap, deaf-mutism, and insanity. He evaluated familial-environmental sources of familial aggregation and noted, among nonpsychotic family members in his pedigrees, personalities that might reflect a "disposition" to insanity.

1 | INTRODUCTION

This essay provides a detailed description of the contributions to the history of Psychiatric Genetics of Ludvig Dahl contained in his 1859 book entitled "Bidrag Til Kundskab om De SindssygeINorge" [Contribution to The Knowledge of the Insane in Norway] (Dahl, 1859) accompanied by the first English translation of the relevant text in an AppendixS1.

This article is divided into four parts. First, we provide, the historical context of Dahl's work with respect to the general development of Psychiatry and particularly that of the very young science of psychiatric genetics. Second, we give a brief biography of Dahl. Third, we review the sections of his book dealing with his findings in the genetics of insanity. Finally, we review the prior material, seeking to place Dahl within the broader history of the field of psychiatric genetics.

2 | HISTORICAL CONTEXT

Nineteen years before the publication of Dahls' monograph, the pupil of Pinel (1745–1826) and the last great figure in early 19th century French Psychiatry, Jean-Étienne Dominique Esquirol (1772–1840), died. Five years earlier in 1854, the French alienist Falret published an article describing a syndrome that would evolve into what we now term bipolar disorder (Sedler, 1983). Three years earlier, in 1856, Emil Kraepelin (1856–1926), to be the dominant figure in late 19th and early 20th century European psychiatry, was born. In the same year as Dahl's publication, in 1859, the first edition of the most influential psychiatric textbook in mid-19th century Britain "A Manual of Psychological Medicine" by John Bucknill (1817–1897) and Daniel Tuke (1827–1895) was published (Bucknill & Tuke, 1858). Six years later, in

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1865, Wilhelm Griesinger (1817–1868), who began the brain and neuropathology focused-tradition in German alienism, would be appointed to the first university chair of Psychiatry in German-speaking Europe. Twelve years later, the monograph on hebephrenia written by Ewald Hecker (1843–1909), one of the foundational texts for Kraepelin's later synthesis of Dementia Praecox, was published (Hecker, 1871; Kendler, 2020).

As outlined elsewhere (Kenneth S Kendler, 2021a; Porter, 2018), descriptions of the familial nature of mental illness can be dated back to the very beginnings of Western psychiatry in the 1780s. What was originally a series of clinical observations had, by the mid-19th century, begun to develop a modest research tradition (Kenneth S Kendler, 2021b). Using family history reports in asylum records, patterns of transmission of insanity were explored. Early researchers were, for example, interested in whether men or women were more likely to transmit, or were more sensitive to, a hereditary predisposition (HP) to insanity. Other topics explored included the relationship between an HP and recurrence and whether the transmission of mental illness within families was largely homogeneous (i.e., "like transmits to like" in families) or heterogeneous (that close relatives of mentally ill probands had a wide variety of psychiatric conditions) (Kenneth S Kendler, 2021a). The concept of a controlled study–that would test whether psychiatric disorders were concentrated in the families of mentally ill patients compared to an appropriate comparison group–were not considered. Indeed, the first such controlled investigation was not published until 1895 (Kendler & Klee, 2020). For readers interested in more details of the background to Dahl's genetic studies should consult Porter's recent book, the only modern English source we have found which discusses Dahl's work (Porter, 2018) (chapter 6).

3 | DAHL BIOGRAPHY

Ludvig Wilhelm Dahl was born in Bergen in 1826 into an upper middle-class family graduating from medical school in 1851. In 1855, he married a daughter of the mayor of Trondheim (Norway's third largest city). They had 13 children. From 1875, he became the first Director General of Health and Chief Medical Officer (CMO) of Norway and died in what is now called Oslo, Norway in 1890, but was termed Christiania up until 1897, then Kristiania until 1924, when Oslo became its official name.

Insanity was not part of the health care system in Norway until 1848 when the so-called Insanity Act was passed in the parliament: *Law of the treatment and care of the insane*. This moved the responsibility for the insane to the state and its health care system and led to the establishment of a National asylum system. Three asylums were built in Norway from 1855 to 1881.

Norwegian physicians were not well prepared for the task of running these asylums as they had no training in psychiatry. So, the asylums had to create a first generation of alienists. One of the main tasks in the development of new psychiatric health care system was to determine the prevalence of insanity. In 1824, a commission in the parliament initiated the first counting of the insane in Norway. This began in 1826 in parallel with the census, and with the help of the clergy. Professor Holst, a well-known professor of medicine, was the leader of the commission. Counts were done in 1835, 1845, and 1855.

From 1853 to 1854, Dahl traveled extensively in Germany, Austria, and France, and visited a number of asylums for the insane. In 1855, he became a member of the building committee for the first new asylum in the capital, Oslo (at that time called Kristiania). When the asylum opened in 1855, he was appointed senior resident physician, a position he held until 1861. From 1861 to 1864, he held different positions in the Norwegian Correctional Service and in the Civil service related to the care of the insane. In 1862, he proposed the building of asylums in two large cities in Norway (Trondheim and Christiansand) and from 1871 to 1875 he was the first director for Rotvoll asylum in Trondheim.

Dahl participated actively in a large number of international meetings and associations. He also continued to travel abroad and in Norway to study Insanity and the conditions, which the insane were living under. In 1856 he went to Denmark, Holland, Belgium, and the UK, and again in 1864, 1873 and 1883, he visited asylums in Switzerland, Germany, Austria, and Italy to study the newest advances in the care of the insane. He was, at that time, well informed and up to date with regard to the latest international developments in the classification, care, and treatment of the insane.

It was therefore not surprising that he was asked by the Ministry of the Interior in 1857 and 1858 to make an investigation into the prevalence of insanity and the conditions for the insane in Norway, after the count in 1855 had shown high numbers. He spent more than 7 months visiting different parts of Norway and carrying out very thorough and detailed investigations. This resulted in the publication,, of the monograph we review here in 1859.

4 | COMMENTARY ON DAHL'S MONOGRAPH

The first major section of Dahl's monograph examines the prevalence of insanity in Norway and contains an extensive discussion of diagnostic categories then in use, particular mania, melancholia, dementia, and idiocy, which were the categories employed in the population counts of 1826 and 1835. However, in the counts done in 1845 and 1855, only two diagnoses were used: furious (rasende), and fools (fjanter), with the latter subdivided into two kinds, according to whether the feeblemindedness was congenital or developed later in life. We will not review those detailed discussion here, although they are included in our translation in the Appendix S1. We should note, however, that these terms cannot be interpreted as equivalent to their modern counterparts. Briefly, mania was any kind of generalized insanity associated with agitation. Melancholia reflected a narrower form of psychotic illness–typically one major delusional theme–often but not always associated with sadness and suicidality. Dementia represented a substantial loss of cognitive functioning and practical living skills but did not have the connotation of irreversibility that term has today. Idiocy was a congenital form of sever cognitive dysfunction.

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In his extensive field work, Dahl relied on official Norwegian census data, but that was supplemented by extensive consultation with local Lutheran Priests, Sheriffs, and School-teachers serving as key-informants. In some cases, he was assisted in pedigree tracing by cooperative family members. Dahl was able to personally examine a substantial proportion of the living members of the pedigrees he investigated, particularly those reported to have mental illness or mental handicap. He did note that some of the insane subjects were unwilling to be seen by him or lived in such remote locations that personal contact was not feasible. For most of those he was unable to see, he reported being able to gather sufficient informant information to be able to assign a diagnosis with confidence.

In this section of his book, Dahl describes at some length his diagnostic approach trying, for example, to discriminate those who have been fools from birth versus after birth. But in his work with pedigrees with high concentrations of mental illness, we only see two common diagnoses, idiocy, and insanity, with deaf-mutism noted in one of them. It is not possible with the information available to us to give a clear modern "translation" of his category of insanity. However, it is clear that Dahl distinguished between what he sometimes termed "acquired insanity," by which he meant with an onset during or after adolescence, as distinct from a range of congenital mental problems that began in early childhood. It is likely that he reserved this term for those who would, in his historical era, be diagnosed with mania and melancholia. In modern terms, this would translate into those with major nonaffective and affective psychotic illnesses and perhaps some nonpsychotic forms of depression and mania. Given his clinical experience, it seems unlikely that his category of insanity included any appreciable number of individuals whose primary neurologic disorders or various forms of uncomplicated mental handicap.

Dahl's next section was entitled "Common causes for Acquired Insanity and Idiocy" which he begins as follows:

> *I consider heredity, perhaps in conjunction with marriage between close relatives, as well as the enjoyment of spirits, to be the most important of the common causes* (Dahl, 1859) *p. 76.*

His first subsection, entitled "Hereditary Factors" is the major focus of our inquiry with our emphasis on his results with respect to insanity. He begins with a discussion of the definition of heredity. As background, we should note that in European culture, the concept of "inheritance" of diseases developed from the more general concept of the inheritance of wealth and land (López-Beltrán, 1992). Therefore, the predominant model of inheritance in early scientific efforts was direct lineal transmission–that is, from parent to children. There was with much less interest in siblings or collateral relatives such as like aunts or uncles. Dahl questions this approach, concluding that it is too narrow:

> *If one, as some Norwegian authors have done … limits oneself to only take heredity to be proved in the cases where the disease has been present in parents [of the mentally ill]… then this cause would be considered only in a very small number of insane. However, … I will, in what follows, present examples, that most clearly demonstrate the heredity of the discussed conditions, even where no such straight forward transmission can be shown to have taken place* (Dahl, 1859) *p. 76.*

He then goes on to argue the even vertical transmission cannot guarantee that heredity is operating. He here makes the point, rarely noted up previously psychiatric genetic writings, that environmental sources of risk could cause disorders to cluster in families (Kenneth S Kendler, 2021b):

> *Even when both parents and children suffer from the same condition, the latter do not need to have received any predisposition from the former. Random causes can have acted simultaneously on members of the same family. Only where a certain disease or class of diseases occurs within a kin group (slækt) with a far greater frequency than outside, without this being able to be explained by the action of other known causes, can one be justified in assuming that this can be attributed a HP (Dahl, 1859) Pp. 76–77 (emphasis added here and elsewhere in the essay).*

That is, Dahl not only makes the important observation that familial aggregation alone is not sufficient evidence for the operation of heredity, but he then proposes the specifics of a test for "HP." To be confident that hereditary factors are at work, he suggests it is necessary to (i) show that the illness is much more common within than outside a family (or large pedigree) and (ii) rule out possible nonhereditary (e.g., environmental) influences. Dahl is here implicitly criticizing much of the prior work in the field which has assumed, with near but not complete uniformity (Kenneth S Kendler, 2021a), that a family history of mental illness in an asylum record constitutes sufficient proof for the operation of hereditary factors.

Dahl then goes on to a more typical presentation of his ascertained subjects with insanity. Of the 151 insane patients he studied, 58 (38%) had "insane relatives," but only 18 (12%) had affected parents, thus documenting his above point–hereditary influences on insanity would be substantially underestimated if only parent-offspring transmission were considered.

The remainder of this section of his book is oriented around a series of pedigrees focused mostly but not entirely on those with a high density of insanity. Dahl's first pedigree (Figure 1) comes from Storelvdalen in Østerdalen, about 200 km due north of Oslo. No proband is specified but for simplicity, we will assume it was Haaken. Haaken had six insane relatives: one uncle, one great-uncle, and four niece/nephews all offspring of different siblings. In addition, Haaken had one brother and two children with idiocy. Dahl was able to personally examine 4 of the ill relatives and confirmed the diagnosis in each of them. He describes this pedigree as follows:

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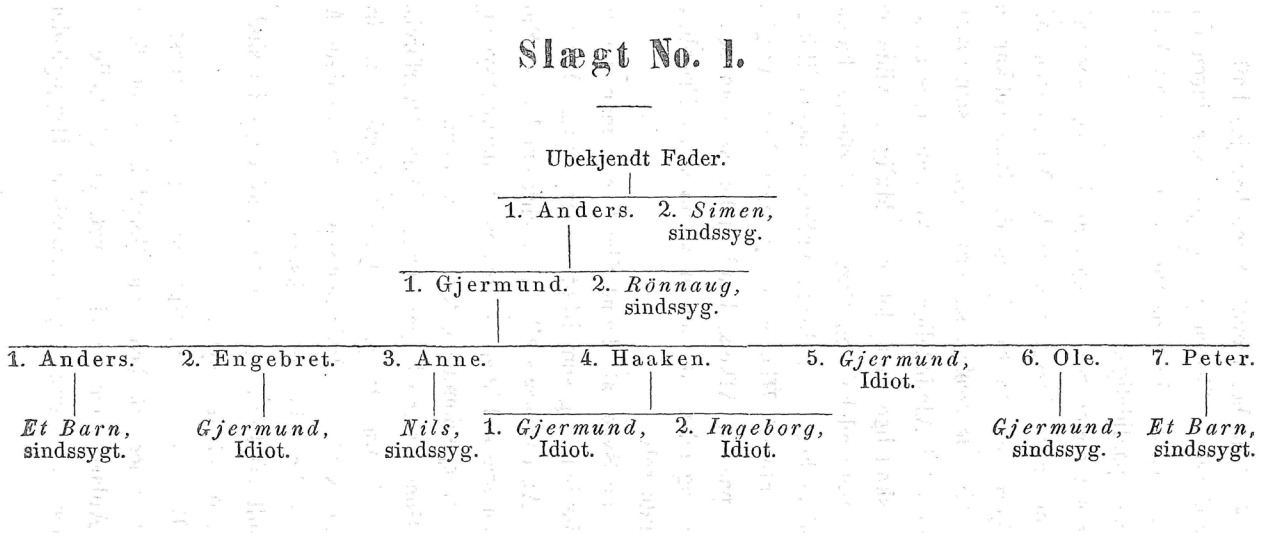

FIGURE 1 Pedigree # 1 from Dahl's monograph. Translations of diagnoses in this and subsequent pedigrees: Sindssyg - Insane; Friske - healthy; dovstum - deaf-mute; barnlos – childless; fjantede – fools; Fjantet - fool

> *Despite none of the ill in this family seeming to be descended from someone insane, the action of a common sickly seed appears so frequently that there can be no doubt regarding its presence, and even less that the general circumstances of the family which suggest that the family was not said to be exposed to other harmful influences…. However, there is nothing preventing the transfer of an illness seed to one's offspring, both without oneself having received it (i.e., from one's parents), and without it breaking through in those transferring it*. (Dahl, 1859) *p. 80*.

Dahl here directly addresses two central questions that appears over and over in the in the 19th century psychiatric genetics literature. First, is it insanity or the liability to insanity that is transmitted in families (Kenneth S Kendler, 2021b)? This was a struggle because while common sense might suggest that it must be the disorder itself, too many clinicians had seen that only one or a few members of a sibship would be ill or that the illness would seem to skip generations, as we see in pedigree # 1. Dahl clearly comes down on the side of the liability theory, as the patterns observed in the pedigrees would be difficult to understand if the disorder itself was transmitted. Note this position strongly supports his view that defining inheritance as requiring parent-offspring transmission of illness is far too narrow. The difference between Dahl's discussion of these issues and most other writers in the field in his historical era is that he bases his conclusions on the result of well documented pedigrees rather than on the more abstract bases of "clinical experience."

Dahl also addresses a second critical question raised by many 19th century authors noted above: is the transmission within families of insanity homogenous or heterogenous (Kenneth S Kendler, 2021a)? His response is "In the ill in this family, one can see this seed reveal itself as idiocy, or in other forms of insanity (Dahl, 1859) p. 80."

Dahl then turns to consider pedigree # 2 (Figure 2), which was collected in the region of Flekkefjord about 400 km south-west of Oslo on the coast. This 3-generation family was brought to Dahl's attention by the district doctor who admitted one of the affected members to the Gaustad hospital in Oslo. As Dahl summarizes "Among the 30 children and grandchildren of a noninsane man, 11 became insane (Dahl, 1859) p. 80." Only 21 are given in the pedigree and this includes 4 of the 8 offspring of Ole. Of the 13 grandchildren seen on the pedigree, all from three of his offspring–two healthy and one insane–6 were themselves insane. Dahl comments on the two kinds of transmission seen in the pedigree, from affected to affected (e.g., Dominikus to Jens, Tobias, Andreas, and Daniel) and from unaffected to affected (Brynild to Rakel and Malene to Gurine). In this pedigree, almost all the depicted transmission are homogenous from insanity to insanity with the exception of Brynild, which has one child with idiocy.

Dahl then returns to his novel but simple suggestion of testing empirically the familial aggregation of insanity.

> *The most certain way … to determine the significance and strength of such a predisposition within a family is to count all living members of the same, and compare the frequency of the illness among them with the frequency of the illness in the greater community in which they lived* (Dahl, 1859) Pp. *80–81*.

He performs this test on pedigree # 3 – a 5 generation family from Flesberg in Numedal, approximately 90 km due west of Oslo (Figure 2). We quote in full this rather remarkable paragraph in which Dahl calculates the degree of concentration of both insanity and deaf-mutism in the descendants of Ejvind–the progenitor of this pedigree:

> *In this family, there are nine that are or had been insane or idiots, four deaf-mutes, and one epileptic. All four deaf-mutes and eight of the insane are alive. In Flesberg parish,*

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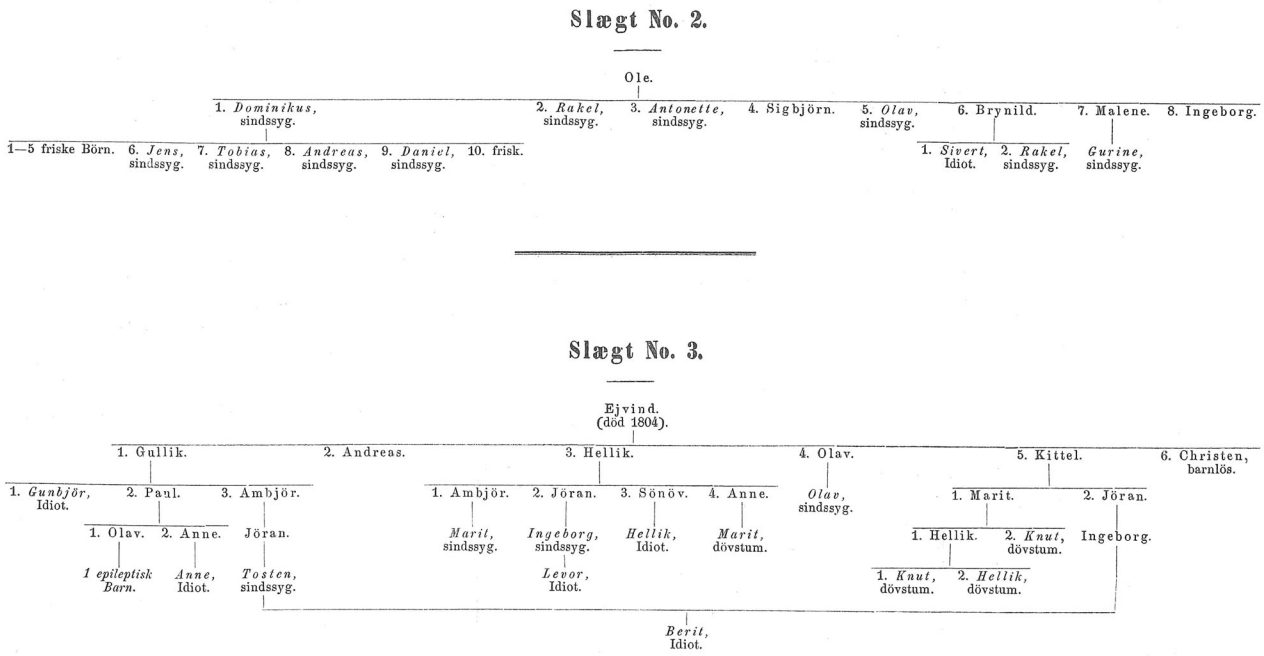

FIGURE 2 Pedigrees # 2 and 3 from Dahl's monograph

> *I have, in total, found 24 insane and 6 deaf-mutes. According to the census, there are listed 11 deaf-mutes, but 5 of these were also counted among the idiots. Therefore 12, or two-fifths of all the 30 in the parish belonged to the insane and deaf-mutes in this one family. To be able to determine how widespread this family could be, I took it upon myself to count precisely living descendants of Ejvind in the parish. With the help of one of his grandchildren and several other members of the family, I found their number to be 126. Flesberg has a population of 2,920. While the relationship within this family is 1 insane or deaf-mute in 10 or 11 individuals, for the other 2,794 (in Flesberg) the ratio is 1 in 155. The predisposition for these conditions or the probability of suffering from them is therefore 15 times greater within this family than outside it. If this relationship is taken for the insane, it can be seen that [the ratio is] 8 out of 126 [within the pedigree], and 16 out of 2,974 (outside it) or 1 in 16 versus 1 in 175, approximately 11 times greater within than outside the family (Dahl, 1859) p. 81.*

His efforts to enumerate all the family members are important and his calculations are straight-forward. While the development of formal statistical tests for such results (e.g., chi-square) were decades in the future, the results are quite convincing. Although compared to the first two pedigrees, the density of insanity in pedigree 3 is considerably less, and no insanity is observed in Ejvind or his children, the concentration of insanity compared to the community rates is striking. Dahl also shows that *both* idiocy and deaf-mutism are also concentrated in this family.

Dahl comments on some of the additional information he gathered on this family. This family is "one of the richest in the parish." He continues:

> *Ejvind himself is described as a competent and reasonably upstanding man. Of his sons, Kittel and to some extent Gullik is described as labile to party and drink. The strangest row is Hellik's daughters, who are described as having dark and hefty characters, and partly to have treated their children harshly (Dahl, 1859) p. 82.*

In dismissing the possibility that the concentration of illness in this pedigree arose from environmental causes, Dahl writes "The family was not gathered in any one place, and no particular influences are known to have acted upon its members rather than on the other residents of the parish (Dahl, 1859). p. 82."

The largest pedigree with a high density of insanity presented by Dahl is number 4, which includes 6 generations–beginning with the priest Preder Lögit–and several inter-marriages (Figure 3). This family contains, according to Dahl a "total [of] 27 members, whereof about 20 living, who are or have been insane (Dahl, 1859) p. 83." (of which 18 are seen in the pedigree). He notes that he was not able to enumerate all living members of the parish, which prevented him from calculating rates of illness within versus outside the family as he did with pedigree 4.

This large pedigree contains only a single transmission of insanity to insanity (Lars to Lars on the far right side of the pedigree) and

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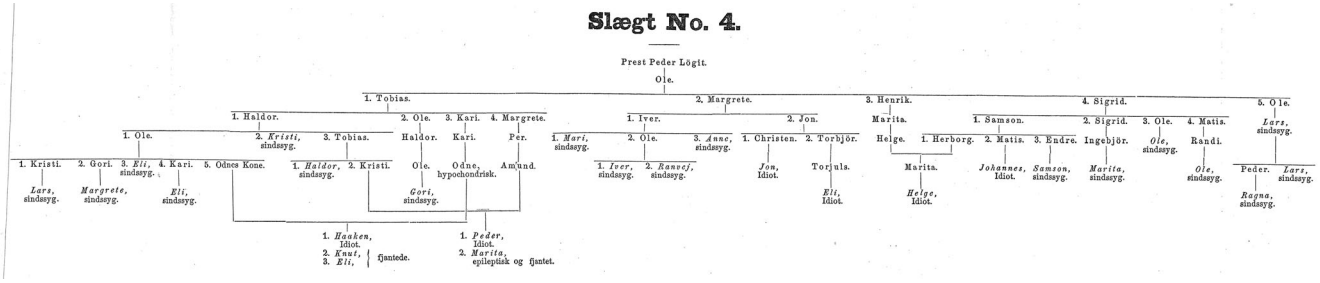

FIGURE 3 Pedigree # 4 from Dahl's monograph

otherwise all cases of insanity lack affected parents. This is likely partly explained by the low fertility of the insane members of the family. Insanity co-segregates with idiocy in several parts of the pedigree. An elevated level of idiocy and "fools" is seen in the offspring of the inter-marriages, something Dahl comments on later in his monograph, in sections we do not review here.

Dahl presents a few other pedigrees, but these focus on other disorders especially albinism although they sometimes contain one or more members with insanity. From his group of investigated families, he comes clearly down on the side of heterogenous transmission, noting that "it appears that the same sickly disposition among different members of a kin can cause insanity, idiocy, deaf-muteness, and albinism (Dahl, 1859) p. 88."

Before concluding this section, Dahl deals with two further issues. The first is what we would now call "spectrum" conditions where the liability to psychotic illness is reflected in personality traits and not psychotic illness. This has been observed by multiple authors in the 19th century, typically noting high rates of oddness or eccentricity in the relatives of their hospitalized patients (Kenneth S Kendler, 2021b). Dahl writes.

> *Naturally, it is not always in fully developed forms of illness that the heritable dispositions become visible. There are certain properties, like in the apparently healthy members of these families, that suggest the presence of the traits (Dahl, 1859) p. 89.*

Dahl goes on to enumerate a range of possible "spectrum" traits from a "dark and hefty" character, "a sad and introverted mind," "a strong sex drive and masturbation" and a tendency to hallucinations.

Second, he returns to the concern about environmental sources of familial transmission with an example of a particular group –the Taters which spoke the Romani language–which he describes:

> *I would mention drunkenness in the parents, maltreatment of the mothers during pregnancy, the hardships and dangers of traveling, both for the fertile mothers and for the newborn, the poor childcare and all the suffering and damaging influences, that the small children are early subjected to. All this is reason enough for the frequent occurrence of these conditions among the Taters, without having to resort to any HPs (Dahl, 1859) p. 93.*

He wishes to remind readers that certain families can be exposed to such degrees of disorganization and abuse that disorders like insanity and idiocy could arise solely from environmental factors.

Dahl provides a list of conclusions from his section on heredity which we quote in part:

a. *Acquired insanity and idiocy frequently occurs together in the same kinfolk (slægt). Deaf muteness also occurs frequently and albinism occasionally in such families…*
b. *Predisposition for these conditions can partly be transferred onto the children from parents who suffer from the same condition, partly be transmitted to the children without the disease having broken out in any of the parents in a directly ascending line. Immediate transfer from parents to children seems though to be the rarest. Often the presence of the predisposition in healthy members of the kin group (Slægten) can be detected in certain mental and bodily attributes.*
c. *Where other causes are known to have acted preferably on members of a family, the frequent occurrence of these disorders within the same cannot be taken to prove heredity. Likewise, the circumstances that parents and children suffer from the same condition need not always prove heredity.*
d. *In certain very widespread kin groups (Slægter), the predisposition can have such force that the probability of suffering the disease is 15 times as great within as outside the family…*
e. *Marriages between members of such a family increases the frequency of disease occurrence in the children…*
f. *Numerical estimates of the frequency of the heredity of insanity must always remain uncertain, as they rely on a somewhat uncertain judgment (Dahl, 1859) pp 94–95.*

5 | DISCUSSION

Dahl addresses, in his book, many of the critical extant *scientific* issues in the young discipline of psychiatric genetics of the mid-19th century

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(Kenneth S Kendler, 2021a). Of these, five are worthy of particular comment.

First, he explores the nature of the transmission of insanity. Despite a high-density of illness in his pedigrees, insanity most typically arises when neither parent is affected. That finding dictates that the liability to the insanity must be transmitted in families, not the disorder itself.

Second, he presents strong evidence that confining the concept of hereditary influences to only parents is empirically unjustified. Such an approach would, he argues, substantially underestimate the proportion of insane individuals with a heredity predisposition.

Third, Dahl examines in his pedigrees the question of the homogeneity versus heterogeneity of familial transmission of insanity. Both idiocy and deaf-mutism co-segregate with insanity in his pedigrees suggesting that the familial liability to insanity also impacts risk for other disorders.

Fourth, in accord with a number of other 19th century observers, Dahl's notes that nonpsychotic relatives of insane individuals in his families have an excess of unusual personality traits that might reflect forme fruste of the familial liability.

Fifth, Dahl argues that familial transmission need not arise from hereditary factors but could emerge from pervasive environmental stressors and/or deprivations.

Dahl made two major *methodological* contributions to the field. First, he collected data on his pedigrees through field work performed far outside the asylum walls. He utilized a combination of at least four information sources: (i) records and personal contact with personnel at the Gaustad hospital in Oslo, (ii) key informants in these rural communities (ministers, teachers etc.), (iii) reports from relatives, and (iv) personal examination of the living relatives when feasible.

Our research suggests that Dahl's pedigrees are the first ever published in the psychiatric literature and perhaps more broadly in human genetics. The historian, Porter, described Dahl's "pedigree tables of inherited conditions [as], apparently the first such table ever printed (Porter, 2018) p 136." In a review of the literature on pedigrees in psychiatric genetic research in the 19th and early 20th century, Gausemeier (Gausemeier, 2015) (apparently unaware of Dahl's research) notes the earliest report of presented pedigree-like data was in 1869, a decade after Dahl, by the French alienist Doutrebente (Doutrebente, 1869). Doutrebente was attempting to provide evidence in favor of the degeneration theories, popular at that time in France, and so, according to Gausemeier (Gausemeier, 2015), "… presented (mostly rather fragmentary) pedigrees which all suggested that incurable mental diseases originated from minor nervous disorders in preceding generations (Gausemeier, 2015) p. 475." Larger and more thorough pedigrees of psychiatric patients were not available until the 20th century (Gausemeier, 2015).

Second, in his most innovative methodological contribution, Dahl proposed a controlled study to evaluate the familial aggregation of insanity and then utilizes the method in an extended family, part of which is contained in pedigree 4. He showed an approximately 10-fold concentration of insanity in that extended pedigree compared to that seen in the population of the parish within which they live. The next controlled study of the familial aggregation of insanity was not published for over 35 years (Kendler & Klee, 2020; Koller, 1895).

Dahl does not follow current confidentiality guidelines in providing names of individuals in his pedigrees. However, this does not raise current ethical concerns given that the book was published over 160 years ago, and has been available to scholars during this entire time period.

Finally, how well known is Dahl's study? Its importance was recognized by Gerhard von dem Busch, a German physician and translator who published, in 1864, a detailed report of Dahl's work, with a complete translation into German of his genetics section (Busch, 1864). However, no reference to Dahl's work was found in the next pedigree study of psychiatric illness a decade later in France (Doutrebente, 1869), nor was it referenced in the first classical case control study of HP to psychiatric illness conducted by Koller in 1895 (Koller, 1895). More remarkably still, as noted above, in Gausemeier's, 2015 review "Pedigrees of madness: the study of heredity in nineteenth and early twentieth century psychiatry," Dahl's study is not mentioned. A search in Google Scholar focused on Dahl's genetic studies uncovers multiple references of Dahl's work by Porter from 2018 onward and one reference that refers to his studies on the genetics of idiocy published in 1877 (Ireland, 1877). We also located a reference to this work in a 1913 history of Forensic Psychiatry in Norway (Winge, 1913) and in a 2003 thesis on the history of mental illness in the late 19th century in Norway (Skålevåg, 2003). We can conclude with some confidence that Dahl's innovative work in psychiatric genetics has, over the more than 150 years since its publication, been poorly recognized and infrequently cited.

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CONFLICT OF INTEREST

The author reports no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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