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CHARLES DAVENPORT AND EPILEPSY AS A SINGLE GENE DISORDER

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ABSTRACT

Charles Davenport's genetic studies had eugenic biases. His work on epilepsy seems particularly questionable, with one gene underlying all cases, even with trauma, and also causing social pathologies. However, he was replacing a medical model with Mendelism, with findings well within the mainstream when he wrote, and for decades after. Physicians had seen heredity as a transmitted factor that stabilized and controlled anatomy and physiology. Diatheses, tendencies towards diseases, were part of heredity, and interacted with other influences, giving transmitted changes. Epilepsy was one of many possibilities reflecting a nervous system diathesis- *neuropathic heredity*, a weakness, or taint. Multiple problems reflected *polymorphism*, "a unitary something in the neuropathic or psychopathic inheritance that makes itself manifest under many forms" (Myerson 1925: 271). Once heredity weakened, it was subject to further defects via *degenerate heredity*. Medical views of diseases with multiple contributing causes complimented and supported this. Here, neuropathic heredity predisposed to epilepsy and other findings, none inevitable, accounting for all cases, even with trauma, well past mid-century! Ultimately, this was standard medicine where Davenport essentially used a single gene- in a sense, a progressive attempt to provide genetic justifications for medical findings. It has been suggested that facts are constructed by professional communities, rather than discovered, which we see here. For genetics to be used effectively, new facts had to be constructed and older facts that reflected experience based medical beliefs eliminated. This took decades to accomplish, and much was never disproved, but became irrelevant and forgotten.

KEYWORDS: Charles Davenport, Degeneration, Diathesis, Epilepsy, Eugenics, Heredity, Mendelism, Neuropathic Heredity, Polymorphism.

INTRODUCTION

Charles Davenport, the guiding hand behind American eugenics, is something of a poster child for abuses of science (Kevles 1985). His work often seems hard to credit, as with his attribution of complex psychological and neurological issues to single Mendelian genes, e.g., thalassophilia (love of the sea), as a recessive sex limited condition that explained why men, not women, ran off to sea, and a necessary condition for success as a naval officer (1919).

His conclusions on the inheritance of epilepsy seem especially shaky, attributing all cases, even with trauma, to a single gene with multiple effects- listing alcoholic, blind, crippled, criminalistic, chorea, deaf, dwarf, eccentric, heart disease, tubercular, and vagrant, among others, in pedigrees, and a particular connection to feeble-mindedness (1911: 73-5).

However, despite a deficient methodology (Kendler 2022), his conclusions were actually consistent with mainstream medical science. Most work on this topic came from physicians, and Davenport's phenotypic analysis reflected standard medical views, not only at the time, but for decades after. Here, early medical ideas about the inheritance of epilepsy are reviewed to provide a context for his analysis.

Medical Background. Epilepsy is a tendency towards seizures: Classically, sudden uncontrollable shaking of the limbs, with loss of consciousness. There are many causes and types, inborn and acquired. Idiopathic cases, with no apparent cause, are common, and about 3% of all people have the diagnosis at some point in their lives (Hauser, Annegers, Kurland 1993). Today, we see it as a general symptom, rather than a specific disease.

Traditional Approaches: The Greeks originally thought that epilepsy came from the gods, but a Hippocratic text, *On the Sacred Disease*, argued for a natural origin from excess phlegmatic humors. This made seizures amenable to study,

and even intervention. Also, since the Greeks generally saw all natural disorders as hereditary in some sense, it raised the same possibility for seizures.

Despite lay concerns over demonic possession in the Christian era, physicians continued to accept a natural process, including a role for heredity (Temkin 1945: 3, 51, 131). Boerhaave (1678-1738), one of the great Dutch doctors, noted one cause as "hereditary, from a family taint of the father, mother, relations or ancestors; the disease frequently lying dormant in the father, while it is derived from the grandfather to the grandchild." Similarly, in 1770, Tissot saw an hereditary nature, and felt that epileptics should not marry (Temkin 1945: 219). A few downplayed inheritance: In 1719 one anonymous author saw it "chiefly occasioned by some posterous accident," and two studies found "hereditary taint" in only (!) about 10% of cases.

Still, as the 19th century progressed, epilepsy was increasingly seen as "pre-eminently an hereditary affliction" (Reynolds 1861: 123-4), which makes little sense in modern terms: Most causes- head injury, stroke, brain tumors, etc. -are not genetic, and the overall risk to relatives, while increased, is still low (Baraitser 1985: 67-8). Why, then, did physicians feel that heredity was so important?

The answer lies in a medical system with non-specific connections between what we now call genotype and phenotype, a system very different from genetics. Instead of distinct unit traits, there were *diatheses*, general (and mutable) predispositions that could act throughout life (Hutchinson, 1884)- even today, poor blood clotting is described as a bleeding diathesis (Saes et al. 2019). For epilepsy, one such tendency, *neuropathic heredity*, was especially important. But it wasn't linked to epilepsy alone- it involved *polymorphism*, "a unitary something in the neuropathic or psychopathic inheritance that makes itself manifest under many forms" (Myerson 1925: 271), with variably defined combinations of epilepsy, insanity, mental retardation and other abnormalities. The "taint" of neuropathic heredity could also segue into *degenerate heredity*, where the range of problems and their severity could worsen over time, or with transmission (Lubinsky 1993).

The effects of that portion of heredity responsible for seizures were expanded from the 1850s on. Before then, some had limited it to a predisposition to epilepsy alone, but others included additional findings, ranging from alcoholism to typhoid fever and phthisis (a form of tuberculosis). By the 1880s, opinion clearly favored a multitude of possibilities (Dowbiggin 1985), and, as we shall see, more were added over time.

None of these findings, including epilepsy, were inevitable when the taint was present. This meant that heredity could be an important cause of convulsions even though seizures were infrequent manifestations- just as we would consider head trauma an important cause, even though most such injuries do not result in seizures. And here, neuropathic heredity predisposed to multiple problems. We can see some of these implications by substituting head injuries for inheritance as a cause. Such injuries can have a variety of outcomes: 1. No findings; 2. Seizures; 3. Seizures *and* other findings (paralysis, coma, mental difficulties, etc); 4. Other findings without seizures. While injuries are an important cause of epilepsy, they seldom result in seizures, and are more likely to show other outcomes.

In this context, alcoholism in the grandfather of an epileptic was evidence of neuropathic heredity. Using different findings, and different relatives, studies gave inconstant findings: 31 of 110 epileptics with ancestors and/or children with epilepsy, insanity, imbecility and hysteria in 1825; 7 of 106 with similarly affected relatives, plus other brain diseases in 8 in 1843; Practically all hereditary, based on relatives with any sort of nervous problem, or of phthisis in 1854 (Temkin 1945: 250-1); 30% hereditary, either with fits or "nervous derangements" (Reynolds 1861: 123).

In his classic monograph on epilepsy (1881), Sir William Gowers, one of neurology's greats, saw "few diseases in the production of which inheritance has greater influence." The same factor could also cause "insanity…, chorea [a movement disorder], hysteria, and some forms of disease of the spinal cord. Intemperance is probably also due, in many cases, to a neuropathic disposition, but is so common among the poor that its existence can hardly be taken as evidence of disease." In 1,218 epileptics, he found neuropathic inheritance in 35%, slightly higher than in two smaller contemporary studies with 31% and 28%.

While Gowers saw neuropathic heredity as casting a wide net, he rejected "heterogenous heredity," which extended findings further, particularly to rheumatism and phthisis (p. 8). There were other limits also, for "we cannot regard all forms of disease of the central nervous system as evidence of its existence. Many... are the result of morbid states… far removed from the nervous system. Thus we cannot regard apoplexy [stroke] and hemiplegia [a sign of brain damage] as related to epilepsy." So, for Gowers, damage that secondarily caused seizures didn't involve neuropathic heredity, a caution that others later rejected.

By 1900, "the enormous influence of hereditary factors [in producing epilepsy was]... hardly questioned..., usually there is... a direct or indirect dissimilar heredity, i.e., in the direct ancestors or in a collateral line of ascendants there are other affections, which also develop upon the foundations of a neuropsychiatric predisposition, namely neuroses and psychoses of the most varied kind" (Vorkastner 1909).

But, as a predisposition, heredity left a role for other causative factors, such as "chronic intoxications and chronic diseases of the germ plasma." Alcoholism in ancestors occurred "with extraordinary frequency. Formerly

to acute alcoholic intoxication was ascribed a deleterious influence upon the germ plasm, and etiologic importance was attached to procreation under the influence of liquor. Recently emphasis is laid upon the point that neurotic and psychopathic individuals readily tend toward alcoholic excesses, also that they are intolerant of alcohol and in the condition of alcoholic excess show a special tendency to sexual activity. Among other chronic intoxications there are… chronic lead poisoning and morphinism [morphine addiction]; among chronic diseases, syphilis, tuberculosis, arthritis deformans, malaria, diseases of the blood, gout and diabetes… [The last two] also regarded by some authors as the expression of the neuropathic predisposition. Among the causes of acquired inferiority of the germ plasma... [is] advanced age of the parents, especially of the mother" (Vorkastner 1909). Consanguinity also had an influence, as it "so vitiated the general stamina as to induce epilepsy" (Spratling 1910).

Still, there was one primary cause, "a hereditary, constitutional, neuropathic predisposition upon the foundation of which epilepsy develops. Any damage [as with trauma]... is sufficient to set the ball rolling" (Vorkastner 1909). George Still, a founder of modern pediatrics, noted "how largely epilepsy is an inborn vice. The exciting cause is but the spark that fires the train. The necessary antecedent is the congenital tendency, for which in a large proportion of the cases heredity is responsible; not necessarily the inheritance of an epileptic strain, but often only of a neuropathic taint, which in the child's parents or near relatives may have shown itself as insanity or alcoholism, or in less pronounced degree as asthma, migraine, or neurosis of one kind or another... The epileptic in fact is, in the majority of cases, an epileptic from birth, albeit the first manifestations of his morbid tendency may be delayed for months or years" (1915: 670).

We see this basically unchanged decades later in Bing's authoritative neurology text: "Most patients who have had severe cranial trauma do not develop epilepsy. That heredity plays an important role is evident in the statistics of Cobb. He found in relatives of nonepileptic persons an incidence of 2.6 per thousand and in relatives of traumatic epileptics, 14 per thousand. According to F. Ryan, almost 60 per cent of those developing post traumatic epilepsy are predisposed to epilepsy on an hereditary basis. In many of these there is other evidence that 'the field for epilepsy is prepared.' Thus, it has been pointed out... that developmental disturbances are relatively common in these individuals" (1939: 708).

As part of this, epileptics had "an easily impressionable nervous system. They possess a fervour or zeal, a susceptibility which influences their character, and this is familiar to every observer... [They] are sensitive, suspicious and irritable... Most... undergo some mental changes in consequence of their epilepsy, and two types result, either the irritable, distrustful, sober and resentful, or the shallow, obsequious and the effusive. The general tendency, however, of repeated epilepsy is towards mental enfeeblement ending in dementia" (Jones 1912).

There was also a connection with madness and genius, "a matter of common experience, that in families in which insanity is hereditary we meet with examples of the greatest intelligence, and with epileptics and imbeciles" (Wagner 1876: 41). So, high intelligence, "sometimes the highest, is said to be associated… [Epilepsy] implies an uneven and irregular discharge of nervous power. A certain instability is necessary to nervous action, and genius may co-exist with morbid instability" (Jones 1912). Later, in a chapter on "Talent and Psychopathy" in a book on human heredity, Lenz stated that "morbidity is... one of the accompaniments of the conditions that have hitherto cooperated to give rise to genius" (1931: 621), and that a form of epilepsy could be part of the spectrum.

There was even an epileptic psychosis, which some felt was so distinct that it could be used to diagnose epilepsy before the onset of seizures. Despite increasing skepticism (Bruens 1974: 593), even in 1974, "the vitality of these concepts is attested by their still being in current use… many authors describe a characteristic irritability... with sudden and unpredictable variations in mood: they are suspicious, quarrelsome, egocentric, circumstantial, egocentric, religious... [plus] a slowness and stickiness of thought that borders onto mental subnormality" (Pond 1974: 580-81). Some continued to believe that the epileptic personality could be seen without seizures in relatives of epileptics (p. 581).

So, in the early 20th century, physicians generally accepted a strong role for heredity in the origins of epilepsy. Gowers's 35% was often quoted, but even higher figures were given: Over half (Vorkastner 1909), or 60 to 65% (although only 16% from parent to child) (Spratling 1910).

These studies typically invoked indirect effects and evidence. Osler was "surprised to find in the list of my cases that hereditary influences played so small a part." But, accepting current wisdom, he added that "it may be said that direct inheritance [parent to child] is comparatively uncommon, yet the children of neurotic families in which neuralgia, insanity, and hysteria prevail are more liable to fall victims to this disease" (1905: 1059).

Others were more skeptical. Sachs saw attributions to heredity as falsely high (1895: 70), and Jelliffe and White were highly doubtful (1917: 30). Myerson wrote a particularly detailed critique (1925: Chapt. 8), with "harsh criticism… more against [Davenport's] medical collaborators than against [Davenport]" (p. 65).

Still, support was well entrenched, often with the same reasoning in the 20th century as in the nineteenth. For example, Burr saw increased insanity, criminality, chorea and alcoholism in relatives of epileptics, but little parent to child transmission, suggesting an indirect and general effect

of heredity (1922). For Thom and Walker, an inherited lack of normal nervous system stability caused mental deficiency, insanity, a variety of neuro-psychological conditions, and seizures from a variety of factors that would not have affected a normal system (1922). Russell Brain, an eminent neurologist, studied 200 epileptics and saw the disease in other family members in 28%, indicating an inherited predisposition. (1926).

In the 1930s, a new source of data appeared: the electroencephalogram (EEG), which could show abnormal electrical brain activity with epilepsy. Clinical findings could now be correlated with electrical ones, and abnormal activity recognized even without actual physical seizures, opening up whole new avenues of investigation.

This included brain wave studies of relatives of epileptics. Löwenbach (1939) found abnormalities in 17 of 37 clinically normal parents and siblings; Robinson (1940) 36%, with 27% questionably normal. Lennox, Gibbs and Gibbs, in the most extensive work, saw findings in 54% of parents and 6% of controls. When both parents were studied, at least one was affected in 94% of families. A larger study showed similar findings, and 70% concordance for epilepsy in identical twins, with the same pattern, normal or abnormal, even when one had trauma induced epilepsy! Conrad found 66.6% concordance in identical, and 3.1% in non-identical twins (cited by Kallman 1953: 189).

This again supported a single factor underlying epilepsy. A 1941 Lancet editorial summarized new data, so that "in the majority of cases there is an interplay of an inherited factor with some local want of development, trauma, or disease of the brain." EEG dysrhythmia was "more frequent among the relatives of females than of male patients and among the female relatives than the males. The heritable factor is therefore stronger in the female." Overall, "epilepsy is usually regarded as a Mendelian recessive trait, but this widespread dysrhythm looks more like a dominant." There was even speculation that gene carriers "might show mutual attraction of similar personalities." Lennox ultimately felt that his data supported a single recessive with incomplete penetrance, although he could not rule out other mechanisms. He also found that 23.9% of epileptics had a family history of migraine, substantially greater than in controls, suggesting a common constitutional and genetic basis for both (Lennox, Lennox 1960: 573).

Some types of epilepsy are due to single genes, like the Baltic myoclonic epilepsy studied by Lundborg, described as a recessive early on (OMIM #254800). But even here, there was still support for older ideas- Lundborg also found "other pathological conditions, including nervous and mental diseases, epilepsy, weak mentality, idiocy, and debility," and concluded that "the tendency to degenerative diseases is due to bad racial quality in this part of Sweden, to much inbreeding... and to the strong use of alcohol. The high birth-rate and the excess of coffee drunk were also regarded as unfavourable forces" (Gates 1929: 251-2).

In fact, even as Mendelism was cited in EEG studies, older approaches persisted. This is nicely seen in some papers (1934-1943) by Paskind and Brown at Northwestern University. They were thoughtful physicians who wanted to dispel myths about high rates of serious problems with epilepsy, especially non-institutionalized patients, and published in first rate journals: The Archives of Neurology and Psychiatry, the Journal of the American Medical Association, and the American Journal of Psychiatry.

Pashkind noted a "special relationship" of epilepsy and migraine going back to 1779, with a dozen later supporting opinions, plus a few doubters (1934). Specific studies were rare, but both Ely and Cobb found overlaps, and increased migraine in relatives of epileptics. Paskind himself had two controls: *Non-neurological*, and *Neurological*, with various disorders: Manic-depressive psychosis, trigeminal neuralgia [a form of nerve pain], psychasthenia [nervous exhaustion], schizophrenia, tic, constitutional inferiority, and paranoia. Epileptics and their families had more migraines than standard controls, but there were similar or higher rates in many of the other disorders, suggesting no special or specific relationship between migraine and epilepsy.

In 1936, Paskind and Brown found parent to child transmission of epilepsy from 0.8% to over 50% in 21 studies from 1826 to 1933, but felt most were biased by concentrating on "deteriorated" institutionalized patients. In their own series of 342 children of epileptic patients in a private practice, only one also had the disorder.

The next year, they studied neuropathic heredity in deteriorated and non-deteriorated patients. From 1858 to 1928, neuropathic heredity was seen in 11.1 to 81.2% of epileptics. In their own study of 331 out-patients with normal mentality and no focal neurological signs, 58% had evidence of hereditary taint, 41% in parents, 10.4% in grandparents, uncles and aunts, and 6.6% of siblings. Findings were psychoses, nervous disorders such as epilepsy, migraine and nervousness, alcoholism, stroke, senility, psychopathic personality, and suicide. Comparison with other studies showed a higher familial incidence of epilepsy, alcoholism and insanity in institutionalized patients, and the opposite for migraine and nervousness. They concluded that "the parents of the more malignant, deteriorated, institutionalized patients are much more burdened with neuropathy" (Paskind and Brown 1937).

They also compared physical findings of neuropathy in the groups, starting with stigma of degeneracy (1936a), primitive structures reflecting problems with development. Quoting Turner, with whom most authors agreed, from 1907: "These point to a latent neuropathic disposition, which may exert a potent influence upon the causation, type of course and treatment of nervous and mental disease. They are of immense value as an index of the intensity or degree of the hereditary degenerative disposition. In the

most pronounced forms of mental deficiency… anatomical variations from the normal are common and often of a pronounced type. In the slighter forms of neuroses… the stigmata are less frequent and less pronounced." They then compared physical measurements (1939), finger prints (1940), handwriting (reflecting early cerebral writing center development) (1940a), and finger nail fold capillaries (1943). Significant differences in all cases supported inborn constitutional issues, so susceptibility to deteriorative epilepsy reflected a larger problem. While not necessarily hereditary by their studies, it still arose very early in development.

Negative stereotypes about epilepsy, especially a tendency towards deterioration, were ongoing issues. Grinker and Bucy felt it necessary to distinguish the vast majority of epileptics who did well from a mostly institutionalized minority with complications and deterioration. They emphasized that either lack of seizure control, or associated cerebral diseases, and not seizures *per se*, gave the so-called epileptic personality and facies, "characterized by a rigid egocentric, selfish, seclusive personality with explosive outbursts. The typical facial appearance is a heavy dull expression without much emotional lability" (1949: 856-7).

In 1952, Ford's authoritative pediatric neurology text, summing up heredity in epilepsy, noted a traditional "*constitutional tendency* to convulsive reactions which is regarded as an inherited peculiarity, and a number of *precipitating factors* such as acquired injuries and diseases of the brain. Morbid heredity has always been emphasized, although there is a great deal of difference of opinion about its importance... There seems to be little doubt that there is an inherited factor of importance… [I]diocy, insanity and various types of neuropathic personality are commonly found among the ascendants of epileptics and on the basis of such observations it has been said that although epilepsy is rarely inherited directly from the parents, it is none the less the outgrowth of a neuropathic stock... It is probable that [EEG] dysrhythmia is an expression of the constitutional factor... Most cerebral injuries are not followed by convulsions no matter where the lesion is placed or how severe it may be and it is for this reason that the underlying constitutional factor had been postulated. Cobb regards the various cerebral injuries as capable of inducing epilepsy only in those in whom there is a latent tendency to the disease" (pp. 1026-7).

These concepts were remarkably resilient. In 1975, in a book compiled by the Epilepsy Foundation of America, language and conclusions are little changed from decades before. So, "those who accept the role of heredity outnumber those who see heredity as unimportant in susceptibility... [The former] state that 'without doubt there is a hereditary constitutional factor in epilepsy as there is in almost all other diseases'" (p. 25). And "Lennox [1960] points out that... all kinds of epilepsy have a common factor, diathesis or the predisposition to seizure" (p. 17).

The consensus was still that genetic factors controlled susceptibility, with one or a few genes accounting for most epilepsy, although the nature of that gene was unclear, with the following citations: Age dependent autosomal, dominant genes- 1965; simple autosomal dominant- 1969, recessive- 1969, and sex-linkage- 1971! Also, in 1969, two different genes or polygenic systems- one for a very low convulsive threshold, the other an isolated predisposition, with low penetrance. Possible mechanisms included a developmental defect of neuroectoderm, a problem with the tissue that gives rise to the nervous system that would certainly allow for other neurological findings (Epilepsy Foundation of America 1975).

DISCUSSION

Overall, eugenics was a social movement that attempted to draw legitimacy from science, and often abused it as well. However, Davenport's work on the inheritance of epilepsy, which seems at first like a classic example of bias, actually suggests a far more complex picture.

He basically saw a single factor accounting for seizures. It didn't quite cause everything- epilepsy, hysteria, and mania were separate from a dominant factor for violent temper, although they could facilitate its expression (1915)- but seemed to come close. He actually had some support from other geneticists: Castle, a leading Mendelian, felt that this stood up to analysis, with epilepsy and feeble-mindedness "merely different manifestations due to a single cause" (1916: 249-53), while Little, another basic scientist, also accepted this relationship (1923).

Still, his data gathering methods had very real problems (Myerson 1925: 64-7). Medical studies, with varied definitions of relatives and heredity, were often worse, with the same second-hand information, and the same penchants for bias.

But here, Davenport had two sources of validation. One was Mendelism, where he was a pioneer- despite interest in heredity and epilepsy, medical uses of genetics lagged, and Myerson listed only one "Mendelian" in 1925: Charles Davenport (pp. 56-72). His attributions of essentially everything inherited to Mendelian mechanisms was extreme, but hardly out of line with the polarization that characterized early genetics-biometry debates (Carlson 1966: 9-13). A single gene was also far from radical: In 1960, EEG pioneers felt that their data did not exclude a single recessive with incomplete penetrance (Lennox, Lennox 1960: 573). Even in 1975, the consensus was still that one or a few genes accounted for most epilepsy (Epilepsy Foundation of America).

The second source of support was medical. Davenport was well within the mainstream in terms of his methods, data, not only for when he wrote, but for many decades after. Neuropathic heredity was *the* cause of epilepsy, and Davenport basically gave this a genetic origin.

Here, he asked whether external "causes are sufficient and exist... A fall on the ice may result in a child's first epileptic fit but thousands fall on the ice without more than temporary discomfort; it was not the fall merely but the fall plus the too delicate nervous organization" (1911: 72). But this just reflected the medical literature, as we have seen. Again: "Epilepsy is an inborn vice. The exciting cause is but the spark that fires the train" (Still 1915: 670); "there is an interplay of an inherited factor with some local want of development, trauma, or disease of the brain" (Lancet editorial 1941) Or, Cobb's view of "injuries as capable of inducing epilepsy only in those in whom there is a latent tendency to the disease" (Ford 1952: 1026-7).

There were also the typical diverse effects of the neuropathic taint, as we saw in authors ranging from Gowers to Paskind and Brown. So, "Mendelism in Davenport's work is merely polymorphism of the widest type" (Myerson et al. 1936: 137).

Davenport's "facts" were mainstream, and respected researchers continued to try to explain them genetically. Unfortunately, they reflected medical ideas of heredity as variable, and diseases as mutable entities, concepts that fit poorly into Mendelian analyses based on stable traits uniquely linked to discrete unit factors.

Integrating genetics into medicine was more than just applying a new theory to old data. Kuhn (1970) felt that facts are constructed by professional communities, rather than discovered, and we see this with epilepsy. To use genetics, new facts had to be constructed, and old facts rejected. However, the old "facts" reflected ideas and experiences integral to medicine, and took decades to change. Interestingly, much that didn't fit with genetics (e.g., the work of Paskind and Brown) was never disproved, but became irrelevant and, ultimately, forgotten and ignored.

Generally, the scientific justifications of eugenics are given little credence today. Certainly Davenport's methodology was deficient (Kendler 2022), and arguments based on degeneration are definitely rejected now, but medical studies of the inheritance of epilepsy up to his time indicate that his phenotypic conclusions were, in fact, mainstream. We also see single gene inheritance supported by others during the first half of the 20th century. However, because these approaches were rooted in medical ideas that have been superseded, they are easily overlooked. Not that medicine was bias free but, by the standards of the day, this was "good" science, and well accepted.

It is easy to make eugenics into a morality play of bad science and scientists, and there is much to criticize in Davenport's work (Kevles 1985; Kendler 2022). However, even good science may not be correct. Perhaps the lesson here is that scientific legitimacy is no guarantee against misuse or harm, and that applications to complicated social issues need to be done with caution and humility.

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