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Review Article

Infectious Diseases And Traditional Medical Ideas About Inheritance. I. General Considerations.

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Running head: Infection and Heredity

Abstract

Today, infection and inheritance are generally seen as medical opposites- environment vs. heredity. However, they actually influenced and supported each other well into the 20th century. Which category disorders belonged to wasn't always clear, and the same rules could apply to both: Diseases could need both "seed and soil," making them infectious and hereditary. Infections supported a medical alternative to genetics with variable traits with interacting causes, a view of what was inherited that undermined recognition of the distinct unit traits of Mendelian inheritance. This had four basic components that also applied to infectious diseases.: 1. Heredity as a force; 2. Degeneration, an often progressive weakening of that force; 3. Diatheses (predispositions); and 4. Polymorphism- variable entities that could transform into one another. Successes against infectious diseases changed medical experiences, undermining non-Mendelian ideas. With this, genetic concepts became increasingly dominant in medicine, and separate from infections.

Keywords : *Heredity, Infection, Degeneration, Diathesis, Polymorphism.*

INTRODUCTION

Medically, infection and inheritance seem like opposite ends of a spectrum, "Huntington disease at one end, tuberculosis at the other" (Lindee 2002), so that a focus on one detracts from the other: Neel (1976) felt that more treatable infections (and deficiency diseases) distracted physicians from genetic disorders, and Rushton saw infections as over-shadowing genetics, especially after 1910 (1994: 134). For Kevles, doctors "perceived no value in genetic knowledge for the treatment of disease; if a malady was hereditary, the prevailing medical attitude had it, it must be neither treatable nor preventable" (1985: 177).

Actually, ideas about infection and inheritance influenced and supported each other well into the 20th century, and the same rules could apply to both. Causes were unclear, and a disease could have infectious and inherited etiologies, needing both "seed and soil." Infections also traditionally emphasized variable findings and interacting causes, undermining recognition of the distinct unit traits of modern genetics, and supporting non-Mendelian medical alternatives.

Analyses here include conditions seen as infectious or hereditary now or in the past, even if mistaken. Information about specific genetic disorders is referenced in OMIM (2024).

THE MISSING DICHOTOMY

Modern medical approaches to infection began in 1864, with Lister's ideas on antisepsis, but in a context very different from today's (Cochran et al. 2000). Neel's categories- infection, deficiency, genetic (1976)- weren't well defined, and were often problematic (Mendelsohn 2005), e.g., for leprosy, contagion, diet, heredity, and sanitation, were all suggested, all widely and variously defined! There was enough of a case for heredity in 1848 for Norway to considered compulsory sterilization and, in 1895, Hansen, who discovered the bacillus, saw "hardly anything on earth, or between it and heaven, which has not been regarded as the cause of leprosy" (Pandaya 1998), including "climatic influences, unwholesome and putrid food, want of salt, a fish diet, malaria, heredity, contagion, syphilis and insanitation" (Tebb 1893, Chapt. 3). Aycock still argued for the primacy of inherited susceptibility in 1941, as "doubt has been thrown upon the belief that contagion is the major determinant."

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There were also complex interactions. For Friedreich ataxia, first seen as combining heredity and syphilis (Friedreich 1877), "often the first symptoms of the disease have been observed after some acute infection… Starr is even of the opinion that in hereditary ataxia there is less of a congenital lack of development than an affection of the entire nervous system appearing in connection with an infectious disease… We may assume that the tracts which serve coördination are especially exposed to the toxic actions of the acute infections… [if] an infectious disease (enteric fever) develops, the course and severity of the clinical picture are influenced in an exceedingly unfavorable manner" (Lüthje 1907).

So, "what we know of the conditions which cause internal diseases does not, for the most part, relate to causes as defined within the discipline of logic, in other words causae sufficientes, causes which always and alone produce the effect, but to complex conditions, under whose influence, sometimes frequently, sometimes rarely, diseases become manifest" (Uhle and Wagner 1913). These factors were particularly likely to be apply to infections.

And here, heredity and infection were often combined, "that in all infections there are two factors, the exogenic virus and soil in which it grows" (Bulloch 1909: 19). So, "it is misleading to assert that the tubercule bacillus is the cause of tuberculosis, and many prefer to say that the entry into, and multiplication of tubercule bacilli in a predisposed body is the cause of the disease… the two factors, the soil and the seed, the external and the internal, must both be taken into account if we are to form any adequate conception of the nature and causation of any disease" (Faber 1930: 38). And that soil typically had an inherited component.

So, pellagra [a vitamin B3 deficiency disease] was "probably communicable, but just how the communicable germ of this disease shall progress in the body depends in part on constitutional factors," which could be inherited (Little 1923: 234). For goiter and cretinism (an enlarged thyroid with growth and mental retardation, typically from iodine deficiency), "hard" water was long suspected, as well as infection (Lancet 1911), with a marked "influence of heredity in the development of the disease… Transmission of the disease through the drinking water is a thoroughly established fact" (Janney 1922: 457-8). Such models probably delayed recognition of its true nature for over forty years (Mellanby 1937).

Other erroneous attributions abounded, e.g., Fibiger won a 1926 Nobel Prize for "proving" that a parasite caused cancer (Stolt et al. 2004), alkaptonuria, a recessive metabolic disorder, was first seen as "a special form of infection of the alimentary tract" (Garrod 1901) and multiple affected family members suggested that breast cancer was infectious (Donegan 2002).

In short, an infection-heredity dichotomy was far from obvious as doctors saw complex etiologies, with heredity, infection and other factors all involved in a single disorder. In this situation, infections drew medical attention to heredity, not away from it.

Inheritance in Medicine

Interactions were supported by ideas about inheritance antithetical to modern genetics, with a force transmitted across generations that controlled both anatomy and lifelong physiology. This arose from the work of Blumenbach, a famed German physician-anthropologist, who proposed a formative force (*Bildungstrieb*, or *nisus formativus*) in 1781. This "initially bestows on creatures their form, then preserves it, and, if they become injured, where possible restores their form… It shows itself to be one of the first causes of all generation, nutrition, and reproduction" (Richards 2000). Here, heredity referred to the force *and* to its function, making transmission, development, and physiology inseparable.

With this, heredity and variation were opposites (Brooks 1899: 75), inheritance continuity, "merely a form of growth" (Wilson 1900: 398): Acorns made oaks just as oaks made oak leaves. Variation was a "partial failure of heredity" (Fisher 1911: 50) extending to degeneration, a weakened inherited force vulnerable to further deterioration (Lubinsky 1993).

Heredity included diatheses, predispositions that, under different names, became units of inheritance that could manifest as specific constitutions, or as idiosyncrasies (Solis-Cohen 1894). They interacted with internal and external factors, including other diatheses, giving inherited changes (Hutchinson 1884). Boundaries were uncertain, and polymorphism "a unitary something… that makes itself manifest under many forms" (Myerson 1925: 271) explained variations and transformations of what we now call the phenotype.

Applications to Infections

The doctrines that governed inheritance- heredity, degeneration, diatheses, and polymorphism- also applied to infectious diseases.

Polymorphism was a prime source of confusion. In 1852, Cazenave and Schedel felt that "we cannot tell why the existing cause should in one case produce a pustule, in another a vesicle, in a third a papule" (pp. 22-3), and that, for a hereditary tendency, "a predisposing cause of much importance… it by no means follows that the same disease must be passed down from father to son: thus the parent may have had a scaly affection of the skin, and the children be attacked by a pustular or vesicular one" (p. 19).

In the early Lister era, Paget, an influential surgeon, saw "transformation of specific diseases… in their transference from one person to another, whether by inheritance, or infection or contagion. [Note: Toxins, such as poison ivy, could be transferred by contact, giving contagion *without* infection (Cochran et al. 2000)]. A parent with one form of secondary

syphilis may have a child with another form, the child of a parent with scirrhous cancer may have an epithelial, a colloid, or a medullary cancer: the inoculation of several persons with the matter from one primary syphilitic sore may produce somewhat different forms of the primary disease and very different consecutive phenomena; the same contagion of small-pox, measles, or scarlet-fever, may produce in different subjects all the modifications of which these diseases are severally capable; the puerperal [laboring] woman, or the patient who has sustained a severe accidental or surgical injury, may modify, or as it were, colour with the peculiarities of her own condition whatever epidemic or other zymotic [infectious] disease she may incur" (1870: 63).

Hutchinson, another famed Victorian surgeon, noted "hereditary susceptibility to special forms of disease, resulting either from contagion or some other external influences... [and that] exanthema [general diseases with skin findings, like measles], during the same epidemic, are apt to affect some families with greater severity than others; and that sometimes in the same family several individuals may suffer very severely and others very lightly, the difference probably being due to inherited peculiarity of constitution" (1890-91: 259).

Despite beliefs in polymorphism, it could be difficult to link different manifestations to a single cause- in 1880, Pasteur's finding that one bacterium caused boils *and* bone infections [osteomyelitis] "was greeted with silence by the more polite, so absurd did it seem" (Farber 1930: 99).

There was also blastophoria, "a large class of diseases in which poisons or infections during prenatal life induce feeble or neuropathic constitutions, which make the descendant liable to acquire the parental disease. Such diseases are tuberculosis and syphilis" (Barker 1925). Or, as Batten, an illustrious neurologist, noted for spinal muscular atrophy (a recessive muscle problem) in 1911, "evidence pointing... to a toxic agent. On the other hand, the occurrence of the disease in families points to... 'a defect of vital endurance.'"

Pearl, an influential biostatistician, opposed "the wild statements now being made in the medical press that the whole problem of phthisis was one of infection" (1920: 296-7), and emphasized constitutional properties (1933). Later, Myrianthopoulos (1956) felt that "the role which heredity plays in constitutional predisposition to infectious diseases is becoming all the more important in the light of recent research work in the fields of immunology and epidemiology... We have evidence that predisposition to at least some infectious diseases is indeed inherited." Some of this was Mendelian- a 1941 lecture cited scarlet fever, diphtheria and tuberculosis as recessive susceptibilities (Snyder: 83-4). And, while inherited susceptibilities are accepted now, diatheses and degeneration give a strange cast to earlier arguments.

The mutations publicized by deVries (1901-3) emphasized hereditary alterations, and evolutionary theory supported change as conditions varied, which some doctors saw as an important lesson (Collins 1920).

Infections also pointed to changes in heredity. In the 1880s, bacteria came to be seen as having separate species, but with variability that could become hereditary. Vaccines showed that bacteria could be made less virulent, and there were often shifts in culture from one form to another (Mendelsohn 2005)- *Escherichia coli*, a common intestinal bacteria, had *mutabile* added to its name for this reason (Brock 1990: 50-53). New disease strains appeared, and the 1918-9 influenza pandemic, 25 times more deadly than any before, and killing tens of millions (Kolata 1999), was a powerful example.

But, if bacteria could change, why not humans? For Castle, a genetics pioneer, since heat or chemicals could decrease the virulence of disease germs, "the same possibility may exist in the higher animals and plants, provided agencies capable of producing change are allowed to act on the germinal substance. It is the sheltered position of the germ-cells which seems ordinarily to exempt them from direct modification, but we cannot safely assume that they are in all cases free from such modification" (1916: 45).

With these issues, acceptance of Mendelism was delayed as alternatives supported by classic medical ideas and experience opposed genetics. The new science might explain the inheritance of a few rare disorders, but it left a wide range of common observations apparently unexplained. We can see more of this with specific disorders (Lubinsky 2024).

A New Era

Ideas changed as medicine evolved. Better sanitation, nutrition, and care tamed the childhood illness that had been routine killers. The advent of antibiotics in the 1940s reduced the incidences of TB, syphilis, and rheumatic fever, and pneumococcal pneumonia, a common acute killer, became easily curable. Vaccinations ended whooping cough and diphtheria epidemics, and the polio wards vanished.

Survival in inherited conditions increased when infections affected mortality. For Wiskott-Aldrich syndrome, an X-linked immune problem, it went from 8 months before 1935 to 6.5 years after 1964 (Perry et al. 1980), and a variety of inherited immune disorders became apparent in the 1950s: Familial hemophagocytic lymphohistiocytosis and Bruton agammaglobulinemia in 1952, congenital neutropenia in 1956, chronic granulomatous disease in 1957, severe combined immunodeficiency in 1958, and Hermansky Pudlak syndrome in 1959 (OMIM 2024). Laboratory advances played a role, but without decreases in "routine" infections and infant deaths, observations of rare genetic disorders would have been generally obscured.

With this, medical awareness of genetic disorders increased as infections became less of a model of complexity and of "seed

and soil." So, the form of TB- scrofula, intestinal, spinal, lung, etc. -no longer mattered, and age, sex, physique, and history became irrelevant: Everybody got streptomycin. Similarly, the complex typing of pneumococcus strains for serum therapy (which strengthened host immunity) (Casadevall, Scharff, 1994) gave way to penicillin. Now, genetics and infections became opposites on a spectrum, "Huntington disease at one end, tuberculosis at the other" (Lindee 2002).

DISCUSSION

Instead of distracting physicians from heredity, infections traditionally reinforced interest in the subject, and profoundly influenced how they approached such issues- the two were seed and soil, intertwined, and the same doctrines, now outmoded, applied to both.

Treatment was hardly an issue- aside from surgery, medicine wasn't very good at cures until well into the 20th century, and therapies relied upon combined factors to influence diseases colored by individual reactions. Even vaccines strengthened constitutional properties by supporting the host's ability to respond to infection. Osler's text, the model for medicine in 1900, emphasized support and comfort (Hogan 1999), general and symptomatic measures that applied to genetic disorders as well as any others. Even unfavorable prognoses were considered important- Egyptian papyri had discussed ailments "not to be treated" (Breasted 1930), and neurologists were fascinated by diseases with irremediably grim outcomes, many inherited and named for physicians: Tay-Sachs disease, Duchenne muscular dystrophy, Huntington disease, Friedreich ataxia, and many others.

Inheritance wasn't even necessarily a reason for despair. Gowers found that epilepsy with a familial component was generally more treatable (1881), and Pearl felt that genetic analysis would be important in controlling tuberculosis (1920: 296-7). Rosenau noted that "It is now perfectly evident that heredity is one of the fundamental factors in preventive medicine" (1917: 470), often based on eugenics, e.g., Barker approvingly cited a "reduction of cretinistic imbecility… [by] segregating the cretins and preventing them from marrying" (1923).

Experiences with infections affected how physicians thought about diseases in general, and inheritance as well. By emphasizing variable and overlapping entities with multiple causes, they also obscured the specific factors and traits of Mendelism, and supported alternatives.

Intrinsic susceptibility and resistance varied and interacted with extrinsic factors, supporting a variable force instead of the presence or absence of a single gene, which was hard to show for infections- for TB, at least four genes contribute, and other determinants are undoubtedly involved (OMIM 2024). For polio and diphtheria, suggestions of a major role for a single gene have not been confirmed (OMIM 2024). Rheumatic fever may be an exception, with a good fit for an autosomal recessive (Sit 1990).

One cause could have many manifestations- TB, syphilis and RF, all "great imitators," had uncertain boundaries and unusual and confusing presentations. "Classic" cases were obvious, but some findings could be caused by any of the three- one finding could have different causes (Lubinsky 2024).

For older ideas to lose support, concepts supported by a wealth of experience had to be rejected, and this occurred as infections decreased. For Chicago, Ill., for example, in 1870, 50% of children died before their fifth birthday, mostly in the first year; In 1900, the figure was still 25%. However by the late 1930s, infant deaths were down to 5%, and in 1945, were only about a quarter of 1915 (Chicago Public Library Web Site 2022).

The relative importance of congenital anomalies rose as infections decreased. In England and Wales, as neonatal mortality from "debility, atrophy & marasmus" roughly halved from 1890 to 1917, congenital defects as causes of death went from 3.2 to 5.9/1000 (Newsholme 1920). Right before the antibiotic era began, the main causes of infant mortality in Northern Ireland were still "respiratory infections, gastro-enteritis and prematurity. Diarrhoea and enteritis were most prevalent among the poorest." But, even then, it was "prematurity and congenital malformations among the richest" (Lancet 1944). Infections fell into a new reductionist model of disease with the availability of antibiotics, improved sanitation, and new laboratory techniques defining disorders more definitively (Lubinsky, 2024). Vaccines, which once supported bacterial variability (Mendelsohn 2005), became another sort of "magic bullet" emphasizing specificity. With this, old ideas lost their justifications, and genetics, increasingly supported by molecular findings, became the new paradigm.

CONCLUSIONS

1. An environment-genetics dichotomy is relatively recent: Ideas about infection and inheritance influenced and reinforced each other well into the 20th century.
2. Infections created a model that determined how physicians defined traits, a view of *what* was inherited that undermined reductionist Mendelian approaches while supporting alternatives.
3. This model emphasized uncertain definitions, variable manifestations, nonspecific findings, and interacting causes.
4. It was supported by a persisting pre-Mendelian system of inheritance with diffuse factors such as heredity, degeneration, diatheses, and polymorphism.
5. The same factors also applied to infections.

6. Infections as alternative causes often interfered with the recognition of specific Mendelian disorders.
7. Changes in experience with infections around the middle of the 20th century, plus a new molecular biology, paved the way for the rejection of older ideas and the acceptance of genetics.

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