# 2 The Origin(*s*) of Interaction: Interaction in the Eugenics Controversy



In the early decades of the twentieth century, the eugenics movement swept across the United States and the United Kingdom.[1] Eugenicists worried that the "unfit" were outbreeding the "fit." In nature, natural selection ensured that the fit passed their traits on to the next generation more often than the unfit. But in human populations, eugenicists saw something different happening. In Britain, for eugenicists like Karl Pearson, who became the first Francis Galton Chair of Eugenics at University College London in 1911, the focus was on class; the concern was that governmental and charitable support for poor and "feebleminded" persons in the lower classes incentivized them to have more and more children, while cultural pressures on professional and cultural elites in the upper classes incentivized them to have fewer and fewer children (Porter 2004).[2] In the United States, for eugenicists like Charles Davenport, the focus was on race and ethnicity; the concern was that educated, upstanding whites were being out-bred by blacks and immigrants (Kevles 1995). What was the eugenicist to do?



Eugenicists on both sides of the Atlantic took a number of steps to spread the eugenic gospel, establishing institutes of eugenic research, eugenic societies, and eugenic journals. In Britain, Pearson set up the Eugenics Laboratory at University College London in order to collect human data and apply statistics to eugenic concerns (Porter 2004). In the United States, Davenport established the Eugenics Record Office at Cold Spring Harbor in Long Island, New York, to similarly collect data on the inheritance of wanted and unwanted traits (Allen 1986). In Britain, eugenicists set up the Eugenics Education Society with its associated *Eugenics Review* journal (Mazumdar 1992). In the United States, eugenicists founded the American Eugenics Society, which had its own *Eugenical News* (Osborn 1974). The gospel spread. Eugenic themes worked their way into everything from films, with titles like "Are You Fit to Marry?," to Sunday morning

sermons, with society-sponsored eugenic sermon contests (Pernick 1996; Rosen 2004).

These efforts at educating the public, however, did not satisfy the eugenicists. More needed to be done. And so eugenicists began working closely with members of government to pass and enforce eugenically inspired laws. In Britain, members of the Eugenics Society helped pass the U.K. Mental Deficiency Act of 1913, which allowed for the forced institutionalization of feebleminded persons; in the 1930s, they fought to pass an act encouraging the voluntary sterilization of the feebleminded (Mazumdar 1992). In the United States, eugenicists helped pass the Immigration Restriction Act of 1924, which severely curtailed the influx of immigrants; in 1927, they worked with Virginia officials to defend the state's policy of involuntary sterilization of the feebleminded before the U.S. Supreme Court and won *Buck v. Bell*, a decision that sanctioned sterilization across the United States (Kevles 1995).

Ronald Aylmer Fisher (1890–1962) was a giant of twentieth-century science. He was the first to demonstrate a mathematical relationship between Darwinian natural selection and Mendelian genetics. He also created many of the statistical methodologies that continue to be taught in statistics classes around the world.

Fisher was also an ardent eugenicist. He was a member of the British Eugenics Society and even helped establish the Society's Committee for Legalising Sterilisation (Mazumdar 1992, 204). His statistical innovations were developed in part to make eugenic assessments of the relative importance of nature and nurture when it came to evaluating traits like feeblemindedness. In the process of developing those statistical innovations, Fisher quickly realized that the interaction of nature and nurture (or what he called the "deviations from summation formula") posed a potential problem for his methodologies; however, it was a problem he felt confident dismissing.

Lancelot Thomas Hogben (1895–1975), though not so well known as Fisher, made equally lasting contributions to science. A trained statistician and biologist, Hogben developed the African clawed frog (*Xenopus laevis*) as a model organism for both experimental research and pregnancy testing; *Xenopus* remains one of the most commonly used model organisms in biology (Gurdon and Hopwood 2000; Olszynko-Gryn 2013). He also founded the Society for Experimental Biology, which still exists today (Erlingsson 2009).

Hogben was also a fervent anti-eugenicist. Throughout the 1930s, Hogben led a vicious assault on the British eugenics movement generally

and Fisher specifically. As Hogben saw it, the interaction of nature and nurture (or the "interdependence of nature and nurture," as he called it) was indeed a problem for Fisher's statistics and posed a serious challenge to the eugenicist.

A debate between Fisher and Hogben emerged, first in private communication and then in public, over this interaction of nature and nurture. What was interaction? Why and how should interaction be investigated? And what was the empirical evidence for it? Fisher and Hogben had radically different answers to these three questions. Where Fisher saw a potential but unproven nuisance, Hogben saw a fundamentally devastating critique. What explains Fisher and Hogben's different assessments of interaction? Was it simply that Fisher and Hogben were influenced by different religious or political motivations? A religious/political divide was surely part of this story, but only part. Fisher and Hogben, I argue, also faced an explanatory divide. I will show this by examining their separate routes to first recognizing the interaction of nature and nurture as a phenomenon. These routes were quite distinct and, as a result, when they finally came to discuss interaction, they understood the phenomenon quite differently: different concepts, different approaches to investigating it, and different verdicts on its prevalence in nature. This chapter about the origin of interaction is, thus, really a story about *origins*: Fisher's origin, and Hogben's origin.

The debate between Fisher and Hogben had a lasting impact on them personally and on the nature/nurture debate generally. On a personal level, the debate changed their relationship forever. Before the debate, they were cordial; afterward, they detested one another. On a wider level, though, Fisher and Hogben's separate routes to interaction created two very different ways of understanding the phenomenon that have persisted all the way to the present and shaped subsequent debates. The legacies of Fisher's route and Hogben's route, as well as the debates that ensued, will be the subject of the next two chapters. To understand those subsequent debates, though, we must start with the two scientists who laid the groundwork for debating interaction.

## R. A. Fisher and the "Deviations from Summation Formula"

Fisher showed signs of mathematical genius early in life. As Joan Fisher Box, his daughter and biographer, tells it, he was interested in fractions by age three, considered astronomical calculations by age six, and enrolled in classes with boys years older than him by age ten (Box 1978, 12–13).[3]

Fisher's early achievement was especially remarkable given the fact that he suffered from extremely poor eyesight. "He began to wear spectacles of increasing power until the lenses were so thick that they resembled transparent pebbles," Box writes (ibid., 14). Because of his poor vision, Fisher developed an unorthodox approach to solving mathematical problems. Rather than writing out a mathematical proof in standard sequential steps, Fisher often worked the problem through in his head visually and then simply stated the answer. This was a source of some tension between Fisher and his teachers early in life; later in his life, it occasionally left his students and readers puzzling to follow his innovative reasoning (ibid.).

Despite his unconventional approach to problem solving, Fisher thrived at mathematics and matriculated at Gonville and Caius College, Cambridge, in 1909 on scholarship. He continued his mathematical studies there, adding physics toward the end of his stay (Hodge 1992). Fisher also became an enthusiastic eugenicist. He helped create the Cambridge University Eugenics Society in 1911, hosting meetings in his rooms, organizing public lectures by well-known eugenicists, assisting at the First International Eugenics Congress, and even delivering his own eugenic lectures (Mazumdar 1992, 97–105). After graduating in 1913, Fisher was eager to join England's military, which was preparing for World War I. Poor eyesight, however, disqualified him (Yates and Mather 1963, 92). Instead, he spent the war years performing a variety of jobs around London related to mathematics and eugenics. He also worked for the Eugenics Education Society, contributing to the society's eugenics bibliography and reviewing books for its *Eugenics Review* (Mazumdar 1992, 105).



### "Analyzing the Causes of Variability"

In October 1918, at only 28 years of age, Fisher published "The Correlation between Relatives on the Supposition of Mendelian Inheritance" (Fisher 1918). At the time, Darwin's theory of natural selection and Mendel's principles of genetics were thought to be incompatible; the former treated variation in nature as gradual and continuous, while the latter treated variation as sporadic and discontinuous. A heated dispute emerged between biometricians, like Pearson, who backed Darwinian continuous variation, and Mendelians, like William Bateson, who backed Mendelian discontinuous variation.[4] Fisher's project was the resolution of this perceived incompatibility. It was an idea he first presented to the Cambridge University Eugenics Society in 1911.[5] Far from being incompatible, Fisher showed that the biometricians' statistical data on continuous variation could be derived from Mendelian inheritance (Norton 1978; Provine 2001).[6] Fisher's

formulation of a mathematical relationship between Darwinian evolution and Mendelian genetics was the first in a series of contributions to what Julian Huxley later termed the "modern evolutionary synthesis" (Huxley 1942). He was soon joined in this work by the British polymath J. B. S. Haldane and the American geneticist Sewall Wright, who collectively created the field of population genetics (Provine 2001). This achievement alone would have secured Fisher's place in any history of twentieth-century science.[7]

Remarkably, synthesizing Darwinian evolution and Mendelian genetics was not the only feat accomplished in Fisher's 1918 article. In the process of deriving the mathematical relationship between Darwin and Mendel, Fisher also introduced a new statistical concept (Moran and Smith 1966; Kempthorne 1974; Box 1978). Fisher was interested in accounting for the sources of variation in a population. Traditionally, populations were statistically evaluated solely with an eye toward averages, but averages shed no light on variation. Fisher found that if a trait under investigation, such as height in humans, manifested itself in a population with a normal distribution (i.e., a bell curve), then the mean could be calculated along with the standard deviation. Fisher's novel contribution to the statistical analysis of variation in a population was to go beyond the standard deviation and analyze the square of the standard deviation. "When there are two independent causes of variability capable of producing in an otherwise uniform population distributions with standard deviations $\sigma_1$ and $\sigma_2$," Fisher wrote, "it is found that the distribution, when both causes act together, has a standard deviation $\sqrt{(\sigma_1^2 + \sigma_2^2)}$. It is therefore desirable in analyzing the causes of variability to deal with the square of the standard deviation as the measure of variability" (Fisher 1918, 399). Fisher, having introduced a new statistical measure, knew he had to simultaneously introduce a new statistical concept to capture this measure, and so he continued, "We shall term this quantity the Variance of the normal population to which it refers, and we may now ascribe to the constituent causes fractions or percentages of the total variance which they together produce" (ibid.). *Variance* was now available as a measurement for the statistician and the eugenicist.

The earlier generation of biometricians had already introduced the concept of the correlation coefficient as a numerical measure of association (Norton 1975; MacKenzie 1981b). Thus, correlation tables were, by 1918, common; and parental correlations (associations between parent and offspring) along with fraternal correlations (associations between siblings) were frequently calculated from these correlation tables by the

biometricians. Fisher utilized this data for partitioning sources of variation in 1918 as a means toward assessing the relative importance of hereditary and environmental causes of variation when it came to height in humans, explaining, "For stature the coefficient of correlation between brothers is about .54, which we may interpret by saying that 54 per cent. of their variance is accounted for by ancestry alone, and that 46 per cent. must have some other explanation" (Fisher 1918, 400).

To what cause should this remaining 46% be attributed? Perhaps an environmental cause of variation? No! Fisher, the eugenicist, was quick to eliminate that possibility from the mind of his reader. "It is not sufficient to ascribe this last residue to the effects of environment. Numerous investigations by Galton and Pearson have shown that all measurable environment has much less effect on such measurements as stature" (ibid.). So with environmental variation expunged from the list of possible causes of variation, Fisher had to find another explanation for the 46% of the total variance left unaccounted. That other explanation came from Mendel's principles of segregation and dominance. Fisher calculated the variance between siblings attributable to Mendelian segregation and the effects of dominance. With variances due to ancestry, segregation ($1/2\ \tau^2$), and dominance ($3/4\ \varepsilon^2$) all accounted for, Fisher could finally sum up the sources of the total variance (ibid., 424):

| | | |
|---|---|---|
| Ancestry | | 54 per cent |
| Variance of sibship | | |
| ½ $\tau^2$ | 31 per cent | |
| ¾ $\varepsilon^2$ | 15 " | |
| Other causes | — | |
| | | 46 " |
| | | 100 per cent |

Fisher, referencing the negligible variance left for "other causes," concluded, "it is very unlikely that so much as 5 per cent. of the total variance is due to causes not heritable, especially as every irregularity of inheritance would, in the above analysis, appear as such a cause" (ibid.). For Fisher in 1918, it was a clear win for the nature side of the nature/nurture debate.

As mentioned above, commentators have pointed to Fisher's 1918 paper for its dual achievement of (a) synthesizing Darwinian evolution and Mendelian genetics, and (b) introducing the statistical concept of variance. Less noticed, though crucial for the discussion of this book, is an additional

phrase Fisher introduced to science in 1918—"cause of variability." Fisher wasn't just measuring variation; he was measuring *causes* of variation. The phrase pervaded his paper, as the quotes above attest: "when there are two independent causes of variability ... "; "it is therefore desirable in analyzing the causes of variability ... "; "it is very unlikely that so much as 5 per cent. of the total variance is due to causes not heritable." Fisher called his calculation above an "analysis of variance"; the great achievement, as he saw it, was not just that he introduced a measure of variation, but rather that he introduced a method for partitioning the *causes* of variation. This attention to identifying and partitioning causes of variability remained a constant throughout Fisher's career.



### "A Great Complication"

An assessment of Fisher's evaluation of the relationship between hereditary and environmental causes of variation would be incomplete if it terminated with his conclusions made in 1918. Historians of genetics and eugenics have often characterized Fisher as a "reformed" eugenicist, emphasizing his ultimate recognition of the potential importance of environmental causes of variation (Soloway 1990; Barkan 1991; Mazumdar 1992; Kevles 1995). Pauline Mazumdar, in particular, has carefully detailed the evolution in Fisher's thought (Mazumdar 1992). According to Mazumdar, Fisher's 1918 paper was, from the very beginning, designed to accommodate the ideals of the Eugenics Society: (a) the compatibility of Darwin and Mendel, and (b) the negligible importance of environmental causes of variation (ibid., 110). But in 1919, Fisher left Cambridge to join the Rothamsted Agricultural Research Station in Harpenden (Box 1978; MacKenzie 1981b; Mazumdar 1992). At Rothamsted, Fisher's job was to examine environmental causes of variation rather than assume them to be a randomly distributed variable, as he had in his 1918 publication. So it was at Rothamsted that Fisher created many of the statistical methodologies, such as tests of significance and the design of experiments that continue to be used by statisticians today (Yates 1964; Box 1978; Johnstone 1987; Preece 1990). And it was there that Fisher first came to consider the problem posed by interacting causes of variation.



In 1918, Fisher explained that sources of variation could be summed as long as the causes of variability were independent. Prior to undertaking his work at Rothamsted, the environment could be treated as independent for the simple reason that Fisher took it to be negligible. In making no contribution to variability, there was no need for Fisher to concern himself with how environmental variation might be related to the other causes of

variation. But at Rothamsted, with nurture now on the list of possible sources of variation, Fisher had to also consider the relationship between environmental variation and hereditary variation. He judged this possible complication in 1923 with Winifred A. Mackenzie, where they studied different potato varieties' responses to different manure-based fertilizer treatments (Fisher and Mackenzie 1923).[8] Fisher began by warning, "if important differences exist in the manurial response of varieties a great complication is introduced into both variety and manurial tests; and the practical application of the results of past tests becomes attended with considerable hazard" (ibid., 311). The "great complication" that posed a "considerable hazard" was the interaction of nature and nurture, with potato variety representing nature and fertilizer representing nurture in this case. If certain potato varieties always performed better than other potato varieties, or if certain fertilizers always performed better than other fertilizers, then the scientist (and the farmer) had a very easy decision to make when it came time to select which potato variety to plant and in which fertilizer. But if different potato varieties performed differently in different fertilizers, then there wasn't necessarily a "best potato" or a "best fertilizer"; the "best potato" depended on which fertilizer was used, and the "best fertilizer" depended on which variety of potato was planted. "Only if such differences are non-existent, or quite unimportant," Fisher cautioned, "can variety tests conducted with a single manurial treatment give conclusive evidence as to the relative value of different varieties, or manurial tests conducted with a single variety give conclusive evidence as to the relative value of different manures" (ibid.).

To test for this great complication, Fisher devised a method to evaluate the fertilizer responses of 12 different potato varieties. He divided a relatively small field (0.162 acres) into two equal parts, one part receiving a manurial treatment, the other receiving no treatment. Each half was then itself divided into 36 plots, and each of the 12 potato varieties then planted in triplicate within each field. Finally, each individual plot was divided again, so that three rows of seven plants were set in each plot; one row received only the basal manuring of the series to which it belonged, while the other two rows received in addition either potassium sulfate (muriate of sulfate) or potassium chloride (muriate of potash). With this experimental design, Fisher measured the weight of produce lifted from each of the rows, determining both the mean yield of each of the 12 varieties irrespective of the fertilizer applied (the "main effect" of variety), and the mean yield of each of the fertilizer treatments irrespective of the variety grown (the "main effect" of fertilizer). What followed was the first presentation

| Variation due to | Degrees of freedom | Sum of squares | Mean square | Standard deviation |
|---|---|---|---|---|
| Manuring ... ... ... ... | 5 | 6,158 | 1231·6 | 35·09 |
| Variety ... ... ... ... | 11 | 2,843 | 258·5 | 16·07 |
| Deviations from summation formula | 55 | 981 | 17·84 | 4·22 |
| Variation between parallel plots ... | 141 | 1,758 | 12·47 | 3·53 |
| Total | 212 | 11,740 | — | — |

**Figure 2.1**
Fisher's analysis of variance due to fertilizer manuring, potato variety, deviations from summation formula, and variation between parallel plots (reproduced from Fisher and Mackenzie 1923, table III).

of an analysis of variance table, listing the various causes of variation along with their respective contribution to the total variation in crop yield (see figure 2.1; Box 1978, 111).

The "deviations from summation formula" category was the measure of the differences between the potato varieties in their fertilizer response—the measure, that is, of interaction. It was called a "deviation" because Fisher's analysis of variance was designed to attribute variation first to the main effects (in this case, potato variety and fertilizer). If the variation due to potato variety and the variation due to fertilizer summed up to account for the total variation in crop yield, then there would simply be no variation left to attribute elsewhere, and so the result was referred to as "additive" because the main effects added up. Interaction arose in the analysis of variance if there was a deviation from the main effects adding up to fully accounting for the variation, in which case there was "non-additivity." So the question for Fisher was whether or not there was a deviation from this additive summation. In yet another innovative leap in this same article, Fisher determined that the deviations from the summation formula were not significantly greater than would occur by chance, leading him to conclude, "In the present material evidently the varieties show no difference in their reaction to different manurial conditions" (Fisher and Mackenzie 1923, 317). This comparison of a source of variation against chance was an early test of statistical significance, or what is now called an "F-test" in honor of Fisher's development of the method. For Fisher, in 1923, it justified dismissing the problem.

Fisher paid little attention to the great complication posed by interacting causes of variation for the next ten years; the considerable hazard had been examined and dismissed. In *Statistical Methods for Research Workers*, his extremely influential how-to guide for statistics, Fisher made only passing reference to the "interaction of causes" just to point to the results

of his 1923 publication with Mackenzie and then dismiss the phenomenon (Fisher 1925, 209). And in his eugenic writings from this period on the sterilization of the feebleminded and the high fertility of people in lower classes, Fisher made no reference to interaction (Fisher 1924a, 1924b, 1926a, 1926b). He was not concerned that people in a lower class might become people of a higher class in a different environment, or that the feebleminded might become less feeble in a different environment. It took a letter from Hogben, a decade later, for Fisher to reconsider the issue.

## Lancelot Hogben and the "Interdependence of Nature and Nurture"

Hogben described his "larval existence" like that of many prominent biologists: obsessively collecting beetles and butterflies, identifying birds and recognizing them by their eggs, and exploring local geography. "I wanted to be a biologist long before I was twelve," he recalled 60 years later (Hogben 1998, 2).[9] Biology, however, was not what God intended for Hogben … at least that was how his mother saw it. He was born two months prematurely, and to ensure that he would survive the ordeal, his mother dedicated him from birth to becoming a missionary (ibid., 1). This religious devotion was no less powerful on the paternal side of his parenting. Thomas Hogben,[10] a self-employed Methodist preacher, spent his days ministering to seamen at the local port under a banner extolling the benefits and burdens of the Christian God. "In the foreground was the lake of brimstone and fire. Across the middle was the edge of a cliff where stood the theatre, the brothel, the casino, the racecourse, the tavern, the *Palais de Danse* and other haunts of Satan. From the edge of the cliff the lost departed were falling in different stages of incandescence. Above the cliff was a solitary pilgrim pursuing a winding road to the rising sun; and, ironically, below it across the flames the legend: *God is Love*" (ibid., 4). Fortunately, the young Hogben and his parents were able to reach a compromise during these formative years; the field of medicine allotted the boy the time to study biology while also preparing him for service as a medical missionary (ibid., 13).

Largely self-educated at the Stoke Newington Public Library in northeast London, Hogben excelled academically and won a scholarship to attend Trinity College, Cambridge, in 1913, just as Fisher was graduating (ibid., 24–25). At Cambridge, Hogben cultivated his biological interests and replaced his parents' religious zeal with a devotion to socialism. He studied botany, physiology, zoology, and embryology (ibid., 40–41). And he entered social life with an equal vigor. Assessing the social societies available to him at the time, Hogben recalled, "I still regard the Union Debating Society of

Cambridge (even more that of Oxford) as a potting shed for the cultivation of mentally retarded politicians. The most lively discussions at an intellectually high level were those which took place at the *Moral Sciences*, colloquially *Moral Stinks, Club*, where Bertrand Russell and [G. E.] Moore minced words with their philosophical competitors, in the Fabian Society and its study circles, and in the *Heretics* founded by C. K. Ogden of Basic English fame" (ibid., 33). The Fabian Society was a particularly accommodating match for Hogben; he met his first wife, Enid Charles, there and eventually became its secretary, changing the society's name to the University Socialist Society (ibid., 51).

Unlike Fisher, the pacifist Hogben actively avoided military combat. At the outset of World War I, Hogben joined noncombatant Quaker relief organizations. When the British government introduced compulsory military service in 1916, Hogben protested this action as a conscientious objector and spent several months imprisoned in London's Wormwood Scrubs for the decision (ibid., chapter 7). After the war, Hogben entered academic life, teaching and leading research in London at Birkbeck and the Royal College of Science (1917–1922), in Edinburgh at the Animal Breeding Research Laboratory (1922–1925), in Montreal at McGill University (1925–1927), and at the University of Cape Town in South Africa (1927–1930).

In his early career at Birkbeck, Edinburgh, McGill, and Cape Town, Hogben was primarily devoted to experimental embryology and physiology. He studied amphibian metamorphosis with Julian Huxley and amphibian pigmentation with Frank Winton (Huxley and Hogben 1922; Hogben and Winton 1922a, 1922b, 1923). These investigations were interventionist in design, attempting to elucidate the mechanisms responsible for phenomena; for example, he isolated the role of the pituitary in the amphibian pigmentory effector system by surgically going through the roofs of frogs' mouths and removing various portions of the gland, then noting the associated lack of pigmentation. Hogben also helped create the Society for Experimental Biology and its accompanying *British Journal of Experimental Biology*, which still exists today as the *Journal of Experimental Biology* (Crews et al. 1923; Erlingsson 2009). Hogben, with these achievements, quickly rose up the ranks of the British biological elite, joining the likes of Haldane and Huxley. Influential British geneticist Cyril D. Darlington recalled after Hogben's death, "When I was very young, Galdane, Guxley, and Gogben (as the Russians called them), seemed to be the three Magi."[11]

It was Hogben's seven years (1930–1937) at the London School of Economics and Political Science (LSE) that produced his most lasting contributions to science and society. During these years he wrote *Mathematics*

*for the Million* (1937) and *Science for the Citizen* (1938); both books were hugely successful, designed for lay readers to teach themselves practical mathematics and science just as Hogben taught himself at the public library years earlier. While at the LSE, he also attacked Britain's eugenics movement with a tenacity unmatched even by the standards of other anti-eugenicists of his day (Blacker 1952; Ludmerer 1972; Werskey 1978; Soloway 1990; Barkan 1991; Mazumdar 1992; Kevles 1995; Paul 1995, 1998). Sir (later Lord) William Beveridge, director of the LSE, sought to bridge the divide between the natural and the social sciences and so announced the search for a Chair of Social Biology in 1929 (Dahrendorf 1995; Renwick 2013). Fisher actually inquired about filling the position, even envisioning whom he would hire as subordinates.[12] But it was Hogben whom Beveridge ultimately invited to take the post. In his autobiography years later, Hogben recalled this vocational victory with glee, noting, "the brass hats of the Eugenics Society were already congratulating themselves on the prospect of one of their co-religionists getting the job" (Hogben 1998, 121). Hogben, however, only agreed to take the appointment after some reluctance, later explaining, "At that time human genetics was a morass of surmise and superstition. ... Should I prosper in the Herculean task of cleaning the Augean stables of human heredity, I should be contributing to the overdue disposal of a manure heap of insanitary superstitions" (ibid., 122). Ultimately it was one of Hogben's fellow "Magi" who convinced him to take on the responsibility. "Conversation with J. B. S. Haldane jerked me out of indecision concerning my fitness for the task" (ibid.). Hogben accepted the position and left Cape Town, joining the LSE in 1930.



### "A Third Class of Variability"

Hogben was not the first to formulate anti-eugenic arguments. Psychologists argued that the intelligence tests hailed by eugenicists to distinguish the fit from the unfit were biased; anthropologists claimed that the categories upon which eugenicists put so much emphasis—class, race, and ethnicity—were cultural constructs, not biological ones; even Catholics criticized eugenics on religious grounds, claiming marriage was a moral matter, not a eugenic one (Kevles 1995). Hogben instead attacked the methodological foundations of eugenic science. His first full-fledged assault on eugenics came with the publication of his *Genetic Principles in Medicine and Social Science* (Hogben 1932).[13] "This book does not undertake to set down all that is known and has been surmised about human inheritance," Hogben admitted. Instead, it was the first step in his "Herculean task." He explained:

> It is an attempt to separate the wheat from the tares, to indicate where a sound foundation of accredited data is available, to discuss what methods can be applied to the extremely elusive nature of the material with which the human geneticist deals, and to re-examine some of the biological concepts which have invaded other fields of inquiry in the light of modern advances in experimental genetics. (ibid., 9)

The underlying thread that guided the discussion was his persistent emphasis on the role the environment played in the development of organisms. When it came to "a sound foundation of accredited data," Hogben emphasized the importance of the nutritional environment contributing to diseases such as rickets (ibid., 64). And when cognitive developmental disorders were discussed, Hogben drew attention to the effect of birth order on the incidences of the traits (ibid., 99–103).



When it came to reexamining "biological concepts," Hogben claimed, "Genetical science has outgrown the false antithesis between heredity and environment productive of so much futile controversy in the past" (ibid., 201). Since every trait is the end product of an immensely complicated series of developmental reactions between the environment and the hereditary material, "Differences can be described as determined predominantly by hereditary or predominantly by environmental agencies if, and only if, the conditions of development are specified" (ibid., 98). Hogben, to drive this point home, pointed out that variation in a population arose from hereditary variation (emphasized by eugenicists), environmental variation (emphasized by anti-eugenicists), and an often-ignored *third class of variability*: that which "arises from the combination of a particular hereditary constitution with a particular kind of environment" (ibid.). This "third class of variability" became especially important when Hogben employed it to criticize Fisher the following year.



But in 1932, Hogben had not yet criticized Fisher, and in his review of *Genetic Principles*, Fisher welcomed Hogben's hiring at the LSE (Fisher 1932).[14] Fisher began, "[Hogben's] recent appointment as Professor of Social Biology at the London School of Economics gave the welcome assurance that his keenly analytic brain, and training in a severe experimental discipline, would be put to important service in the study of the biology of man" (ibid., 147). Compliments aside, Fisher then complained that Hogben's attention to "purely academic considerations" too often led to an exclusion of "aspects of more practical importance" (ibid.). In particular, Fisher criticized Hogben's tendency to demand experimental investigations into the genetic and environmental causes of development before intervening on those causes of variation (with, for example, eugenic policies). Fisher sarcastically noted:

Throughout the book, those who consider that the practical importance of the problem renders it urgent, will receive a disturbing impression that they are being asked to wait, in solemn hush, outside the laboratory door, until the Professor sees fit to announce that the ultimate truth has at last been revealed. (ibid., 147–148)[15]

### "An Inherent Relativity in the Concepts of Nature and Nurture"

That same year, the medical faculty at the University of Birmingham invited Hogben to deliver their William Withering Memorial Lectures, and Hogben chose medical genetics as the theme of his lectures. Hogben, in preparation for the lectures, contacted Fisher, who was about to leave Rothamsted for University College London, succeeding Pearson in the Galton Chair of Eugenics. "Dear Fisher," Hogben wrote, "I am at present engaged in preparing a course of lectures in which I shall be dealing with your own contributions to the genetic theory of correlation. There is one point in your 1918 paper which worries me very much." Hogben was concerned about Fisher's talk of "cause." He asked, "When you speak of the contribution of hereditable and nonhereditable causes of variance in a population, what exactly do you mean? I often use the same form of words myself and lately I have been searching for a more explicit formulation of the problem." To explain the source of his concern, Hogben provided a quantitative example:

Suppose you say that 90 per cent of the observed variance is due to heredity, do you mean that the variance would only be reduced ten percent, if the environment were uniform? Do you mean that the variance would be reduced by 90 per cent, if all genetic differences were eliminated? Perhaps you will think the question silly; but if you could suggest an alternative form of words, it might help.[16]



Fisher responded the following day.

Dear Hogben, Your question is a very sound one. The point is this:-If the differential effects of environment and heredity are not correlated, i.e. if each genotype has an equal chance of experiencing with their proper probabilities, each of the available kinds of environment, then the variance is additive, and the statements you have are equivalent.[17]

Fisher took Hogben's question to be one concerning the correlation of heredity and environment, and so he answered Hogben's question with a discussion of heredity's "chance of experiencing" a particular environment. This, however, was not Hogben's target, and so Hogben took several days to construct a lengthy rebuttal. "Dear Fisher," he wrote, "I don't think you quite got the difficulty which I am trying to raise. It concerns an inherent relativity in the concepts of nature and nurture."[18] To clarify, Hogben

introduced research by American embryologist Joseph Krafka (1920), who raised different strains of fruit fly (one called "low bar" and another called "ultra bar") at different temperatures and then counted the number of eye facets that each population developed on their compound eyes:

From Krafka's data you will see the following values for facet number are given at 15° and 25° C.

| | Low bar | Ultra bar |
|---|---|---|
| 15° C | 189 | 52 |
| 25° C | 74 | 25 |



Consider the elementary population with the following structure. The genotypes are low bar and ultra bar in equal numbers, equally distributed between two environments, namely an incubator at 15° C and one at 25° C. There is zero correlation between the distribution of environmental and genetic variables. Yet I cannot agree that the two statements "y per cent of the variance is due to environment," and "the variance would be reduced by y per cent if all differences of environment were eliminated," are equivalent nor that there is equivalence between the two statements "x per cent of the variance is due to heredity" and "the variance would be reduced by x per cent if there were no genetic differences."

The reason, Hogben pointed out, was that there was a "lack of singularity in the problem," since differences in heredity and differences in environment could be eliminated in any number of ways. "Let us abolish all differences of environment," Hogben proposed:

We can do this in an infinite number of ways. One would be to culture all flies at 15° C. Result: mean 120.5 and variance 4692. Another is to culture them all at 25° C. Result mean 49.5 and variance 600. Which of these two variances has priority as an estimate of the "contribution" of environment to the observed variance in the fourfold population? Again we eliminate all genetic differences by killing off all ultra bar flies. Result: mean 131.5 and variance 3306. We could alternatively kill off all low bar flies. Result: mean 38.5 and variance 182. Which of these gives the contribution of heredity to the observed variance?

Hogben's point was this: an analysis of variance that partitioned causes of variation in a population did not necessarily translate into a single answer about how the population would respond to an intervention on those causes of variation; it depended on how the hereditary difference was acted upon, and how the environmental difference was acted upon. Hogben closed by making the source of his concern explicit: "What I am worried about is a more intimate sense in which differences of genetic constitution are related to the external situation in the process of development."

Hogben's letter on the 23rd of February, 1933, marked the dawn of interaction being utilized as a critical tool to attack the summing of hereditary and environmental causes of variation.[19] The fruit fly example from Krafka also became the empirical backbone of Hogben's last William Withering lecture, entitled "The Interdependence of Nature and Nurture" (Hogben 1933a, 1933b). It was, in short, an all-out attack on Fisher.

There, Hogben admitted that Fisher's statistical techniques could be "used to detect the existence of differences due to environment and differences due to heredity" (Hogben 1933a, 93). However, moving beyond the detection of such differences, "The difficulties of interpretation begin when we attempt to clarify what is meant by calculating 'the numerical influence … of the total genetic and non-genetic causes of variability'" (ibid., 94–95). Hogben drew on his Cambridge philosophical hero to make this point. "In his illuminating essay on the *Notion of Cause* Bertrand Russell has pointed out that few words are used with greater ambiguity in scientific discussion" (ibid., 95).[20] What Hogben had in mind here was an extension of the critique he first made in his *Genetic Principles*. "The biometrical treatment of variability," Hogben argued,

> inherited from Galton a tradition of discourse in which the ambiguity of the concept of causation completely obscured the basic relativity of nature and nurture. Since then this relativity has become increasingly recognised through experiments involving the use of inbred stocks in physiological laboratories, especially in connexion with experimental work on diet. It is therefore necessary to examine with great care what we mean when we make measurements of a genetic difference and a difference due to environment. (ibid.)

Hogben, to explain "what we mean" when experimental biologists talk about "genetic difference" and "environmental difference," introduced to his reader the same case he introduced to Fisher in correspondence earlier that year—Krafka's data on different strains of fruit fly raised at different temperatures. This time, though, Hogben provided both the data and an image depicting the different responses of the low bar and the ultra bar strains to the different temperatures (see figure 2.2). Figure 2.2 is a norm of reaction graph; it plots the differential response of different genetic groups (their "norm of reaction") to different environments. On such a norm of reaction graph, if the norm of reaction lines are parallel, then there is no interaction of nature and nurture; if the lines are not parallel, however, then there is interaction in the population. In figure 2.2, notice that the norm of reaction line for low bar and the norm of reaction line for ultra bar are clearly not parallel, indicating that there was interaction in that

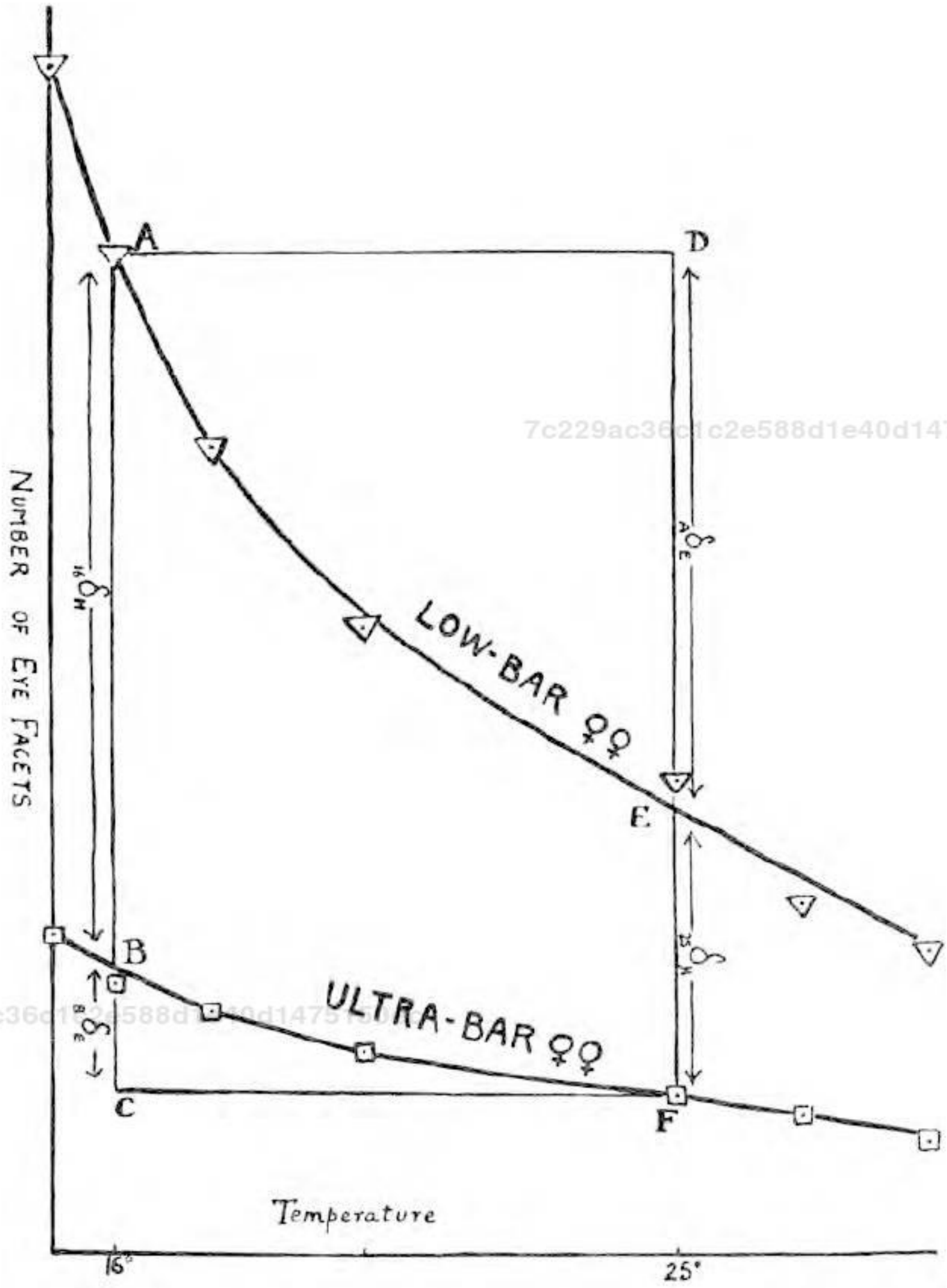


**Figure 2.2**

Hogben's norm of reaction graph for number of eye facets (y-axis) for low bar and ultra bar *Drosophila* strains raised at different temperatures (x-axis; reproduced from Hogben 1933a, figure 2).

population. The differences between points A and B and between points E and F corresponded to what Hogben claimed experimental biologists meant by a genetic difference. The differences between points B and C and between points D and E corresponded to what Hogben claimed experimental biologists meant by a difference due to environment. Hogben granted, "Clearly we are on safe ground when we speak of a genetic difference between two groups measured in one and the same environment or in speaking of a difference due to environment when identical stocks are measured under different conditions of development." But then he continued, questioning, "Are we on equally safe ground when we speak of the contribution of heredity and environment to the measurements of genetically different individuals or groups measured in different kinds of environment?" (ibid., 97). Hogben asked his reader to consider a low bar stock kept at 16° C and an ultra bar stock kept at 25° C, creating the observed differences AC or DF. "How much of AC or DF is due to heredity and how much to environment? The question is easily seen to be devoid of a definite meaning" (ibid.).

Yet it was precisely such meaning, Hogben claimed, that eugenicists attached to Fisher's analysis of variance (or what Hogben called Fisher's "balance sheet of nature and nurture"). Recall that Fisher, in 1918, wrote with regard to stature, "it is very unlikely that so much as 5 per cent. of the total variance is due to causes not heritable," to which Hogben retorted, "The only practical significance which Fisher's analysis of variance seems to admit is that, if it were correct, we could only reduce variance with respect to stature in a human population by 5 per cent. or less if the environment were perfectly uniform" (ibid., 114). But the lesson from the different strains of fruit flies raised at different temperatures, Hogben argued, applied equally well to different classes of humans raised in different medical, nutritional, and educational environments. The human environment could be made uniform in any number of different ways, some ways tending to magnify differences between classes and some tending to diminish differences between classes (ibid., 116). Hogben concluded,

> In whatever sense Fisher himself intended his balance sheet to be interpreted, there is no doubt that many writers on human biology entertain the belief that biometrical estimates of this kind do entitle us to set such limits. On the basis of such statements as the previous quotation about stature, it is often argued that the results of legislation directed to a more equitable distribution of medical care must be small, and that in consequence we must look to selection for any noteworthy improvement in a population. This is rather like saying that the difference between black and

white is negligible because an inkpot thrown into a tank of china clay has very little effect on the latter. (ibid., 116–117)

We can only assume that Fisher felt little gratitude when Hogben concluded his essay, "It is a great pleasure to acknowledge the courtesy with which Dr. Fisher has replied to communications in which some of the issues raised in this discussion have been explored" (Hogben 1933b, 405).

## The Explanatory Divide

Although Hogben never mentioned it, Fisher responded to Hogben's letter discussing the Krafka data just days later:

Dear Hogben, I think I see your point now. You are on the question of non-linear interaction of environment and heredity. The analysis of variance and covariance is only a quadratic analysis and as such only considers additive effects. Academically one could proceed in theory, though in a theory not yet developed, to corresponding analyses of the third and higher degrees. Practically it would be very difficult to find a case for which this would be of the least use, as exceptional types of interaction are best treated on their merits, and many become additive or so nearly so as to cause no trouble when you choose a more appropriate metric. Thus facet number shows its sweet reasonableness when measured in 'proportional units' or in other words on a logarithmic scale. However perhaps the main point is that you are under no obligation to analyse variance into parts if it does not come apart easily, and its unwillingness to do so naturally indicates that one's line of approach is not very fruitful.[21]

Fisher's appraisal of interaction here reveals much about his understanding of the phenomenon by the time Hogben used it against him. Fisher saw Hogben now to be worrying about the " non-linear interaction of environment and heredity." Fisher, of course, was familiar with the problem, having taken up his study of potato varieties and different fertilizers in 1923 with the sole purpose of testing for the complication. With the conclusions of that study in mind, notice how Fisher responded to Hogben: his concern was written off as "academic," while "practically it would be very difficult to find a case for which this would be of the least use." Fisher's investigation at Rothamsted led him to believe that such cases of interacting causes of variation were not the norm in nature; and, if they did arise, they could be eliminated by altering the scale on which the environment was measured (a transformation of scale), such as with the fruit fly data's "sweet reasonableness" with a transformation to a logarithmic scale. Notice also that Fisher's letter bore a striking resemblance to his review of *Genetic Principles* a year earlier, in which he complained that Hogben's attention

to "purely academic considerations" led to an exclusion of "aspects of more practical importance." Hogben could pursue a study of interactions for academic reasons, Fisher granted, but such a study would have no practical application.

Fisher's congeniality in this correspondence, which predated Hogben's published assault on Fisher, can be contrasted with a letter he wrote to fellow-eugenicist John A. Fraser Roberts in 1935 after Hogben published the last William Withering lecture, "The Interdependence of Nature and Nurture." There, Fisher predicted, "There is one point in which Hogben and his associates are riding for a fall, and that is in making a great song about the possible, but unproved, importance of non-linear interactions between hereditary and environmental factors. J.B.S. Haldane seems tempted to join in this."[22] Fisher here assessed the interaction of nature and nurture quite explicitly: it was of "possible, but unproved, importance." "Possible" because, as Fisher recognized in his potato/fertilizer study, the non-linear interactions would greatly complicate his summing of the causes of variation. But also "unproved" because Fisher found no such deviations from the summation formula.

Hogben came to quite a different conclusion. Krafka's research was a clear example, and Hogben took full advantage of its implications in his anti-eugenics lectures and publications. Importantly, Hogben emphasized that the Krafka data was not an isolated instance, writing, "The literature of experimental physiology is not wanting in examples of such divergent curves representing the measurement of a character and the strength of the environment" (Hogben 1933b, 385). He drew on the research of Norman Taylor (1931) and Frank Winton (1927), his former colleague and co-author from Edinburgh, who examined variation in the sinus beat of frogs exposed to different temperatures, and variation in the mortality rate of rats exposed to different levels of rat poison, respectively (Hogben 1933b, 385).[23]

## The Variation-Partitioning and the Mechanism-Elucidation Approaches to Nature and Nurture

When it came to judging the empirical evidence of interaction in nature, Fisher and Hogben evaluated the situation quite differently. Where Fisher saw a phenomenon of "possible, but unproved importance," Hogben saw a phenomenon he took to be common in nature. Why? Why, that is, did Fisher and Hogben see the situation so differently?

Historians who have reflected on the divergent scientific views of Fisher and Hogben have tended to emphasize the religious and political positions

that motivated Fisher's eugenics and Hogben's anti-eugenics. Fisher was a devout Anglican who published on the relationship between science and Christianity (Fisher 1955). Historians have highlighted how Fisher's religion supported his eugenics; the two domains were united in their emphases on family and in their emphases on human progress (Ruse 1996; Bartley 1994; Moore 2007). Fisher was also a proud patriot (eager to fight for his country) and a political conservative. He saw a clear divide between the upper and lower classes in Britain, and he thought eugenics could improve his country by increasing the former and decreasing the latter (Mazumdar 1992; Kevles 1995).

Hogben, in stark contrast, was an evangelical atheist. "I'm an atheist, thank God!" he often said (Wells 1978, 190). His perspective on religion aligned well with his socialist politics. He was critical of Britain's class system, a system which he spent his life demonstrating was no guarantee of performance. In 1931, at the Second International Congress on the History of Science, a Soviet delegation led by Nikolai Bukharin introduced Marxism to the British scientific community. Historians have noted how influential this visit was for left-wing scientists of the day, such as Hogben and Haldane. As Mazumdar explained:

> Hogben's thinking on the problems of social biology did not take a completely new direction following his contact with Marxism, but the Marxist analysis both sharpened his perception of the class-bound nature of the eugenic program, and also provided a theoretical support for his campaign against the over-emphasis of the biological in human society. (1992, 161)

Gary Werskey similarly offered:

> Rather than completely sacrifice [Hogben's] outside political interests to the demands of scientific life, he consciously brought his politics to bear on the kind of science he did. As a feminist who was also an experimental biologist, Hogben was drawn in the early twenties to the new field of comparative endocrinology, in order to study the hormonal bases of sex differences. As a socialist, he likewise found himself attracted to the social biology of class and racial differences. (1978, 105; see also Kevles 1995; Bowler 2001)

These appeals to religion and politics certainly help us to appreciate the motivations for Fisher's eugenics and Hogben's anti-eugenics. But the separate routes that Fisher and Hogben took to first considering the interaction of nature and nurture revealed something else that divided them. In addition to any religious or political divide, they also faced an explanatory divide. They faced this explanatory divide because, although they shared a common interest in understanding the relationship between nature and

nurture, they employed very different approaches to studying that relationship. I use "approach" here in a manner similar to Thomas Kuhn's "paradigm" (1962), Imre Lakatos' "research programme" (1977), Larry Laudan's "research tradition" (1977), or Ian Hacking's "style of scientific theorizing" (1994). These various philosophical accounts emphasize how different scientists can attempt to explain an apparently common phenomenon and yet approach that phenomenon with very different questions, concepts, problems, and methodologies. My emphasis is on how Fisher and Hogben, though they studied a common phenomenon—the relationship between nature and nurture—approached that phenomenon with different explanatory frameworks. That is, though they were both interested in the relationship between nature and nurture, they identified different things that needed explaining (what philosophers call the "explanandum"), they asked different causal questions about the explanandum, they appealed to different things that did the explaining (what philosophers call the "explanans"), and they pointed to different methodologies that generated the explanans. Fisher and Hogben faced an explanatory divide because they diverged on each of these components: thing to be explained, causal question, thing that does the explaining, and methodology. Let me explain.

Fisher took what I call the variation-partitioning approach to studying nature and nurture (see table 2.1). For him, the thing that needed explaining was variation in a population or, more specifically, the relative contributions of nature and nurture to variation in a population. Fisher asked, how much of the variation was due to hereditary causes of variation, and how much was due to environmental causes of variation? What did the explaining when it came to these how-much questions were the causes of variation responsible for variation in a population. Fisher, in turn, developed many of the statistical methodologies, such as the analysis of variance, that allowed for partitioning these causes of variation in order to attribute to each its relative contribution.

Hogben, in contrast, took what I call the mechanism-elucidation approach to studying nature and nurture (see table 2.1). For Hogben, the thing that needed explaining was the developmental process or, more specifically, the developmental relationship between nature and nurture that played out during the developmental process. Hogben asked, how do differences in heredity and how do differences in environment interact during development to create variation separate from hereditary and environmental variation alone? What did the explaining when it came to these how questions were the causal mechanisms responsible for the

**Table 2.1**
The Components of the Explanatory Divide

| | Variation-partitioning approach | Mechanism-elucidation approach |
|---|---|---|
| **Thing to be explained** | Variation in a population | Developmental process |
| **Causal question** | How much? | How? |
| **Thing that does the explaining** | Cause of variation | Causal mechanism |
| **Methodology** | Statistical | Interventionist |
| **Concept of interaction** | Biometric | Developmental |



development of a trait. Hogben, for his part, thought Fisher's statistical methodologies were useful as a starting point for detecting the relevant variables in the causal mechanisms, but those statistical methodologies had to be followed up with more interventionist experiments designed to probe the system in order to elucidate how those variables made their difference in the developmental mechanisms.[24]

### Different Answers to the Conceptual, Investigative, and Evidential Questions

Fisher and Hogben shared a common interest in studying the relationship between nature and nurture, but they faced an explanatory divide because they took very different approaches to that study. Fisher's variation-partitioning approach attended to the causes of variation responsible for variation in a population, while Hogben's mechanism-elucidation approach attended to the causal mechanisms responsible for the development of a trait. Their routes to first considering the interaction of nature and nurture were situated in these approaches. And because they approached the study of nature and nurture so differently, it followed that their considerations of interaction were different, too. Fisher and Hogben ultimately had very different answers to the following three questions:



- the conceptual question—what is interaction?
- the investigative question—why and how should interaction be investigated?
- the evidential question—what is the empirical evidence for interaction?

When it came to the conceptual question, Fisher defined interaction as a "deviation from summation" and also as a "non-linear interaction." For

Fisher, notice, the interaction of nature and nurture was defined as an *absence*—an absence of summation, an absence of linearity, an absence of additivity between the main effects. It was also a purely statistical concept, a product of his statistical analysis of variance. Interaction arose in an analysis of variance when the main effects of nature and nurture failed to add up to the total variation. Fisher conceptualized interaction as *a statistical measure of the breakdown in additivity between the main effects of nature and nurture.* Because, for Fisher, interaction was a statistical phenomenon, I call this the "biometric concept of interaction."

Fisher's biometric concept of interaction influenced his answer to the investigative question. Interaction had to be considered because it posed a "great complication" to the task of measuring the relative contributions of nature and nurture to variation in a population. Partitioning those relative contributions was quite easy if the causes of variation were independent. But if they were not independent, if the variation caused by heredity was affected by the variation caused by environment and vice versa, then the statistician and the eugenicist suddenly faced a "considerable hazard." Fortunately, as far as Fisher was concerned, there was a solution to the problem if it arose. If the analysis of variance did indeed find a non-additive deviation from the summation formula, then the statistician could alter the scale on which the environmental variable was measured (to, for example, a logarithmic scale) in order to eliminate the non-linear interaction and get back to assessing the relative contributions of the main effects.

Even more fortunate, as far as Fisher was concerned, the transformation of scale wasn't commonly needed for the simple reason that non-linear interactions were the exception and not the rule in nature. They were a "possible, but unproved" obstacle that he had considered and then dismissed in 1923 when he studied the different potato varieties' responses to different fertilizers. This was Fisher's answer to the evidential question. And it explains why he wrote off Hogben's concern as "academic." In nature, where scientists and eugenicists with "practical" concerns focused their attention, Fisher saw no reason to be concerned by the prospect of that great complication.[25]

Hogben, coming to consider interaction within a mechanism-elucidation approach, answered the conceptual, investigative, and evidential questions much differently. Hogben clearly did not get the original idea from Fisher. Hogben never referenced Fisher's 1923 publication on the deviations from summation; and, when Hogben first raised the issue with Fisher in correspondence, remember that he admitted, "Perhaps you will think the

question silly." Hogben, in his letter to Fisher, explained quite clearly the origin of the issue for him: "What I am worried about is a more intimate sense in which differences of genetic constitution are related to the external situation in the process of development." This was his third class of variability—variation due to the combination of a particular hereditary constitution with a particular kind of environment. Hogben's answer to the conceptual question was thus much different from Fisher's answer. Interaction, according to Hogben, was not an absence; it was a *presence*—a presence of the third class of variability, a presence of a source of variation generated by the developmental relationship between nature and nurture. Hogben conceptualized interaction as *variation that resulted from differences in unique, developmental combinations of nature and nurture*. Because, for Hogben, interaction was a developmental phenomenon, I call this the "developmental concept of interaction."

Hogben's developmental concept of interaction influenced his answer to the investigative question. The interaction of nature and nurture was to be sought out, according to Hogben, because of the information it provided about the causal mechanisms of the developmental process. Statistical methodologies, such as Fisher's analysis of variance, were a useful starting point to "detect the existence of" hereditary differences and environmental differences, but then the scientist had to transition to interventionist experiments that elucidated how those differences made their difference in the causal mechanisms of development.[26]

Because, according to Hogben, the interaction of nature and nurture was a developmental phenomenon, it was to be commonly expected in nature. And this is why he answered the evidential question so differently from Fisher. There was Krafka's (1920) example, and Hogben added Taylor (1931) and Winton (1927), surmising, "The literature of experimental physiology is not wanting in examples." The interaction of nature and nurture, understood as Hogben conceptualized it, was the rule and not the exception.

In Fisher's last letter to Hogben, he began, "I think I see your point now. You are on the question of non-linear interaction of environment and heredity." The thesis of this chapter about the origins of interaction, in the plural, is that Fisher was partially right but also partially wrong in this statement. There was a sense in which Fisher did "see" Hogben's point—he realized Hogben was concerned about the interaction of nature and nurture. There was also a sense, though, in which Fisher did not "see" Hogben's point—he did not conceptualize, investigate, or judge the empirical evidence of that interaction at all like Hogben. Part of the divide between

Fisher and Hogben was surely political. But I have argued here that there was also an explanatory divide between them. They answered the conceptual, the investigative, and the evidential questions so differently because they came to consider the interaction of nature and nurture from two very different approaches, each with its own thing to be explained, causal question, thing that does the explaining, and methodology.[27]

## The Legacy of Fisher versus Hogben

The exchange between Fisher and Hogben evidently took its toll on their relationship. In 1932, when reviewing Hogben's *Genetic Principles*, Fisher welcomed Hogben's appointment to the Chair of Social Biology at the LSE. But in an unpublished review of Hogben's *Nature and Nurture*, where Hogben attacked Fisher, Fisher chided,

> Many of those, who had hopes that the establishment of a Chair of Social Biology at the London School of Economics would lead to a scientific and unbaissed [*sic*] attack on the social problems in this field, must by now be realising, in various degrees, their disappointment. For the functions of an advocate and of an investigator seem to be incompatible; and though one may be always amused and sometimes stimulated to thought when a brilliant journalist, such as Mr. G. K. Chesterton, sets out to show what a good forensic case can be made in opposition to the weight of scientific evidence and opinion, Professor Hogben lacks the charm of style needed to make confusion of thought seem luminous, or his facetiousness seem penetrating.[28]

Fisher's disdain for Hogben was by no means confined to the years of their debate. Almost thirty years later, when there was some confusion over whether an article in *Nature* was written by Anthony W. F. Edwards (Fisher's student) or his brother John Edwards (Hogben's student), Fisher wrote of the matter to his former University College London colleague R. R. Race:

> It was the thought that it was he [i.e., Anthony W. F. Edwards] that annoyed me, for the estimates published in *Nature* were manifestly incompetent, and I feared that one of my own pupils was running amok, and adding unnecessarily to darkness and confusion. However, I understand he [i.e., John Edwards] is only one of Hogben's, so all is explained.[29]

Fisher's Edwards, in fact, personally witnessed his mentor's disdain for Hogben upon the arrival of the paperback edition of Fisher's *The Genetical Theory of Natural Selection* (1958). "I was standing in the departmental office when Fisher opened the parcel of author's copies," Edwards recalled. "'Hmph,' [Fisher] said at his first sight of the cover, 'Looks like a book by Hogben'"[30] (Edwards 1990, 278).

Hogben lost no less love. In discussing the downfall of the Nazi Party in an unpublished portion of his autobiography, Hogben judged:

> After the war, the Nuremberg justices of the peace had [Nazi Alfred] Rosenberg hanged. If I believed in hanging people for their opinions, the only extenuating circumstances I might enter with a clear conscience as a plan for mercy on behalf of the late Sir R. A. Fisher would be that he did not occupy a government post with responsibility for implementing his convictions.[31]

That from an avowed pacifist!

Importantly, the debate between Fisher and Hogben did not just affect their personal relationship. Their separate routes to the interaction of nature and nurture generated two different concepts of interaction, two different assessments of why and how interaction should be investigated, and two different judgments of the empirical evidence for it. These different answers to the conceptual, investigative, and evidential questions outlived the debate between Fisher and Hogben and even outlived them. When the next debate over interaction erupted in 1969, Fisher was dead and Hogben was beginning his retirement in Glyn Ceiriog, Wales. And yet the explanatory divide arose again, this time surrounding the controversial question, "Why do African Americans perform poorer on IQ tests than Caucasians?"