Timofeeff-Ressovsky, N. (1940). Mutations and geographical variation, in J. Huxley, ed., *The New Systematics*: 73–135.
Volterra, V. (1931). *Leçons sur la théorie mathématique de la lutte pour la vie*, rédigées par Marcel Brelot, Paris, Editions Gauthier-Villars. Fac-simile: Paris, Editions Jacques Gabay, 1990. The lessons were actually given in Paris between 1928 and 1929.
Wright, S. (1931). Evolution in Mendelian Populations, *Genetics*, 16:97–159.
Wright, S. (1932). The Roles of Mutation, Inbreeding, Crossbreeding, and Selection in Evolution, *Proceedings of the 6th International Congress of Genetics*, 1:352–66.
Wright, S. (1939). *Statistical Genetics in Relation to Evolution*, Paris, Hermann.
Wright, S. (1977). *Evolution and the Genetics of Natural Populations*, vol. 3, *Experimental Results and Evolutionary Deductions*, Chicago and London, The Chicago University Press.

Historical Studies

Boesiger, E. (1980). Evolutionary biology in France at the time of the evolutionary synthesis, in E. Mayr and W. B. Provine, 1980:309–21.
Burian, R. M., Gayon, J., et Zallen, D. (1988). The Singular Fate of Genetics in the History of French Biology, 1900–1940, *Journal of the History of Biology*, 21:357–402.
Burian, R. M., and Gayon, J. (1989–90). Genetics after World War II: The Laboratories at Gif, in *Cahiers pour l'histoire du CNRS*, 6, 1989:108–10; 7, 1990:25–48.
Dorst, J. (1974). Notice sur la vie et l'oeuvre de Georges Teissier (1900–1972), membre de la section de zoologie, Paris, Palais de l'Institut.
Gayon, J. (1992). *Darwin et l'après-Darwin: une histoire de l'hypothèse de sélection dans la théorie de l'évolution*, Paris, Kimé.
Gillois, M. (1996). Malécot, in P. Tort, ed., *Dictionnaire du darwinisme et de l'évolution*, Paris, Presses Universitaires de France: 1768–1785.
Krimbas, C. (1995). Resistance and acceptance: Tracing Dobzhansky's influence in Europe. *Genetics of Natural Populations: The Continuing Importance of Theodosius Dobzhansky*, L. Levine, ed., New York, Columbia University Press: 23–36.
Lewontin, R. C. (1974). *The Genetic Basis of Evolutionary Change*. New York, Columbia University Press.
Lewontin, R. C. (1981). Introduction: The Scientific Work of Th. Dobzhansky, in *Dobzhansky's Genetics of Natural Populations I–XLIII*, R. C. Lewontin, J. A. Moore, W. B. Provine, and B. Wallace eds., New York, Columbia University Press: 93–115.
L'Héritier, P. (1981). Souvenirs d'un généticien-Revue de Synthèse, 102:331–50.
Mayr, E. (1980). The Arrival of Neo-Darwinism in France, in E. Mayr and W. B. Provine, 1980:321–2.
Mayr, E., and Provine, W. B., eds. (1980). *The Evolutionary Synthesis: Perspectives on the Unification of Biology*, Cambridge, MA, Harvard University Press.
Petit, C. (1995). L'implantation de la génétique des populations en France, in *Nature, histoire, société; Essais en hommage à Jacques Roger*, C. Blanckaert, J. L. Fischer, R. Rey, eds., Paris, Klinsieck, pp. 147–60.
Provine, W. B. (1986). *Sewall Wright and Evolutionary Biology*, Chicago, The University of Chicago Press.

In Thinking about Evolution: Historical, Philosophical, and Political Perspectives, R.S. Singh, C.B. Krimbas, D.B. Paul, and J. Beatty, eds. (Cambridge: Cambridge University Press, 2001), 103-118.

CHAPTER FIVE

Did Eugenics Rest on an Elementary Mistake?

DIANE B. PAUL AND HAMISH G. SPENCER

On the evidence of many genetics texts, of books on biology and society, and of histories of science, eugenicists were guilty of an astoundingly simple mistake. According to conventional accounts, which vary only in details, eugenics enthusiasts thought they could eliminate mental deficiency by segregating or sterilizing affected individuals. But a basic understanding of the Hardy–Weinberg principle suffices to destroy that illusion.

Eugenicists in the 1910s and 1920s attributed most mental defect to a recessive Mendelian factor (or in today's parlance, allele). But it is evident from the simple equation $p^2 + 2pq + q^2 = 1$ that if a trait is rare, the vast majority of deleterious genes will be hidden in apparently normal carriers. Selection against the affected themselves will thus be ineffectual. For example, even if all the affected were prevented from breeding, in a single generation the incidence of a trait at an initial frequency of 0.000100 would be reduced to just 0.000098 (and the allele frequency from 0.0100 to 0.0099). To reduce the incidence to half its original value (i.e., 0.000050) would require some 41 generations, or about 1000 years. Tables in numerous genetics textbooks serve to make the point that hundreds of generations are required before a rare deleterious trait would disappear. Because a human generation lasts about 25 years, eugenical selection would be futile over any meaningful period. P. B. and J. S. Medawar express a common view: the eugenicists were ignorant and muddled (as well as foolish and inhumane). "Only a minority of the offending genes are locked up in the mentally deficient themselves," they explain, "so sterilizing them would not be effective" (Medawar and Medawar 1977, p. 60).

That selection is slow when genes are rare is not a new insight. Indeed, Roll-Hansen (1980) has noted that, as early as 1914, the Norwegian psychiatrist Ragnar Vogt had emphasized the difference between "positive" (dominant) and "negative" (recessive) hereditary diseases, noting that the former could be eradicated by preventing those who have the disease from reproducing, whereas the latter "are transmitted through one or more healthy intermediate links, and only a small part of the diseased individuals have diseased parents. The socially most important hereditary diseases, such as certain kinds of deaf-muteness, feeblemindedness, and mental illness appear to behave like negative (recessive)

hereditary diseases, and one can therefore not achieve any appreciable eugenic effect by preventing the diseased individuals from having children through prohibition of marriage or similar means" (Vogt 1914, pp. 4–5). Vogt's point seems not to have been appreciated at the time, but in 1917 it was independently apprehended by Harvard geneticist Edward Murray East. East's argument was then refined by R. C. Punnett of Cambridge University, and Punnett's version was popularized by J. B. S. Haldane in Britain and H. S. Jennings in the United States. "To merely cancel the deficient individuals themselves – those actually feebleminded – makes almost no progress toward getting rid of feeblemindedness for later generations," wrote the latter (Jennings 1927, p. 273; Haldane 1928 [pub. in 1932], p. 105). If the futility of sterilization and segregation were exposed so early and often, it might seem that the numerous geneticists who endorsed these policies were a remarkably dim-witted lot.

Whatever their personal and political failings, this explanation is implausible. R. A. Fisher was a social reactionary, as well as ardent eugenicist. But his worst enemies did not think him stupid. He unquestionably understood the implications of Hardy–Weinberg. Moreover, when Punnett first articulated these implications, he did so in an effort to expand eugenics' scope, not demonstrate its futility. Indeed, in the 1920s and 1930s, nearly all geneticists, including those traditionally characterized as opponents of eugenics, took it for granted that "mental defectives" should be prevented from breeding. To see why few geneticists of that period drew the conclusions that seem so obvious to their present-day successors, let us review the original arguments about the threat represented by carriers.

5.1 The "Real Menace" of the Feebleminded

In his 1917 essay "Hidden Feeblemindedness," East argued that neither the character nor scope of the problem of mental defect had been fully appreciated. Although lauding efforts to cut off the stream of "defective germplasm" through segregation or sterilization of the affected, East thought the primary danger lay elsewhere in the vast mass of invisible heterozygotes.

He had been influenced in this view by the American psychologist Henry H. Goddard, author of *The Kallikak Family: A Study in the Heredity of Feeblemindedness* (1912), an impressionistic study of a "degenerate" rural clan, and *Feeble-Mindedness: Its Causes and Consequences* (1914), a much longer work that discussed the meaning of the data for theories of inheritance. In the latter book, Goddard had argued that "normal-mindedness is dominant and is transmitted in accordance with the Mendelian law of inheritance" (Goddard 1914, p. 556). His views were widely accepted. Thus Punnett could write in 1925 that no one "who has studied the numerous pedigrees collected by Goddard and others [could] fail to draw the conclusion that this mental state behaves as a simple recessive to the normal" (Punnett 1925, p. 704). William E. Castle also praised Goddard's research and uncritically reported his results. "Goddard's evidence," he wrote in an influential textbook, "indicates that feeble-mindedness is a recessive unit-character" (Castle 1927, p. 355). As late as 1930, Jennings was

able to assert that feeblemindedness was "the clearest case" of a recessive single gene defect (Jennings 1930, p. 238). Although Paul Popenoe and R. H. Johnson did criticize Goddard's assumption that feeblemindedness resulted from a single gene, they accepted his claims that at least two-thirds of those affected owed their condition directly to heredity and that they numbered at least 300,000 (Popenoe and Johnson 1918, pp. 105, 175).

Biometricians such as David Heron of the Galton Laboratory in London disparaged both the methods and logic used to reach this conclusion. In a passionate response to the stream of publications coming out of Charles B. Davenport's Eugenics Record Office in Cold Spring Harbor, Heron attacked almost every aspect of the Americans' work. Although his essay predated publication of *Feeblemindedness*, Heron's critique was as applicable to Goddard as Davenport. He concluded "that the material has been collected in an unsatisfactory manner, that the data have been tabled in a most slipshod fashion, and that the Mendelian conclusions drawn have no justification whatsoever" (Heron 1913, p. 61). Heron and the other biometricians were themselves ardent eugenicists, with "the highest hopes for the new science" (p. 4). But they feared that eugenics would be crippled at birth by the American Mendelians' crude errors. Perhaps because of their unremitting anti-Mendelian rhetoric and personal style of attack, the biometricians' critiques were largely ignored by Mendelian geneticists on both sides of the Atlantic (Spencer and Paul 1998).

Davenport was one of the few Mendelian geneticists to criticize the category of feeblemindedness, which he characterized as a "lumber room" of different (and separately inherited) mental deficiences (Davenport 1912, 1915). He also noted the illogic of expecting a socially defined trait – a feebleminded person was considered "incapable of performing his duties as a member of society in the position of life to which he is born" (Popenoe 1915, p. 32) – to be inherited as a simple Mendelian recessive (Davenport 1912, p. 286; see also Holmes 1923, pp. 121–33; Wiggam 1924, pp. 56–8). But these were minor quarrels. Until the mid-1930s, Thomas Hunt Morgan was the only Mendelian geneticist consistently to repudiate Goddard's claim that social deviance was largely due to bad heredity (Barker 1989). In the 1925 edition of *Evolution and Genetics*, Morgan argued that much of the behavior tagged with that label was probably due to "demoralizing social conditions" rather than to heredity (Morgan 1925, p. 201). But Morgan's critique, like Heron's, had little impact.

East was thus one of many geneticists to conclude that feeblemindedness was genetic and transmitted as a Mendelian recessive. But he was the first to see the implications for eugenics. Even without benefit of Hardy–Weinberg, East understood that the number of apparently normal carriers must be much larger than those affected. In 1912 Davenport could offer the following advice:

> Prevent the feebleminded, drunkards, paupers, sex-offenders, and criminalistic from marrying their like or cousins or any person belonging to a neuropathic strain. Practically it might be well to segregate such persons during the reproductive period for one generation. Then the crop of defectives will be reduced to practically nothing (Davenport 1912, p. 286).

Two years later, a committee of the American Breeders Association concluded almost as optimistically that two generations of segregation and sterilization would largely "eliminate from the race the source of supply of the great anti-social human varieties" (Laughlin 1914, p. 60). East realized that these predictions were wrong. The "real menace" of the feebleminded lay in the huge heterozygotic reserve, which constituted about 7% of the American population, or one in every fourteen individuals. He warned: "Our modern Red Cross Knights have glimpsed but the face of the dragon" (East 1917, p. 215).

East's point was echoed by Punnett, who earlier had suggested that feeblemindedness could be brought under immediate control. Like many other geneticists, he felt "there is every reason to expect that a policy of strict segregation would rapidly bring about the elimination of this character" (Punnett 1912, p. 137). But as a consequence of work for his influential 1915 book, *Mimicry in Butterflies*, he changed his mind.

For his mimicry work, Punnett needed to know how fast a Mendelian factor would spread through a population (Provine 1971, p. 137; Bennett 1983, pp. 8–10). He turned to his Cambridge mathematics colleague H. T. J. Norton for help. Norton prepared a table (which appears as an appendix to Punnett's book) displaying the number of generations required to change the frequency of completely dominant or recessive factors at different selection intensities (Punnett 1915). From the table, Punnett learned both that selection could act with surprising speed and that, when the recessive factor was rare, extreme slowness. Two years after *Mimicry in Butterflies* appeared, Punnett called attention to the implications of the latter point for eugenics. Policies aimed at the affected, he argued, would take a distressingly long time to work.

He employed a relatively well-understood condition to illustrate the nature of the problem:

> Albinism, for example, behaves on whole as a recessive. Nevertheless, albinos appear among the offspring in an appreciable proportion of matings where either one or both parents are normal, and where no consanguinity can be detected. The same is true of feeblemindedness. This becomes less difficult to understand when we realize that the heterozygotes are bound greatly to outnumber the recessives whenever these form a small proportion of a stable population (Punnett 1917, p. 465).[1]

Although that argument had already been made by East, Punnett was able to work out its implications with much greater precision. Applying the Hardy–Weinberg formula, he concluded that over 10% of the population carried the gene for feeblemindedness. With G. H. Hardy's help, he also estimated the rate at which a population could be freed from mental defect by a policy of segregating or sterilizing the affected. He found the results depressing. Even under the unrealistic assumption that all the feebleminded could be prevented from breeding, their proportion in the population would only decline from

1 in 100 to 1 in 1000 in 22 generations
1 in 1000 to 1 in 10,000 in 68 generations

1 in 10,000 to 1 in 100,000 in 216 generations
1 in 100,000 to 1 in 1,000,000 in 684 generations.

In other words, given Goddard's (unchallenged) estimate that about three in every thousand Americans were feebleminded as a result of genetic defect, it would take over 8000 years before their numbers were reduced to 1 in 100,000. Punnett thus concluded that eugenic segregation did not, contrary to his initial belief, offer a hopeful prospect.

Punnett, who served with Fisher on the Council of the Cambridge University Eugenics Society, did not intend to provide an argument against eugenics (Bennett 1983, p. 12). Like East, he concluded that if "that most desirable goal of a world rid of the feeble-minded is to be reached in a reasonable time, some method other than that of the elimination of the feeble-minded themselves must eventually be found" (Punnett 1917, p. 464). That method would take advantage of the phenomenon of partial dominance. East had noted that complete dominance was rare among the characters studied by plant and animal breeders. He speculated that intelligence tests (which Goddard had introduced to America in 1908) could be used to identify heterozygotes, who would likely exhibit a lower mentality than the "pure normals." Punnett took up the suggestion, concluding his paper with a call for research to focus on carriers of defective genes.

Whatever his intention, Punnett's claim about the inefficacy of selection was seized on by critics of eugenic segregation and sterilization. For example, in 1923 the Central Association for Mental Welfare issued a pamphlet opposing sterilization, which it asserted "would have only a very limited effect in preventing mental deficiency" (Central Association for Mental Welfare, 1923, p. 12). In the same year, the Section of Medical Sociology of the British Medical Association sponsored a discussion on the issue of sterilizing mental defectives. The opponents of such a policy were clearly familiar with Punnett's argument. Thus, Dr. Joseph Prideaux, the mental and neurological inspector of the Ministry of Pensions, argued that if the proportion of mental defectives in the population were 3 or 4 per 1000, it would be necessary to sterilize "some 10 percent of the population, who were carriers of mental defect" (a policy he thought absurd) and that, moreover, "no really good result would be forthcoming until a very long period had elapsed" (Prideaux 1923, p. 231). Dr. H. B. Brackenbury, the Section's president, ended his summary of the discussion by remarking that the more the hereditary impact of rigorous segregation "was looked into the more certain aspects of it appeared to be disappointing," and noting that it had formerly been hoped that complete segregation or sterilization would rapidly eliminate the mentally defective population, "but this was not so" (Brackenbury 1923, pp. 233–4).

R. A. Fisher (Fisher 1924) realized that Punnett's calculations were misleading and easily employed to subvert the eugenic goals that he and Punnett shared. If the goal were to rid the world of the last few mental defectives, Fisher noted, the fact that thousands of generations are required to reduce their number to

one in a billion would be meaningful. But if the calculations were extended to this point, "the reader would perhaps see the catch, and recognize that it would not matter if it took ten thousand generations to rid the world of its last lone feebleminded individual!" (p. 114). Even on Punnett's unrealistic assumptions of a single gene for mental defect and random mating, Fisher argued, substantial progress could be achieved in the first few generations if affected individuals were prevented from breeding. Expressing the frequency of the defectives as so many per 10,000 easily demonstrates the point:

From 100 to 82.6 in 1 generation
From 82.6 to 69.4 in 1 generation
From 69.4 to 59.2 in 1 generation.

Hence, in the first generation alone, selection could remove more than 17% of the affected persons.

Fisher's estimate is derived from Hardy's table, which represented an abstract calculation of the effects of selection, given assumptions about the initial number of affected. But the starting figures were chosen for ease of presentation rather than their assumed fit with reality. A standard estimate – and the one used by Punnett – was that three in a thousand individuals were feebleminded. Punnett's table could have been even more dramatic had he the skill to recalculate Hardy's numbers based on the lower initial frequency. But he understood little math. (In his 1916 referee's report on Fisher's classic paper, "The Correlation between Relatives on the Supposition of Mendelian Inheritance," Punnett wrote that it was of little interest to biologists but added; "frankly I do not follow it owing to my ignorance of mathematics" (Bennett 1983, p. 116, n. 12). If Fisher had used Punnett's estimate of the frequency of mental defect, the reduction in the first generation would have been about 10%.[2]

Fisher also examined the effects of relaxing Punnett's assumption of random mating. This time, however, the result was more favorable to eugenics. Fisher assumed that the feebleminded constituted a larger proportion (one sixteenth) of a smaller subsection (5%) of the population whose members mated only with others in that subsection. Hence, he incorporated a form of assortative mating into the model. Although it seems reasonable to assume that the feebleminded would tend to mate among themselves, the 5% figure dramatically decreases the frequency of carriers, thus increasing the efficacy of selection. Even starting from the standard frequency of 30 affecteds per 10,000 people, Fisher calculated that mental defect could be reduced by 36% in one generation (p. 115). Nevertheless, Fisher had shown for the first time that any form of assortative mating could help the eugenics cause.

Fisher's argument is often treated dismissively (see Kevles 1985, p. 165; Barker 1989). But Fisher diverged from Punnett and Jennings only in claiming that the affected tended to mate with each other (which would increase the frequency of homozygotes and thus speed selection) and that the trait was multifactorial. Both claims were eminently reasonable and at least as defensible as those of Punnett or Jennings – the conventional heroes of this historical

fable – who were also more alarmist than Fisher. It was the progressive Jennings who asserted that "a defective gene – such a thing as produces diabetes, cretinism, feeblemindedness – is a frightful thing; it is the embodiment, the material realization of a demon of evil; a living self-perpetuating creature, invisible, impalpable, that blasts the human being in bud or leaf. Such a thing must be stopped wherever it is recognized" (Jennings 1927, p. 274).

Fisher's primary criticism was leveled at the use of Hardy's table to demonstrate the inefficacy of selection. He was surely right in claiming that it was deceptive. What mattered to most eugenicists was the potential progress of selection in the next few generations. Here, Fisher demonstrated that eugenical policies could make a substantial difference. Even on Punnett's assumption of random mating, a substantial reduction in a single generation was possible.

In fact, all the geneticists agreed that the incidence of mental defect could be reduced by about 10% in the first generation (and on the same reasoning, 19 and 26% by the second and third generations, respectively). Even Haldane, who regarded compulsory sterilization "as a piece of crude Americanism" thought it "would probably cut down the supply of mental defectives in the next generation by something of the order of 10 percent." (Haldane 1938, pp. 80, 88). If some degree of assortative mating is assumed, the estimates would of course be higher. According to Jennings, the ostensible critic: "A reduction in the number of feebleminded by eleven percent [on the assumption of random mating], or still more, a reduction by thirty or forty percent [if mating is assortative], would be a very great achievement. And it could be brought about in no other way than by stopping propagation of the feeble-minded persons" (Jennings 1930, p. 242).

Why are the estimates so high? It is often said that eugenics was based on a mistake about the efficacy of selection against rare genes. But this was not the eugenicists' error. The crucial point is that feeblemindedness was not considered rare, at least in comparison with a trait like albinism. Thus, Davenport wrote that eugenics was prompted by recognition of the "great proportional increase in feeble-mindedness in its protean forms – a great spread of animalistic traits – and of insanity" (Davenport 1912, p. 308). Indeed, the raison d'etre of the eugenics movement was the perceived threat of swamping by a large class of mental defectives. Numerous British and American studies and an increase in the institutionalized population seemed to indicate that the problem was rapidly worsening. In America it was commonly believed that from 300,000 to 1,000,000 persons were feebleminded as a result of genetic defect (Popenoe and Johnson 1918); those figures tended to increase as mental tests came into wider use to evaluate students, prisoners, inmates of poorhouses and training schools, immigrants at Ellis Island, and army draftees. In 1912, Goddard tested New York City schoolchildren and estimated that 2% were probably feebleminded (Goddard 1912). The results of tests administered to army recruits during World War I were even more alarming, for they indicated that nearly half (47.3%) of the white draft – and 89% of the black – was feebleminded

(Yerkes 1921). Moreover, it seemed that the feebleminded were particularly prolific. For example, the British Royal Commission on the Care and Control of the Feeble-Minded reported in 1908 that defectives averaged seven children, whereas normal couples averaged only four; many other studies came to similar conclusions (see Paul 1995, pp. 78, 62).

Contemporary advocates of the futility of eugenics often mention Tay–Sachs disease, phenylketonuria (PKU), or albinism. Selection against such diseases is certainly futile. But these textbook examples are almost invariably rare conditions whose effects are either lethal or minor. Both their frequency and consequences ensure that they would be of little interest to a eugenicist. Individuals with Tay–Sachs and (with a few exceptions) untreated PKU do not leave offspring. Albinos and treated phenylketonurics do reproduce, but these conditions are not disabling. The frequent employment of albinism in texts is probably an unconscious inheritance from Punnett's original article. In 1917, Punnett had few examples to choose among.

Applied to the historical eugenics movement, the argument about the futility of selection against rare genes is simply irrelevant. Given widely shared assumptions about the causes and incidence of mental defect, eugenic policies could be expected to substantially reduce the number of affected. In any case, geneticists in the 1920s would generally have favored such policies whatever their exact effect. In *Heredity and Eugenics*, Ruggles Gates summarized Punnett's argument, concluding that even if all mental defectives were prevented from reproducing, "the most difficult part of the process of eliminating feeblemindedness from the germ plasm of the population would scarcely have begun" (1923, p. 159). But he ends the same chapter with a call for "the prevention of reproduction on the part of undesirables, such as the feebleminded," reasoning that, "such measures are necessary, not so much for the improvement of the race, as for arresting its rapid deterioration through the multiplication of the unfit" (p. 251). Indeed, most geneticists would have assented to Jennings' claim that "to stop the propagation of the feebleminded, by thoroughly effective measures, is a procedure for the welfare of future generations that should be supported by all enlightened persons. Even though it may get rid of but a small proportion of the defective genes, every case saved is a gain, is worth while in itself" (Jennings 1930, p. 238).

Like Jennings, Lancelot Hogben is often portrayed as an opponent of eugenics. He did criticize some advocates of sterilization for exaggerating the urgency of the problem and the results they could achieve – fearing that overstatement would harm the cause. He also invoked Fisher to argue that there is no need to overstate potential results. That we cannot do everything "is not a valid reason for neglecting to do what little can be done" (Hogben 1931, p. 207). That point was echoed by Edwin G. Conklin, who like Jennings and Hogben, criticized some eugenic proposals. Conklin once remarked that sterilizing all the inmates of public institutions was "like burning down a house to get rid of the rats" (Conklin 1916, p. 438). But he did not oppose sterilization

of the feebleminded. On the contrary, he asserted that "all modern geneticists approve the segregation or sterilization of those who are known to have serious hereditary defects, such as hereditary feeblemindedness, insanity, etc." Conklin asked of the American Eugenics Society's proposed sterilization policy; "Can any serious objection be urged to such a law?" (Conklin 1930, pp. 577–8). In 1930, this question was unambiguously rhetorical.

Nearly all geneticists of the 1920s and 1930s – including those traditionally characterized as opponents of eugenics – took for granted that the "feebleminded" should be prevented from breeding. Moreover, nearly everyone agreed on the scientific facts. Punnett, East, Fisher, Jennings, and even Haldane made roughly the same estimates as to the speed and scope of eugenical selection. But in respect to social policy, the facts did not speak for themselves. They required interpretation in light of other assumptions and goals. Thus, Haldane opposed sterilization, arguing that "with mental defects as with physical defects, if you once deem it desirable to sterilize I think it is a little difficult to know where you are to stop" (Haldane 1938, p. 89). That is a powerful argument. But it is a social, not a scientific one. Lionel Penrose was an even more vehement and consistent critic of eugenics. An expert in the genetics of mental deficiency, he stressed the heterogeneity of its causes and the modest influence of eugenic measures in reducing its incidence. But his main argument was ethical. Penrose maintained that the best index of a society's health is its willingness to provide adequate care for those unable to care for themselves (Penrose 1949; see also Kevles 1985, esp. 151–63).

The Hardy–Weinberg theorem meant different things to different people. To those already disposed against eugenics, it proved that policies to prevent the feebleminded from breeding were not worth the effort. There is no reason that those disposed in favor of eugenics should draw the same conclusion. Whether a 10% reduction in incidence is large or small is not a question science can answer. Indeed, one may concede that the percent reduction is small yet still think it worthwhile. Thus, at the close of a long discussion of the implications of Hardy–Weinberg, Curt Stern remarked: "To state that reproductive selection against severe physical and mental abnormalities will reduce the number of affected from one generation to the next by only a few percent does not alter the fact that these few percent may mean tens of thousands of unfortunate individuals who, if never born, will be saved untold sorrow" (Stern 1949, p. 538). A similar point was made by the Swedish Commission on Population in its 1936 report on sterilization. After acknowledging the falsity of the earlier belief that sterilization would result in a rapid improvement of the population, the authors note that it would still result in gradual improvement while preventing possible deterioration and that "whenever an eugenic sterilization is carried out... in the specific case the operation will prevent the birth of sick or inferior children or descendants. Owing to this, sterilization of hereditarily sick or inferior human beings is still a justified measure, beneficial to the individual as well as to society" (quoted in Broberg and Tydén 1996, p. 106). Thus,

it may not matter if the reduction in absolute numbers is miniscule. Indeed, if one assumes with Jennings that "the prevention of propagation of even one congenitally defective individual puts a period to at least one line of operation of this devil" and that "to fail to do at least so much would be a crime," the rate of selection is simply beside the point (Jennings 1927, p. 274).

We began by asking whether eugenics was based on an elementary mistake. To the extent that support for eugenical segregation and sterilization was based on the assumption that "it would be possible at one fell stroke [to] cut off practically all of the cacogenic varieties of the race," (Laughlin 1914, p. 47) a loose definition of "feeblemindedness," as well as acceptance of Goddard's shoddy data and defective logic, the answer is yes. But it was possible to recognize these flaws and still remain a eugenicist, as the example of David Heron demonstrates. Moreover, what is usually characterized as the eugenicists' most obvious error – a failure to understand the implications of the Hardy–Weinberg theorem – was a mistake few geneticists made after 1917. By the 1920s, they well understood that the bulk of genes for mental defects would be hidden in apparently normal carriers. For most geneticists, this appeared a better reason to widen eugenic efforts than to abandon them.

It is often said that support for eugenics declined in the 1930s as its scientific errors were exposed. But the eugenics movement grew stronger during the Depression (see Paul 1995, pp. 72–90). In the United States, the number of sterilizations climbed. The procedure was legalized in Germany (1933), the Canadian Province of British Columbia (1933), Norway (1934), Sweden (1934), Finland (1935), Estonia (1936), Iceland (1938) and Japan (1940). Denmark, which in 1929 had legalized "voluntary" sterilization, permitted its coercive use on mental defectives in 1934. These laws were generally applauded by geneticists.

In 1918, Popenoe and Johnson wrote that "so few people would now contend that two feeble-minded or epileptic persons have any 'right' to marry and perpetuate their kind, that it is hardly worth while to argue the point" (Popenoe and Johnson 1918, p. 170). Assumptions we now take for granted they thought too absurd even to require challenging. The inversion of these assumptions in recent decades is best explained by political developments. Revelations of Nazi atrocities, the trend toward respect for patients' rights in medicine, and the rise of feminism have converged to make reproductive autonomy a dominant value in our culture. In 1914, a committee of the American Breeders Association asserted that "society must look upon germ-plasm as belonging to society and not solely to the individual who carries it" (Laughlin 1914, p. 16). Few today would profess such a view. A change in values, and not the progress of science, explains why contemporary Swedes would be unlikely to concur with the 1936 commission that criticized as "extremely individualistic" the concept that individuals have a right to control their own bodies (Broberg and Tydén 1996).

It is not our superior quantitative skills that explain why we today draw very different implications from the Hardy–Weinberg theorem. There was nothing wrong with most eugenicists' math. Our concept of rights, however, is much more expansive than theirs. That is why the same equation holds different lessons for them than it does for us.

5.2 Acknowledgments

Having met as research associate (Diane B. Paul) and graduate student (Hamish G. Spencer) in Richard Lewontin's laboratory, the authors are especially grateful to him and for his having outlined for us some of the arguments in the appendix. We also thank Evelyn Fox Keller, J. Maynard Smith, Michael Ruse, Edna Seaman, and Graham Wallis for reading the manuscript; Leila Zenderland for comments on Goddard; Nils Roll-Hansen for bringing Ragnar Vogt's argument to our attention; the Division of Sciences, University of Otago, for financial support; and the staff of the Science Library, University of Otago, for help with references.

APPENDIX

In this appendix we compare the efficacy of selection under Goddard's single-locus model with that under a simple quantitative model as suggested by Fisher. The argument we use in examining the latter follows suggestions made to us by R. C. Lewontin.

Let us first examine the dynamics of the Mendelian model in which the feebleminded are homozygous for a recessive allele. Let F be the proportion of feebleminded in the population, and thus the frequency q of the dysgenic allele is the square root of F. On the assumption that none of the feebleminded reproduce (i.e., that eugenic selection is complete), the allele frequency after one generation is given by the equation

$$q' = \frac{q}{1+q}.$$

Hence, the percentage drop in the incidence of feeblemindedness is given by

$$\begin{aligned} 100(F' - F)/F &= 100(q'^2 - q^2)/q^2 \\ &= -100q(2+q)/(1+q)^2 \\ &= -100\sqrt{F}(2+\sqrt{F})/(1+\sqrt{F})^2 \end{aligned}$$

This function is shown in Figure 5.A1. We can also use this model to see how long eugenic selection takes to reduce the proportion of feebleminded by a certain fraction, say 50%. The allele frequency after n generations of selection is given by

$$q_n = (n + q^{-1})^{-1},$$

which can be rearranged to give

$$n = q_n^{-1} - q^{-1}.$$

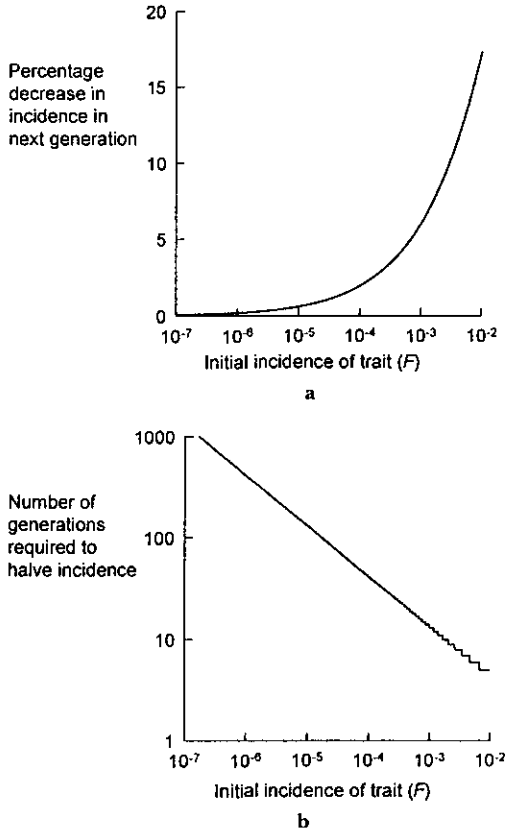

Figure 5.A1. (a) The effect of eugenic selection against a recessive Mendelian trait in one generation. (b) The time required to at least halve the frequency of a dysgenic recessive Mendelian trait. (The stepped nature of the graph in b is because there is no integer solution to the equation; see text.) Note the logarithmic x-axis in both figures and the logarithmic y-axis in b. We have assumed that the eugenic selection is complete (i.e., no affected individuals have children). The frequency q of the dysgenic allele is the square root of the incidence F.

Because halving the incidence of a trait reduces the allele frequency by a factor of $\sqrt{2}$, the number of generations required to halve the incidence is given by

$$(q/\sqrt{2})^{-1} - q^{-1} = (\sqrt{2} - 1)/\sqrt{F}$$

Because n will not in general be a whole number (especially for high incidences), the number of generations required to at least halve the incidence is given by rounding n upwards, that is $[n+1]$. This function is also shown in Figure 5.A1.

Let us now examine the quantitative model in which individuals with a mental ability below a threshold t are considered to be the feebleminded. Suppose that

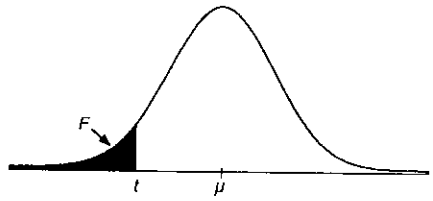

Figure 5.A2. The distribution of mental ability modeled as a quantitative trait with mean μ. The feebleminded are those with mental ability below the threshold t and make up a proportion F of the population equal to the shaded area under the curve.

mental ability has a heritability of 1.0 and is determined by a large number of additive loci, which are two assumptions most favorable to Fisher's argument. The trait will then be normally distributed, with a mean μ and a standard deviation σ. The proportion of feebleminded in the population, F, is the area under the normal curve to the left of t, as shown in Figure 5.A2. This value is easily found from tables of the standard normal distribution. In symbols,

$$F = \int_{-\infty}^{t} \phi(x)\, dx,$$

in which $\phi(x)$ is the normal density function. If all the feebleminded are prevented from breeding, the mean in the subsequent generation, μ', will be larger:

$$\mu' = \mu + h^2(\mu_s - \mu),$$

in which h^2 is the heritability of the trait, and μ_S is the mean of the selected or breeding population. The difference in parentheses is easily found from the properties of the normal distribution, is well known to quantitative geneticists (Falconer 1989, p. 191), and is usually denoted as S:

$$S = \mu_S - \mu = \sigma^2\phi(t)/(1 - F)$$

Now, because the heritability is 1.0, the distribution of the subsequent generation will be normal with a mean μ_S. That is, the distribution of mental ability is moved a distance S to the right. The proportion of feebleminded in the subsequent generation, F', is thus given by

$$F' = \int_{-\infty}^{t} \phi(x - S)\, dx,$$

with the proportional reduction in the proportion of feebleminded given by

$$(F - F')/F.$$

Using the values relevant to Fisher's argument, we find that if $F = 0.01$, then $\phi(t) = 0.0266$ and $S = 0.0269\sigma^2$, giving $F' = 0.0093$. The quantitative model predicts a proportional decrease in feeblemindedness in one generation of eugenic selection, therefore, of about 7% compared with the 17% reduction predicted from the single-locus Mendelian model. The proportional changes for other values of F are shown in Table 5.A1.

Table 5.A1. *Percentage Decrease in Feeblemindedness under*

F	Mendelian Model	Quantitative Model
0.010	17.36	6.95
0.003	10.12	2.76
0.001	6.04	1.13

Notes

1. Indeed most texts continue to treat albinism – as Punnett did – as a single-locus defect. Falconer (1989) is one of the few exceptions. But it has long been known that albinism arises from the actions of recessive genes from at least two loci (McKusick 1992). Consequently, the incidence of homozygous recessives for a particular locus is lower than most texts suggest, and eugenic selection against albinism would be even less efficacious.
2. In emphasizing that his presentation was based on Punnett's assumptions, Fisher traded on this weakness. A reader could easily presume that Fisher employed assumptions favorable to Punnett's case. He did not. "In a single generation," Fisher wrote, "the load of public expenditure and personal misery caused by feeblemindedness would be reduced by over 17 percent." If based on the figures in Punnett's table, this estimate is correct but also misleading. Fisher did add that if the starting point had been thirty instead of a hundred (per ten thousand), the reduction in one generation would be "over 11 per cent" (p. 114). But he failed to note that this is the relevant figure. In fact, Fisher's 11% figure is still inflated. The true reduction is approximately 10.1%. Haldane (1938, p. 88) discreetly gives the correct value – "something of the order of 10 percent" – but less mathematically inclined writers, such as Jennings (1930, p. 242), appear not to have noticed Fisher's error.

REFERENCES

Barker, D. (1989). The biology of stupidity: Genetics, eugenics and mental deficiency in the inter-war years. *Brit. J. Hist. Sci.* 22:347–75.

Bennett, J. H. (1983). *Introduction to Natural Selection, Heredity, and Eugenics.* Oxford, UK: Clarendon Press.

Brackenbury, H. B. (1923). The President's Summary of the Discussion. *British Medical Journal* 2:233–4.

Broberg, G., and Tydén, M. (1996). "Eugenics in Sweden: Efficient Care." In *Eugenics and the Welfare State: Sterilization Policy in Denmark, Sweden, Norway, and Finland,* eds. G. Broberg and N. Roll-Hansen. East Lansing: Michigan State University Press.

Castle, W. E. (1927). *Genetics and Eugenics.* Cambridge, MA: Harvard University Press.

Central Association for Mental Welfare (1923). Sterilization and mental deficiency. *Studies in Mental Inefficiency* 4:10.

Conklin, E. G. (1916). *Heredity and Environment in the Development of Man,* 2d edition. Princeton, NJ: Princeton University Press.

Conklin, E. G. (1930). The purposive improvement of the human race. In *Human Biology and Population Improvement,* ed. E. V. Cowdry, pp. 566–88. New York: Hoeber.

Cowan, R. S. (1977). Nature and nurture: The inter-play of biology and politics in the work of Francis Galton. In *Studies in the History of Biology,* eds. W. Coleman and C. Limoges, pp. 133–208. Baltimore: Johns Hopkins University Press.

Darwin, L. (1928). *What is Eugenics?* London: Watts.

Davenport, C. B. (1912). The inheritance of physical and mental traits of man and their application to eugenics. In *Heredity and Eugenics,* eds. W. E. Castle, J. M. Coulter, C. B. Davenport, E. M. East, W. L. Porter, pp. 269–88. Chicago: University of Chicago Press.

Davenport, C. B. (1915). Review of H. H. Goddard, Feeblemindedness: Its Causes and Consequences. *Science* 42:837–8.

East, E. M. (1917). Hidden feeblemindedness. *Journal of Heredity* 8:215–17.

Falconer, D. S. (1989). *Introduction to Quantitative Genetics,* 3d ed. Harlow, UK: Longman.

Fisher, R. A. (1924). The elimination of mental defect. *Eugenics Review* 26:114–16. (Reprinted in *Collected Papers of R. A. Fisher I,* ed. J. H. Bennett, 1971 edition. Adelaide, Australia: University of Adelaide.)

Gates, R. R. (1923). *Heredity and Eugenics.* London: Constable.

Goddard, H. H. (1912). *The Kallikak Family: A Study in the Heredity of Feeblemindedness.* New York: MacMillan.

Goddard, H. H. (1914). *Feeblemindedness: Its Causes and Consequences.* New York: MacMillan.

Haldane, J. B. S. (1932). *The Inequality of Man and Other Essays.* London: Chatto and Windus.

Haldane, J. B. S. (1938). *Heredity and Politics.* London: Unwin.

Heron, D. (1913). Mendelism and the problem of mental defect. I. A criticism of recent American work. In *Questions of the Day and of the Fray No. VII.* London: Cambridge University Press.

Hogben, L. (1931). *Genetic Principles in Medicine and Social Science.* London: Williams and Norgate.

Holmes, S. J. (1923). *Studies in Evolution and Genetics.* New York: Harcourt, Brace.

Jennings, H. S. (1927). Health progress and race progress: Are they incompatible? *Journal of Heredity* 18:271–6.

Jennings, H. S. (1930). *The Biological Basis of Human Nature.* London: Faber and Faber.

Kevles, D. J. (1985). *In the Name of Eugenics: Genetics and the Uses of Human Heredity.* New York: Knopf.

Laughlin, H. H. (1914). Report of the Committee to Study and to Report on the Best Practical Means of Cutting Off the Defective Germ-Plasm in the American Population. I. The Scope of the Committee's Work. Eugenics Record Office, Bulletin No. 10A. Cold Spring Harbor, New York.

McKusick, V. A. (1992). *Mendelian Inheritance in Man: Catalogue of Autosomal Dominant, Autosomal Recessive, and X-Linked Phenotypes.* 10th ed. Baltimore: Johns Hopkins University Press.

Medawar, P. B., and Medawar, J. S. (1977). *The Life Sciences: Current Ideas in Biology.* New York: Harper and Row.

Morgan, T. H. (1925). *Evolution and Genetics,* 2d ed. Princeton, NJ: Princeton University Press.

Paul, D. B. (1995). *Controlling Human Heredity: 1865 to the Present.* Amherst, NY: Humanity Press.

Penrose, L. S. (1949). *Biology of Mental Defect.* New York: Grune and Stratton.
Popenoe, P. (1915). Feeblemindedness. *Journal of Heredity* 6:32–6.
Popenoe, P., and Johnson, R. H. (1918). *Applied Eugenics.* New York: MacMillan.
Prideaux, J. F. E. (1923). Comment. *British Medical Journal* 2:231–2.
Provine, W. B. (1971). *The Origins of Theoretical Population Genetics.* Chicago: University of Chicago Press.
Punnett, R. C. (1912). Genetics and eugenics. In *Problems in Eugenics.* First International Eugenics Congress, 1912, pp. 137–8. New York and London: Garland Publishing, 1984.
Punnett, R. C. (1915). *Mimicry in Butterflies.* Cambridge, UK: Cambridge University Press.
Punnett, R. C. (1917). Eliminating feeblemindedness. *Journal of Heredity* 8:464–5.
Punnett, R. C. (1925). As a biologist sees it. *The Nineteenth Century* 97:697–707.
Roll-Hansen, N. (1980). Eugenics before World War II. The case of Norway. *History and Philosophy of the Life Sciences* 2:269–98.
Spencer, H. G., and Paul, D. B. (1998). The failure of a scientific critique: David Heron, Karl Pearson and Mendelian eugenics. *British Journal for the History of Science* 31:441–52.
Stern, C. (1949). *Principles of Human Genetics.* San Francisco: Freeman.
Vogt, R. (1914). Det Norske Videnskabsakademi i Kristiania, Forhandlinger (13 Feb.):4–5.
Wiggam, A. (1924). *The Fruit of the Family Tree.* Indianapolis, IN: Bobbs–Merrill.
Yerkes, R. M. (1921). *Psychological Examining in the United States Army.* vol. 15. Memoirs of the National Academy of Sciences. Washington, D.C.

CHAPTER SIX

Can the Norm of Reaction Save the Gene Concept?

RAPHAEL FALK

The term *norm-of-reaction* was born in the same year as that of the gene. In June 1909, Richard Woltereck (1877–1944) presented his research *Weitere experimentelle Untersuchungen über Artveränderung, speziell über das Wesen quantitativer Artunterschiede bei Daphniden* (Further investigations on change of species, specifically on the nature of quantitative species-differences in Daphnides) to the German Zoological Society. In it Woltereck declared his objective to provide a rejoinder to what he called the Mendelian teaching in the footsteps of Weismann and de Vries, or the de Vries–Johannsen conception of the origin of specific types (specific-kinds, biotypes)[1] by abrupt hereditary changes, that is, by mutations rather than by continuous small changes (Woltereck 1909). Woltereck's work was specifically intended to be a contribution to the reestablishment of the Darwinian conception of gradual and continuous evolution of species as opposed to the growing sentiment for evolution by saltations.

In his *Die Mutationstheorie* de Vries resurrected a typological conception of taxonomy, reaching down to the level of the individual organism. He claimed that until the time of Linnaeus it was actually the genus (*Gattung*) that was the unit of taxonomy: To avoid the lengthy and tiresome lists of variations that accompanied the descriptions of such genera, Linnaeus "raised" species (*Arten*) to the basic entities of taxonomy (de Vries 1902–3, I. pp. 12–13). If so, there was no compelling need to accept the species as the fundamental entity of taxonomy, and the distinct segregating Mendelian unit-characters might be the ones that denote the fundamental discontinuous *type* of taxonomy. It was, however, Wilhelm Johannsen who threatened to consolidate a new typological taxonomy on the foundation offered by genotypic Mendelian discontinuity versus apparent phenotypic continuity (Johannsen 1909). Johannsen's studies in pure lines were closely linked to his concept of grouping and classification of natural entities. His notion was that the genotype, the "constant form-type" of the pure lines, being the purest realization of the basic unit of classification, was actually a direct implementation of Aristotelian typology (Roll-Hansen 1978, pp. 221 ff.). The discontinuity between types (genotypes) is biologically true and meaningful. It reflects the *discontinuous* or saltational character, as opposed to the *continuous* character, of the Darwinian intra- as