# The hidden science of eugenics

Diane B. Paul and Hamish G. Spencer

The early eugenicists were not stupid, but they did not share our social values. The rise and fall of the eugenics movement is a history that modern medical geneticists would do well to heed.

Many textbooks suggest that eugenicists were guilty of an astoundingly simple mistake. According to conventional accounts, enthusiasts about eugenics 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 clear from the equation  $p^2 + 2pq + q^2 = 1$  that if a trait is rare, most deleterious genes will be hidden in apparently normal carriers. Selection against those actually affected will thus be ineffective. Tables and formulas in many general biology and genetics textbooks (for example, refs 1–4) serve to make the point that hundreds of generations are required before a rare deleterious trait would disappear.

It is true that many eugenicists were muddled about genetics. But what about the host of respected geneticists, such as R. A. Fisher in the United Kingdom, Erwin Bauer in Germany, Herman Nilsson-Ehle in Sweden and Edward Murray East in the United States, who championed eugenics long after the implications of the Hardy-Weinberg principle were understood? The insight that selection is slow when genes are rare originated in 1917 and was popularized in the 1920s by J. B. S. Haldane in the United Kingdom and H. S. Jennings in the United States. Yet 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. Moreover, the geneticists who first discussed the social implications of the Hardy-Weinberg principle did so in an effort to expand the scope of eugenics, not demonstrate its futility.

#### **Invisible danger**

In his 1917 essay "Hidden Feeblemindedness"<sup>5</sup>, the Harvard geneticist East lauded efforts to cut off the stream of "defective germplasm" through segregation or sterilization of the affected. But East thought that the primary danger lay elsewhere, in the vast mass of invisible heterozygotes.

East had been strongly influenced by the psychologist Henry H. Goddard, author of *The Kallikak Family: A Study in* the Heredity of Feeble-Mindedness, a chronicle based on data collected by a field-worker who traced family members and assessed their mental and moral state. Many family members were of course dead or could not be located. Their mentality and character were assessed on the basis of hearsay. Judgements of both the living and dead were swift and subjective.

Two years later, in 1914, Goddard published Feeble-Mindedness: Its Causes and Consequences, in which he discussed the meaning of the Kallikak data for theories of inheritance. He argued that "normal-mindedness" is a dominant trait inherited in a Mendelian fashion; an individual lacking the factor for normal mentality would be feebleminded — "incapable of performing his duties as a member of society in the position of life to which he is born" 6.

The recessive theory of mental defect was widely accepted by Mendelians. The Cambridge geneticist R. C. Punnett spoke for many when he wrote that no one "who has studied the numerous pedigrees collected by Goddard and others [could] fail to draw the conclusion that this mental state [that is, feeblemindedness] behaves as a simple recessive to the normal"<sup>7</sup>. Charles Davenport did note the illogicality of expecting a socially defined trait to be inherited as a simple Mendelian recessive; he thought there were many different (and separately inherited) mental deficiencies8. A few geneticists also contested Goddard's claim that feeblemindedness was caused by a single Mendelian factor9. But these were minor quarrels. With the exception of Thomas Hunt Morgan, who argued that much of the behaviour associated with feeblemindedness arose from "demoralizing social conditions"10, no Mendelian geneticist before the 1930s rejected Goddard's claim that social deviance was largely due to bad recessive — heredity11.

In 1912 Davenport offered 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.

At the time, such predictions were common. Even without the benefit of Hardy-Weinberg, East realized that they were wrong. The "real menace" of the feebleminded, he argued, lay in the huge heterozygotic reserve, constituting about seven per cent of the US population, or one in every fourteen individuals. East sounded an alarm: "Our modern Red Cross Knights have glimpsed but the face of the dragon." 5

### A question of time

His point was echoed by Punnett. For his influential Mimicry in Butterflies (1915), Punnett needed to know how fast a Mendelian factor would spread through a population<sup>12</sup>. His Cambridge mathematics colleague, H. T. J. Norton, prepared a table displaying the number of generations required to change the frequency of completely dominant or recessive factors at different selection intensities. Punnett called attention to the table's implications for eugenics. Policies aimed at the affected, he argued, would take a distressingly long time to work. The Hardy-Weinberg formula indicated that more than ten per cent 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 defects by segregating or sterilizing the affected. Even under the unrealistic assumption that all the feebleminded could be prevented from breeding, it would take more than 8,000 years before their numbers were reduced to 1 in 100,000, given Goddard's estimate that about 3 in 1,000 Americans were genetically feebleminded. Punnett concluded that eugenic segregation did not, contrary to his initial belief, seem hopeful<sup>13</sup>.

Punnett, who served with Fisher on the Council of the Cambridge University Eugenics Society, did not intend to provide an argument against eugenics. In fact, he explicitly endorsed both East's scientific point and his policy proposals. Like East, he aimed to convince his readers of the need to identify the carriers of defective genes. "Clearly if that most desirable goal of a world rid of the feebleminded is to be reached in a reasonable time," he asserted, "some method other than that of the elimination of the feebleminded themselves must eventually be found." 13

According to Fisher, Punnett's goal was subverted by opponents of eugenics, who seized on his table to argue that segregation and sterilization worked too slowly to justify the effort. In a 1924 article, "The Elimination of Mental Defect", Fisher argued that Punnett's calculations

NATURE · VOL 374 · 23 MARCH 1995

obscured the fact that selection would initially be rapid. And for all practical purposes, he noted, that is what matters. Even under Punnett's unrealistic assumptions of a single gene for mental defect and of random mating, he argued, substantial progress could be achieved in the first few generations if affected individuals were prevented from breeding. In the first generation alone, the reduction would be more than 11 per cent<sup>14</sup>.

Notwithstanding some technical differences, Jennings, Punnett and Fisher agreed that mental defectives should be prevented from breeding. Jennings, who is sometimes portrayed as an opponent of eugenics, asserted that a gene that produces feeblemindedness "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."15 Fisher diverged from Punnett and Jennings (who asserted as late as 1930 that feeblemindedness was "the clearest case" of a recessive single gene defect<sup>16</sup>) only in claiming that the affected tended to mate with each other and that the trait was multifactorial. All agreed that the incidence of mental defect could be reduced by at least 10 per cent in the first generation and by 19 and 26 per cent 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"<sup>17</sup>.

Why were the estimates so high? It is often said that eugenics was based on a mistake about the efficacy of selection against rare genes. But few geneticists made this error, at least after 1917. Feeblemindedness was not considered to be rare. Indeed, the *raison d'être* of the eugenics movement was the perceived threat of swamping by a large and rapidly growing class of mental defectives.

According to eugenicists, the number of physical and mental defectives would once have been kept in check by natural selection. But in civilized societies, it seemed that the process had practically ceased. Medicine and public charity now kept the 'unfit' alive. Worse, these failures were now reproducing faster than their betters. In the first three decades of this century, a raft of studies seemed to demonstrate the high fertility of the feebleminded. For example, the British Royal Commission on the Care and Control of the Feeble-Minded reported in

1908 that defectives averaged seven children, normal couples only four. In the United States it was commonly believed that between 300,000 and 1,000,000 people were feebleminded as a result of genetic defects<sup>9</sup>; the figures tended to increase as mental tests came into wider use. In 1912, Goddard tested New York City schoolchildren and estimated that two per cent were probably feebleminded. The results of tests given to army recruits during the First World War were even

## IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

Breeding propaganda — London Eugenics Society poster, c. 1935.

more dramatic: they indicated that 47.3 per cent of the white draft and 89 per cent of the black draft were feebleminded<sup>18</sup>.

Contemporary textbook examples of the futility of eugenics often mention Tay-Sachs disease, phenylketonuria and albinism. Selection against such conditions is certainly futile. But they are usually rare and their effects either lethal or minor. Victims of Tay-Sachs and (with a few exceptions) untreated phenylketonuria do not leave offspring. Albinos and treated phenylketonuriacs reproduce, but these conditions are not disabling. And with regard to the potential increase in genes for treatable genetic diseases, the Hardy-Weinberg argument is relevant<sup>19</sup>. Most single-gene disorders are extremely rare and would spread slowly in the population. Moreover, some disease genes may be maintained at high frequency because of heterozygote advantage; although this is difficult to demonstrate, some geneticists believe it explains not only the well-confirmed case of sickle-cell anaemia, but also cystic fibrosis and Tay-Sachs (in Ashkenazi Jews), among other diseases<sup>20</sup>. Selection here is futile.

But these arguments do not apply to the early eugenics movements. Given widely shared assumptions about the incidence and causes of feeblemindedness, eugenic policies could be expected to reduce substantially the number of affected individuals. In the famous case of Buck v. Bell (1927), decided in the wake of US Army mental tests, Justice Holmes upheld the Virginia sterilization law "to prevent our being swamped with incompetence". Commenting on this passage, one author remarks that "what such reasoning fails to take into account is that. . . sterilization will have almost no effect on the frequency of the disease"1. The point is illustrated by a recessive disorder affecting 1 in 40,000 individuals.

In any case, geneticists in the 1920s would generally have favoured eugenic policies whatever their exact effect. Most would have assented to Jennings's 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 worthwhile in itself" 16.

Like Jennings, Lancelot Hogben is often portrayed as an opponent of eugenics. Hogben did criticize some advocates of sterilization for exaggerating both the extent of the problem and efficacy of their solution. He also argued that the fact that we cannot do everything "is not a valid reason for neglecting to do what little can be done"21. His point was echoed by E. G. Conklin, who, like Jennings and Hogben, criticized some eugenic proposals. But Conklin approved of the segregation or sterilization of the feebleminded. He asked of the American Eugenics Society's proposed sterilization policy: "Can any serious objection be urged to such a law?"2

Punnett, East, Fisher, Jennings and even Haldane made roughly the same estimates of the speed and scope of eugenic selection. But the facts did not speak for themselves. They required interpretation in the 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"<sup>17</sup>. This is a powerful argument. But it is a social, not a scientific one. Lionel Penrose was the most vehement geneticist 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<sup>23</sup>.

The Hardy-Weinberg theorem meant different things to different people. For example, Curt Stern once 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 per cent does not alter the fact that these few per cent may mean tens of thousands of unfortunate individuals who, if never born, will be saved untold sorrow"24. It may not even matter if the reduction in absolute numbers is minuscule: the rate of selection is immaterial 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. To fail to do at least so much would be a crime."

Many advocates of sterilization employed a loose definition of 'feeblemindedness', accepted Goddard's defective data and logic or assumed that "it would be possible at one fell stroke [to] cut off practically all of the cacogenic varieties of the race"25. But it was possible to recognize all these flaws and still remain a eugenicist. After 1920, it was well understood that most genes for mental defects would be hidden in apparently normal carriers. For most geneticists this seemed to be a good reason to widen eugenic efforts rather than abandon them.

This implication makes sense in the light of social values. 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"9.

Politics changed these assumptions. During the 1940s and 1950s, many geneticists tried to distinguish the race- and class-biased eugenics of the past from a new eugenics that focused on disease. But attempts to distinguish good from bad eugenics were ultimately unsuccessful. Nazi atrocities gave eugenics of any kind a bad name and produced a backlash against the view that the state had a legitimate interest in who reproduced.

That reproduction should be a private matter was strongly reinforced by a trend towards respect for patients' medical rights, the development of a broad jurisprudence of privacy and the rise of feminism. By the 1960s, reproductive autonomy had become a dominant cultural value. This was a far cry from the assertion of a 1914 committee of the American Breeders Association: "Society must look upon germ-plasm as belonging to society and not solely to the individual who carries it"25. A change in values, and not the progress of science, explains why few Swedes would now agree with the 1936 commission that criticized as "extremely individualistic" the notion that individuals have a right to control their own bodies<sup>26</sup>.

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. In the United States, the number of sterilizations increased. Sterilization was legalized in Germany (1933), British Columbia, Canada (1933), Norway (1934), Sweden (1934), Finland (1935), Estonia (1936) and Iceland (1938). Denmark, which in 1929 had legalized 'voluntary' sterilization, permitted its coercive use on mental defectives in 1934. These laws were generally applauded by geneticists.

How then do we account for the popularity of the claim that eugenics was based on a technical error? We suggest two related reasons. In the first quarter of this century, nearly all geneticists were enthusiastic proponents of a movement that is now generally held in contempt. In Germany, not one geneticist criticized the interwar eugenics movements<sup>27</sup>. After the Nazis came to power, genetics was invoked on behalf of ever more extreme measures of racial purification. Nevertheless, most of Germany's leading geneticists, including those who before 1933 had criticized antisemitism, actively helped to build the racial state. They served on important commissions, provided opinions on racial ancestry and participated in the drafting of racial laws. More than half of all academic biologists joined the Nazi Party, the highest membership rate of any professional group<sup>28</sup>. In other countries, too, eugenicists promoted policies such as immigration restriction that reflected strong class and racial biases. So the history of the field is the source of some embarrassment (and defensiveness). It is far more comforting to think that eugenics' decline was also due to geneticists. The myth rights the historical balance.

#### **Backdoor eugenics**

The claim also enables textbook writers and college teachers to avoid controversial issues. If eugenics is assumed to have rested on a technical error, it no longer raises thorny ethical questions. Geneticists can therefore condemn eugenics without questioning any of the aims of genetic testing. As Arthur Caplan points out, when the State of California ruled that screening for maternal serum α-fetoprotein should be offered to all pregnant women, it did so "in the hope that some of those who are found to have children with neural tube defects will choose not to bring them to term. . . thereby preventing the state from having to bear the burden of their care"29. There is similar costbenefit reasoning in the 1990 guidelines of the International Huntington Association and the World Federation of Neurology, which deem it acceptable to refuse to test women who "do not give complete assurance that they will terminate a pregnancy where there is an increased risk" of Huntington's disease<sup>30</sup>. Those who made this recommendation certainly did not think they were promoting eugenics. Assuming that eugenics is dead is one way to dispose of deep social, political and ethical questions. But it may not be the best one.

Diane B. Paul is in the Department of Political Science, University of Massachusetts, Boston, Massachusetts 02125, USA; Hamish G. Spencer is in the Department of Zoology, University of Otago, PO Box 56, Dunedin, New Zealand.

ACKNOWLEDGEMENTS. We thank the Division of Sciences. University of Otago, for financial support; E. F. Keller, R. C. Lewontin, J. Maynard Smith, M. Ruse, E. Seaman and G. P. Wallis for reading the manuscript; L. Zenderland for comments on Goddard; and the staff of the Science Library. University of Otago, for help with references

19. Paul, D. B. & Spencer, H. G. Am. J. hum. Genet. 43,

- 344-346 (1988). 20. Emery, A. E. H. & Mueller, R. F. Elements of Medical Genetics 8th edn (Churchill Livingstone, Edinburgh, 1992).
- 21. Hogben, L. Genetic Principles in Medicine and Social Science 207 (Williams & Norgate, London, 1931).
- 22. Conklin, E. G. in Human Biology and Population Improvement (ed. Cowdry, E. V.) 577-578 (Hoeber, New York, 1930). 23. Penrose, L. S. The Biology of Mental Defect (Sidgwick &
- Jackson, London, 1949).
- 24. Stern, C. Principles of Human Genetics 538 (W. H. Freeman, San Francisco, 1949).
- 25. Laughlin, H. H. 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 - Vol. 1: The Scope of the Committee's Work 47, 16 (Eugenics Record Office, Bulletin No. 10A, Cold Spring Harbor, New York, 1914).
- 26. Broberg, G. in Eugenics and the Welfare State. Sterilization Policy in Denmark, Sweden, Norway, and Finland (eds Broberg, G. & Roll-Hansen, N.) (Michigan State Univ. Press, East Lansing, in the press)
- Harwood, J. Brit, J. Hist. Sci. 22, 257–265 (1989). 28. Deichmann, U. & Müller-Hill, B. in Science, Technology and National Socialism (eds Renneberg, M. & Walker, M.) 160–183 (CUP, Cambridge, 1994).
- 29. Caplan, A. L. in Prescribing Our Future: Ethical Challenges in Genetic Counselling (ed. Bartels, D.) 149-165 (Aldine de Gruyter, Hawthorne, New York,
- 30. Ethical Issues Policy Statement on Huntington's Disease Molecular Genetics Predictive Test J. Med. Genet. 27. 34-38 (1990).

- 1. Cummings, M. R. Human Heredity: Principles and Issues 2nd edn, 436-437 (West, St Paul, 1991).
- Curtis, H. & Barnes, N. S. Biology 5th edn, 987 (Worth, New York, 1989).
- 3. Griffiths, A. J. F., Miller, J. H., Suzuki, D. T., Lewontin, R. C. & Gelbart, W. M. An Introduction to Genetic Analysis 5th edn. 759-760 (W. H. Freeman, New York, 1993).
- 4. Strickberger, M. W. Genetics 3rd edn, 693 (Macmillan, New York, 1985).
- East, E. M. J. Hered. 8, 215–217 (1917).
  Popenoe, P. J. Hered. 6, 32–36 (1915).
- Punnett, R. C. The Nineteenth Century 97, 697-707 (1925).
- Davenport, C. B. in Heredity and Eugenics (eds Castle, W. E., Coulter, J. M., Davenport, C. B., East, E. M. & Porter, W. L.) 286 (Univ. Chicago Press, Chicago, 1912).
- Popenoe, P. & Johnson, R. H. Applied Eugenics 105. 170-176 (Macmillan, New York, 1918) 10. Morgan, T. H. Evolution and Genetics 2nd edn, 201
- (Princeton Univ. Press, Princeton, 1925). Barker, D. Brit. J. Hist. Sci. 22, 347-375 (1989).
- 12. Provine, W. B. The Origins of Theoretical Population Genetics 137 (Univ. Chicago Press, Chicago, 1971).
- Punnett, R. C. J. Hered. 8, 464-465 (1917) 14. Fisher, R. A. Eugenics Rev. 26, 114–116 (1924). (Reprinted in Collected Papers of R. A. Fisher (ed. Bennett, J. H.) Vol. 1, 580-582 (Univ. Adelaide, South
- Australia, 1971).) 15. Jennings, H. S. J. Hered. 18, 271–276 (1927).
- 16. Jennings, H. S. The Biological Basis of Human Nature 238-242 (Faber & Faber, London, 1930).
- Haldane, J. B. S. Heredity and Politics 80-89 (Unwin, London, 1938).
- Brigham, C. C. A Study of American Intelligence (Princeton Univ. Press, Princeton, 1923).