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The failure of a scientific critique: David Heron, Karl Pearson and Mendelian eugenics

HAMISH G. SPENCER* and DIANE B. PAUL†

The bitterness and protracted character of the biometrician–Mendelian debate has long aroused the interest of historians of biology.¹ In this paper, we focus on another and much less discussed facet of the controversy: competing interpretations of the inheritance of mental defect. Today, the views of the early Mendelians, such as Charles B. Davenport and Henry H. Goddard, are universally seen to be mistaken. Some historians assume that the Mendelians' errors were exposed by advances in the science of genetics. Others believe that their mistakes could have been identified by contemporaries. Neither interpretation takes account of the fact that the lapses for which the Mendelian eugenicists are now notorious were, in fact, mostly identified at the time by the biometricians David Heron and Karl Pearson. In this paper we ask why their objections had so little impact. We think the answer illustrates an important general point about the social prerequisites for effective scientific critique.

In the latter years of the biometrician–Mendelian debate, the inheritance of mental defect in American families was intensively studied by Davenport, Goddard, H. H. Laughlin, David F. Weeks, A. J. Rosanoff and others. Mental defect, then often termed 'feeble-mindedness', was defined socially, as the incapacity of an individual to perform 'duties as a member of society in the position of life to which he is born'.² Davenport headed the Eugenics Record Office (ERO) on Long Island, New York, which published the Eugenics Record Office Bulletin in which much of the work appeared. The best known of

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1 See for example W. B. Provine, *The Origins of Theoretical Population Genetics*, Chicago, 1971; P. Froggatt and N. C. Nevin, 'The "Law of Ancestral Heredity" and the Mendelian–ancestrarian controversy in England, 1889–1906', *Journal of Medical Genetics* (1971), 8, 1–36; B. J. Norton, 'Biology and philosophy: the methodological foundations of biometry', *Journal of the History of Biology* (1975), 8, 85–93; D. MacKenzie, 'Sociobiologies in competition: the biometrician–Mendelian debate', in *Biology, Medicine and Society 1840–1940* (ed. C. Webster), Cambridge, 1981, 243–88; R. Olby, 'The dimensions of scientific controversy: the biometric–Mendelian debate', *BJHS* (1988), 22, 299–320; P. J. Bowler, *The Mendelian Revolution*, London, 1989, 71–2.

2 P. Popenoe, 'Feeble-mindedness', *Journal of Heredity* (1915), 6, 32–6.

the school's publications, however, was Goddard's *The Kallikak Family*,³ a descriptive account of a rural New Jersey clan in which feeble-mindedness was apparently rampant.

By 1912 Davenport had become convinced that feeble-mindedness was inherited as a simple Mendelian recessive, and he began to argue the point in books and talks. For example, in *Heredity and Eugenics*, a collection of lectures by several eminent geneticists including W. E. Castle and E. M. East, he claimed, on the basis of Goddard's data, that 'when both parents are feeble-minded all of the children will be so likewise; this condition has been tested again and again'.⁴ He concluded his essay with the advice that individuals suffering from 'weakness in any trait should marry strength in that trait and strength may marry weakness'.⁵ In this way, feeble-mindedness – for this is the 'weakness' that most concerned eugenicists – would be prevented by its recessive nature from being expressed in the following generation.

Two years later, Goddard, who was director of the Research Laboratory of the Training School for Feeble-minded Girls and Boys at Vineland, New Jersey, published *Feeble-mindedness, Its Causes and Consequences*, a longer and more formal Mendelian analysis of the Kallikak and other data. Goddard professed to have started out believing that feeble-mindedness was not a Mendelian character, but changed his mind after the data were analysed. The conclusion was 'forced upon us by the figures' even though it was 'difficult to make agree with previous conceptions'.⁶ As we will argue below, this book was fundamentally important in the controversy.

The embrace of the Mendelian model by the American eugenicists was noticed by participants on both sides of the debate in England. Among the Mendelians, Reginald C. Punnett, Balfour Professor of Genetics at the University of Cambridge, enthusiastically supported the model, telling the International Eugenics Congress held in London that feeble-mindedness was 'a case of simple Mendelian inheritance'.⁷ Although William Bateson, first director of the John Innes Horticultural Institution at Merton and the leading English Mendelian of his day, was more cautious, he told the 1913 International Medical Congress that the Americans had shown feeble-mindedness to have 'at least one of the marked features of a recessive condition'.⁸

Members of the opposing biometrical school, however, were dismayed. In 1913 and 1914, the Galton Eugenics Laboratory headed by Karl Pearson published a three-part response, under the overall title *Mendelism and the Problem of Mental Defect*. The author of Part I, subtitled *A Criticism of Recent American Work*, was David Heron, the Galton Research Fellow. Heron's essay was a direct attack on the work of the ERO and, in

3 H. H. Goddard, *The Kallikak Family: A Study in the Heredity of Feeble-mindedness*, New York, 1912.

4 C. B. Davenport, 'The inheritance of physical and mental traits of man and their application to eugenics', in *Heredity and Eugenics* (ed. W. E. Castle, J. M. Coulter, C. B. Davenport, E. M. East and W. L. Tower), Chicago, 1912, 281.

5 Davenport, op. cit. (4), 288.

6 H. H. Goddard, *Feeble-mindedness: Its Causes and Consequences*, New York, 1914, p. ix.

7 R. C. Punnett, 'Genetics and eugenics', in *Problems in Eugenics. Papers Communicated to the First International Eugenics Congress Held at the University of London, July 24th to 30th, 1912*, 2 vols., London, 1912, i, 137.

8 W. Bateson, 'Address on heredity', *British Medical Journal* (1913), 2, 360. Bateson went on to say, however, 'It is nevertheless difficult to regard this condition as a simple recessive.'

particular, the single-gene Mendelian model. Heron's paper purported to show that the material on which the ERO's papers were based 'has been collected with a decided bias in favour of a particular theory of heredity; that it is presented with extraordinary carelessness; that it is, on internal evidence, repeatedly contradictory; that it is not treated in any adequate statistical manner, and that the conclusions reached are not justified by the data'.⁹ Part II, by Pearson and Gustav A. Jaederholm, and Part III, by Pearson alone, were more moderate in tone, presenting the biometricians' positive view of how the genetics of feeble-mindedness should be approached.

It is important to realize that Heron and Pearson were not opposed to eugenics. Indeed, Donald MacKenzie has argued that there was a methodological link (as well as a financial one) between biometry and eugenics.¹⁰ Their critique was motivated by the fear that the slipshod methods (and, to a lesser extent, data) of the ERO would cripple the progress of eugenics. Heron spoke for them all when he wrote, 'those of us who have the highest hopes for the new science of Eugenics in the future are not a little alarmed by many of the recent contributions to the subject which threaten to place Eugenics with the older "social science" and much of modern sociology – entirely outside the pale of true science'.¹¹

Certainly by today's standards, the Mendelian theory of mental defect is easily refuted. But it could equally well have been rejected by geneticists of the time. David Barker put it bluntly: 'Since the standards [of criticism] are those which could and should have been used by an undergraduate geneticist in 1914, it is difficult to see why it was not immediately pronounced unseaworthy on "internalist" or scientific grounds'.¹² Barker then went on to suggest reasons why the Mendelian theory remained so influential for so long. But he seems not to have been aware that the theory had, in fact, been subjected to rigorous critique. The important question is why this critique counted for so little in the long term. After examining the three papers in the 'Mendelism and the Problem of Mental Defect' series, we propose an answer.

Heron began his paper by attacking Davenport's advice. Even if the Mendelian model were true, he noted, the effect of 'weakness' marrying 'strength' is but a temporary masking for one generation of the defective alleles and the production of heterozygous carriers (when 'strength' is homozygous), or the production of 50 per cent 'weakness'

9 D. Heron, 'Mendelism and the problem of mental defect. I. A criticism of recent American work', *Questions of the Day and of the Fray* No. VII, London, 1913, 12.

10 MacKenzie, op cit. (1), 271. Eileen Magnello has disputed this link, however, arguing that the work carried out in the Drapers' Biometric Laboratory was very different both in subject and in methodology from that performed in the Galton Eugenics Laboratory (M. E. Magnello, 'Karl Pearson's methodological innovations: the Drapers' Biometric Laboratory and the Galton Eugenics Laboratory', *History of Science*, in press). One area of overlap, however, was work on heredity and Mendelian genetics, and it is noteworthy that, although Heron was appointed to the Galton Laboratory, he was also trained in Pearsonian biometrics and published several papers in *Biometrika*, the primary journal of Drapers' Laboratory. Moreover, Heron was seen by Davenport and other Mendelians as a biometrician and labelled as such (see note 63 below).

11 Heron, op. cit. (9), 4. Heron was a foundation member of the Eugenics Education Society and in 1909 was elected to its Council (P. M. H. Mazumdar, *Eugenics, Human Genetics and Human Failings: The Eugenics Society, its Sources and its Critics in Britain*, London, 1992).

12 D. Barker, 'The biology of stupidity: genetics, eugenics and mental deficiency in the inter-war years', *BJHS* (1989), 22, 359.

(when ‘strength’ is heterozygous). Sooner or later two feeble-minded alleles will be united, and the ‘weakness’ expressed. Davenport clearly did not understand that the heterozygous carriers were a fresh source of the defect every generation and that his advice would greatly increase their number.

Heron then proceeded to examine in detail a number of ERO Bulletins, and, briefly, *The Kallikak Family*. (*Feeble-mindedness* had not yet been published.) He discovered a plethora of problems. First, much of the data had obviously been collected with the Mendelian hypothesis in mind. Thus field workers were instructed to make a special effort to find feeble-mindedness in the ancestry of two normal-minded – but by hypothesis heterozygous – parents of a feeble-minded child, thus proving the heterozygosity. When these ancestors could not be traced, heterozygosity was simply assumed. If neither the parents nor children were feeble-minded, no search was made in their ancestry, and they were all considered homozygous for the normal-minded allele. As Berkeley zoologist Samuel J. Holmes also noted, the classification of normal-minded individuals as either homozygous normal or heterozygous was made ‘according to whatever assumption is necessary to bring facts into accord with theory’.¹³

Secondly, there were several inconsistencies between the tabulated data and the text (for example, descriptions in tables and the text of individuals with the same case numbers did not match). Some of these contradictions could have reflected simple typographical errors, but Heron found innumerable instances which suggested systematic sloppiness. Sometimes heterozygotes were considered intermediate in their mental level; in other places normal-mindedness was considered completely dominant. Several of the observed proportions of different phenotypes did not match the Mendelian expectations, in spite of assertions that they did.

Particularly telling were the examples of normal-minded offspring of feeble-minded parents, children who should not have existed if the single-gene Mendelian recessive model were correct, and whose existence had been explicitly denied by Davenport. Heron thought Goddard particularly gifted at explaining away two of these exceptions:

Either there is a mistake in calling them normal, or a mistake in calling the parents feeble-minded; or else there was illegitimacy somewhere and these two children did not have the same father as others in the family. Or we may turn to the Mendelian law and we discover that according to that law there might be in rare instances such a combination of circumstances that a normal child might be born from two parents that function as feeble-minded.¹⁴

Heron remarked, ‘Thus the facts are to be considered as elastic, and if that fails we are to make the theory plastic enough to cover the facts’.¹⁵

At least one of the points raised by Heron was genetically sophisticated: he noted the

13 S. J. Holmes, *The Trend of the Race: A Study of Present Tendencies in the Biological Development of Civilized Mankind*, New York, 1921, 38.

14 Goddard, op. cit. (3), 114.

15 Heron, op. cit. (9), 61.

existence of what would come to be called ascertainment bias.¹⁶ For example, some data had been tabulated and analysed only when at least one child was defective in each family, which by the Mendelian hypothesis meant that neither parent was homozygous normal. But Heron noted that under Mendelian assumptions this restriction excludes some families that by chance have no defective children in spite of both parents being heterozygous. This bias creates an excess of defectives, but in several cases the data showed too few.

Heron also ridiculed the classification of individuals as abnormal. For instance, in one study of the inheritance of a 'neuropathic constitution', he quoted some of the ostensible manifestations of this condition, such as 'is said to have died of homesickness', 'quick tempered', 'very queer, lives alone, boards out cats', 'restless', 'fidgety, cannot keep still', 'worrier', 'insomnia, neuralgia', 'odd, very quiet disposition'. Every characteristic from a very long list was attributed to the individual being homozygous for a recessive allele. Heron concluded, 'It is a matter of surprise that there are any "normal" individuals at all; it is, indeed, a fortunate circumstance that the Mendelian theory requires the presence of *some* normal individuals.'¹⁷ (We should point out that there were other contemporary criticisms of Davenport and Goddard's Mendelian theory of feeble-mindedness, but none were as detailed or direct, or showed such a clear understanding of Mendelian principles as Heron's.)¹⁸

Although the two papers of which Pearson was an author were much more moderate in tone, he also highlighted inconsistencies: 'The fact is that alcoholism is by the American Mendelians treated as equivalent to normal, to latent amentia or to patent amentia just as may be needed to twist the facts to fit the theory.'¹⁹ After making original criticisms, he went on to analyse data collected by Jaederholm and others in what he considered to be

16 As far as we are aware, Heron was the first to describe ascertainment bias in English, although he did not use that term. Wilhelm Weinberg, after whom the Hardy-Weinberg Principle is named, had written about the subject in terms of conditional probabilities as early as 1908, but his work was not widely known in Britain until 1931 when Hogben applied these ideas to human pedigrees (Mazumdar, *op. cit.* (11)). Thus, Barker's claim that any undergraduate would have been able to make this (and other) criticisms is perhaps a little exaggerated.

17 Heron, *op. cit.* (9), 38, italics in original.

18 Psychologists in particular were doubtful, but few had the genetic training to make telling points. For example, the psychiatrist Abraham Myerson ('Psychiatric family studies', *American Journal of Insanity* (1917), 73, 355-486) also claimed (in two pages of a seventy-page article) that Davenport's facts had been made to fit Mendelian theory: the category of feeble-mindedness was polymorphic and the evidence for a single-gene defect was weak. But his criticism was mostly simple assertion of his doubts, and there was no response from the ERO. Popenoe and Johnson in their often-reprinted text *Applied Eugenics* (P. Popenoe and R. H. Johnson, New York, 1918), also expressed reservations about the single-gene model, but again the criticism was brief and they did not doubt that most feeble-mindedness was hereditary. Popenoe, as editor of the *Journal of Heredity*, may have been an influential critic, but by 1918 the Mendelian-chromosome theory was vindicated. Ironically, Davenport himself made one of the most relevant criticisms. In a surprisingly lukewarm review of *Feeble-mindedness: Its Causes and Consequences*. By H. H. Goodard', *Science* (1915), 42, 837-8), Davenport queried how a socially defined trait could be inherited as a Mendelian factor: 'since feeble-mindedness is a social and not a biological term, it would seem almost absurd to seek to find a law of its inheritance'.

19 K. Pearson, 'Mendelism and the problem of mental defect. III. On the graduated character of mental defect and on the need for standardising judgments as to the grade of social inefficiency which shall involve segregation', *Questions of the Day and of the Fray* No. IX, London, 1914, 16.

the appropriate, biometrical, way. The second paper in the series²⁰ confines most of its criticism to Davenport's classification of feeble-mindedness. Both in this paper and in Part III, Pearson argued that the feeble-minded and the normals were both part of a single continuous distribution of intelligence, rather than distinct classes separated by some intellectual boundary. Moreover, he claimed that since mental defectives were not differentiable from normals by mentality, but rather were 'socially inefficient', distinguishing the two was not simply a medical problem solvable with simple tests. Pearson²¹ was one of the first to argue that feeble-mindedness might not be completely due to heredity – a remarkable observation for a geneticist of that time – and he noted that correlations between degraded environments and feeble-mindedness told us nothing about causality.

Like Heron, Pearson understood Mendelian theory clearly,²² and showed how its application to the feeble-minded data could lead to absurdities. He examined the effect of 'weakness marrying strength' over four generations and argued that it would work only if the resultant carriers possessed a far greater knowledge of their ancestry than could be expected. He also examined several of the pedigrees used by Davenport, and noted the absurdly high frequency of marriages between carriers and defectives. Based on the alleged frequency of the feeble-minded allele in the general population, Pearson calculated that the chance of a single such marriage was about one in eight. (Most marriages between the feeble-minded and normals should involve homozygous normals.) One pedigree had five such marriages, a chance Pearson estimated at 32,767 to 1. Even allowing for some assortative mating, these long odds were not credible. And like Heron, he noted the exceptions to the 'iron-clad rule' of two feeble-minded parents having only feeble-minded children.²³

The response by the Americans to Heron's paper was swift.²⁴ The 1914 ERO Bulletin No. 11 contained detailed replies by Davenport and Rosanoff, as well as an extract from a letter by Weeks to Davenport discussing some of Heron's criticisms.²⁵ Davenport also wrote to *Science*.²⁶ Extracts of Heron's paper appeared in the *New York Times* in November 1913, along with a vigorous response from Davenport, to which Heron in turn

20 K. Pearson and G. A. Jaederholm, 'Mendelism and the problem of mental defect. II. On the continuity of mental defect', *Questions of the Day and of the Fray* No. VIII, London, 1914.

21 Pearson, op. cit. (19), 5–8.

22 Pearson (and, presumably, Heron as well) accepted Mendelian explanations for discrete characters (such as blue and brown eye colour), but he considered these cases rare exceptions rather than the rule (M. E. Magnello, 'Karl Pearson's mathematization of inheritance: from ancestral heredity to Mendelian genetics (1895–1909)', *Annals of Science* (1998), 55, 35–94; see also Froggatt and Nevin, op. cit. (1)).

23 Pearson, op. cit. (19), 20.

24 The two papers by Pearson appeared at approximately the same time as these responses and were apparently ignored by Davenport and his co-workers.

25 C. B. Davenport, 'A discussion of the methods and results of Dr. Heron's critique', in *Reply to the Criticism of Recent American Work by Dr. Heron of the Galton Laboratory*, Eugenics Record Office Bulletin No. 11, Cold Spring Harbor, New York, 1914, 3–24; A. J. Rosanoff, 'Mendelism and neuropathic heredity: a reply to some of Dr. David Heron's criticisms of recent American work', in *ibid.*, 27–43 (Reprinted from *American Journal of Insanity* (1914), 70, 571–87); D. F. Weeks, 'Extract from a letter to C. B. Davenport from Dr. David F. Weeks, Superintendent of the New Jersey State Village for Epileptics at Skillman', in *ibid.*, 25.

26 C. B. Davenport, 'A reply to Dr. Heron's strictures', *Science* (1913), 38, 773–4.

replied in January.²⁷ Goddard did not respond explicitly, going on to publish *Feeble-mindedness*, but it is worth noting that Heron mentioned *The Kallikak Family* only once, towards the end of his critique, spending most of his time dealing with various ERO Bulletins and attacking Davenport by name. Nevertheless, most of Heron's points could equally well have been directed at Goddard's work.

Heron's and Pearson's criticisms had almost no long-term impact on the debate about the genetics of feeble-mindedness. Seventeen years later, Johns Hopkins biologist H. H. Jennings, who was portrayed by Daniel J. Kevles²⁸ as the leading American opponent of mainline eugenics, wrote that feeble-mindedness was the clearest case of a single-gene defect in humans.²⁹ Writing in the popular *Harper's Monthly* in 1931, Julian Huxley also sanctioned the theory.³⁰ Even such a prominent critic of eugenics as J. B. S. Haldane tacitly accepted the single-gene Mendelian model when he admitted that eugenic measures against the feeble-minded would probably reduce the proportion 'by something of the order of 10 per cent'.³¹ Numerous respected geneticists, including Castle³² and East,³³ explicitly endorsed Goddard's methods of data collection and analysis well after its deficiencies had been pointed out by Heron and Pearson.³⁴ Into the 1960s, Goddard's Kallikak pedigrees could still be found in college psychology textbooks.³⁵

Why was Heron's critique so ineffective? We suggest several reasons. The first concerns its style. The paper was extremely hostile in tone, repetitive (for example the Davenport quotation, 'strength may marry weakness', occurs at least once on every page of pp. 5–8, and is paraphrased on these and other pages as well), intemperate ('We cannot conceive of a greater evil than that expressed in the teaching above [that "strength may marry weakness," etc.]') and highly personal ('we are not prepared to dissent from the view that the citation of a pedigree by Dr Davenport is a disqualification for its future use').³⁶ In addition, in many places, Heron was criticizing minute inconsistencies in the data, in long paragraphs of dense prose. It may be that few readers could stomach sixty-two pages of this. In support of this explanation, we have discovered very few citations of Heron's paper within or outside the genetics community. Given the fact that it was published in a widely

27 D. Heron, 'English expert attacks American eugenic work', *New York Times*, 9 November 1913, part V, 1; C. B. Davenport, 'American work strongly defended', *ibid.*, 1–2; D. Heron, 'English eugenics expert again attacks Davenport', *ibid.*, 4 January 1914, part V, 1.

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

29 H. S. Jennings, *The Biological Basis of Human Nature*, New York, 1930, 238.

30 J. S. Huxley, 'The vital importance of eugenics', *Harper's Monthly Magazine* (1931), 163, 324–31.

31 J. B. S. Haldane, *Heredity and Politics*, New York, 1938, 94, but see also D. B. Paul and H. G. Spencer, 'The hidden science of eugenics', *Nature* (1995), 374, 302–4 and D. B. Paul and H. G. Spencer, 'Did eugenics rest on an elementary mistake?', in *Thinking about Evolution: Historical, Philosophical and Political Perspectives* (ed. J. Beatty, C. Krimbas, D. B. Paul and R. S. Singh), in press. Diane Paul has noted that while all three of these geneticists (Jennings, Huxley and Haldane) were critical of conventional eugenics, they retained many eugenic views their whole lives (D. B. Paul, 'Eugenics and the left', *Journal of the History of Ideas* (1984), 45, 567–90).

32 W. E. Castle, *Genetics and Eugenics*, 4th edn, Cambridge, MA, 1931, 381.

33 E. M. East, 'Review *Genetic Principles in Medicine and the Social Sciences*. By L. T. Hogben', *Economica* (1932), 12, 235–8.

34 See Barker, *op. cit.* (12), for a fuller treatment.

35 For example H. E. Garrett and H. Bonner, *General Psychology*, 2nd edn, New York, 1961.

36 Heron, *op. cit.* (9), 6, 38.

known series and publicized in the leading American newspaper,³⁷ we do not think that the paper was simply overlooked.³⁸

Secondly, Heron's paper was technically difficult, requiring a reasonable understanding of Mendelian genetics. Many of the arguments could have been used by anti-eugenic critics, for example the Roman Catholic writer G. K. Chesterton, who wrote numerous articles on the subject,³⁹ or others opposed to sterilization, such as Dr Joseph Prideaux, the Mental and Neurological Inspector of the British Ministry of Pensions.⁴⁰ Although some did use Punnett's argument about the inefficacy of sterilizing the feeble-minded,⁴¹ no one took up Heron's points. His paper was doubtless too difficult for non-specialists, and there were no actively anti-eugenic geneticists at the time. Indeed, we suspect Heron remains unread to this day: few historians go beyond remarking that Heron was a critic of the Mendelian model.

A third reason suggests itself: scientists on the edge of the debate may have been satisfied that Heron's critique was unjustified. Davenport and Rosanoff had replied at length, and both Weeks and Davenport had made shorter responses – Davenport in *Science*. The respondents were more politically astute: their language was measured, they emphasized the personal nature of Heron's attack and complained of its generally immoderate tone, and they concentrated on responding to a small subset of Heron's less-damaging points. Davenport characterized the Mendelians as 'biological ... students of genetics', in contrast to the biometricians who were 'mathematical students' from whose work 'no biological laws could be deduced',⁴² and he effectively belittled the role of the critic, saying that Heron should instead be 'making positive discoveries in a field where so little is known and where the need of utilisable knowledge is so great'.⁴³

Davenport defended the data collection methods as standard, derived in fact from

37 The *New York Times* treatment (op. cit. (27)) also played on their readers' patriotism, headlining their extracts from Heron's paper 'English expert attacks American eugenic work' and introducing it saying that Heron had made 'a spirited attack ... upon the entire body of American Eugenics'. In his adjacent reply entitled 'American work strongly defended', Davenport continued this line, claiming 'We in America ... have abandoned the Old World scholasticism' so that 'the United States is the only place where on a large scale, eugenics is being worked out' and which is 'attempting with any measure of success to put the findings of eugenics experts into practical everyday use'. Only at the end of the reply does he mention the biometrician–Mendelian debate, carefully phrasing it in terms of the main protagonists, 'Our Pearsonian critics absorbed with their mathematical tables.' Heron was careful in his response two months later to start out by saying that 'so much excellent scientific work has been done in America', but the paper's subeditors placed it under the headline 'English eugenics expert again attacks Davenport'.

Davenport did not have it all his own way, however. The extract from Heron's paper contained a one-sentence assertion that Davenport favoured inter-racial marriages. In his reply, Davenport denied this view, but Heron's response expanded the accusation, quoting Davenport's comments on how hybridizing would allow the white population to gain various characteristics (for example 'keen sense of humour') supposedly superior in blacks.

38 Popenoe (op. cit. (2), 34) noted at the time that the controversy between Heron and the ERO members was 'fairly well aired in the daily as well as the scientific press'. Heron's monograph was (favourably) reviewed in the *Times Literary Supplement*, 3 September 1914, 410.

39 See for example G. K. Chesterton, *Eugenics and Other Evils*, London, 1922.

40 J. F. E. Prideaux, *British Medical Journal* (1923), 2, 231.

41 R. C. Punnett, 'Eliminating feeble-mindedness', *Journal of Heredity* (1917), 8, 464–5. See also Paul and Spencer, op. cit. (31).

42 Davenport, op. cit. (25), 23.

43 Davenport, op. cit. (26), 774.

Galton himself.⁴⁴ He argued that it made economic sense to follow up the ancestry of particular cases, denying that this method led to a bias. In his 1914 paper, Davenport did acknowledge a number of typographical errors, blaming them on typesetters, typewriters, poor proof-reading and joint authorship, although just the year before he had argued that most of Heron's claimed errors were misunderstandings of the ERO's methods. For instance, he claimed that case numbers were altered to preserve patient anonymity and so would not necessarily match up.⁴⁵ He then accused Heron of invalid methods of scientific criticism, for example 'making unjustifiable assumptions and then refuting them', 'leaving false impressions by partial statements', 'multiplication of "errors" by adopting the worst construction' and 'listing as errors-of-omission data which were not used because not sufficiently critical'.⁴⁶

Davenport explained the exceptional cases of normal children of feeble-minded parents by analogy with blue-eyed parents having a brown-eyed child. The Mendelians' claim was of the nature of a 'first statement', he argued, an approximation whose 'limiting conditions are worked out by further critical study'. The exceptions all concerned parents who were 'defective in one or more specific traits and not generally'. Moreover, other workers had independently confirmed their work: Lundborg, for example, had argued for the Mendelian inheritance of 'progressive myoclonic epilepsy', which Davenport claimed was a slight modification of the 'genuine epilepsy' studied by him and Weeks.⁴⁷ And Rosanoff explained these exceptional cases as children who had not yet reached the age of onset of neuropathy.⁴⁸

Davenport also complained that he was misquoted, and his responses to Heron's substantive criticisms were carefully targeted. For example, his staunchest defence of the 'weakness marrying strength' aphorism appeared in the *New York Times*, where he claimed that 'practically the entire monograph [that is, Heron's attack] is based on this sentence'.⁴⁹ Not only was this assertion untrue – at most ten of Heron's sixty-two pages were on the subject – but it cleverly dismissed many other equally serious criticisms. Davenport also asserted that his aphorism was qualified by other statements in the paragraph in which it was embedded. In fact, as Heron justifiably complained,⁵⁰ it was not: the paragraph did not appear in the source Heron quoted. Davenport's later assertion⁵¹ that the phrase also appeared in a 1910 paper (from which the missing paragraph also came) was untrue.

More importantly, Davenport responded that weakness marrying strength was the only practical rule given that virtually everyone is a carrier of some heritable weakness.⁵² Since

44 Davenport, op. cit. (25), 6.

45 Davenport, op. cit. (26) and op. cit. (25), 16.

46 Davenport, op. cit. (25), 11–16.

47 Davenport, op. cit. (25), 18, 22.

48 Rosanoff, op. cit. (25), 35.

49 Davenport, op. cit. (27).

50 Heron, op. cit. (27).

51 Davenport, op. cit. (25), 20.

52 This argument is particularly interesting because it has metamorphosed into one of the standard anti-eugenic responses: no one is free from some bad genes. For example, Haldane (op. cit. (31), 95–6) argued that 'With mental defects as with physical defects, if you once deem it desirable to sterilise I think it is a little difficult to know where you are to stop.'

the advice was intended for a general university audience,⁵³ it needed to be put in its simplest and ‘briefest form, comparable to a folk-saying’.⁵⁴ Heron’s reply was weak. While he pointed out that the application of Davenport’s rule would lead to ‘the contamination of stocks that are mentally normal’,⁵⁵ he did not explain why this mattered in the long term. In spite of quoting the very passage that revealed Davenport’s failure to recognize the future threat of the pool of resulting heterozygotes – and thus his poor understanding of Mendelism⁵⁶ – Heron proceeded to ask where the weak found mates if the strong were so rare. In doing so, he confused a situation involving single traits, to which Davenport’s aphorism clearly applied, with one involving overall strengths and weaknesses.

Rosanoff defended the apparently eclectic ‘neuropathic constitution’ category, claiming that its manifestations varied from one generation to the next since they were all the result of ‘instability of the nervous system’. This instability was, by implication, the true Mendelian trait. Rosanoff then pointed out that in a previous paper, Heron too had amalgamated, into a single class, patients with different clinical diagnoses.⁵⁷

Although all these factors may have contributed to Heron’s ineffectiveness, we suggest that the primary explanation is that his scientific claims soon seemed irrelevant, given the subsequent apparent confirmation of the Mendelian model of feeble-mindedness, and the triumph of the general Mendelian-chromosome theory. The most important apparent confirmation of the theory of mental defect must surely have been the publication of Goddard’s *Feeble-mindedness* in 1914, hard on the heels of Heron’s critique. It would have reassured many who may have had some initial doubts about the ERO’s work. This book was much more substantial than *The Kallikak Family*, with many more pedigrees, and importantly, free of the more obvious errors that Heron had noted.⁵⁸ Goddard also tried to correct for the problem of ascertainment bias.⁵⁹

Some observers may have concluded that even if Davenport’s (and Goddard’s) original data and methods were a little suspect, this new work was much more careful – indeed it was – and so Heron’s critique was no longer scientifically germane. In support of this argument, we note that it is Goddard’s work rather than Davenport’s that was usually cited when the single-gene Mendelian model was discussed, for example by East and Fisher in their papers about the efficacy of eugenic selection.⁶⁰ In their influential text, *Applied Eugenics*, Popenoe and Johnson explicitly cite Goddard’s research, not Davenport’s, as the main evidence that eugenicists subscribed to that model.⁶¹ And this view is still held by

53 This point was made clear in the preface to the published version of the lecture (J. M. Coulter, F. R. Little and W. L. Tower, ‘Preface’, in *Heredity and Eugenics* (ed. W. E. Castle, J. M. Coulter, C. B. Davenport, E. M. East and W. L. Tower), Chicago, 1912, pp. v–vi).

54 Davenport, op. cit. (27), 2.

55 Heron, op. cit. (27), 1.

56 See Paul and Spencer, op. cit. (31).

57 Rosanoff, op. cit. (25).

58 See Barker, op. cit. (12), 352.

59 This action may have appeared fair, but as Barker (op. cit. (12), 357) noted, the correction was still erroneous and fortuitously made the fit with the Mendelian model better.

60 E. M. East, ‘Hidden feeble-mindedness’, *Journal of Heredity* (1917), 8, 215–17; R. A. Fisher, ‘The elimination of mental defect’, *Eugenics Review* (1924), 16, 114–16.

61 Popenoe and Johnson, op. cit. (18), 105.

historians of science: Barker stated that ‘the genetic theory of mental defect was primarily the work of H. H. Goddard’ in *Feeble-mindedness*.⁶²

The ascendancy of the Mendelian school from about 1914 may also have proved reassuring to observers. Davenport asserted in all his responses⁶³ that the real motivation for Heron’s criticisms was to further the biometricians’ anti-Mendelian campaign.⁶⁴ The years 1914–18 were those in which Thomas Hunt Morgan’s group at Columbia was demonstrating that numerous characters in *Drosophila* were inherited as Mendelian factors.⁶⁵ And in human genetics, one form of mental defectiveness, Huntington’s chorea, had been shown to be due to a dominant Mendelian factor by Davenport himself.⁶⁶ For all these reasons, criticisms of Mendelism would have seemed dated. And there was no other group of geneticists who might have taken up Heron’s anti-Mendelian arguments. In particular, there was no biometrical school in the United States corresponding to that of Pearson’s in Britain. The publication of Fisher’s paper resolving the theoretical issues of the biometrician–Mendelian debate⁶⁷ also may have convinced scientists that Heron’s (and Pearson’s)⁶⁸ anti-Mendelian argument was unimportant – and, by association, their particular criticism of the Mendelian model for feeble-mindedness as well. Given the widespread acceptance – even by critics of the single-gene model – that the vast majority of cases of feeble-mindedness were due to genetic defect,⁶⁹ the triumph of the Mendelian model for genetics generally carried the implication that the Mendelian model for mental defect was also right. Indeed, Holmes made this argument explicit in his defence of the feeble-mindedness model.⁷⁰ And in replying to Heron, Rosanoff argued that since

62 Barker, op. cit. (12), 351.

63 For example, Davenport, op. cit. (25), 22–4.

64 Ludmerer has argued that Davenport and his colleagues misinterpreted Heron’s true motivation. Heron, he claimed, was merely encouraging ‘Americans working in the field to employ greater caution’ (K. M. Ludmerer, *Genetics and American Society: A Historical Appraisal*, Baltimore, 1972, 61). We disagree: the wholesale criticism of the Mendelian model shows quite clearly that Heron was attacking the method and not just the data.

65 G. E. Allen, *Thomas Hunt Morgan, the Man and his Science*, Princeton, 1978.

66 C. B. Davenport and E. B. Muncey, *Huntington’s Chorea in Relation to Heredity and Eugenics*, Eugenics Record Office Bulletin No. 17, Cold Spring Harbor, New York, 1916.

67 R. A. Fisher, ‘The correlation between relatives on the supposition of Mendelian inheritance’, *Transactions of the Royal Society of Edinburgh* (1918), 52, 399–433. As MacKenzie (op. cit. (1)), Froggatt and Nevin (op. cit. (1)), Magnello (op. cit. (22)) and others have noted, previous reconciliations were published (for example G. U. Yule, ‘Mendel’s laws and their probable relations to intra-racial heredity’, *New Phytologist* (1902), 1, 193–207, 222–38; K. Pearson, ‘Mathematical contributions to the theory of evolution – XII. On a generalised theory of alternative inheritance, with special reference to Mendel’s laws’, *Philosophical Transactions of the Royal Society of London A* (1904), 203, 53–86; K. Pearson, ‘On the ancestral gametic correlations of a Mendelian population mating at random’, *Proceedings of the Royal Society of London B* (1909), 81, 225–9; E. M. East, ‘A Mendelian interpretation of variation that is apparently continuous’, *American Naturalist* (1910), 44, 65–82), but they had little effect on terminating the debate. While the biometricians considered that Mendelism could apply to discrete data, they thought such data were very rare and did not accept that Mendelism offered a full explanation of heredity, especially for continuous characters. Moreover, many Mendelians consistently portrayed their opponents as rejecting Mendelism *in toto* (Magnello, op. cit. (22)).

68 We have also found few citations of Pearson’s two papers. If scientists were convinced by Davenport’s response (and the rise of Mendelism in general), they may have remained largely unread. Their publication was not accompanied by the publicity that Heron achieved. Moreover, Heron’s style may have tarnished all three critiques.

69 Popenoe, op. cit. (2), Kevles, op. cit. (28), 71.

70 Holmes, op. cit. (13), 37.

Mendelism was now known to be correct, exceptions to its predictions about mental defect should be handled by special explanations (for example delayed onset of neuropathy).⁷¹

The fate of Heron's now-obscure paper shows that even the soundest scientific arguments will fail to convince in the absence of a suitable culture in which the argument can take root. In the quarter-century following publication of Heron's paper, the Mendelians vanquished the biometricians.⁷² Thus no scientific group had an interest in calling attention to Heron's critique. While opponents of eugenics would have had such an incentive, there were none amongst the Mendelian geneticists.⁷³ And non-scientific critics would surely have been baffled by the technical arguments.

But what explains the fate of Heron's critique at the hands of historians? While his paper is widely cited, no one has actually analysed the arguments, which have been seriously misconstrued.⁷⁴ We propose two related reasons. First, the Heron case undermines the conventional linkage between the progress of genetics and the decline of eugenics. After all, the most compelling scientific criticisms were there from the start. Secondly, it also undermines a strong inclination to dispose of eugenics on technical grounds. Since the critical arguments were advanced by *eugenicists*, the Heron case shows that scientific sophistication was (and is) compatible with policy choices that most historians now disdain. Thus historians have had as little interest as scientists in taking Heron seriously.

71 Rosanoff, *op. cit.* (25).

72 We do not wish to imply that biometrical genetics disappeared completely. But after 1918, when Fisher (*op. cit.* (67)) showed how continuous traits could be explained by several Mendelian factors acting simultaneously, the synthesis of the chromosome theory with Mendelism provided the only viable explanatory theory of genetic inheritance. Sturtevant and Beadle's standard text mentions 'continuously variable characters' in a single paragraph of the final chapter (a historical review of the development of genetics) (A. H. Sturtevant and G. W. Beadle, *An Introduction to Genetics*, Philadelphia, 1939, 362). And of the forty-three major articles appearing in the first six months of 1930 in the *Journal of Heredity*, just one could be seen as explicitly biometric in its approach. Ironically, as Magnello (*op. cit.* (22)) has pointed out, many of Pearson's biometrical techniques, especially his chi-square goodness-of-fit test, became standard tools of Mendelian analysis.

73 Bateson was probably the closest thing that existed to an anti-eugenic Mendelian: he never joined the Eugenics Education Society, nor did he think eugenics was his job, seeing it as a 'serious nuisance diverting attention' from 'real Genetics' (letter dated 28 January 1915, in response to an invitation to lecture in Cambridge on eugenics, quoted in *William Bateson, F.R.S., Naturalist: His Essays and Addresses* (ed. B. Bateson), Cambridge, 1928, 388). Nevertheless, he was proud to deliver the 1919 Galton Lecture to the Eugenics Education Society and he endorsed the state putting 'such control on the feeble-minded members of the population as to prevent their propagation' (W. Bateson, 'Common-sense in racial problems', reprinted in *ibid.*, 371–87). This speech was written just two years after his close collaborator, R. C. Punnett, had shown (*op. cit.* (41)) that, under the Mendelian model, such a policy would take an eternity to eliminate the condition.

74 For example, see Ludmerer, *op. cit.* (64).