FEATURE ARTICLE

The Misuse of Pedigree Analysis in the Eugenics Movement

MARK SHOTWELL

ABSTRACT
Pedigree analysis has long been an essential tool in human genetics as well as a staple of genetics education. Students of genetics might be surprised to learn that human pedigrees were first popularized in the United States by proponents of eugenics, the pseudoscientific social movement aimed at improving the genetic quality of the human race. Notably, the influential eugenicist Charles B. Davenport employed pedigree charts to support his belief that not only were such medical conditions as Huntington disease and albinism inherited in a simple Mendelian fashion, but so too were such characteristics as alcoholism, criminality, and “feeblemindedness.” We now see the flaws in Davenport’s pedigree analysis, but at the time, it was the latest scientific advance. The misuse of pedigree analysis during the eugenics era may serve as a cautionary tale for those who are now harnessing the latest genetic technologies to solve complex problems.

Key Words: eugenics movement; pedigree analysis; Charles B. Davenport.

Introduction
Humans make terrible subjects for genetic studies. That’s because the primary experimental tool of genetics, the controlled cross, is simply not possible for humans. There is no such thing as a highly homozygous “true-breeding line” of humans, and even if there were, it would be impossible (or at the very least highly unethical) to compel two people to mate with each other to reveal the pattern of inheritance of a particular trait. Almost since the dawn of genetics at the turn of the 20th century, therefore, human geneticists have relied on a much less powerful (but at least feasible) method, the analysis of pedigree charts.

Pedigree charts are branching diagrams that depict the appearance of a trait in a family (Resta, 1993). If the family is large enough, comprising several generations with multiple affected persons, it may be possible to infer how the trait is inherited, whether the responsible gene resides on the X chromosome or an autosome, and whether it is dominant or recessive.

Genealogical charts were drawn for centuries before they became so important in human genetics. At first, they were simply graphical representations of family relationships. The name is derived from the Middle French pied de grue, literally meaning “foot of crane,” because some early diagrams contained so many branching diagonal lines that they resembled a bird’s foot. One of the earliest surviving pedigrees showed the inheritance of hemophilia A, an X-linked recessive condition, in the royal families of Europe.

Pedigrees have grown in sophistication in recent decades, and may now incorporate information from genetic testing as specific as single-nucleotide differences in genes. But their basic construction has changed little since the first decades of the 20th century. What most students of genetics are unaware of, however, is that pedigree analysis was developed in this country by proponents of eugenics, the pseudoscientific social movement with the aim of controlling human heredity (Resta, 1993). Eugenicists employed pedigrees to bolster their claims that not only were physical traits strictly inherited as simple Mendelian characters, but so too were mental, emotional, and behavioral characteristics. As we will see, their analysis was deeply flawed, based on slipshod data collection, unsupported assumptions, and circular reasoning.

Pedigree Construction
Pedigree construction became standardized in the United States in 1911 with the publication of the book Heredity in Relation to Eugenics by the leading American geneticist and eugenicist Charles B.
Davenport (Davenport, 1911). Until that time, pedigree charts used a variety of symbols; from that time onward, almost all were made with the symbols shown in Figure 1 (at least in the United States).

Davenport’s pedigree of a family with Huntington disease illustrates the key features of pedigree construction (Figure 2). Females are indicated with circles and males with squares. (Previously, ♀ and ♂ were used for female and male, respectively.) Affected persons are denoted by shaded symbols. The generations each occupy a separate line, with the most ancestral at the top, and are numbered with Roman numerals. Individuals within a generation are numbered left to right with Arabic numerals. Each person in the pedigree may thus be identified by a Roman numeral and an Arabic numeral (e.g., II-3). A single line connects a female and male to represent a mating, and a vertical line of descent drops from the mating line to the sibship line, which connects the offspring in their order of birth.

**Modes of Inheritance**

The goal when examining a pedigree is to infer the mode of inheritance of the trait. Does the pattern of appearance of the trait suggest autosomal dominant, autosomal recessive, X-linked dominant, or X-linked recessive inheritance? The first step is to determine whether there are any skipped generations; that is, are there any instances of two unaffected parents with an affected child? For traits determined by a recessive allele, it is possible, even common, for two unaffected parents to have an affected child. Both parents must be heterozygous, however (Figure 3). If the trait is determined by a dominant allele, two unaffected parents cannot have an affected child. One or the other of the parents would have to carry the dominant allele for a child to be affected (Figure 4). Skipped generations are thus diagnostic of a recessive mode of inheritance. The absence of skipped generations, when every affected person has at least one affected parent, is evidence for dominant inheritance.

Once dominant vs. recessive inheritance has been established, the next task is to decide whether the responsible gene resides on an autosome or on the X chromosome. We’ll start with the recessive mode. For the X-linked recessive mode of inheritance, females with genotypes AA and Aa are unaffected, and only genotype aa is affected. In males, who have only one X chromosome and therefore are hemizygous for all X-linked genes, AY is unaffected and aY is affected. The genotypes and the associated pedigree symbols are shown in Figure 5.

Consider a mating between a heterozygous unaffected female (“carrier female”) and a hemizygous dominant unaffected male (Figure 6). The possible outcomes of this mating are given in the Punnett square in Figure 7. Note that this is an example of two unaffected parents having an affected child, a “skipped generation.”
the diagnostic element of recessive inheritance. Note further that the expected phenotype ratio is three unaffected to one affected, but all of the affected are male. In other words, the recessive phenotype co-segregates with sex.

X-linked recessive pedigrees thus show two distinguishing features:

1. The appearance of the phenotype usually (but not always) skips generations. That is, affected persons almost always have parents who are both unaffected.
2. The phenotype appears predominantly in males. For a rare condition, affected females will almost never be found.

The pedigree depicting the occurrence of hemophilia in the royal families of Europe (mentioned above; the pedigree can easily be found online) shows these two characteristic features. So does the pedigree in Figure 8, which depicts the appearance of Duchenne muscular dystrophy in a small three-generation family (Bundey, 1978).

If there are skipped generations in a pedigree, but the trait is far more common in males, it may be concluded that the mode is autosomal recessive. Davenport's pedigree of a family with astigmatism shows the characteristic features of autosomal recessive inheritance (Figure 9).

Of the four possible modes of inheritance, X-linked dominant is by far the least common. Only a very few X-linked dominant conditions are known; incontinentia pigmenti and Rett syndrome are two on a very short list. Given its rarity, X-linked dominant inheritance will not be discussed here.

### Interpreting Pedigrees

The first steps in analyzing a human pedigree are to look for skipped generations and any patterns characteristic of X-linkage:

1. If there are no skipped generations (i.e., no instances of two unaffected parents with at least one affected child), autosomal dominant inheritance may be assumed. (X-linked dominant inheritance can be ruled out by the appearance of either an affected father with an affected son or an affected father with an unaffected daughter.)
2. If there is at least one skipped generation (i.e., one or more instances of two unaffected parents with an affected child), recessive inheritance is assumed.
   a. If the phenotype is much more frequent in males than in females (or appears only in males), X-linked recessive
inheritance may be assumed. All unaffected females must have at least one dominant allele ($A^-$). The affected males are hemizygous recessive ($aY$) and the unaffected males are hemizygous dominant ($AY$).

b. If the trait is not much more frequent in males than in females, autosomal recessive inheritance is assumed. Everyone unaffected must have at least one dominant allele ($A^-$), and all the affected persons are homozygous recessive ($aa$).

Consider the pedigree in Figure 10, showing the appearance of alkaptonuria, a defect in the gene encoding the enzyme homogentisic acid oxygenase (Cuthbert, 1923). The first question we ask ourselves about this pedigree is whether there are any skipped generations. The answer to this question is yes: III-1 and III-2 are both unaffected and have three affected children (IV-1, IV-3, and IV-4). We therefore conclude that alkaptonuria is a “recessive trait.”

Our second question is whether the phenotype is much more common in males than in females. In this case, there are two affected males (IV-1 and IV-3) and one affected female (IV-4). A ratio of two affected males to one affected female is far too low for this to be X-linked recessive. We thus conclude that the mode of inheritance is autosomal recessive.

Alkaptonuria (black urine disease) was the first so-called inborn error of metabolism described by Archibald Garrod (Garrod, 1902). Garrod himself suggested that alkaptonuria was a recessive condition (based on his discussions with William Bateson). Garrod also noted that 60% of the cases of alkaptonuria he had identified appeared in the children of first-cousin marriages. This is another hallmark of the autosomal recessive mode of inheritance, a higher incidence in the offspring of consanguineous matings, most commonly between first and second cousins. Such matings are indicated by a double mating line, as shown in Figure 10.

**Charles B. Davenport**

Charles Benedict Davenport (Figure 11) was a Harvard-educated zoologist who helped extend Mendelian principles to animals, including poultry, mice, and horses, in the first decade of the 20th century. During that time, he also carried out a series of studies of eye color, hair form, and hair color in humans, and proposed that skin color is determined by two interacting genes. Consequently, Davenport may rightly be considered the first true human geneticist.

With equal justification, Davenport may also be regarded as the leading eugenicist in the United States. Eugenics was a pseudo-scientific social movement whose goal was to improve the hereditary quality of the human race by controlling breeding, encouraging the hereditarily “superior” to have more children and discouraging (or preventing altogether) the reproduction of the genetically “inferior.” Eugenics originated in the writings of the English polymath Francis Galton (Charles Darwin’s half-cousin) and flourished in the first three decades of the 20th century, stimulated by the rediscovery of Mendel’s principles of heredity in 1900. Eugenics programs sprung up throughout Europe, Scandinavia, and South America, in addition to England and the United States. In the United States, eugenics found widespread favor, supported by prominent authors (e.g., H. G. Wells), journalists (e.g., Albert E. Wiggam), industrialists (e.g., John Harvey Kellogg), inventors (e.g., Alexander Graham Bell), birth control advocates (e.g., Margaret Sanger), psychologists (e.g., Henry H. Goddard), religious leaders (e.g., William R. Inge), and politicians (e.g., Theodore Roosevelt). It was also promoted by the leading geneticists of the day, including R.A. Fisher in England, Erwin Baur in Germany, Herman Nilsson-Ehle in Sweden, and Edward M. East in the United States, among many others (Paul & Spencer, 1995). For a time, eugenics was backed by the pioneering geneticist Thomas Hunt Morgan, whose work with fruit flies first
showed that genes reside on chromosomes. Though lukewarm in his support, Morgan did serve as a founding scientific director of the Eugenics Record Office (Lombardo, 2001). He soon grew disillusioned with the methods of the eugenic movement, criticizing them as reckless and unreliable to Davenport (Ludmerer, 1972, pp. 82–83). In 1915 he resigned in protest from the American Breeders’ Association (Kevels, 1985, p. 122), which, under Davenport’s influence, had begun strongly promoting eugenics (Kimmelman, 1983, p. 185).

Davenport published influential articles and books, edited journals, founded professional societies, and organized meetings of like-minded enthusiasts of scientific breeding in humans. His most significant contribution to the field, however, was acquiring generous funding to promote eugenics. In 1910 he persuaded Mary Harriman, the widow of the railroad baron E. H. Harriman, to contribute a large sum of money (equivalent to half a million of today’s dollars annually) to establish the Eugenics Record Office at Cold Spring Harbor, New York (Figure 12). Later financial support came from oil magnate John D. Rockefeller Jr. and the Carnegie Institute of Washington. The ERO had a twofold purpose: (1) to advance research in human genetics and, in particular, to elucidate the manner of inheritance of specific human traits; and (2) to educate the public about eugenics by disseminating findings and supporting nationwide eugenic education efforts (Allen, 1986). More than 250 fieldworkers were trained at the ERO (the majority of them young college-educated women) to administer mental tests, record physical measurements, and recognize insanity, criminality, epilepsy, and other conditions. Their training included doing genetic crosses in maize so that they would see Mendelian patterns of inheritance for themselves, patterns they were expected to find in the families they studied (Wilson, 2002). They fanned out to state hospitals, insane asylums, poorhouses, and reformatory schools, as well as people’s homes, ultimately amassing hundreds of thousands of pedigree charts, family histories, and other hereditary data, which were catalogued and stored at the ERO. The ERO’s pedigrees collected by Davenport and those constructed by the psychologist Henry H. Goddard were disseminated widely, appearing in hundreds of biology textbooks (Largent, 2008, p. 129) as well as in publications intended for a more general audience. Ostensibly serving an educational purpose, these pedigree charts became tools of propaganda, persuading the public of the dangers of hereditary degeneration and the urgent need for legislative remedies (Lombardo, 2001).

As a staunch Mendelian, Davenport considered every human characteristic to be controlled by a single gene with dominant and recessive alleles whose expression is not fundamentally altered by the environment (to be contrasted with a multifactorial trait, whose expression is affected by alleles of several − or even many − genes as well as by environmental conditions). Hair color and texture, eye color, temperament, mathematical ability, alcoholism, musical talent, muscular strength, nervousness, and “feebblemindedness” − all were equally simple Mendelian characters to Davenport, each determined by a single gene with a dominant and a recessive allele.

The first fruits of the ERO’s data-collection effort appeared in Heredity in Relation to Eugenics (Davenport, 1911). It contained just about all there was to know about human genetics at the time and included more than 150 pedigree charts, for everything from cataracts to criminality, from epilepsy to eroticism, from ichthyosis to insanity. Some of Davenport’s pedigrees did, in fact, reveal the mode of inheritance of a trait. He correctly deduced that not only was Huntington’s disease inherited as a dominant trait but so were achondroplasia and polydactyly. Albinism was found to be recessively inherited. His pedigree for albinism is reproduced in Figure 13. Examination of this pedigree reveals multiple skipped generations, a substantial proportion of affected females (9 out of 19), and several consanguineous matings, all characteristic of the autosomal recessive mode of inheritance. Note that this pedigree shows females and males not as circles and squares, but as the symbols ♀ and ♂, respectively, a convention followed by English eugenicists such as Karl Pearson (Resta, 1993).

Many of the pedigree charts Davenport presented in Heredity in Relation to Eugenics were of little value, however. Figure 14, showing the appearance of criminality in a family, serves to illustrate some of the problems in Davenport’s analysis. From the skipped generation in this pedigree, Davenport concluded that criminality is inherited as a recessive trait, “like most neuroses.” Upon closer examination, we find the basis for this inference very shaky indeed. What was the evidence for criminality in this family? Davenport related that the first affected person (I-3) was “a western desperado, drank hard and was involved in a murder.” His grandson (person II-2) was reported to have hitched a ride on a train at age three, run away from reform school 13 times, lied habitually, reneged on debts, and been convicted of burglary.

In Heredity in Relation to Eugenics, Davenport wrote that many other traits were mainly, if not entirely, inherited in a recessive mode, including musical, artistic, and literary abilities (p. 61), “bodily energy” (p. 93), epilepsy (p. 72), insanity (“neuro-pathic taint,” p. 77), alcoholism (“a strong hereditary bias toward alcohol,” p. 83), feebblemindedness (pp. 65–72), and sexual immorality (pp. 90–92).

In a later book, Davenport presented exhaustive evidence that the trait thalassophilia (literally “love of the sea”), a type of maritime wanderlust, was inherited in families of naval officers (Davenport & Scudder, 1919). One such family was that of British Vice-Admiral Cuthbert Collingwood, the hero of the Battle of Trafalgar in 1805 (Figure 13). On the basis of this and many additional pedigrees, Davenport concluded that thalassophilia was recessive and sex-limited because it often skipped a generation and occurred only in males. (His research had failed to uncover any naval officers who were women.)

Figure 12. The Eugenics Record Office archives room. Photo credit: www.eugenicsarchive.org.
Problems with Davenport’s Pedigrees Analysis

There are many problems with the pedigrees Charles Davenport constructed and interpreted using data collected by ERO field-workers. First, although many of the traits he considered were straightforward conditions such as albinism, polydactyly, and color blindness, many were complex and ill-defined, like alcoholism, musical ability, pauperism, mechanical skill, general mental ability, nervousness, and, of course, “feeblemindedness.” For pedigree analysis to be successful, the trait under study must be well defined and easily discriminated.

Second, the fact that a trait runs in a family does not prove that the trait has a genetic basis. It is not uncommon for traits that at first appear to be inherited to be found to have an environmental basis. (Such an environmentally caused phenotype that mimics a genetic condition is called a phenocopy.) Multiple occurrences of a disease in a family may be due to a bacterial or viral infection, a nutritional deficiency, or the exposure to an environmental toxin. It is important to remember that just because a trait is familial (i.e., running in a family), that does not necessarily mean it is genetic.

The list of familial traits that are unlikely to have a genetic underpinning is long: language and dialect, religious belief (or lack thereof), political affiliation, and even sports team allegiance. Other characteristics develop through a complex interplay of genetic predispositions and life experiences, notably musical talent, athletic ability, alcoholism, and mental illness. Two criteria must be met to establish a genetic causation (Mange & Mange, 1999):

1. The trait must occur more frequently among the genetic relatives of an affected person than in the general population.
2. The trait must not spread to unrelated persons exposed to similar environmental conditions.
Another indicator is when identical twins (who have 100% of their genes in common) share the phenotype more often than nonidentical (fraternal) twins (who share 50% of their genes).

A third mistake Davenport and others made is assuming that even those traits with a substantial genetic foundation were simple Mendelian characters. In other words, he believed that each trait was controlled by a single gene with a dominant allele and a recessive allele, and that the genetic cause was the same in every family. For example, Davenport presupposed that polydactyly, which he showed to be a dominant condition, had the same underlying genetic basis in every family in which it occurred. In fact, it is now known to be caused by mutations in at least six different genes (PAPA1, PAPA2, PAPA6, SRPS2A, PPD2, and SRPS3) (Online Mendelian Inheritance in Man), most of which are dominant but incompletely penetrant. This is an example of genetic heterogeneity, when the same phenotype may result from different genotypes. It is well known that mutations in the BRCA1 and BRCA2 genes increase a woman’s risk of developing breast cancer, but at least 11 other genes also raise breast cancer susceptibility when mutated (McClellan & King, 2010).

An even more extreme case is inherited deafness. At least 120 genes have been identified that, when mutated, result in deafness (Nance, 2003). The inheritance pattern may be autosomal dominant (i.e., gene GJB6), autosomal recessive (i.e., gene GJB2), or X-linked recessive (i.e., gene POU3F4). Hearing loss may even result from mutations in genes residing not on a chromosome but in the DNA of the mitochondrion (Kokotas et al., 2007).

There were also many problems in the way the ERO fieldworkers collected the data Davenport analyzed. Often the information gathered was subjective, based on cursory observation and guesswork by the poorly trained fieldworkers. Even worse, fieldworkers commonly made records of people they had never met, because they were either geographically distant or no longer living. These records were based on unreliable personal recollections of family members and published sources of questionable veracity.

Compounding these issues was that many of Davenport’s pedigrees were quite small, comprising a dozen or so persons in three, or sometimes only two, generations, with many family members unaccounted for. Figure 16 is one example – a pedigree showing “heart trouble.” Based on this one pedigree, the mode of inheritance of “heart trouble” could be autosomal dominant, autosomal recessive, or X-linked dominant. Only X-linked recessive is ruled out. Such small families make it difficult, if not impossible, to discern the mode of inheritance of a trait.

Yet another problem was that Davenport used anecdotes to support his hereditary claims. To him, the fact that the three Brontë sisters (Charlotte, Emily, and Anne) had each written a famous book was strong evidence of the hereditary nature of literary ability. He likewise took the similar professional accomplishments of John Roebling, the designer of the Brooklyn Bridge, and his sons, who finished the bridge and later supplied cable for the Golden Gate Bridge, as evidence that mechanical skill is inherited. Anecdotes, no matter how suggestive, do not count as scientific evidence.

Finally, Davenport and other eugenicists used circular reasoning in their analysis of human pedigrees. Here’s how it worked: First, a pedigree was produced that purported to show that a particular trait was inherited in a simple Mendelian fashion with a clear-cut mode of inheritance. Next, when this trait was found in another family, it was assumed that it displayed the same straightforward mechanism of inheritance as in that first pedigree. This new information was interpreted in such a way as to fit in this simple model, even when it plainly did not. This data manipulation lent credence to the original pedigree analysis, and elevated its conclusions to “scientific fact.”

Eventually, the flaws in the pedigree data gathered by ERO fieldworkers and analyzed by Charles Davenport became apparent. After reviewing the ERO in 1935, the year after Davenport’s retirement, a scientific committee concluded that this information was of little scientific value (Kevles, 1985, p. 199). The ERO was quietly shut down four years later, its influence having all but evaporated (Paul, 1998, p. 120).

Eugenics Old & New

The eugenics movement did not end when the Eugenics Record Office closed, however. As is well known, the most extreme expression of eugenics took place in Nazi Germany. Eugenics started slowly in German and for many years lagged behind the movements in England and the United States. German eugenicists monitored the developments in these countries closely and maintained close ties with such prominent American figures as Charles Davenport; his deputy, Harry Laughlin; the anthropologist Clarence G. Campbell; and, especially, the agriculturist and eugenicist Paul Poppenoe, from whom they gained inspiration (Kühl, 1994, p. 19).

One who followed American eugenics closely was future chancellor Adolf Hitler. A mere two months after he came to power in 1933, the Law for the Prevention of Genetically Diseased Progeny was enacted. Patterned on statutes in California and other states, which were themselves based on Laughlin’s Model Eugenical Sterilization Law of 1922, it mandated the sterilization of those with “feeblemindedness,” mental illness, epilepsy, hereditary blindness and deafness, Huntington’s disease, alcoholism, and physical deformities (Kühl, 1994, p. 39). In the first three years the law was in effect, 225,000 people were sterilized (Kevles, 1985, p. 117). Later, its reach was extended to the “socially feebleminded,” as determined by Hereditary Health Courts.

As the Third Reich gained momentum, German eugenicists took ever more sinister turns, as it became subsumed by Nazi racial policies. Forced sterilizations all but ceased after 1939, replaced by a program of euthanasia. The first to die in the gas chambers were mental patients, followed by homosexuals, other social and political outcasts, about 500,000 Gypsies, and, most horrifically, 6 million Jews.

The first published histories of the eugenics movement suggested that advances in genetics gradually eroded the scientific foundation of the eugenic program, exposing the movement’s leaders to ridicule. This was accompanied by a waning of interest in eugenics during the Great Depression of the 1930s when the country had more pressing concerns than controlling human heredity. The movement was finally laid to rest when the atrocities of the Nazis came to light at the end of World War II. More recent scholarship has challenged this narrative (Paul, 2016).

Compulsory sterilization of the “unfit,” the blunt instrument of negative eugenics, did not stop when the original eugenics

Figure 16. Davenport’s pedigree showing the appearance of “heart trouble.”
movement ran out of steam and the ERO shut down. In fact, eugenic sterilizations increased during the depression of the 1930s as new state laws were enacted and older ones were more vigorously enforced. Sterilizations peaked in 1933 and did not begin declining until 1944 (Largent, 2008, p. 77). Coerced sterilizations continued to be performed for more than two decades after the end of World War II, although they were increasingly targeted at the poor and ethnic minorities rather than the “feebleminded” (Largent, 2008).

At the same time, many so-called reform eugenicists began making common cause with birth-control advocates in the United States, England, and elsewhere. Their goal in providing information and distributing contraceptive devices was not so much granting women control over their reproductive lives as it was reducing the fertility of the poor (Klausen & Bashford, 2012).

In the years just before and after World War II, eugenically minded geneticists helped develop a new medical specialty, which became known as genetic counseling. As with Davenport thirty years earlier, the pioneers in the field relied heavily on pedigree construction and analysis, although they were careful to confine themselves to conditions with a clear hereditary basis (Largent, 2008). A hallmark of genetic counseling today is the principle of nondirectiveness; counselors present information and outline options to couples but do not recommend a course of action. But in the formative days of the profession, counselors took into account not only the welfare of the couples seeking assistance but also the future hereditary health of the entire population (Resta, 1997). These early hereditary clinics were thus intended to have a role in shaping human evolution by preventing the occurrence of inherited disease (Paul, 1998, pp. 125–127; Comfort, 2012, p. 119).

Interestingly, Davenport’s *Heredity in Relation to Eugenics* may be considered the first handbook on genetic counseling (Reilly, 2008, p. 160). In it, he advised those with a family history of an inherited disorder not to have children (a practice that today we would call directive counseling). He recommended against marriages between first cousins and other close relatives and went so far as to suggest that sisters of hemophiliacs should opt to remain childless (see Davenport, 1911, pp. 118 and 157).

It is now clear that eugenics did not dwindle in the 1930s and disappear altogether at the end of World War II. Rather, it was reconceived, adapting to new developments in biology and medicine. Along the way, it was rebranded, shedding the unpalatable label “eugenics” in favor of such terms as “medical genetics” and “social biology,” all the while adhering to many of the goals of the original movement (Paul, 2016).

More than a century after Charles Davenport standardized the construction of pedigrees charts, pedigrees remain an essential tool in human genetics. But in recent decades, a host of genetic and reproductive technologies have been invented and perfected, including in vitro fertilization, preimplantation genetic testing, gene therapies, stem cell treatments, and, most recently, CRISPR gene editing. These technologies make possible a new eugenics of scope and power unimaginable to Davenport. It will be driven not by propaganda campaigns and state-directed programs, but by consumer preferences stoked by a vast biotechnology industry. It will be, in other words, not an authoritarian eugenics but an individualistic eugenics.

Few would dispute that modern genetic technologies hold enormous promise for the diagnosis, treatment, and prevention of inherited diseases and that this promise has only partially been realized. But the use of these technologies raises many thorny questions (see, e.g., Baylis, 2019). Can we harness the powerful new genetic tools to cure disease without engendering a new era of eugenics (Comfort, 2018)? Or is a 21st-century eugenics inevitable? If it is, will its benefits be equitably distributed throughout society, or will only the wealthy stand to gain, along with the industry that caters to their needs? Will genetic knowledge be used more wisely than it was a century ago, when pedigree charts served as the “scientific” justification for discrimination against the disadvantaged, extending so far as institutionalization and sexual sterilization? Have we learned the lessons of the original eugenics movement well enough to navigate the ethical waters that lie ahead? Or will future generations judge us for our ethical lapses as critically as we now judge Charles Davenport (Paul, 2014)? Only time will tell.

### References


MARK SHOTWELL is an Associate Professor of Biology at Slippery Rock University, Slippery Rock, PA 16057; e-mail: mark.shotwell@sru.edu.