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Teaching School Genetics in the 2020s: Why "Naive" Mendelian Genetics Has to Go

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Whereas Mendelian genetics is an important research program in the life sciences, its school version is problematic. On the one hand, it contains stereotypical representations of Gregor Mendel's work that misrepresent his findings and the historical context. This deprives students from gaining an authentic picture of how science is done. On the other hand, what most students end up learning in schools are extremely simplistic accounts of heredity, whereby alleles directly control traits and phenotypes, and thus exclusively depend on which allele an individual has. Such oversimplifications of Mendelian genetics as those that we still teach in schools were exploited by ideologues in the beginning of the twentieth century to provide the presumed "scientific" basis for eugenics. This paper addresses these problems of the school version of Mendelian genetics, which I call "naive" Mendelian genetics. It also proposes a shift in school education from teaching how the science of genetics is done using model systems to teaching the complexities of development through which heredity is materialized.

Mendelian genetics is a research program that dominated the first half of the twentieth century, whereas it continued to be important during its second half. Nowadays, it is still important in research in medical genetics, population genetics, and elsewhere. The respective undergraduate teaching usually begins with the friar Gregor Mendel and his work and then moves on to more complicated models and analyses. However, when it comes to school genetics, Mendel and his work with peas, as well as some similar simple models about genetic inheritance, is all that students ever learn. The most advanced level school students usually reach is being able to resolve problems with crosses using Punnett squares. Students are usually told that there exist alleles "for" traits, which they represent on these Punnett squares and the respective crosses, without a deeper reflection about the underlying developmental processes that connect allele expression to a phenotype.

As a result of this, instead of teaching school students about heredity and development, they end up being taught about the models that we can use to study genetic inheritance. They also learn that a heroic figure, Gregor Mendel, singlehandedly figured out how heredity takes place while being way ahead of his time and his contemporaries. The way we teach Mendelian genetics in schools thus deprives school students from gaining an authentic image of both hereditary phenomena and of the nature of science. Because

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of this, I hereafter call the school version of Mendelian genetics "naive" Mendelian genetics. The adjective "naive" does not have a derogatory meaning here, but only indicates the lack of depth and sophistication that characterizes school Mendelian genetics compared to the respective scientific research program. In this paper,[1] I argue that there are two kinds of problems with the teaching of "naive" Mendelian genetics in schools.

The first problem I call "anachronistic Mendelism." The story of Mendel as a lonely genius ahead of his time not only makes historically inaccurate statements about the foundations of the science of genetics, but also distorts how science is actually done by scientific communities. Science is a social process, not the outcome of eureka moments of individual (usually male and white) geniuses. The study of the history of science clearly shows that any scientist who has ever made any important advancement has had to rely on the work of numerous others, both predecessors and contemporaries.

The second problem I call "social Mendelism," a term borrowed from the historian Amir Teicher (2020). This relates to the fact that Mendelian genetics, as it developed during the early twentieth century, was primarily and predominantly based on William Bateson's interpretation of Mendel's paper. From this, simplistic models of Mendelian inheritance emerged that were invoked in eugenics and eventually in the racial hygiene of the Nazis to account for complex mental human traits, even though there was already at the time abundant evidence to the contrary. My concern is that very similarly simplistic models of Mendelian inheritance are still taught in schools today.

Let us then look at these two problems in more detail.

ANACHRONISTIC MENDELISM

Consider the following excerpt from a popular science book written by a Pulitzer-prize winning author:

> We begin with Mendel's pea-flower garden, in an obscure Moravian monastery in 1864, where the "gene" is discovered and then quickly forgotten.… Had Mendel stopped his experiments here, he would already have made a major contribution to a theory of heredity. The existence of dominant and recessive alleles for a trait contradicted nineteenth-century theories of blending inheritance; the hybrids that Mendel had generated did not possess intermediate features. Only one allele had asserted itself in the hybrid, forcing the other variant trait to vanish.… Each trait was unitary—distinct, separate, and indelible. Mendel did not give this unit of heredity a name, but he had discovered the most essential features of a gene.
>
> —Mukherjee (2016, pp. 13, 51, 53)

This is the account of Mendel's life and work that we can also find in most textbooks, or after a quick web search about Gregor Mendel. This is the stereotypical account of Mendelian genetics, which comprises at least the following ideas: that the study of genetics began with Mendel, that no one understood Mendel's paper that contained the entire basis of modern genetics, that he postulated the existence of particulate factors—later called genes—which are transmitted from parents to offspring, and that he also found how this transmission occurs by what became known as the law of segregation and the law of independent assortment. While all this is presented, the stereotype of a lonely genius being ahead of his time comes to life: the lonely monk (he was actually a friar, and the difference is important) in the garden conducting experiments while being entirely isolated from his social and scientific context. The problem with this account is that it distorts both the history of science and science itself.

This account has been questioned at least since 1979 by historian Robert Olby and sociologist Augustine Brannigan (Brannigan 1979, 1981; Olby 1979, 1985). Throughout his 1866 paper, Mendel referred to characters, not any underlying factors or alleles, and he used the term "hybrid traits" to refer to a character being a hybrid, not the traits of hybrids (Müller-Wille and Hall 2020). What was dominant and recessive in his view were the characters themselves. Mendel wrote about characters and character frequencies, and what he managed to observe

[1]This essay is a summary of the historical evidence and the arguments presented in complete form in my book *How We Get Mendel Wrong and Why It Matters* (Kampourakis 2024).

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was the segregation and independent assortment of characters, not alleles (even though he regarded the visible characters as the upshot of material in the gametes). In short, Mendel did not have the perspective of what later came to be known as Mendelian genetics (Olby 1979). This becomes evident from the striking differences between the way Mendel presented his results and their modern representations, as well as from the differences in terminology (Table 1).

Mendel's paper was not an attempt to develop a theory of heredity; rather, he was interested in hybridization in the agricultural context of Brno in which he was working. Therefore, Mendel was not a man ahead of his time, discovering laws none of his contemporaries understood, but a man of his time working on particular practical problems of agricultural interest that were important for the society in which he lived. Mendel was, in fact, working in the experimental hybridist tradition of Kölreuter, Gärtner, and others, which inadvertently led to a focus on the inheritance of individual traits (Müller-Wille and Orel 2007; see also Olby 1985). Some conclusions about Mendel from his paper are therefore that:

- He intended to study hybridization in particular and not heredity in general,
- He studied how traits were passed on from one generation to the next one, and nowhere did he write about hereditary particles that determine these traits,
- He was able to observe the segregation and independent assortment of traits, but he did not conclude anything about the segregation and the independent assortment of alleles (Kampourakis 2015).

A careful study of history also makes clear that Mendel's work was not ignored, but that it was just irrelevant to the attempts of various scholars to develop a theory of heredity at the end of the nineteenth century. Beginning with Charles Darwin himself, Herbert Spencer, Francis Galton, William Keith Brooks, Carl von Nägeli, August Weismann, and Hugo de Vries attempted to develop theories of heredity under an evolutionary perspective. Whereas they were all influenced by one another in various ways, only Nägeli became aware of Mendel's experimental work, which was nevertheless referenced in various journals and books widely read at the time. Attention was not paid to it, perhaps because it was not an explicit attempt to develop a theory of heredity, as several others did. But in 1900, Mendel's paper was read under a new light, in part because Galton and Weismann

Table 1. Differences in the presentation of experimental results of a two-character (or "dihybrid" cross), between Mendel's paper (left) and modern representations of his work (right)

Experimental result: 315 round/yellow: 101 angular/yellow: 108 round/green: 32 angular/green	
Mendel's terminology (A and a are characters): • "A" was the dominant character that remained constant through generations • "a" was the recessive character • "Aa" was the hybrid form that exhibited the dominant character but which somehow included the recessive one as well, as they both segregated in offspring	Modern terminology (A and a are alleles in the genotype): • "aa" is the genotype homozygous for the recessive allele • "AA" is the genotype homozygous for the dominant allele • "Aa" is the genotype of the heterozygote
Mendel's ratios	**Modern ratios**
Two classes resembled each of the parental ones (AB and ab):	Nine round/yellow:
Two classes had one character from each parental form (Ab and aB):	Three angular/yellow:
Four classes appeared twice and had a parental and a hybrid character (ABb, aBb, AaB, Aab):	Three round/green:
One class, hybrid in both characters (AaBb), appeared four times	One angular/green
or 1AB: 1ab: 1Ab: 1aB: 2ABb: 2aBb: 2AaB: 2Aab: 4AaBb	or 9A*B*: 3A*bb: 3aaB*: 1aabb (*: either allele could be there)

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had developed a new framework of discontinuous variation and germline inheritance in which Mendel's experimental approach could nicely fit (Robinson 1979; Kampourakis 2013).

Brannigan has also explained that Mendel was brought back to the scene in 1900 to resolve a potential priority dispute between two of his "rediscoverers," Hugo de Vries and Carl Correns. Correns was probably long aware of Mendel's paper through his teacher Nägeli with whom Mendel had a long correspondence. Correns was also the first to refer to Mendel in his 1900 paper (Correns 1950). As de Vries had not done so in his first article presenting the Mendelian ratios (de Vries 1950), Correns might have thought that de Vries was claiming Mendel's results for his own. By labeling the discovery "Mendel's Law" in his article title, Correns insisted on Mendel's priority over both de Vries and himself. The title that Correns used in his paper is perhaps the most important factor in the reification of Mendel as the founder of genetics (Brannigan 1979).

It was William Bateson's interpretation of Mendel's paper that established the notations that are still used in Mendelian genetics. Bateson (1902, p. 80) actually commented that Mendel's notation (such as those used in the left part of Table 1) was not accurate and it had better be replaced by the one he proposed (such as the one used in the right part of Table 1). Despite criticisms by Raphael Weldon and others, Bateson persisted, and, following Weldon's untimely death in 1906, his interpretation eventually prevailed. In essence, what we call today Mendelian genetics had better be called Batesonian genetics, as it was Bateson who coined most of the key terms and notations, and it was his work with his collaborators that established the model of Mendelian genetics as used in research. His close collaborator, Reginald Punnett, was also the person behind the scheme that came to be known as Punnett's square. By adopting a model based on the presence and absence of factors, Bateson and his colleagues were able to explain the inheritance of several features. When this was not possible, new concepts, such as epistasis, were invented to accommodate the new findings (Radick 2023).

We thus see that the true story of Mendel is more complex and more interesting than the stereotypical account of the lonely genius being ahead of this time. This account provides a severely distorted image of science that does not present it as what it really is: a social process. But the distortion of how science is done is not the most important problem with the "naive" Mendelian genetics taught in schools. There is a more important problem to address when it comes to representations about the way alleles and traits are related. In the past, such simplistic representations formed the basis for extreme abuse. It all started with another person born, like Mendel, in 1822: Francis Galton.

SOCIAL MENDELISM

Galton was a prolific writer and a polymath but nowadays is mostly remembered as the founder of eugenics. He was mostly interested in what came to be called "positive" eugenics: encouraging "superior" individuals (whatever that meant) to mate and produce children. However, as it was soon realized that "inferior" individuals (of lower classes in England or of non-white races in the United States) were reproducing at a higher pace than the "superior" ones, many eugenicists became advocates of what came to be called "negative" eugenics. This was about preventing "inferior" individuals from reproduction, which was possible either by segregation (that is, social isolation, usually by restricting them in asylums) or by sterilization. Only the former of these practices was legislated in Britain, where the idea of eugenics was born. When, in 1907, Galton stepped down as Director of the Eugenics Record Office at University College London (UCL), the statistician Karl Pearson took over. Pearson renamed it the Galton Laboratory for National Eugenics and became the first Galton Professor of Eugenics at UCL. Pearson thus became the most prominent eugenicist in Britain (Kevles 1995).

Pearson was a fervent and vocal anti-Mendelian, and a close friend and collaborator of Raphael Weldon. He became interested in eugenics around 1900 and, being a lot more competent than Galton in mathematics, he was in-

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strumental in developing key statistical methods for the analysis of data. Like Galton's, Pearson's approach to eugenics was primarily mathematical, and it had absolutely nothing to do with Mendelian genetics. In fact, Pearson and Weldon jointly battled Bateson and the Mendelians in the 1900s until Weldon's untimely death in 1906. An important eugenic concern for Pearson was the problem of "indiscriminate immigration" of Polish and Russian Jews to London, as well as to poor areas in other towns in the country. He considered indiscriminate immigration destructive for the goal of the nation reaching a superior level because of the "influx" of inferior material. A 1925 paper, with Pearson as its first author (Pearson and Moul 1925), stated that:

> The whole problem of immigration is fundamental for the rational teaching of national eugenics. What purpose would there be in endeavouring to legislate for a superior breed of men, if at any moment it could be swamped by the influx of immigrants of an inferior race, hastening to profit by the higher civilisation of an improved humanity? To the eugenist permission for indiscriminate immigration is and must be destructive of all true progress. Such progress is only possible where intra-racial selection is combined with a large measure of isolation.

Since the Mental Deficiency Act of 1913, detention in institutions was implemented as the only measure to address this problem.

The situation was quite different in the United States. By the late 1900s, several states already had sterilization laws. At the same time, many geneticists had become proponents of eugenics, arguing that the propagation of defective traits, inherited as Mendelian recessive, should be stopped. The first and most important proponent of this view was Charles Davenport, who founded the Eugenics Record Office in 1910 at Cold Spring Harbor, and who for years collected and analyzed pedigree data to account for the inheritance of human traits. Here is what Davenport wrote in 1909 (pp. 20–21):

> First, the scandal of illegitimate reproduction among imbeciles must be prevented. That class often shows a frightful fecundity. If segregation is inadequate protection and since reason cannot overcome the sentiment against destruction of the lowest-grade imbeciles, at least operations should be required that will prevent the reproduction of their vicious germ plasm.
>
> Second, the old idea that there is in society any class that is superior to any other class should be abandoned. It is the characteristics of the-germ plasm and not individuals as a whole that are favorable or prejudicial to human society. The way to improve the race is first to get facts as to the inheritance of different characteristics and then by acquainting people with the facts lead them to make for themselves suitable matings. The only rule, a very general one, that can be given at present is that a person should select as consort one who is strong in those desirable characters in which he is himself weak, but may be weak where he is strong.

One trait that many geneticists, including Bateson's close collaborator Punnett, considered important to deal with was "feeblemindedness," which is harder to define than to spell. The presumed threat of this trait became popularized via a 1912 book by Henry Goddard, *The Kallikak Family* (Goddard 1912). Even though Goddard stated that he was not sure whether "feeblemindedness" was inherited as a Mendelian recessive trait, the whole book is an argument about this, as it shows the two sides of a family with the trait prevailing in the one and missing in the other (Paul 1995). It was the close interaction between Davenport and Goddard that marked the beginning of what can be described as "Mendelian eugenics," that is, the eugenics based on Mendelian genetics (and actually very simplistic ones). Davenport and Goddard looked for evidence that confirmed that "feeblemindedness" was inherited as a Mendelian recessive trait, and they collected all the data that stood as evidence for this. So, even though eugenics was not right from the start Mendelian, it took the Mendelian turn around 1909 through the work of Davenport and Goddard. This matters because as historian Nathaniel Comfort nicely put it: "Eugenics became evil when it became Mendelian" (Comfort 2023, p. 3).

The reason for this was that Mendelian eugenics provided the "scientific" background for sterilization laws in the United States. Although many states had adopted such laws, these were

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not really implemented, with the exception of California. However, after the Supreme Court of the United States approved the sterilization of Carrie Buck in 1927, more sterilizations were made. The decision by Justice Holmes is chilling:

> It is better for all the world if, instead of waiting to execute degenerate offspring for crime or to let them starve for their imbecility, society can prevent those who are manifestly unfit from continuing their kind. The principle that sustains compulsory vaccination is broad enough to cover cutting the Fallopian tubes. Jacobson v. Massachusetts, 197 U.S. 11. Three generations of imbeciles are enough (Buck v. Bell, 274 U.S. 200, 1927; https://supreme.justia.com/cases/federal/us/274/200/).

Not all scientists agreed with the assumptions of Mendelian eugenics. Thomas Hunt Morgan and his colleagues were sensitive to the limitations of Mendelian genetics (Allen 1978). In 1915, they provided a clear conception of what genes might do and how they might do it, that is, their function as difference-makers for phenotypes:

> Mendelian heredity has taught us that the germ cells must contain many factors that affect the same character. Red eye color in *Drosophila*, for example, must be due to a large number of factors, for as many as 25 mutations for eye color at different loci have already come to light…. One can therefore easily imagine that when one of these 25 factors changes, a different result is produced, such as pink eyes, or vermillion eyes, or white eyes or eosin eyes. Each such color may be the product of 25 factors (probably of many more) and each set of 25 or more differs from the normal in a different factor. It is this one different factor that we regard as the "unit factor" for this particular effect, but obviously it is only one of the 25 unit factors that are producing this effect.

This view is something that even today we fail to explain to students (Kampourakis 2021a). You can compare this to the following excerpt from the popular science book already mentioned in the previous section: "In genetic terms, this suggests a peculiar paradox. Sex, one of the most complex of human traits, is unlikely to be encoded by multiple genes. Rather, a single gene, buried rather precariously in the Y chromosome, must be the master regulator of maleness.… Is all of sex just one gene, then? Almost." (Mukherjee 2016, pp. 359–360, 362). Well, as Morgan and his collaborators had already explained in 1915, no character can ever be the product of a single gene.

Some geneticists such as Edward Murray East and Punnett argued that to deal with this issue, it was not enough to identify the "feebleminded" persons and preclude them from reproduction. To eliminate this trait, it would be necessary to identify the many more heterozygote carriers of the trait who would keep spreading it in the populations. Others like Ronald Fisher criticized such views, arguing that already dealing with the "feebleminded" persons themselves would bring about a big change. But other geneticists and scientists began criticizing the simplistic explanations for the inheritance of "feeblemindedness," starting with Morgan in 1925. Others soon followed, including Lancelot Hogben and J.B.S. Haldane (Barker 1989). The first devastating critique of Goddard's theory was made in 1925 by psychiatrist Abraham Myerson (Myerson 1925), with Goddard himself subsequently accepting in print that he might have been wrong (Goddard 1927). Gradually, support for eugenics was lost for various reasons (Zenderland 2001).

What is less well known is that the Nazis relied significantly on Mendelian genetics for their eugenic policies. It was actually in Nazi Germany where "Mendelian" eugenics took its most extreme form, with thousands of sterilizations and mass exterminations of people. The concept of "recessive factors" was key in this case. What was of concern to the Nazis was the potential to transmit such recessive factors related to pathologies to the next generations, not whether a person showed such pathologies. The discussions that led to the 1935 race laws (Fig. 1) began with considerations based on Mendelian genetics. Arthur Gütt, Head of the Public Health Department in the Reich Ministry of the Interior, and chairman of the Expert Advisory Board for Population and Racial Policy of the Ministry, submitted a memo during the discussions that included explanations of Mende-

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Figure 1. A 1936 chart explaining the Nuremberg Laws. It distinguishes the hierarchal difference between German-blooded, Jews, and Mischling (part Jewish) based on their grandparent's "race." Mischling are further broken down into two grades: first class, who are half-Jewish (having two Jewish grandparents); and second class, who are one-quarter Jewish (having one Jewish grandparent). These three "races" were codified by the Reich Citizenship Law and the Law for the Protection of German Blood and German Honor, collectively known as the Nuremberg Laws, which were passed in Germany in September 1935. These laws prohibited "race defiling," that is, marriage and sexual relations between pure Germans and Jews. The chart states that Jews are not German citizens and can marry only other Jews and Mischling. The laws rejected the traditional view of Jews as members of a religious or cultural community and claimed that they were a race defined by birth and blood. This new definition included tens of thousands of people who had no Jewish cultural affiliations, such as those who had converted to Christianity. The chart was designed by Willi Hackenbergeer, the propaganda leader of the Reich Committee for Public Health Service, and it was available for purchase for one Reichsmark from the Reich Committee for Public Health Service. (Chart provided by the U.S. Holocaust Memorial Museum Collection, gift of Virginia Ehrbar through Hillel at Kent State University.)

lian genetics, beginning with a Punnett square on crosses between guinea pigs (the memo is titled "Department of Public Health-Reich and Prussian Ministry of the Interior, 25.9.1935, Principles of Mendelian Inheritance"). A central concern for the Nazis was to prevent the unwanted results of "mendeling out" (herausmendeln): the sudden reappearance of ancestral traits that had been hidden for several generations in a mixed population. There was only one way to avoid this in Mendelian terms: achieving purity via homozygosity (Teicher 2020). As is evident in Figure 1, Mendelian genetics formed the basis for the explanation of the race laws, with being Jewish considered as a Mendelian recessive trait.

The textbooks and teaching aids of the Nazi regime were also the outcome of a fusion of biology facts with Nazi ideology. Alfred Vogel, a curriculum developer and school headmaster, produced in 1938 a series of teaching aids that conveyed the ideological messages of the Nazis. Topics for biology education included the heredity of physical, mental, or spiritual character-

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istics, the heredity of disease, the heredity of physical and mental characteristics of the German race, the "law of selection," and "the Jews and the German people" (Pine 2010, pp. 43–44). To create a racially pure German nation, all those who did not belong to the Nordic race should be excluded. Therefore, to achieve this and therefore racial purity, students had to be educated about the deteriorating effects of mating with non-Nordic people. In one example, Vogel drew parallels between crossbreeding in plants and racial mixing. By comparing "pure" flower varieties with "mixed" ones, he aimed to show how the racial purity of the German people could be maintained if they mated only with one another or deteriorate if they mated with people from other races (Fig. 2; see also Wegner 2002, Chap. 3). Unfortunately, this happened when most geneticists had become critical of the "naive" Mendelian genetics initially invoked in support of eugenics. Mendel is not to blame, of course, and his name and legacy were abused. It is the simplicity of "naive" Mendelian genetics that allowed the Nazis to do so. This simplicity also still leads to misunderstandings today.

If "naive" Mendelian genetics paved the way for eugenics and racial hygiene, why do we still teach it in schools? I am not suggesting that by teaching Mendelian genetics in schools today, there is the danger of a new Holocaust. However, letting most students who finish school have as their main knowledge an oversimplified account of Mendel's laws and Punnett squares deprives them of a deeper understanding of heredity, as most of them will not become biologists and will likely not be taught anything further. This deeper understanding is necessary for a deeper understanding of ourselves and our life for at least two reasons.

On the one hand, many racist arguments have been based on mistaken assumptions of the biological/genetic superiority of some groups over others. As we saw, eugenics was based on this false assumption, with tragic consequences. Unfortunately, we continue to talk about genetic differences between human populations who are distinguished from one another primarily on the basis of cultural characteristics and some, few and obvious, biological characteristics, such as skin color, overlooking the fact that all humans have 99.9% of the same DNA (Kampourakis 2023).

On the other hand, the overemphasis of the potential of genes can have an important negative consequence: it relieves both the individual and society of responsibility for the manifestation of socially negative traits and diseases. It is interesting how convenient it is to attribute such traits to genes and fatalistically accept the result. By saying this, of course, I do not mean that our DNA has no role in the manifestation of such traits; obviously, it does, especially in the case of single-gene traits, which are not the norm. It is just that DNA is not the only factor contributing to these traits (Kampourakis 2021b).

CONCLUSIONS

There is evidence that the teaching of genetics that is based on the usual representations of genetics in textbooks can cause problems. This has been shown by the work of Brian Donovan and his colleagues. Overall, their research—based on randomized control trials—has shown that when secondary students are taught about the prevalence of monogenic disorders in different racial groups, their genetic essentialist beliefs (that traits are due to inner essences, with genes being the usual placeholder for this) are strengthened. In one study with 8th-grade students, the learning outcomes of two groups were compared: the students in one group had been taught about the prevalence of genetic disease, such as sickle cell anemia, in different races, whereas the students in the other group had been taught about the same diseases without any reference to race. The comparison showed that the students in the former group were more likely to believe that each race must be genetically homogeneous and discrete from others. These findings were replicated with 7th- and 9th-grade students in subsequent studies. In another study with 8th- and 9th-grade students, Donovan and colleagues showed that students' belief in genetic essentialism could be reduced through a five-lesson sequence about how low human genetic diversity is among groups, that is, that most genetic variation is found within rather than between continental groups. These results were replicated in two

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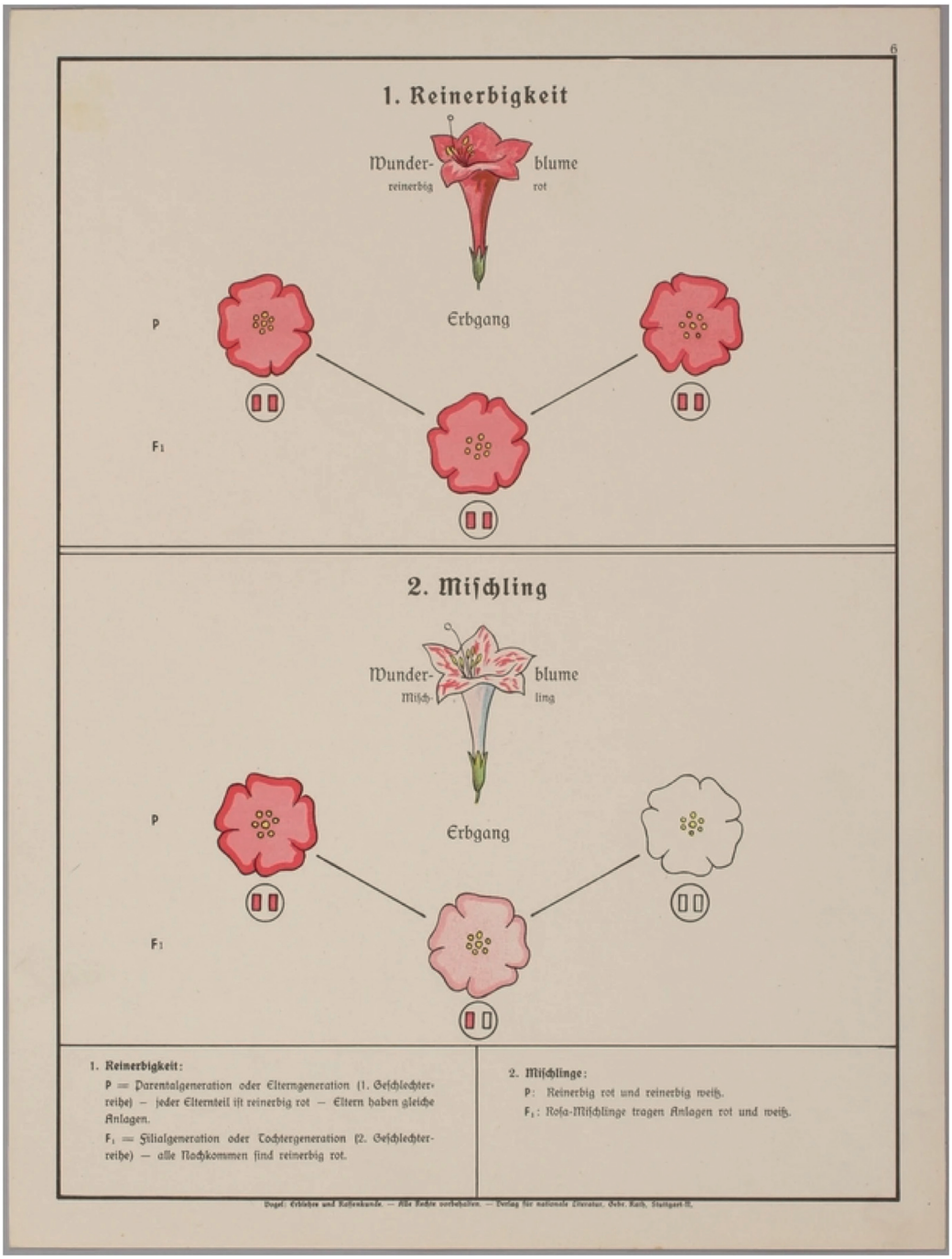

Figure 2. Poster from the 1938 racial science textbook, *Erblehre und Rassenkunde für die Grund-und Hauptschule (Heredity and Race Studies for Primary and Secondary Schools)*, by Alfred Vogel. The *upper* half depicts genetic "purity," the *bottom* half "mixed-breed" (Image provided by the United States Holocaust Memorial Museum Collection.)

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more studies with adults and 9th–12th graders. The researchers concluded that in all three studies, the intervention about human genetic variation resulted in a reduction of students' belief in genetic essentialism by changing their perception of the uniformity and the discreteness of races. Finally, the researchers compared the effect of this teaching about human variation that emphasizes multifactorial genetic inheritance and human genetic diversity to the material typically taught in schools with groups of 7th- to 12th-grade students. The comparison showed that the students in the former condition showed a reduction in their genetic essentialism beliefs (Donovan 2022).

There is, therefore, an urgent need to reconceptualize school genetics; instead of teaching deceptively simple models that implicitly convey the message that genotypes determine phenotypes, we had better make a shift toward the teaching of heredity in schools (see Kampourakis 2024 for specific suggestions). Two hundred years after Mendel's birth, we have failed to appreciate his real contribution. Mendel did not provide a mechanism of heredity in 1866; however, he developed an experimental approach that would become the foundation of the science of genetics. The acceptance of Mendel's work depended not simply on the discovery of facts, but rather on the development of a new conceptual framework. Mendel's paper had an impact in 1900 because the context in which it was read had changed. Galton and Weismann had created a framework that supported the idea of "hard" heredity, with discontinuous variation and nonblending characters. In addition, cytologists supported the view that particles inside the cells might have been controlling the emergence of traits. Thus, in 1900, Mendel's paper could have been regarded as bringing together the results of breeding experiments with the facts of cytology while also providing an experimental approach for the study of heredity. Still, in 2022, some scientists tend to ignore the work of historians that explain this and devote themselves to a hero-worshipping of Mendel (e.g., Nasmyth 2022).

Furthermore, teaching Mendelian genetics in schools misrepresents what we currently know about heredity and the relationship between traits and genes, as well as the history of science with respect to what Mendel actually did. In the current postgenomic era, where genetics is still misused to justify discrimination with respect to race, sex, ethnicity, and other identities, we had better give students a sense of the complexity of heredity, which is masked entirely by "naive" Mendelian genetics. Concepts, such as the Mendelian gene, can have a heuristic value in research, yet this does not require that they correspond to real entities; even though the "gene" was initially conceived as a difference-maker, that is, one of the several different causes of a phenomenon, it eventually evolved to being considered as the main cause. As a result, traits are presented as being dichotomous, even though they are usually not—natural variation is ignored. Furthermore, the relation between traits and genes is presented as being one-to-one; even though it is really many-to-many, single genes are difference makers, they do not determine traits.

Mendel was an ingenious plant breeder of his time, not a lonely genius ahead of his time who understood heredity. Mendel genetics as usually taught in schools provides oversimplified, and often inaccurate, explanations for hereditary phenomena. Perhaps we should drop Mendelian genetics altogether from genetics curricula in schools. If we teach about Mendel, we should teach about what he did and why he did it in its own context, and not present his work in anachronistic terms (Radick 2016). Mendel's story, as it is currently taught in schools, can provide useful lessons neither for understanding science, nor for understanding today's genetics.

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