

Mending Mendelism

Douglas Allchin

EVER wonder why genetics is so challenging to teach? And why problems remain a full century after the landmark revival of Mendel's work in 1900? I note, in particular, certain widespread, perhaps notorious, misconceptions that frequently persist even after explicit instruction. Such notions as: some traits are more likely to be inherited than others; dominant traits are more frequent in the population; adaptive traits eventually become dominant through natural selection; mutations or "abnormal" genes are recessive; dominant alleles subdue or control recessive ones. In every case the non-technical meaning of dominance—based on power, strength and value—shapes student thinking (Donovan 1997). Here I explore the problem of dominance as a concept, trace its persistence to the history of our mythic images of Mendel, then suggest how we can mend the flaws, while providing at the same time a valuable lesson in the history and nature of science (BSCS 1993). We may thus honor Mendel in this centenary year by appreciating the historical import of one of his concepts.

Three Problematic Conceptions

(1) **First, consider a common student question: how does dominance "work"?** Just as the Mendelian principles of segregation and recombination "reduce to" the behavior of chromosomes in meiosis and fertilization, so too do we expect a lower level explanation for dominance. Ultimately, though, there is no general mechanism for dominance in molecular or cellular terms. This reveals one of three major conceptions about dominance (at least, among non-geneticists).

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Textbooks rarely address this matter. As a result, two conceptions typically emerge—both mistaken. First some conceive dominance as a form of gene regulation.

That is, the dominant allele somehow inhibits or suppresses the expression of the recessive allele. One gene *dominates* the other. While plausible, this does not in fact occur. No protein or messenger molecule mediates the behavior of homologous alleles (except in immune cells). Rather, they are expressed independently, in parallel.

Others conceive dominance and recessiveness as the presence or absence of a trait, protein or gene product (e.g. Lewin 2000, p. 19). Here, one sees the phenotype as switched on or off. Geneticists in the early 1900s actively debated this theory. While it describes some cases well, it is misleading as a general model (Rothwell 1983, pp. 14–15). First, the recessive allele is generally transcribed into mRNA. Depending on the specific allele, it also produces a polypeptide. The function of the resulting protein may vary. In some cases, an enzyme may lose its catalytic activity [see Guilfoile (1997) on the classic traits smooth/wrinkled seed in peas and white-eye in fruit flies]. In other cases, however, the alternate protein may serve as a different product, catalyze a different reaction, accelerate or slow down its reaction rate, or perhaps modify multimeric assembly. In sickle-cell anemia, for example, the alternate hemoglobin molecules carry oxygen like their normal counterparts, but only at high partial pressures. Where oxygen is scarce, the proteins bind to each other in long chains, alter cell shape, and block capillary blood flow. Sickle-cell anemia as a disease is hardly well described as the absence of hemoglobin or its oxygen-carrying capacity. Consider also osteogenesis imperfecta. Patients with this condition have a deficit of collagen fibers. They are extremely susceptible to bone injury. One allele alone can generate the life-threatening condition. The presence-absence model

"The Nine Lives of Gregor Mendel," by Douglas Allchin.

The Andy Warholesque image helps mark the centenary of the revival of Mendel's work and the iconographic status of Mendel in biology education today. The title comes from an essay by historian Jan Sapp, who describes how Mendel's ideas are portrayed in nine different—and conflicting—ways by various biologists and historians. In each case, personal perspectives and theoretical commitments shape how individuals interpret Mendel as they seek to garner his prestige and bolster the authority of their own claims. In this article, Allchin considers how the concept of dominance has been modified and entrenched in the century since its "rediscovery." His analysis ultimately leads to new ways to address the widely recognized challenge of teaching genetics, while providing a striking lesson in the history and nature of science.

implies, awkwardly, that the healthy condition (recessive) is only the “absence” of the disease. As these cases demonstrate, not all recessive traits are non-functional. A recessive phenotype is an *alternative* phenotype. Dominant and recessive traits do not map simply onto a trait’s presence or absence.

Expecting dominance to exhibit some universal mechanism—whether gene regulation or presence-and-absence—reflects a deeper misconception: that dominance is a causal property inherent in the trait or allele itself. Rather, dominance is just an observed pattern. It emerges only distantly by viewing the *coupled* expression of *two* alleles. Labeling traits or alleles as individually dominant or recessive merely encourages students to think—erroneously—that one has the property of dominating, the other of receding.

(2) Dominance is also widely misconstrued as the norm. Most introductory textbooks present dominance as the core of “Mendelian” genetics. Codominance and incomplete dominance, meanwhile, are exceptions to some basic rule. One might well infer—incorrectly—that dominance is most frequent. Not so. As early as 1907 Hurst noted that incomplete dominance is twice as frequent as complete dominance. Informed breeders and geneticists knew that dichotomous characters, like Mendel’s tall/dwarf or green/yellow, are relatively rare. A recent estimate (Rodgers 1991) indicates that fewer than one-third of human clinical genetic conditions fit the dominant-recessive pattern. Consider, too, the lack of substantive examples of dominant traits in humans. Textbooks often appeal to such trivial traits as “attached earlobe,” “hitchhiker’s thumb,” “widow’s peak,” “tongue-curling” and “PTC-tasting.” Nearly all significant cases have more complex stories. Dominance is a special case, not the norm. When presented as a basic model, it is misleading. We need to view “non-Mendelian” inheritance as primary, and place Mendelian dominance in the background.

(3) Finally, the concept of dominance has been misplaced. Dominance is not about the transmission of genetic material, but about phenotypic expression. It is not about inheritance, Mendelian or otherwise. It concerns development in diploid organisms. All alleles, whether the traits are dominant or recessive, are inherited the same way.

Because Mendel introduced dominance in his original paper, some assume that it is essential for understanding the basic principles of heredity. But early 20th-century geneticists discovered that dominance only muddied the waters. For example, William Bateson, Mendel’s strongest advocate in England, first interpreted Mendel’s rules of sorting and recombination as applying only to discontinuous traits (Olby 1987). Thus Bateson (like many students of genetics in the century since!) initially confused the hybrid appearance of a phenotype with the mixture of genetic

material itself. Bateson, of course, soon realized his error:

The degree of blending in the heterozygotes has nothing to do with the purity of the gametes. (1902, p. 152)

Curtis and Guthrie echoed in their 1933 textbook:

The course of inheritance for characteristics that do not exhibit dominance, therefore, is in no way different from that for characteristics in which dominance occurs. (1933, p. 185)

Even Nobel prize-winning geneticist Thomas Hunt Morgan refrained from including dominance as one of the basic principles in his synoptic *Theory of the Gene* in 1926. The early Mendelians learned that Mendel’s concept of dominance, ironically perhaps, was not fundamental to what we normally construe now as *Mendelian* genetics.

In summary, (1) dominance is not a special property that has a clear uniform explanation on the molecular level. (2) It is not most prevalent as a pattern of expression among pairs of alleles. (3) Most important, it is not requisite for understanding basic “Mendelian” inheritance (segregation and recombination of discrete genes). For these reasons, we should dissolve this conventional dichotomy between Mendelian and non-Mendelian genetics. We should embrace instead a simplified, more unified conceptual structure.

The Nine Lives of Gregor Mendel

But how did our current concepts become entrenched? The term ‘dominance’ originated, of course, in Gregor Mendel’s now classic 1865 paper. Those who read the original paper over a century later are often impressed with its clarity and modern style. That immediacy can be deceptive, though. One may assume, for instance, that Mendel’s conception of dominance was the same as ours now. It belongs to *Mendelian* genetics, after all. Interpreting Mendel, however, involves understanding his purpose and the context in which he wrote, often poorly represented in biology texts (Olby 1985, 1997; Monaghan & Corcos 1990; Hartl & Orel 1992; Corcos & Monaghan 1993). More important, interpreting *Mendelism* involves seeing how geneticists since Mendel have interpreted, and sometimes transformed, his work (Brannigan 1981; Olby 1979, 1985; Sapp 1990).

Curiously, perhaps, Mendel only used an adjective (30 mentions). He never used a corresponding noun or verb, or described a general principle or relationship between two characters as “dominance.” Rather, he merely labeled one character of a given pair based on the visible appearance of hybrid offspring. It was a linguistic tool, not a claim about inherent properties. He never said, for example, that a trait appears in hybrids *because* it is dominant, as others since have used the term (e.g. Campbell et al. 1999, p. 242;

Lewin 2000, p. 53). Also, whereas Mendel referred only to traits as dominant, now the term commonly applies to genes, or alleles (e.g. Campbell et al. 1999, pp. 242, G-7, G-18; Lewin 2000). The subtle shift of reference from phenotype to genotype facilitates conceiving “dominance” as causal—with profound effect (see below). Ironically, then, the current “Mendelian” concept of dominance was not Mendel’s at all.

Mendel himself seemed aware that his conclusions were limited. For example, he admitted that dominance was not the exclusive norm. Before introducing dominant traits he noted that:

with some of the more striking characters, those, for instance, which relate to the form and size of the leaves, the pubescence of the several parts, etc., the intermediate, indeed, is nearly always to be seen. (1866, §4)

Mendel noted other exceptions in peas: stem length (the hybrids were actually *longer*, §4), seed coat color (hybrids were more frequently spotted, §4), flowering time and peduncle length (§8). Mendel’s sequel work (1869) on hawkweed (*Hieracium*) certainly showed that one could not easily generalize his results on peas (*Pisum*). For Mendel, his law of hybrid development applied only to “those differentiating characters, which admit of easy and certain recognition” (1866, §8). Other characters followed another, different rule or law of hybrids. For Mendel, dominant traits were not universal.

Mendel’s successors, it seems, were more ambitious than Mendel. They recast his modest “law of hybridization” as a general scheme of heredity (Monaghan & Corcos 1990; Hartl & Orel 1992). Ultimately, the current concepts of gene, segregation and independent assortment generalized well. Dominance did not. Mendel’s work, of course, became a guide—a virtual touchstone—in the early decades of the 1900s. The pioneers of genetics thus treated dominance as a general rule. Still, they did not uniformly endorse it. Bateson, one of Mendel’s first and most ardent advocates, rejected any such principle or law. He later identified the “Andalusian” trait in fowl (blue-grey hybrids of black and white parents), which textbooks still use now to illustrate incomplete dominance. In promoting the legacy of Mendelism, textbooks conveyed a mixed message about dominance. For example, a 1921 text praised Mendel’s discovery, then added ironically, “of course breeding is not so simple as this, and some characteristics do blend or average in the hybrids” (Moon 1921, p. 543). A 1969 text presented “Mendel’s law of dominance,” thereby granting it stature equivalent to Mendel’s other laws. But the authors cited two classic exceptions, adding:

Since Mendel’s time, we have found that the law of dominance does not always hold. . . . It is clear that we cannot speak of a “law” of dominance even though


dominance occurs frequently (Kroeber, Wolff and Weaver, 1969, pp. 412–413).

Dominance was both a law and not a law. Such has been the ambivalence towards dominance.

Remarkably, despite the persistent criticisms, textbooks still preserve dominance at the core of *Mendelian* genetics. Other patterns (incomplete dominance, etc.) are secondary. That is, texts consistently introduce the eponymous Mendelism first, even when just a few pages later they “undo” dominance. Why? I contend that the conceptual organization reflects Mendel’s authority, even where we know it misleads students. The problem is thus embedded in our very respect for Mendel. Historically, geneticists and teachers have seemed unable to separate dominance from the image of Mendel as a great scientist. Biologists have revered Mendel, and hence also the version of genetics that now bears his name.

The aura of Mendel’s authority is vividly portrayed by Jan Sapp in his “Nine Lives of Gregor Mendel” (1990, available on-line at MendelWeb). Sapp notes how biologists and historians present Mendel as supporting nine different, sometimes contradictory, claims. Despite their disagreement, however, all the authors appeal to Mendel to bolster their own claims. Mendel’s verdict matters. Indeed, their goal of securing Mendel’s “voice” seems to explain their contrary interpretations of his work. Hence, for Sapp, one should look for bias behind any “Mendelian” label.

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We should assess the structure of Mendel's authority. Similarly, the image on page 632—like Andy Warhol's deliberate re-renderings of cultural images—invites us to rethink Mendel as an icon in biology.

Textbooks, too, have their biases in how they interpret Mendel. While history is generally absent from most biology texts, few fail to mention Mendel. They portray him as an exemplary scientist, with implicit morals about the nature of science. For example, he worked alone in an Austrian monastery: scientists modestly seek the truth; they do not pursue fame or wealth. Mendel used peas; scientists choose "the right organism for the job." He counted his peas: scientists are quantitative. He counted his peas for many generations over many years: scientists are patient. He counted thousands and thousands of peas: scientists are hard-working. After all this, Mendel was neglected by his peers, who failed to appreciate the significance of his work, but was later and justly "rediscovered": ultimately, scientific truth triumphs over social prejudice. Above all, Mendel was right: scientists do not err. That is, textbooks portray Mendel as "an ideal type of scientist wrapped in monastic and vocational virtues" (Sapp 1990). Mendel is mythic in proportion. The net result is: *any* concept that bears Mendel's name is virtually sacrosanct. Texts have thus helped perpetuate a view that all things Mendelian (whether Mendel's or not!) are immune to criticism. Because dominance was part of Mendel's original scheme and, at the same time, we revere Mendel, we continue to include dominance with basic genetics. Dominance has become entrenched in the romantic lore of Mendel.

One Potent Metaphor

Some may suggest that the concept of dominance (or the term) may not be ideal—and many people misuse it—but it is harmless enough. I contend, however, that the language itself matters. The *connotations* and *implicit meanings* of dominance shape thinking (Lakoff & Johnson 1980). As noted above, they engender the worst conceptions students develop. Teachers need to appreciate how the metaphor of power in dominance affects other concepts, as well, in biology and culture alike.

Even biologists have misinterpreted dominance. The nontechnical meaning has contributed to the shift from dominance as an effect to dominance as a property of alleles that governs gene expression. That is, biologists have *reified* dominance in accord with its nonbiological meanings. As a result, many evolutionary biologists—and many geneticists, too—have viewed dominance as a material property that can evolve. They believed, for example, that a "fit" recessive mutant would eventually become dominant through natural selection (or some other mechanism).

Mathematician Ronald A. Fisher (1928), the chief architect of population genetics, introduced the notion of heritable dominance "modifiers" to deal with the problem of deleterious mutants. Sewall Wright (1969, p. 69), co-founder of the Modern Synthesis, later discussed these at more length. Were the modifiers themselves dominant or recessive? Bernard Kettlewell (1973), of peppered moth fame, like his colleagues in ecological genetics, assumed that selectively advantageous traits would become dominant (e.g. Ford 1964, pp. 87–89, 120–121, 127, 269–71). And so they were puzzled why, in natural populations of moths from nonpolluted environments, melanistic forms were nevertheless dominant. When one properly understands dominance at a molecular level, however, one realizes how all these biologists made unsubstantiated assumptions. I believe these renowned scientists responded just as our students do to the language of dominance.

The effects of the dominance metaphor are even more profound for nonbiologists. Nonscientists' perceptions of scientific concepts become models of "how the world is." People adapt their behavior and structure society accordingly. The Mendelian model reinforces cultural tendencies to interpret social issues in dualistic terms. It allows concepts of either-or, winner-take-all competition to seem "natural." Only one option is expressed. That is, equal voice or cooperation are "unnatural." This view of dominance helps shape views of society, from Congressional politics and international relations to marriages and school sports. Compromise, sharing of authority, or creative options rarely emerge. When one teaches codominance or multiple alleles as "non-Mendelian," for example, one makes unintended ideological claims about what is standard and what is not. Teachers should recognize that the concept of dominance carries with it implicit cultural overtones—and ensure that science does not participate in even appearing to justify such cultural biases.

The metaphor of dominance is potent. It fosters many misconceptions. For example, if one conceives dominant alleles as having more "force," one infers that they are inherited more often (by a margin of three-to-one, usually!). Accordingly, dominant traits fill the population. To reconcile this with natural selection, adaptive traits must be (or become) dominant. Mutant ("abnormal") traits, likewise, must be inherently recessive. All these naive conceptions mutually reinforce one another—and all trace their origin to the dominance metaphor. Of course, no professional geneticist believes these things. But a teacher's concern is how the public, not some isolated community of experts, understands science. These conceptions cascade into and misinform public discussions involving genetics. The case of biologists misconstruing dominance illustrates the strength of

the metaphor. If science teachers are jointly responsible for developing an informed public, then the challenge is great indeed. We thus need a *simple* model of genetics that can inform everyone, while misleading as few as possible. This is why we urgently need to mend Mendelism.

Three Strategies

Given the scope and depth of the problem of dominance, a solution is surprisingly easy. While one might *correct* misconceptions through further instruction, we can *avoid* them altogether. Here are three simple remedies:

(1) **First, teach absence of dominance as the initial norm.** The most difficult concept for students is generally segregation (also true historically). In beginner's terms, genes are *paired* in humans. Each parent contributes *only one* of each pair. These are distributed equally, but separately in the gametes. The separation is difficult to observe with dominance because the recessive trait is not plainly visible in heterozygotes. One can only infer its presence indirectly. The behavior of the genes is more transparent, however, when each genotype has its own, clearly corresponding phenotype—namely, in cases where there is no dominance. So, I suggest, *begin with blood type*. Or, better yet, *begin with pink flowers* (perhaps the classic case of four o'clocks). According to naive notions of “blending inheritance,” pink parents can only produce pink offspring. The emergence of true-breeding red and white traits from self-crossing is counterintuitive (a discrepant event). It shows vividly that the hereditary material was never fully blended at all. Pink still reflects a mixture of half red, half white, but of discrete units. The pigment genes sorted themselves out and then recombined. This was Bateson's crucial epiphany.

With this basic model, one can easily address several popular misleading expressions. For example, it becomes harder to say that traits mystically “skip a generation.” Rather, they pass *through* generations. One can also reveal the false assumptions in such remarks as, “oh, you have your mother's eyes!” or “you get that chin from your father's side of the family!” These imply that for any trait an individual has only one active gene from one parent only. The corollary is that genes from the parents “compete.” A model without dominance underscores that genes from both parents are present and that phenotypes are, fundamentally, two overlapping traits.

(2) **Second, when discussing basic Mendelian models, adopt the language and notation currently used for blood types.** By eliminating aspects of phenotypic expression, the language of multiple alleles with no dominance helps students focus on alleles and how they segregate and recombine. Moreover,

one need not change the language to accommodate additional alleles, however many. Thus, with no extra step we prepare students to deal with the great diversity within gene pools [for instance, OMIM (1997) now documents 370 alleles for beta hemoglobin].

This way of speaking reflects an assumption that each allele is expressed. It underscores that phenotype is based on two alleles, the whole genotype. Accordingly, we would adopt an image of phenotype (in diploid organisms) as *compound*. That is, two alleles yield two “traits.” Sometimes, the two traits may be the same. When different, one trait may sometimes “mask” its homolog. Two distinct genotypes may thereby appear ostensibly the same, as in Mendel's dominant-recessive pairs in peas. One need only characterize each trait carefully. Certain recessive traits may appropriately be described as *null* or *truncated* (but as “traits” nonetheless). That is, in context the trait may be “blank” or nonfunctional, *even when fully expressed*. In general, one may ask whether a given trait is *manifest* at a certain phenotypic level, assuming that every trait is generally expressed at some level. One need never introduce the term ‘dominant’—or any equivalent—to convey the essential information.

This approach enjoys an additional benefit. Namely, when one refrains from casting dominance as a

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fundamental model, all its “exceptions” *dissolve*. That is, one no longer need teach incomplete dominance, codominance, multiple alleles, expressivity or penetrance. All follow the *basic* principles of segregation, recombination and independent (parallel) expression. We can thereby begin to unify Mendelian and non-Mendelian genetics in one scheme. Genetics becomes *simplified*.

(3) Third, teach phenotypic expression and its patterns as part of development (molecular genetics), not inheritance. That is, while dominance has become entrenched in Mendelian genetics, the phenomenon is not about the transmission of genetic material at all. It is about how genes in diploid organisms (once inherited) are expressed in pairs. Two minor adjustments are appropriate.

First, teachers should consistently highlight the complete pathway from gene to trait(s). Texts typically scatter the elements across various chapters. One chapter discusses molecular genetics, generally stopping at polypeptide production. Another discusses the function of proteins. Yet another links cellular functions to observable physiological traits. Teachers need simply to relate these chapters more explicitly. A few well-chosen examples can illustrate the link between genes, proteins and physiological traits. Garrod’s (1908) inborn errors of metabolism (alkaptonuria, albinism, phenylketonuria), of course, are classic cases. Ideally, one might not mention any genetic trait without also profiling its corresponding protein (or RNA). Explanation of pleiotropy fits here naturally, too.

Through such discussion, students learn the nature of a heritable “trait.” How do biologists reduce the organism into *functional*, selective units? Smooth and wrinkled seeds in peas, for example, are indicators of a trait that is really better characterized as endosperm starch/sugar content (Guilfoile 1997). Likewise, the human body is an assemblage of traits. Which are exemplary: “widow’s peak” and “attached earlobe,” or physiological and developmental functions associated with proteins, such as myosin, insulin and norepinephrine receptors?

Second, teachers need to integrate the concept of diploidy into discussion of gene expression. That is, texts typically portray the pathway of gene expression from DNA to phenotype along a single line. No parallel pathway intersects from any homologous allele. A fuller, more accurate account includes how the presence of two gene products may modify function or shape observable traits, or how they potentially interact. That is, how do compound phenotypes develop? Sometimes, a “double dose” (or triple dose) may be physiologically distinct from a single one (Rodgers 1991). A model that does not assume dominance as the norm—but is still thoroughly Mendelian—prepares students better to interpret all possible patterns of two alleles.

Conclusion

- how concepts can change through time
- how new results (here, molecular understanding of dominance) can revolutionize (not just add to) earlier knowledge
- how scientific authority is established, exercised and borrowed
- how scientists are human and can err, or can be biased by cognitive frameworks
- how scientists discover and correct error
- how science simplified for educational contexts can mislead
- how public understanding of science (whether accurate or not), can affect culture (e.g. Toumey 1996).

Acknowledgments

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