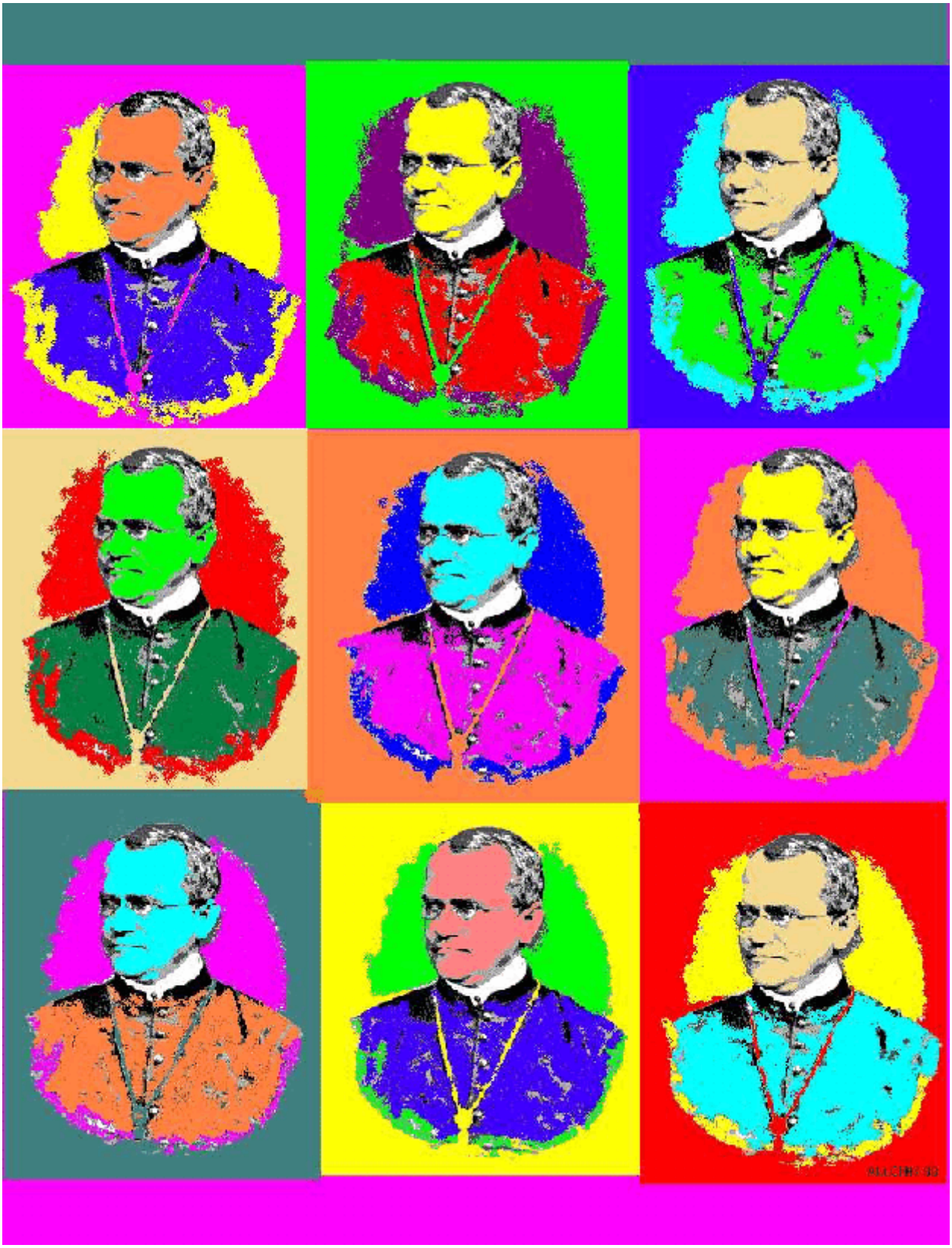

"The Nine Lives of Gregor Mendel," by Douglas Allchin.
This Andy Warholesque image was inspired by an essay by historian Jan Sapp. Sapp describes how Mendel is interpreted in nine different— and conflicting ways—by various biologists and historians. He details how personal perspectives and theoretical commitments shape how individuals interpret Mendel historically, while also borrowing from Mendel's prestige to bolster the authority of their own claims. Allchin elaborates on this theme by discussing the iconographic role of Mendel in biology education. Allchin profiles flaws in the concept of dominance and then suggests how reverence for Mendel contributed to the concept's historical entrenchment.

Mending Mendelism

The history of dominance can guide how to address misconceptions in genetics.

Douglas Allchin

Ever wonder why genetics is so challenging to teach? I note, in particular, certain widespread, perhaps notorious, misconceptions that frequently persist even after explicit instruction. Such notions as: some traits are inherently more likely to be inherited than others; dominant traits are more prevalent in the population; adaptive traits eventually become dominant through natural selection; mutations or "abnormal" genes are recessive; dominant alleles subdue or control recessive ones. (I trust these sound all too painfully familiar. So pervasive are these notions, that some textbooks explicitly warn against them; e.g., Rothwell 1983, pp. 15-16, Campbell 1996, pp. 248-249.) In every case the non-technical meaning of dominance—based on power, strength and value—shapes student thinking. Not long ago Michael Donovan (1997), past SCST President, noted this infelicitous language, but he despaired of ever fixing it. I am less sanguine about the prospects. Here I explore the problem of dominance as a concept, trace its persistence to the history of our mythic images of Mendel (who originated the term), then profile how we can mend the flaws. As a bonus, the case provides an excellent lesson in the history and nature of science.

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. What is the general mechanism for dominance in molecular or cellular terms? Most people are surprised to learn that there is none. This reveals one of three major conceptions needing realignment (at least, among non-geneticists).

Textbooks rarely address this matter. In the virtual silence, 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 additional protein or messenger molecule mediates the behavior of corresponding genes on separate chromosomes. Homologous alleles do not interact directly. Rather, they are expressed independently. One errs if one views dominance as some property distinct from two genes merely acting in parallel.

Others conceive dominance and recessiveness as the presence or absence of a trait, protein or gene product (e.g., Lewin et al 1997, p. 62). 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 (e.g., 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's [1997]

informative account in *ABT* of two 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 characterized as the absence of hemoglobin or of 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 alternate protein actively disrupts the assembly of the subunits into polymers, even when the normally functioning protein is present). The presence-absence model implies, incongruously, that the healthy condition (here, recessive) is no more than the "absence" of the disease. As these cases demonstrate, not all recessive traits are non-functional. Rather, a recessive phenotype is an *alternative* phenotype. It corresponds to a variant protein. 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 (mis)conception: 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. It is one among many such patterns. 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. Students use our language to guide their thinking.

(2) Dominance is also widely (mis)construed as the norm. Most introductory textbooks present dominance as constitutive of "Mendelian" genetics. Codominance and incomplete dominance, meanwhile, are "*non-Mendelian.*" That is, they are exceptions to some basic rule. One might well infer—incorrectly—that dominance is most frequent. Not so. As early as 1907 Hurst observed that incomplete dominance is twice as frequent as complete dominance (Darden 1991, p. 68). Informed breeders and geneticists knew that characters like Mendel's tall/dwarf or green/yellow, which take only two forms, are relatively rare. Exceptions to dominance were widely cited as a reason for rejecting Mendelism outright. A more recent estimate (Rodgers 1991, p. 3) likewise indicates that fewer than one-third of human clinical genetic conditions follow the dominant-recessive rule. Here is a different kind of "Mendelian ratio," although no deeply systematic study seems to document it. An indirect and informal measure of the prevalence of dominance, though, might be the textbook examples of dominant traits in humans. To illustrate Mendelian inheritance, they often appeal to "attached earlobe," "hitchhiker's thumb," "widow's peak," "tongue-curling," "PTC-tasting." Why so much reliance on such trivial traits? Nearly all the interesting or significant cases have more complex stories. Indeed, Online Mendelian Inheritance in Man (OMIM™ 1997), the major reference for human genetics discontinued

classifying traits as dominant and recessive in 1994. Dominance is a special case, not the norm. When presented as a basic model, it is misleading. One may profit, therefore, by considering the inverse gestalt: namely, viewing "non-Mendelian" inheritance as primary and placing Mendelian dominance in the background.

Many textbooks suggest that Mendel's choice of dichotomous traits (whether deliberate or fortuitious) helped him decipher the patterns of inheritance. Clarity from simplicity, so the motto goes. One tends to imagine, therefore, that focusing on such dominant/recessive pairs will likewise help our students appreciate segregation and recombination. I believe we should reassess this assumption. Indeed, I suspect that highlighting dominant/recessive character pairs merely promotes students viewing certain traits has having more "force" in inheritance—precisely the opposite of Mendel's insights. Students tend to tally visible phenotypes, not invisible genes. They observe the famous 3:1 ratio and infer that the dominant trait is, literally, dominating. Dominant traits obscure the underlying genetics, confounding interpretation. For example, an organism showing the dominant trait can be either homozygous or heterozygous. It has a "double signification," as Mendel put it (1866, §5). Students cannot see segregation directly. The cryptic relationship between phenotype and genotype adds an extra layer of complexity. The key to Mendel's insight was focusing on a unit trait, not a dominant/recessive character pair.

Contrary to common assumptions, then, dominance is not most frequent. Nor is it simplest. It is not most elementary. Students learning genetics deserve a better introductory model.

(3) Finally, the concept of dominance has been *misplaced*. Ultimately, dominance is not about rules for transmitting genetic material, but about phenotypic expression. That is, it is not about inheritance, Mendelian or otherwise. It is about development in diploid organisms. All alleles, whether the traits are dominant or recessive, are inherited from one generation to the next in precisely the same manner. Where, therefore, should textbooks place dominance?

Since Mendel introduced dominance in his original paper, one may assume perhaps that it must be integral to understanding the basic principles of heredity. But early in the 20th century, geneticists soon discovered that dominance only muddied the waters. William Bateson, Mendel's strongest advocate in England, for example, was himself confused at the outset. He interpreted Mendel's rules of sorting and recombination to apply only to discontinuous traits (Olby 1987, pp. 414-417). Thus Bateson (like so many students of genetics in the century since!) initially conflated the hybrid appearance of a phenotype with the irrevocable mixture of genetic material. Bateson, of course, soon realized his error, and announced his discovery:

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

In other words, Mendel's rules of inheritance concerned the integrity of the genetic material, regardless of whether the phenotype appeared "pure" (as in his dominant-recessive pairs). Curtis and Guthrie expressed the conclusion succinctly 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 (Darden 1991, p. 72). The early Mendelians learned that Mendel's concept of dominance, ironically perhaps, was not fundamental to what we normally construe now as *Mendelian* genetics (more on this apparent paradox below).

In summary, (1) dominance is not a special property that has a clear uniform explanation on the molecular or cellular level. Indeed, it is not a property at all. It is, at best, a shorthand descriptive label. (2) It is not most prevalent as a pattern of expression among pairs of alleles. As a model, it is thus grossly misleading. (3) Most important, it is not requisite for understanding basic "Mendelian" inheritance (segregation and recombination of discrete genes). For these reasons, I think we benefit by gently excising dominance from the conceptual repertoire of basic genetics.

By erasing the concept of dominance, we also dissolve a conventional (false) dichotomy between Mendelian and non-Mendelian genetics. We embrace instead a simplified, more unified conceptual structure (see Strategies below). This gestalt switch may seem radical—subversive for some. One would be well advised, therefore, to also consider the history of the concept that has been with us for so long. Elsewise, perhaps, we abandon it at our peril.

The Nine Lives of Gregor Mendel

Today's term 'dominance' originated, of course, in Gregor Mendel's now classic 1865 paper on "Experiments on Plant Hybrids" (1866). Those who read the original paper over a century later are often impressed with its clarity and modern style. Even high school students today seem to understand it without much trouble. 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 and Corcos 1990, Hartl and Orel 1992, Corcos and Monaghan 1993). More importantly, interpreting *Mendelism* involves seeing how geneticists since Mendel have interpreted, and sometimes transformed, his work (Brannigan 1981, Olby 1979, 1985, Sapp 1990).

'Dominant' is an English translation of Mendel's *dominirend(e)*. The original German term meant at the time "coming to the fore" or "overweighing." The complementary traits were "latent," Mendel said; he called them '*recessive'* because “the characters thereby designated withdraw or entirely disappear” (1866, §4). Accordingly, a more precise (if indirect) current translation of Mendel's *dominirende* might be 'manifest'. That is, Mendel's concept had none of the connotations of power we now associate with the words 'dominant' and 'recessive' (see "Metaphor" below). The language has evolved.

Curiously, perhaps, Mendel only used an adjective (30 mentions). He never used a corresponding noun or verb form. That is, he never described a general principle or relationship between two characters as "dominance." Nor did he refer to one trait as "dominating" another. Rather, he merely denoted one character of a given pair based on the visible appearance of hybrid offspring. He *defined* a dominant trait operationally. It was a linguistic device, 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 1996, p. 241; Lewin et al 1997, p. 55). Also, whereas Mendel referred only to traits as dominant (never his 'elementes'), now the term commonly applies to genes, or alleles (e.g., Campbell 1996, pp. 241, G-7, G-18; Lewin et al 1997, pp. 54-55, 62). The subtle referential shift (from phenotype to genotype) has facilitated conceiving "dominance" as causal—with profound effect (see "Metaphor" 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 acknowledged that dominance was not the exclusive norm. Before introducing dominant traits he first 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)

Later he commented:

> as regards flowering time of the hybrids, . . . the time stands almost exactly between those of the seed and pollen parents. (§8)

Mendel noted other exceptions in stem length (the hybrids were actually *longer*, §4), seed coat color (hybrids were more frequently spotted, §4) and peduncle length (§8) in *Pisum*, and seed size, flower color and seed coat color in *Phaseolus* (§10). Mendel's sequel work (1869) on hawkweed (*Hieracium*) certainly showed that one could not easily generalize his results on peas (*Pisum*). For Mendel this meant that 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 were, in a sense, more ambitious than Mendel. What Mendel presented as only a modest "law of hybridization" (marked in his title, for instance), the Mendelians cast as a general scheme of heredity (Monaghan and Corcos 1990, Hartl and Orel 1992). Ultimately, the current concepts of gene, segregation and independent assortment generalized securely. 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 implicitly treated Mendel's dominance as a general rule. At the same time, they did not uniformly endorse it (Darden 1991, pp. 56-57). Bateson, one of Mendel's first and most ardent advocates, rejected any such principle or law:

> In the *Pisum* cases the heterozygote normally exhibits only one of the allelomorphs [alternative phenotypic forms] clearly, which is therefore called the dominant. It is, however, clear from what we know of cross-breeding that

> such exclusive exhibition of one allelomorph in its totality is by no means a universal phenomenon. Even in the pea it is not the case that the heterozygote always shows the dominant allelomorph as clearly and in the same intensity as the pure dominant . . . (Bateson 1902, p. 129)

Bateson later identified the "Andalusian" trait in fowl (blue-grey hybrids of black and white parents), which textbooks later often used to illustrate incomplete dominance. In carrying forward the legacy of Mendelism, textbooks equivocated on the status of dominance as a universal "law" or basic model. For example, a 1921 text praised Mendel's discovery of dominance, 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 1933 zoology text, too, followed its description of dominance with a cautionary note: "dominance and recessiveness do not, however, characterize all cases of inheritance" (Curtis and Gurthrie 1933, p. 184). 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. In today's textbook conventions, inheritance is both Mendelian and non-Mendelian. Such has been the ambivalence towards dominance.

Remarkably, despite the persistent criticisms, textbooks still preserve dominance at the core of genetics. Dominance remains central and primary—constitutive of *Mendelian* genetics. Other patterns (incomplete dominance, codominance, expressivity and penetrance) are peripheral and secondary (*non*-Mendelian). That is, texts consistently introduce the eponymous Mendelism first, even when just a few pages later they "undo" dominance. Why? Why have texts not embraced a simplified, more basic model of genetics at the outset? I contend that the conceptual organization shows the authority of Mendel's original formulation (even where we know it is misleading). The problem is thus embedded in our very respect for Mendel himself. Historically, geneticists and teachers have seemed unable (or unwilling) to disengage 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 essay, "The Nine Lives of Gregor Mendel" (1990, available online at MendelWeb). Sapp notes how biologists and historians present Mendel as supporting nine different, sometimes contradictory claims. Thus, for some, Mendel rejected Darwin or even tried to disprove his theory, while for others his results were throughly Darwinian. For yet others, Mendel set questions of evolution wholly aside. One finds that Mendel falsified his data and also that he did not falsify his data. Mendel was the founder of modern genetics at the same time that he was no Mendelian (that is, he neither intended to describe a general model, nor stated the principles we now call Mendelian). Despite their disagreement, however, all the authors appeal to Mendel as a decisive authority to bolster their own claims. Mendel's

verdict matters. Indeed, their goal of securing Mendel's "voice" seems to explain why their interpretations of his work are contrary. Hence, for Sapp, one should look for bias behind any "Mendelian" label and analyze the architecture of Mendel's authority. Similarly, this month's *ABT* cover image—like Andy Warhol's paintings, in its deliberate re-rendering of an iconic image—invites us to rethink Mendel as an icon in biology. We should appreciate how the very image of Mendel can shape our thinking.

Textbooks, too, have their biases in how they interpret Mendel. While history is generally absent from most biology texts, few fail to mention Mendel. Most profile his work with garden peas. 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 ambitiously 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 unfairly 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 secure from criticism.

Historically, then, dominance began as a modest descriptive label for certain trait pairs (Mendel's 1865 paper). It has since become transformed into a property of alleles, frequently deemed causal, that anchors a general model of genetics (current usage). Textbooks consistently found this model wanting. Yet dominance persisted at the core of genetics, helping to establish Mendelian and non-Mendelian genetics as separate categories. Because dominance was part of Mendel's original scheme and, at the same time, we honor Mendel almost religiously, we continue to include dominance with basic genetics. Dominance has become entrenched in the romantic lore of Mendel.

One Potent Metaphor

Some suggest that overhauling our genetics curricula is hardly worth the effort. The concept of dominance, or the term, may not be ideal—and many people misuse it—they contend, but it is harmless enough (Fritz 1999, anonymous reviewer). I disagree. The language itself matters. The term has not been idle conceptually. The *connotations* and *implicit meanings* of dominance shape thinking (Lakoff and Johnson 1980). As noted above, they engender the worst conceptions students develop. We show disrespect for students if we continue to prime these misconceptions. One needs to appreciate how the metaphor of power in dominance affects other concepts, as well, in biology and culture alike.

One might well imagine that biologists would not misinterpret dominance. However,

the non-technical meaning of dominance seems to have contributed to the shift from dominance as an effect, or artifact, to dominance as a property of alleles that governs genic expression. That is, biologists have *reified* dominance in accord with its non-biological 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 dominance modifiers, heritable factors that affect the "dominance" interaction of two alleles. He postulated such modifiers in order to reconcile his theoretical equations with observed rates of evolution in the field and to deal with the problem of deleterious mutants. Sewall Wright (1969, Vol. 2, p. 69), co-founder of the Modern Synthesis, later discussed these at more length. Were the modifers themselves dominant or recessive? (Wright did not address the implicit problem of endless recursion.) Bernard Kettlewell (1973), of peppered moth fame, like his colleagues in ecological genetics, assumed as a matter of course 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 non-polluted environments, melanistic forms were nevertheless dominant. When one properly understands dominance at a molecular level, however, one realizes how all these biologists succumbed to unsubstantiated assumptions. We should pause and wonder what could have misled so many scientists of such renown. I believe they responded (just as students do) to the language of dominance. No, it is not "harmless" or idle.

The downstream effects of the dominance metaphor are even more profound for non-biologists. Scientific concepts of nature—or, rather, non-scientists' *perceptions of* such concepts—become models of "how the world is." People adapt their behavior and structure society accordingly. So, for example, many people nowadays see Social Darwinism as the state of nature described by evolutionary biology. They do not conceive it as an ideology outside science which humans either adopt or reject. They *assume* that science says we live in a "society red in tooth and claw" and then live with that expectation (even if they fight against it). The Mendelian model, by portraying nature in simple dualities, reinforces cultural tendencies to interpret social issues in bipolar terms. The notion that dominance is the norm gives a naturalness to casting such issues in either-or, winner-take-all terms. Only one option is expressed. All others are totally eclipsed. That is, equal voice or synergy are "unnatural" alternatives. This view of dominance helps shape or reinforce our view of society, from Congressional politics and international relations to marriages and school sports. Compromise, sharing of authority or creative third options rarely emerge as default alternatives. When one teaches codominance or multiple alleles as "non-Mendelian," for example, one makes unintended normative claims about what is standard, what is not. We should recognize that the concept of dominance carries with it implicit cultural overtones—and endeavor to ensure that science does not participate in promoting or even appearing to justify such cultural biases.

The metaphor of dominance is potent. It precipitates many misconceptions. For example, if one perceives that dominant alleles have more "force," then one infers that they

are inherited more often (by a margin of three-to-one, usually!). Accordingly, dominant traits fill the population (are *predominant*, the language seems to confirm). 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 my concern is not some isolated community of experts. Rather, how does the public understand science? These conceptions cascade into and misinform public discussions involving genetics, now rising in frequency and importance. The case of biologists misconstruing dominance is intended not as a direct criticism, but as an illustration of the strength of the dominance metaphor. If science teachers are jointly responsible for developing an informed public, then the challenge is great indeed. Our teaching must aim to reach all students, even the weakest ones. We thus need a *simple* model of genetics that informs, while misleading as little as possible. That is why we urgently need to mend Mendelism.

Three Strategies

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

First, *teach absence of dominance* ("codominance") *first, as the norm*. In my experience (and others can speak to this), the most difficult concept for students is segregation (also true historically; see Darden 1991, pp. 57-60). In beginner's terms, each parent contributes only *half* its genes. Genes are *paired* in humans; any individual offspring receives *only one* of each pair. These traits are distributed equally, but separately among gametes. The separation is difficult to observe with dominant traits 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 about Mendel. Hence, a case such as this helps isolate and reveal the critical first concept of Mendelism, segregation.

With this basic model, one can easily address several popular misleading expressions. For example, because one can follow the genes through the phenotypes, it becomes harder to maintain that traits mystically "skip a generation." Rather, they pass

through generations. One can also reveal the false assumptions in such statements as, "oh, you have your mother's eyes!" or, "you get that chin from your father's side of the family!" These remarks imply that at some level, for any trait, an individual has only one gene and that it comes from one parent only. The corollary is that genes from the parents "compete" (whether in inheritance or expression). A model without dominance underscores that genes from both parents are present and that phenotypes are, fundamentally, two overlapping traits (see Strategies #2 and #3, below).

This means, too, of course, that Mendel's landmark work on peas may no longer be ideal for introducing heredity. Given the confusions that it generated historically among *professional* biologists, however, one should not be surprised that students might be equally challenged in interpreting his results, at least initially. Mendel's studies may have an important pedagogical role elsewhere instead.

Second, when discussing basic Mendelian models, *adopt the language and notation currently used for blood types*: that is, multiple alleles with no dominance ("codominance"). By neutralizing elements of phenotypic expression, this language allows students to 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 diveristy within gene pools, now becoming much more evident (for instance, OMIM [1997] now documents 370 alleles for beta hemoglobin).

This way of speaking reflects a null hypothesis that each allele is expressed. It underscores that phenotype is based on two alleles, the whole genotype. It advances no assumptions about individual alleles. 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. Thus, dominant-recessive pairs, including Mendel's 1865 work on peas, fit comfortably into this linguistic framework. 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 non-functional, *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 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*.

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 elucidates how cellular functions relate to observable physiological traits. Teachers need simply to link 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 excellent 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 begin to learn the nature of a heritable "trait," and ultimately how biologists reduce the organism into *functional*, selective units. Smooth and wrinkled seeds in peas, for example, are pleiotropic indicators of a trait that is better characterized as endosperm starch/sugar content (Guilfoile 1997, also noted in 1903 by Gregory). Likewise, how do we want to portray the human body? It is an assemblage of traits. Which are exemplary: "widow's peak" and "attached earlobe," or physiological and developmental functions associated with proteins, such as dystrophin, insulin receptors and neurotransmitter-degrading enzymes?

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, with no parallel or intersecting pathway 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 (e.g., Wright 1968, Vol 1, pp. 63-70). That is, how do compound phenotypes develop? For example, the story of osteogenesis imperfecta (above, "Problematic Conceptions" #1) involves knowing that the disease protein can interfere with function even when the functioning protein is present. 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 dual alleles.

Conclusion

The perceptive reader may have noticed and appreciated the 9:3:3:1 theme that inspired my section titles. The intent, here, is not to discredit Mendel. Rather, I want to mend a concept that we attribute to Mendel (but which, in fact, he never held). Indeed, I think our respect for Mendel demands it.

At the same time, I hope to sharpen the acumen of teachers in considering the

histories of science that enter the science classroom. One tendency, epitomized in textbook renderings of Mendel, is to romanticize or idolize great scientists—often to the point of distorting how science really happens. We err, however, if we only conceive scientists as either heroes or fools (Gould 1977). Our goal should be to convey science honestly—noting its flaws and limitations, as well as celebrating its achievements. Our histories, therefore, need supportive evidence as much as any claim in science, especially when we use them to portray science to students.

The history of dominance is certainly in part a cautionary tale. But it also offers compelling lessons on the history and nature of science for students (see also Hagen, Allchin and Singer 1996). These include, at least (as detailed and referenced above):

- how concepts can change through time
- how scientists build on, and sometimes transform, earlier scientists' work
- how new results (here, molecular understanding of dominance) can revolutionize (not just add to) earlier knowledge (Kuhn 1970)
- how scientists communicate (including problems of language and history)
- 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 is presented in educational contexts
- how the public understands science (whether accurately or not), and how this can affect culture (e.g., Toumey 1996).

Viewed appropriately, the Warholesque image of "The Nine Lives of Gregor Mendel" (cover) symbolizes all these potential lessons. It is a tribute and critique at the same time. The mindful teacher, I trust, appreciates both views—and knows how to balance them well.

Acknowledgements. Many, many thanks to Louise Paquin (for a good ear and professional counsel) and to Patti Soderberg, Bob Olby, and Rachel Ankeny for their support.

References

Bateson, W. (1902). The facts of heredity in the light of Mendel's discovery, *Reports to the Evolution Committee of the Royal Society, London 1,* 125-160.

Blumberg, R. (ed.). (1997). MendelWeb. URL: www.netspace.org/MendelWeb.

Brannigan, A. (1981). The law valid for *Pisum* and the reification of Mendel. In *The Social Basis of Scientific Discoveries*, Cambridge Universtiy Press, Cambridge, pp. 89-119.

Campbell, N.A. (1996). *Biology*, 4th ed. Menlo Park, CA: Benjamin Cummings.

Corcos, A. and Monaghan, F.V. (1993). *Gregor Mendel's Experiments on Plant Hybrids: A Guided Study*. New Brunswick: Rutgers University Press.

Curtis, W. C. and Guthrie, M. J. (1933). *Textbook of General Zoology*, 2d. ed., New York:

John Wiley & Sons.
Darden, L. (1991). *Theory Change in Science: Strategies from Mendelian Genetics*, Oxford: Oxford University Press.
Donovan, M. P. (1997). The vocabulary of biology and the problem of semantics, *Journal of College Science Teaching 26*, 381-382.
Fisher, R.A. (1928). The possible modification of the response of the wild-type to recurrent mutations. *American Naturalist 62*, 115-126.
Ford, E.B. (1964). *Ecological Genetics*. London: Methuen.
Fritz, G. (1999). Allchin's many mistakes. *BioQuest Notes 10*(1), 9-12.
Garrod, A. (1908). *Inborn Errors of Metabolism*. Oxford: Oxford University Press.
Gould, S.J. (1977). On heroes and fools in science. In *Ever Since Darwin*, New York: W.W. Norton.
Guilfoile, P. (1997). Wrinkled peas and white-eyed fruit flies: The molecular basis of two classical genetic traits, *American Biology Teacher 59*,92-95.
Hagen, J., Allchin, D., & Singer, F. (1996). *Doing Biology*. Glenview, IL: Harper Collins.
Hartl, D. L. and Orel, V. (1992). 'What did Gregor Mendel think he discovered?' *Genetics 131*, 245-253. Available at Blumberg (1997).
Kettlewell, H.B.D. (1973). *The Evolution of Melanism: The Study of Recurring Necessity; with Special Reference to Industrial Melanism in the* Lepidoptera*.* Oxford: Clarendon Press.
Kroeber, E., Wolff, W. H. and Weaver, R. L. (1969). *Biology*, 2d ed. Lexington, MA: D.C. Heath.
Kuhn, T.S. (1970). *The Structure of Scientific Revolutions*, 2d ed. Chicago: University of Chicago Press.
Lakoff, G. and Johnson, M. (1980). *Metaphors We Live By*. Chicago: University of Chicago Press.
Lewin, B., Siliciano, P. & Klotz, M. (1997). *Genes VI*. New York: Oxford University Press.
Mendel, G. (1866). Versuche über Pflanzenhybriden [Experiments on plant hybrids], reprinted in J. Kříženecký (ed.), *Fundamenta Genetica*, Oosterhout: Anthropological Publications; Brno: Moravian Museum; and Prague: Czech Academy of Sciences (1965), pp. 57-92. English translation reprinted in C. Stern and E. Sherwood (eds.)*, The Origin of Genetics: A Mendel Source Book*, San Francisco: W.H. Freeman (1966), pp. 1-48. Translation by Druery (1901) available at MendelWeb, URL: www.netspace.org/MendelWeb.
Monaghan, F.V. and Corcos, A. (1990). The real objective of Mendel's paper. *Biology and Philosophy 5*, 267-292.
Moon, T. J. (1921). *Biology for Beginners*. New York: Henry Holt.
Olby, R.C. (1979). Mendel no Mendelian? *Hist. Sci. 17*, 53-72.
Olby, R.C. (1987). William Bateson's introduction of Mendelism to England: a reassessment, *British Journal for the History of Science 20*, 399-420.
Olby, R.C. (1985). *The Origins of Mendelism*, 2d ed. Chicago: University of Chicago

Press.
Olby, R.C. (1997). Mendel, Mendelism and genetics. MendelWeb, URL: www.netspace.org/MendelWeb/MWolby.intro.html.
OMIM™ (2000). Online Mendelian Inheritance in Man. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine (Bethesda, MD), URL: www.ncbi.nlm.nih.gov/omim/
Rodgers, J. (1991). Mechanisms Mendel never knew, *Mosaic 22*(3), 2-11.
Rothwell, N.V. (1983). *Understanding Genetics*, 3rd ed. New York: Oxford University Press.
Sapp, J. (1990). The nine lives of Gregor Mendel , in H. E. Le Grand (ed.), *Experimental Inquiries*, Kluwer Academic, Dordrecht, pp. 137-166. Also available at Blumberg (1997).
Toumey, C.P. (1996). *Conjuring Science*. New Brunswick NJ: Rutgers University Press.
Wright, S. (1968-69). *Evolution and the Genetics of Populations, Vols. 1-2*. Chicago: University of Chicago Press.

This article originally appeared in American Biology Teacher ***62****: 632-639*.