Putting Mendel in His Place: How Curriculum Reform in Genetics and Counterfactual History of Science Can Work Together

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1 Introduction

Classical or transmission genetics is the study of patterns of inheritance: how traits in an organism – and, either implicitly or explicitly, the genes that give rise to those traits – are passed from one generation to the next. For most undergraduate students, their first (and sometimes only) experience of the study of inheritance is bound up with Mendelian principles. Anyone who has studied genetics at high school or first year at university will be very familiar with Mendel's peas: varieties of the garden pea with clear-cut, dichotomous traits such as smooth seeds versus wrinkled, yellow seeds versus green, or tall habit of growth versus short. Explanations of the way these traits are transmitted between generations, especially in the pedagogical context, have traditionally been framed around the concepts of *dominance* and *recessiveness*. For example, when a smooth-seeded plant is crossed with a wrinkled-seeded one, the offspring are all smooth-seeded. Smooth is therefore said to be *dominant* to its *recessive* partner, wrinkled. In the same way, yellow is dominant to green, and tall is dominant to short. Further, the 'gene for' the smooth trait is taken to be dominant to the 'gene for' the wrinkled trait, and so on.

This Mendelian approach in the teaching of genetics has persisted into the twenty-first century despite the increasing recognition in many disciplines – genetics, molecular biology and neuroscience amongst others – that, contrary to the Mendel's-peas picture, genes should not be regarded as the sole causes of organismic ('phenotypic') traits, but rather as elements in a complex network of factors involved in the development of an organism. Of course teaching methods and materials in the sciences always lag behind the sciences themselves, for well-known reasons. In the genetics case, moreover, the start-with-Mendel pedagogic strategy has its defenders

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even among those who appreciate that biology has moved on.[1] A Mendelian conceptual foundation, they will say, gives students precisely the basis they need in order to cope with the messiness of the rest of biology. To stick with Mendel's peas, in the defenders' eyes, is thus to embrace the venerable wisdom of the good teacher who knows that it is always best to start with something simple, and only gradually thereafter to introduce difficulty, complexity, complications, exceptions. Start simply, and your students will proceed with understanding and confidence. Do otherwise, and you risk overwhelming them, leaving them confused and demoralized.

But even the most useful of strategies can be problematic. To a growing number of critics, including geneticists, historians and educators (though these are by no means mutually exclusive groups), Mendel's peas no longer look fit for purpose as a starting point for organizing knowledge of inheritance, because the simplifications they bring with them – and which have made them so attractive to teachers of genetics for so long – may ultimately do more harm than good. The Mendelian concept of dominance in particular has come under suspicion, as generating a highly exaggerated but also deeply engrained sense of the determinative power of genes.[2] Such a stance toward genes can adversely affect students' ability to understand and do genetics in the age of genomics and epigenetics. Just as importantly, it can affect students' perceptions of genetics in wider contexts, including those that bear on important social issues (see Moore, this volume).

These critics do not, it should be emphasized, necessarily have Mendel himself in their sights. In his original papers, he used the term 'dominant' – or rather, its German counterpart – to describe the behaviour of a certain trait in a specific context, not as a defining quality of that trait (Falk 2001; Allchin 2005, p. 440). However, within the modern usage of 'Mendelian', dominance has come to be perceived as a fixed, unvarying quality of a gene variant (an 'allele'), where X is dominant to Y in any and all circumstances, and the 'heterozygote' (having X and Y) will always display the dominant phenotype, which overrides the recessive phenotype. As early as 1900, it was recognized that this view of dominance matches reality only in special cases, and fails utterly to account for phenotypic variability (see Allchin 2002, 2005). This recognition in turn underpinned the introduction, after 1900, of a number of variations on the concept of dominance, including:

- Partial or incomplete dominance, which results in offspring with a range of intermediate phenotypes in the heterozygote. The classic example is the Andalusian Blue fowl, with blue-grey plumage, which is the result of crossing a pure-breeding black with a pure-breeding white bird. The Andalusian Blue is not, itself, pure-breeding because it is inevitably heterozygous.

[1] See Skopek (2011) on the birth and early career of the start-with-Mendel strategy among writers of textbooks in genetics.

[2] Although we shall concentrate on dominance here, other aspects of traditional Mendelian pedagogy have also been criticized. As Kampourakis (2013) has shown, the presentation of the process of science is also flawed, promoting, as it does, a view of Mendel as a lone pioneer, rather than as the author of one contribution to the social activity that is science.

- Co-dominance, as seen in the ABO blood types system, where both parental phenotypes are expressed simultaneously
- Over-dominance, or heterosis, where the heterozygous phenotype is outside the parental range, and can provide a survival advantage with respect to certain characteristics, as seen in the increased resistance to malaria conferred by the sickle cell trait in the heterozygote

Such exceptions to Mendelian patterns of inheritance are collectively characterized as 'non-Mendelian inheritance', including, in addition to the above, pleiotropy (a single gene having effects on multiple traits), expressivity (the variation in the degree of expression of a given trait in individuals with a given genotype), penetrance (the proportion of individuals with a given genotype that express the expected phenotype), phenotypic plasticity (changes in the phenotype of an adult organism in response to its environment) and epigenetic factors (heritable changes in phenotype which do not involve alterations to the nuclear DNA sequence) (see Love, this volume; see Uller, this volume). Even so, this dichotomous categorization – into whatever is Mendelian and whatever is not – tends to reinforce the perception that straightforward Mendelian dominance and recessiveness is the usual or normal case. A similar situation holds with respect to two versus more-than-two alleles, where one gene with two alleles is presented as 'normal' and genes with multiple alleles as the exception.

A look at current university-level textbooks suggests that the critics' concerns are not groundless, though neither should the textbooks be caricatured. A typical example, Peter Russell's *iGenetics: A Mendelian Approach* – commonly recommended as reading for undergraduate courses – begins with the premise that "Mendel's work constituted the foundation of modern genetics" (Russell 2006, p. 2). By page 4, the student is introduced to the concepts of dominant and recessive traits. Although gene/environment interactions, and the idea that genes confer only the potential for the development of a certain trait (rather than determining developmental fate absolutely), are touched upon, they are no more than touched upon, briefly. Thus is the precedence of the Mendelian perspective established at the very beginning, at least among students inclined, in the familiar way, to cling to the most straightforward explanation, and to give less weight to complexities presented as side issues (an impression reinforced, with respect to gene-environment interactions, by their not being fully explained at this stage). In the standard way, the textbook goes on to use the language of a "gene for…" a particular characteristic, and of genes as "controlling"/"being responsible for" phenotypic properties. By such language, arguably, genes are directly connected to the adult phenotype, bypassing the complexities of development. Furthermore, 'dominant' is defined as "describing an allele or phenotype that is expressed in either the homozygous or heterozygous state" and 'recessive' as "an allele or phenotype that is expressed only in the homozygous state" (Russell 2006, p. 733 and p. 742) – a conflation of 'allele' and 'phenotype' that may well encourage students to accept the more readily that dominant alleles mask or overpower recessive ones.

There are attempts to present genetics in a different way. Russell himself also publishes a version of his textbook that is subtitled *A Molecular Approach* (Russell 2010), and that postpones bringing Mendelian genetics into the story until chapter 11. However, this represents merely a reordering of topics, not a different presentation of the subject. Once students travelling the molecular route emerge from the details of DNA, RNA and so on to consider how all of that affects the organism, they encounter exactly the same descriptions and definitions of dominance and recessiveness as do the students approaching the material via Mendel's peas.

In this chapter we wish to propose and explore a more far-reaching option, at once new and old, for reorganizing the genetics curriculum. It is new in that it is not represented in the current debate on that curriculum. But it is old in that it revives a concept of dominance – and a way of thinking about inheritance generally – from more than a century ago, before the Mendelian perspective took off and took over. They are to be found in the work of the very first critic of the Mendelian perspective, W. F. R. Weldon (1860–1906), in particular in an unpublished manuscript *Theory of Inheritance* (1904–1905) where Weldon expressed concerns about the dogmatic nature of Mendelism. In his attempt to achieve a better understanding of inheritance by combining the best of both Mendelism and 'biometry' (the statistical biological studies for which he was best known), Weldon adopted, as we shall see, a contextual interpretation of dominance of the sort that many people now seem to be looking for.

We shall proceed from here as follows. The next section offers a more in-depth review of present-day discussion of dominance and its discontents. In Sect. 3 we will describe the alternative conception of dominance proposed by Weldon in his *Theory of Inheritance*. Section 4 will then consider how Weldon's ideas – and ideas about the genetics that might have been had Weldon and his allies not lost their battle over Mendelism – can be useful now in devising a genetics curriculum suitable for the twenty-first century.

2 The Trouble with Tradition

Concerns about the difficulties of teaching genetics, including the role of misinterpretations of dominance, have been expressed for decades (see, for example, Stewart 1982 and Collins and Stewart 1989). This is not surprising, since the evolution of the concept has been highly complex, and scientific understandings of dominance have been subject to much debate (see Falk 2001 for a history of the concept). The pedagogic problems have been most clearly analysed by Douglas Allchin in a series of papers (see Allchin 2000, 2002, 2005) in which he emphasizes the power of language to influence thinking, and the way that an everyday understanding of dominance – suggesting both power and prevalence – can colour understanding of

genetics and genetic issues (see especially Allchin 2005). Common misconceptions based on everyday interpretations of the word include:[3]

- Dominant traits are 'stronger' than recessive ones
- Dominant traits are more likely to be inherited than recessive ones
- Dominant traits completely mask or overpower recessive ones
- Dominant mutations occur more frequently than recessive ones
- Dominant mutations are more likely to survive than recessive ones
- For any trait the 'wild-type' tends to be dominant whereas any mutations will be recessive

Even though more advanced genetics teaching modifies the concept of dominance, the very fact that these modifications are overtly framed as 'non-Mendelian' implies, as noted above, that they are exceptions to the fundamental rule, thus propping up the deterministic interpretation of dominance. Furthermore, since a substantial proportion of students will study only elementary genetics – a single introductory genetics module in a general biology degree, say – they will never encounter these exceptions, thus perpetuating the exaggerations surrounding the subject.

It is not exclusively in the discipline of genetics that the Mendelian concept of dominance is problematic. Consider, for example, how 'dominance' talk can produce misunderstandings about the relationship between genetics and evolution. In the evolutionary context, the commonplace interpretation of 'dominance' can lead students to assume that if the 'fittest' individuals survive, surely they must be the dominant ones. In a similar vein, if an allele for a trait confers increased fitness and thus becomes more prevalent in the population, the linguistic and cultural implications discussed above can lead to the inference that the selected allele must therefore be dominant to its counterpart, or even that its increased occurrence is caused by its dominance over the 'wild-type' allele. These misconceptions can lead to a reduced understanding of both genetics and evolution. We see here too how the forging of an educational link between Darwin and Mendel, whilst clearly attractive, can exacerbate the problem of misunderstandings of genetics and must therefore be handled carefully in a new curriculum (see Bizzo and El-Hani 2009 for a discussion of this problematic relationship in the context of high school biology teaching).

'Dominance' talk in other biological disciplines can also add to the confusion. For example, in behavioural ecology, dominance can imply a power relationship between individuals, whereby one exerts physical or social power over another to gain resources, and the dominated individuals are defeated, give way, or submit to dominant ones. Dominance in this context is not only in accord with our everyday usage but is also presented as an "implicit natural model" (Allchin 2005, p. 431), promoting competition and conflict. If students are studying behavioural ecology and genetics simultaneously, as may well be the case in many modular courses, it would not be surprising if their grasp of the genetic meaning of 'dominance' is somewhat

[3] Allchin (2002), p. 50, citing also Donovan (1997).

unsophisticated and deterministic. Allchin (2005, pp. 441–446) further argues that the reification of dominance as an essential quality has shaped biological practice as well as pedagogy throughout the twentieth century. The perception of dominance as "a heritable property" of a trait or allele, rather than a contextual epiphenomenon, leads readily into a perception of dominance (and by association, recessiveness) as pre-requisites of natural order. In fact, dominance in nature is a special case; the majority of traits do not show simple dominance (Allchin 2005, p. 441 and 433).

Allchin (2005, p. 435) also highlights the inadequacy of even more sophisticated models of dominance, such as the so-called linear model for sickle-cell anaemia. On conventional representations of its genetics, the disease is treated as the dominant phenotype, with its expression being a matter of degree. If you are homozygous for 'normal' hemoglobin, you will not have the disease; if you are homozygous for sickled hemoglobin, you will have the disease; and if you are heterozygous, you are somewhere in between, perhaps suffering some symptoms under certain circumstances. What such a scaling scheme fails to capture, however, is the equally legitimate sense in which the heterozygous phenotype – sickle trait, as it is known – is dominant, since, for well-understood reasons, it is the heterozygous condition that confers resistance to malarial infection.

Another major problem arising from the traditional method of teaching genetics is that of oversimplification. The simple patterns of inheritance shown in Mendel's peas – smooth vs. wrinkled, yellow vs. green, and so on – encourage the view that one gene controls one trait. As Lewis (2011) argues, however, such a view is contrary to the understanding which has emerged through more recent research in genomics, according to which single-gene characteristics are very rare. Instead of focussing on these uncommon single-gene traits and diseases in our teaching, and thereby potentially promoting a hard-line determinism, we need, say Lewis and other critics, to move towards a genomic approach which encompasses the interactions of the entire genome with both the internal and external environments, and which acknowledges the complex nature of the vast majority of traits (see also Burian and Kampourakis, this volume).

There are also concerns about the impact on public understanding of science (Allchin 2000, 2005, p. 430). Increasingly, people are required to make personal and political decisions that touch upon genetics – decisions about pre- or post-natal testing, or about social policy regarding the results – and it is essential that we should have a well-educated population to cope. The problem does not first manifest in higher education but is rooted much earlier. As Mills-Shaw et al. (2008) have shown, misconceptions about a range of genetic issues begin when education about genetics begins, in high school or earlier, including misunderstandings of patterns of inheritance, the genetic basis of disease and genetic determinism.[4] This is not surprising when we consider that genetic determinism is a "general phenomenon" in school textbooks, as recently argued by Gericke et al. (2012). In their study

[4]For more on the increasingly inadequate public understanding of genetic issues, see Condit et al. (1998), Smerecnik (2010), Condit (2011), and Lewis (2011).

of 38 high-school textbooks from six countries, these authors show that due, in large part, to oversimplification in the way that the nature of the gene is presented in these books, students are in effect encouraged to develop a very reductionist, deterministic view of genetics. Mills-Shaw and her co-workers highlight a further area of concern, noted too more recently by Lewis (2012, citing Nowgen 2011), which is that secondary-level teachers do not always feel that they have the necessary knowledge and expertise to teach this subject properly. The potential problems of inadequately educated teachers in this area are explored in an international study by Castéra and Clément (2012) who show that the level of teachers' training and understanding in genetics affects not only the grip of their students on genetic facts and concepts but the attitudes that the students acquire to gender, ethnicity and other socially sensitive topics.

Taken together, the above constitute compelling reasons to improve the presentation of inheritance in both school and university curricula. It is in everyone's interests for school teachers to feel better able to teach genetics (and genuinely to be so), for those students who do not move on to higher education to have a better grounding in important genetic issues, and for those students who do move on to be better prepared for the transition from school to university biology studies. The result would be a better-informed citizenry, more capable of making decisions involving genetics, and with a more subtle attitude to human differences, genetic or otherwise.

One way forward, proposed by Allchin (2005, p. 436) would be to abandon the terminology of 'dominance' altogether and to teach genetics without it. An early emphasis on what Allchin describes as the 'haplophenotype' – that is, consideration of the expression of each allele separately – would remove the need for the dominant/recessive dichotomy (see further discussion in Sect. 4). Other suggestions of how to alleviate the problems by devising new curricula include the development of an "inverted" curriculum, where complex traits are addressed first, before later moving on to the more simple patterns seen in dichotomous traits such as smooth and wrinkled, which are then presented as exceptional examples of discrete variation, rather than as the norm (Dougherty 2009). In this way, students are discouraged from relying on the (over)simplified situation seen in Mendelism as traditionally taught. This approach is not entirely new; J.B.S. Haldane and Julian Huxley, in their 1927 *Animal Biology,* aimed at school boys, began their account of Mendelism with the example of the Blue Andalusian fowl, in which, as noted above, the hybrid of black and white strains shows an intermediate colour. Only after this did they briefly refer to the idea of dominance, to demonstrate that there are *some* instances where one allele can "mask the appearance" of the other in the first hybrid generation.

It is nevertheless striking how persistent old Mendeldian habits of thinking and talking have remained, even among those aiming to update the genetics curriculum. Guilfoile (1997), for example, provides an informative analysis of the molecular basis of the round/wrinkled trait in peas, presenting this as a classical example of a dominant/recessive trait, molecular understanding of which can help students to integrate classical and molecular genetics. His approach certainly contributes to resolving that particular problem. But it does so at the price of overstating the overlap between Mendelian and molecular descriptions of the phenotype. At the molecular level, it turns out, there are not two things, a gene 'for' roundness and a gene

'for' wrinkledness. There is, as far as DNA is concerned, mainly just one thing: DNA encoding an enzyme that coverts sugar into starch. Depending on the number of functional copies of that sort of DNA in a given pea plant, the seeds on that plant will have different quantities of the enzyme, hence different quantities of starch, hence – for reasons to do with the effects on water absorption – different seed shapes. Guilfoile draws no attention to it, but the molecular account he supplies should lead us to expect to find that real pea seeds are not either round or wrinkled, as per textbook Mendelism, but instead show every gradation, from extreme wrinkledness to full roundness. That expectation would no doubt only get stronger with supplementary attention to how other genes in the pea genome, ambient temperature and pressure, mineral content in the absorbed water, and so forth also affect seed shape. And indeed, there are many degrees of wrinkledness (and of other traits) in real pea seeds, as – we shall see shortly – was clearly demonstrated by Weldon in 1902 (Weldon 1902a, b).

The point can be stated more generally. The further we explore the molecular basis of traits, the more unwieldy the simple concept of dominance and recessiveness becomes, since we become less and less able to identify dichotomous phenotypes. As our knowledge increases, through the Human Genome Project and other efforts, we increasingly recognize that the vast majority of human genetic traits are multifactorial, involving the interaction between different genes within the genome and between the genome and the internal and external environments. Mendel's classic examples are idealized models which, on the one hand, can help students to grasp basic concepts but, on the other, can promote deep-seated misconceptions which can interfere with the ability of students to engage with the full complexity of inheritance.

We need to teach genetics in such a way as to leave students in no doubt that dominance, as Allchin (2005, p. 437) writes, "… is not a property inherent in any isolated allele, but rather varies with context." And this is precisely what Weldon, more than a century ago, went to great lengths to demonstrate by analogy, in development and regeneration, as the next section will recount.

3 The Recovery of an Alternative View of Dominance (And How It Got Lost)

It helps, in understanding Weldon's views and how and why he came to hold them, to place him in relation to another English biologist of the same generation, William Bateson. Born in 1860 and 1861 respectively, they met as undergraduates at Cambridge University, where they studied zoology in the heyday of evolutionary 'morphology', and so undertook extensive study and research in embryology. They became friends, though there was always an instability in the relationship, in that Weldon, a year older, seems always to have outshone Bateson, in one respect or another – and this was an instability that increased in the 1890s as their careers progressed, and Weldon developed a habit of criticizing Bateson in print whenever

he thought Bateson's work merited it. By 1900, they appeared to be heading for very different, and unequal, professional futures; for where Weldon had become the Linacre Professor of Zoology at Oxford, Bateson was, in professional terms, hanging on by his fingertips, with a relatively low-status position at a Cambridge college (though both by this time were sufficiently distinguished to be members of the Royal Society). But 1900 saw changes that brought about quite a reversal of fortune for the both of them.

1900 is famous for biologists and for historians of biology as the year in which Mendel's paper, which was published in 1866, was 'rediscovered'. The word has to go in quotation marks in part because the paper was never completely forgotten among specialists. What happened in 1900 was that the paper suddenly became a talking point throughout European botany. Bateson and Weldon responded in very different ways to this development. Between 1900 and 1902, Bateson became increasingly persuaded that Mendel's paper represented a new foundation for a truly scientific science of inheritance, which would be experimentally precise as well as quantitative. Weldon, by contrast, came to think that any attempt to put Mendel's work at the centre of the understanding of inheritance was wrongheaded, and indeed a huge backward step for biology. In 1902, he published a critique of the Mendelian perspective in the journal *Biometrika* (Weldon 1902a). Within the historiography of biology, the tradition, right up to the present, has been to treat Weldon's critique as wilfully obstructive and deeply confused (Schwartz 2008, ch. 7). It was neither of these things; and an effort to reinhabit Weldon's point of view is well worth making.

We can usefully attend first of all to two photographic plates that accompanied his 1902 article, and with which he aimed to show readers that Mendel's laws of inheritance do not seem to work even for peas. Weldon took it upon himself to collect hybrid pea varieties and to study them, conveying some of his results photographically to emphasize his point. The top of his first plate shows a line of peas; the leftmost peas are green and the rightmost peas are yellow. But in between, the peas range from greenish yellow to yellowish green. It appears that nature actually presents a continuum of colours. Certainly no one else's peas seemed to look the way Mendel reported his peas as looking. We see a similar situation with wrinkledness, the subject of Weldon's second plate, which shows peas ranging from smooth to wrinkled, in gradually increasing degrees. Another thing you would never imagine with a Mendelian mindset, Weldon suggests, is that, sometimes, descendant pea varieties in their wrinkledness recall not their immediate ancestors but, as his evidence shows, their more distant ancestors.

What is happening here? It may look like nitpicking. In the textbooks, Mendel is celebrated precisely for the brilliant methods that allowed him to cut through all of the complexity that nature presents and find an underlying simplicity and order. His stroke of genius was his insight that for his hybridization experiments to yield clean results he had to purify his starting materials, which took him years – years spent ensuring that his white-flowering pea plants only ever gave rise to white-flowering pea plants, and that his purple-flowering pea plants only ever gave rise to purple-flowering pea plants, and so on. It is hard to view all of that effort with anything

other than reverence. One way into salutary irreverence is to recall an old philosophy-of-science joke. It's night-time. A man is walking down the street, and he sees a second man down on his knees, searching the ground around him:

> First man: "What are you doing?"
> Second man: "Oh, I dropped my keys on the other side of the street."
> First man: "So why are you looking for them over here?"
> Second man: "Well, the light is better here."

As Weldon saw it, performing experiments à la Mendel in the name of understanding inheritance is an exercise in looking where the light is better, rather than where the keys are – the keys that unlock the most profound mysteries about inheritance. It suits *us*, the investigators, to eliminate all of the variability that creates complexity, and thus to generate patterns which are simple, so much so that they can be treated with simple combinatorial mathematics. But there is no reason, in Weldon's view, for regarding what is produced thereby as somehow basic, foundational, fundamental. It is just arbitrary to do that. Furthermore, in taking that step – in treating that arbitrary order as God-given – we cease to take a serious interest in variability and complexity and the lessons they might hold about how real inheritance (as distinct from an artificially engineered and arbitrarily simplified version of it) really works.

Weldon himself sought illustrations not in the world of jokes but in the history of science. When he tried to engage his audience – notably students on a summer course at Oxford in 1905 – he recalled an episode from the recent history of physics and chemistry. The middle years of the 1890s saw a new column added to the Periodic Table: the noble gases, starting with argon. As Weldon told the story, this development resulted from William Ramsey and Lord Rayleigh being unwilling to let themselves off the hook when confronted with a discrepancy in their data, unwilling to fictionalize it away – in this case, a difference of hundredths of a gram between nitrogen collected from nitrogen-bearing compounds and nitrogen isolated from the atmosphere, after all the other then-known gases had been removed from a sample. Atmospheric nitrogen was just a little bit heavier – again, just hundredths of a gram – but not forgetting about that difference, indeed taking it very seriously, worrying about it and at it, led to the discovery of new elements (and, in 1904, a Nobel Prize). That, in Weldon's view, is great science; it depends on not turning a blind eye to the complexity you experience, and not idealizing it into conceptual oblivion.

In drawing the reader's attention to all the natural variability whose existence is ignored in Mendel's exposition, Weldon was trying to suggest to biologists that it was absolutely vital – when it came to identifying new patterns, when it came to avoiding misleading organizing concepts – to bear in mind exactly what nature presents them with. And he drove the point home in the paper with an example now strongly associated with Mendelism: eye colour in humans. We now live in a world where educated people think they know that dark-coloured eyes are dominant and light-coloured eyes recessive, with family lineages showing classic Mendelian patterns. A light-eyed child from dark-eyed parents? The parents must be heterozygous for eye colour. A dark-eyed child from light-eyed parents? Well, that cannot happen, unless… So widespread is the latter pattern, and the related chain of Mendelian reasoning (and suspicions of adultery), that one can now easily find on

the web popular-science columns reassuring parents that yes, light-eyed parents *can* have dark-eyed children! Because, unlike what educated people tend to have learned, eye colour is now known to be the result of interaction between many genes, whose collective effects can result in any of the possible colours we see in humans.[5]

Here is Weldon in 1902 (our emphasis):

> It would almost certainly be possible, by selecting cases of marriage between men and women of appropriate ancestry, to demonstrate for their families a law of dominance of dark over light eye-colour, *or of light over dark. Such a law might be as valid for the families of selected ancestry as Mendel's laws are for his peas and for other peas of probably similar ancestral history, but it would fail when applied to dark and light-eyed parents in general – that is, to parents of any ancestry who happen to possess eyes of given colour.* (1902a, p. 242)

In other words, the experimental investigator could justify either 'law' – dark dominant over light or light dominant over dark – depending on the starting materials chosen (and excluded). And one remembers in coming across this passage that one of Weldon's own greatest scientific heroes, Francis Galton, had in his collection a tin box of glass eyes, whose iris colours divided up not into two kinds, light and dark, but into a spectrum comprising 16 different kinds.

With the publication of Weldon's 1902 critique, the "biometrician-Mendelian" debate, as it has subsequently become known, was launched. What followed constitutes one of the most ferocious controversies in the whole of the history of science (on this debate see MacKenzie and Barnes 1974; Olby 1988; Kim 1994). A more recent name, 'the Mendel wars', captures the tenor. For Bateson's part, he was very energetic and successful at recruiting clever allies to his cause, such as his Cambridge colleague Reginald Punnett, famous to genetics students the world over for the Punnett square, a diagrammatic way of keeping track of the various outcomes of crosses and their probabilities. He also published the first textbook on Mendelian inheritance, *Mendel's Principles of Heredity: A Defence*. It came out in the late spring of 1902 and was intended as a rebuttal – a very rude rebuttal – both to Weldon's attack and to Weldon personally. As the book went on into successive editions, the direct attack on Weldon dropped away. But what was left became a template which has moulded genetics textbooks right through to the present: Mendel's experiments show us the first step; the rest is extension; and when the extensions do not work in a straightforward way there follows allowance for exceptions and exculpatory explanations. But it all starts with Mendel and his peas. For Bateson, the peas were important in more than just a theoretical sense. He always emphasized the utility of Mendelism, and was actively engaged in marketing the new Mendelian genetics ("genetics" was Bateson's own coinage) to farmers and animal and plant breeders (Charnley and Radick 2013).

From the time of Francis Bacon, our culture has come to expect that from the finding of true principles there will flow useful techniques; and Bateson accordingly

[5]For a review of the complex, polygenic nature of the inheritance of eye colour, see Sturm and Frudakis (2004); for discussion of the reasons why determinist explanations of patterns of inheritance are attractive, see Moore (2008).

represented the Mendelian perspective as giving to the breeder at last the kind of power the chemist had to plug qualities in and out. Bateson promised in his 1902 book that the breeder would no longer have to trudge benightedly along the paths of tradition, because breeding would have a new, scientific basis. Experimental plots were set up at Cambridge and elsewhere aiming precisely to show the power of Mendelian views in generating new and commercially attractive plant and animal varieties. And in extolling the usefulness of Mendelism, Bateson did not shy away from stressing its usefulness to eugenics – the breeding of better people through science. His was an era, of course, in which more or less everyone with scientific interests was supportive of eugenics, and Bateson was no exception. Even so, Mendelism was seized upon by eugenicists as especially apt for their cause, suggesting as it did that quite complex traits could be governed by single genes and, by extension, would therefore be amenable to selective breeding (see, for instance, the inheritance-of-eye-colour genealogical charts that adorned walls in Nazi Germany).

What about Weldon? Weldon was different. He spent the years from 1902 to 1906 trying to develop an alternative science of inheritance. He knew what he was against: the placing of Mendel's experiments at the centre. But what was he for? He set down his ideas most fully in a remarkable manuscript entitled *Theory of Inheritance* which, though never completed, nevertheless serves to suggest vividly where Weldon was going (and, for anyone who knows the standard secondary literature on Weldon, where he was going comes as an immense surprise).[6] Two features of Weldon's vision are especially relevant for present purposes. One – and this is unsurprising to anyone familiar with Weldon's biometrical allegiance – is the commitment to statistical description. Statistics are essential, Weldon thought, because it is only with statistical language that biologists can describe biological variability precisely. And again, he thought that the really momentous insights in science, the great leaps forward, depend on keeping visible all of the data in all of their complexity – and statistics is the means.

A second more surprising feature is his emphasis on experimental embryology. Successor to the morphological embryology in which Weldon trained, experimental embryology was one of the premier sciences of the late nineteenth century; and a significant proportion of the manuscript reviews experiments involving artificially induced regeneration, as when the parts of an individual *Stentor* (a protozoan) cut into three each went on to develop into a fully formed individual. The lesson that Weldon drew from all of this extraordinarily detailed experimental work, by himself and others – which, fascinatingly, he regarded as merely confirming the theoretical insights of Galton decades before – was that dominance is not something permanently associated with a biological character. Rather, what is expressed by a biological tissue depends essentially on the tissues surrounding it. Expression is fundamentally context-dependent. One would never guess that the middle region of the *Stentor* had the capacity to become an entire *Stentor* until the tissues surrounding that region are removed experimentally. Similarly, higher up the animal scale, a cell that would normally always

[6]The authors are currently working on an annotated edition of Weldon's manuscript.

develop into an anterior structure will, when relocated to the posterior of the animal during a certain stage of development, develop as a posterior structure. And lower down, at the level of the hereditary factors – Weldon favoured Weismann's term "determinants" – one and the same character can be dominant (i.e. expressed), or recessive (i.e. not expressed), or falling between those categories, depending on the company kept. Weldon's is a view of dominance and recessiveness emerging from *interaction*: interaction among the hereditary determinants, interaction in turn with their environments, biological and physico-chemical.[7]

Before we go on to suggest how this recovered Weldonian perspective might prove fruitful for reorganizing the genetics curriculum in the age of genomics, and so remedying some of the shortcomings surveyed in the previous section, we offer a few quotations from the manuscript, not least because Weldon's clarity on these issues is so striking (our emphases throughout):

> The group of properties which normally becomes dominant in the case of any particular unit is determined by its position relatively to the other units which make up the body; for we have seen that any group of units, forming the cut surface of a divided *Stentor*, can be made to exhibit dominance of any given group of properties, so that it can be made to produce any given set of organs, if we remove certain adjacent portions of the body, so as to leave the units in question in a suitable relation of position with regard to the next. (Weldon, ch2a, p. 6)

> If we disturb the normal relation of these tissue elements to their neighbours, as we do by removing them from the body, *we can render dominant properties which were previously recessive, and vice versa*. (Weldon, ch2a, p. 21)

> Whatever may happen during the process of inheritance, it is clear that during the life and growth of single individuals such as those we have examined, *Mendel's conception of dominance as a property permanently belonging to the determinants of certain characters, wherever these are in the presence of certain others, is altogether inadequate*. The tissues of these animals are neither "pure" in the sense that they contain only determinants representing a single character or group of characters, nor constant in the sense that some of the determinants they contain are of necessity dominant over the others: on the contrary, *each tissue can be shown by experiment to be in the condition indicated by Galton's hypothesis, behaving as if it contained determinant elements which represent a large series of characters, any one or more of which can become either dominant or recessive, according to circumstances*. Experiments of the kind we have described show that one factor which determines the dominance or latency of characters in a tissue is the relation between the tissue itself and other parts of the body, and apparently in many cases, as in reversed grafts of the Hydra, an essential factor is rather relative position than any of the more complicated relations connected with nutritive or other processes. (Weldon, ch3, pp. 18–19)

In the spring of 1906, this debate over Mendelism – but above all, as we have seen, over the concept of dominance that ought to dominate in the science of inheritance – more or less came to an end with the unexpected death of Weldon at the age of 46 (from pneumonia brought on, some said, by overwork and stress). Weldon's manuscript was never finished and never published, and the field was left open for Bateson to develop his programme to the pitch of textbook glory that it later achieved.

[7] On heredity-environment interactions in Galton's work and Mendel's, see Radick (2011).

4 What if? Remaking the Curriculum Along Weldonian Lines

A question that naturally arises in cases where someone who might have changed things dies or is otherwise incapacitated before finishing the job is to ask: what if? Or, more precisely, what might have been? What might biology have looked liked had Weldon lived longer and – to give this speculation a little spice – been more successful than he actually was at recruiting clever young allies and at marketing his point of view to agriculturalists and doctors? Could there have been a successful Weldonian science of inheritance? What would it have looked like? And would it have been anything like as successful as the science of inheritance that we actually have?

We are persuaded that these sorts of what-if or counterfactual questions are very important, for all of us, but for historians and philosophers of science especially. They are important historically because if we do not pose, and try to answer, these questions, we cannot weigh the significance of past events; and to that extent we cannot really explain why things happened as they did and not otherwise. Did Weldon's death matter for the history of biology? Not much, if Mendelian genetics was destined to emerge in one form or another and eventually settle down to something like its current form; it matters a great deal, however, if that was *not* the inevitable outcome. Counterfactual history-of-science questions are also important philosophically because at stake in confronting them is our sense of what scientific knowledge is, and in particular what makes it worthy of the esteem in which that knowledge is held. Underpinning the widespread notion that scientific knowledge represents human reason at its most objective is the further notion that scientific knowledge is independent of local historical conditions. One group of investigators may, for contingent reasons, come to hold one view as true; another equally competent group may, for different contingent reasons, come to hold another, incompatible view. But in the end, if science is working well, all views will converge on the truth. But will they really? Why should we think so?[8] There are puzzles here aplenty for philosophers, however historically inclined. And for scientists too. Suppose, for argument's sake, that a distinctively Weldonian science of inheritance could have developed out of Weldon's manuscript. And suppose that that science might have yielded insights into inheritance that our science now lacks because of the particular pathway it followed. What might we be missing?

An obvious way to find out, it seems to us, is to try to create the Weldonian science that never was. One can imagine doing this on various scales, from the grand to the modest. At a modest scale, a promising focus is precisely the problems in current genetics pedagogy discussed above. Again, the textbook tradition has been remarkably conservative. From Bateson's day to the present, students start with Mendel's peas; and all of the complex information about inheritance accumulated by the biological sciences in the intervening century is treated as an add-on, tucked

[8] On counterfactuals and the history of science in general, see Radick 2008; on the case for a counterfactual history of genetics, with Weldon as a focus, see Radick 2005.

in along the way. And so we find ourselves in a situation where the points that Weldon was making about the interactive nature of hereditary factors with each other and with their environments are now widely acknowledged, even taken for granted, among biologists, and yet the textbooks remain, by and large, organized around Mendelian phenomena which – and this is their traditional attraction – can be made sense of without considering environmental interactions of any kind.

Does it have to be like that? What are the alternatives? Is it possible to imagine a genetics pedagogy which, in a Weldonian spirit, set off differently? Suppose, instead of starting with Mendel and his peas, we start with something genuinely representative of how genes function in bodies,[9] and even better, representative of the sort of genetic causation that may actually matter to the lives of a large number of people – say, the contribution that genes make to the condition of a human heart. Here is a case of interaction on a massive scale: lots of genes interacting with one another, and in complex ways with exercise and diet (including ingestion of pollutants from air and water), and the whole collocation changing over time as the individual person matures. What would happen if you set beginning students to thinking about cases like that in the first instance; and if, as you developed your instruction, week by week, chapter by chapter, you foregrounded gene-environment interaction as pervasive and primary, not secondary and selectively present? And suppose further that the students, having followed the curriculum for several weeks and assiduously worked their way through the chapters of this imagined textbook, eventually – let us say, around chapter 8 in the textbook (roughly where they meet gene-interactions now) – they get to Mendel and his peas. But they see the pea case as a special case. These patterns do arise; but they arise only under special conditions, notably when humans have engineered artificially purified lineages into being, by deliberately excluding unwanted variability (and note here how reliant genetics textbooks are on *domesticated* plants and animals, rather than their wild and genetically untidy counterparts; the specific strains of fruit flies, zebra fish and mice, for example, used in the experiments described in textbooks do not – indeed, often could not – exist outside the laboratory).

What would those students be like? One prospect is that they might have a more sceptical attitude than is generally the case when it comes to the notion, familiar enough outside biologists' circles, that genes are 'super' causes – 'genetic determinism', in the philosophers' jargon. They might be that bit more prepared to insist, when confronted with claims for the discovery of a gene for a particular trait, that they be told about the range of environments, genetic and otherwise, in play in the course of the investigations, because they will know, in their bones as it were, that any such claims are implicitly founded on observations made within such a (limited) range.[10]

[9]The strategy outlined here has affinities with that advocated in Dougherty 2009. Of course, the very concept of the 'gene' is problematic; see Burian and Kampourakis, this volume, for discussion of an alternative way to conceptualize the material of heredity.

[10]On the problems of genetic determinism see Lewontin (1993); see also Moore, this volume.

It is instructive, in the light of that possibility, to look back with renewed attention at how determinism leaks into genetics textbooks even as their authors attempt to disabuse students of determinist attitudes. Consider the following, witty back-of-the-chapter problem from a textbook already discussed, Russell's *iGenetics: A Mendelian Approach*:

> After a few years of marriage, a woman comes to believe that, among all of the reasonable relatives in her and her husband's families, her husband, her mother-in-law, and her father have so many similarities in their unreasonableness that they must share a mutation. A friend taking a genetics course assures her that it is unlikely that this trait has a genetic basis and that, even if it did, all of her children would be reasonable. Diagram and analyze the relevant pedigree to evaluate whether the friend's advice is accurate (Russell 2006, p. 38).

The friend scoffs at the possibility that something as complex as unreasonableness could have "a genetic basis". But notice what happens when the friend – and, in the friend's footsteps, the student attempting to solve the problem – decides to suppose, in a hypothetical spirit, that unreasonableness does have a genetic basis, and furthermore that the nature of that basis can be disclosed through pedigree analysis. There is, of course, a textbook-sanctioned right answer to the question; and it can be reached only by supposing that there is a gene for unreasonableness, and that it behaves exactly as a gene for seed colour or flower colour in peas is supposed, by Mendelian textbooks, to behave. But consider again that phrase, "genetic basis." It suggests, of course, a trait caused by genes interacting with environments. On a certain view, one might with equal justification describe the trait as having an "environmental basis" – but nothing so described would tempt the student toward the just-like-Mendel's-peas problem-solving paradigm. The approved answer to this question, by the way, is that the friend's advice is wrong: the woman's children would have a 50 % chance of being unreasonable whether the gene for unreasonableness is dominant or recessive.[11]

Where current genetics pedagogy remains still so much the product of its Mendelian history, the alternative pedagogy we have outlined is rooted in the Weldonian history that never happened (but might have). Another statement from Weldon's *Theory of Inheritance* manuscript can serve as a credo:

> Since the character of any organ depends, not only upon a specific something transmitted to it through the germ-cells out of which it was developed, that is to say upon something inherited, but also on two sets of conditions external to the organ itself, namely its relation to the parts of the body to which it belongs, and its relation to the environment in which that body exists, we may say that every character of every animal is both 'inherited' and 'acquired'. (ch. 5, p. 24, Jamieson transcription).

Recently, a small group at the University of Leeds, including ourselves and a distinguished genetics education specialist, Dr Jenny Lewis (whose work we have already mentioned), has come to be in a position to translate this vision into reality. Over the

[11] On the critique of "genetic basis" talk see Kitcher (1997, ch. 11, esp. p. 251); on the concept of heritability, and why the nature/nurture dichotomy no longer holds explanatory power in biology, see Moore, this volume.

course of a 2-year project now underway, we aim, in pilot-study form, to devise a curriculum for genetics that will, in Allchin's phrase, "dissolve dominance" in order to give students a genetics education fit for purpose in the twenty-first century.

The challenges before us are not to be underestimated. In closing let us mention three. An obvious one is to find a way to combine a simple, pragmatic language, accessible to the non-specialist, with content that is still sufficiently detailed and accurate to avoid the current problems with misconceptions and misunderstandings. One promising strategy here is Allchin's suggested emphasis on haplosufficiency. To teach with that concept in mind would be to convey to students the message that many "nonfunctioning" alleles produce something. It might be a 'faulty' product; but it is nevertheless present; and every allele product has some action (albeit imperceptible at the phenotypic level in some cases). Furthermore, for some physiological processes, one normal product is sufficient for normal function/phenotype, whereas others need two fully functioning alleles, and still others will have some intermediate state for the heterozygote. As we noted above in our comments on the molecular genetics of both sickle-cell anemia and seed shape in peas, such an approach will help students to avoid the presence/absence implications of classical Mendelian analysis.

A second challenge is, in a Weldonian spirit, to keep the focus on development, and the extent to which organisms develop as they do thanks to a set of complexly interacting factors, 'inheritance' being only one of these, and in itself subject to the vagaries of time and place as much as everything else biological. Consider, for these purposes, the pedgagic potential of maternal inheritance and maternal effect, both of which display 'non-Mendelian' inheritance in the phenotype. Maternal inheritance is the sum of the transmissible characteristics carried on the mitochondrial genome, and hence entirely matrilineal and unaffected by any paternal contribution. Maternal effect is the effect of the products of maternal genes present in the cytoplasm of the egg – genes which control fundamental early developmental processes, such as body axis formation, before the zygotic genome begins to be expressed. Critical early events in development are thus under the influence of a different genome than that of the zygote. Development also provides the opportunity to emphasize the influence of environmental effects. Neural tube closure, for example, is dependent on a number of known genes and also on a variety of environmental factors, some well understood – folic acid, for example – others still quite mysterious, such as socioeconomic group (Gilbert 2010, pp. 340–341). Such examples, introduced very early in the curriculum, could do much to encourage in students an appreciation for the multiple factors involved in development, rather than genetic determinism.

A third, quite different but no less important challenge is to build in scope for problem solving. Students on a traditional genetics course are expected to be able to predict the outcome of specified crosses, and to analyse pedigrees so as to establish patterns of transmission of traits. Manifestly, the dominance/recessiveness dichotomy is very powerful in this context, underpinning a set of problem-solving techniques which, over the century-plus during which Mendelian pedagogy has been honed, have become partnered to problem sets well-calibrated to instilling mastery of those techniques and so to distinguishing degrees of mastery in the students. Any alternative curriculum must either ensure that it provides commensurately ample

scope for prediction and analysis or find appropriate surrogates. Perhaps, in our own attempt at such a curriculum, it will be sufficient to introduce dominance/recessiveness at a later stage, along with a sample of the standard problem sets, having made sure the students are firmly grounded in gene-environment interactionism and the contingent nature of development, so that they clearly grasp that the concept of dominance is merely a tool and not a fact of biology.

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