The Evolution of Biological Theories: Explaining the Success of Mendelian Genetics, Darwin's Theory of Natural Selection and their Synthesis

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A minor dissertation submitted in partial fullfilment
of the requirements for the award of the degree of Master of Philosophy

Faculty of the Humanities
University of Cape Town
2019

Compulsory Declaration

This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, and has been cited and referenced.

Signed by candidate

9th of February 2019

1 Many thanks to Dr. Jack Ritchie for extensive comments and advice.

Abstract

Darwin's theory of natural selection was not widely accepted in the biological community until its synthesis with Mendelian genetics. I investigate the history of both sciences, with the aim discovering why Mendelian genetics and the synthesis were scientifically successful. One possible explanation for this is given by constructivism, the view that developments in science are decided not by rational reasons, but by contingent factors. A sophisticated version of this view is defended by Gregory Radick, who argues that Weldonian biometry, a rival theory of inheritance, could have supplanted Mendelism. For Radick, the success of Mendelism and the corresponding decline of biometry can be explained by historical circumstances, such as Weldon's untimely death and his inability to recruit talented students. Another popular philosophical explanation of scientific developments is scientific realism, whose proponents argue that scientific success can be explained by the truth of scientific theories. More sophisticated versions of realism, such as Weisberg's, take the routine scientific distortion of truth (idealization) into account. I argue from the history of genetics that neither constructivism nor realism, sophisticated or otherwise, can help us understand the success of Mendelian genetics. Instead, I argue that there were rational reasons in favor of Mendelian genetics, even if it was not a true theory of inheritance. I further conclude that the synthesis was successful because Mendelian genetics theoretically enriched Darwin's theory of natural selection. This enrichment solved serious empirical and conceptual problems for Darwin's theory, showing that we can also understand the success of the synthesis without appeal to broad realist or constructivist views.

Introduction

The middle of the 19th century saw the publication of two extremely important works for the biological sciences. The first, Charles Darwin's "On the Origins of Species by Means of Natural Selection" published in 1859, introduced Darwin's views on descent and his theory of natural selection to the scientific community. Just 7 years later, Gregor Mendel published his paper "Experiments on Plant Hybrids", in which he presented his findings on inheritance. Today both Darwin and Mendel are thought of as the progenitors of evolutionary biology and genetics respectively. But before their later successes, the history of Darwinian and Mendelian science takes some surprising turns.

Although most biologists were convinced by Darwin's view that species evolved from common ancestors, Darwin's theory of natural selection, his proposed explanation for how evolution occurred, was less popular. The idea that natural selection was the driving force behind evolution only became the consensus view during the synthesis of Darwin's theory with Mendelian genetics (Sarkar, 2017: 753), which started in the 1920s (Sarkar, 2017: 754).

Mendel's work languished in relative obscurity until it resurfaced in British biology at the start of the 20th century. At that point it received significant criticism from the biometrical school of biology, sparking a controversy between the biometricians and the early Mendelians. Despite a difficult start, Mendelian genetics eventually asserted itself as the dominant theory of inheritance and variation in British biology. Later Mendelian genetics was further developed and synthesized with the theory of natural selection.

In this paper, I will try to discover why these two theories came to be accepted in the scientific community. In particular, I want to explain why Darwin's theory of natural selection was only widely accepted once it was combined with Mendelian genetics in the synthesis. In doing so, I will counter realism and constructivism, two popular philosophical views of scientific theory acceptance. To both develop my own view and rebut these alternatives, I will discuss the history of Mendelian genetics and Darwin's theory of natural selection in detail.

First let us consider scientific realism. Realists believe that empirical success, and in turn the widespread acceptance of a theory in the scientific community, can be explained by that theory's (approximate) truthfulness. A famous philosophical defense of this view was given by Hillary Putnam and what came to be known as the 'No Miracles' argument. For Putnam, it would be a miracle if a scientific theory were empirically, predictively and explanatorily successful without being true (Chakravartty, 2017). So according to Putnam successful scientific theories are very likely to be true, and since the scientific community accepts successful theories, those theories that are the consensus are also most likely true. If Putnam's theory applies to our case, then we can explain the success, and thereby the acceptance, of Mendelian genetics and Darwinism by their truth.

The other general view I will argue against is constructivism. Constructivists, unlike realists, think that rational reasons (often) do not determine which theories become accepted in the scientific community. Instead, the course of scientific history can be determined by historical accidents and contingent circumstances. For example, a good explanation of the fact that Darwin's views were initially more popular than Mendel's is that Darwin was already a well known and respected biologist, while Mendel was not. A more exciting

and controversial constructivist view has been defended by historian and philosopher of science Gregory Radick. He argues that the biometricians could have triumphed over their Mendelian rivals, and that Weldon, a prominent biometrician, could have developed a successful alternative theory of inheritance (Radick, 2016: 159). According to Radick, Weldon had good arguments against Mendel (Radick, 2016: 158), but ultimately it was contingent circumstances, such Weldon's untimely death, that decided the debate in the Mendelians' favor (Radick, 2016: 159).

Of course both realism and constructivism are very broad attitudes about science. It is possible to have a realist or constructivist view of a certain scientific episode without having a similar view about the entirety of science. This also means that constructivism and realism are not mutually exclusive. For example, Radick is a constructivist about the controversy between the biometricians and the Mendelians, but is also a scientific realist. Although I will argue that constructivism and realism are broadly unhelpful for our purposes, I will conclude that they can explain some details of the history of genetical and evolutionary biology.

A view that I think is much more useful for understanding the significance of the synthesis comes from Bas C. van Fraassen's paper the "The Perils of Perrin, in the Hands of Philosophers". Van Fraassen discusses the history of the atomic theory, which he thinks faced serious empirical difficulties before Perrin's famous experiments with colloids (Van Fraassen, 2009: 25). For van Fraassen, the crucial problem for the atomic theory was that it was impossible to derive important parameters, such as the atomic mass, from measurements. Perrin's achievement consisted in *enriching* the atomic theory with new theoretical hypotheses, which allowed him to derive the elusive parameters (Van Fraassen, 2009: 25). I will argue that a simplified version of van Fraassen's story, where a theory's empirical difficulties are solved by enriching it with new theory, can help us understand why Darwin's theory became more widely accepted after the synthesis.[2]

In the first chapter I will discuss Darwin's theory of natural selection and why it was initially relatively unpopular in the scientific community. A naive constructivist explanation for this is that scientists were blinded by their creationist biases. But that explanation doesn't make sense of the fact scientists warmed to Darwin's evidence for common descent, which also contradicts creationism, much more quickly. In fact, I will argue that constructivism per se is not going to be helpful here, because there were good reasons to be skeptical of Darwin's theory of natural selection. These problems arose for Darwin due to the lack of a good theory of inheritance and variation, showing that Darwin's work needed to be enriched by such a theory (like Mendelian genetics). I conclude that this is a good preliminary explanation for the success of the synthesis.

The next two chapters are devoted to explaining the early history of Mendelian genetics, and highlighting the rational reasons in favor of it. In chapter 2 we will see that Mendel's experimental work was characterized by the care he took in abstracting away from irrelevant observations. Mendel further introduced two important theoretical ideas to the study of inheritance. The first is that inheritance is controlled by heritable particles (later known as genes). The second is the phenomena of dominance, where one gene prevents another from affecting the traits of an organism. I will argue that the former idea proved

2 We should also keep van Fraassen's empiricism in mind, since we will want to say that certain episodes in the history of biology were driven by the need to solve empirical problems. However, I will not apply van Fraassen's interesting views on empirical grounding in this paper.

more important to later Mendelians, while his view of dominance was quickly qualified. So we should think of the success of Mendelism as largely resting on the particulate genetical theory, rather than on Mendel's view of dominance.

The third chapter features my evaluation of Radick's discussion of the controversy between the biometricians and the Mendelians. I will argue that there were Mendelian successes after the debate which Radick ignores or downplays. Furthermore, I will show that the arguments that Weldon, one of the leading biometricians, brought to bear against Mendel could have been answered by later Mendelians. So I am skeptical of Radick's sophisticated constructivist story as well.

At this point the discussion will appear to favor the realist view. It might seem that, since the rational reasons favored Mendel's theory, the success of his particulate theory can be attributed to its approximate truth. This realist approach might ascribe the success of the synthesis to the combination of two approximately true theories of inheritance and evolution. But I think this approach is both false and unilluminating. It is false, because, as I will argue in the fourth chapter, modern genetics shows that Mendelian theory was not even approximately true. It is unhelpful because it obscures the real reasons that the synthesis was a success, which I will discuss in my final chapter on Fisher's book, "The genetical theory of natural selection". Here we will see how Fisher uses Mendelian genetics to solve Darwin's empirical and conceptual problems. We will also see that Fisher's work is aided by the introduction of various mathematical and statistical techniques, an important independent trend in biology.

I will conclude that theoretical enrichment is important to understanding the history of Mendelian genetics and Darwin's theory of natural selection. While I will have argued against broad realist and constructivist explanations of this scientific history, I will conclude that both philosophical views can help explain some elements of biological history. So ultimately doing philosophy of science well is more difficult and complicated than accepting a single, overarching philosophical theory.

Chapter 1: The Dangers for Darwin

Darwin's most famous work, his 1859 "On the Origins of Species", included both his views on descent and his theory of natural selection. Darwin's work on descent argued that species evolved from common ancestors, while his theory of natural selection provided an explanation of how said evolution worked. The theory of natural selection did not enjoy widespread scientific support until the evolutionary synthesis between Mendelian genetics and Darwinism (Allen, 1968: 113). While there was certainly creationist opposition to Darwin's view that species evolved, many of Darwin's scientific critics believed in evolution themselves. So we can't ascribe opposition to Darwin's theory purely to religious biases. In fact I don't think constructivism per se will be helpful in this chapter, because I will show that there were rational reasons to doubt Darwin's theory of natural selection.

I will argue that Darwin's theory faced empirical and conceptual difficulties because it lacked a good theory of inheritance and variation. Firstly, we will see how Darwin's theoretical statement in "the Origins of Species" referred to inheritance and variation. However, while Darwin provided an impressive amount of facts about inheritance and variation in the wild and in domesticated species, he admitted ignorance of the causes of and laws governing these processes. So our discussion of Darwin's book shows that his theory was conceptually incomplete. This incompleteness led to empirical problems when Darwin employed bad theories of inheritance. As an example, I will look at Fisher's discussion of how Darwin failed to derive correct predictions when he (tacitly) assumed the blending theory of inheritance.

Darwin's "On the Origins of Species" begins with three chapters dedicated to explaining the building blocks of his theory of natural selection. The first two chapters discuss inheritance and variation both in the wild and in domesticated species. Darwin argues that human breeders can effectively select and promote traits in animals and plants, which tacitly suggests that the (potentially) analogous process of natural selection is also possible. Furthermore, Darwin argued that there are many different traits that can appear in domestic animals, and that the same is true, to a lesser extent, for wild animals (Darwin, 1872: 80). We will refer to these new or different traits as variations. Darwin claimed that inheritance could be faithful enough to allow for these variations to be transferred from parent to child (Darwin, 1872: 80). Together variation and inheritance make selection possible, because a human breeder can select preferable variations, which can then be inherited to form a new breed. The same holds true for the (potentially) analogous process of natural selection. So we can already see that Darwin makes a number of claims about inheritance and variation, which suggests that a theory of both is essential to an account of natural selection.

The third chapter is dedicated to another important component of Darwin's theory of natural selection: the struggle for existence. Darwin notes that in nature, individuals typically struggle to survive, and that the survival of one individual is often incompatible with the survival of another. For example, so many plants might grow in an area that new sprouts cannot get enough sunlight to photosynthesize. As this example illustrates, a struggle for existence in Darwin's sense need not involve anything as dramatic as predation or violent competition. However, it is nonetheless the case that survival in the wild is very difficult, and that therefore the difference between life and death can depend on very thin margins. Those individuals who are successful in the struggle for life are also more likely

to reproduce (because they aren't dead), meaning that differential survival can lead to differential reproductive output.[3]

Now that Darwin has all the pieces in place, he moves on to explain his theory of natural selection, as can be seen in the following passage from the fourth chapter:

"Can it, then, be thought improbable, seeing that variations useful to man have undoubtedly occurred, that other variations useful in some way to each being in the great and complex battle of life, should sometimes occur in the course of thousands of generations? If such do occur, can we doubt (remembering that many more individuals are born that can possibly survive) that individuals having any advantage, however slight, over others, would have the best chance of surviving and of procreating their kind? On the other hand, we may feel sure that any variation in the least degree injurious would be rigidly destroyed. This preservation of favorable variations and the rejection of injurious variations, I call Natural Selection." (Darwin, 1872: 80)

First Darwin reminds us of the great degree of variation in wild and domesticated species, and concludes that, since so many new variations occur over time, it is likely that new traits arise which could aid its carrier in surviving. Due to the struggle for existence, these advantageous traits can mean the difference between life and death. But that means that its carriers have a better chance both of surviving and of living to reproduce. So on average carriers of the trait will produce more offspring, and since we have already learned that traits can be faithfully inherited, the advantageous trait will likely become more prevalent in the next generation. So Darwin's explanations of variation, inheritance and the struggle for existence allow him to conclude that advantageous mutations are likely to be preserved and promoted across generations. As Darwin indicates at the end of the quote, the same reasoning can also be used to show that disadvantageous mutations are likely to be eliminated, because their carriers will be less likely to survive the intense struggle for existence.

As we can see, Darwin's theory of natural selection requires that inheritance and variation take place. Much more importantly, it makes substantive claims about inheritance and variation, which is a problem because Darwin had no solid theoretical understanding of these processes. I think that Darwin must have sensed some of this difficulty, given that he was quite open about his relative ignorance of the mechanisms of inheritance. We can see this from the following remark he makes in the first chapter: "The laws governing inheritance are quite unknown; no one can say why the same peculiarity in different individuals of the same species, and in individuals of different species, is sometimes inherited and sometimes not so; …" (Darwin, 1872: 13)

Now that we understand the conceptual problem that a lack of theoretical knowledge of inheritance presented for Darwin, I want to argue that said lack of knowledge also led to empirical problems for him. In order to make predictions, Darwin needed to support his theory of natural selection with a theory of inheritance. However, Darwin only had access to bad theories of inheritance, and so he was unable to make accurate predictions. In the

3 In fact differential reproductive output is so important that modern Darwinist (following Pearson) prefer to speak of fitness (the organisms likelihood to reproduce, or to reproduce many times) than of differential survival. Furthermore, after Darwin the struggle for existence was eventually not thought of as necessary for differential survival. See for instance pages 43 and 44 of Fisher's "the genetical theory of natural selection" for an example the latter objection.

following example from Fisher's book, we will see how Darwin's theory failed to make accurate predictions, due to his tacit assumption of the blending theory of inheritance. Here I follow Fisher's reconstruction of Darwin's reasoning from a number of sources. (Fisher, 1930: 2)

The blending theory of inheritance holds that the heritable material of both parents is mixed in the child. The child will have traits that are intermediaries between those of the parents, so, for example, two parents with different heights will produce children with height somewhere between the upper and lower limits given by the parents. As Fisher recounts, Darwin noticed that (blended) inheritance will cause populations to rapidly tend towards uniformity in traits. According to the blending theory of inheritance, the child will tend to inherit an intermediary trait from its parents. Therefore, it is quite unlikely that unusual traits, such as extreme tallness, will be inherited across one generation. Since this holds for every act of procreation, traits of unusual magnitude (such as being taller or shorter than the average) will be very likely to disappear as a number of generations pass. So we can conclude, as Fisher's reconstruction of Darwin's argument does, that "with blending inheritance bisexual reproduction will tend rapidly to produce uniformity". (Fisher, 1930: 2)

It follows that evolution would, on this theory of inheritance, be constantly stymied by the effects of blended inheritance. So if we accept the blending theory, we have to explain the variety in plants and animals, and the evolution of these species, by appeal to a theory of variation. Since blending tends so quickly to uniformity, we need variations to be numerous enough to potentially overpower the blunting effect of blended inheritance. Darwin's idea was that new variations were very common, which meant that a new, favorable variation could arise multiple times, and then be naturally selected despite inheritance's strong tendency towards uniformity. Once the new variation becomes normal, the population will have evolved. Similarly, we can explain the apparent diversity in individual's traits by arguing that each individual carries (many) new variations. So on this scientific picture of nature, the theory of variation does a lot of work. We can say that the blending theory of inheritance necessitated this view of variation, since it would otherwise fail to account for diversity of species, and be unable to allow for evolution by natural selection.

However, this theory of inheritance and variation led Darwin into empirical difficulties. Darwin believed that new variations were caused primarily by changes of environmental conditions (like climate) or by changes of the amount of available food. But as Darwin noticed (Fisher, 1930: 7), these causes were poor explanations for variation amongst wild and domesticated species. For example, Darwin wrote that, although domesticated species show a great deal of variation, food could not have increased enough to cause so much variation over such a long period of time (Fisher, 1930: 7). There were similar problems with attributing changes in variation to changes of the plant or animal's environment. Darwin wrote that "it does not appear that a change of climate, whether more or less genial, is one of the most potent causes of variability; for regard to plants Alph. De Candolle, in his Geographie Botanique, repeatedly shows that the native country of a plant, where in most cases it has been longest cultivated, is that where it has yielded the greatest number of varieties" (Fisher, 1930: 7). Instead, Darwin would have expected plants to show more

variation if they were introduced to new countries. Darwin's proposed causes of variation could not account for the empirical observations.[4]

So Darwin's incorrect views about inheritance, variation and the causes of variation led to inaccurate predictions from the theory of natural selection. This suggests that a better theory of inheritance and variation would have solved Darwin's empirical problems. As we will see in our discussion of Fisher, the synthesis provided a much better model of inheritance and variation than the blending theory, and solved the empirical difficulties that Darwin faced. So we will conclude later that the theory of natural selection needed to be enriched by a good theory of inheritance and variation to reach empirical success, and that Mendelian genetics was such a theory. This provides a partial explanation of the success of the synthesis.

Both Darwin's empirical problem and their solution in the synthesis are, according to my analysis both here and later in this paper, driven by rational, scientific reasons. Scientists initially had reasons to doubt Darwin's theory, but during the synthesis these problems, as we shall see later, were answered. This means that my explanation of Darwinism's relative lack of popularity is opposed to constructivism, since constructivists believe that rational reasons do not decide which theories become widely accepted. So far, my story is also consistent with realism, because realists emphasize the importance of rational, truth-apt reasons. But of course we will see in chapter four that this position is not tenable either.

Now that we know why Darwin's theory of natural selection struggled, we can understand why many biologists objected to it: either because of the bad theories of inheritance that Darwinians employed or because the objectors subscribed to a theory of inheritance that was incompatible with Darwinism. Darwinians, including Darwin, frequently made use of dubious theories of inheritance, such as Lamarck's theory (Allen, 1968: 114). This reliance on various poor theories of inheritance not only may have discredited Darwinians in some critics eyes, but led to significant confusion amongst critics as to what the theory of natural selection claimed (as opposed to what the auxiliary theories of inheritance and variations claimed) (Allen, 1968: 115). Others objected to Darwinism because their views of inheritance and variation gave them reason to. A historically important example of this comes from William Bateson, who became the leading British Mendelian at the start of the 19th century. Before that, in 1894, he published his book "Materials for the Study of Variation", where he argued that evolution occurred in discontinuous jumps, because heritable variations came in large, discontinuous jumps (Allen, 1968: 117p). Therefore Bateson thought it was impossible for small variations to slowly add up to a large evolutionary change, which is precisely how Darwin thought most traits evolved. So Darwinism's lack of a good theory of inheritance and variation led not only to internal empirical and theoretical problems, but also left it vulnerable to reasonable (if ultimately mistaken) criticism.

4 Of course this example of Fisher's does not, by any means, exhaust the scope of Darwin's theorizing about inheritance. Darwin ended up devising and accepting his theory of pangenesis. See Ute Deichmann's paper "Gemmules and Elements: On Darwin's and Mendel's Concepts and Methods in Heredity" for reasons why Darwin's theorizing on pangenesis was methodologically suspect.

Chapter 2: Mendel, Morgan and the particulate theory

In the previous chapter we saw that there were rational reasons to doubt Darwin's theory of natural selection. We also learned that these problems arose because Darwin lacked a solid theoretical understanding of inheritance and variation, which suggests that the synthesis was a success for Darwinism because Mendelian genetics provided such an understanding.[5] This proposed explanation is incompatible with a broad constructivist stance, because it implies that rational reasons decided the course of biological history. It is also prima facie conducive to realism, since a realist could claim that the synthesis was a success because Mendelian genetics was an approximately true theory. To refute this view, I will argue in chapter four that modern genetics shows that Mendelian theory is not approximately true (and so that the synthesis was not a combination between two approximately true theories). To do so, I will first need to clarify what Mendel, and Mendelians, believed about inheritance.

In this chapter we will first discuss Mendel's 1866 paper "Experiments on Plant Hybrids", with the aim of clarifying what was distinctive about his work. I will argue that Mendel carefully and methodically examined empirical phenomena. His exemplary empirical method included abstraction away from what Mendel thought were irrelevant factors, and the introduction of combination series to describe his experimental results. Mendel concluded that there were particles (later known as genes) responsible for the inheritance of traits, and that these genes came in dominant-recessive pairs. Next I will argue that, while the Mendel's theorizing about dominant and recessive traits was certainly influential, his views and results were qualified by later Mendelians. In particular, I will show that the idea that traits were controlled by particulate genes was historically a more tenacious and important feature of Mendelian genetics.

Mendel's work is characterized by the care he takes to avoid irrelevant observations. What makes an observation relevant or irrelevant? That depends on the aims of the experimental scientist(s). In the introduction to his paper, Mendel claims that the "striking regularities" (Mendel, 2016: 3) in the inheritance of plant hybrids motivated his investigation. To derive these regularities from experiment, Mendel was careful to notice and avoid various experimental difficulties. This is seen from Mendel's own comments: "the value and the validity of any experiment are conditioned by the suitability of the means used for it, as well as by the appropriate application of the same" (Mendel, 2016: 5). Consequently, "it cannot be immaterial which plant species are chosen as support for the experiments and in which ways these experiments were carried out" (Mendel, 2016: 5).[6]

Mendel lists three conditions that his experimental plants absolutely must meet to be useful. Firstly, they must "possess constantly differing traits" (Mendel, 2016: 5). Secondly, it must be possible to reliably and easily shield the plants from foreign pollen during their flowering

5 We will see in chapter 5 exactly how Mendelian genetics was theoretically invaluable to the theory of natural selection.

6 The idea of carefully choosing experimental plants seems at odds with Mendel's aim of finding regularities. Regularities are supposed to hold for a great number of cases, so what good does it do to focus on a narrow case? In fact Mendel appreciated that accuracy was more important, and more difficult to achieve, than scope. Many other biological theorists failed because they tried to account for too many phenomena without guaranteeing that those observations where empirically accurate. For example, see Deichmann's paper "Gemmules and Elements: On Darwin's and Mendel's Concepts and Methods in Heredity", where she argues that, among other things, Mendel's theory was superior to Darwin's pangenesis because of its focus on accuracy over scope.

period. Lastly, the plants and their descendants cannot suffer serious disturbances in their fertility as the experimental breeding continues. Since Mendel's experiment will require that plants are crossed and observed for many generations, an inability to cross plants due to a loss of fertility would bring the experiment to a screeching halt. An inability to control and know which plants cross with which plants through the invasion of foreign pollen could be equally devastating, since it could lead to false conclusions about which traits have been inherited from which plants. Lastly, if plants do not posses clearly differentiable characters it will be difficult (or impossible) to determine when they have been inherited.

With these criteria in mind, Mendel selected the genus Pisum of the Leguminosae plants. He writes that "some entirely autonomous forms from this genus possess constant, easily and clearly distinguishable traits and, once crossed with each other, render completely fertile descendants in their hybrids. Also, disturbance by foreign pollen cannot easily occur, since the fertilization organs are tightly enclosed by the keel, and since the anthers already burst within the bud, whereby the stigma is covered by pollen even before the flower opens. This circumstance is of particular importance. Other advantages worth mentioning are the ease with which these plants are cultivated both outdoors and in pots, as well as their relatively short vegetation period. Artificial fertilization is admittedly somewhat cumbersome, yet nearly always succeeds" (Mendel, 2016: 6) So Mendel's choice of experimental plant expertly sidesteps the three pressing practical difficulties we discussed earlier: There are no problems with fertility, the traits are easily distinguished and there is little danger of the plant being fertilized by the wrong pollen. By Mendel's own admission, his experiment is carefully designed to make it possible to reach his results. This illustrates that good experimental science is not simply about observing the natural world. Often, the relevant phenomena and regularities need to be teased out by the scientist, in this case by eliminating interfering natural processes like chaotic fertilization, the development of sterility in offspring and the obtuseness or invisibility of traits.

Even when the real experiments begin, Mendel doesn't take every novel observation to be theoretically relevant. For example, he writes that "in the case of one sort, some considerably deviating forms were noticed among a larger number of like plants. These did not vary however in the following year, and agreed exactly with another species derived from the same seed shop; undoubtedly, the seeds had been admixed purely by accident." So Mendel also had to discard irrelevant and misleading observations made during the experiment, again showing how scientific observation requires practical and theoretical knowledge. This also shows that the three problems Mendel outlines can also serve as explanations for (theoretically) unexpected results. The fact that certain offspring take erratic forms is not a refutation of Mendel's ratios, rather it can be explained away by appeal to the idea that foreign pollen was introduced.[7]

The parents of hybrids possess (at least) two different traits of the same type, such as round and wrinkled pods in Mendel's experimental peas. Mendel's aim is to discover the rules that govern the inheritance of traits in the hybrid offspring, and his initial results show that some of his (carefully selected) traits reappeared in the next generation, while the rest did not. Mendel categorizes the former as dominant and the latter as recessive traits, then proceeds to show that they appear in fixed ratios in the offspring. So the initial experiments

7 And indeed the second scenario seems much more likely given the other reasons he gives in the quote. Mendel argues that the strange traits agreed with those of other plants from the same storage area, and since the deviating forms were shown not to be hybrids (ie. They were constant forms), they most likely stem from the experimenters using the wrong seeds.

motivated the introduction of a distinction, which is then useful for categorizing and making sense of the final experimental observation.

Mendel's first set of experiments aim to show that, despite the fact that recessive traits disappear entirely in the initial hybrid offspring, the children of the hybrids posses the dominant and recessive traits in an average ratio of 3:1, while there are no intermediate forms between the recessive and dominant traits (Mendel, 2016: 12). Mendel derives the approximate ratio for a number of different pairs of traits, showing that the same ratio can be reached by a number of different experiments. The different methods of reaching the ratio lead to approximately the same result, as can be seen from the first three trials:

"1st trial: Seed shape. From 253 hybrids 7324 seeds were obtained in the second year of the experiment. Among these, 5474 were round or roundish and 1850 were angular and wrinkled. This results in a proportion of 2.96 : 1." (Mendel, 2016: 12)

"2nd trial: Colouration of the albumen." yellow and green, "stand in a proportion of 3.01:1" (Mendel, 2016: 12)

"3rd trial: Colour of the seed coat." violet-red flowers and grey-brown seed coats, "gives a proportion of 3.15 : 1" (Mendel, 2016: 14)

The average of all these experiments, "2.98 : 1", is very close to the theoretical parameter of "3 : 1", showing how successful Mendel was at deriving his theoretical ratio from the experimental results.[8]

Mendel proceeds with the same method in the next generation, although his results are different. Those plants which exhibited recessive traits in the first trial passed this character on to their descendants when bred amongst each other, as did a third of the dominant character exhibiting plants of the first generation (so a quarter of the plants of this generation). However, approximately two thirds of the plants with the dominant characteristics once again manifested a 3 : 1 ratio of the dominant and recessive traits in their descendants, as Mendel demonstrates with the same kind of procedure as earlier (Mendel, 2016: 15p). This pattern continued for the children of the hybrid forms (those that were neither constantly recessive nor dominant), leading Mendel to conclude that "in each generation, the descendants of the hybrids divided themselves in proportions of 2 : 1 : 1 into hybrid and constant forms" (Mendel, 2016: 16). The constant forms are those which, when bred with each other, continue to produce offspring with the same recessive (or dominant) trait.

At this point in the paper Mendel is ready to introduce his theoretical nomenclature: "If A denotes one of the two constant traits, for example the dominating one, a the recessive one, and Aa the hybrid form in which both are united, then the expression A + 2Aa + a yields the developmental series for the progeny of hybrids with two differing traits each." (Mendel, 2016: 17) This developmental series, A + 2Aa + a, is a mathematical representation of the distribution of hybrid and constant forms in the second generation of offspring. The series A + 2Aa + a means that constant dominant (A), hybrids (Aa) and constant recessive (a) forms appear in a 1 : 2 : 1 ratio. This mathematical representation

8 In fact Mendel was so successful that many have claimed that Mendel's results were unlikely enough to warrant allegations of fraud. Here I will assume the standard view that Mendel did not consciously adjust his data (Radick, 2015: 160), although that is not entirely uncontroversial.

allows Mendel to neatly summarize his results for numerous generations (and also to generalize for arbitrarily many generations). Mendel's tabulation leads to the following ratios, for a given generation n: (2^n) - 1 : 2 : (2^n) - 1, where the first and last columns represent A and a, while the middle column stands for Aa. Mendel can therefore use his developmental series to predict the relative occurrence of hybrids in future generations, and to model the inheritance of traits in successive generations. However, it is again clear that this doesn't come without its share of caveats, such as that fertility doesn't change and that all of the plants produce the same number of offspring (Mendel, 2016: 17p). Given that Mendels' experimental set up was designed to tease out regularities while trying to avoid and control for all the distracting natural processes, Mendel's theoretical machinery can be seen as an idealized, or simplified, model of inheritance of traits in nature. Idealization occurs when scientists make simplifying or false assumptions in their theories or models. In this case, Mendel is ignoring various phenomena, precisely because he knows that they have nothing to do with inheritance, the process he is interested in. For example, young plants might not manifest their traits because they are ravaged by insects, but cases like these are correctly ignored in Mendel's theory. So one virtue of idealizing like this is that it can single out the kinds of phenomena, or processes, the scientist is interested in studying or theorizing about. Another advantage of idealizing like this is that it simplifies the task of mathematically modeling the phenomena. And as we have seen, the mathematics of combination series is very helpful not just for representing the ratios of hybrids and constant forms, but also for generalizing results, or for deducing which ratios should arise. For example, we could use the combination series (2^n) - 1 : 2 : (2^n) - 1 to deduce the the ratio of hybrid and constant forms at any given generation. So Mendel's idealization allowed him to introduce quite a helpful and powerful piece of mathematics.

We have seen so far how Mendel's work has been purposefully specific, for example by trying to isolate single, easily identifiable traits. In doing so, Mendel initially sidelined problem cases where he could not easily identify traits, for example where they are not clearly demarcated or difficult to detect. Concerning these cases, Mendel writes that "the perfect concordance that all the characteristics subject to the experiment show certainly permits and justifies the assumption that the same behavior would also hold for those remaining traits that do not emerge as sharply pronounced in the plants and could therefore not be admitted to the individual trials" (Mendel, 2016: 24). So for Mendel, his success in some specific cases gives a good reason to expect that his theory can be applied to other cases where the ratios do not emerge as sharply. I think the idea here is that the prior success of the theory permits the assumption that experimental difficulties, and not Mendel's theory, are to blame for the less conclusive results from other experiments.

Having thoroughly investigated Pisum, Mendel turns his attention to less straightforward cases. In dealing with other plants, Mendel would show that his theory has the potential to be universally applicable. However, as Mendel indicates in the above quote, there are obvious practical difficulties presented by the fact that not every plant is suited for this kind of experiment, and so Mendel's three problems arise and make the accurate determination of his ratios more difficult. That being said, the issue with these cases is more about experimental accuracy, and Mendel's theory need not be changed to overcome them. However, there is another case Mendel considers that requires a more theoretical approach.

Mendel recounts how, in the experimental plant type Ph. multifloras, colors were inherited in a pattern unlike the 3:1 ratios: "With respect to the colour traits it seems difficult, however, to reveal some sufficient agreement. Apart from the fact that from the conjunction of white and purple-red colouration a whole range of colours from purple to

pale-violet and white emerges, the circumstance must also attract attention that among 31 flowering plants only one possessed the recessive white characteristic, whilst in Pisum this is the case fourth plant on average already" (Mendel, 2016: 34). So there were multiple hybrid forms in multifloras, and the recessive white color was much rarer than it would have been in Pisum.

This is clearly a different kind of problem case for Mendel; one that can't be explained away by appeal to experimental difficulties. To show how it could be solved, Mendel requires some theoretical finesse, but in so doing indicates how his theory has the resources to turn apparent problems into theoretical successes. A substantial part of Mendel's paper is devoted to experimentally showing that the inheritance of multiple kinds of traits can be accounted for by multiplying his developmental series. Recall that Mendel uses the expression "A + 2Aa + a" to represent the inheritance of two traits (A is dominant, a recessive) of the same kind. Suppose that there is another pair of traits of a different kind represented by "B + 2Bb + b". Then if we cross two plants, one containing A and B and another with a and b, the hybrid offspring will inherit in ratios given by the expression obtained by multiplying both expressions: "A + 2Aa + a" x "B + 2Bb + b" = "AB + Ab + aB + 2ABb + 2aBb + 2AaB + 2Aab + 4AaBb" (Mendel, 2016: 21). Similarly, for three pairs of traits three simple series are multiplied (Mendel, 2016: 22). Mendel supported these ratios with experiments earlier in his paper, and now he can employ his theoretical mathematics to provide a possible explanation for the difficult problem case.

He begins his solution with "these puzzling phenomena might probably be explained according to the law that is valid for Pisum, if one were allowed to presuppose that the flower and seed color of Ph. multifloras was composed of two or more entirely autonomous colours, each of which behaves individually in the same way as every other constant trait in the plant" (Mendel, 2016: 35). He proposes that the colour "A" (purple-red) of the first parent flower is actually made up of multiple independent traits "A1 + A2 + …". The recessive white color of the other parent is given by "a", meaning that the hybrids developmental series are given by the multiplication of the following two terms:

"A1 + 2A1a + a" and "A2+2A2a + a"

In the term obtained by said multiplication, only one of 16 possible combinations features the "aa" term required for the recessive trait to manifest. If we add a third trait in the purple-red flower (A3), then only one in 64 hybrid offspring will manifest the recessive white color (Mendel, 2016: 35). So Mendel can explain, using his developmental series, why some recessive traits manifest themselves quite rarely. This is the start of an explanation of the ratios of colors of Ph. multifloras, although Mendel would require further experiments (which he does not provide in the paper) to show that the color traits of the flower really do follow the patterns he stipulates. Even though Mendel does not finish said investigation here, it is clear that his theory has resources to try to explain more complicated cases like these, even though they might initially have appeared to easily refute his theory.

Mendel's interests appear to extend to the composition of the reproductive cells. He aims to argue that germ and pollen reproductive cells are not different, and that there are precisely as many reproductive cells "as constant combination forms are possible" (Mendel, 2016: 24). For example, let us consider the standard Mendelian case where there are two constant forms. Then Mendel claims that there are two distinct reproductive cells in each reproductive organ responsible for these traits. So Mendel claims that there are two separate factors in each reproductive organ, with two of the same kind leading to a

constant form.[9] Therefore Mendel's paper is not just about regularities and combination series, but also involves the claim that there are heritable units (later known genes or Mendel's factors) responsible for these patterns of inheritance.

By this point we have seen that Mendel carefully idealized away from irrelevant observations in his experimental setup. We also saw that he derived his ratios for the dominance and recessiveness of multiple different traits, then introduced a neat mathematical representation of his findings with his combination series. Manipulation of these combination series allowed Mendel not just to summarize and generalize his results, but also to try to explain away seemingly problematic cases. Careful experimental design, idealization, mathematical representation and the ability to turn apparent counterexamples into theoretical successes are all hallmarks of good scientific technique. So I think we can attribute some of the Mendelian successes described above to the good (but not infallible) scientific approach of its founder.

Now that we have a good understanding of what Mendel achieved in his paper, we have to ask which components of Mendel's theorizing were particularly influential, and which were quickly dropped or amended by later Mendelians. I think we can now distinguish two very important theoretical elements of Mendel's theory. The first is Mendel's view that traits can come in dominant and recessive pairs. The second is the idea that the inheritance of traits can be modeled by, or thought of as, the inheritance of particles which control the appearance of traits. In other words, there are particulate factors responsible for the inheritance of traits. While these views fit together very nicely, they are reasonably independent from each other. For example, one can think that one trait is dominant over another even if they arise from (or are best understood by) a mechanism very unlike the particulate, factorial model.

My claim is that the dominant-recessive distinction was often strongly adapted or weakened by later Mendelians, and that the particulate model was Mendel's most influential and enduring contribution to genetic theory. To see why this is the case, allow me to provide a few important passages from "the mechanism of mendelian heredity", a work cowritten by Morgan, one of the most important experimental Mendelian geneticists. The great innovation that Morgan et al build on is the idea that chromosomes are carriers of Mendel's particulate factors, which was first suggested by Sutton in 1902 (Martins, 1999). The addition of chromosomal reasoning helped expand on the particulate model and calculus introduced by Mendel, as we can see in the following examples.

Both examples from the book involve a kind of phenomena which the addition of chromosomal theorizing helps explain. Bateson and Punnett's 1906 experiments showed that certain Mendelian factors tended to be inherited together (Morgan et al, 1915: 5). Morgan et al explain this observation by arguing that the factors were located on the same chromosome. So the observation that various traits are frequently inherited together is explained by the organization of the Mendelian factors in the chromosomes.

The second example is about sex linked traits. Amongst fruit flies, red and white eyes can be inherited in more complicated patterns than the standard 3:1 ratios. When (pure bred) red eyed females and white eyed males are bred, their grandchildren exhibit red and white eye colors in a 3:1 ratio. Of those grandchildren, two out of three red eyed flies

9 See pages 24p of the BSHS translation of "Experiments on Plant Hybrids" for Mendel's argument for this conclusion

will be female. The other is male, and will always be a hybrid form with both red and white factors. Why is this the case? There is precisely one kind of Y chromosome, one that contains the white eyed factor, in the population. This means that males can only come in hybrid forms, or with two factors leading to white eyes. This means that red eyed fruit flies in the populations must be hybrids, which explains the observation that, when any red eyed male is bred with a pure bred white eyed female, the grandchildren will have red and white eyes in a 1:1 ratio. So the addition of chromosomal reasoning to the particulate theory helps explain why certain configurations of traits cannot arise in individuals, and why it is impossible to generate the 3:1 ratio with a red eyed male fruitfully in this population (Morgan et al, 1915: 16pp).

However, these developments in no way invalidated or sidelined the particulate model or the factorial mathematics. If anything, the improvements made by Morgan et al help the particulate model and the factorial mathematics account for more cases successfully. So the modifications to Mendel's particulate model and his factorial systems in no way diminished their importance. On the other hand, Mendel's views on dominance where quickly qualified. Consider the following passage to see that Morgan et al don't think of much Mendel's conception of dominance:

"Whether a character is completely dominant or not appears to be a matter of no special significance. In fact the failure of many characters to show complete dominance raises a doubt as to whether there is such a condition as complete dominance. Some cases approach so nearly to that condition that special tests may be required to show that the hybrid is affected by the recessive factor. For instance, in flies the factor for white eyes seems to produce no effect when white is bred to red. The F1 reds are indistinguishable from pure reds. But by weakening the red by adding recessive factors other than white, the influence of white can be demonstrated, as Morgan and Bridges have shown. Therefore although the effect of the white factor can not be detected in the single combination with red, it is reasonable to suppose that some effect is really present. Similarly, conditions were found in which the effect of heterozygosis for eosin, vermillion, or pink could be demonstrated. While the question is one of only subsidiary importance, yet in the separation of classes it is often useful to able to distinguish the pure from the hybrid form; but whether this can or can not be done in any given case does not affect the fundamental principle of segregation which is the essential feature of Mendel's discovery." (Morgan et al, 1915: 31p)

So here we see not only a denial of the view that all dominance is as Mendel described it as, but also an argument to conclude that there are no cases of dominance so complete that they fit Mendel's description. Furthermore, in Morgan et al's view the fact that dominance per se might not be detectable would be not be a theoretical problem for the Mendelians. Instead, these Mendelians were clearly much impressed with Mendel's factorial scheme and the particulate model for genes. This is reinforced by further discussions in the chapter, which highlight relations between genes and traits other than dominance. This includes not only cases where the intermediate state between a pair of genes is much more messy than the dominant relationship described by Mendel, but also the idea that a single factor, or gene, can cause a number of different traits.

So Mendel's view that inheritance is controlled by particulate factors proved to be more central to later Mendelian thought than the discovery of dominance. Although dominance was certainly not irrelevant to Mendelian genetics, Mendel's views on this topic were weakened by later Mendelians. In fact Morgan et all seem to view (incomplete) dominance

as just another phenomena, without any claim to special theoretical importance. The factorial, particulate model, on the other hand, enjoyed a far more prominent status. For this reason, I like to follow Fisher in thinking of Mendelism as the particulate theory of inheritance.[10] I think this characterization of Mendelian theory will be helpful in assessing whether constructivism or realism can help us understand the success of Mendelian genetics and its proper role in the synthesized theory.

10 More precisely, Fisher claimed that "In the future, the revolutionary effect of Mendelism will be seen to flow from the particulate character of the hereditary elements." (Fisher, 1930: ix)

Chapter 3: The Controversy about the Controversy

After Mendel published his work on hybrids, it was initially not very well received. However, at the start of the twentieth century it suddenly became popular amongst British biologists (Radick, 2016: 154). The upsurge of interest in Mendel led to a dispute between the early Mendelians and the biometrical school of biology. For Radick, this dispute can largely be understood as a clash between two historically important personalities: William Bateson, the most prominent early defender of Mendelism, and Raphael Weldon, a leading biometrician who was trying to develop a rival theory of inheritance to Mendelian genetics (Radick, 2016: 54pp). According to Radick, this dispute could have ended with either a Mendelian or a biometrical victory, and the winners were decided by contingent circumstances instead of rational reasons (Radick, 2016: 157). That is not to say that there were no rational reasons in play, but rather that none of them were decisive. Instead historical and social factors, like Bateson's ability to persuade researchers and breeders of the value of Mendelian genetics and Weldon's untimely death, determined the outcome of the controversy (Radick, 2016: 157). To understand Radick's position, we first need to understand his view of the history of the controversy.

As Radick explains, Mendel's paper suddenly became popular in 1900 (Radick, 2016: 145). William Bateson and Raphael Weldon, two British biologist who had studied at Cambridge together, both became aware of this paper (Radick, 2016: 154). Bateson was quickly convinced of the importance of Mendel's work, and recruited allies to help him continue Mendelian research. Weldon instead had established himself as a leading biometrician, and had helped found "Biometrika", a journal dedicated to "the statistical study of biological problems" (Radick, 2016: 154). Weldon quickly became the most prominent critic of Mendel and Bateson (Radick, 2016: 154).

Biometry was a school of biological thought that originated from the work of Francis Galton, and was championed by Weldon and Pearson by the start of the 1900s (Radick, 2005: 34). The biometricians were interested in pursuing "the statistical study of inheritance at the level of the population" (Radick, 2005: 34), and for Radick the best example of this is given by a 1893 collaborative paper from Pearson and Weldon (Radick, 2005: 35). They investigated various traits of female shore crabs, and showed that the traits in the population were distributed into bell curves, and the only exception could be analyzed with two bell curves (Radick, 2005: 35). Lastly, Weldon and Pearson showed that the variation of certain features where statistically correlated with variations in other features (Radick, 2005: 35). So the defining feature of biometrical research was that it involved the use of statistics to analyze inheritance in biological populations. This is similar to Mendel's experiments on hybrids, since an empirical investigation led to the introduction of a useful piece of mathematics. However, this work differs from Mendel's work on hybrids in that none of these analyses suggested a view of inheritance. While not every Mendelian thought that there really are heritable factors, or genes,[11] many did think of Mendelian genetics as showing what the heritable material was really like. On the other hand, Weldon and Pearson's work on the crabs doesn't have any possibly realist implications.

11 Consider for example Morgan's comment in his 1934 Nobel Lecture that "There is no consensus of opinion amongst geneticists as to what the genes are – whether they are real or purely fictions – because at the level at which the genetic experiments lie, it does not make the slightest difference whether the gene is a hypothetical unit, or whether the gene is a material particle." (Morgan, 1934: 315)

The same is true for Galton's law of ancestry, the most important theoretical claim of the biometricians. Galton thought that ½ of the heritable material of a child came from its parents, a ¼ from its grandparents, a ⅛ from its great-grandparents, and so on (Radick, 2005: 35). Of course, the sequence ½ + ¼ + ⅛ + … tends to 1, meaning that Galton could account for the entirety of heritable material in the child (Radick, 2005: 35). As Radick correctly notes, this law of ancestry did not make any claims about the nature of inheritance either (Radick, 2005: 35p).

So unlike for Mendelian genetics, the exemplary early work in biometry was silent on the nature of inheritance. According to Radick, Pearson was not bothered by this due to his positivist philosophy of science, since he felt that only the law's ability to predict observations was relevant (Radick, 2005: 36).[12] However, Weldon and Galton disagreed, and Weldon sought to remedy the situation by developing his own theory of inheritance to provide a "mechanism … of heredity" (Radick, 2005: 36). Unfortunately, Weldon died unexpectedly of pneumonia in 1906, and so was unable to finish his book on inheritance (Radick, 2005: 37). So it will never be entirely clear what Weldon's theory of inheritance, or a science based on his work, would have looked like. However, Radick does explain some interesting features of Weldon's views of inheritance, which can give us a broad, if imprecise, understanding of what Weldon thought about inheritance. Weldon believed that there were 'determinants', located in the chromosomes for higher animals, which were responsible for the inheritance of traits. Weldon "insisted on the influence of the chemical and physical environments on the form of hereditary characters. He also envisaged a kind of contest among the chromosomal determinants. In general, the more active or vigorous an individual determinant, the greater its share in the character. At the extreme, one determinant could dominate wholly" (Radick, 2005: 37).

As Radick recounts, Weldon had a number of interesting objections to Mendelism. Weldon was the first to argue that Mendel's experimental data was, statistically speaking, suspiciously good. Of course, Radick is aware that most modern historians do not think that Mendel faked his data (Radick, 2015: 160). Furthermore, even if these allegations reflected poorly on Mendel, that would not necessarily discredit Mendelism. That being said, Weldon developed other, more interesting objections against Mendel's theory.

Weldon examined peas just like Mendel's, though he suggested that Mendel was mistaken to treat them as either being green or yellow. Instead, Weldon suggested that there were various intermediate colors, such as yellowish green (Radick, 2016: 155). So there was a continuum of colors, which Mendel illegitimately ignored. Weldon and Radick conclude from this that the Mendelians simplified away from the complexity of real variations – that pairs of traits were just convenient, but misleading, fictions for the experimental Mendelians (Radick, 2016: 156).

Weldon's second major criticism of Mendelism is that it ignored the law of ancestry (Radick, 2016: 155). Remember that Galton's law states that the parents contribute one half of the heritable material in the child, the grandparents one quarter, the great-grandparents one eight, and so on (so that ½ + ¼ + ⅛ + … tends to 1). As we have seen, Mendel, and the particulate theory of genetics, sought to account for the traits of the child purely through

12 I suspect that Pearson instead had a methodological preference for doing statistical analysis before searching for a material explanation, as is indicated by the following quote from Pearson: "The numerical laws for the intensity of inheritance must first be discovered from wide observation before plasmic mechanics can be anything but the purest hypothetical speculation" (Froggatt et al, 1971: 8)

the genetic material of the parents. In other words, Mendelians thought that half of the heritable material came from the mother and half from the father. So Mendelism and the particulate theory of genetics are incompatible with Galton's law of inheritance, and so Weldon, as a follower of Galton, saw this as an excellent reason to reject Mendelism.

Lastly, we also know that Weldon felt that environmental effects could influence which traits were expressed. Furthermore, different hereditary elements could affect each other, leading to different visible traits. Evidently both Weldon and Radick thought of this stance as incompatible with Mendelism (Radick, 2016: 155p).

Radick's conclusion from these three arguments is this:

"The upshot for Weldon was that the Mendelian picture of 'dominance' as absolute – so, for example, of yellowness as dominant to greenness, no matter the context – was deeply misleading. In Weldon's view, to run a Mendelian experiment was deliberately to exclude all of the variability that would otherwise produce different kinds of pattern. If an experimentalist were so minded, he thought, a race of peas could be established in which greenness was dominant to yellowness. It all depended on the choices made, the contexts built. And to declare one pattern the natural one, and the other patterns as somehow deviant, was just arbitrary. No, what biologists needed was a concept of dominance which treated it as context-dependent" (Radick, 2016: 156).

So for Radick, Weldon's objections show that Mendelians were unjustifiably dogmatic. In particular, Mendelians' ignorance of context led them to believe that traits were caused purely by the effects of genes. In other words, Mendelians, according to Radick, thought that genes alone determined traits – and so were genetic deterministes. I think that, for Radick, this dogmatism, and genetic determinism, pervade the history of Mendelian genetics, as I think can be seen from the following quote:

"From early days, Mendelism and a kind of simple-minded determinism travelled hand in hand… During the Great War, Pearson kept a diary in which he recorded an encounter with a Mendelian. On Pearson's exasperated account, the latest hypothesis from this permanently misguided fellow was that 'music in the Welshman was a "Mendelian recessive"'. That kind of reductive determinism incensed Pearson." (Radick, 2016: 165)

This humorous story of Pearson's is supposed to show how Mendelians naively thought they could explain (nonexistent) traits purely by reference to a single gene. This was especially bad, because genetic determinism, while refuted by modern genetics, continues to pervade popular thinking about genetics:

"In our day, textbook Mendelian-determinism continues, entrenched. At the the research frontier in biology and bio-medicine, meanwhile, interaction is where the action is at." (Radick, 2016: 165)

As we will see in our later discussion of realism and modern genetics, Radick is right to claim that modern geneticists, like Weldon, focus on the interactivity of the genetic material.[13] He is further correct in pointing out that genetic determinism is an unfortunate

13 As far as I can tell, Radick doesn't infer from this truthful element of Weldon's theory that it would have been a real, or superior, alternative to Mendelian genetics. Suffice to say that I don't think this inference would be very good.

legacy of Mendel's success. However, I think this a legacy we should probably blame modern science educators, and not now outdated but previously successful science, for.

So Radick's view is that there were plenty of rational reasons in favor of Weldonian biometry, and that it could have led to a fruitful, alternative history of biology. Weldon's project failed to take off, not because Mendelian genetics had scientific advantages over it, but due to contingencies like Weldon's unfortunate early death. Furthermore, it seems to me that Radick often suggests that it would have been better if Weldon's biometry had succeeded, because, for example, genetical determinism would have been, both presently and historically, less popular.

My response to these criticisms, made by Weldon and endorsed by Radick, is that, even if they might be true of Mendel, they cannot be fair criticisms of the entirety of Mendelism. As we have seen in our discussion of Morgan et all, Mendelism is best described as the particulate theory of inheritance. This is because the idea that units of inheritance are, or are best described by, Mendelian factors proved much more influential and important than Mendel's views on dominance. I will argue that a Mendelian who has read Morgan et al's book, published only nine years after Weldon's death, would have been entirely able to respond to Weldon's arguments. This would mean that the disadvantages of Mendelism that Weldon highlights were transient, and so that these were not rational reasons to prefer Weldon's possible alternative over Mendelism (at least when we allow the theory enough time to be developed). Furthermore, I will show that later Mendelians actually agreed with Weldon on a number of issues raised in these arguments. This suggests, rather surprisingly, that the Weldonian alternative would not have been as distinct from Mendelism as Radick suggests.

First let's consider the last of Weldon's objections, that outside environments and multiple genetical elements can affect which traits arise. To see how this criticism fails to refute Mendelism, let's look at the following passage from Morgan et al's "The Mechanisms of Mendelian inheritance":

"It would have been indeed strange if Mendelian factor-differences had not been found that require special conditions – environmental, developmental, or factorial – in order to produce a given effect, or any effect at all. For Mendelian factors may cause or influence all sorts of characters – that is, any or all kinds of developmental or physiological reactions; and many of these reactions are known to be affected by age, temperature, region of the body, and so forth. The facts given above are in no possible sense subversive to Mendelian principles. On the contrary they illustrate to great advantage the previously given interpretation of all hereditary characters – namely, that every character is the realized result of the reaction of hereditary factors with each other and with their environment. Failure to understand this viewpoint has led to some futile criticism by the opponents of the modern mendelian interpretation in terms of unit factors. This criticism is as pointless as it would be to criticize the atomic theory on the ground that oxygen does not, under all conditions, and in all compounds, give rise to substances with the same properties." (Morgan et al, 1915: 46)

So it appears that Morgan et al are in complete agreement with Weldon about the importance of external and genetic environments, which is rather puzzling if we are to conclude with Radick that there were rational reasons to doubt Mendelism. Furthermore Morgan et al, with their recognition of the importance of environmental and developmental conditions, can hardly be accused of being genetic determinists. Again we can safely say that Weldon's

criticism can fairly be made of Mendel, without it having to apply to a more developed Mendelian or particulate theory of genetics.

The first criticism is similarly overstated. Remember that Weldon complained that real peas did not look like Mendel's peas insofar as there was a continuum of colors between green and yellow. Recall also that Morgan et al emphatically rejected the idea that all traits are dominant in the strong sense championed by Mendel. Furthermore, we would have to account for the developmental factors that Morgan et al (and Weldon) were cognizant of, and also of the experimental difficulties and impurities that Mendel was so wary of. It is possible that, once all of these are accounted for, Weldon's peas represent a counterexample to Mendel. But we should also acknowledge that the Mendelians were far less simplistic than Radick implies, and they had far more resources at their disposal to deal with these difficulties than Radick suggests. Because, as I will argue later, the particulate theory is false, I cannot hope to argue that there are no counterexamples to it. What I can argue is that there were rational reasons, arguments, experiments and theoretical successes in favor of Mendelism. We should also note here that many of the later Mendelians shared Weldon's desire to a find different concepts of dominance, which were easier to reconcile with the facts than Mendel's stricter conception of dominance. Once again it seem like Weldon and the later Mendelians were not as different as Radick suggests.[14]

I've left the second criticism for last, and that is because I think it is by far the weakest. Galton's law of ancestry was abandoned both by Mendelians and later schools of genetics both because of its falsehood, but also, I think, because it wasn't especially empirically helpful. The Mendelians were able to do much more with the correct assumption that the parents together contributed all of the heritable material in the child. So I think Weldon's observation that Mendelian genetics is incompatible with Galton's law need have troubled the Mendelians, unless there was some particularly good piece of evidence for Galton's law. But, as far as I know, neither Radick nor Weldon have provided a compelling argument for Galton's principle, and so I think we can dismiss this second argument.

If you're keeping score at home, that's at most ½ out of 3 in favor of Weldon. So later Mendelians, like Morgan et al, were certainly in a position to respond to Weldon. I also think that the Mendelians managed to explain the biometricians' data using their theory of inheritance. For example, recall the biometricians' greatest success: the introduction of bell curves (and the accompanying statistical analysis) to the biological description of populations. Remember that Radick explained the importance and results of Pearson's and Weldon's observations of the traits of female shore crabs, which could be shown to appear in distributions best modeled by bell-shaped curves. In the following quote, Fisher argues that Mendelians could account for bell curved distributions of traits in populations (in this case using the example of human height):

"The biometrical facts as to the inheritance of stature and other human measurements, though at first regarded as incompatible with the Mendelian system, have since been shown to be in complete accordance with it, and to reveal features not easily explicable on any other view. The approximately normal distribution of the measurements themselves may be deduced from the simple supposition that the factors affecting human stature are approximately additive in their effects. The correlations found between relatives of different degrees of kinship are, within their sampling errors, of the magnitudes which

14 Of course not all Mendelians were this critical of the concept of dominance, and Weldon's complaints would have been fair critiques of those Mendelians.

would be deduced from the assumption that the measurement is principally determined by inheritance, and the factors controlling it show, like most Mendelian factors, complete or almost complete dominance. The presence of dominance is a Mendelian feature, which is shown in the biometrical data by the well-established fact that children of the same parents are, on the average, somewhat more alike than are parents and offspring." (Fisher, 1930: 17p)[15]

So the Mendelians managed to explain biometrical phenomena using their theory's resources. The ability to turn apparent failures of the particulate theory into successes provides a strong empirical reason for favoring it. In the section on Fisher, we will see other, more complicated, examples of theoretical and empirical success. These successes are especially impressive because it seems in no way guaranteed that the Mendelians could have found theoretical explanations for these phenomena.

In fact, we needn't even think of Mendelism and biometry as necessarily opposed to each other. Other than Weldon's unpublished work on inheritance, the biometricians did not really provide theories of the material basis of inheritance. Galton's law and bell curves do not explain what the heritable material is – instead they make statistical predictions. And Mendelian genetics, which was a theory of the heritable material, could make excellent use of good statistical techniques (even if Bateson did not appreciate this fact). During the controversy, Pearson considered the possibility that Mendelian genetics and ancestrian biometry might be made compatible with each other. One of his suggestions was to drop the Mendelian concept of dominance (Froggatt et al, 1971: 20p), the same move that we saw Morgan et al make. So ultimately a continuation of the conflict might have followed Pearson's path, with a conscious synthesis of both schools of biology.

So really the arguments Weldon had against the Mendelians might have had some bite against Mendel, but not much against later, more refined versions of Mendelism, which abandoned the strict concept of dominance. This story also ignores many later Mendelian theoretical and experimental successes, not least their explanation of the bell-curves introduced by the biometricians. This strongly suggests, I think, that Radick has not shown that the rational reasons favored Weldon, or even that they were in the balance. Furthermore, there were reason to doubt the ancestral aspect of Weldon's theorizing, while he agreed with later Mendelians on a number of issues. This might bring us to doubt that Weldon's work really could have been as distinct an alternative to Mendelian genetics as Radick suggests. For all of these reasons I am skeptical of Radick's constructivist story.

It seems to me that the main weakness of Radick's current work on the topic is that he doesn't really touch on scientific developments after the controversy. Certainly there are many interesting elements to the controversy, which Radick brings attention to. However, many of the features of the debate, its sciences and its participants, were transient in nature. For example, Bateson was not very mathematically inclined, but Fisher, one of the greatest statisticians of all time, was a Mendelian. We also saw how many of the theoretical elements of Mendelism that Weldon and Radick criticized were also transient. Our discussion of Mendel and Morgan, and our conclusion that we should think of Mendelism as the particulate theory of inheritance, allowed us to appreciate how criticisms of Mendel need not reflect badly on Mendelism. Perhaps there are good arguments available to Radick

15 It is worth noting that Fisher views dominance much more favorably than Morgan et al, and so would have been more susceptible to Weldon's criticism about dominance. Perhaps we can attribute this difference to Morgan et al's greater experimental knowledge.

that take the later history into account. But in the absence of explicit discussion of these later arguments, I think we should conclude that the rational reasons did not appear to be balanced or in favor of Weldonian biometry.

So far I have argued that Mendelian theory had resources to defend itself from Weldon's criticisms. Since these argumentative resources would have been available within a decade of Weldon's death, we must assume that, for Radick's alternate biometrical history to have taken place, Weldon and his allies must have won the debate decisively and quickly. Otherwise it stands to reason that the debate might have continued for a while, until the Mendelians developed good counterarguments. Since we have seen that those counterarguments exist, and that the greatest strength of biometry, its statistical methods, could be used by Mendelians just as well (if not better – after all the Mendelians managed to explain the bell-curves using their theory of inheritance), it seems to me that the Mendelians would be in a better position to win the debate.[16]

With those criticisms out of the way, I want to end our discussion on a positive note. I think the biometricians effectively championed the trend of mathematizing biology with new statistical techniques. This trend continues, as we will see, strongly in Fisher's work, and mathematics continues to be an important component of modern evolutionary and genetic biology. So I think that the biometricians deserve high praise for their pioneering statistical approach.

16 To refute my argument, I think Radick would either have to show that there are good reasons to think that the biometricians could have won over the long term, or good reasons to think that the debate would have ended decisively and quickly. I further wonder why Radick does not think that the synthesis between Mendelian genetics and biometry, as Pearson envisaged it, was not a possible outcome.

Chapter 4: No Miracles and Idealization

In the previous chapter we considered the most interesting and developed constructivist history of Mendelian genetics. Now I think we should turn to the question of whether realism can account for its success. As with constructivism, we will be considering both a more naive and a more sophisticated view. First we will look at Putnam's "No Miracles" position. Afterwards, we will consider whether realism can be made compatible with the apparent falsity of the particulate theory by a sophisticated view of idealization. Here our discussion of will be aided by the work of Michael Weisberg and his paper "three kinds of idealization". To evaluate any kind of realist position on genetics, we need to know, or at least have some idea of, what a true theory of genetics looks like. To do so, we would have to look at contemporary practice and theory of genetics, and here we will rely on the work of historian and philosopher of genetics Evelyn Fox-Keller. I will use her most recent papers on the subject to explain why the particulate theory of genetics is not true (at least according to modern genetics). It follows of course that a false theory can be successful, leading to either a qualification or a refutation of the no miracles view. Next we will see how Weisberg's realist view of idealization is also not helpful in discussing this case. I also want to try to diagnose some problems of Weisberg's views. After this section, we will have concluded our discussion of views I want to criticize. At that point I want to examine Fisher's work in the synthesis as an interesting and important case of scientific success – both for Mendelian genetics and Darwinian evolutionary theory.

Putnam's view is that it would be miraculous if a false theory reached a sufficient level of scientific success, where we should understand a miracle as an extremely unlikely event. This extreme unlikelihood of false, but successful, scientific theories makes it highly probable that theories reaching a certain level of success are true. So the no miracles position implies that, practically speaking, successful theories are very likely to be true. This also means that, most likely, theoretical entities of successful theories have to exist in the ways that the theory postulates that they do, since otherwise it would be difficult to make sense of the theory being true or approximately true. And it seems to me that the existence of genes is the only thing that realists and anti-realists about Mendelism could really disagree about.

So really Putnam's view means, for our purposes, that sufficient success means that the existence of theoretical entities is very likely. There are two ways that this view could fail to help us understand the history of Mendelian genetics. The first, obvious way is that it could be incorrect, and unfortunately philosophy has much less use for false views than science does. Secondly, it could be the case that Mendelian genetics never met the high standard of success required by the no miracles argument. This second reason is important to bear in mind, because Putnam's argument is quite vague in this way. However I think we shall see that Putnam's argument is unhelpful for our purposes, no matter how charitably we interpret the word 'success'.

The first question to answer is whether the particulate theory is true, or at least approximately true. If it is, then its success might indicate its truth. On the other hand, if the particulate theory isn't true, Putnam's theory will not be applicable to the current case. We will try to answer this question by briefly examining the state of contemporary genetics and comparing it with Mendelian genetics.

If the particulate theory is to be (approximately) true, then it needs to be the case that there are segregated units of inheritance responsible for the formation of traits. While the Mendelians thought that environmental and developmental influences could affect the organism's traits, these processes would not change the composition or arrangement of the heritable material. To see whether modern genetics suggests that this is the case, we will follow Fox Keller's analysis of the history, and current trajectory, of modern genetics.

Of course the Mendelians did not have a good understanding of what the genetic material was. As we have discussed, they theorized that genes were located in the chromosomes, but had little understanding beyond that. It was only much later that geneticists came to think of DNA as the genetic material. In 1943 Avery, MacLeod and McCarthy showed that DNA carried biological information in bacteria (Keller, 2002: 3). But it was Watson and Crick's 1953 experiment that "convinced biologists not only that genes are real molecules but also that they constituted of nothing more mysterious than deoxyribonucleic acid" (Keller, 2002: 3). This new scientific direction initially seemed quite consistent with the particulate theory. Scientists identified genes with protein-coding stretches of DNA (Keller, 2012: 134), and thought that they could explain all kinds of heritable traits by analyzing these genes. So at this point, modern genetics might look like a continuation of the Mendelian project. Having identified genes at the molecular level, biologists hoped to explain all heritable traits with them, much like how the Mendelians hoped to explain all heritable traits with particulate factors.[17] If this project continued to the present day, we might be inclined to think of the particulate theory as approximately true. However, as Fox Keller shows, modern research has sharply deviated from this understanding of inheritance.

Identifying genes with protein coding DNA was not without its problems. In 1968 it was shown that there were "large amounts of repetitive DNA" (Keller, 2012: 134), while there further appeared to be no correlation between "the amount of DNA in an organism and its complexity" (Keller, 2012: 134). Furthermore, it was demonstrated in 1977 that genes could be discontinuous – so that there could be apparently useless tracts of non-coding DNA inbetween the segments of DNA that made up a gene (Keller, 2012: 134). The upshot of these examples, among others, is that very many DNA sequences do not code proteins, and so a huge portion of DNA would not consist of genes (Keller, 2012: 134). The consensus response to this problem soon become that these tracts were "junk DNA" of little to no consequence for inheritance and variation. This phenomena of "junk DNA" was given an evolutionary explanation by appeal to Dawkin's concept of "selfish DNA", which simply propagated itself without contributing to the organism.

However, as Fox Keller explains, this way of thinking about DNA has been, since the 1990s, gradually supplanted by genomics (Keller, 2012: 136). The findings of genomics research showed that there were important genetical elements other than protein coding DNA stretches. Instead genomics focused on the entirety of the genome, so on the entirety of the DNA molecule[18]. As we shall see, genomics shows that modern genetics is incompatible with the particulate theory.

The first important result was given by the human genome project, which demonstrated that there are not nearly as many genes in the human genome as scientists had predicted

17 This analogy is a little inexact, but lets assume it for charity's sake.

18 As Fox Keller explains in her paper, "genes, genomes, and genomics", the word 'genome' has multiple meanings. However, I think that for our purposes we can just think of it as referring to the whole DNA molecule.

(Keller, 2012: 136). Genomics research further showed that most non protein coding DNA was in fact transcribed into RNA (Keller, 2012: 136). The resulting ncRNA (non-coding RNA, since it is transcribed from DNA that does not code proteins), was shown to be responsible for regulating how, when and if at all genes coded for proteins (Keller, 2012: 136). So a proper understanding of genetical material requires more information than which genes code for which proteins. Instead we also need to know about ncRNA that could regulate or otherwise act on these protein coding stretches of DNA. This means that the particulate model must be mistaken. There are no single kind of genetic entity responsible for an organisms' traits. Instead, there are coding stretches of DNA, non-coding stretches of DNA, protein coding RNA molecules and noncoding RNA, so four different kinds of entities, interacting with each other to give rise to traits.

Perhaps the realist might respond that, while there is no single genetic entity, we can think of the Mendelian gene as a placeholder for the more complicated physiological reality. And since these entities together behave sufficiently like Mendelian genes, we can think of the particulate theory as being approximately true. However, the obvious problem with this response is that the genetical material does not behave like particulate factors would. A good example is that gene expression can change based on environmental cues. So the actual composition of and behavior of the genetic material, and not just the traits of the organism, are responsive to environmental and developmental effects. Mendelian genes, on the other hand, are supposed to be atomic entities that are not themselves changed by said influences.

Gene expression can be altered by input from the nervous system. For example, it was observed that people who feel lonely or stressed are more susceptible to viral infections (Cole, 2009: 132). The genomic explanation for this fact was given by the observation that the genes of people who felt stressed or lonely were consistently expressed differently (Cole, 2009: 132). This explains the early onset of viral diseases particularly well, because viruses are parasites that hijack the cell's ability to reproduce, and have evolved together with our genome (Cole, 2009: 132). This example also shows that the genetic material is not atomic, and that it can be changed in response to cues from the organism's environment. But an organism's Mendelian genes are supposed to remain the same atomic units from its conception to its death.[19] So we can't think of particulate factors as placeholders for collections of many genetic entities, because the latter changes in response to environmental stimulus while the former does not.

So the particulate theory isn't true. There is no single kind of particle or genetic entity responsible for the inheritance of and variation in traits. So we can conclude that Mendelism's success did not guarantee its truth. Perhaps this was a miracle, but it is far more likely that either a) the theory was not successful enough to guarantee truth, or b) Putnam's view is mistaken. Either way, it is clear that Putnam's No Miracles argument is unhelpful for our purpose of understanding the success of Mendelian genetics.

But perhaps this is barking up the wrong tree. It is well known that scientific theories frequently involve falsehoods and simplifications. In other words, science involves idealizations, so distortions or untruths in theories and models. For example, physicists and applied mathematicians often discuss frictionless planes and bottomless oceans in their models, although neither of those things exist. Idealizations seem like an appropriate thing to investigate for two reasons. Firstly, idealization presents an obvious prima facie

19 With the possible exception of mutation as result of intense cellular damage.

problem for realists. If science routinely involves falsehoods, how can we say that science is about discovering the truth? So sophisticated versions of realism will have to take idealization into account, and sophisticated versions of realism are the most important for us to address. Secondly, it seems to me that we can think of Mendel as having practiced idealization. Recall that Mendel took extreme care to avoid what he felt were irrelevant observations. So Mendel's experimental design abstracts away from the complexity of traits in plants. And abstraction is a common rationale and justification for idealization. So there are good reasons to think that Mendels' work involves idealization, and if that is the case, then maybe a sophisticated realist view of idealization can explain how Mendelian theory could both be false and explained by a realist view of science.

Our sophisticated realist view will come from Michael Weisberg's paper "three kinds of idealization". According to Weisberg, we should not think of idealizations as properties of theories, but rather as a scientific activity or practice (Weisberg, 2007: 2). Weisberg thinks that there are three different kinds of idealization, corresponding to three different kinds of scientific practices. These practices have different aims, and therefore have different justifications. We will examine each to see if any of them can help us understand the success of Mendelian genetics in a realist fashion.

The first is Galilean Idealization. In this practice, the scientist already knows what a correct theory would look like. However, they are unable to make the necessary computations and calculations with a fully correct theory. So Galilean idealizers introduce simplifications and falsehoods into a model to be able to make the necessary calculations (Weisberg, 2007: 3). The justification for this kind of idealization is that it enables otherwise impossible computational procedures. Once these false assumptions are no longer necessary (because the scientists have access to better computational methods and devices), the scientists will remove the idealizations from their theory (Weisberg, 2007: 3). For example, Galileo wanted to investigate the effects of gravity in a frictionless medium, and since he clearly did not have access to such experimental conditions, he investigated mediums with low resistance instead (Weisberg, 2007: 3).

The second kind of practice is Minimalist Idealization, where scientists are interested in capturing only the factors which are the primary causes of the phenomena under investigation. The aim of minimalist idealization is to abstract away from features irrelevant to the scientific investigation. It can also aid in increasing scientific understanding by providing a simple and abstract model. Since these cognitive benefits are not related to computational ability, the aim is not to eventually replace these idealizations with more accurate and complicated models (Weisberg, 2007: 7p). Weisberg gives the example of harmonic oscillators, which scientists use to "model the vibrational properties of a covalent bond" (Weisberg, 2007: 7). This idealization can be justified by the fact that other information is irrelevant to understanding the vibrational phenomena (Weisberg, 2007: 8).

The final kind of practice is Multiple-Models Idealization (or MMI). In this case, there is no obvious choice as to which idealized model to employ, because each option has its own shortcomings. So scientists use multiple models instead. For our purposes, it is clear that MMI is not applicable, because the Mendelians used precisely one theoretical model to explain inheritance. So we have to see whether we can understand the particulate theory as a Galilean or a minimalist idealization. Before we do so, we should quickly discuss why these two forms of idealization are compatible with realism. In Galilean Idealization, scientists abstract away from what they know the world is like in favor of computational simplicity. So really Galilean idealization is just a computational crutch, and in no way

displays ignorance or disregard of the truth on behalf of the scientists. With minimal models, the aim is to represent the core causes of relevant phenomena, so to give scientists a picture of how something really works. So a minimal model helps to truly understand or explain the dominant causes for an observation arising. So the distortions of a minimalist models are aimed at uncovering more important truths.

Let's first consider whether Galilean Idealization can help us understand the success of Mendelism. The first problem with this approach is that the Mendelians did not understand what a correct theory of inheritance would look like. So it is doubtful that the Mendelians were consciously simplifying the truth for computational simplicity. It is further doubtful that the untruths of the Mendelian theory could be understood as being purely in the service of computational efficiency. If that were the case, then historical changes in the history of genetics would have been driven by improvements in computational methods. But that description does not match the history we have previously discussed. Instead, it looks like the big breakthroughs after Mendelism have had to do with acquiring a better understanding of the genetic material. For example, consider Watson and Crick's demonstration that the DNA molecule has a double helix structure, or the fairly recent discovery that "junk DNA" has important regulatory functions. In each case genetics progressed because scientists acquired a better understanding of what the genetic material is like. This is prima facie quite a good reason to have a realist attitude about modern genetics, but it also means that Galilean idealization is unhelpful for understanding the success of Mendelian genetics.

Minimalist idealization seems like a better candidate, because Mendel's experimental approach did appear to abstract away from irrelevant features with the aim of understanding the core mechanisms of inheritance. However, it is not the case that the Mendelian view of inheritance tracks the real core causal factors. Arguably there are stretches of DNA we can identify as genes (although these already behave quite differently to Mendelian factors). But a large, and very important part of the genome consists of factors concerned with regulating these genes and their transcriptions. So we can't understand Mendelian theory as truly describing the core causal factors, because it leaves out too many important features.

So we can't think of Mendelian genetics' idealizations as allowing for the theory to be true. It seems to me that Weisberg's treatment of idealization is tailored to modern science, where scientists often (but definitely not always) have a good idea of how the phenomena they are investigating really work. But a lot of science involves, and has involved, situations where nobody really has a clear understanding of what is going on (even if the scientists believed they did). So I don't think Weisberg's realism, or even realism in general, is going to be helpful in explaining the success of the particulate theory. That being said, the discussion of minimal idealization does seem helpful to understanding Mendel's methodology. We can see that Mendel, and his successors, did intend to abstract away from irrelevant observations, and often sought to understand only the most crucial causes of inheritance. But as we have seen, and as I think we will continue to see in the following discussion of Fisher's synthesis, not understanding the core causes of inheritance did not prevent Mendelian genetics from attaining empirical successes. In fact, given the failure of a realist explanation for the success of Mendelian genetics, I think we can attribute it in part to the excellent empirical methods of its practitioners.

Chapter 5: Fisher's synthesis

At this point we have considered and criticized a number of views that could explain why Mendelian genetics was successful. We also have looked at a number of important historical texts and developments. Now I want to begin to provide an explanation of why the synthesis was a success. To do that, we will look at Fisher's "The Genetical Theory of Natural Selection", our final historical text. But first, we need to clarify how the synthesis could lead to a success for the theory of natural selection. Recall that Darwin had two pressing problems. Firstly, Darwin's theory faced the conceptual problem of making reference to inheritance and variation, while Darwin himself had no sound understanding of either process. The second problem was that Darwin lacked the ability to make accurate predictions about inheritance, variation and the pace of evolutionary change. Furthermore, Darwinians therefore had difficulty in defending their theory from legitimate counterarguments.[20] In this chapter, I want to show how Fisher solved both of these problems for Darwinism, and to thereby illustrate how the synthesis served as a successful enrichment for the theory of natural selection.

Fisher's book contains a number of interesting arguments and achievements. We will first look at how Fisher argues against alternative pictures of variation and inheritance. This shows how Mendelian genetics was the best theory of inheritance and variation at the time, and Mendelian genetics, as Fisher argues, clearly supported the theory of natural selection. I think Fisher further argued that natural selection deserved a central role in theorizing about evolution, so he showed not just that the theory of natural selection is compatible with Mendelism, but that the synthesis catapulted Darwinism into a much more important theoretical status. With respect to the empirical problem, Fisher provides a Mendelian model of inheritance and variation, and demonstrates mathematically that natural selection is still possible when a variation (or rather mutation) is rare. So Fisher's work shows how Darwinians were now in a position to understand inheritance, variation and the power of natural selection. This conceptual understanding allowed them to provide explanations that they were previously unable to. So we will conclude that the synthesis led Darwin's theory to conceptual and empirical success.

Let's first see how Fisher tries to sweep away various alternate views of inheritance and variation. Fisher is partly concerned with rebutting the blending theory of inheritance, which, as he have seen, Fisher thought was responsible for some of Darwin's empirical difficulties. As we noted in that discussion, the blending theorist needed to postulate that many variations, or rather mutations, took place in order to explain the possibility of evolution. So the evolution of a trait would require that it appears a great number of times in a population. On this view, the type and frequency of mutations is very relevant to determining which traits evolve. If mutations are very rare, they cannot hope to become prevalent in a population by being naturally selected. In other views, such as that an organ's use or disuse in the parent affects its size or vigor in the child (Fisher, 1930: 12), the mechanism of mutation is supposed to fully explain the direction of evolutionary change. In Fisher's words, views like this claim that "the direction of evolutionary change is governed by the predominant direction in which mutations are taking place" (Fisher, 1930: 13), instead of by the effects of selective advantages. If these kinds of views were correct, the theoretical importance of the theory of natural selection would either weakened or

20 Or at least those counterarguments that made reference to variation and inheritance. But since the lack of knowledge of inheritance and variation was an obvious weak point of Darwinism, it makes sense that many objections were made on these grounds.

nonexistent, since the direction of mutation would be a powerful evolutionary force independent of natural selection (and capable of overpowering or sidelining it). So the refutation of these views would allow for the theory of natural selection to take a central role in explaining the direction of evolutionary change.

Fisher presents a number of objections to the views that inheritance is blended and that the predominant direction of mutation determines the (possible) direction(s) of evolution. Of these counterarguments, I think two are particularly instructive and helpful. The first is that both theories contradict the results of Mendelian pure line experiments, which is a quick, empirical rebuttal to both views. It will also remind us what predictions the Mendelians made about inheritance and variation. Secondly, Fisher demonstrates mathematically that even very rare mutations with small benefits to the organism are likely to be naturally selected. This example shows that the predominant direction of mutation is largely theoretically irrelevant, because the effects of natural selection can overpower the direction of mutation. In fact we will see that natural selection is the most important factor for explaining why certain variations are prevalent in the population.

Let's first consider the pure line experiments. Recall that Mendel thought that there were pure and hybrid forms of plants for a particular trait, where a pure form contained two identical factors, or genes in the modern language, responsible for the given trait. Later Mendelian researchers showed that it was possible to breed self-fertilizing beans so that children consistently shared a trait with their parents (Fisher, 1930: 18) – so the Mendelians concluded that the beans were in pure forms with respect to these traits. These pure forms could be established for a large number of different traits, and variations of traits, in the beans, which suggests that there was a great deal of variation in genes (or rather traits) in the beans to begin with (Fisher, 1930: 18). This observation is not in favor of the blending theory, since, as we have seen in the section on Darwin, it predicted that a population would quickly tend towards uniformity in traits. Therefore, the variation in traits of the beans must have been caused recently by common, numerous mutations. However, in this case the lines of pure breeding should have should have featured a great number of mutations. Instead, the researchers found new mutations to be quite rare (Fisher, 1930: 18). This shows that the variation in traits of the beans could not have been the result of recent mutations, since the mutation rate was quite low. Instead, these new traits must have arisen longer ago, and have been preserved by faithful inheritance over long stretches of time. So the blending theory cannot explain the variation of traits in the beans, because new variations were shown not to arise quickly enough to overpower the blunting effect of blending. Furthermore, the fact that mutations are quite rare strongly suggests that the predominant direction of mutation could not drive evolution, because mutations are not numerous enough to drive evolutionary change. This point is made even more forcefully in Fisher's discussion of experiments on fruit flies:

"The frequency of individual mutations in Drosophila is certainly seldom greater than one in 100,000 of individuals, and we may take this figure to illustrate the inefficiency of any agent, which merely controls the predominant direction of mutation, to determine the predominant direction of evolutionary change" (Fisher, 1930: 20)

From this example, we can also see what basic predictions a Mendelian would make about inheritance and variation. As we have seen from the above examples, Mendelian research showed that mutations had to be very rare. On the other hand, it was quite easy for a Mendelian factor to be inherited, since genes come in pairs, and so each parent would

have a 50% chance of passing on a gene to their child. So, all other things being equal, new mutations were as likely to become more than less prevalent in the following generation. Unlike the blending theory, according to Mendelian genetics there was "no inherent tendency for the variability to diminish" (Fisher, 1930: 9).[21] So Mendelian theory allowed that various different genes could be maintained in the population for long periods of time. This is fortunate for Darwinism, since we now know that evolution by natural selection can take a reasonably long time. However, the low mutation rates appear, prima facie, to be a problem. How is it possible for evolution to take place, if so few mutations occur?

Another apparent problem is this: are there evolutionary changes which natural selection cannot account for? This second problem led Weissman, a prominent Darwininian, to postulate a mechanism by which the direction of mutation could sometimes decide the course of evolution. Weissman felt that there were certain long-term evolutionary processes which could not be explained by natural selection alone (Fisher, 1930: 13). For example, many complex organs, such as the eyes and the brain, slowly evolved, but Weissman thought that not every small evolutionary step could have been sufficiently advantageous to the organism for natural selection to have taken effect. To explain these steps, and to explain the process of continuous organ evolution or disappearance, Weissman argued that mutations could gather momentum. If the same adaptive process was consistently favored by natural selection in the past, then that process would continue even when these steps themselves did not lead to any (appreciable) advantage. Furthermore, mutations having momentum could explain how some traits could reach the point of conferring an advantage. If a mutation appeared, but was not yet significant enough to give its carrier an advantage, then it could still gather (or start with) enough momentum to reach a point where it could benefit the organism's descendants.

Fisher's reaction to both these problems, and Weissman's theory, was, I think, both instructive and important. Fisher's response to Weissman was to argue that there are selective advantages even for small steps in a longer evolutionary process. According to Fisher, we should think that selective advantages vary continuously, even if we do not notice it. So if a large mutation leads to a selection advantage, a smaller mutation of the same sort will lead to a fraction of that advantage (Fisher, 1930: 15). Fisher goes further in arguing that it doesn't matter to the theory of natural selection how small or inappreciable these mutations are: "The rate at which a mutation increases in numbers at the expense of its allelomorph will indeed depend on the selective advantage it confers, but the rate at which a species responds to selection in favor of any increase or decrease of parts depends on the total heritable variance available, and not on whether this is supplied by large or small mutations. There is no limen of appreciable selection value to be considered" (Fisher, 1930: 15p). So according to Fisher, even the smallest advantages matter for evolution by natural selection, and so Weissman's proposed mechanism is theoretically redundant. But this counterargument raises another important question: how can we know that very small advantages can be selected for? This question is related to our earlier one about how evolution can occur if mutations are so rare. In both cases, we need a reason to believe that natural selection is powerful enough to drive evolution, even when the new mutation being selected for only provides a small advantage and/or is quite rare.

Fisher provides an answer to both of these questions in his statistical work. In order to approximate how likely mildly beneficial genes are to spread across the population, Fisher

21 see pages 87–88 of Fisher's book for more precise reasoning on the stability of variation in populations

uses Taylor and Poisson series. He reaches the following conclusion:[22]

"The fact that a mutation conferring an advantage of 1 per cent in survival has itself a chance of about 1 in 50 of establishing itself and sweeping over the entire species, shows that such mutations cannot occur with any great total frequency before this event is realized, or at least rendered certain, by the initial success of one of their number. The odds are over 100 to 1 against the first 250 mutation of such a favorable type all perishing. Consequently the success of such a mutation must become established at a time when the mutation rate of the mutation in question is extremely low, for in a species in which 1,000,000,000 come in each generation to maturity, a mutation rate of 1 in a thousand million will produce one mutant in every generation, and thus establish the superiority of the new type in less than 250 generations, and quite possibly in less than 10, from the first occurrence of the mutation; whereas, if the new mutation started with the more familiar mutation rate of 1 in 1,000,000 the whole business would be settled, with a considerable margin to spare, in the first generation." (Fisher, 1930: 77p)

So Fisher showed that it is possible for a mutation to both be rare and provide a minimal advantage, and still become very prevalent in the population. As Fisher argues, the prevalent direction of mutation should not matter, because natural selection should act before the advantageous trait has the chance to become very prevalent.

So now we have an excellent argument to answer our previously pressing questions. Neither the rarity nor the small effect of beneficial mutations are significant roadblocks to evolution by natural selection.

At this point, I think we can appreciate that Darwin's problems have been solved. Fisher has shown us both what a Mendelian picture of inheritance and variation looks like, but also that it is entirely consistent with the theory of natural selection. Fisher goes even further, and argues that natural selection is the most important mechanism of evolution (for instance by showing how effective natural selection can be in the above passage). This solves the conceptual issue, that Darwin's theory made reference to inheritance and variation, while Darwin lacked a theoretical understanding of them. Not only does Mendel's theory provide such an understanding, but Fisher shows that the Mendelian picture of inheritance sets Darwin's theory centre stage.

Fisher's work also shows how the empirical problem was solved. Darwinians were now in a position to make predictions and provide explanations that required an empirically accurate theory of inheritance. For example, Darwin's empirical problems in chapter 1 evaporate once we understand that traits can easily be faithfully inherited. Lastly, the synthesis undercut any attempt to argue against Darwinism via appeal to an incompatible theory of inheritance. Particulate genetics was, at this point, the consensus best scientific theory of inheritance, and it was shown in Fisher's work (among others) to be entirely consistent with the theory of natural selection.

We have also seen how Mendel's theorizing involved idealization. Mendel and many of his successors thought that they were describing the important features of inheritance. This is similar to Weisberg's view of minimal models, which capture the real causal processes. But we have seen that Mendelism did not correctly describe the real causal processes. Instead, I think idealization was necessary because it allowed Mendel to get to grips with a very

22 This argument assumes that there are genes that appear in pairs, and so is obviously Mendelian

complicated problem in a fruitful way. This is not quite the same as Weisberg's Galilean idealization, because, when genetics advanced to genomics, it did not so much strip away the idealizations from the Mendelian model as completely abandon it. However, I think the idea that Mendel's idealization made computations possible is not that far off. However, it did this by introducing, or allowing for the introduction of, new mathematical or theoretical techniques. Remember how Mendel's factorial model allowed the introduction of combination series, and how Fisher's above argument assumes that inheritance is Mendelian. This shows that Mendel's theory was well suited for mathematical treatment, which I think goes beyond Weisberg's idea of making computations possible. Instead, it seems to me that Mendel's combination series and Fisher's statistics are themselves parts of Mendelian theory, whose manipulation allows not just for calculations, but also for new arguments and explanations. So I think a crucial advantage of Mendelian idealization is that it allowed for the development of a theory that in turn allowed for mathematical theorizing, which included the use of mathematics to (help) make calculations, provide arguments and explanations. In other words, I think Mendel's theory was conducive to mathematical enrichment because of idealization. Therefore I disagree with Weisberg, since I think that the pragmatic benefits of idealization are more numerous than making calculations possible.

So we have seen how the enrichment with Mendelian genetics, and the addition of new (at least for biology) mathematical techniques, solved serious problems for Darwin's theory of natural selection. Evidently mathematics remained relevant to both genetics and the theory of natural selection, despite the particulate theory falling out of favor. So it would be interesting to know whether the mathematical techniques Fisher uses all depend on the Mendelian model of inheritance and variation. If not, then perhaps we should view mathematization as another important, but somewhat independent trend in the history of biology.

Let's look at one of Fisher's most famous arguments, which explains why the ratio of adult males and females is very close to 50% each in most species. Here Fisher uses an equilibrium argument to show that the sex ratio will tend towards 50/50 due to natural selection.

The phenomena to be explained is this: Why are there, all other things being equal, approximately the same number of adult males as females in most species? This does not mean that there an equal number of children born of these sexes. For example, human female fetuses are somewhat more fragile than male fetuses, and therefore slightly more male children are born, despite an equal number of male and female children being conceived (Orzack et al, 2015). However, male children are somewhat more fragile than female children, so more likely to die early, and so in the end effect there are approximately the same amount of young males and females of reproductive age.[23] Since equality is, in the case of humans, achieved only at reproductive age, we might think that it is something about reproductive age that explains this symmetry. Indeed, Fisher does show that selective advantages (which have to do with reproduction) can explain why this symmetry arises. Suppose that there were more females than males at the age of reproduction. Then the average male would be more likely to find a mate then the average female, and so male children would have a reproductive advantage. Now we can further suppose that there are individuals that are more likely to have male children. These individuals will tend to have more children with a reproductive advantage, meaning that they have a kind of second

23 At least insofar as we ignore advances in medicine and artificial selection of children's sexes.

generation advantage. So they will have more grandchildren than other parents, and so their heritable trait of being more likely to have male children will become more prevalent in the population. The same pattern holds if there are more males of reproductive age than females, so there is a tendency for the sex ratio to move to a point where neither parents who have more male children nor parents who have more female children will have an advantage. In other words, the sex ratio will tend to an equilibrium where both sexes are approximately equally represented at the reproductive age.[24]

As we can see, the argument doesn't make assumptions about the nature of inheritance other than that natural selection does frequently take place. Therefore Mendelism is not strictly speaking necessary for this line of reasoning (provided we already have a good general understanding of the power of natural selection). So I think we can conclude that not every mathematical argument in Fisher depended on the Mendelian framework of inheritance. This shows that the increased mathematization of biology was a relatively independent trend. Of course, we have seen that Mendelian genetics lended itself well to mathematical treatment, such as the combination series, which certainly was an advantage of the theory. However, although the relationship between Mendelian genetics and applied mathematics was favorable, that does not mean that they were entirely codependent.

Another interesting feature of this argument is that it idealizes away from various details – for instance the physiological explanations of which adaptions lead particular organisms to have a balanced sex ratio. Fisher's argument doesn't need the information that, for example, female fetuses are more fragile in the womb, but human male children are less robust after birth. Instead, it abstracts away from the specific physiological causes of the sex ratio to explain why it developed – and why it generally must develop. In this way it seems unlike many of Weisberg's examples of minimal models, since it abstracts away from the core physiological causes of the sex ratio, and focuses on the developmental factors and equilibria instead.

24 Fisher's argument is actually a bit more complicated than this, since it takes parental expenditure in birthing and raising the child into account (Fisher, 1930: 142).

Conclusion

We have seen that there are ways of understanding the direction and/or success of science which are not very helpful for understanding the history of Mendelian genetics. I argued that Radick's constructivist story about the biometrician-Mendelian controversy is unconvincing, because does not accounted for later Mendelian successes. These successes provided rational reasons for preferring Mendelism and good responses to Weldon's critiques, even if they were not available at Weldon's time. On the other hand, common realist views were not very helpful either. Putnam's view that successful theories are very likely to be true was inapplicable, either because it is false or because Mendelian genetics was not successful enough. Weisberg's treatment of idealization gave some insight into Mendel's method of abstraction, but did not really help us to think of Mendelism as approximately true. All in all, I think we can rule out the idea that Mendelism was successful because of its theoretical truth.

That being said, the fact that these global constructivist and realist explanations fail does not mean that there could not be realist and constructivist elements to an explanation of the history of biology. For example, the fact that Morgan et al understood the structure of the chromosome no doubt was responsible for many of their successes. And the fact that Darwin was already an established scientist, and Mendel was not, no doubt caused his ideas to gain traction in the scientific community much more quickly. But that does not answer our main question: Why was Mendel's factorial, particulate theory of inheritance so successful?

Unsurprisingly, I think an important part of Mendelism was the empirical methodology. For example, recall that Mendel took great care in his experimental setup, and carefully tracked his traits across many generations. But I think there is a much more interesting way in which the Mendelian theory was empirically responsive. Recall our brief discussion of van Fraassen and theoretical enrichment at the start of this paper: when a theory is faced with empirical difficulties, a good response is often to introduce new theoretical postulates to solve the empirical problem. I think that enrichments have played a crucial part in the history of Mendelian genetics and the theory of natural selection. These enrichments were not limited to the addition of theoretical postulates, but included new styles of reasoning. To see how the early histories of Darwinism and genetics were shaped by enrichment, let's consider the historical developments and sources we have discussed in this paper:

Recall that Darwin's theory of natural selection was initially much less popular than his views of descent. Since Darwin's views on descent contradict creationism, we cannot attribute this inequality to the existence of creationist biases. Rather, we learned that Darwin's theory suffered because it required a theory of inheritance and variation, but Darwin couldn't supply a satisfactory one. This was a problem for Darwin because he was aware of the empirical difficulties his theory faced; of its generation of inaccurate predictions. For other members of the scientific community, the absence of a widely accepted theory of inheritance that supported the theory of natural selection meant that one could doubt whether evolution by natural selection was possible. As we see in Fisher, the idea that mutations drove the direction of evolution was a possible, popular alternative. So Darwin's theory needed to be enriched by a theory of inheritance and variation that could solve its empirical difficulties, but also improve its standing in the scientific community, by demonstrating that the best theory of inheritance and variation was consistent with Darwin's theory of evolution. It also needed to be shown that views

of inheritance inconsistent with the theory of natural selection, and other pictures of evolution stemming from those views, were untenable.

In fact this is precisely the kind of work we see Fisher do. We saw how he argued against the general alternative view that evolution was driven by mutation and thoroughly dismantled it. Fisher's more positive project consisted not only in demonstrating the compatibility of Mendelian genetics and the theory of natural selection, but also in creating a general model of inheritance, variation and selection. As Fisher explains, the (Mendelian) genetical theory of natural selection predicts that new mutations are quite rare, that they could be quite easily be transferred across generations, and that natural selection would then be likely (albeit slow) to act upon them. This general model was then shown to be consistent with independent, mathematical arguments, such as the statistical proof that mutations with small selective advantages were likely to spread across a population (according to the particulate theory of inheritance). Looking at both Fisher and Darwin, I think we can view the synthesis as a successful enrichment of Darwinian theory by Mendelian/particulate genetics.

So much for the broad scientific development. But I think we can also see how smaller shifts in the history of Mendelian genetics involved enrichment. Recall our discussion of Weldon's criticisms of Mendel and how, by Morgan's time, Mendelians had already adjusted their theory so that many of these criticisms fell flat (or at least were not as compelling as Radick made them seem). I argued that Weldon's weakest objection was his observation that Mendelian theory contradicted the Galton's law of ancestry, but thought that his other objections were more compelling. No doubt this was due to the empirical nature of Weldon's complaints about environmental effects and dominance, and of course we saw that Mendelians adapted their theory to these empirical difficulties (whether they were aware of Weldon's critique or not). At this point theoretical modification took a different route: instead of enriching their theory, Mendelians cut out problematic elements of Mendel's original approach. In particular, Mendel's treatment of dominance, as a state where the effects of recessive genes were completely negated, fell out of fashion. So sometimes the appropriate response to empirical challenges is not to add to the theory, but to strip away a problematic or limiting element of the theory. But this kind of development is not unrelated to enrichment. Firstly, we can think of both as different facets of the same kind of scientific development, where scientists modify their current theory rather than abandoning it for something quite different. Secondly, the loosening of Mendelian understanding of dominance coincided with the addition of new, more fruitful ways of classifying the relationships between factors [25]. So the removal of certain theoretical elements can coincide with enrichments. In this case a the removal of a restricting concept of dominance allowed for the theoretical description of a number of different patters of inheritance.

Of course there was a better example of enrichment of Mendelian theory in Morgan et al's book. The introduction of chromosomal reasoning made it possible for Mendelians to explain linkage and 1:1 ratios of eye color in fruit flies, when they are crossed in certain ways. So not only was Mendelian genetics successfully used to enrich Darwinian theory, but Mendelian theory was itself improved by various theoretical enrichments. These enrichments included new mathematical and statistical techniques, as we saw in our discussion of Fisher. However, as we noted in our discussion of the sex ratio argument, the mathematization of biology cannot be thought of purely as an enrichment of Mendelian

25 Such as the confusingly named co-dominance and partial dominance.

genetics. Instead, we should think of mathematization as representing a reasonably independent trend in the history of genetics and evolutionary theory.

So we can really understand the early histories of the theory of evolution by natural selection and genetics as a history of enrichments. Why were enrichments so helpful? When we think of the synthesis, the answer seems simple: Darwin's theory needed to make predictions about inheritance and variation, which Mendelian genetics was able to. In the cases we have considered, theoretical enrichments have aided theories in achieving empirical success. I think we can say that the practice of enrichments can make theories more empirically responsive; so it can allow scientists to adapt or improve their theory to account for various empirical phenomena.

On the other hand, our discussion of modern genetics showed that this incremental, enriching approach is not always appropriate. Modern discoveries about the roles of 'non-genic' DNA and RNA show that the particulate model of inheritance cannot be reconciled with the modern understanding of the genome. So sometimes there are problems that can only be solved by sweeping old theory away and taking a different approach.[26]

Although I have argued that theoretical enrichment is very important to understanding the history of genetics and evolutionary theory, I think it is appropriate to end on a cautionary note. As we have seen in the above example on genomics, theoretical enrichment was not always an appropriate scientific strategy. We can also appreciate that not every aspect of Darwin and Mendel's success and failure can be explained by empirical problems and enrichment (for example, remember that we attributed Mendel's work's initial obscurity to the fact that Mendel, unlike Darwin, was not already a well known figure in the scientific community). I think these remarks illustrate that, even when a philosophical theory of science does nicely apply to the scientific history, we should not generally expect science to be simple enough to completely fit an overarching philosophical narrative. Instead, we should always seek to understand a portion of the history of science before finding or devising a piece of philosophy to understand it (and not the other way around).

So I conclude with a healthy, but undogmatic, skepticism of overarching philosophical narratives. I argued that broad realists and constructivist stories are unhelpful in understanding a specific episode of scientific history, while a broad view of empirical enrichment was. However, I attribute this discrepancy not to the absolute superiority of one style of philosophical explanation over another, but to the specifics of the early histories of Mendelian genetics, Darwinism and the synthesis. But even if science doesn't have any essential characteristics (Fine, 1996: 174), we can still use philosophical theories to understand it – provided that we are appropriately sensitive to the history of the scientific field, success or development that we are investigating.

26 This claim does not, I think, require us to commit to a realist or anti-realist understanding of molecular genetics. We might say, following an intuitive realist view of molecular genetics, that the problem is that particulate theories incorrectly describe the behavior of the heritable material. Or we might, following van Fraassen, say that observations under the microscope are just different empirical phenomena that the old genetical theory could not account for – without having to commit ourselves to the claim that genetical science is about discovering what the genetical material really is like. Either way, I think we can say there was a time when biologists managed to attain an impressive degree of empirical success, without even having a good idea of what kind of molecules were responsible for the inheritance of traits. And this success was attained in part via good experimental methods, but also by the practice of theoretical enrichment.

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