Of stirps and chromosomes: Generality through detail

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A B S T R A C T

One claim found in the received historiography of the biometrical school (comprised primarily of Francis Galton, Karl Pearson, and W. F. R. Weldon) is that one of the biometricians' great flaws was their inability to look past their population-focused, statistical, gradualist understanding of evolutionary change – which led, in part, to their ignoring developments in cellular biology around 1900. I will argue, on the contrary, that the work of the biometricians was, from its earliest days, fundamentally concerned with connections between statistical patterns of inheritance and the underlying cellular features that gave rise to them. Such work remained current with contemporary knowledge of chromosomes, cytology, and development; in this article, I explore the first case. The biometricians were thus well positioned to understand the relationship between the patterns of Mendelian inheritance and the statistical distributions with which they primarily occupied themselves. Ignorance of this connection, then, is not the reason why they rejected Mendelism. Further, both Galton and Weldon – though each in their own unique way – decided to turn to biological detail as a way to better justify the generality of their statistical approaches to heredity. Perhaps paradoxically, then, for these biometricians, detail offered an approach to theoretical generality.

A classic historical narrative of the science of 'inheritance' or 'heredity' during the first years of the twentieth century (during which time it would be rechristened 'genetics') draws our focus inexorably to a grand and, it is claimed, fruitless debate, pitting against one another two groups of biologists who each have things half-right. In one corner, the young geneticists, who correctly see in Mendel's results a path toward the understanding of, to borrow modern parlance, the biochemical foundations of the inheritance of traits from parents to offspring and the nature of traits as varying alleles at different genetic loci – but are, lamentably, too focused on large-scale mutation and too neglectful of slow, adaptive evolutionary change. In the other corner, the biometricians, partisans of a statistically grounded and strictly gradualist understanding of natural selection, who were too hidebound within their own research tradition to see the ways in which Mendel's results could be brought within the fold of their own work. We would have to await the genius of R. A. Fisher, Sewall Wright, and the other architects of the Modern Synthesis to transcend this impasse, by uniting the good and discarding the bad arising from both approaches.

Such a view, which focuses our attention on the deeply personal "biometry-Mendelism debate" (and, secondarily, on a debate over the importance of continuous vs. discontinuous variation in evolution), can be found in a number of classic historical works presenting the history of genetics. Perhaps most famously, Provine's celebrated history of population genetics describes work in this period as follows:

After 1900, when Bateson became a champion of Mendelism and Pearson named his science biometry, the controversy became known to the public as the conflict between the Mendelians and biometricians. The conflict drove a wedge between Mendel's theory of heredity and Darwin's theory of continuous evolution and consequently delayed the synthesis of these theories into population genetics. (Provine, 1971, p. 56)

Olby writes, with martial flair, that "it was blending heredity and Galton's formulation of the Ancestral Law which the biometricians backed to the hilt in their fight against Mendelism" (Olby, 1966, p. 82). Bowler writes that while "the most important factor limiting the success of the biometricians was their clash with the newly emerging Mendelian genetics" and "the biometricians were misguided in their rejection of Mendelism," they were nonetheless "ultimately justified in their support for the selection of continuous variations as the mechanism of evolution" (Bowler, 1989, p. 256). "This personal quarrel," writes Sturtevant, "which came to involve Pearson as an ally of Weldon, seems to have been a chief reason for the anti-Mendelian stand of both Weldon and Pearson" (Sturtevant, [1965] 2001, p. 58).

Thankfully, it is now becoming a commonplace among historians of biology that such a view of this pivotal period is too narrow-minded. A number of examples might be cited, but to mention just a few: William

Bateson's early training and relationship to statistical biology makes him difficult to classify (Peterson, 2008; Radick, 2012); the way in which W. F. R. Weldon approached these questions is much more complex than had been previously assumed (Pence, 2011; Radick, 2005, 2011); and, in general, the reductive effort to simplify the entire period to a story of one particular conflict is bound to run afoul of sound historiography (Porter, 2014; Shan, 2020; Vicedo, 1995).

In this paper, I will target another way in which this classic tale falls short of the facts on the ground. One element of the "biometry-Mendelism debate" story emphasizes the extent to which the Mendelians were equipped to consider the rapid advances in cellular biology in the first decade of the twentieth century – especially developments in our understanding of chromosome structure, cell division, developmental biology, and other features involved in the underlying mechanisms of character transmission – that the biometricians, with their statistical, distant view of large-scale population phenomena, could not fully appreciate. Bateson and the Mendelians thus had the advantage of being able to keep pace with a whole host of empirical data that was invisible to Pearson and Weldon.

Again, this is a claim that one finds across histories of the period. Provine's reconstruction of the real point of difference between Bateson and Karl Pearson over the latter's theory of homotyposis, for example, centers on characteristics of the germ cells to which Mendel's theory clearly pointed, and which biometrical work passed over (Provine, 1971, pp. 60–61).[1] Sturtevant glosses the critiques of biometry offered by Bateson, Johannsen, "and others" as pointing out that their results "were only valid statistically, were of no help in individual families, and *gave no insight into the mechanisms involved*" (Sturtevant, [1965] 2001, p. 59, emph. added). Bowler writes that the biometricians' "problem was that without a suitable theory of *how heredity worked*, they could not exploit this insight [a statistical approach to inheritance] in the defense of Darwinism" (Bowler, 1989, p. 257, emph. added). Olby, writing about the impact of discoveries concerning the chromosomes, writes that the Weismannian idea that the germ cells of an individual could have different internal constitutions "was precisely the point which Naegeli and Darwin were not prepared to accept" (Olby, 1966, p. 137),[2] in which claim he seems to be joined by no less than R. A. Fisher, who wrote that "the revolutionary effect of Mendelism will be seen to flow from the particulate character of the hereditary elements," ascribing the discovery of such a position to Mendel and its advocacy to Bateson, whom he otherwise held in fairly low opinion (Fisher, 1930, p. ix).[3]

One might thus develop the impression at a glance that the biometricians, in their pursuit of a statistical, populational theory of evolution, had no use whatsoever for the kinds of particular cellular details that so interested the early Mendelians. This would be mistaken. On the contrary, the details of the transmission of characters from parents to offspring was a recurrent motif throughout the work of the biometrical school, though it was put to various uses by different authors (and, obviously, changed dramatically over the period from 1890 to 1910, as knowledge about those underlying cellular processes improved). This fact has not escaped notice, of course: a variety of commentators have

remarked upon the extent to which speculations and theories about underlying systems of heredity were an important part of the stock-in-trade for a host of authors writing in the period between the death of Darwin and the Modern Synthesis. Churchill (1987), for instance, underlines the importance of developments in German cellular biology of the 1880s for theories of heredity. Müller-Wille and Rheinberger (2012) discuss a number of such speculations, including dedicating a significant amount of space to the work of Galton, about which more below. That said, the work of the biometrical authors is often minimized or dismissed. Churchill, for example, refers to Galton's work as one of a set of "inconsequential trial balloons" (Churchill, 1987, p. 360) of the period.

In what follows, I hope to expand on these arguments, though I will limit my focus here to one theoretical tool – chromosome theory – and two authors commonly taken to be far at the "biometrical" end of the biometry-Mendelism spectrum, Francis Galton and W. F. R. Weldon.[4] Galton's initial take on the question is – as is typical for Galton's theorizing in general – suggestive and schematic. Galton's view of inheritance replaces Darwin's theory of pangenesis with his theory of the "stirp," a hypothesized physical carrier of the elements responsible for the development of particular characters, transmitted from parents to offspring via a sort of random sampling process.

These suggestions were taken up aggressively and innovatively by Weldon as he turned his sights to offering an explanation of Mendelian-style empirical results (*alternative inheritance*, in his terminology). Weldon's work, which was aided by engagement with the extensive work on cellular and developmental biology in progress around 1900, could ground Galton's idea of a stirp more concretely in the chromosomes, and proposed a more sophisticated mathematical apparatus to derive the patterns of transmission of elements over time (though he would not, in the end, succeed at developing this formalism).

In short, while their thoughts on these questions have not been the subject of much historical attention (especially Weldon's), the work on the underlying mechanisms of inheritance by the biometrical school was varied, vibrant, and entirely consonant with the study of cellular structure at the time.

I will close by considering the reasons that these two authors might have approached a statistical theory of evolution in such a way. Put simply, if we are armed with a statistical, population-level theory of natural selection, why bother with getting into the weeds of cytology? Couldn't we simply argue instead that this statistical theory functioned as an idealization, highlighting important features of biological populations that nonetheless did not need to be tightly connected with cellular detail? I will argue that both authors felt that obtaining further data about the underlying processes of heredity was important not for purposes of reduction, or in the pursuit of some kind of fundamentality or physics-envy, but rather to make their statistical theories *more general*. To do so, I canvas a number of ways in which generality has been discussed in the philosophy of science, and, using recent work on scientific idealization by Angela Potochnik (2017), I will consider what might have pushed Galton and Weldon to pursue generalized theories of heredity by means of their chromosomal basis. Both authors, I claim, rejected an idealization-based approach to evolution because they had good, well-founded reasons for thinking a statistical explanation needed, nonetheless, to refer to those lower-level details.

The historical story that I tell here, then, is intended to ground a much more holistic and less antagonistic perspective on the kind of science done by the biometricians. As was the case for many of their national and international colleagues, they were deeply motivated by a desire to understand evolutionary change from the population to the cellular level. Illuminating that story can help us to understand better the broader

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[1] The story is a bit more complex, in fact: Bateson "*could have* devastated Pearson's theory" using the implications of Mendel's results for the constitution of the germ cells, Provine writes, but Bateson chose not to do so, as he was also hesitant to adopt a material basis for heredity (Provine, 1971, p. 61, emphasis added). So much the worse for both of them, apparently.

[2] We will see that the biometricians were not only ready to accept this point, but had already done so as early as Galton.

[3] A more sophisticated lens, though still a version of the same critique, can be found in Bulmer's biography of Galton, when he notes that an alternative way to construct this worry about a blind-spot in biometry is to note that Galton seems to have been unaware that the Law of Ancestral Heredity "has two different interpretations: as a prediction equation, and as a representation of the separate contributions of each ancestor to the phenotype of the offspring" (Bulmer, 2003, p. 249).

[4] I will unfortunately have to largely pass over the work of Karl Pearson, who obviously constitutes a natural third member of this set, for reasons of space. I hope to return to Pearson's approach to these questions in more detail in later work.

context of this period, beyond the constrictive framing of the "biometry-Mendelism" debate.

1. Galton's 'stirps'

We should begin, then, with the founder of the biometrical school, the first author to present a statistical conception of the inheritance of character traits from parents to offspring: Francis Galton.

Galton's engagement with the concept of inheritance begins when he reads his cousin Darwin's *Origin of Species*. He would write in his autobiography that the work "made a marked epoch in my own mental development, as it did in that of human thought generally" (Galton, 1908, p. 287). His focus increasingly turned toward inheritance, first in domesticated animals and then – especially as his interest in eugenics increased – in man. Just as this transformation was underway, Galton would be exposed to statistics for the first time, via the work of Quetelet. And just as Galton was preparing his initial study of the inheritance of "genius" in man, Darwin published his theory of pangenesis.

All in all, these events combine to form the first presentation of a statistical approach to character transmission and evolution, in the last chapter of Galton's *Hereditary Genius* (1869). As Porter puts it, "the statistical study of heredity was anchored from the beginning in Pangenesis, which, at least in Galton's hands, was clearly a particulate theory" of inheritance (Porter, 2014, p. 130). Galton commits fully to Darwin's new mechanism for the inheritance of characters:

> [Pangenesis] gives a key that unlocks every one of the hitherto unopened barriers to our comprehension of [heredity's] nature; it binds within the compass of a singularly simple law, the multifarious forms of reproduction, witnessed in the wide range of organic life, and it brings all these forms of reproduction under the same conditions as govern the ordinary growth of each individual. (Galton, 1869, p. 364)

There are a number of significant features of pangenesis that attracted Galton. First, he finds in Darwin an aspect of inheritance that he takes to be central, and broadly neglected by prior authors: the distinction between what he will call *patent* and *latent* characters. Darwin's theory allows for the existence of "a vastly larger number of capabilities in every living being, than ever find expression, and for every *patent* element there are countless *latent* ones" (Galton, 1869, p. 367). Galton believes that such a distinction is important for a number of reasons. First, he always holds that evolution by natural selection will have to be *less* gradual than Darwin had thought (though for more on Galton's rather confusing brand of saltationism, see Bowler, 2014). The presence of numerous latent characters, many of which might be simultaneously activated after a number of generations of apparent absence, could allow for the right kind of "sports" to emerge to drive larger, non-Darwinian episodes of evolutionary change. Further, Galton is continually impressed by the phenomenon of occasional reversion to the characters expressed in distant ancestors (as Darwin himself had been in pigeons in the *Origin*); the constant presence of latent characters offers a handy explanation for this phenomenon as well.

Additionally, and perhaps more importantly in the long run, Galton recognizes that "the theory of Pangenesis brings all the influences that bear on heredity into a form, that is appropriate for the grasp of mathematical analysis" (Galton, 1869, p. 373). Presaging his later work on the Law of Ancestral Heredity (Bulmer, 1998; Galton, 1897), Galton writes that

> It becomes an interesting inquiry to determine how much of a person's constitution is due, on average, to the unchanged gifts of a remote ancestry, and how much to the accumulation of individual variations. The doctrine of Pangenesis gives excellent materials for mathematical formulæ, the constants of which might be supplied through averages of facts, like those contained in my tables, if they were prepared for the purpose. My own data are too lax to go upon; the averages ought to refer to some simple physical characteristic,

unmistakeable in its quality, and not subject to the doubts which attend the appraisement of ability. (Galton, 1869, p. 371)

Indeed, he writes later, there is no reason that such a formula could not allow for the prediction of the distribution of characters in offspring from their distribution in the parents.

All well and good – though soon to be dramatically upended by Galton's own experimental work, which would call into doubt the fundamentals of pangenesis (Galton, 1871). His experiments on the transfusion of blood within rabbits indicated, at the very least, that gemmules like those Darwin described did not circulate in the blood, and they were taken by the broader community to be even more damaging than that to pangenesis.

Galton thus finds himself in the position of being in possession of more and more observational and experimental data that he takes to confirm his general approach to understanding heredity (by statistical comparison between parents and offspring), while at the same time having no clear explanation for why evolving populations would give rise to such statistical distributions in the first place. He clearly believes that in particular instances – his data on Basset Hound coat color would be commonly referred to for decades – he can offer very compelling statistical descriptions of population change. But it seems as though the failure of pangenesis leaves him without the kind of *explanation* of these changes for which he was searching.

He returns to the question in a number of articles in the mid-1870s, searching for a workable vocabulary for talking about the transmission of heritable elements between cells.[5] In order to interpret almost any of the facts of heredity, he argues, it must be the case "that each of the enormous number of quasi-independent units of which the body consists, has a separate origin, or germ" (Galton, 1876, p. 331), but there is no clear sense of how such germs might function. Of course, this is not necessarily a problem. As Porter has noted, for Galton (and this would remain true for Weldon, as we will see in the next section),

> Nobody had ever detected a gemmule, or what de Vries called a pangene, and nothing was known about physiological processes that might produce one. They were hypothetical entities, whose presumed processes were inferred in a somewhat circular fashion from the phenomena they were designed to explain, rather than tangible objects susceptible to laboratory manipulation. (Porter, 2014, p. 129)

Galton was thus readily able to soldier on with a somewhat sketchy interpretation of the "germ" as that which gives rise, in the end, to differentiated cell types.

In that spirit, then, we also know that the characters must be capable somehow of being united together in the germ cells. Both the patent and latent characters in an organism, he writes, must "diverge from a common group and converge to a common contribution, because they were both evolved out of elements contained in a structureless ovum, and they, jointly, contribute the elements which form the structureless ova of their offspring" (Galton, 1872, p. 394).[6] He will refer to these unions as *stirps*:

> I beg permission to use, in a special sense, the short word "stirp," derived from the Latin *stirpes,* a root, to express the sum-total of the germs, gemmules, or whatever they may be called, which are to be found, according to every theory of organic units, in the newly fertilized ovum – that is, in the earliest pre-embryonic stage – from which time it receives nothing further from its parents, not even from its mother, than mere nutriment. (Galton, 1876, p. 330)

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[5] For a particularly clear presentation of Galton's theorizing between the 1870s and 1889, see Bulmer (2003, pp. 119–131), though I disagree with Bulmer that there is a clear distinction to be drawn between the theories of the 1870s and the theory as laid out in *Natural Inheritance*.

[6] While detailing it would take me too far afield here, the conception of matter at work in Galton's referring to the ova as 'structureless' was an element in a rather heated debate between Galton and Maxwell; see Radick (2011).

Stirps will thus bear both the latent and the patent characters of organisms, and be somehow implicated in the process of the creation of germ cells.

How does Galton believe the elements carried by stirps actually segregate? Galton here returns to an analogy he first deployed in *Hereditary Genius*, between the elements within the chromosome and representative governments.[7] "Since for each place" among the active elements, he argues, "there have been many unsuccessful but qualified competitors [i.e., among the latent elements], it must have been on some principle whose effects may be described as those of '*Class Representation*,'" though explicitly without introducing any particular theory as to the details of how this process in fact operates (Galton, 1872, p. 395).[8] Each character trait, that is, which is required to construct an organism must thus have some kind of "representative" taken (elected?) from among the collection of elements that could have given rise instead to alternatives for that trait. Of course, he argues, we do not know the average number of "candidates," whether or not "the same person is eligible for, or may represent at the same time, more than one place, nor whether the result of the elections at one place may not influence those at another" (Galton, 1872, p. 395).

Later, he writes a few more concrete ideas about the process of segregation – the most thorough treatment he considered himself able to provide of the subject:

The conditions under which each element in the sample became selected are, of course, unknown, but it is reasonable to expect they would fall under one or the other of the following agencies: first, self-selection, where each element selects its most suitable neighbour, as in the theory of pangenesis; secondly, general co-ordination, or the influence exerted on each element by many or all of the remaining ones, whether in its immediate neighbourhood or not; finally, a group of diverse agencies, alike only in the fact that they are not uniformly helpful or harmful, that they influence with no constant purpose – in philosophical language, that they are not teleological; in popular language, that they are accidents or chances. Their inclusion renders it impossible to predict the peculiarities of individual children, though it does not prevent the prediction of average results. (Galton, 1885, p. 1213)

Of course, this is at best a simple taxonomy of the possible kinds of influences that could impact segregation. They could either be directed at maintaining the fitness of the organism, and within that category either local (involving only immediate neighbor-elements) or global (involving a more general structuring cause), or they could be random. We know, however, that given the stability of species over the long term, the perturbing or random influences must on the average cancel themselves out.

Finally, how does Galton think the elements carried by stirps will influence development in the actual organism? He never makes many claims here, or at least none more significant than that each part of the organism will have an element which is responsible for its character, but he does frequently allude to the idea that the *position* of these elements with respect to one another is vitally important for both patency and for the process of growth itself. He writes that "organisation wholly depends on the mutual affinities and repulsions of the separate germs; first in their earliest stirpal stage, and subsequently during all the processes of their development" (Galton, 1876, p. 331).

There is much that could be said about Galton's use of stirps as a grounding for his statistical theorizing about inheritance; I will focus here only a theme that relates to the more general argument about theoretical generality to which I will return in Section 3. The way in which Galton chooses to present these claims in his *opus magnum, Natural Inheritance* (1889), is telling. After discussing the basics of the process of inheritance, and introducing something like the competition for places among the patent elements mentioned above, he notes that this entails that "the step by step development of the embryo cannot fail to be influenced by an incalculable number of small and mostly unknown circumstances" (Galton, 1889, p. 9).

Such an invocation will doubtless be familiar to anyone who has read either Galton or his statistical mentor, Adolphe Quetelet. As Galton himself will make precise later in the work, the fact that development is subject to so many small causal influences is quite meaningful:

The incalculable number of petty accidents that concur to produce variability among brothers, make it impossible to predict the exact qualities of any individual from hereditary data. But we may predict average results with great certainty …. [This chapter's] intention has been to show the large part that is always played by chance in the course of hereditary transmission, and to establish the importance of an intelligent use of the laws of chance and of the statistical methods that are based upon them, in expressing the conditions under which heredity acts. (Galton, 1889, pp. 16–17)

For Galton, as for Quetelet before him, precisely what grounds the use of statistical methods in the first place is the traditional derivation of the law of error. In Galton's words, when "a number of small and independent accidents" are accumulated, then "in rare cases, a long run of luck continues to favor a particular [element] toward [the extremes], but in the large majority of instances the number of accidents that cause Deviation to the right, balance in a greater or less degree those that cause Deviation to the left" (Galton, 1889, pp. 64–65). Any such process will produce, in the end, normal distributions of the outcomes that result.

Thus, for Galton, the construction of heredity as the net influence of a number of independent, small accidents that befall each of the elements of a stirp responsible (or latent, but potentially responsible) for the development of a character trait is crucial. For it is not merely by their sheer number, or their potential to be quantified, that the stirp picture enables the development of a mathematized theory of heredity. If these were the only relevant properties of the stirps, we could perhaps write down entirely abstract formulas describing their transmission, but we would have precious little way to reason with them. It is the structure of development as arising from the small interactions of and modifications to the elements of the stirps that allow for them to be statistically treated in the first place.

In that sense, I argue, Galton's very understanding of the nature of statistics requires that he ground the presence of statistical distributions and evolutionary change in a very particular set of characteristics of the underlying heritable material. Something like a stirp must exist, not only for Galton's approach to provide satisfying explanations of statistical population change, but even for that approach to be feasible in the first place. Galton's understanding of statistics means that a theory of the underlying details of inheritance is a condition for the possibility of a general, statistical theory of evolution.

Galton's theory, of course, is laid down before any significant results concerning chromosomes or other features which might have given rise to that underlying biology of inheritance are known. Theodor Boveri's celebrated experiments on chromosomes, for instance, only began in the mid-1880s (well after Galton has formulated his entire understanding of heredity).[9] The lack of biological detail in his theory is thus entirely

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[7] The various socially and culturally situated metaphors that Galton used to discuss heredity, including this one, are discussed in detail by Müller-Wille and Rheinberger (2012), pp. 6–12).

[8] A few years later, as Olby has highlighted, Galton would write to Darwin, imagining a case on which the elements within stirps were paired up combinatorially. Setting aside Olby's rather Whiggish interpretation that "here we find all the elements of the Mendelian explanation save the independent segregation of different pairs of characters" (Olby, 1966, p. 72), Galton unfortunately never put this more mathematically precise view down in print.

[9] Punnett, interestingly, described Boveri's hypothesis of the individuality of the chromosomes as an *impediment* to the recognition of their function by the early geneticists, in interesting contrast to Weldon below (Punnett, 1950, p. 10).

explicable; his description of the germ cells as structureless is, in essence, a promissory note constituting the best knowledge of his day.

Things would change rapidly, however, as Galton's work was extended by the now fledgling biometrical school. While that group's best known member, Karl Pearson, was never particularly interested by the biological fundamentals grounding his use of a statistical theory of heredity, they would become crucial for Pearson's colleague and close collaborator, Raphael Weldon.[10]

2. Weldon on chromosomes

The 1890s were, for W. F. R. Weldon, a furious period of experimental and theoretical work on biometry. Early empirical successes, particularly in the measurement of characters of crabs (Weldon, 1893, 1895), were supported by mathematical discoveries arriving at a breakneck pace (typified by Pearson's series of "Mathematical Contributions" articles, beginning with Pearson, 1894). It is in this intellectually turbulent environment that we find Weldon's first engagement with chromosome theory.

2.1. Weldon's Weismannian and Galtonian context

In May or June 1896, seven years after Galton's *Natural Inheritance*, he receives a long letter from Galton, describing Galton's then-current understanding of the cellular properties underlying the transmission of characters from parents to offspring. Unfortunately, I have been unable to find Galton's own letter (measuring at least ten pages) in the archive (Weldon likely destroyed it, as he did not often preserve his incoming correspondence). We are thus left only with Weldon's ten-page response, written on June 6, 1896 (Weldon, 1896).

Weldon begins by narrating his understanding of the state of research on cell division and chromosome duplication. Galton, apparently following Weismann,[11] has advanced two hypotheses: in Weldon's words, first, "that the character of the nuclear chromosomes 'determines' the nature of a cell," and second, "that the separation of chromosomes during embryonic development is always selective" – that is, that "the segregation of the egg is accompanied by a sorting of the determinants into 'suits' [i.e., of cards], one 'suit' going to one group of cells, which is thereby rendered capable of becoming say nervous tissue, while another 'suit' being dealt to a second group of cells forces these cells to become muscle-cells; and so on" (Weldon, 1896, f. 264). Weldon is extremely dubious about this second claim. In addition to the apparent similarity of the cells produced after these divisions, Weldon lays out the results of Driesch, Chabry, and Roux on the development of sea-urchin eggs (and the replication of similar results by Haeckel in jellyfish), as well as evidence concerning the regeneration of lost parts, implying that plasticity, and not "sorting," remains the order of the day for a number of divisions during the development of a new embryo.[12] He closes the letter in a postscript by defending Boveri's observation of the development of denucleated eggs (see Laubichler & Davidson, 2008): "people who have

not his excellent habit" of experimental technique "may easily undervalue his statements," Weldon writes (1896, f. 271).

In short, while Weldon does clearly think that chromosomes are important, he is quick to quash any theory "which involves the belief that egg-segmentation is only a sorting out of hereditary substances" (Weldon, 1896, f. 270). On Weismann's (and, presumably, Galton's) theory, "determinants" are formed from collections of smaller "biophores," and the former were responsible for cellular differentiation. Each cell receives the determinants responsible for the type into which it will develop, along with those for the types of the more distal offspring cells to which it will give rise. (Determinants, in turn, are packaged into "ids," which are packaged into "idants," the analogue of chromosomes.) "Thus," Esposito writes, for Weismann "development was essentially conceived of as a process of distribution and progressive parceling of *determinants* following cellular differentiation" (Esposito, 2013, p. 519). Of course, as Esposito discusses in some detail, the phenomena of regeneration pose an immediate and obvious problem for any such interpretation of development, as they imply that cells need to contain more determinants than those they are currently expressing – Weldon, unlike Weismann, believed that this problem was fatal.[13]

Importantly for our purposes, though, is the level of sophistication that Weldon brings to the argument: he is clearly fully informed of the developmental and cellular biology of his day. What Galton has lost track of, Weldon thinks, is his own theory's former insistence "that this government [of cell growth by chromosomal elements] is a function alike of the composition of the nuclear substance *and of its position in the embryo.* So far as I know, Driesch and I are the only people who habitually preach this: but I do not see any alternative" (Weldon, 1896, f. 266, original emphasis).

2.2. Responding to Mendelism

Weldon would not pursue his own theory of the connection between chromosomes and inheritance for a few more years, until an external disruption in his research program provided the needed impetus. In the immediate aftermath of the "rediscovery" of Mendel's work in late 1900 by Bateson, Carl Correns, and Erich von Tschermak, it was business as usual for the biometrical school. Weldon had recently moved from University College, London, to take up a professorship at Oxford, while Karl Pearson stayed behind (and would eventually head the Galton Eugenics Laboratory there). In 1898, Weldon turned more seriously to the analysis of natural selection, spurred by what appeared to be a very rapid adaptive change taking place in crabs found in Plymouth Sound, near the laboratory of the Marine Biological Association (Weldon, 1898).

On October 16, 1900, Weldon wrote to Pearson that he had read a copy of Mendel's paper for the first time, summarizing the basic gist, and offering to bring the Royal Society's copy of the article to Pearson if he'd like (Weldon, 1900a). What strikes one most about the letter, given the outsized importance with which this period would be treated by the later history of biology, is its entirely quotidian nature: Mendel's paper is just another topic of daily conversation, an interesting result about breeding in peas, sandwiched between other letters concerning personal tribulations and extensive discussions of breeding in just about any other system for which Pearson and Weldon could obtain data.

In the context of research in the biometrical school, however, this makes perfect sense. To Weldon, Mendel's results didn't appear to be more than a special case of their extant research. As he would later explain his view of the matter, it was surely a novel collection of results that was interesting as a launching pad for further work, particularly insofar as it pointed toward an asymmetry in biometrical understanding of the various modes by which inheritance might take place. The data collection undertaken by Weldon and Pearson had focused largely on

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[10] For a subtle approach to Pearson's thoughts on the underlying basis of heredity, see Porter (2014).

[11] Weismann's work was widely disseminated in the early 1890s (Weismann, 1891), and we know that Weismann and Galton corresponded. Galton found in Weismann a fellow traveller, and Weismann described one of Galton's first papers on heredity (Galton, 1865a,b) as "in one essential point nearly allied to the main idea contained in my theory of the continuity of the germ-plasm" (Weismann to Galton, February 23, 1899, quoted in Pearson, 1930, p. 341). As Bulmer notes, however, we should not misinterpret the two men's theories as identical: Galton has no equivalent for Weismann's concept that "the germ-plasm of the zygote is doubled, with one part being reserved for the formation of the germ cells" (Bulmer, 2003, p. 133).

[12] Note that, in general, there was much debate around how to interpret the results of these experiments before around 1903 or 1904; see Baxter and Farley (1979); Esposito (2013).

[13] An illuminating discussion of Weismann's response to the challenge of regeneration can be found in Esposito (2013, pp. 519–523).

(usually normally distributed) *blending* inheritance. For instance, the dimension of crab morphology which Weldon took to be evidence for the action of natural selection (a measure across the front of the crab which Pearson persistently referred to as its 'forehead') was clearly a blending character. But it was well known to any student of inheritance that characters were often passed on in at least two other primary manners. The least common is what Weldon called *particulate* inheritance, not to be confused with theories about the underlying material basis of heredity (the sense in which 'particulate' would be used by authors including Galton and Fisher).[14] For the biometricians, this describes the production of variegated patterns, which combine, without mixing, both of the elements received from the parent.

The data provided by Mendel follow a third pattern, which Weldon calls *alternative* inheritance, where the two characters possessed by each parent form mutually exclusive alternatives for the offspring. The most common, everyday example of alternative inheritance that featured in biometrical discussions was eye color, though perhaps the best described case, already alluded to above, was Galton's data on the inheritance of coat colors in Basset Hounds (Galton, 1897).

Weldon, writing a few years later but clearly expressing the position that had always been the default for the biometricians, would accurately note that unlike in the case of blending inheritance,

> Our knowledge of *particulate* or mosaic inheritance, and of *alternative* inheritance, is however still rudimentary, and there is so much contradiction between the results obtained by different observers, that the evidence available is difficult to appreciate. (Weldon, 1902c, p. 228)

Mendel's work, thus interpreted, forms a welcome corrective and addition to the program of research that the biometricians were already undertaking.

We see the same story repeated when we investigate Weldon's correspondence with Pearson in the immediate aftermath of their first encounter with Mendel's papers. By some six weeks later, Weldon is contemplating how to incorporate these new alternative-inheritance results into their standard theoretical framework, in particular considering the ways in which the unions of different kinds of germ-cells (whether 'legitimate' or 'illegitimate', in his terminology) might produce results like those of Mendel (Weldon, 1900b,c).

But the "business-as-usual" frame begins to break up rather quickly, as Mendel became a subject of biological debate. On the 12th of December, now two months after their first encounter with Mendel, we see for the first time a glimmer of the program that will become a vital part of Weldon's research for the remainder of his life, until his untimely death in 1906. Weldon turns to questions about the *composition of gametes*:

> If I understand what you mean by gemmules, I certainly think they are necessary.
>
> I think that there must be an element in each gamete corresponding to every quality transmitted by it; some of these may blend with the corresponding elements of the other, some may exclude corresponding elements of the other, some may make a patchwork resulting in a particulate inheritance. (Weldon, 1900d, f. 1)

Weldon, that is, is developing the conviction that the kind of phenomena pointed out by Mendel – and, more generally, the various sorts that will need to be accounted for if the biometricians are going to successfully handle the full complexity of known modes of inheritance – indicates a missing link in the biometricians' theoretical system. They will need to develop, he argues, an account of the ways in which characters are carried by parents, assort into gametes, and then govern the

development of offspring. In short, just as Galton had been pushed to do a decade prior to ground his use of statistics, Weldon in turn is looking to the material grounding of inheritance, this time in order to make room for Mendel's results.

Over the next few years, though, Weldon does not make any significant advances on this project. He and Pearson are quite heavily occupied with the program of editing their fledgling journal, *Biometrika*. When Weldon does turn to analyzing Mendel's data in more detail, he's concerned with the way in which Mendel categorizes the phenomena, particularly the worry that the relevant strains of peas do not in fact separate as cleanly into groups like "green" and "yellow" as Mendel's theory required (Weldon, 1902c).

His thinking about the material basis of heredity, however, is jump-started by the publication of Bateson's *Mendel's Principles of Heredity: A Defence* (1902), which includes Bateson's most extensive and direct assault on Weldon's theoretical work. He realizes, I suspect, that the only way to fend off such attacks for good is to complete the work he had outlined several years before: to demonstrate that Mendel's results really do arise as nothing more than a special case of broader biometrical theoretical principles, grounded in the transmission of characters. As he puts the matter in a letter to Pearson,

> What Bateson does, and what all Mendelians do, is to take the diagram of frequency [Fig. 1] and to call a range AB one "character," and the range BC another "character" of a Mendelian pair.
>
> [...]
>
> There must be a simple relation between AB, BC, and the [standard deviation] of the original system, which would make the chance that a grandchild falls within BC $= \frac{1}{4}$? (Weldon, 1902b, f. 2r–3r)

Surely, Weldon argues, there must exist some way in which we can describe Mendelian inheritance in terms of the decomposition of the classic frequency curves with which the biometricians had already been concerned. If "recessive individuals" is just a name we give to a particularly extreme region of these bell curves, then we might be able to give an account in terms of the underlying elements that give rise to those bell curves which can reproduce Mendelian population ratios over time. And further, he goes on to argue, if we could then use such knowledge of the material processes of inheritance to understand why it is that some species transmit character traits in a Mendelian manner and others do not, such knowledge might serve as the basis of a predictive science of inheritance, one that would give us the ability to determine, for a particular individual case, the variability of offspring given knowledge of the variability of parents.

Once again, however, the project enters a period of dormancy, as Weldon returns to data collection on a vast number of new biometrical target species. The next major series of moves comes in 1905. Weldon has been invited to give a series of lectures (there would be seven in total) at University College, London, on the topic of "Current Theories of the Hereditary Process." While I will return in more detail to the content of those lectures in Section 2.4, for the moment it is important only to note that, prior to presenting Mendel's theory, he hopes to finally get clear on the question of segregation and assortment of elements.[15] To see how he hopes to do so, we should look back to early 1902, and add one more element to our understanding of Weldon's context.

2.3. Weldon and Correns

As we have already seen, the question of the transmission of characters from parents to offspring is by no means a new preoccupation for

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[14] In fact, Galton himself believed that pure blending and particulate inheritance, in his sense, formed something like two ends of a spectrum; see Porter (2014, pp. 128–129) and, for helpful context, Krashniak and Lamm (2017).

[15] Weldon never settled on terminology for these "elements," referring to them at times as 'chromomeres', 'gemmules' (though he dislikes the connection to Darwin's pangenesis), or collectively with Galton's term 'stirp'.
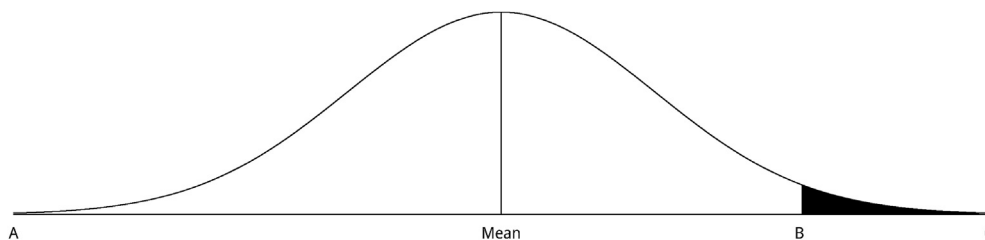
Fig. 1. Weldon's normal curve, redrawn from Weldon's letter to Pearson of June 23, 1902 (Weldon, 1902b).

Weldon – his late treatments of the question, both in his lectures and in the final notebooks and manuscripts to which I will turn in a moment, are broadly congruent with what Weldon had written as early as 1896 in his letter to Galton with which we began this section. One question that *is* difficult to answer, however, is just exactly what underlying account of the nature of the chromosomes Weldon has in mind. He never cites one, partly because he never published his thoughts on chromosomes in a finalized form, and partly because he was generally lax about citation. One connection of which we can be sure is perhaps representative. In a letter from April of 1902, Weldon mentions having read a paper by Correns which will "show exactly when the characters in the chromosomes arrange themselves according to Mendel" (Weldon, 1902a, f. 2r). I follow Sloan in reading this as a reference to Correns's recently published 1902 work on chromosomal segregation, which was at least therefore *among* Weldon's sources on the nature of the chromosomes (Sloan, 2000, p. 1076). Thin gruel, perhaps, but it will at least serve to give an idea of the kinds of resources that were available to Weldon as he developed his theory over 1905 and early 1906.[16]

Correns is directly considering precisely the question that has interested Weldon: how the elements (*Anlagen*) present in the chromosomes might segregate and assort over the process of cell divisions (both mitotic and meiotic). First, he describes the processes underlying normal divisions:

> We assume that, in the same chromosome, the two elements of each pair of traits lie next to each other (*A* next to *a*, *B* next to *b*, etc.), and the pairs of elements themselves lie in a row. A picture of this can be found in [Fig. 2, 'Fig. 1']. *A*, *B*, *C*, *D*, *E*, etc. are the elements of the first parent, *a*, *b*, *c*, *d*, *e*, etc. are those of the second parent. In a normal cell- or nucleus-division, which supplies similar products [i.e., similar parent and offspring cells], the longitudinal splitting of the chromosome takes place in such a way that each element is divided in half, in our picture in the plane of the paper. Each half then contains all the elements…. In germ cell formation, on the other hand, a longitudinal cleavage takes place once, which separates the attachments of the individual pairs … in our image thus *perpendicular* to the plane of the paper, in the line *xx*. (Correns, 1902, p. 304)[17]

Such a chromosome division could produce something resembling Mendelian segregation. On the other hand, we know that on occasion, characters appear to assort randomly. This would require an extension of the model, and the introduction of a second process, by which the individual pairs (*Aa*, *Bb*, etc.) are permitted to "spin" randomly around the central axis *xx*, producing any one of the 32 possible random combinations that could result (like the labeled 'Fig. 2' and 'Fig. 3' in my Fig. 2) with equal probability. If such spinning could stop at 90 or 270 degrees' orientation, not merely 0 or 180, then such a model could even account for cases in which some characters appear to assort randomly and others do not.

Fig. 2. Correns's model of the construction of chromosomes from their elements. Originally from Correns (1902); reprinted from *Comptes Rendus de l'Académie des Sciences: Series III: Sciences de la Vie*, Vol. 323, Hans-Jörg Rheinberger, Mendelian Inheritance in Germany between 1900 and 1910. The Case of Carl Correns (1864–1933), pp. 1089–1096, Copyright 2000, with permission from Académie des sciences/Éditions scientifiques et médicales Elsevier SAS.

It is obvious that, for Correns (and Weldon as well), this is not intended to be a theory of the ways in which elements in fact chemically behave when aggregated into chromosomes; no one is genuinely proposing that such pairs would spin around their central axis. That said, the general idea is particularly amenable to Weldon's aims. It retains the Galtonian idea that the relative position of elements within the stirp is vitally important, and it also offers a wide variety of processes by which the assortment itself could be modified, perhaps enough to reproduce the various different systems of blending and alternative heredity to which Weldon hopes to extend his theoretical scope.[18]

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[16] There is an extensive secondary literature on Correns's work, and I lack the space to carefully engage with it here. Correns himself is engaged in an interesting process of theoretical generalization. See especially the work of Hans-Jörg Rheinberger (2000, 2003, 2008).

[17] Translation is my own, rendering Correns's *Anlagen* as 'elements'.
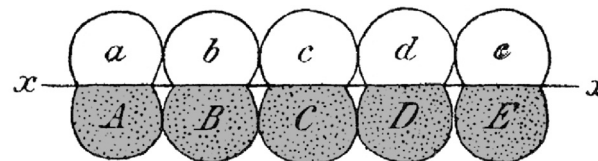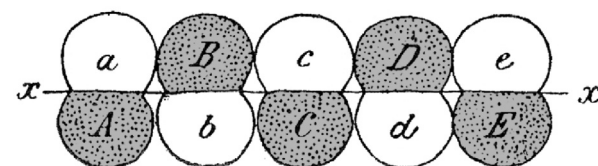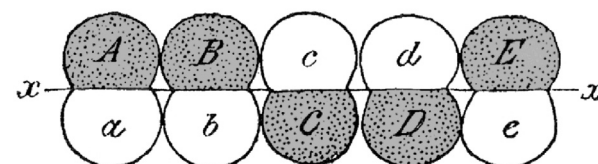
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[18] As he puts the matter in a letter to Pearson, Weldon believes that "a long account of the 'stirp' … wants doing, in answer to Bateson's statement that the Law of Ancestral Heredity is incomplete, because it does not give a theory of the constitution of gametes" (Weldon, 1905l, f. 2–3). This letter, dated April 17, 1905, is the last time that Weldon's work on the subject would appear in his correspondence with Pearson.

2.4. The 1904–1905 lectures and Theory of Inheritance

Let's return to Weldon's late work on his new approach to chromosomes and the transmission of characters. A full narrative detailing all of Weldon's own theoretical adaptations of a chromosomal basis for inheritance from 1904 to 1906 would run well beyond my available space, but I want in the remainder of this section to explore it in three different ways. In this subsection, we will consider the lectures, mentioned above, that Weldon delivered at University College over the course of 1904 and 1905. As it turns out, the structure of these lectures closely parallels the content of the book manuscript, *Theory of Inheritance*, that Weldon would leave unfinished upon his death; I thus consider that manuscript in this subsection as well. In Section 2.5, I look at a series of research notebooks in which Weldon attempted to work out some of the finer mathematical details of the theory. Finally, in Section 2.6, I look at a reconstruction of this theory that Pearson published in *Biometrika* in 1908.

Unfortunately, we have precious little information available to reconstruct the content of Weldon's lectures. The syllabus is available in the archives, but it contains only their titles and a short list of major subjects considered in each (Weldon, 1904). Anonymous accounts of the lectures were published in *The Lancet* over the course of 1905 (Weldon, 1905a,b,c,d,e,f,g,h), which give us a bit more detail.[19] It seems clear that the structure of the lectures was to roughly parallel the structure of a book manuscript that Weldon left unfinished at his death, tentatively entitled *Theory of Inheritance.* We only have a few chapters of this manuscript, however, and many of the most crucial moves for the story I am telling here were never written.

But what we see in these somewhat sketchy sources is already sufficient to give us an idea of Weldon's commitment to a vital role for cellular biology.[20] The very first lecture is billed in the syllabus as commencing with the "necessity of examining the visible phenomena of Reproduction and Development before considering the possible nature of hereditary processes," and explores cellular and developmental phenomena across hydra, sea urchins, fish, mollusks, and more – focusing on particularly strange cases such as repair and regeneration (Weldon, 1904, f. 2). The author recounting the lecture writes that while Weldon noted that a few facts about regression and population averages could perhaps be derived only using statistical tools, "when an explanation was sought of the mechanism or *modus operandi* of heredity, one passed, he said, outside the domain of statistics and had to picture the invisible organic processes accompanying the growth and the reproduction of animals" (Weldon, 1905a, p. 42).

The fourth and seventh lectures were those most directly covering questions of chromosome theory as we have discussed it here. The syllabus promises in the fourth lecture a comparison of two conceptions of heredity, the "pangenetic," represented by Darwin, Nägeli, de Vries, and (in a modified form) Galton, and the "segregative," represented by Mendel, Roux, and Weismann.[21] The roles of "nuclear phenomena" and the germ-plasm hypothesis were also to be discussed (Weldon, 1904, f. 2). In the seventh, Weldon proposed a "comparison between the behaviour attributed by Galton to the 'stirp,' and that actually demonstrated in the elements of a Sea-urchin's embryo" (Weldon, 1904, f. 3). Unfortunately, Weldon seems to have come somewhat apart from his syllabus by the time the lectures were delivered (a phenomenon with which readers are doubtless familiar), and the content promised for the fourth lecture was delivered in the fifth – apparently a fairly standard presentation of the treatments of chromosome theory by Weismann, Roux, and de Vries, without much direct connection to their evolutionary consequences (Weldon, 1905e). The empirically grounded comparison promised for the seventh lecture never materialized. A brief sketch apparently made it into the eighth and last lecture, which was described in the *Lancet* account as follows, recalling our discussion of Correns above:

> A formula of inheritance propounded and developed by Francis Galton gave better results [than Mendelian segregation]. The Galtonian theory postulated the presence of "elements" in the germ cells of one generation, which were of two kinds—viz., [active and latent. …] If the chromosomes of the conjugating germ cells divided always transversely into equal segments … the Mendelian hypothesis … would be always realized. But if the cleavage of chromosomes were horizontal qualitative difference would appear … and "mixture" of characters would invariably appear when different races or varieties were crossed. (Weldon, 1905h, p. 810)

Unfortunately, the corresponding portion of the *Theory of Inheritance* manuscript was never written, so we cannot turn to them for clarification here. We have there only the promissory note, after the introduction of Galton's basic idea of latent and patent elements, that "we shall see in a future chapter how far the suggestion made can be brought into harmony with the facts of germinal structure which have been discovered during the last thirty years" (Weldon, 1905n, ch. 2, p. 3, f. 67r). None of these resources, then, give us that much clarity concerning Weldon's thought on the relationship between chromosomes and inheritance.

2.5. The late theoretical notebooks

The best source available to us for reconstructing the detailed argument that Weldon hoped to advance in these lectures and the book is a series of papers and research notebooks that were written over the course of 1905.[22] After opening a research notebook in January of that year by noting that phenomena of regeneration entail that it is impossible to support a theory whereby a given nucleus can produce exactly one type of cell (that is, exactly the same complaint he had made to Galton in 1896), he continues by returning to Galton:

> 2. — The above facts do not invalidate [the] conception of nuclear elements as a series of *stirps,* in Galton's sense, each containing something capable of exciting the development of *any* of the somatic characters, according to its position in the organism.
>
> 3. — It seems necessary to regard a *stirp* as capable of exciting, not only somatic characters like those of its parents, but characters like those of its more remote ancestors, under certain circumstances.
>
> 4. — It is evident, from the facts of growth and regeneration, that the characters of any one stirp which become active in any one generation are determined by the position of that stirp with reference to the rest – i.e., by a process of the same nature as Mendelian "dominance." (Weldon, 1905m, f. 1r)

Weldon thus remains convinced that a series of elements, arranged into chromosomes, can play exactly the role that Galton had laid out for a stirp, and can account both for phenomena of ancestral inheritance (by instantiating the Galtonian properties of patency and latency in each element) and phenomena of Mendelian dominance (via the repetition of relationships between stirps across multiple generations).

What now remains to be done is the mathematical heavy lifting.

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[19] Note that I follow common practice in attributing these sources to Weldon himself, as he is the author of the lectures, but the real author of the anonymous accounts is not known, even to the journal's editors, who normally retained authorship records for anonymous pieces (pers. comm.).

[20] And, perhaps of equal importance, to developmental biology, a connection that I hope to pursue in future work.

[21] It is interesting to note here the impact of Weismann's theory of the sorting of determinants, discussed in Section 2.1, on Weldon's reading of Mendel.

[22] Interested readers may find a reconstruction containing the full mathematical details in the scholarly edition of Weldon's *Theory of Inheritance* manuscript (Radick et al., in preparation), and a broader philosophical treatment in Pence (2022a, ch. 4).

Weldon assumes that the gamete is composed of n chromosomes, each of which is constructed from p different elements. He also considers two hypotheses by which chromosomes themselves might be built – one on which the physical chromosome remains intact across cell divisions, and another on which, during cell divisions, the chromosomes themselves dissociate into their separate elements, which are then recombined into groups of p, independent of their prior chromosomal membership. It's clear that Weldon hopes that by varying the values of n and p, and the processes of chromosomal formation, he can produce dramatically different resulting distributions of characters – some of which could lead to Mendelian ratios for dominant and recessive transmission, and others of which could lead to the traditional patterns of blending inheritance with which Weldon was used to working.[23]

There is, unfortunately, a problem: the mathematics simply don't work. The difference between a normal distribution of characters on blending inheritance and a Mendelian distribution is significant – that is, we would need to see *radically* different results from a hypothetical breeding experiment as we varied the model of chromosome formation and the values of n and p. The trouble is, we don't – and this leads to the production of patterns of inheritance that don't offer nearly enough "pure" dominant or recessive individuals. After having chosen one method of chromosome formation and having performed the copious, long-hand arithmetical calculations of offspring trait distributions, Weldon writes to Pearson that

> I have laboriously worried through the effect of supposing the chromosomes to retain their individual constitution right on from the moment of fertilisation to the formation of new germ-cells; and it does not give anything like a proper segregation: there are very few "pure" individuals, among either "dominants" or "recessives." (Weldon, 1905i, f. 1)

In Fig. 3, I have reproduced three of the example cases with which Weldon worked. Two assume that the elements remain connected into chromosomes throughout the process of reproduction (the "individuality" or "individual constitution" of the chromosomes), with two different sizes of chromosome; a third assumes that all the elements disassociate, are divided between the germ cells, and then re-form into chromosomes.[24] But all three of these graphs only differ very slightly. We do not yet have a mathematical structure that can yield biometrical results for some cases and Mendelian results for others.

Weldon has, notably, a proposal for how to fix this – by reintroducing yet another Galtonian insight (Weldon, 1905j). Absent from any of this discussion, so far, has been Galton's assertion that the fact of an element's being patent in the current generation somehow renders that element more likely to be patent in future generations. This "valency," as Weldon dubs it, could – he hopes – offer us a way to more significantly alter the curves of element distribution. But it would require mathematical treatment that Weldon does not know how to perform. Weldon's project, at least as he was conceiving of it in these notebooks and letters, has failed to produce a formal system that can be tractably analyzed by the kinds of mathematical tools that were available to him at the time. And what's worse, he's out of time: he has to deliver his lectures on Mendel, and so he shelves the calculations for the time being.

Anyone familiar with the history of biometry will know that, unfortunately, further work on the question was never done. After dedicating himself for several months to the analysis of volumes and volumes of horse-coat data, dramatically overworking himself in spite of illness, Weldon dies of pneumonia in April 1906, at the age of forty-six.

2.6. Pearson's reconstruction

Pearson attempted, to the best of his ability, to print what work he could salvage from the notes of his late colleague, including both Weldon's notes on the inheritance of mouse color (another experimental program Weldon had extensively pursued before his death; Pearson, 1907) as well as Weldon's notes on chromosomes (Pearson, 1908). We learn from Pearson's paper that he had prepared a "mathematical draft" on the basis of their conversations about chromosomes, which "was finally taken by Weldon from Ilsley to Oxford to be rewritten with proper biological terminology" (Pearson, 1908, p. 80); this revision was never completed, and as far as I am aware the document to which Pearson refers no longer survives in the archives. Pearson notes that he has worked largely from Weldon's notes (the same notes which I have reconstructed above), and in the absence of any discussion with "a cytologist with an interest in and a knowledge of the theory of chance at all comparable with Weldon's" (Pearson, 1908, p. 80). What we have, then, is Pearson's attempt to give a coherent reconstruction of the mathematics that he found in Weldon's notebooks.

Of the three kinds of cases that I described above in Fig. 3, Pearson picks up a generalized case encompassing two of them (I suspect because the mathematics for the third were either more challenging, or because Pearson suspected that Weldon had made errors in his analysis, though Pearson's reason for doing so is not clearly stated).[25] In his own terms, Pearson reworks and cleans up the mathematical derivations performed by Weldon (Pearson, 1908, pp. 83–84), and then provides a number of fully worked-out examples. Notably, he considers several cases that were *not* present in Weldon's notes, some of which approach more closely Mendelian segregation patterns than any that Weldon had developed himself.

He then turns to a question that Weldon had posed in hopes of getting traction on the evolutionary dynamics of these cases: what would the standard deviation of these distribution curves be, and how closely would they approximate normal curves? (If nothing else, determining this could make possible the application of some of the analytical tools which Pearson and the other biometricians had developed for normal curves to this new case.) Pearson then moves beyond anything that Weldon had pursued in his notebooks, noting that the form of normal curve derived here parallels a case that was developed elsewhere by Pearson, and then deriving some initial values for correlations between parents and offspring.

The biological conclusions that Pearson derives on this basis are somewhat peculiar. Deriving the consequence that, on this view, there will be a correlation between features of somatic cells and those of gametes, he explains this correlation with the following gloss:

> … from this standpoint the somatic cell precedes the germ cell of the individual, and the somatic cell might, under the proper stimulus, give rise to a germ cell. These germ cells are not of one type; they are variable, but correlated with the originating somatic cell. It is difficult, if we look at matters for the time being from this aspect, to find any basis for a "continuity of the germ plasm." (Pearson, 1908, p. 91)

It is not at all clear why Pearson believes that this biological grounding is a reasonable extrapolation from the existence of a correlation between somatic cells and gametes. Given the way in which Weldon had discussed Weismann's work (see Section 2.1), I think that Weldon would have likely disagreed. Pearson closes the work by extending the correlations to later generations.

Setting aside the odd biological conclusions which Pearson draws, he describes the overall goal of Weldon's project in a very similar way to that

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[23] A version of this structure for the relationship between chromosomes and characters is presented in a letter to Pearson as a kind of card game; see Weldon (1905k).

[24] As is by now well known, the question of chromosome individuality, as a portion of the broader argument concerning the continuity of inherited structure or material across generations, was regularly discussed in the cellular biology of this period (e.g., Churchill, 1987, pp. 354–357).

[25] More precisely, Pearson ignores the case marked "no individuality" in Fig. 3; he is assuming that chromosomes remain intact throughout the processes of cell division, rather than passing through a phase in which the elements of which they are constituted separate from one another and form a kind of single, shared "pool" of elements.
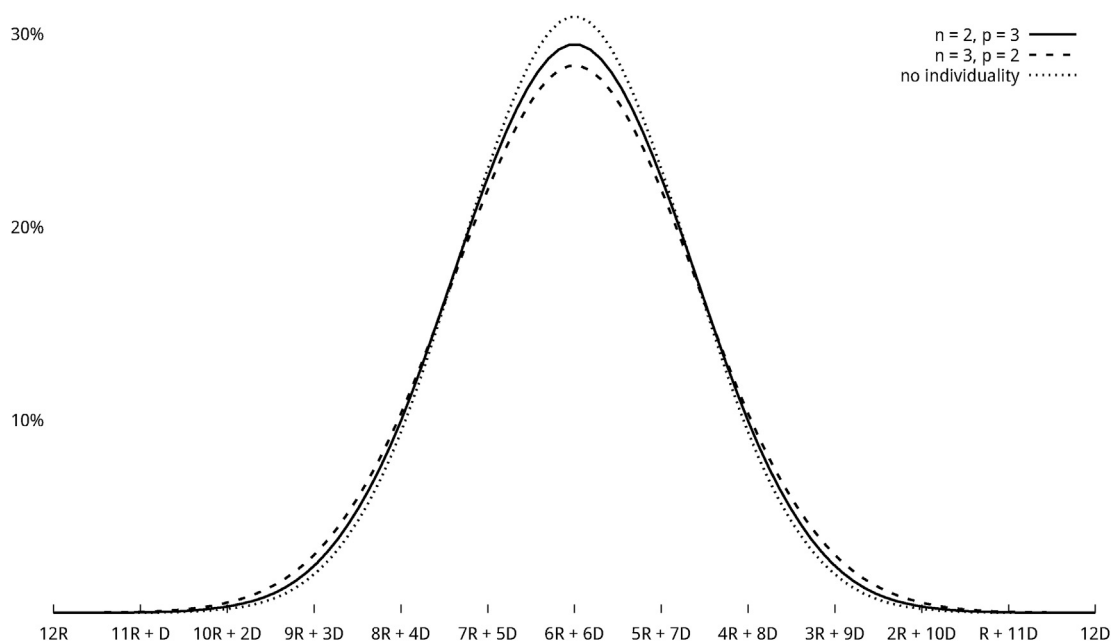
Fig. 3. The distributions of elements in offspring gametes, according to the three hypotheses for chromosomal size and constitution which Weldon considers in his research notebooks of 1905. The x-axis describes the distribution of elements in chromosomes, where, e.g., "7R + 5D" indicates a chromosome formed of seven "recessive" and five "dominant" elements. They produce ratios of "dominant" individuals differing by less than 2%.

which I have laid out thus far. In his words, "we see a continuous transition from simple Mendelism, through various phases of pseudo-Mendelism to distributions closely following the normal curve. […] If the hypothesis here dealt with were correct, it would follow that the Mendelians were merely working at one end of the scale, the biometricians somewhat further down" (Pearson, 1908, p. 93). It is notable that this concession to Mendelism (and a further note a few sentences later that "Mendelian literature for the careful reader may provide answers" to questions about Mendelian ratios in non-true-breeding populations) represent some of the most conciliatory moments in all of Pearson's writings.

3. From underlying details to generalizations

Let's pause to briefly sum up. Galton, in search of a replacement for the role that he had hoped Darwin's pangenesis would play in his statistical account of inheritance and natural selection, proposes the *stirp* as the carrier of whatever physical substance would be responsible for the transmission of characters from parents to offspring. As we saw in his exchanges with Weldon, he seems to go back and forth on the question of how exactly the content of this stirp is responsible for producing differentiated cells: sometimes this is due to the position of elements within the stirp, sometimes via a process of sorting of elements responsible for a particular cell type during division. With respect to the transmission of those stirps from parents to offspring, he relies upon analogies with colonial governance to give us a vague idea of the random sampling procedure that he seems to have in mind.

Weldon, with the aid of a few very important further years of development in cellular biology, can be more precise. The elements of Galton's stirps are carried by chromosomes, and experiments in developmental biology mean, he thinks, that strict "sorting" must not be taking place – every stirp can, at least potentially, produce any somatic cell type, depending especially on position-based effects. Further, Weldon hopes (though these hopes are never fulfilled) to derive a formula which could, given the number of elements present within a chromosome and the number of chromosomes, reproduce both Mendelian and non-Mendelian, blending patterns of inheritance.

Already, one claim for which I hoped to argue in this paper should by now be fairly straightforwardly established. The underlying material

basis of the transmission of characters was a constant theme throughout biometrical work – by no means did biometrical authors want to simply abstract away from and ignore any such detail by working exclusively at the population level, or exclusively with statistical distributions of character traits. Further, they remained in dialogue with the latest advances in cellular and developmental biology. Galton, who predated many of the most important empirical advances, works first with Darwin's pangenesis, and then with the best abstracted account he can muster in the theoretical vacuum which follows the rejection of pangenesis by the broader biological community. Weldon's thinking about chromosomes is taken at least in part from Correns, and he was hoping to theorize in an entirely innovative way about the properties of chromosomal elements just prior to his death.

In this last section, I want to consider a commonality that underlies both Galton and Weldon's having made exactly the same kind of turn toward the material basis of their otherwise statistically phrased understandings of inheritance. In both cases, I claim, these authors were pushed to think about the material basis of heredity in part by the search for *generality* for their theories.

3.1. Generality in the philosophy of science

To begin, I should pause to introduce the concept of generality and consider some of the roles that it has played across the philosophy of science. First, generality has often served as a desideratum in scientific explanation. Beginning, for instance, with the traditional deductive-nomological model as offered by the late logical positivists (Hempel & Oppenheim, 1948) and continuing through theories like Kitcher's approach to scientific explanation as unification (Kitcher, 1981), generality or generalization is taken to be important to the very act of offering a scientific explanation, although the function that this generality plays differs. For Hempel and Oppenheim, generality allows us to connect explanation with prediction. As they write,

> Only to the extent that we are able to explain empirical facts can we attain the major objective of scientific research, namely … to learn from [phenomena], by basing upon them theoretical generalizations which enable us to anticipate new occurrences and to control, at least

to some extent, the changes in our environment. (Hempel & Oppenheim, 1948, p. 138)

It is thus theoretical generalizations that allow us, by permitting extrapolation from precise details, to pass from explanations of phenomena to prediction and control, and in so doing to arrive at the true aim of science.

For Kitcher, generalization – the construction and usage of general argument patterns – is introduced as part of the explication of the concept of *unification*. By generalizing the *patterns of argument* present in Newtonian mechanics, and thus searching for other explanations that could be constructed in terms of inertial states and perturbing forces, early Newtonians were engaged in precisely the search for explanatory unification that Kitcher thinks is essential to the scientific process:

> Newton's successors were trying to generalize the pattern of argument presented in *Principia*, so that one "kind of reasoning" would suffice to derive all phenomena of motion. If, furthermore, the facts studied by chemistry, optics, physiology and so forth, could be related to facts about particle motion, then one general pattern of argument would be used in the derivation of all phenomena. (Kitcher, 1981, p. 514)

For Kitcher, then, generalization helps us construct theories, by giving us a reasonably economical collection of arguments which we can draw upon to offer explanations in a particular context. This implicit appeal to economy resonates with Pearson's own approach to the philosophy of science. While Pearson would construct his response to the question on a largely anti-realist foundation, claiming that scientific theories make reference only to "the contents of the mind, the 'inside' world" (Pearson, 1900, p. 67), he would nonetheless argue that "the object discovered by the discovery of [scientific] laws is the economy of thought" (Pearson, 1900, p. 78), and that "the regular course of scientific progress" shows us that "the scientific law … is always liable to be replaced by a wider generalisation" (Pearson, 1900, p. 99).

Even pragmatist or deflationary accounts of scientific explanation – such as, most famously, that of van Fraassen (1977, 1980) – still underline the importance of generalization in scientific knowledge. If explanation is, on such a view, (nothing more than) the providing of satisfactory answers to why-questions, then at least part of what might make such an answer satisfactory is the demonstration that a phenomenon follows in virtue of the same kinds of causes, laws, or principles that we think are at work in other, related cases (van Fraassen, 1980, p. 154).

We can turn then from this more epistemic understanding of generalization – generalization in the service of explanation – to generality as a putatively metaphysical feature of the world, often connected to the consideration of the discovery of *real patterns* in nature. Of course, as Potochnik notes, following Giere, "patterns are not universal and exceptionless but limited in scope and permit deviations and exceptions" (Potochnik, 2017, p. 25). In order to offer a theory of generality in this sense, then, we must stop to consider the ways in which the patterns in the world described by science might fail to be general. As Potochnik describes it, restrictions on the scope of scientific generalities are two-fold: such generalizations "hold only in limited circumstances, and most also have deviations and exceptions even within those circumstances" (Potochnik, 2017, p. 26). This arises directly from a tension that the invocation of generality in explanation makes clear: the more that we abstract or simplify a generalization in order to make it illuminating or epistemically useful, the more likely we will be, in turn, to require exceptions to those very generalizations.

Generality is therefore a complex scientific concept, which has played a role both in our understanding of scientific explanation and in the attempt to understand the real patterns in the natural world which science attempts to describe. It is particularly apt for philosophical analysis, as it clearly demonstrates a sort of classic tension – one perhaps most familiar from debates over scientific pluralism (Cartwright, 1994) – between scientific theories as manageable, illuminating objects of human construction and scientific theories as accurate and complete descriptions of the natural world. Many philosophers of science now agree that theories meeting both these desiderata will be extremely rare, if not impossible to find, and thus there is substantial work to be done in examining the trade-off between them from a philosophical perspective.

3.2. Generality in Galton and Weldon

Let's return to the particular case of the authors that I have explored here. Both Galton and Weldon seem to have made deliberate theoretical moves, with the aim of increasing the generality of their theories. In this sub-section, I'll detail exactly what those turns toward generality looked like, before turning in the next sub-section to the question of how to place the concept of generality that I find there in dialogue with the philosophical discussions above.

Galton. For Galton, any statistical theory – and, *a fortiori*, any statistical theory that could be generalized and applied to a large number of cases in heredity and evolution – had to be legitimated by demonstrating that the underlying units of which a population was composed behaved in the appropriate sort of way. On his Quetelet-inspired approach to statistics, it had to be shown that the elements to be treated statistically have a very peculiar set of properties and interactions – that is, that they either behave something like the aggregation of a large number of independent, small effects, or (which Galton believed to be more likely later in his career) that we have an explanation for how they can produce normal distributions *in spite of* the fact that they don't behave in such a way.

In the case of heredity, however, this effort to produce a statistical theory of the transmission of characters from parents to offspring didn't work. There were two primary problems. First, this interpretation of inheritance as behaving something like a "law of error" is, in essence, phrased as a conditional: *if* the distribution of hereditary elements in offspring germ cells is the result of a large number of small causal influences, *then* this would guarantee the (re-)production in the future of normally distributed offspring characters. But Galton recognized that it would in reality be quite difficult to consider the impact of each element on later organismic development as "small":

> [A]lthough characteristics of plants and animals conform to the law, the reason of their doing so is as yet totally unexplained. The essence of the law is that differences should be wholly due to the collective actions of a host of independent *petty* influences in various combinations…. Now the processes of heredity … are not petty influences, but very important ones. (Galton, 1877, p. 512)

Galton therefore needed a way to show that, in spite of the fact that elements might have very significant impacts on the future characters of an organism, they will, in the end, follow the laws of statistics nonetheless. He never offered any such explanation, relying only on either physical analogies like his quincunx device (Galton, 1889, pp. 63–65) or on vague conceptual analogies such as the relationship between the people of a country and its elected representatives, as already discussed above. Despite Galton's insistence that "these are not idle metaphors, but strict analogies" (Galton, 1876, p. 336), they still do not give us anything like an argument for the claim that the processes of character transmission will behave in such a way as to license the inferences that Galton draws from them.

The second reason for Galton's failure to produce a statistical theory, as briefly mentioned before – again a trait inherited from his reliance on Quetelet's statistics – was his emphasis on the idea that it is the *stasis* of the natural world that is remarkable. Species are largely unchanged over generational time, and the same sorts of normal curves reappear regardless of the apparent differences between individual parents and their offspring. (One is reminded of Quetelet's insistence that the number of murderers in Paris was a matter of mathematical law, calculable in advance.) But this leaves Galton able only with difficulty to approach evolutionary change in general, and natural selection in particular. He

thus was left with a kind of unstable saltationist approach to selection, on which centers of organic stability, represented by different possible normal distributions of characters, would rapidly change from one to the other, driven by selection (Bowler, 2014). It isn't clear whether Galton could have resolved this apparent paradox without a different approach to the foundations of statistics.[26]

Weldon. For Weldon, on the other hand, the acute need to generalize the biometrical theory with which he had been working was brought on by the arrival of Mendelism. Biometry – which had largely been preoccupied with blending inheritance – needed a way to incorporate alternative inheritance, at least as a special case. At least some instances of Mendelism in natural populations were simply too well confirmed to be ignored, even by opponents as convinced as Pearson and Weldon, and Weldon saw in a careful treatment of the transmission of particular numbers of chromosomal elements from parents to offspring a potential way to recover the widely varied patterns of inheritance that the biometricians had so carefully catalogued in the preceding years.

This effort to encompass Mendelism into the biometrical fold appears to have fared little better than Galton's construction of a general theory of inheritance. Weldon's initial mathematical failure, as I have described it, left him with a "generalized theory" of chromosomal inheritance that actually wasn't very general at all, insofar as it did not produce relationships between parents and offspring that differed significantly from those already discussed in biometry – indeed, it had an unwelcome tendency to stubbornly produce exactly the same kind of normal curve time and again. This theory (as I can confirm, having re-derived the mathematics even with the aid of today's computer programs for statistical calculation) is cumbersome and unwieldy, and adding the kind of "valency" to it that Weldon had proposed at the end of his life would only have served to compound these problems, introducing into the model a number of new free parameters to describe the ways in which the latency and patency of elements would be preserved or attenuated over time.

As Morrison (2002) has compellingly argued, however, this is perhaps to have been expected. For Pearson, she writes, we consider the nature of inheritance by a process of generalizing or averaging that starts with real-world individuals, "not only with respect to the individuals we are investigating, but also the experiences we have of them, no two of which exactly agree" (Morrison, 2002, p. 64). In this way, we find ourselves with statistical descriptions of characters, but not *idealized* descriptions – we remain closely tied to the real-world population with which we began.[27]

In Weldon's theorizing, this took the shape of beginning with the constitution of the gametes of two types of parents and deriving the distribution of all the characters present in (or, better, patent in) all of the possible offspring to which they might give rise.[28] Morrison notes that this aspect of the biometrical world-view constitutes an important distinction between the biometricians and the statistical approach of someone like R. A. Fisher, for whom populations become the focal, now idealized object, capable of possessing properties like hypothetical frequencies or behavior in the infinite limit of population size. This kind of understanding of a population is simply not on the table for Weldon, and thus one might reasonably infer that his mathematical efforts to generalize a simple theory of blending inheritance on the basis of the transmission of chromosomal elements were always, in some sense, doomed to fail.

3.3. Generality through detail

There thus appears in these two cases a kind of common thread – though one that might at first appear paradoxical. Both Galton and Weldon are faced with threats to the kind of generality that they hoped their theories could reach, whether as a generally applicable statistical theory of heredity or as the search to integrate Mendelism with biometry. One particularly obvious way in which they *might* have decided (but as we will see below, did not in fact decide) to guarantee the generality of their theories has been regularly discussed in the philosophical literature. Either Galton or Weldon could have chosen to double down on the role of their speculations about chromosomes as *idealizations*. As Potochnik writes, "*idealizations* are assumptions made without regard for whether they are true and often with full knowledge they are false" (Potochnik, 2017, p. 42). Applied to the case here, one might have thought that these authors would have argued for the abstract nature of their higher-level, statistical descriptions of inheritance and natural selection, and in so doing both admit and dismiss the fact that these explanations could not be cashed out in terms of the underlying details of biological populations.[29]

But in neither case did these authors make this move, further distancing their statistical explanations from the underlying biological details. Both, on the contrary, thought that the route to statistical generality passed exclusively through a *more comprehensive* grounding in biological detail. Thus the paradox: when we talked about generality above, we saw emphases on prediction, on understanding and economy of thought, and on the balance between capturing real-world complexity and (cognitively beneficial) simplification. Neither Galton nor Weldon seems to approach generality in this way (which has, it would seem, more to do with idealization in Potochnik's sense). Somewhat unexpectedly, both of them turn instead back toward chromosomal and cytological detail, apparently the opposite of idealization.

Why might this have been the case – why might these authors have rejected a picture of their chromosome work as idealization? Helpful light can be shed on the question by returning to Potochnik's discussion of the motivations for idealization. As she notes, there are a plethora of reasons for which one might decide to idealize a system rather than studying it in its full complexity (Potochnik, 2017, p. 48).[30] One might hope to avoid the obligation to engage with an exceedingly complex causal structure, or be limited by computational power (a restriction that we saw would have likely been relevant for Weldon). One might think that our cognitive limits or pedagogical goals require us to idealize. More importantly for my case here, one might think that the idealization suffices to capture the core causal influences at work in the system, or one might think that the idealization enables a desirable, general application of the theory.

It is in these last two potential motivations for idealization that I think we see most clearly, by contrast, the reasons that Galton and Weldon would have chosen *not* to idealize. For Galton, because of his understanding of the foundations of statistics, an idealization *wouldn't* have permitted a more general application of his theory of heredity – at least, it would have left open, perhaps permanently and fatally open, the question of the conformity of the processes of inheritance with his statistical tools. For Weldon, an idealization *wouldn't* have captured the core causal influences at work in inheritance. Because he remained so thoroughly preoccupied with cellular and chromosomal structure, he simply didn't seem to think that any theory that idealized away from these influences could possibly offer us a useful way in which to approach a theory of heredity.

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[26] For an illuminating discussion of the counterfactual history in which Galton does develop such a theory, one can consult the discussion of "Galton's Law as it should have been" in his biography by Bulmer (2003, pp. 247–250).

[27] I will return to the relationship between these non-idealized generalizations and Potochnik's theory of idealization in the next subsection.

[28] Notably, this is a methodological step very similar to that taken by the Mendelians, who drew this same kind of individualized approach to inheritance from Mendel himself.

[29] Such a reading of abstracted statistical explanations has, I think misleadingly, often been attributed to Galton (Ariew et al., 2015; Hacking, 1990). For a complete argument against this interpretation of Galton's work, see Pence (2022a, ch. 2).

[30] I will only mention a selection of the reasons that she presents here, and Potochnik argues that even the full list that she presents is not definitive.

What kind of inferential role could these non-idealized generalizations then be able to play? This is a difficult question, as it brings us toward a large and complex literature on the role of representation and inference in scientific models, which would take me too far afield to engage with here (e.g., Frigg, 2006; Hughes, 1997; Matthewson & Weisberg, 2009; Weisberg, 2013). Again, I think a comparison with the inferential role described by Potochnik for idealizations is illuminating. She notes that representation in the case of idealizations seems not to be representation of the system as it is, but rather *representation as-if*, on which we "represent a system as if it were ideal" in some particular sense (being frictionless, being infinite, etc. Potochnik, 2017, p. 52). It is in this way that idealizations can help us "indicate the nature of a factor's relevance to the focal causal pattern" (Potochnik, 2017, p. 54), by, for instance, comparing the behavior of the system as it is with the behavior it would exhibit if it were idealized.

Continuing the comparison, then, the kind of generalization for which Galton and Weldon were searching is not representative in this kind of way – it very much does not seek to represent the populations *as if* they acted in some way in which they in fact do not, or as if they possessed some kind of idealized property. Rather, both Galton and Weldon became convinced, for independent reasons, that they would be able to deploy non-idealized statistical generalizations, generalizations that were constructed starting from the basis of cellular fundamentals.

Perhaps the most compelling explanation for their choice is in terms of epistemic and non-epistemic value differences – an explanation for differing preferences surrounding idealization already explored by Potochnik. As she notes,

> The emphasis that different research programs place on different causal patterns results in different choices of idealizations and of elements that are intended to represent realistically (in some regard, to some extent). Different standards govern the epistemic acceptability of posits playing these different roles. (Potochnik, 2017, p. 200)

As the philosophical context in which the two men worked changed dramatically over the end of the nineteenth and beginning of the twentieth centuries, we might expect, then, a corresponding change in the ways in which they would understand idealization.[31]

In sum, considering the turn toward chromosomal and cellular details in the work of Galton and Pearson has allowed us to see a genuine continuity which lies beneath a profound difference between the theoretical approaches of the two men. Galton's heavy debt to Quetelet and his lack of available empirical results in cellular biology meant that his push for a detail-grounded conception of evolution remained at the level of what we might call a suggestive metaphor. While Weldon's more sophisticated (one might say Pearsonian) understanding of statistics allowed him to avoid Galton's uneasy commitment to saltationism and stasis, his "individualistic" understanding of character transmission meant that his effort at grounding a general theory of statistical biology was, in turn, never capable of getting off the ground. But the two were linked by a rejection of idealization, and a concomitant drive to consider the nature of the hereditary elements underlying their theories in the pursuit of building more general and generalizable theories of evolution.

4. Conclusion

I hope to have demonstrated here one particular inadequacy of some of the traditional historiography surrounding the "biometrical school." The work undertaken by the biometricians centrally involved much more than the narrow search for population-level statistical patterns. They were well informed about a wide swath of contemporary biological work, including cellular and developmental biology. They intended not only to produce theories of statistical evolutionary change, but also pursued – just as the

Mendelians of their day did as well – highly general approaches to the constitution and changes of evolving systems, at every level from the cell to the population. And these two enterprises were related: the necessity of deploying these approaches to the elements of inheritance was produced precisely by the drive for theoretical generality.

At a historiographic level, then, the ways in which we have conceived of this period in the history of the life sciences seem to fall short when we explore the work done by these biologists in anything like the detail it deserves. Important distinctions – for instance, between the different members of the biometrical school – are glossed over, and important commonalities are left unexplored. For example, after an analysis like the one I have sketched here, the possibility is opened for an examination of the different ways in which biometricians and Mendelians alike chose to take up results from early cellular and developmental biology like those of Boveri and Driesch.

Most broadly of all, we see here two authors engaging with a perennial question in the philosophy of biology: Do the underlying details that produce a statistical phenomenon like evolutionary change actually matter? If they do, how? Far from being an esoteric question in the philosophy or metaphysics of science, this is a worry that arises for both Galton and Weldon as an integral part of their practical work, and I think their example can offer us the prospect of pursuing this question across the history of biology.

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[31] I have considered a similar comparison of this sort, although in a different context, elsewhere; see Pence (2022b).

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