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August Weismann on Germ-Plasm Variation *

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Abstract. August Weismann is famous for having argued against the inheritance of acquired characters. However, an analysis of his work indicates that Weismann always held that changes in external conditions, acting during development, were the necessary causes of variation in the hereditary material. For much of his career he held that acquired germ-plasm variation *was* inherited. An irony, which is in tension with much of the standard twentieth-century history of biology, thus exists – Weismann was not a Weismannian. I distinguish three claims regarding the germ-plasm: (1) its continuity, (2) its morphological sequestration, and (3) its variational sequestration. With respect to changes in Weismann's views on the cause of variation, I divide his career into four stages. For each stage I analyze his beliefs on the relative importance of changes in external conditions and sexual reproduction as causes of variation in the hereditary material. Weismann believed, and Weismannism denies, that variation, heredity, and development were deeply intertwined processes. This article is part of a larger project comparing commitments regarding variation during the latter half of the nineteenth century.

Keywords: August Weismann, development, evolutionary developmental biology, externalism, genetics, germ-plasm, heredity, inheritance of acquired characters, nineteenth century, sexual reproduction, variation

August Weismann is remembered mainly for three theses that he putatively expounded: (1) acquired characters are not inherited, (2) variation in the hereditary material arises solely as a consequence of internal hereditary processes, such as sexual reproduction, and (3) the germ-plasm is continuous within and between generations. Geneticist and embryologist T. H. Morgan alluded to the first two theses in a lecture he gave as the president of the Sixth International Congress of Genetics in 1932:

> Weismann's theoretical contributions have also played an important historical rôle. "The Continuity of the Germ-plasm" [1885] served to

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> counteract the all-too-prevalent influence of Lamarck and his successors, whose views if correct would undermine all that Mendel's principles have taught us. Weismann's speculations on the origin of new variations by recombination of elements in the chromosomes, while not to-day acceptable as stated by him, nevertheless focused attention on an important subject. His discussion of the interpretation of the maturation divisions played, I believe, a leading rôle in directing attention to a subject that was destined very soon to have great importance for genetics.[1]

A generation later, the historian of biology William Coleman wrote: "By removing the germ-plasm from the cell at large and assigning it to the nucleus, Weismann reversed his earlier views and turned to exclusively internal causes of development, heredity, and variation ... Variation could not be induced directly from the exterior but required an internal source."[2]

The "earlier views" Coleman referred to were those presented in Weismann's *Studies in the Theory of Descent* (1882; German publication, 1875–1876). Coleman held that the mature Weismann ascribed variation solely to internal sources. These two examples, originating from important authorities, indicate that theses (1) and (2) were, and are, understood as central to Weismann's beliefs about heredity and variation.

August Weismann's views are succinctly expressed in a passage from the English translation of his 1892 book, *Das Keimplasma. Eine Theorie der Vererbung*: "The ultimate source ['letzte Wurzel'] of *variation* is always the effect of external influences ['äussere Einflüsse']. Were it possible for growth to take place under absolutely constant external influences, variation would not occur; but as this is impossible, all growth is connected with smaller or greater deviations from the inherited developmental tendency. When these deviations only affect the soma, they give rise to temporary non-hereditary variations; but when they occur in the germ-plasm, they are transmitted to the next generation and cause corresponding *hereditary variations in the body*."[3]

Variation both in the soma and the germ-plasm required pervasive and perennial changes in external influences. Although somatic variations were not heritable, germ-plasm variations were. Germ-plasm variations caused by changes in external influences, Weismann believed, were the only kind of genuinely novel germ-plasm variations. Weismann was thus not a Weismannian – he championed what I will call "the inheritance of acquired germ-plasm variations."[4] He was a nineteenth-century biologist who, like

[1] Morgan, 1932, p. 263; see also Morgan, 1926, pp. 30–31.

[2] Coleman, 1 965, pp. 153–154.

[3] Weismann 1893a, p. 463 and, for the German, 1892b, p. 609.

[4] Weismann used "inheritance of acquired characters" to mean *only* "inheritance of acquired *somatic* characters." These were cases, disparaged by Weismann, in which external

Darwin and others,[5] believed that changes in external conditions were the ultimate necessary causes of variation in the hereditary material.

In order to fully understand Weismann's theory of variation, we have to distinguish the continuity of the germ-plasm from its morphological and variational sequestration. These are three different concepts, as we shall see. Furthermore, we have to distinguish between changes in external influences and sexual reproduction as causes of variation in the hereditary material. Weismann championed the role of changes in external influences. However, he also held that sexual reproduction was important for the production of variation in this sense: it rearranged preexisting externally-caused variation in the hereditary material. *Changes in external conditions, acting during development*, ultimately caused all novel variation in the hereditary material.

Weismann synthesized an *externalist* view on the causes of variation with a *developmental* conception of heredity and variation. Externalism is the thesis that properties of, and relations in, organic systems can be explained in terms of properties of, and relations in, the conditions external to them.[6] An externalist hypothesis uses outsides to explain insides; an internalist one uses insides to explain other insides. Before his formulation of the germ-plasm concept in 1883, Weismann held that any phenomenon outside of the *organism* was external; he implied that any change in such phenomena could cause variation in the hereditary material. After articulating the germ-plasm concept, Weismann generally argued that any object or process outside of the *germ-plasm* counted as external conditions. The hierarchy of external conditions included the *soma* – germ-cell cytoplasm, somatic cells in general,

conditions caused a variation in the soma that was then transmitted to the germ-plasm and, subsequently, to the offspring [on his restrictive usage see, e.g., Weismann, 1891a (1883 essay), pp. 80–81; 1891a (1888 essay), pp. 425–426; 1893a, p. 392; 1904a, v.2 p. 63]. However, I will deviate from Weismann's narrow usage since the term "inheritance of acquired germ-plasm variations" accurately describes his views. In general, I prefer to use the term "variations," instead of the term "characters," to refer to changed properties of either the germ-plasm or soma, but these terms are interchangeable in this context. *The important distinction here is whether changes in external conditions cause a variation in the germ-plasm or in the soma*. Weismann's theory of the germ-plasm and Darwin's Pangenesis – each one a theory of generation which synthesized development, heredity, and variation in a unique manner – employed distinct postulated mechanisms and structures. Despite these differences, the inheritance of acquired germ-plasm variations, in Weismann's theory, is analogous to the inheritance of what I call "germinally-mediated variations" in Darwin's theory. Similarly, the inheritance of acquired somatic variations, in Weismann's theory, corresponds to the inheritance of what I term "somatically-mediated variations" in Darwin's theory, even if Weismann dismissed, whereas Darwin accepted, such inheritance. (I describe Darwin's views in Winther, 2000.) See Jablonka and Lamb, 1995; Sarkar, 1991 for recent philosophical discussion on the meaning of "inheritance of acquired characters."

[5] See Churchill, 1987; Gerson, 1998; Robinson, 1979; Winther, 2000.

[6] See Godfrey-Smith, 1996, p. 30; Gould, 1977, p. 2; Winther, 2000, p. 427.

and acellular (neither a cell nor contained in a cell) bodily substances – and the extra-organismic *environment*.[7] Only changes in the nutrition, temperature, or other external conditions directly required for germ-plasm growth and development could cause germ-plasm variation. Weismann rarely specified the sources and causal pathways, within the hierarchy of external conditions, of such changes. Nevertheless, he clearly held that germ-plasm growth and development were intertwined with somatic growth and development, as well as with the extra-organismic environment; changes in external conditions could thus lead to germ-plasm variation. Churchill notes that Weismann had a "developmental frame-of-mind."[8] Hodge argues that Weismann's synthetic theory was, in many respects, "cell-theoretic, growth-theoretic and generation-theoretic."[9] Development, heredity, and variation were intimately linked for Weismann.

An irony, which is in tension with much of the standard twentieth-century history of biology, thus exists. One of the most important biologists of the nineteenth century, famous for successfully arguing against the inheritance of acquired *somatic* variations, actually championed the inheritance of acquired *germ-plasm* variations. Weismann was not a Weismannian. My article complements Griesemer and Wimsatt's argument that Weismann was not a Weismannian in the domains of heredity and development. They argue that he did not conceptually cleave the processes of heredity and development, which Weismannism does.[10] Weismannism *also* denies the inheritance of acquired variations of any kind. Here I show that since Weismann accepted the inheritance of acquired germ-plasm variations, he was not a Weismannian in the domain of variation.

The rise of genetics markedly changed the theoretical linkages among development, heredity, and variation, not for Weismann, but for subsequent

[7] Weismann initially labelled *organisms* as internal: "internal influences, i.e. the underlying physical nature of the organism" (1882, p. 653 and, for the German, 1875–1876, p. 289). He subsequently identified the *germ-plasm* as internal: "purely internal [causes], viz., . . . the composition of the germ-plasm" (1893a, p. 422 and, for the German, 1892b, p. 553). Weismann also explicitly defined "external conditions" as anything outside of the germ-plasm: "One needs to comprehend the concept of external conditions ['äussere Umstände'] broadly and one needs to understand that *everything which is not the germ-plasm itself, falls under this concept*" (1891a (1888 essay), p. 408 and, for the German, 1892b, p. 477 (emphasis mine)). In contrast to the notion of the germ-plasm, articulated in 1883, the concepts of external conditions and soma remained relatively unexplored in Weismann's ontology (see also note 45 below). However, these passages justify my usage of the term "external conditions" in the manner described above.

[8] Churchill, 1999, p. 749.

[9] Hodge, 1989, p. 274.

[10] Griesemer and Wimsatt, 1989; see also Griesemer, 2000b.

biology, which adopted Weismannism.[11] Weismannism also ties the issue of the relation between development and heredity with the problem of variation, but in a manner diametrically opposed to Weismann's linkage. Weismannism, in its extreme form, is committed to the idea that precisely *because* development and heredity (transmission) are separable and since changes in external conditions act during development, acquired variations of any kind cannot be inherited; variations in the hereditary material are caused by hereditary dynamics internal to the continuous and sequestered germ – recombination and "spontaneous" mutations caused by internal replication errors, for example.[12] Weismann championed, and Weismannism denies, the view of development, heredity, and variation as integrally and necessarily intertwined.

Today we are moving away from Weismannism and returning to Weismann's synthetic problem structure in our current efforts to integrate our understanding of the processes of evolution, development, heredity, and variation into a coherent theoretical, methodological, and empirical framework.[13] Thus, an analysis of Weismann's views on variation is important not only for the history of biology but also for evolutionary developmental biology, which is the field attempting a synthesis conceptually similar to, but methodologically and empirically far more advanced than, the efforts Weismann and other biologists made during the latter half of the nineteenth century.

[11] Weismannism has a complicated history, which I cannot trace here (see Griesemer and Wimsatt, 1989 for a part of that history). During the first three decades of the twentieth century there was active debate concerning (1) the variational sequestration of the germ-plasm as well as (2) the relation between the processes, and disciplinary organization, of heredity/genetics and development. As a tentative hypothesis requiring further exploration, I suggest that Weismannism became the dominant *perspective* in fields investigating generation – embryology, cytology, and genetics – as well as the standard *interpretation* of Weismann's views, during the 1920s and 1930s (see, e.g., Haldane, 1990 (1932), pp. 8, 20; Morgan, 1926, pp. 30–31; Morgan, 1932, p. 263; Wilson, 1925, pp. 11–13; on perspectives see Griesemer, 2000a; Wimsatt, 1974; Winther, 2002 in press).

[12] Biologists acknowledged mutagenic agents such as radiation and some chemicals as early as the 1920s. These were cases of acquired germ-plasm variations so the acceptance of such phenomena seems to indicate that biologists did not hold Weismannism in its extreme form. But this belief requires further historical and philosophical investigation. As a provisional hypothesis, I suggest that the extreme form of Weismannism was indeed powerful and coordinated much biological work. With the rise of genetics, biologists no longer held that external variation was *necessary* for variation in the hereditary material, nor were they searching for a *general* mechanistic theory of variation, externalist or otherwise, as nineteenth-century biologists such as Darwin, Haeckel, and Weismann had.

[13] See Buss, 1987; Griesemer, 2000a; Griesemer, 2000b; Oyama, 2000; Raff, 1996; Robert et al., 2001; Wagner et al., 2000; Winther, 2001, 2002 in press.

In this article, I will show that Weismann's views regarding the origin of variation were significantly more subtle than the first two of the three theses attributed to him: (1) acquired variations (characters) are not inherited, and (2) internal hereditary processes, particularly sexual reproduction, are the sole cause of variation in the hereditary material. There is a lacuna in the secondary literature concerning these theses.[14] Since my focus is on variation, I will not be directly concerned with thesis (3), the germ-plasm is intra- and inter-generationally continuous, which has been discussed at length; this is precisely the thesis pertinent to the relation between development and heredity.[15] Weismann always argued that changes in external conditions were necessary causes of variation in the hereditary material. Subsequent natural selection on this variation was required to produce organismal adaptation.[16] I will argue that two more subtle externalist and developmental theses more accurately describe his views: (1′) acquired somatic variations are not inherited, but acquired germ-plasm variations *are* inherited and are the only kind of novel germ-plasm variation, and (2′) although the hereditary *and* developmental process of sexual reproduction recombines existing variation in the hereditary material, changes in external conditions are the *ultimate necessary* causes of novel variation in the hereditary material.

Weismann's detailed views on the cause of variation changed as a consequence of novel experimental methods and data, debates with his critics, and the continual elaboration of his germ-plasm theory of development, heredity, and variation. I divide his career into four stages and, in what follows, analyze each in turn. The four stages are: (1) vague externalism (1875–1884); (2) phylogenetic externalism (1885–1891); (3) hierarchical externalism (1892–1895); and (4) externalist selectionism (1895–1914). Weismann consistently held thesis (2′) throughout the four stages and adopted thesis (1′) unequivocally only during the last two stages. My periodization provides a framework for examining the particularities of each stage as well as for inferring generalities across stages. I will focus on each particular

[14] However, see Bowler, 1979 and Hodge, 1989, both of which briefly discuss Weismann's externalist views on variation in the hereditary material. Churchill, 1999 and Mayr, 1985 describe germinal selection. My paper complements these articles by providing a systematic and detailed analysis of Weismann's views on variation in the hereditary material.

[15] See Berill and Liu, 1948; Buss, 1987; Churchill, 1968, 1985, 1986, 1987, 1999; Coleman, 1965; Griesemer, 2000b; Griesemer and Wimsatt, 1989; Jablonka and Lamb, 1995; Maynard Smith, 1989; Mayr, 1985; Robinson, 1979, chap. 7; Webster and Goodwin, 1982.

[16] Except for a lecture in 1895 (Weismann, 1896b), Weismann always held that externally-caused variation was random with respect to adaptive direction at the organism level. No mechanism existed for transferring organismally adaptive structures and processes to the germ-plasm. In this paper I therefore use the term "acquired" to refer to a change caused by an external difference, whether the change is adaptive or not.

stage and will suggest tentative reasons for his shifts between stages in the conclusion.

Vague Externalism (1875–1884)

In the "Preface to the English Edition," written in November 1881, of his *Studies in the Theory of Descent*, Weismann opined that "Nor can the transforming influence of direct action, as upheld by Lamarck, be called in question, although its extent cannot as yet be estimated with any certainty."[17] Although this can be understood as an endorsement of Lamarckism by Weismann, as Mayr has discussed,[18] the term "Lamarckism" is a vague one haunted with myriad connotations. I suggest, therefore, that we not use it to describe Weismann's position in 1875.[19] Instead, by carefully analyzing the last chapter of Weismann's *Studies*, entitled "On the Mechanical Conception of Nature," we can arrive at a more detailed understanding of his position regarding the causes of variation in the hereditary material.

In order to account for the origin of "variability," Weismann felt that he needed to provide a "mechanical explanation." This would avoid the "smuggling in [of] a teleological power."[20] His mechanical explanation referred to external differences: "All dissimilarities of organisms must depend upon the individuals having been affected by dissimilar external influences during the course of the development of organic nature."[21] Weismann remained committed to this position for the remainder of his career. The strength of this commitment stemmed, in part, from his adherence to mechanism and his aversion to explanations employing teleology or vitalism.[22] Any spontaneous change – change without prior change in external influences – would imply non-mechanistic activity.

Let us explore the ways in which differences external to the organism caused change. Weismann characterized the germ as carrying "developmental directions."[23] Organisms of the same species, as zygotes, contained different combinations of developmental directions. But they are furthermore "always exposed to unequal external conditions with respect to nutrition and

[17] Weismann, 1882, p. xvii.

[18] Mayr, 1985.

[19] In his *Studies*, Weismann also emphasized the importance of natural selection in causing evolutionary change.

[20] Weismann, 1882, p. 677.

[21] Weismann, 1882, p. 677; see also p. 115.

[22] Churchill, 1999, p. 750; Churchill, pers. com.; Mayr, 1985.

[23] See, e.g., Weismann, 1882, p. 680. In German, Weismann uses "Entwicklungsrichtung," 1875–76, pp. 304–305.

pressure."[24] Thus, differences between organisms arose both from inherited ("ererbten"[25]) dissimilarities and acquired ("erworbenen"[26]) dissimilarities.[27] Unequal external influences that acted upon the *ancestors* of a particular organism caused differences that the particular organism inherited. Furthermore, inherited dissimilarity "must arise to a greater extent in sexual than in asexual reproduction."[28] Before his explicit theory of reduction-division and amphimixis (developed during the subsequent phylogenetic externalism stage), Weismann already believed that sexual reproduction was a source of variation. Note that although he held, in the *Studies*, that developmental directions were mixed, he did not anywhere hint at a reduction-division during sexual reproduction. In the vague externalism period Weismann held that all acquired organismal dissimilarities were heritable, although he strongly denied this in the next three stages.

Weismann provided what I will call a "primordial organism" thought experiment to illustrate his argument regarding inherited and acquired differences. If we assume a "single primordial organism," all the individual differences of its offspring would be caused by "dissimilar external influences."[29] However, the offspring of these offspring would be different both because they were exposed to different external influences and because their parents, or pairs of parents, in cases of sexually-reproducing organisms, were different from each other. Since any mechanism resembling reduction-division was absent, Weismann also implied that the number of different developmental directions would accumulate in subsequent generations of sexually-reproducing organisms. Ultimately, all organismal differences came from external differences.

Weismann's example concerned the first simple and homogenous unicellular organism. Such an organism, to be anachronistic with respect to Weismann's thought, would have no separation between germ-plasm and somatoplasm[30] simply because it was a homogenous single-cell. All induced change would be heritable. Could his argument regarding the external origin of organismal differences be made for organisms with distinct germ-plasm and somatoplasm? This question cannot be answered because Weismann did not distinguish germ-plasm from somatoplasm during this stage. He therefore did not distinguish heritable from non-heritable variation either in this

[24] Weismann, 1882, p. 680.
[25] Weismann, 1875–76, p. 313.
[26] Weismann, 1875–76, p. 313.
[27] Weismann, 1882, pp. 679, 692.
[28] Weismann, 1882, p. 681.
[29] Weismann, 1882, pp. 679–680.
[30] In this case the somatoplasm would be the cytoplasm.

example or elsewhere in his *Studies*. All variation caused by changes in external conditions acting on the organism or the germ[31] was heritable.

Weismann's view regarding the external sources of variation was merely a conceptual sketch, which, he claimed, had not been verified by experiment. Weismann noted that a "mechanical theory of reproduction," which would explain the causes of variation, was yet to be articulated.[32] Thus, his view was a *vague externalist* sketch.

In his 1883 essay "On Heredity," Weismann formulated further distinctions: reproductive versus somatic *cells*,[33] *substance* ("plasm"),[34] and *molecules*.[35] Weismann was unclear about where in the cell these substances (e.g. germ-plasm) or these molecules resided. In fact, since he wrote about "reproductive protoplasm"[36] and "the substance of germ-cells"[37] he seems to have implied that the *whole* reproductive cell contained reproductive substance.

In this essay, Weismann mounted his first attack on the inheritance of acquired characters. For him, this term meant: "the transmission of changes, produced by the direct action of external forces upon the somatic cells."[38] He criticized the view that molecules could be transmitted from the somatic to the reproductive cells. He concluded that such a view would require the existence of unnecessary new forces.[39] Instead, he proposed that all differences in characters were due to "primary changes in the germ."[40]

But what caused these changes in the germ? Again, Weismann explained the changes by reference to changes in external influences. For example, in elaborating on nourishment affecting the germ-cells, he wrote, "For the germ-cells are contained in the organism, and the external influences which affect them [the germ-cells] are intimately connected with the state of the organism in which they are safely contained."[41] However, the kind of change induced on the germ-cell would be different from the kind of change induced on the body. Weismann also referred to the last chapter of his *Studies*, analyzed

[31] In 1882, Weismann did use the loose word "germ" to describe germ-cells, but he did not distinguish it from anything else. Furthermore, it was just a part, subordinate in importance to the whole organism.

[32] Weismann, 1882, p. 692.

[33] Weismann, 1891a (1883 essay), p. 77.

[34] Weismann, 1891a (1883 essay), pp. 80, 105.

[35] Weismann, 1891a (1883 essay), p. 75.

[36] Weismann, 1891a (1883 essay), p. 74.

[37] Weismann, 1891a (1883 essay), p. 105.

[38] Weismann, 1891a (1883 essay), p. 80. See note 4 above.

[39] Weismann, 1891a (1883 essay), pp. 76–78.

[40] Weismann, 1891a (1883 essay), p. 78.

[41] Weismann, 1891a (1883 essay), p. 105, and, for the German, 1892c, p. 119.

above, for an explanation of his opinion regarding the source of variations in the germ. Thus, in his 1883 essay, Weismann still only provided a vague externalist outline of the source of variation in the hereditary material. He had, however, now distinguished heritable (germ-plasm) from non-heritable (soma) variation and his interests were turning from the organism as a whole to the germ-plasm and its properties and processes.

Phylogenetic Externalism (1885–1891)

(1) *Three Distinctions: Continuity of the Germ-Plasm; and Morphological Sequestration versus Variational Sequestration of the Germ-Plasm*

Weismann developed a crucial set of distinctions and concepts in his 1885 essay. In this essay he argued that the germ-plasm, not the germ-cells, was continuous and was found in all multicellular organisms.[42] The intra-generational and inter-generational *continuity* of the germ-plasm became one of Weismann's celebrated theses. Furthermore, in this essay he noted that the germ-plasm was specifically located in the nucleus of the cell, and not throughout the whole cell.[43] Weismann distinguished idioplasm (nuclear substance) from somatoplasm (cytoplasm).[44] Furthermore, he distinguished two idioplasms: germ-plasm, found in germ cells, and somatic idioplasm, found in somatic cells.[45] Thus, Weismann now argued for what I will call the *morphological sequestration* of the germ-plasm; germ-plasm was *spatially* separated from both germ-cell cytoplasm and somatic cells, which contained both somatic idioplasm and somatic cytoplasm. Continuity of, and morphological sequestration of, the germ-plasm are distinct from what I will call the *variational sequestration* of the germ-plasm. Variational sequestration existed when changes in external conditions, which included changes in the germ-cell somatoplasm (cytoplasm), the somatic cells in general, acellular bodily substances, and the environment outside of the organism, *did not cause any variation* in the germ-plasm. Put differently, when the germ-plasm was stable despite exposure to changes in external influences, it was variationally sequestered. After 1884, Weismann ardently championed the continuity and morphological sequestration of the germ-plasm; starting in 1892, he denied

[42] Weismann, 1891a (1885 essay), pp. 167–176.

[43] Weismann, 1891a (1885 essay), pp. 181–182.

[44] Weismann, 1891a (1885 essay), pp. 183–184.

[45] Weismann, 1891a (1885 essay), pp. 183–187. "Soma" thus was, and remained, an ambiguous term, which Weismann used frequently. It could refer to the *nuclear material* in the somatic cells, the *cytoplasm* of somatic or germ cells, somatic *cells*, or *acellular* bodily *substances*. Weismann did, however, consistently differentiate germ *cells* from germ-*plasm*.

its variational sequestration. Only in his phylogenetic externalism period did he hypothesize an (almost) completely variationally-sequestered germ-plasm.

(2) *Variational Sequestration of the Germ-Plasm*

Although Weismann had partly argued for the morphological sequestration of the germ-plasm in his 1883 essay, he still held that it was not variationally sequestered. Starting in his 1885 essay, "The Continuity of the Germ-Plasm as the Foundation of a Theory of Heredity," and until 1891, Weismann argued for the variational sequestration of the germ-plasm. During this stage he argued that the main immediate source of germ-plasm variation in all multicellular organisms was sexual reproduction.

Weismann did, however, remain uncertain about whether changes in external influences might not produce some small effect on the germ-plasm. In his 1885 essay, for example, he was "far from asserting that the germ-plasm – which, as I hold, is transmitted as the basis of heredity from one generation to another – is absolutely unchangeable or totally uninfluenced by forces residing in the organism within which it is transformed into germ-cells." Weismann, however, noted that even if changes in external conditions could affect the germ-plasm, they "cannot act in the manner in which it is usually assumed."[46] This was because the "quality of the change in the germ-plasm can have nothing to do with the quality of the acquired character." Thus, he allowed for the possibility that changes in external conditions could affect the germ-plasm, but the kind of changes in the soma and germ-plasm would be different. Despite these caveats, Weismann concluded that "it has never been proved that any changes in general nutrition can modify the molecular structure of the germ-plasm."[47]

We see his uncertainty concerning variational sequestration continue in his 1886 essay, "The Significance of Sexual Reproduction in the Theory of Natural Selection." Here Weismann also rejected the inheritance of acquired somatic variations and furthermore argued for the "extreme stability" of the germ-plasm, which "absorbs nourishment and grows enormously without the least change in its complex molecular structure."[48] Even if the germ-plasm did, on occasion, change in response to external fluctuations, the stochastic nature of the fluctuations ensured that the germ-plasm would not change structurally in a systematic fashion.[49] Despite his insistence on the variational sequestration of the germ-plasm throughout the body of the essay, in the

[46] Weismann, 1891a (1885 essay), p. 172.
[47] Weismann, 1891a (1885 essay), p. 173.
[48] Weismann, 1891a (1886 essay), p. 278.
[49] Weismann, 1891a (1886 essay), pp. 278–279.

appendix he hinted at another view in his discussion of the ideas of the American biologist William Keith Brooks. Weismann believed "that permanent hereditary variability can only have arisen through some direct change in the germ-plasm effected by external influences, or following from the varied combinations which are due to the mixture of two individually distinct germ-plasms at each act of fertilization."[50] Which source of germ-plasm variation, changes in external conditions or sexual reproduction, was more common? Given his emphasis, during his phylogenetic externalism stage (cf. 1886 and 1891 essays), on sexual reproduction as a cause of germ-plasm variation, it is clear that he considered this source of germ-plasm variation significantly more frequent and, therefore, more important than changes in external conditions acting on the germ-plasm.[51]

In these essays, Weismann struggled with the extent of variational sequestration of the germ-plasm. Although he was loathe to deny any possibility of changes in nutrition, temperature, moisture, etc. – stemming from the extra-organismal environment or the soma – causing germ-plasm variation, he attributed the vast majority of germ-plasm differences to the process of sexual reproduction. Furthermore, Weismann articulated a set of qualifications for how changes in external conditions could act, if at all: (1) they could affect the rate of growth of the germ-plasm;[52] (2) although they could induce systematic change in all individuals of a species,[53] they could not account for individual differences since the effects of nutritional micro-fluctuations would mutually cancel[54] (he abandoned this position in the *Germ-Plasm*); (3) most important, the kind of change induced in the soma would be different from the kind of change, if any, induced in the germ-plasm because there is no mechanism for transmitting the change, hence the impossibility of the inheritance of acquired somatic variations.[55] Weismann's reluctance to admit any significant external influence on the germ-plasm during this period stood in stark contrast to his views during and after 1892.

[50] Weismann, 1891a (1886 essay), p. 336.

[51] For explicit statements to this effect, see Weismann, 1892a (1890 essay), p. 95; Weismann, 1890a, p. 322. In the latter, he discusses variation in parthenogenetic fungi, which was brought to his attention by the German botanist and mycologist Anton de Bary.

[52] Weismann, 1891a (1886 essay), pp. 278–279.

[53] Weismann, 1892a (1891 essay), p. 190.

[54] Weismann, 1891a (1886 essay), pp. 278–279; Weismann, 1892a (1891 essay), p. 190.

[55] Weismann, 1891a (1885 essay), p. 173; Weismann, 1891a (1886 essay), pp. 326–328; Weismann, 1891a (1889 essay), pp. 449–450; see also Weismann, 1891a (1883 essay), pp. 76–78.

(3) *The Production of Germ-Plasm Variation: The Ambiguous Role of Sexual Reproduction*

Since Weismann used the terms "reduction-division," "amphimixis," and "sexual reproduction" inconsistently, a brief explanation of how I will use them is necessary. I use "reduction-division" to refer to the formation of germ-cells each with one-half the number of ancestral plasms (ids).[56] Here I use "amphimixis" to refer to fertilization of the germ-cells of two sexually-reproducing multicellular organisms, or conjugation in two unicellular organisms exchanging nuclear material.[57] "Sexual reproduction" includes both reduction-division, as just defined, and amphimixis, as it pertains to fertilization. Further discussion of Weismann's employment of, and changes in, these distinctions and concepts can be found in the secondary literature.[58]

A puzzle regarding the role of sexual reproduction exists in the essays of this stage. In some places, Weismann argued that sexual reproduction could both create new, as well as rearrange preexisting, germ-plasm variation, whereas in others he claimed that it *only* rearranged preexisting germ-plasm variation. In what follows I explore each of these two strands of argument. This puzzle is not solvable in this period, I will suggest, due to the opaqueness of Weismann's views on novel variation and the lack of an explicit germ-plasm architecture.

(3a) *Sexual reproduction as creating and rearranging germ-plasm variation*
In his 1886 essay, Weismann suggested what I will call an "asexual-sexual comparison" thought experiment similar to the primordial organism one presented in his *Studies*. With asexual lineages, regardless of whether they were similar or different, natural selection could "never produce a new species."[59] This was because natural selection could not "create new characters."[60] Note that he was assuming variational sequestration. However, with the presence of sexual reproduction, the "origin of hereditary individual variability" in "man and the higher animals" was explained.[61] With the variation produced by sexual reproduction, natural selection could transform species. Weismann also argued that all species could not "have been included as vari-

[56] Weismann, 1892a (1891 essay), p. 126.
[57] Weismann, 1892a (1891 essay), p. 180.
[58] Baxter and Farley, 1979; Churchill, 1968, 1970, 1985, 1999; Coleman, 1965; Farley, 1982, chap. 6 and 7; Robinson, 1979, chap. 7.
[59] Weismann, 1891a (1886 essay), p. 281.
[60] Weismann, 1891a (1886 essay), p. 282.
[61] Weismann, 1891a (1886 essay), p. 284.

ations of the first species."[62] Thus, Weismann implied, sexual reproduction created, as well as rearranged, germ-plasm variation.

In explaining how sexual reproduction created new germ-plasm variation, Weismann provided what I will call an "anti-regression to the mean" argument: "If, for instance, the same part of the body is strongly developed in both parents, the experience of breeders tells us that the part in question is likely to be even more strongly developed in the offspring; and that weakly developed parts will in the same manner tend to become still weaker."[63] Nowhere in this essay did he provide a mechanism for this position. It is therefore not clear, for example, whether this phenomenon could provide sufficient variation for speciation. Surprisingly, Weismann did not mention this anti-regression to the mean argument in either the 1887 or 1891 essays.

A presentation of some of the words employed in the original German will provide a sense of the creative role that Weismann believed sexual reproduction could play in the formation of germ-plasm variation. In the 1887 essay on polar bodies, Weismann stated that the purpose of sexual reproduction was the "preserv[ation]" ["Erhaltung"] as well as the re-formation ["Neugestaltung"],[64] of individual variation. In his 1891 essay on amphimixis, he focused on the process of sexual reproduction recombining "hereditary tendencies."[65] But sexual reproduction was also involved in the "creation" ["Schaffung"][66] of variation and it had a generating effect ["erzeugenden Wirkung"][67] on variation; such germ-plasm variation was advantageous (i.e. natural selection could act on it) for the preservation ["Erhaltung"];[68] as well as the alteration and re-modeling ["Veränderung" and "Umbildung"][69] of species.[70] Thus, through the anti-regression to the mean argument in the 1886 essay, and through his word choice in the 1887 and 1891 essays, it can be seen that

62 Weismann, 1891a (1886 essay), p. 281.

63 Weismann, 1891a (1886 essay), p. 286.

64 Weismann, 1891a (1887 essay), p. 384 and, for the German, 1892c, p. 449. Schönland incorrectly translated "Neugestaltung" as "call forth."

65 Weismann, 1892a (1891 essay), pp. 132, 136, 186, 193.

66 Weismann, 1892a (1891 essay), p. 195 and, for the German, 1891b, p. 135.

67 Weismann, 1892a (1891 essay), p. 195 and, for the German, 1891b, p. 136. The translation is not completely true to the German grammar here.

68 Weismann, 1892a (1891 essay), p. 222 and, for the German, 1891b, p. 176. The translators used the word "maintain" instead.

69 Weismann, 1892a (1891 essay), pp. 199 and 222 and, for the German, 1891b, pp. 141 and 176. The translators used the vague word "modification" for both German words. I thank Sacha Willsey for help translating German to English.

70 Note that he understood sexual reproduction as a mechanism both for maintaining species boundaries and for the evolution of new species. He did not, however, discuss the former process in any detail; he only did this in his last, externalist selectionism, stage.

Weismann held that sexual reproduction created new, as well as rearranged preexisting, germ-plasm variation.[71]

(3b) *Sexual reproduction as only rearranging germ-plasm variation*

The second strand of argument regarding sexual reproduction was that it reorganized preexisting germ-plasm variation, but did not create it. Experimental results on reduction-division during the 1880s convinced Weismann that chromosomes in egg and sperm could be divided in many different combinations,[72] thereby explaining why all the germ-cells of a given individual were not identical. Sexual reproduction rearranged variation by halving and then combining the units of hereditary tendencies – the ids[73] – of each parent in the offspring.

In his 1886 essay, Weismann insisted on finding the "origin ['Ursprung'] of hereditary individual variability."[74] He wrote, "We have ... shown that hereditary differences, when they have once appeared, would, through sexual reproduction, undergo development into the diverse forms which actually exist; but this conclusion affords us no explanation of the source whence such differences have been derived."[75] Weismann's worry about the original source of differences implied that variation in the hereditary material must first be created and only then could sexual reproduction rearrange it. Since the germ-plasm was variationally sequestered in modern multicellular organisms, Weismann suggested that the source of variation could be found in their unicellular ancestors: the "origin of hereditary individual variability cannot indeed be found in the higher organisms – the Metazoa and Metaphyta; but it is to be sought for in the lowest – the unicellular organisms."[76] Because such ancestors lack a "distinction between body-cell and germ-cell" and because "the child [of such ancestors] is a part, and usually half, of the parent," all change caused "by some external influence" would be heritable.[77] He did not clarify whether changes induced *anywhere* in the cell could be inherited. He gave the example of a moneron (a bacterium) "gain[ing] a somewhat

[71] Weismann, 1891a (1886 essay), 1891a (1887 essay), 1892a (1891 essay).

[72] Weismann, 1892a (1891 essay), pp. 132–135; see secondary literature in note 58 above.

[73] Weismann, 1892a (1891 essay). Note that prior to the 1891 essay "hereditary tendencies" were rearranged. In the 1891 essay, Weismann more specifically argued that "ids" were rearranged. He did not develop the units of "biophors" and "determinants" until the *Germ-Plasm*.

[74] Weismann, 1891a (1886 essay), p. 284 and, for the German, 1892c, p. 338.

[75] Weismann, 1891a (1886 essay), p. 284.

[76] Weismann, 1891a (1886 essay), pp. 284–285.

[77] Weismann, 1891a (1886 essay), p. 285.

coarser and more resistent protoplasm"[78] that would subsequently be transmitted. Such an organism, as he pointed out in 1891, lacked, he believed, a nucleus with germ-plasm and hence a change induced anywhere in it would be heritable. Again, as in his 1882 book, through a particular choice of example, Weismann glossed over the case of unicellular organisms with distinct cytoplasm and germ-plasm in a nucleus.

In 1891, however, he distinguished unicellular organisms with a nucleus (Protozoa) from those without one (Monera).[79] Concerning Monera, he still held that "All variations which have arisen in them, by the operation of any causes whatever, must be inherited, and their hereditary individual variability is due to the direct influence of the external world." It was in the Monera-like ancestors of multicellular organisms that variation, which was subsequently recombined, had arisen.[80] Regarding Protozoa, Weismann now held that their nucleus contained germ-plasm, which was variationally sequestered.[81] In general, Weismann argued that sexual reproduction only rearranged variation in the hereditary material caused by external differences that had acted on ancestral unicellular organisms; this is why I call this stage *phylogenetic externalism*. Weismann did not explain how germ-plasm and somatoplasm (cytoplasm) *differentiated evolutionarily* from the substance found in Monera. Weismann also did not describe the nature of this substance, but to be consistent, it could not have been germ-plasm because, if it had been, then it would have been variationally sequestered and could therefore not have changed in response to changes in external influences.

(3c) *A recalcitrant puzzle: Weismann's Theory of Variation from 1885–1891*
Did sexual reproduction create new, as well as rearrange preexisting, germ-plasm variation, or did it *only* rearrange germ-plasm variation? If the former, then why did Weismann insist on accounting for the original source of germ-plasm variation – why could sexual reproduction not produce novel germ-plasm variation as well? If the latter, then why did Weismann provide an anti-regression to the mean argument, which clearly granted sexual reproduction a creative role? This is a puzzle.

In the *Germ-Plasm*, Weismann specifically criticized his anti-regression to the mean argument. He noted that sexual reproduction was "incapable of giving rise to ['schaffen'] new variations."[82] Now that he had articulated his germ-plasm ontology he could clarify where and how reduction-division,

[78] Weismann, 1891a (1886 essay), p. 285; in German he wrote "Moner" (Weismann, 1892c, p. 338) although this was mistranslated as "Protozoon."
[79] Weismann, 1892a (1891 essay), pp. 190–191.
[80] Weismann, 1892a (1891 essay), p. 193.
[81] Weismann, 1892a (1891 essay), p. 192.
[82] Weismann, 1893a, p. 414 and, for the German, 1892b, p. 543.

amphimixis, and changes in external influences could affect the germ-plasm. Before this book, he had not worked out the hierarchy of biophors, determinants, ids, and idants. Without this explicit germ-plasm ontology during the phylogenetic externalism period, Weismann could not clarify whether sexual reproduction alone could create germ-plasm variation.

Hierarchical Externalism (1892–1895)

In the *Germ-Plasm*, the germ-plasm was not variationally sequestered and sexual reproduction only rearranged germ-plasm variation. These views, which predate Weismann's mechanism of germinal selection, were further formulated in the 1894 Romanes lecture, the 1896 paper on seasonal dimorphism in butterflies, and in two essays (1893, 1895) responding to Herbert Spencer. Only in the 1894 lecture, as well as in his second response to Spencer, did he start to develop germinal selection. Thus, there is no precise date when he turned from hierarchical externalism to his final view of externalist selectionism.

The cause of variation was a key problem in the *Germ-Plasm*. In introducing the last chapter of the *Germ-Plasm*, entitled "Variation," Weismann claimed that "*variation . . . forms an integral part of heredity*, *for the latter always includes the former*."[83] Unlike some biologists a generation earlier, such as Prosper Lucas and, to an extent, Darwin, Weismann did not see variation and heredity as opposing forces.[84] He felt that "A theory of heredity must . . . include a theoretical substantiation of variation."[85] Let us now explore Weismann's substantiation of variation during his stage of hierarchical externalism.

(1) *The Hierarchical Structure of the Germ-Plasm*

In the first chapter of the *Germ-Plasm*, Weismann described the germ-plasm as consisting of four compositional levels. Idants generally corresponded to chromosomes and were composed of ids. Ids were arranged linearly along the idant and each id of germ-plasm contained all the determinants necessary for the formation of an organism.[86] Determinants "correspond[ed] to

[83] Weismann, 1893a, p. 410.

[84] Churchill, 1987; Winther, 2000.

[85] Weismann, 1893a, p. 410.

[86] Weismann postulated that during development, ids underwent a division parallel to the length of the idant, which thereby partitioned different determinant-kinds into the two daughter germ-plasms (e.g., AB vs. C in Figure 1). Somatic idioplasm – the nuclear material of somatic cells – had fewer (eventually only one per cell) determinants than germ-plasm (Weismann,

	Id1	Id2	Id3	Id4	Id5
Determinant-Kind A	A1	A2	A1	A1	A1
Determinant-Kind B	B2	B3	B3	B3	B2
Determinant-Kind C	C1	C3	C1	C3	C1

Length of Idant (Chromosome)

Figure 1. One idant (chromosome) of this hypothetical organism consists of five ids and three determinant-kinds. The other idants (chromosomes) may consist of different numbers of ids but they will all contain these three determinant-kinds (A, B, C). The ids are arranged sequentially along the length of the chromosome. The determinant-kinds are arranged within each id orthogonally to the sequence of ids. Each determinant-kind (e.g., B) has two determinant-variants-of-a-kind (e.g., B2, B3) in this depiction. Many other determinant-variants-of-a-kind (e.g., B1, B4, B5) could be present in other organisms in the population or even on other idants of this organism.[90]

and determin[ed]" particular "cells, or groups of cells which are independently variable from the germ onwards" and which he called "determinates."[87] Intestinal cells or the hair in particular bodily regions were examples of determinates.[88] Finally, determinants were composed of groups of molecules, or biophors, which were the "bearers of vitality, [and] possess the power of growth and of multiplication by fission."[89] In fact, all these levels possessed the power of growth and multiplication. Fluctuations in external conditions during development caused variation in the growing germ-plasm.

In order to frame the following discussion, I distinguish a *determinant-variant-of-a-kind* from a *determinant-kind* (Figure 1). A determinant-variant-of-a-kind produces a particular variety of a given kind of determinate. For example, one determinant-variant-of-a-kind might produce orange-red eyebrow hairs whereas another determinant-variant-of-a-kind might produce brown eyebrow hairs. A determinant-kind produces a distinct kind of determinate. For example, one determinate-kind might produce eyebrow hairs whereas another might produce intestinal cells. In more familiar language,

1893a, pp. 60–75). This explanation of mosaic development and ontogenetic differentiation became known as the Roux-Weismann theory of qualitative nuclear division (e.g., Wilson, 1925, pp. 1057–1062).

87 Weismann, 1893a, p. 57.

88 Weismann, 1893a, p. 58.

89 Weismann, 1893a, p. 42; for three other descriptions of the hierarchical germ-plasm architecture see Churchill, 1999, pp. 761–762; Jablonka and Lamb, 1995, pp. 37–42; Maynard Smith, 1989, pp. 2–3.

90 See Weismann, 1893a, pp. 280, 308 for verbal descriptions of the figure; Frederick Churchill, pers. com., provided a three-dimensional "log-model" which helped me understand the germ-plasm architecture.

determinant-variants-of-a-kind correspond abstractly to alleles whereas determinant-kinds correspond to genes. Weismann distinguished homologous from heterologous determinants. The former were determinants of a particular determinant-kind; the latter were determinants of different determinant-kinds.[91] He also contrasted homodynamous and heterodynamous determinants. Both were determinants of the same determinant-kind, but the former were the same determinant-variant-of-a-kind whereas the latter were different determinant-variants-of-a-kind.[92] Again, Weismann's heterologous (distinct determinant-kinds) and heterodynamous (distinct determinant-variants-of-a-kind) determinants corresponded, abstractly, to the genes and alleles of twentieth-century genetics. However, Weismann also believed in: (1) a one-to-one causal relation between determinant-kinds and the cells or groups of cells – the determinates – they caused, (2) as many copies of each determinant-kind as there were ids – this was on the order of hundreds within the germ-plasm of a single individual, and (3) the potential construction of an organism from just one id – the ancestral unit of individuality.[93] In contrast, genetics adopted pleiotropy and polygeny, asserted only two copies of each gene per individual, and claimed that the whole germ-plasm was required to construct the organism.[94]

(2) *Growth as a Variation-Inducing Process: Germ-Plasm is not Variationally Sequestered*

In the *Germ-Plasm*, and in all other subsequent work, Weismann asserted that the germ-plasm was not variationally sequestered. As an organism grew, cell divisions occurred and the germ-plasm multiplied as it traveled inside the cells of the germ-track and as it was distributed during gamete formation.[95] "Complete uniformity as regards nutrition existing during growth" was impossible.[96] Therefore "*the elements of the germ-plasm* – i.e., the biophors and determinants – *are subject to continual changes of composition*

[91] Weismann, 1893a, pp. 264–268.

[92] Weismann, 1893a, pp. 264–266, 274, 278.

[93] Point (3) was a subject of debate between Weismann and Theodor Boveri. Boveri asserted the position subsequently adopted by genetics: the entire complement of chromosomes is required to build an organism, and each chromosome has individuality; see Robinson, 1979, pp. 189–190. In response to Boveri, Weismann partially changed his view on this matter; see Churchill, in prep.; Weismann, 1913.

[94] Further comparisons between Weismann's views and the views developed after the rediscovery of Mendel is beyond the scope of the paper. This is relatively unexplored territory. But see Allen, 1978; Churchill, in prep; Olby, 1966. Churchill's analysis is the most detailed one on this matter.

[95] Weismann, 1893a, chap. 3–6, 14.

[96] Weismann, 1893a, p. 417.

during their almost uninterrupted growth . . . *these very minute fluctuations, which are imperceptible to us, are the ultimate source* ['letzte Wurzel'] *of the greater deviations in the determinants, which we finally observe in the form of individual variations*."[97] Differences in nutrition led to differences in the constitution of the parts of the germ-plasm. However, nutritional differences induced variation that is imperceptible both to selection and to us. Amphimixis was required to *amplify*[98] the variation. This is one reason for calling this period *hierarchical externalism*: the processes that induced and amplified germ-plasm variation were occurring at different levels.

Weismann noted disingenuously that he had assumed a changing germ-plasm in his 1886 paper. But, he claimed, not only had he overestimated the stability of the germ-plasm, he had also not considered the role of amphimixis in amplifying variation.[99] In a passage that I quote fully at the very beginning of this article, Weismann wrote, "The ultimate source ['letzte Wurzel'] of *variation* is always the effect of external influences ['äussere Einflüsse']. Were it possible for growth to take place under absolutely constant external influences, variation would not occur; but as this is impossible, all growth is connected with smaller or greater deviations from the inherited developmental tendency."[100] Germ-plasm variation was *necessarily* caused by external differences.

Weismann explained how micro-fluctuations in nutrition were supposed to cause variation: "[The] variability [of biophors and determinants] . . . is due to the *dissimilar composition of the elements in the growing substance*. If the determinants consisted of masses which were all exactly alike, inequality of nutrition could never transform determinant A into A_1: – it could only alter its rate of growth. They are, however, composed of biophors of different kinds, which react unequally ['ungleich'] under different conditions of growth. This renders possible a disarrangement of the proportional numbers of the different biophors in a determinant, and consequently also the variation of the latter."[101]

Two conditions were necessary for inducing variation: differences in the germ-plasm elements and differences in nutrition. A determinant which previously had, say, 50 biophors of kind X and 70 biophors of kind Y, might be transformed into a new determinant with 60 biophors of kind X and 60 biophors of kind Y. Note that this was *not* germinal selection. The variation was not directed, nutrition was not limited, and capacity for growth of

[97] Weismann, 1893a, p. 417 and, for the German, 1892b, pp. 546–547.
[98] As we shall see below, amplification is a kind of rearrangement.
[99] Weismann, 1893a, p. 417.
[100] Weismann, 1893a, p. 463 and, for the German, 1892b, p. 609.
[101] Weismann, 1893a, p. 418 and, for the German, 1892b, p. 548.

different biophors and determinants was not different; these, however, were all elements of Weismann's germinal selection theory.

In the other passage where Weismann carefully depicted the process of variation production, he wrote: "If a determinant N differs slightly in every id, it will also vary a little during growth if exposed to [change-inducing] influences ['abändernde Einflüsse']; so that, for example, the determinant N may remain unaltered in id A, while N_1 varies in id B. On the other hand, the [change-inducing] influence of nutrition may very likely be slightly different in id A and in id B, and may produce a variation in N, while N_1 in id B remains unaltered."[102]

In the first example, he considered a given change-inducing influence acting on two distinct determinants, whereas in the second case he contemplated *two* change-inducing influences acting on two distinct determinants. In both examples the variation arose from a combination of both determinant and nutritional differences, but in the second example the nutritional differences were different from each other.

The meanings of "*inequality* of nutrition" and "*change-inducing* influences," both of which are changes in conditions external to the germ-plasm, are unclear.[103] Was Weismann comparing spatially – distinct influences at a time-slice or fluctuations of influences over time in the same, or even different, locations? In a similar fashion, Darwin was vague about how he used "changes in the conditions of life."[104] Here I will assume that a change-inducing influence exists if influences outside of the germ-plasm vary temporally or spatially or both. Inequality of nutrition is a particular kind of change-inducing influence involving nutrition. Weismann was vague regarding these meanings, but my assessment is consistent with his work.

In both his 1896 paper on seasonal dimorphism in butterflies and in the *Germ-Plasm*, Weismann also attributed changes in the germ-plasm to changes in temperature. He wrote, "These determinants [in *Chrysophanus plæas*], which determine the various scales of the wing, are found in the germ-plasm of the reproductive cells, and in the rudiments of the wings of the pupa; and it is easy to assume, that they are struck by the heat in both places, and influenced in a similar way, though not to an equal extent."[105] Neither in this paper nor in the book did he provide further details on how changes in temperature altered the determinants. He did, however, distinguish two kinds of effects of temperature in the paper. One was to directly alter

[102] Weismann, 1893a, p. 420 and, for the German, 1892b, pp. 550–551.

[103] Here I discuss the meaning of *changes* in external conditions; in the introduction I discussed the meaning of an *external* condition.

[104] Winther, 2000, p. 436.

[105] Weismann, 1896a, p. 37; see also 1893a, pp. 399–409.

the determinants. The other was to serve as the "*stimulus to development*;" temperature effects were "*not the actual causes of such formations, but only the stimulus, which sets their primary constituents free*."[106] This was the process he had analyzed in his 1894 Romanes Lecture entitled *The Effect of External Influences upon Development*. Whereas he called the first kind of effect "direct seasonal dimorphism," the second he named "adaptive seasonal dimorphism."[107] When temperature acted in the second fashion, Weismann argued that selection, and not temperature, had been the agent of determinant organization.[108] Although this essay was published three weeks *after* Weismann delivered his lecture on germinal selection,[109] he was here referring to organismal, not germinal, selection.

Describing temperature and selection as two mechanisms for seasonal dimorphism was misleading. Weismann here confused the origin of variation with the maintenance of variation. Each of these issues should be addressed for each case of seasonal dimorphism. Selection, even at the germinal level, as Weismann pointed out subsequently,[110] does not produce variation – it requires variation on which to act. In championing the role of selection in the 1890s, even *before* he developed germinal selection, Weismann on occasion omitted discussion of this point and implied that selection produced variation.

During his stage of hierarchical externalism, Weismann held that external differences, such as changes in nutrition and temperature, caused germ-plasm variation. As a final piece of evidence for Weismann's denial of variational sequestration, consider the addition, during the first half of 1892, of a telling footnote to a passage in the *Aufsätze über Vererbung*, a collection of his earlier essays. He was also working on the *Germ-Plasm* during this time. The footnote referred to Weismann's 1886 argument for the source of germ-plasm variation in the hereditary material of unicellular ancestors: "This sentence[111] does not apply as soon as one assumes the changeability of the germ-plasms of the multicellular organisms, through changing external influences. Then we do not have to derive the hereditary differences of the multicellular organisms from the primordial organisms ['Urwesen']."[112] In contrast to his earlier view, changes in external influences now caused variation in the hereditary material of *all* organisms, including ones with germ-plasm.

106 Weismann, 1896a, p. 181.
107 Weismann, 1896a, p. 180.
108 Weismann, 1896a, p. 207.
109 Weismann, 1896b.
110 Weismann, 1904a, v. 1 pp. viii–ix, 215, v. 2 p. 380.
111 Weismann, 1891a (1886 essay), p. 286 – sentence starting "Hereditary variability ...."
112 Weismann, 1892c, p. 339; Sacha Willsey provided a translation of the text.

(3) *Sexual Reproduction as Rearranging Germ-Plasm Variation*

Sexual reproduction, as presented in the *Germ-Plasm* and in subsequent work, merely rearranged ids, thereby rearranging the different determinants and biophors. Variation induced by changes in influences was "imperceptible" to natural selection because it tended to only occur in a small number of determinant-variants-of-a-kind.[113] Since the determinant-variant-of-a-kind in the majority determined the particular determinate,[114] the small minority of changed determinant-variants-of-a-kind could not cause a changed determinate. In order for a changed determinate to ensue, the "homodynamous determinants [same determinant-variant-of-a-kind] in different ids and individuals [must be] brought together in *one* germ-plasm by means of the process of 'reducing division' and amphimixis, so that they can thus form a majority."[115] By halving the parental ids in the appropriate fashion and then combining the gametes, sexual reproduction could bring together a majority of a particular determinant-variant-of-a-kind. It amplified the number of a particular determinant-variant-of-a-kind by rearranging them. Natural selection then acted on the advantageous determinate (cell or group of cells) constructed by the majority determinant-variant-of-a-kind.

A strong criticism of Weismann's theory of the production of germ-plasm variation by id rearrangement through sexual reproduction, written by the biologist Marcus Hartog, appeared in the pages of *Nature* on October 29, 1891 and was dated October 12, 1891. It triggered an exchange that highlights Weismann's views on sexual reproduction. In an argument that is not easy to follow, Hartog claimed that Weismann's ontology of ids – ancestral individual germ-plasms – was unclear about whether particular whole ids represented a particular species (e.g. lobster ids) or whether they corresponded to varieties of ancestral protozoans which, in particular combinations, produced particular metazoan species. If the former, then id shuffling through sexual reproduction was both unnecessary and insufficient to explain the origin of species; if the latter, then sexual reproduction could recombine ids so that "a lioness might be expected to bring forth a lobster or a starfish or any other animal."[116] The former, Hartog claimed, was inconsistent with Weismann's premise that sexual reproduction alone could produce germ-plasm variation to form new species; the latter was contrary to fact. Thus, Hartog concluded, Weismann's theory of the sufficiency of sexual reproduction in producing new species was either inconsistent or contrary to fact.

[113] Weismann, 1893a, p. 419.
[114] Weismann, 1893a, chap. 9.
[115] Weismann, 1893a, p. 422.
[116] Hartog, October 29, 1891, p. 613.

In a second letter to the editor dated November 28, 1891, published in the same journal on December 3, 1891, Hartog explained that he had written to Weismann after drafting his first criticism. Hartog desired to know whether he had been fair in his criticism or whether he was merely setting up a straw man. Hartog received a reply from Weismann before October 12 in which Weismann diplomatically stated that Hartog's depiction was accurate. In his letter, Weismann wrote, "You may very well compare sexual reproduction to the shuffling of a deck of cards, from which half of the cards is always removed. *Only it is not to be forgotten that the cards themselves are not fully unchangeable*."[117] Thus, approximately three months after finishing his "Amphimixis" essay,[118] the role of amphimixis was now compared to the shuffling of a deck of cards – it rearranged the cards without directly changing them. This was the same role he was arguing for in the 1891 essay, as we have seen in the phylogenetic externalism section. But now, unlike the 1891 essay, he was also implying the absence of variational sequestration – the cards could change through the effect of changes in external conditions.[119]

(4) *The Development and Evolution of New Units of Variation: The Limits of Weismann's Account*

The evolution of new kinds of organisms and body-plans required the production of new determinant-kinds: "*The number of determinants in an id of germ-plasm has . . . increased considerably, and even enormously, in the course of phyletic development*."[120] Furthermore, the number of ids had also

[117] Quoted, in German, in Hartog, December 3, 1891, p. 102. I thank Sander Gliboff, John den Hartog, and Aage Winther for help translating German to English.

[118] Sent to Gustav Fischer on July 8, 1891, Frederick Churchill, pers. com.

[119] It is unclear whether Hartog's criticisms (of the sufficiency of sexual reproduction alone to produce germ-plasm variation that could account for the origin of species) contributed to Weismann's rejection of the variational sequestration of the germ-plasm. In a letter dated May 3, 1896 to the English botanist, Albert Trow, Weismann claimed that Hartog had played no role in changing his views on variation (Churchill and Risler, 1999, v. 1, pp. 264–265). This is not definitive evidence that Hartog failed to change Weismann's views; self-representation by historical agents is notoriously suspicious evidence for accurate historical narratives. It is also worthwhile noting that Trow was an agent in this debate. He defended Weismann by claiming that Hartog had misunderstood Weismann's postulated mechanism of sexual reproduction (Trow, December 3, 1891); this may have made Weismann less likely to portray Hartog's influence, even if there was one, to Trow. Hartog, on the other hand, certainly believed that he had influenced Weismann (Hartog, May 11, 1893).

[120] Weismann, 1893a, p. 415; see also 1904a, v. 2 p. 187, where he tied this phenomenon to Haeckel's biogenetic law.

increased over phyletic time in order for accessory idioplasm, such as reserve germ-plasm, to exist.[121]

With respect to the numerical increase in determinant-kinds, Weismann only once suggested an explanation for how it could occur: "more abundant nourishment can cause the doubling of a determinant in the germ-plasm."[122] Despite this suggestion, Weismann did not elaborate on how the doubling was to take place or how a new function was to be adopted by the new determinant-kind. As an example, he mentioned a sexually-dimorphic character, the tail-feathers of hummingbirds.[123] Weismann mentioned "double-determinants" in the context of sexually-dimorphic organisms and bilaterally-symmetrical organisms.[124] He assumed that double-determinants had "a common origin" and "[lay] close to one another."[125] They were not, however, functionally-distinct determinant-kinds since each of the two rows of determinants produced the same determinate, one for each sex or one for each side of the body. The evolution of new determinant-kinds required either: (1) a double-determinant abandoning its role in sexual differentiation and *each* determinant becoming a determinant-kind pertinent to all individuals of a species or (2) a determinant-kind involved in producing a determinate present in all individuals of a species or population doubling and its double becoming a new determinant-kind. Weismann did not discuss either of these processes.

With respect to the increase in id number, Weismann considered a number of phenomena for which this had to occur: regeneration, reproduction by fission and gemmation, alternation of generations, and the existence of the germ-track. In addition to its own somatic idioplasm, the nuclear material of specialized somatic cells also contained reserve ids – accessory idioplasm – that had either many (for cases of regeneration) or *all* (for the other cases) of the determinant-kinds of the species; the latter case corresponded to germ-plasm.[126] Weismann did not consider how this doubling of ids occurred either during ontogeny or phylogeny.

Weismann did not provide clear or substantial explanations for either the development or the evolution of *new* determinant-kinds or ids. Such explanations were *not* beyond the limits of Weismann's theory of the germ-plasm, but perhaps they were beyond the limits of Weismann's interests and problem

[121] Accessory idioplasm were ids not used directly by the cell in which they were found; reserve germ-plasm, which was part of the germ-track and was also used in cases of reproduction by fission, is an example.

[122] Weismann, 1893a, p. 428.

[123] Weismann, 1893a, pp. 427–428.

[124] Weismann, 1893a, pp. 285–286, 355–356, 427–428.

[125] Weismann, 1893a, p. 356.

[126] Weismann, 1893a, chap. 2–6.

structure in his book and in subsequent work. This does seem curious in light of the fact that many of his critics, such as Haeckel and Hartog, were arguing with him on exactly these issues.[127]

(5) *The Meanings of "Hierarchical Externalism"*

In this section I have explored the mechanisms of germ-plasm variation that Weismann expounded in his 1892 book and in work over the subsequent three years. External differences, which included differences stemming from the soma, were *required* to produce germ-plasm differences. These external effects could induce variation at a variety of compositional levels: biophors, determinants, and ids. This is one meaning of the term "hierarchical externalism." Another meaning of the term is that amphimixis, a higher-level mechanism, is required to make these biophor, determinant, and id variations large enough for selection to be able to act on them.

Externalist Selectionism (1895–1914)

Starting in 1893, Weismann and Spencer debated over the inheritance of acquired (somatic) characters.[128] This debate has been addressed in the secondary literature.[129] Furthermore, since it was primarily concerned with acquired somatic variations such as mutilations and functional modifications, rather than with acquired germ-plasm variations, I will not here analyze it. However, it is important to note that, in this debate, Weismann developed arguments concerning panmixia[130] and germinal selection.[131] In the *Germ-Plasm*, Weismann had held that germ-plasm variation caused by nutritional fluctuations occurred independently of the selective needs of the organism. By his 1895 lecture, however, he held that germinal selection determined

[127] Hartog, October 29, 1891, December 3, 1891, May 11, 1893. Haeckel criticized Weismann's theory on the grounds that the distinction between germ-plasm and somatoplasm was empirically unfounded and made the inheritance of acquired (somatic) variations impossible. Gliboff argues that Haeckel also believed that Weismann's germ-plasm ontology could not explain the origin of new determinant-kinds: Weismann's ontology did not provide for a creative process. See Gliboff, 2001; Haeckel, 1893. Sander Gliboff pointed out, and helped explain, the Haeckel text to me; Sacha Willsey provided a translation of the text.

[128] Spencer, 1893a,b,c,d, 1894, 1895; Weismann, 1893b, 1895a; see also: Hartog, 1893; Romanes, 1893a,b,c,d.

[129] Blacher, 1982; Churchill, 1978. On Weismann's general role in arguing against the inheritance of acquired (somatic) characters see Churchill, 1976; Greenfield, 1986; Johnston, 1995.

[130] Weismann, 1893b, 1895a.

[131] Weismann, 1895a.

the direction of variation and that, therefore, germ-plasm variation was no longer independent of selective utility. Although Weismann was diametrically opposed to Spencer on the question of the inheritance of acquired (somatic) characters, they shared the common assumption that most variations were useful and that the production of germ-plasm variation was somehow fundamentally connected with its utility. Thus, Weismann's debate with Spencer provided the context for further developments of his externalist views on variation. In this section I will review germinal selection and I will then turn to the role sexual reproduction played in generating germ-plasm variation. During this stage, Weismann combined his externalism with his hyperselectionism, which is why I call it *externalist selectionism*.

(1) *Germinal Selection*

Germinal selection was a process in which the units of the germ-plasm, especially the determinants, competed for limited nutrition and therefore grew at different rates; the ones assimilating nutrition more efficiently fared better. Weismann hinted at this process in his 1894 and 1895 papers[132] and developed it substantially both in a lecture delivered to an audience of zoologists in Leiden, Holland on September 16, 1895, and in his 1904 book.[133]

An outcome of the Weismann-Spencer debate was that Weismann became worried about "*why it happens that useful variations are always present*."[134] That is, why were adaptive germ-plasm variations more frequent than expected by chance? He held that "*some profound connection must exist between the utility of a variation and its actual appearance*, or, in other words, *the direction of the variation of a part must be determined by utility*."[135] Weismann's argument was that a determinant[136] could either assimilate more or less nutrition and thereby grow larger or smaller. There were only two directions of determinant variation: "plus or minus."[137] These variations fluctuated around a "zero-point."[138] A plus determinant would lead to a plus determinate, which, in a number of cases, meant a larger organ. Weismann also argued that although germ-plasm variation was always caused

[132] Weismann, 1894a, pp. 12–17; Weismann, 1895a, pp. 425–426.
[133] Weismann, 1896b, 1904a,b.
[134] Weismann, 1896b, p. 29.
[135] Weismann, 1896b, p. 33.
[136] Although he was opaque on this point, Weismann seems to have held that selection occurred primarily among determinant-variants-of-a-kind. Determinant-kinds could also compete with one another.
[137] Weismann, 1896b, p. 35.
[138] Weismann, 1896b, p. 36.

by a quantitative increase or decrease of biophors or determinants, qualitative determinant or determinate variation could occur "because [their] component parts change their proportions;"[139] quantitative changes were the basis for all qualitative changes.

In his 1895 lecture Weismann argued that selective forces favoring the determinant at the germinal level (e.g., limited nutrition), would also favor the determinant at the personal, or what we would call organismal, level (e.g., predators, parasites, limited food, and limited mates).[140] Utility at both levels were completely correlated. This position is illogical since plus determinants, which would always be favored at the germinal level, could lead to determinates that would be selectively disfavored at the organismal level.

Weismann abandoned this logically inconsistent correlation in his 1904 book. Here he argued that "Whether a determinant falls or rises depends in all cases only on the play of powers ['Spiel der Krafte'] in the interior of the germ-plasm, and certainly not on whether the particular variation-direction ['Variationsrichtung'] is beneficial or harmful [at the organismal level], that is, whether the particular organ, the determinate, does or does not have value."[141] What was favored at the germinal level would not necessarily be favored at the personal level. Consistently with this shift, Weismann had also de-emphasized the prevalence of personally (organismally) adaptive variations. Although he still held that they existed, he no longer believed that they were exceedingly common – germinal selection did not guarantee them. Note, then, that the only point in his career during which he thought that acquired germ-plasm variation was necessarily adaptive at the organismal level was his 1895 lecture. Prior and subsequent to this, he held that organismal adaptation required the operation of natural selection on germ-plasm variation; *this variation was not necessarily adaptive at the organismal level*.

Directly related to the issue of organismal adaptation is Weismann's adoption of Romanes' idea of "selection value."[142] A particular variation, measured along some axis, attained selection value only when it was sufficiently different from other variations for selection to act on it differentially, either positively or negatively. Similar variations, close to each other on the axis, were equivalent and none was any better or worse with respect to selection. Furthermore, selection values across neighboring clusters of equivalent variations were different. Sometimes Weismann characterized these differ-

[139] Weismann, 1904a, v. 2 p. 153; see also Weismann, 1896b, pp. 46–47.
[140] See, e.g., Weismann, 1896b, pp. 50–51, 60.
[141] Weismann, 1904a, v. 2 p. 118 and, for the German, 1904b, v. 2 p. 101.
[142] Weismann, 1893b, p. 324.

ences as matters of life or death; in this case the selective differences could be interpreted typologically.[143] At other times he described the selective differences as gradual continuous differences.[144] Furthermore, in his 1895 lecture, Weismann suggested that variations would quickly attain selective value and thus that the clusters of equivalent variations would be found in narrow bands along the axis chosen.[145] In his 1904 book, Weismann held that variations had to be significantly different before selection value would be attained.[146] The clusters of equivalent variations were broader in his theorizing in 1904 than in 1895. Furthermore, whereas in the 1895 lecture, selective value concerned both the germinal and organismal level, in the 1904 book it only pertained to the organismal level.

Once a determinant began to vary in either an upward or downward direction, it generally continued to do so. I call this claim the "determinant-inertia hypothesis." In 1895, Weismann explained that advantageous determinants continued to increase "because they themselves now oppose a relatively more powerful front to their neighbors, that is, actively absorb more nutriment, and upon the whole increase in vigor and produce more robust descendants."[147] Determinants varied in their capacity to "absorb nutriment" and those with a greater capacity would continue outgrowing those with a lesser capacity. This capacity may seem to be an internal property, so that germ-plasm variation through determinant-inertia may appear internally-caused. However, Weismann tied current differences in nutrition-absorbing capacity to the effect of differences in *past* quantities of nutrition available to determinants.[148] Thus, germ-plasm variation through determinant-inertia was also, ultimately, externally-caused.

Weismann presented similar arguments for this determinant-inertia hypothesis in 1904: "The determinant whose assimilating power is weakened by ever so little is continually being robbed by its neighbours of a part of the nourishment which flows towards it, and must consequently become further weakened."[149] However, here he also noted that "slight fluctuations ... may often alternate and turn in an opposite direction, and thus the upward movement of a determinant may be transformed into a downward one."[150] Thus, in 1904, determinant-inertia was not only a consequence of the capacity to assimilate nourishment, but also depended on a constant overall temporal

[143] Weismann, 1893b, p. 323.
[144] Weismann, 1904a, v. 2 p. 132.
[145] Weismann, 1896b, pp. 60–61.
[146] Weismann, 1904a, v. 2 p. 132.
[147] Weismann, 1896b, p. 45.
[148] Weismann, 1896b, pp. 41–42, 45.
[149] Weismann, 1904a, v. 2 p. 120; see also pp. 117–118.
[150] Weismann, 1904a, v. 2 p. 128.

pattern of changes in external conditions (see note 103 above). More broadly, Weismann now also distinguished between changes in external conditions causing the same variation in the same determinant-kind of all ids ("induced germinal selection") and changes in external conditions causing distinct variations in the same determinant-kind of different ids ("spontaneous germinal selection").[151]

Germinal selection was a hypothesis in which changes in external conditions, pertinent to germ-plasm growth, caused variation. The units of the germ-plasm now competed for limited nutrition, exhibited directed variation, and varied in their capacity to assimilate nutrition and grow. Weismann maintained this hypothesis for the remainder of his career.[152]

In addition to articulating arguments regarding germinal selection, Weismann also presented one of the strongest statements of his externalism in his 1904 book: "The roots of all the transformations of organisms, then, lie in changes of external conditions. Let us suppose for a moment that these conditions might have remained absolutely alike from the epoch of spontaneous generation ['Urzeugung'] onwards; then no variation of any kind and no evolution would have taken place. But as this is inconceivable, since even the mere growth of the first living substance must have exposed the different kinds of biophors composing it to different influences, variation was inevitable, and so also was its result – the evolution of an animate world of organisms."[153]

This position is similar to Darwin's claim that "if it were possible to expose all the individuals of a species during many generations to absolutely uniform conditions of life, there would be no variability."[154] Despite numerous differences in their theories of generation, both Weismann and Darwin held that changes in external conditions were *necessary* to produce heritable variation.

(2) *Two New Processes for Sexual Reproduction: Causing Co-adaptation of Determinant-Kinds and Stabilizing Species*

Although Weismann did not discuss sexual reproduction to any significant extent in his exchanges with Spencer or in his 1895 lecture, he considered it at great length in his 1904 book. Sexual reproduction still rearranged, and

[151] Weismann, 1904a, v. 2 pp. 136–137.

[152] See Weismann, 1909a, pp. 46–54; Weismann, 1913, chap. 25 and 26 – these chapters are equivalent in content to the ones in the 1904 book and will therefore not be separately analyzed here.

[153] Weismann, 1904a, v. 2 p. 380 and, for the German, 1904b, v. 2 pp. 318–319.

[154] Darwin, 1868, v. 2 p. 308. See Winther, 2000, pp. 433–434.

did not create, germ-plasm variation. It was now also involved in two new processes.

Weismann now implicitly distinguished two ways that sexual reproduction could rearrange determinants: it rearranged determinant-variants-of-a-kind both *of* a particular determinant-kind and *across* determinant-kinds. He had already developed the former process in his hierarchical externalist period; this was the process, discussed above, in which reduction division and amphimixis separated and united different combinations of determinant-variants-of-a-kind so that one variant or other was in the majority. The latter process was a new explicit concern.[155] Weismann had argued with Spencer over the mechanisms for co-adaptation of parts. Whereas Spencer championed a Lamarckian mechanism for this process, Weismann argued that sexual reproduction brought ids together with the right combination of advantageous determinant-variants-of-a-kind of *distinct* determinant-kinds. In fact, it was "only through amphimixis that simultaneous harmonious adaptation of many parts becomes possible."[156]

The second new process that Weismann ascribed to sexual reproduction was "that it also leads, by a continual crossing of individuals, simultaneously with the elimination of the less fit, to a gradually increasing constancy of the species."[157] Weismann was here concerned with Quetelet's and Galton's arguments regarding regression to the mean, which he now accepted as a phenomenon to be explained.[158] Thus, sexual reproduction produced two complementary outcomes: individual variation and species constancy. In the context of species constancy, Weismann argued for the occurrence of a "more perfect and stable equilibrium of the whole determinant system."[159] This implied that under some conditions at least, germinal selection would stop. Nevertheless, "the old-established hereditary equilibrium of the germ-plasm must be most easily disturbed when the species is in some way brought into new conditions of existence."[160] It is unclear how often sexual reproduction could establish a species hereditary equilibrium given Weismann's view of the prevalence of changes in external conditions, which disturb the equilibrium.

155 Weismann did briefly mention the issue in the *Germ-Plasm*, 1893a, pp. 431–432.

156 Weismann, 1904a, v. 2 p. 264; see also p. 196.

157 Weismann, 1904a, v. 2 p. 203.

158 Weismann, 1904a, v. 2 pp. 202–209.

159 Weismann, 1904a, v. 2 p. 200.

160 Weismann, 1904a, v. 2 p. 129; this position is again quite similar to Darwin's view – see Winther, 2000.

During his last stage, Weismann developed his theory of germinal selection, which he believed accounted for directed germ-plasm variation. He also articulated two new processes for sexual reproduction.

Weismann's Externalist Views on Germ-Plasm Variation

Although Weismann always held that variation in the hereditary material was necessarily tied to changes in external conditions, his views changed over his career. A summary of Weismann's views is presented in Table 1. Now I will suggest some hypotheses for why Weismann changed his opinions. Perhaps he started with a vague and simple externalism after reading Darwin's *On the Origin of Species by Means of Natural Selection* (1859), or his *Variation of Animals and Plants Under Domestication* (1868), or other works such as Haeckel's *Generelle Morphologie der Organismen* (1866). Perhaps it was the simplest mechanical explanation for the production of variation. A stronger hypothesis can be provided for the change between his vague externalism and his phylogenetic externalism: Oscar Hertwig's, Eduard Strasburger's, and his own cytological work on the cell nucleus[161] led Weismann to account for germ-plasm variation by reduction-division and amphimixis. However, even during his phylogenetic externalism phase, he still held that external differences had caused variation in ancestral unicellular – particularly Monera – organisms. Perhaps he then came to see that the inheritance of acquired germ-plasm variation did not, and does not, imply the inheritance of acquired somatic variation; perhaps he was influenced by the criticisms of Haeckel, Hartog, and others, as described above. Whatever the reason, he denied the variational sequestration of the germ-plasm in the *Germ-Plasm*. Concerning the last shift here examined, the one between hierarchical externalism and externalist selectionism, I have argued that Weismann's debates with Spencer prepared him to fasten upon certain assumptions. He came to believe in the co-adaptation of parts as well as in the intrinsic connection between the utility and the production of variation. Full explanations for Weismann's changes require a future project analyzing in detail his interactions with his critics and context at large, as well as the experimental work shaping his theories.

[161] See Baxter and Farley, 1979; Churchill, 1970, 1987; Coleman, 1965; Farley, 1982, chap. 6 and 7; Robinson, 1979, chap. 7.

[162] Weismann did not develop the germ-plasm concept until his 1883 essay; he did not develop a rich germ-plasm ontology until his *Germ-Plasm* book. It is therefore somewhat anachronistic to ask all these abbreviated questions regarding the first two stages of his career. I have therefore introduced three categories of answers to these questions for these two stages: (1) questions that are "not relevant" because the concepts are too specific and require a germ-

Table 1. A series of abbreviated questions with regard to germ-plasm variation and Weismann's answers during different stages of his career.

Question	Period			
	Vague Externalism[162] (1875–1884)	Phylogenetic Externalism (1885–1891)	Hierarchical Externalism (1892–1895)	Externalist Selectionism (1895–1914)
Variational Sequestration of germ-plasm?	(No)	Yes [almost completely]	No	No
Morphological Sequestration of germ-plasm?	(No – but perhaps in 1883 essay)	Yes	Yes	Yes
Continuity of germ-plasm?	(Maybe)	Yes	Yes	Yes
Nutritional fluctuations cause germ-plasm variation?	(Yes)	Very Little, if at all	Yes	Yes
Limited germ-plasm nutrition?	Not Relevant	Not Relevant	No	Yes
Directed germ-plasm variation?	Not Relevant	Maybe	No	Yes
Sexual reproduction mixes developmental tendencies?	Yes	Yes	Yes	Yes
Sexual reproduction creates germ-plasm variation?	(No)	One Strand of Argument	No	No
Sexual reproduction rearranges germ-plasm variation?	(No)	One Strand of Argument	Yes	Yes
Sexual reproduction rearranges determinant-variants-of-a-kind?	Not Relevant	Not Relevant	Yes	Yes
Sexual reproduction rearranges determinant-kinds?	Not Relevant	Not Relevant	Maybe	Yes
Sexual reproduction as a source of species constancy?	No	Maybe	No	Yes

According to Weismann, development, heredity, and variation were fundamentally intertwined. The continuity of the germ-plasm should be distinguished from its morphological and variational sequestration. Weismann adopted germ-plasm continuity and morphological sequestration during, and subsequent to, his phylogenetic externalism stage. He accepted germ-plasm variational sequestration in his phylogenetic externalism stage and rejected it in his last two stages. However, except for one strand of argument in his phylogenetic externalism stage, he always held that changes in external conditions in the soma – germ-cell cytoplasm, somatic cells in general, and acellular bodily substances – and in the environment outside of the organism, were the ultimate source of all new variation in the hereditary material. Sexual reproduction merely rearranged this variation. Furthermore, except for his 1895 lecture, Weismann held that such variation was not necessarily adaptive at the organismal level. External differences, acting during the development of the germ-plasm, made the inheritance of acquired germ-plasm variations not only possible, but *necessary* in Weismann's theory. Weismann was not a Weismannian.

Weismannism was constructed as the cleaving of hereditary and developmental processes, and the denial of the inheritance of acquired characters. Why did this happen? A number of historians have noted that many biologists increasingly separated heredity from development after the turn of the twentieth century.[163] Weismann's defense of external and developmental sources of germ-plasm variation blurred the strong distinction that biologists, particularly geneticists, were forging. Furthermore, such a source of variation seemed to many to be a form of Lamarckism that disturbed the morphologically and variationally sequestered sanctity of the causally-powerful germ-plasm. Hence, it appears that they reinterpreted Weismann in a manner suitable to their purposes. Such interpretative moves would also favor the advocacy of eugenics and hereditarianism by many biologists.[164] Further work is required to articulate the chronology of, mechanisms for, and reasons for the construction of Weismannism as both the dominant perspective in biological fields pertinent to generation – embryology, cytology, and genetics – and as the standard, but erroneous, interpretation of Weismann's views on germ-plasm variation.

plasm ontology not yet developed; (2) questions to which sufficient discussion of inheritance and variation was present to provide a tentative answer – these answers are surrounded by parentheses; (3) questions to which a definite answer could be provided – these are answered as "yes," "no," or "maybe" without a parenthesis.

[163] Allen, 1985; Churchill, 1980; Gerson, 1998; Maienschein, 1987.

[164] See, e.g., Kevles, 1995, pp. 70–71; Rosenberg, 1997, pp. 47, 215–218.

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