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CHAPTER

## 7 7 The Baldwin Effect and Genetic Assimilation: Reply to Griffiths

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### Abstract

Paul Griffiths — the author of Chapter 6 in this book — argues that the process coined “genetic assimilation” has little connection with the issue C. H. Waddington had in mind when he invented the term. This chapter's response is that Griffiths is running two things together, genetic canalization and genetic assimilation. What he says would make sense if related to an earlier paper by, David Papineau, the author of this chapter. Previously David Papineau had been concerned only with Waddington's notion of genetic canalization, but Waddington's thoughts about the Baldwin Effect involve the more specific idea of genetic assimilation, and it was these thoughts that David Papineau addressed in that paper. Given this, Griffith's criticisms miss their mark.

**Keywords:** [genetic canalization](#), [Waddington](#), [gene](#), [developmental resource](#)

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### 1 Canalization and Assimilation

Paul Griffiths argues that the process I called “genetic assimilation” in Papineau (2005) has little connection with the issue C. H. Waddington had in mind when he coined the term. In order to establish this, he argues that Waddington's understanding of genetic assimilation is “simply not accessible” to someone who conceptualizes genes in the way I did in my article. According to Griffiths, where my article used a notion of “gene-P”—a gene as a difference-maker for a specific phenotype—Waddington's thinking requires a notion of “gene-D”—a gene as a developmental resource that is indeterminate with respect to phenotype.

My response is that Griffiths is running two things together, genetic *canalization* and genetic *assimilation*. What he says would make sense if the focus of my concern with Waddington were genetic canalization. But I

was not discussing genetic canalization, but genetic assimilation, and that is a different matter. (Once these issues are clarified, we shall see that Griffiths points about different gene concepts, interesting as they are, constitute something of a red herring.)

*Canalization* is the phenomenon illustrated by Waddington's famous developmental landscapes. Certain phenotypic outcomes are so important that natural selection has buffered them against environmental (and genetic) disruption. As Griffiths explains, once some part of the developing organism finds itself in one of Waddington's valleys, then it will not easily be deflected from its adaptive destination by unusual environments (or unusual genes).

p. 103 Perhaps the most familiar kind of canalization occurs when developmental sequences that previously depended on specific environmental interactions are brought under genetic control. This kind of “genetic canalization” will be involved in any process that deserves the name “Baldwin effect,” since a Baldwin effect by definition requires that some item that was previously acquired from the environment later comes to depend on genes. However, natural selection for such canalization is only part of what defines the Baldwin effect, and not the most interesting part at that. As Griffiths himself makes very clear at the beginning of his note, the interesting part of the Baldwin effect is the idea that natural selection is sometimes able to bring development under genetic control *specifically as a result* of its previously being under environmental control. It's not just that genetic control is selected over environmental control—it's more specifically that this selection occurs *because* of the prior environmental control (chapter 6 here, p. 101).

However, having made this point clear at the beginning of his comments, Griffiths seems to lose sight of it. Even though he presents himself as discussing Waddington's contribution to the Baldwin debate,<sup>1</sup> he does nothing to show how Waddington's thinking bears on the specific issue just emphasized (that is, the possible existence of evolutionary processes where some aspect of development is brought under genetic control *because* it was previously under environmental control). Rather, Griffiths simply focuses throughout on genetic canalization, which is a far more general phenomenon, as I have just explained. Griffiths makes many fascinating observations about Waddington's thinking on canalization. But by Griffiths's own account, canalization *per se* does not count as a Baldwin effect.

Griffiths would at least score a terminological point if the more general phenomenon of canalization were *all* that Waddington was interested in. Of course, this wouldn't show that I mischaracterize Waddington's thinking about the Baldwin effect, since it wouldn't show that Waddington was thinking about the Baldwin effect at all, given the difference between canalization as such and the more specific Baldwin effect. Still, it would at least argue that I was misguided to use the term “genetic assimilation” to refer to a species of Baldwin effect, as I did in the article Griffiths is commenting on. After all, Waddington coined the term, and, if he wasn't interested in Baldwin effects, then that couldn't have been what he was talking about. However, contrary to this suggestion, there is good reason to suppose that Waddington *was* interested in genuine Baldwin-like processes and, moreover, that he used the term “genetic assimilation” specifically in this connection.

## 2 Waddington's Understanding of “Genetic Assimilation”

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p. 104 Waddington introduced his conception of “genetic assimilation” by reference to his series of laboratory experiments on fruit flies (Waddington, 1953b, 1957, 1961). These experiments did not just show how genetic control can be selected over environmental control but, more specifically, how such a selective process can depend essentially on passing through a stage of environmental control. Moreover, Waddington was often quite specific in emphasizing this point.

Consider the best known of Waddington's experiments, which induced environment-independent "veinless" in fruit flies. Waddington subjected a population of fruit fly pupae to heat shocks (40 degrees Celsius for two to four hours). As a result, some failed to grow crossveins on their wings (he called this trait "veinless"). Waddington then bred selectively from these individuals, and again subjected the pupae to heat shocks. After repeating this process for 12 generations, he was able to isolate a strain of flies that displayed the veinless trait even in the absence of early heat shocks and that subsequently bred true for this trait.

It should be clear that this experiment does not simply show that there can be selective regimes that will favor spontaneous veinlessness over environmentally acquired veinlessness. Indeed, Waddington's eventual "artificial selection" of the spontaneously veinless strain, as such, is a trivial matter. Rather, the interesting phenomenon is that repeated selection of individuals *who acquire the trait environmentally* somehow increases the representation of individuals who display it spontaneously and *thereby* makes them available for selection. The final artificial selection of the spontaneously veinless strain depends essentially on the earlier selection of those who acquire veinlessness environmentally.

Here is how Waddington himself described the significance of these laboratory experiments:

All these experiments demonstrate that if selection takes place *for the occurrence of a character acquired in a particular abnormal environment*, the resulting strains are liable to exhibit that character even when transferred back into the normal environment. That is to say, the process which has been defined as genetic assimilation really occurs. Insofar as this is true, the *appearance of acquired characters* which are of value to an organism in terms of natural selection *will have evolutionary consequences*. *Natural selection for such characters* will lead to the appearance of populations in which the character is an inherited one and will be developed even in environments other than that which originally provoked it and in which it is of adaptive value. We have, therefore, experimental justification for using the notion of genetic assimilation to explain all those evolutionary phenomena which people in the past have been tempted to attribute to the inheritance of acquired characters in the Lamarckian sense. (1961, p. 263, my emphases)

I take it that this passage puts it beyond dispute that Waddington understood "genetic assimilation" to refer to something specifically Baldwin-like, rather than simply to the more general idea of the evolution of genetic canalization. Having said that, it must be said that Waddington was far less clear about the *mechanism* that might be responsible for genetic assimilation. He tends to shy away from this topic, and often suggests that no further explanation is needed beyond the general observation that evolution frequently favors genetic canalization (see Waddington, 1957, 1961). Still, this doesn't alter my immediate point, which is that Waddington clearly uses "genetic assimilation" to refer to the more specific selective processes by which some aspect of development is brought under genetic control *as a result of* previously being under environmental control, even if he doesn't have any good explanation of how this happens.

### 3 An Explanation of Waddington's Experimental Results

Waddington is not alone in supposing that genetic assimilation is somehow self-explanatory. As Patrick Bateson has observed, “frequent references are made to genetic assimilation ... without thought being given to how a usually implicit reference to Waddington might explain what was being proposed” (2004, p. 290). Sometimes commentators will refer to the role of the new environmental factor (for example, the heat shocks in Waddington's experiment) in “revealing” hitherto unexpressed genetic variability (the presence or absence of the genetic factors that yield veinlessness after heat shocks) and thus exposing these factors to selective pressure. But this by itself does not serve to explain Waddington's results, for there is no intrinsic reason why selecting flies that are veinless-if-heat-shocked should yield a population with an increased likelihood of innately veinless flies.

To see this more clearly, it will be helpful to think of the various flies in Waddington's veinlessness experiment as having three sorts of genomes: those that ensure they have crossveins even if heat shocked; those that make them veinless if heat shocked; and those that render them spontaneously veinless. Most of the flies in the original population had the first sort of genome. By subjecting them to heat shocks and selecting for veinlessness, we get a population with the second sort of genome. Now, why should the third sort of genome be more probable in the second population than in the first? Why, so to speak, should the second and third genomes' similarity in phenotypic space—they are both capable of displaying veinlessness—mean that they are similar in genomic space, that is, a population with the second genome makes the appearance of the third more likely? (See Mayley, 1996.) More generally, why should the selection of genes that facilitate the environmental acquisition of the trait be a crucial step along the way to the selection of its spontaneous appearance?

In my earlier article I offered the following baby model of what might be going on. Suppose veinlessness depends on two factors: (1) some developmentally important protein loses its required conformation, and (2) the “heat shock protein” needed to correct this is absent. Both of these factors can be genetically determined, but both genes are originally rare, and so a spontaneously veinless fly is highly improbable. Now think of the extreme heat shocks imposed by Waddington as an alternative nongenetic way of causing these two protein deficiencies. Not all flies subject to the heat shocks will develop these deficiencies, but an appreciable proportion will. Now it is much easier for the two rare genes for protein deficiencies to be selected for producing veinlessness: no longer do they have to find themselves together with the other gene in order to produce the “advantageous” phenotype; either gene on its own will now have a “selective advantage,” since it will mean the phenotype will appear in any case where the other protein deficiency has been environmentally caused.

Obviously, the specifics of this explanation are speculative. But it seems plausible that something of this general kind must lie behind Waddington's experimental results. We need only suppose that his “advantageous” phenotype results from a number of factors, each of which gets produced in some individuals by his experimental manipulation of the pupae, but each of which can also be fixed by some gene. Given these conditions, then the genes in question will individually be selectively favored, because each on its own reduces the environmental contribution needed to produce the phenotype. A quantitative illustration of this process is given in the second chapter of Jablonka and Lamb (1995, pp. 32–6), and they, too, argue that this is the natural explanation for Waddington's results.<sup>2</sup>

It is perhaps worth making it explicit why this model makes Waddington's experiments come out as instances of the Baldwin effect (where “Baldwin effect” is understood, as before, as meaning that some trait comes under genetic control as a result of its previously being environmentally acquired). In the foregoing model, an essential precursor to the eventual spontaneous appearance of the “advantageous” phenotype is the stage where each of the genes is being individually selected because it will cause the phenotype *when the*

*environment is producing the other determinants.* In this sense, the overall phenotype eventually comes entirely under genetic control only in virtue of the fact that previously the environment was producing the various determinants of that phenotype.

## 4 Genetic Assimilation Generalized

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I take this suggested explanation of Waddington's results to instantiate an important general structure. Take any case where it would be biologically advantageous to have some phenotype genetically fixed, rather than dependent on specific environmental stimuli. But suppose also that this requires a complex suite of genes, and that the initial rarity of these genes makes their cooccurrence unlikely (and in any case liable to be undone by sexual reproduction). However, if the various determinants of the phenotype can also be environmentally produced, then this selective obstacle can be surmounted. As soon as the various determinants of the phenotype are *environmentally* produced in a significant number of individuals, then each gene becomes advantageous on its own, even in the absence of the genes at other loci, since it reduces the chancy dependence on the environment by ensuring that that the phenotype will appear as soon as all the *other* determinants are environmentally produced. The selective process that ensues will constitute a Baldwin effect in the sense defined earlier, since the eventual accumulation of genes for the different determinants of the phenotype will hinge essentially on a prior stage where those determinants are also being environmentally produced.

p. 107 In my earlier article I used the term “genetic assimilation” to refer to this general structure. That is why I was happy to include under this heading the kind of case where selection brings some complex *behavior* increasingly under genetic control by cumulatively favoring genes that accelerate its *learning*. As Griffiths explains in his note, I modeled this phenomenon by supposing that any such behavior has a number of subparts, each of which can either be learned or genetically fixed. Even if it is selectively superior to have the whole behavior genetically fixed, initial rarity of the relevant genes will present a *prima facie* evolutionary obstacle. Still, we can see how the genetic fixity of the behavior could evolve if we suppose that the animals involved are also able to *learn* the various parts of the behavior. For then each gene will indeed have an advantage on its own, even in the absence of the others, since it will increase the speed and reliability with which the whole behavior is learned.

I am not alone in commandeering Waddington's terminology of “genetic assimilation” to cover a far wider range of phenomena than he demonstrated in his original fruit fly experiments, including cases where *behavior* is brought under genetic control via the cumulative selection of genes that lighten the amount of *learning* required to acquire the behavior. I learned this usage from Peter Godfrey-Smith (2003), who uses “genetic assimilation” in this broad sense, and he in turn takes it from a flourishing tradition of computer modeling of selective processes (see especially Hinton & Nowlan, 1987; Turney et al., 1996). The rationale for this broad understanding of “genetic assimilation,” as I hope I have made clear, is that the same general structure of interacting genetic and environmental processes is plausibly present in both Waddington's experiments and in cases where learned behavior is brought under genetic control.

## 5 Arguments for Narrowing Genetic Assimilation

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Not everybody is comfortable with this broad understanding of “genetic assimilation.” As we have seen, Paul Griffiths, for one, queries my usage, on the grounds that my account of how learned behavior is brought under genetic control assumes a conception of *gene* that is quite different from Waddington's. I shall discuss Griffiths's concerns in the next section. But first it will be helpful to consider some rather different worries about applying “genetic assimilation” to cases involving learned behavior. A number of other commentators, while not disagreeing with any of my substantial analysis so far, feel that other differences between the learning cases and Waddington's experimental paradigm are so significant that it is misleading to lump both under the same name. For example, Patrick Bateson lists a number of ways the two kinds of case differ, and urges on this basis that we should restrict the term “genetic assimilation” to Waddington's examples, and instead use the term “organic selection” for the behavioral cases (Bateson, 2004). (“Organic selection” was the term originally used in the 1890s by Henry Osborne, Conwy Lloyd Morgan, and James Mark Baldwin himself to describe the way that learning can lead to the genetic selection of behavior.)<sup>3</sup>

p. 108 Of course, there is no substantial issue here. All can agree that there are some similarities between Waddington's examples and the learning cases, along with some differences, and moreover that we can adopt whatever terminology we like, as long as we make it clear what we mean. Still, many of the points raised by Bateson are of interest in their own right. I shall focus on two of them: the *adaptiveness of learning* and the *role of mutation*.

For the first point, note that the initial environmental cause of the novel phenotype works rather differently in the Waddington and behavioral cases. In the Waddington cases, it is due to some novel environmental influence on early development; in the behavioral cases, it is a result of a mature learning process operating in new environmental conditions. In itself, this contrast might not seem to matter to the logic of selection for increased genetic control, but it carries with it a further difference that does so matter: in the behavioral cases, the novel phenotype will characteristically be *adapted* to the novel environmental conditions, whereas in realistic Waddington cases, as opposed to those cooked up in his laboratory, such adaptedness will be a freak.

After all, there is no intrinsic reason why a real-life developmental novelty prompted by a natural analogue of Waddington's heat shocks should be advantageous rather than deleterious. Of course, in his experiments Waddington chose to select for the novelties his developmental shocks provoked. But there is no reason why natural environments should be so cooperative, and in reality, genes that facilitate any given Waddington-style developmental novelty are far more likely to be selected against than for. By contrast, behavioral novelties produced by learning mechanisms will naturally tend to be advantageous, since learning mechanisms are themselves adaptations designed to produce behaviors that are suited to current environments, and so there is a built-in reason why genes that facilitate these behaviors will be selected for.

This is certainly a noteworthy difference. But it does not undermine the point that, whenever a Waddington-style developmental novelty is advantageous, then just the same complex structure of selection as operates in the behavioral cases can bring it under genetic control. Perhaps the fact that advantageous Waddington-style novelties will be the exception rather than the rule argues that Waddington-style cases are a less powerful evolutionary force than learning-based “organic selection.” Still, it is not as if it is alien to natural selection to work with sources of variation that are more likely to produce deleterious variants than advantageous ones (see Jablonka & Lamb, 1995, p. 36).

I turn now to the suggestion that Waddington's cases are different from learning-based “organic selection” because they work with preexisting genetic variability, whereas “organic selection” relies on new genetic mutations. I must say that this does not strike me as a real difference. True, there is direct evidence that

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mutations played no role in Waddington's experiments: when the experiments were tried on inbred strains of flies with no appreciable genetic variability, selection for the novel phenotypes produced no genetically new strains (Waddington, 1961, sec. 4A). Conversely, it is also true that most literature on the learning-based “organic selection” assumes that the eventual genetic changes derive from the selection of new genetic mutations. However, it does not take much analysis to show that there is no real contrast here.

For a start, it just isn't true that learning-based “organic selection” needs new genetic mutations. Mutations may be required for some other learning-involving processes that qualify as the “Baldwin effect,”<sup>4</sup> but if we focus on the kind of case at issue in this essay, where genes are favored because they reduce the amount of learning needed to acquire some complex behavior, then it is clear that they can work with existing genetic variability, and indeed for just the same structural reasons as apply in Waddington's cases. To illustrate, suppose that some animal population begins to display some complex learned behavior (maybe the natural environment changes so as make the behavior useful, or maybe a “culture” newly arises by happenstance). This will then create selective pressure, when there was no such pressure before, for any gene that fixes some element of the behavior and thus reduces the learning load. However, such genes could well have been present in the population all along, prior to their acquiring a selective advantage. In that case, the relevant genetic variability would have been “dormant,” waiting for the emergence of the learned behavior to allow the relevant genes to make a selective difference, just as the genes in the Waddington experiments had to wait for his developmental shocks before they had any real chance of producing his “advantageous” phenotypes.

Conversely, there seems no principled reason why, given enough time, Waddington-style experiments shouldn't depend on mutations rather than preexisting genetic variability. Suppose the experiments on the inbred fruit flies had lasted long enough for the genes that fix the required protein deficiencies to emerge occasionally by chance mutation. Then, as long as the environmental shocks were still producing the protein deficiencies too, these mutant genes would have been selected for, leading eventually to a strain that had both protein deficiencies genetically fixed, just as in the original experiment. So, to sum up this point, *both* learning-based “organic selection” *and* Waddington-style cases seem equally capable of working *both* with preexisting genetic variability *and* with novel mutations.

## 6 Gene-Ps and Gene-Ds

Let me now finally deal with Paul Griffiths's reason for doubting that the phenomenon I call “genetic assimilation” can possibly cover what Waddington had in mind. In Griffiths's view, Waddington's thinking requires the notion of gene to be conceptualized in a way that is quite different from the notion of gene that I assume when discussing “genetic assimilation.”

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Griffiths argues that Waddington's interest in developmental canalization meant that he thought of genes as multipurpose developmental resources, rather than as difference-makers for specific phenotypes. Following Lenny Moss (2001), Griffiths distinguishes between “gene-Ds” and “gene-Ps.” Gene-D is the notion Griffiths ascribes to Waddington. A gene-D is a molecular sequence of DNA, but has no connection with any specific phenotype. The protein it determines (or even the *proteins* it determines, when it has alternative regulators) can play different roles at different stages in development, perhaps folding into different isoforms in different instances, and possibly contributing to the formation of many different kinds of tissue. By contrast, gene-P is the perhaps more familiar notion of a genomic entity whose presence or absence is a reliable sign of some specific phenotype, like blue eyes or cystic fibrosis. Gene-D is Waddington's notion, says Griffiths, whereas he takes it that I need gene-Ps to analyze my kind of “genetic assimilation”.

Well, I am more than happy to agree that Waddington's thinking about genetic *canalization* requires gene-Ds rather than gene-Ps. In his note, Griffiths makes a compelling case that Waddington's ideas about the evolution of canalization requires us to think of genes as alterable constraints on shifting developmental landscapes, rather than as determinants of specific phenotypes. However, as I have stressed throughout this essay, genetic canalization is not the same thing as genetic *assimilation*. So the fact that gene-Ds are required for understanding canalization does not imply that they are required when we are thinking about genetic assimilation.

Thus I see no problem in the fact that my initial models of “genetic assimilation,” involving complex behaviors originally acquired by learning, decompose these behaviors down into various subtraits, and assume that there are genes *for* each of these subtraits—that is, gene-Ps for these subtraits (see the passages quoted by Griffiths, chapter 6 here, pp. 92–94). Since my topic is genetic assimilation, understood as the process whereby some complex trait is brought under genetic control *because* it was previously under environmental control, rather than genetic canalization, which is the topic Griffiths focuses on, I see no objection to my modeling it using a concept of gene different from the one needed in order to think about canalization.

Still, perhaps Griffiths will want to press the issue further. Maybe genetic assimilation is not the same as genetic canalization. But the point remains that *Waddington* was interested in genetic assimilation only because it is one source of genetic canalization, and this in itself argues that the genetic assimilation processes *he* was interested in will need to be analyzed in terms of gene-Ds. So, if my notion of “genetic canalization” demands gene-Ps, then it seems unlikely, once more, that I can mean the same thing as Waddington by “genetic assimilation.”

This would be a good argument if the *only* way of thinking about genetic assimilation in my sense were in terms of gene-Ps. But I do not accept this. While I think that there are cases of genetic assimilation that can happily be dealt with in terms of gene-Ps, like my learned-behavior example that Griffiths focuses on, I am also ready to agree that, when we come to the cases that Waddington was interested in, we need to switch to gene-Ds. And I take it that this is exactly what I do when discussing Waddington's fruit fly experiments. There I don't break the complex phenotype (“veinless”) down into subtraits, each with its own gene-P. Rather I speak of the various protein-level “determinants” of veinlessness, and explain how selection can favor bringing these determinants under genetic control, thereby rendering the development of veinlessness independent of environmental factors. This strikes me as more like gene-D talk than gene-P talk, quite in line with the thought that Waddington's cases of genetic assimilation will resist analysis in terms of gene-Ps.

So, once more, I see no reason to erect a principled distinction between Waddington's concept of genetic assimilation and the way I understand this notion.

## Notes

A precursor of this essay was read at the Workshop on Philosophy of Biology of the Department of Philosophy at the University of Bristol October 8–9, 2004. I thank the participants for their comments. I am also grateful to Pat Bateson for discussing some of these issues with me.

1. Thus Griffiths: “Waddington's process stands out among the other ideas listed earlier (‘organic selection,’ ‘coincident selection,’ ‘autonomization,’ ‘the Baldwin effect’)” (chapter 6 here, 92). Again: “Waddington's genetic assimilation is importantly different from the rest of this ‘Baldwiniana’ because...” (chapter 6 here, p. 101).
2. Bateson (1982) offers an alternative suggestion. Suppose veinlessness normally depends on the very rare homozygote of a rare recessive gene, and suppose further that the heat shock reverses dominance so that even heterozygotes with one allele will display veinlessness; this reversal will then create a significant selective pressure for the veinlessness allele,



where none existed before, and thus increase the allele's frequency to the point where homozygotes—who will display veinlessness even if not heat shocked—will become common. However, this explanation, unlike the one offered in the text, is in tension with some further data reported by Waddington: namely that in most of his experiments the final strain of flies that spontaneously displayed the “advantageous” phenotype differed from the original normal flies at loci on a number of different chromosomes (Waddington, 1961, sec. 4B). Given this, Bateson's explanation requires that the heat shock reverses dominance at all these different loci, not just one.

3. Waddington also sought to distinguish “genetic assimilation” from “organic selection,” but on the rather different grounds that earlier theorists failed sufficiently to appreciate the significance of canalization.
4. This is true, for instance, of the kind of process that Godfrey-Smith (2003) calls “breathing spaces.”