The Baldwin Effect and Genetic Assimilation: Contrasting explanatory foci and gene concepts in two approaches to an evolutionary process

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1. The Papineau Effect

David Papineau (this volume) has discussed the relationship between social learning and the family of postulated evolutionary processes that includes ‘organic selection’, ‘coincident selection’, ‘autonomisation’, ‘the Baldwin effect’ and ‘genetic assimilation’. In all these processes a trait which initially develops in the members of a population as a result of some interaction with the environment comes to develop without that interaction in their descendants. It is uncontroversial that the development of an identical phenotypic trait might depend on an interaction with the environment in one population and not in another. For example, some species of passerine songbirds require exposure to species-

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typical songs in order to reproduce those songs whilst others do not. Hence we can envisage a species beginning with one type of developmental pathway and evolving the other type. If, however, the successive evolution of these two developmental pathways is a mere coincidence, selection first favoring the ability to acquire the trait and later, quite independently, favoring the ability to develop it autonomously, then no distinctive class of evolutionary process is going on, merely two standard instances of natural selection one after the other. George Gaylord Simpson pointed this out in the paper that gave us the term ‘Baldwin effect’ (Simpson 1953). The real interest of the Baldwin effect and its relatives lies in various mechanisms that have been proposed to link the evolution of the two developmental pathways, so that acquiring the trait through interaction with the environment makes it more likely that later generations will evolve the ability to acquire the same trait without that interaction. Papineau argues that one such link can be forged if the interaction with the environment that plays a role in the first pathway takes the specific form of social learning. I am not going to repeat his argument here (see Papineau, this volume) and simply note that he seems to me to have described a plausible evolutionary scenario in which social learning plays a role in a distinctive form of ‘niche-construction’ (Odling-Smee, Laland, and Feldman 2003).

My concern here is with Papineau’s discussion of Conrad H. Waddington’s concept of ‘genetic assimilation’ (Waddington 1942, 1953). Waddington’s process stands out amongst the family of ideas listed above both because its author was able to demonstrate it in laboratory selection experiments and because it was part of his larger vision of the relationship between development and evolution, a vision that has influenced
contemporary work in evolutionary developmental biology or ‘evo-devo’. Papineau defines “Waddington’s Genetic Assimilation” as follows:

“Suppose 5 sub-traits, say, are individually necessary and jointly sufficient for T. Each can either be genetically fixed or acquired through (not necessarily social) learning. (So for sub-trait T^K we have allele K^G which genetically fixes T^K and allele K^L which allows it to be learned.)

Suppose also that at first the K^Gs are rare. Still, those lucky organisms that have some T^Ks genetically fixed by K^Gs will find it easier to learn the rest of T, and so will be favoured by natural selection (assuming that learning is here costly). Selection will thus cumulatively build up the genes K^G which genetically fix T.”
(Papineau 2003)

It is easy to show that this process has little connection with the one described by Waddington². In itself this is neither particularly important nor particularly surprising. Many different processes have been proposed that might free traits from their developmental dependence on some aspect of the environment, and terms like ‘Baldwin effect’ and ‘genetic assimilation’ have been used in numerous senses in this extensive literature (See e.g. Belew and Mitchell 1996; Weber and Depew 2003). In fact, despite calling the process ‘Waddington’s genetic assimilation’ Papineau does not cite

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² It does resemble one version of ‘organic selection’. Patrick Bateson has argued that many learning processes have components which might separately become independent of the environmental conditions originally required for their development and that the efficiency and/or reliability of the learning process might thereby be improved. Like Papineau, he points out that these variations would only be selected if organisms regularly undergo the complete learning process (Bateson 2004, 289).
Waddington’s work as a source, but instead cites a well-known computer simulation of the interaction between learning and inheritance (Hinton and Nowlan 1996). The interesting point is that Waddington’s actual model of genetic assimilation is simply not accessible to anyone who conceptualises genes in the way Papineau does in the passage quoted above. Several recent authors have stressed the need for biologists and philosophers of biology to become more self-conscious about the existence of multiple gene concepts and of the appropriate range of theoretical and experimental contexts in which those concepts should be deployed (Moss 2002; Falk 2000; Stotz, Griffiths, and Knight 2004; Griffiths and Neumann-Held 1999). I will argue here that paying attention to gene concepts helps one to distinguish two radically different approaches to explaining how the development of a phenotypic trait can become independent of certain aspects of the developmental environment. One approach looks to selection to forge a link between the successive evolution of two developmental pathways to the same trait. The other approach, represented by Waddington’s genetic assimilation, looks to developmental biology. This latter approach seeks to explain how the development of a phenotypic trait can become independent of an environmental stimulus (or become dependent on that stimulus) by showing that in certain kinds of developmental systems these transitions can be produced by trivial genetic changes. In the first approach the explanatory focus is on the relative selective advantage of the two developmental pathways. In the second approach the explanatory focus is on the developmental mechanisms that make suitable variants available for selection.
2. Genetic Assimilation and Gene-P

In the passage quoted above Papineau employs a concept of the gene which Lenny Moss has labeled ‘Gene-P’:

“Gene-P is defined by its relationship to a phenotype. … Gene-P is the expression of a kind of instrumental preformationism (thus the “P”). When one speaks of a gene in the sense of Gene-P one simply speaks as if it causes the phenotype. A gene for blue eyes is a Gene-P. What makes it count as a gene for blue eyes is not any definite molecular sequence (after all, it is the absence of a sequence based resource that matters here) nor any knowledge of the developmental pathway that leads to blue eyes (to which the "gene for blue eyes" makes a negligible contribution at most), but only the ability to track the transmission of this gene as a predictor of blue eyes. Thus far Gene-P sounds purely classical, that is, Mendelian as opposed to molecular. But a molecular entity can be treated as a Gene-P as well. BRCA1, the gene for breast cancer, is a Gene-P, as is the gene for cystic fibrosis, even though in both cases phenotypic probabilities based upon pedigrees have become supplanted by probabilities based upon molecular probes.” (Moss 2001, 87-88)

Papineau’s five genes are Gene-Ps, each defined by a specific part (‘sub-trait’) of the phenotypic trait T. I take it that these parts are dispositions to acquire behavioral modifications which together amounts to a disposition to acquire the new behavior T. The process he labels ‘genetic assimilation’ is therefore simply the spread of certain of these phenotypic traits as a result of selection. His trait $K_G$ is selectively superior to $K_L$ because
individuals acquire T more reliably than K individuals. The sought-for link between individuals initially learning the sub-trait K and later individuals possessing K without learning is mediated by a process of niche construction – a change in the selective regime as a result of behavior (K was presumably a selectively neutral variant until behaviour T became common). In contrast, Waddington thought that the link between the ability to reliably acquire an adaptive trait and the appearance of individuals with an intrinsic tendency to exhibit that trait was forged by the typical nature of the development pathways underlying adaptively valuable traits. It was for this reason that he objected to Simpson’s term ‘Baldwin effect’ with its implication that this evolutionary process is a special case. Waddington intended genetic assimilation to be a ubiquitous feature of phenotypic evolution:

“Simpson comes to the conclusion that the Baldwin effect, in the sense he describes it, has probably played a rather small role in evolution. The genetic assimilation mechanism, however, must be a factor in all natural selection, since the properties with which that process is concerned are always phenotypic; properties, that is, which are the products of genotypes interacting with environments.” (Waddington 1953, 386)

According to Waddington the tendency of phenotypes to become genetically assimilated reflects the fact that there is little difference between the actual developmental processes that underlie a highly canalised phenotype that depends on an environmental stimulus and one that has been rendered independent of that stimulus, as I will now try to explain.
3. Genetic Assimilation and Gene-D

Waddington was aware that his vision of development required a conception of the gene which does not intrinsically link genes and specific phenotypic outcome. He made this point in ‘The Evolution of Developmental Systems’, an address delivered in Brisbane in 1951:

“There were, as we now know, two competing theories of development. The preformationists believed that all the characters of the adult were present in the newly fertilized egg, but packed into such a small space that they could not be distinguished with the instruments then available. If we merely consider each gene as a determinant for some definite character in the adult (as when we speak loosely of the ‘gene for blue eyes, or for fair hair’), then the modern theory may appear to be merely a new-fangled version of the old idea. But in the meantime, the embryologists, who are concerned with the direct study of development, have reached a quite different picture of it… This is the theory known as epigenesis, which claims that the characters of the adult do not exist already in the newly fertilized germ, but on the contrary arise gradually through a series of causal interactions between the comparatively simple elements of which the egg is initially composed. There can be no doubt nowadays that this epigenetic point of view is correct.” (Waddington 1952, 155)

In Waddington’s vision of development, the entire collection of genes makes up a developmental system which produces a phenotype. Many features of the phenotype are explained by the dynamical properties of that developmental system as a whole, rather
than by the influence of one or a few specific alleles. Thus, for example, Waddington sought to explain one of the major biological discoveries of his day – the fact that extreme phenotypic uniformity can be observed in many wild populations despite extensive genetic variation in those same populations – by appealing to the global dynamics of developmental systems. A ‘canalised’ developmental system takes development to the same endpoint from many different genetic starting points. The development of wild-type phenotypes can thus be buffered against genetic variation. Waddington represented this idea with his famous ‘developmental landscape’ (Figure 1).

*Insert Figure 1 about here. Figure is a reproduction of Figure 10.1 from (Sterelny and Griffiths 1999).*

In modern terms, Waddington’s ‘developmental landscape’ is a complex system whose parameters are genetic loci and whose state space is a set of phenotypic states. The state space is depicted as a surface, each point of which represents a phenotype. The genetic parameters are depicted as pegs that pull on the surface and thus determine its contours. Epistatic interactions between loci are represented by links between the cords by which those loci pull on the surface. The development of an organism over time is represented by the movement of a ball over the surface, which is dictated by gravity, so that the ball rolls downhill on a path dictated by the contours of the surface. The development of the organism is thus represented by its trajectory over the surface, through successive phenotypic states. The basic point which Waddington uses this representation to make is that if the surface has any significant contours, then the effect of a change at one genetic
locus will be dictated by the overall shape of the landscape, which is a global consequence of the state of all the other genetic loci. Some genetic changes, such as those which affect the tops of inaccessible ‘hills,’ will have no effect on development. Other changes of the same genetic magnitude which affect the entrance of a valley or ‘canal’ will have a massive effect on development. The phenotypic impact of a genetic change is not proportional to the magnitude of the genetic change, but depends on the overall dynamics of development. Furthermore, the phenotypic difference produced by a genetic difference is not explained by that genetic difference in itself, but by how that change interacts with the rest of the developmental system. This picture retains considerably validity in the light of contemporary developmental genetics (Gilbert, Opitz, and Raff 1996; Wilkin 2003)

In Waddington’s vision, phenotypes are global expressions of genomes, but it does not follow that particular parts of the phenotype express particular parts of that genome. The gene concept that fits this thoroughly epigenetic view of development is the one which Moss has labeled ‘Gene-D’:

“Quite unlike Gene-P, Gene-D is defined by its molecular sequence. A Gene-D is a developmental resource (hence the “D”) which in itself is indeterminate with respect to phenotype. … To be a gene for N-CAM, the so-called “neural cell adhesion molecule,” for example, is to contain the specific nucleic acid sequences from which any of a hundred potentially different isoforms of the N-CAM protein may potentially be derived … N-CAM molecules are (despite the name)
expressed in many tissues, at different developmental stages, and in many
different forms. The phenotypes of which N-CAM molecules are co-constitutive
are thus highly variable, contingent upon the larger context, and not germane to
the status of N-CAM as a Gene-D.” (Moss 2001, 88, his emphases)³

To understand Waddington’s vision of development it is essential not to think of genes as
‘genes for’ particular phenotypes or phenotypic differences (Gene-P), but instead to think
of them as parameters of a developmental system (Gene-D). It is necessary to think in
terms of what in Waddington’s day was known as ‘physiological genetics’.

In a series of widely-read papers the philosopher Andre Ariew has used Waddington’s
concept of canalisation to explicate the concept of innateness (Ariew 1996, 1999). Innate
traits, Ariew has argued, are those traits insensitive to environmental variation, or,
equivalently, those traits which are canalised with respect to changes in the
environmental parameters of a developmental system. Unfortunately, Ariew’s work has
led philosophers who know of Waddington only through these papers to use the term
‘canalisation’ and even ‘genetic canalisation’ to mean insensitivity to environmental
variation. In fact, the idea of insensitivity to environmental factors, properly known as
‘environmental canalisation’ (Wagner, Booth, and Homayoun 1997), cannot even be
represented in Waddington classic picture of the developmental landscape (Figure 1).
Environmental parameters are not included in this model, and whether a phenotype is

³ Philosophers will note that Gene-P and Gene-D correspond respectively to ‘descriptive’ and ‘rigid’
readings of the phrase ‘the gene for T’ when this phrase is used in the usual way to report the fact that some
DNA sequence accounts for a large portion of the variance in trait T in some study population (See
canalised in Waddington’s original sense is a question of the dynamical structure of the developmental system, not the relative role of genes and environment⁴. But the model can easily be extended to include environmental parameters, and Waddington himself does so when discussing genetic assimilation, as seen below. If these additional parameters are added, then we can define both ‘environmental canalisation’ and ‘genetic canalisation’. A phenotypic outcome is environmentally canalised if those features of the surface which direct development to that endpoint are relatively insensitive to the manipulation of environmental parameters. A phenotypic outcome is genetically canalised if those features of the surface which direct development to that endpoint are relatively insensitive to the manipulation of genetic parameters. It should be noted, however, that we are not forced to draw this distinction. The idea of canalisation with respect to all the parameters that are included in a model of the developmental system is equally legitimate. It is, after all, far from clear whether to classify many critical parameters, such as the presence of DNA methylation or of maternal gene products in the cytoplasm, as ‘genetic’ or ‘environmental’. The issue of genes versus environment is peripheral to Waddington’s central concern, which is how developmental outcomes can be robust and reliable in the face of variations in developmental parameters.

Like some modern authors, Waddington believed that natural selection would favor the canalisation of important adaptive phenotypes. Developmental systems that produce important adaptive outcomes robustly will be selected over those that are easily

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⁴ The evolutionary developmental biologist Brian Hall has written extensively on Waddington and has stressed that his thought was profoundly ‘gene centered’ in the sense that he saw the developmental system as primarily and predominantly the expression of a potential present in the genome (Hall 2003, 1992, 1999).
perturbed. Although I do not have time to explore this theme fully here, it is important to recall that, like his contemporaries I.I Schmalhausen and Theodosius Dobzhansky, Waddington saw natural selection as optimizing, not the phenotypic character itself, but rather a norm of reaction which specifies a range of phenotypes as a function of genetic backgrounds and environmental conditions: “An animal is, in fact, a developmental system, and it is these systems, not the mere static adult forms which we conventionally take as typical of the species, which become modified in the course of evolution’’ (Waddington 1952, 155). When there is a single, optimal phenotype, ‘stabilising selection’ will operate to select a narrow reaction norm, or, in other words, to canalize the phenotype. In other circumstances, however, selection may favour a broader reaction norm, producing what we describe today as ‘adaptive phenotypic plasticity’. The shape of the norm of reaction is itself a character produced by natural selection.

We are now in a position to see why Waddington thought there would be little difference between the actual developmental processes that underlie a highly canalised phenotype that depends on an environmental stimulus and those that underlie one that has been rendered independent of that stimulus. Waddington writes:

“If natural selection was in this way acting in favour of the ability to respond in a useful way to some environmental stimulus, it would also in time build up a canalised response, so that the most valuable degree of expression was regularly achieved. Once that had been done, the genotype would have been modified so that it determined a new valley on the developmental surface; but it would still
require the push of an environmental stimulus to cause one of the balls in our model to run into it. However, once the valley was formed and canalised, the exact strength of the push, and the exact time at which it was applied, would be of lesser importance. In fact, we might expect that, by this stage in the evolution [sic], there would be a number of mutant genes available in the species which could divert development into the prepared channel; and thus, once the ground had been prepared, as it were, an internal genetic mechanism could take over from the original environmental stimulus. We can thus envisage a mechanism by which a valuable response to the environment could become gradually incorporated into the hereditary endowment of the species.” (Waddington 1952, 159)

I have discussed elsewhere how some of Waddington’s contemporaries, particularly J.B.S Haldane and his wife and collaborator Helen Spurway, saw his work on genetic assimilation as demonstrating that there need be little difference as regards developmental genetics between ‘innate’ and ‘acquired’ traits (Griffiths 2004). Haldane and Spurway drew on Waddington to argue that transitions back and forth between instinct and learning were to be expected in response to the adaptive advantages of these two forms of development in specific environments. A couple of brief quotations will give the flavour of this work:

(discussing passerine song learning) “some of these species must have passed through a stage where the song was learnt by some individuals and was instinctive in others.
As a geneticist I think that it is quite impossible to make a sharp distinction between learnt and unlearnt behavior.” (Haldane 1992 [1955], 605).

“The number of generations during which a learned ethogenesis evolves into an instinctive ethogenesis, if it does so at all, depends on the relative strength of the selection pressures favouring uniformity and variability in development.”(Haldane and Spurway 1954, 275)

4. Gene concepts and explanatory foci

In the evolutionary scenario described by Papineau the gene for trait K (‘allele $K_G$’) spreads through the population in response to a selection pressure caused by the spread of a learnt trait T whose acquisition requires of five separate dispositions to each of which corresponds to a gene (‘allele $K_L$’ and four companions). As I remarked above, these genes are Gene-Ps - DNA elements individuated by the criteria that their presence is a reliable statistical predictor of a phenotypic difference. This, I suggest, is typical of one way of thinking about how the development of a phenotypic trait can become independent of certain aspects of the developmental environment. The evolutionary problem is framed as follows: 1) What are the adaptive advantages of having T conditional on an the environmental factor? 2) What are the adaptive advantages of having T independent of that factor? 3) Does the evolution of the first trait produce new selection pressures which favour the evolution of the second? The genes that feature in
typical scenarios designed to address these questions are Gene-Ps corresponding to the
difference between the first trait and the second.\(^5\)

In contrast, most of the genes that figure in Waddington’s genetic assimilation scenario –
the ‘pegs’ in Figure 1 – are genes that are present both when the trait is dependent on the
environment and when it is independent of the environment. They are the genes (Gene-
Ds) which play a causal role in building the K phenotype, not the genes that differ
between cases where that particular cascade of gene expression is switched on
endogenously and cases where it is switched on exogenously or even the genes that differ
between individuals that have K and those that lack K. The evolutionary problem is
framed as follows: How does a trait come to be such that the initiation of its
development can be readily switched between different triggers? This second way of
thinking about how the development of a phenotypic trait can become independent of
certain aspects of the developmental environment corresponds to some of major themes
in recent evolutionary developmental biology, namely the evolution of developmental
modularity (Gass and Bolker 2003; Wagner 2001) and the evolution of phenotypic
plasticity (Preston and Pigliucci 2004; Gilbert 2001; Brakefield and Wijngaarden 2003).
These evolutionary problems simply cannot be posed if evolution is represented as
change over time in the frequency of ‘genes for’ specific phenotypes (Gene-Ps).

We can compare these two ways of thinking about how the development of a phenotypic
trait can become independent of certain aspects of the environment with two ways to

\(^5\) If the evolutionary problem is posed in this way, then I have argued elsewhere that such ‘Baldwin effects’
are best conceived as a special case of niche construction (Griffiths 2003).
approach the evolution of sexual differentiation. The mammalian SRY gene on the Y chromosome is a Gene-P with respect to the masculinisation of the foetus: individuals who have this gene are very likely to have a male phenotype. It does not follow that the evolution of sexual differentiation should be studied by asking how the SRY gene evolved. The masculinisation of the mammalian foetus is the result of a complex cascade of a gene expression. Both male and female foetuses have almost all the genes involved in this cascade. In Waddington’s terms the developmental landscape has a deep branching valley running across it and the SRY gene simply nudges the foetus into one branch rather than the other. With this picture in mind, it is easy to understand how two groups of rodents have managed to lose the SRY gene (and, indeed, the whole Y chromosome) while retaining the gene expression cascade of mammalian sexual development. Some other gene expression event early in development acts to triggers the same cascade. Furthermore, sexual differentiation is an ancient characteristic of vertebrates and in this larger group cascades of gene expression that are to some degree homologous with those in mammals are triggered in still more diverse ways. Crocodiles and others have environmentally triggered sexual differentiation, some fish retain the capacity for females to be masculinised by an environmental trigger throughout their life cycle, birds have a genetic trigger for sexual differentiation but are reverse heterogametic (males have identical sex chromosomes, females have different sex chromosomes). From the viewpoint of developmental genetics, understanding the evolution of the specific triggering cause in one group or another is not the way to understand the evolution of the cascade of gene expression which constitutes becoming male. Nevertheless, there is nothing wrong with Gene-P thinking in the right context – if we want to ask about the
evolutionary pressures leading to genetic versus environmental sex determination it is appropriate to pose the question in terms of the selection pressures on the specific loci involved in these two modes of triggering. In the same way, we could examine the selection pressures on the specific loci involved in making the switch between the dependence of a behavior on learning and its independence of learning, as Papineau does. But this should not be confused with the quite different project in which Waddington was engaged, namely asking how developmental systems can be such as to make such options readily available to selection.

5. Conclusion

Many evolutionary processes have been described in which a trait that initially develops in the members of a population as a result of some interaction with the environment comes to develop without that interaction in their descendants. Waddington’s genetic assimilation is importantly different from the rest of this ‘Baldwiniana’ because his explanatory focus was not on the selection pressures at the point of transition, but on how developmental systems come to be structured in such a way that these evolutionary transitions are readily accessible to evolving lineages. Waddington’s approach also replaces the simple contrast between ‘acquired’ and ‘innate’ with a non-dichotomous model of developmental canalisation and phenotypic plasticity that is in line with recent work on the evolution of development. From a Waddingtonian perspective evolutionary transitions between ‘innate’ and ‘acquired’ are only to be expected because those categories have little meaning in terms of developmental genetics and in some cases the difference between the ‘innate’ and ‘acquired’ may be genetically trivial. But to see this it is necessary to use a gene concept suitable for thinking about development, and not a
gene concept designed for theoretical population genetics or for the prediction of phenotypic differences in populations.

References

Ariew, André. 1996. Innateness and Canalization. Philosophy of Science 63 (3 (Supplement)):S19-S27.


