Beyond the Baldwin Effect: James Mark Baldwin's 'social heredity', epigenetic inheritance and niche-construction[*].

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1. Introduction

James Mark Baldwin is remembered today almost exclusively for his paper 'A New Factor in Evolution' (Baldwin, 1896a). The new factor, which he called 'organic selection' and which later became known as the Baldwin effect, was a process that could cause 'accomodations' - the acquired adaptive responses of individual organisms - to become hereditary. 'Accomodations' include physiological adaptations like calluses and, of much greater interest to Baldwin, learnt behaviors. The Baldwin effect differed from classic Lamarckian inheritance because it respected August Weismann's doctrine of the 'continuity of the germ plasm' according to which modifications to somatic cell-lines can have no influence on the state of the germ cells. Viewed at the population level, the Baldwin effect would give the impression that the Lamarckian inheritance of acquired characters was taking place, but this impression would be an illusion - no individual

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organism would actually inherit an acquired character from a parent. I am not going to describe how the Baldwin effect is meant to occur. Baldwin himself never had a fully satisfactory theory and his contemporaries offered a number of significantly different theories. Later authors have offered many more. The details of these proposals are described by David Depew (this volume), Stephen Downes (this volume) and Peter Godfrey-Smith (this volume). Rather, in this paper I argue that too much attention has been paid to the Baldwin effect. George Gaylord Simpson was probably right when he said that the effect is theoretically possible and may have actually occurred but that this has no major implications for evolutionary theory (Simpson, 1953). The Baldwin effect is not even central to Baldwin's own account of 'social heredity' and biology-culture co-evolution, an account that in important respects resembles the modern ideas of epigenetic inheritance and niche-construction.

There are two reasons for the excessive attention that has been paid to the Baldwin effect. The first is the confused but enduring idea that the Baldwin effect allows 'mind' to 'direct' evolution and thus saves us from the barren Darwinian vision of a world ruled by chance and necessity. The second motive is less well known and far more interesting. Ever since Weismann, biologists interested in causes of adaptation other than natural selection acting on the germ line - causes which include physiological adaptation, learning and its relatives, and epigenetic, ecological and cultural factors in individual development - have faced the challenge that the processes they study can be of no *evolutionary* significance because any changes in form produced by these means will not be preserved on evolutionary timescales. The Baldwin effect allows these processes to leave a record in the germ line and this explains the continuing interest in (and redefinition of) the Baldwin effect by biologists and psychobiologists. I will argue, however, that this way of gaining recognition for these processes merely obscures their real evolutionary role. As Celia Moore (this volume) shows, epigenetic inheritance can play an important role in evolution without a Baldwin effect to transfer its effects to the germ line. In section five I examine a number of recent arguments designed to minimize the evolutionary significance of epigenetic inheritance. I suggest that, at best, these arguments leave the evolutionary significance of epigenetic inheritance undetermined and a matter for (long overdue) empirical enquiry. In section six I turn from epigenetic inheritance to 'niche construction': the reciprocal influence of evolving lineages on the ecological forces that impinge on those lineages. The importance of niche construction has become increasingly widely recognized in recent years, due largely to the work of John F. Odling-Smee and his collaborators. As Peter Godfrey Smith (this volume) argues, the potentially important evolutionary mechanism described by Terence Deacon as the Baldwin effect (Deacon, 1997) is much closer to niche construction than to the Baldwin effect as conceived by its earlier advocates.

Before entering into these contemporary disputes, however, I want to re-examine the role of the Baldwin effect in Baldwin's own work. I will argue that his primary interests were 'accomodation' as a phenomena in its own right, 'social heredity' and the influence of social heredity on natural selection via the process we now call niche-construction. Even the term Baldwin used for the effect now named after him - 'organic selection' - meant something else in the rest of Baldwin's writings and was merely opportunistically redefined when Baldwin attempted to assert his priority with respect to the ‘new factor in evolution’.

2. Baldwin’s Opportunistic Redefinition of ‘Organic Selection’

In ‘A New Factor in Evolution’, Baldwin defines organic selection as, ‘The process of ontogenetic adaptation considered as keeping single organisms alive and so securing determinate lines of variation in subsequent generations’ (Baldwin, 1996: 78)[1]. In an April 1897 letter to *Nature* organic selection is, ‘the perpetuation and development of congenital variations in consequence of accomodation’ (Baldwin, 1897a: 558). In an almost identical contribution to *Science* that same month he repeats this definition, but with the word ‘congenital’ in brackets (Baldwin, 1897b: 635). So in 1896-7 Baldwin consistently defines organic selection as the process in which a characteristic that was initially produced by the action on the individual organism of its developmental environment becomes a hereditary characteristic in descendants of that organism. Read literally, the *Nature/Science* definition of 1897 would encompass not only the Baldwin effect, but also the straightforward neo-Lamarckian inheritance advocated by Baldwin’s sparring partner Edwin Drinker Cope (Baldwin, 1896c; Cope, 1896) and rejected by Baldwin by this date, although this was presumably not Baldwin’s intention when he gave the definition.

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[1] This issue of *Science* in the University of Pittsburgh library having succumbed to the usual fate of acid-rich Victorian paper, citations are to the centenary reprint.

The almost identical contributions to *Nature* and *Science* in April 1897 were designed to defuse a priority dispute between Baldwin, Conwy Lloyd Morgan and the paleontologist Henry F. Osborn. In these short articles Baldwin lists papers published by the three men in 1896 discussing very similar mechanisms by which acquired characteristics could become hereditary without violating Weismann's doctrine of the continuity of the germ plasm. The three authors had introduced a host of neologisms in their accounts of the new evolutionary process. Baldwin lists nine of these terms and suggests that they be adopted as standard. He attributes four terms to himself, three to Lloyd Morgan and treats the remaining two - 'variation' and 'accomodation' - as needing no special attribution. Despite making a great show of even-handedness, however, Baldwin subtly emphasizes his own priority. Osborn had the first published paper (by eleven days), but the earliest date in the list of papers relating to the new theory is that of Baldwin's verbal presentation at the N.Y Academy of Sciences on January 31st 1896. Among the terms Baldwin attributes to himself is 'organic selection', which is the name he suggests for the new factor in evolution as a whole and the title of both the articles.

Naming the new factor in evolution 'organic selection' reinforced Baldwin's claim to priority in another way, since he had introduced this term some years earlier. This fact is heavily emphasized in 'A New Factor in Evolution' where he remarks, 'I have noted in print ... that Prof. Lloyd Morgan and Prof. H.F Osborn have reached conclusions similar to my main one on Organic Selection. I do not know whether they approve of this name for the "factor"; but as I suggested it in the first edition of my book (April, 1895) and used it earlier, I venture to hope that it may be approved by the biologists' (Baldwin, 1996: 79 fn 9). Earlier in the same article Baldwin cites the source of the concept of organic selection as Chapter VII of his *Mental Development in the Child and the Race: Methods and Processes* (Baldwin, 1895). The clear implication of these passages is that Baldwin has known about the new factor for some years and has, indeed, described it in print. Why then, in the two short contributions to *Science* and *Nature* in the next year does Baldwin not cite the chapter-long discussion of organic selection published in 1895 and thus put his priority beyond doubt? The reason is that in his 1895 book Baldwin had used the term to mean something quite different. The insinuation of priority in 'A New Factor in Evolution' would not have stood up to the scrutiny generated by a dispute with two established scientists.

Chapter VII of *Mental Development...* is titled 'Organic Adaptation in General' and in it Baldwin is concerned to improve upon Alexander Bain and Herbert Spencer's explanation of how animals can acquire new, adaptive behaviors in a single lifetime: 'How is it that we, or the brute, or the amoeba, *can learn to do anything*?'(Baldwin, 1900: 181, his italics) [2]. These adaptive responses cannot be explained by '*Natural selection as operative directly upon individual organisms'* (Baldwin, 1900: 172), by which Baldwin means the conventional Darwinian process of natural selection acting on random variations in congenital behavior. Instead, adaptive responses that arise during a single lifetime must be explained by '*Natural selection as operative upon different*

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[2] Citations are to the second edition, of which Baldwin remarks that he has made 'minor corrections throughout. The only important alteration is to be found in the tables (I. and II.) on p. 52'.

reactions of the same organism' and it is this process that Baldwin terms 'organic selection':

> 'It is necessary to consider further how certain reactions of one single organism can be selected so as to adapt the organism better and give it a life-history. Let us at the outset call this process 'organic selection,' in contrast with the 'natural selection' of whole organisms.' (Baldwin, 1900: 174)

The largest part of Chapter VII is devoted to establishing Baldwin's views of the origin of 'the excess process itself, which gives the movements from which "organic selection" selects the fittest' (Baldwin, 1900: 205); the origin, to speak anachronistically, of operant behaviors. Baldwin is concerned to grant natural selection a larger role than he finds in the accounts of Spencer and Bain in explaining the origin of these 'excess' behaviors and the origin of the ability to respond to pleasure and pain by repeating one 'excess' behavior and suppressing another. In 1895, then, it seems that Baldwin meant by 'organic selection' the acquisition of behaviors through reinforcement learning and that his concern was to establish that this ability arose through natural selection rather than being in some way intrinsic to sensible matter.

Section three of Chapter VII is titled '*Development and Heredity'*, but despite this promising title, it contains no mention of the Baldwin effect. Baldwin's concern in this section is to show that his 'theory would not be affected by the truth or falsity of either of the opposed views of heredity now so bitterly opposed to each other in biological circles' (Baldwin, 1900: 204), these two theories being the 'neo-Darwinian' (e.g. Weismannian) and the ‘neo-Lamarkian’. Baldwin states that his theory can be accepted by ‘neo-Darwinians *[who]* hold that natural selection, operating upon congenital variations, is adequate to explain all progressive race gains’, because, ‘granting the ontogenetic progress required by the Spencer-Bain theory and adopted in my own — the learning of new movements in the way which I have called “organic selection,” — yet the ability to do it may be a congenital variation.’ (Baldwin, 1900: 204-5). In 1895, it seems, Baldwin merely held that the congenital ability to learn from experience allowed organisms to adapt in ontogeny - hardly a ‘new factor in evolution’! There is only one passage in section three which might be read as prefiguring the Baldwin effect, a passage where he argues that:

> ‘all the later acquirements of individual organisms may likewise be considered only the evidence of additional variations from these earlier variations *[those conferring ability to learn]*. So it is only necessary to hold to a view by which variations are cumulative to secure the same results by natural selection as would have been secured by the inheritance of acquired characters from father to son.’ (Baldwin, 1900: 205)

However, the following sentences reveal that this has nothing to do with the Baldwin effect. He informs us that, ‘Mr Spencer and others seem to me to be quite wide of the mark in saying that the only alternative to the inheritance of acquired characters is a doctrine of “special creation” ’ (Baldwin, 1900: 205) and goes on to remark that in the recapitulation of phylogeny by ontogeny we see each feature emerge at the same stage that it emerged in the ancestor. The remark that seemed to prefigure the Baldwin effect is in fact an attack on the neo-Lamarckians. Baldwin is arguing that we should not be surprised that sons exhibit the behaviors their fathers learned, since the congenital variation that allowed the father to learn those behaviors will be inherited by the son and will allow him to learn them too. Hence this phenomenon does not compel us to accept the neo-Lamarckian theory of heredity[3].

As late as May 1896, two months after the publication of part one of ‘Heredity and Instinct’ in which the Baldwin effect is moderately clearly described (Baldwin, 1896b, see esp. 439 & 440-441), Baldwin was still defining ‘organic selection’ in a way that makes no reference to the Baldwin effect. Describing the theory of his 1895 book he says:

> ‘Its main thought is this, that all new movements that are adaptive or “fit” are selected from overproduced movements or movement variations, just as creatures are selected from overproduced variations by the natural selection of those which are fit. This process, as I conceive it, I have called “organic selection,” a phrase which emphasizes the fact that it is the organism which selects from all its overproduced movements those which are adaptive and beneficial.’ (Baldwin, 1896c: 427)

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[3] This reading is reinforced by the fact that in his exchange with Cope in *American Naturalist* Baldwin describes Cope’s neo-Lamarckian view of the origin of adaptive behavior in the individual using exactly the same language. Cope’s view is ‘the doctrine of the special creation of ontogenetic adaptations by consciousness’ and contrasts unfavorably with Baldwin’s own, Darwinian theory of the ‘organic selection’ of adaptive behaviors in the individual (Baldwin, 1896c: 427).

3. Baldwin and 'Social Heredity'

Viewed in the broader context of Baldwin's work, the Baldwin effect is primarily an addendum to Baldwin's theory of social heredity. The initial significance of social heredity for Baldwin may have been mainly as a counter to the arguments of the neo-Lamarckians, who argued that neo-Darwinism was insufficient to account for the progressive tendencies that they saw in evolution:

> 'I have recently argued that Spencer and others are in error in holding that social progress demands the use hypothesis* since the socially-acquired actions of a species, notably man, are socially handed down; giving a sort of 'social heredity' which supplements natural heredity' ...
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> *SCIENCE, August 23, 1895, summarised in Nature, Vol. LII, 1895. p. 627.'
>
> (Baldwin, 1896b: 439)

By the time he wrote *Social and Ethical Interpretations in Mental Development: A Study in Social Psychology* (Baldwin, 1897), however, social heredity loomed large in Baldwin's thought:

> "It is as inexorable as the colour of his eyes and the shape of his nose. He is born into a system of social relationships just as he is born into a certain quality of air. As he grows in body by breathing the one, so he grows in mind by absorbing the other.' (Baldwin, 1906: 69-70)[4]

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[4] Page references are to the fourth edition. Chapter two, 'The Social Person' was not substantially revised in these later editions. As Baldwin notes in the prefaces to these later editions, he is publishing new books

The increased importance of social heredity in human beings as compared to other animals plays a key role in Baldwin's typically optimistic Edwardian account of social progress. Individuals who fail to acquire the level of civilization of the society around them because of some defect in their 'physical heredity' are eliminated by 'social suppression of the unfit'. Anti-social mental traits are eliminated by criminal sanctions - execution and imprisonment - and also by reproductive isolation, since the instincts of anti-social individuals are abnormal and do not 'fit' with the instincts of potential mates. Meanwhile, pro-social mental traits flourish. By this means, once social heredity has evolved, evolution has an intrinsic tendency to move to higher and higher levels of social organization (Baldwin, 1906: 80-90). Leaving aside the theme of 'onwards and upwards' which was common to so many evolutionary theorists at this time (Ruse, 1996), Baldwin's account of social heredity was a theory of what would today be called 'niche-construction' (Odling-Smee, 1988). Social heredity alters the selective forces that act on the units of biological heredity so as to alter the course of biological evolution. This general theory of social heredity figures prominently even in Baldwin's papers devoted specifically to the Baldwin effect. In the *Nature* and *Science* papers of 1897 he argues that his term 'social heredity' is to be preferred to Lloyd Morgan's term 'tradition' because it emphasizes the causal relationship between one human mind and the next (Baldwin, 1897a: 558; 1897b: 636). In 'A New Factor in Evolution' he argues that, not only does social heredity facilitate the Baldwin effect, "it has a farther value. *It keeps alive a series of functions which either are not yet, or never do become, congenital at*

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and so it is not important to revise the old ones to keep up with his changing views (although he is gratified they are still being reprinted!)

all." (Baldwin, 1996: 67, his italics). Most striking of all, when Baldwin describes how social heredity 'tends to set the direction of phylogenetic progess' (Baldwin, 1996: 67), he does not describe a case in which a character initially transmitted by social heredity later becomes congenital (the Baldwin effect) but instead describes a case in which social heredity of one character changes the selection pressures on other, quite different characters of the organism (niche construction). The separation of human racial types now living together in the American South, he argues, is due to the social transmission of 'a repugnance to black-white marriages', which is the '*influence of education, imitation, etc'*. Yet the effects of this process of social heredity will 'appear in our fossils when they are dug up long hence by the paleontologist of the succeeding aeons!' (Baldwin, 1996: 67-68, his italics)[5].

Not only did Baldwin continue to discuss social heredity and its impact on biological evolution by means other than the Baldwin effect in the years 1896-7, his work after 1987 show no sign that he felt that the Baldwin effect had replaced his more general account of the interaction between biological and social heredity. He discusses the Baldwin effect in two later works, but with no sign that he has worked the process out in more detail or applied it to specific cases (Baldwin, 1902, 1909). The primary focus of Baldwin's writing after 1897 continued to be the psychological processes by which the child comes

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[5] This example occurs elsewhere in Baldwin's writing. In *Social and Ethical Interpretations* he enlarges on it and adds that this repugnance is not shared by 'Northern whites' (Baldwin, 1906: 87-8). It is hard not to read these passages in the light of the fact that Baldwin - a Southerner - was dismissed from Johns Hopkins in 1908 on suspicion of having had sexual relations with an African-American prostitute (Richards, 1987).

to cope with its individual environment, processes discussed with a mass of detail in his multi-volume *Genetic Logic* (Baldwin, 1906-11). Simpson claims that Baldwin’s period of active interest in the Baldwin effect ended around 1903 (Simpson, 1996: 100, fn 1.), and this seems a reasonable assessment.

The prominence Baldwin gave to the Baldwin effect in 1896-7 and his redefinition of ‘organic selection’ to refer to the ‘new factor in evolution’ can perhaps best be explained by his interest in obtaining scientific priority. The Baldwin effect was obviously an idea whose time had come and Baldwin wanted credit as its inventor. His tendency to claim priority too readily was evident elsewhere in his career. Baldwin was accused in print of having taken his theory of imitation - the core of his theory of mental development - from the work of the French sociologist M.G Tarde without adequate acknowledgment (Baldwin, 1906: viii, xi-xiv). Robert J. Richards has suggested that Baldwin’s tendency to aggressively assert his priority was not unconnected to the fact that the rest of American psychology showed more than the usual degree of *schadenfreude* in the face of his disgrace and dismissal in 1908 (Richards, 1987).

4. Why has the Baldwin effect been remembered?

As David Depew (this volume) makes clear, the Baldwin effect has been substantially redefined more than once in its century-long career as a controversial, but never completely disreputable aspect of evolutionary theory. Authors including C. H Waddington, G.G Simpson, I. I Schmalhausen, R. Matsuda and M. J West-Eberhard have described diverse mechanisms by which acquired characters could become hereditary consistently with Weismann’s doctrine, and all of these mechanisms have been described as Baldwin effects. Clearly, it is not the details of Baldwin’s theory that have commanded such sustained interest, but rather the possibility of some mechanism or other filling the abstract role he identified.

There are two reasons for the continuing interest in finding some mechanism to fill this role. The first, and better known, is the idea that the Baldwin effect gives ‘mind’ the chance to ‘direct’ evolution and thus saves us from the barren Darwinian vision of a world ruled by chance and necessity. It was this prospect that attracted cultural critics such as George Bernard Shaw (Shaw, 1939) and Arthur Koestler (Koestler, 1972) to the Baldwin effect. But it is entirely unclear to me how, absent some prior commitment to an immaterialist account of mind, the fact that the evolution of intelligence has influenced future evolution is challenging to conventional Darwinism. That fact is exactly on a par with the fact that the evolution of plants led to an increase in atmospheric oxygen and so influenced future evolution. The tendency to find the influence of intelligence on evolution vaguely reassuring persists even today. The evolutionary psychologist Geoffrey Miller, commenting on his theory that human intelligence evolved by sexual selection, reassured a journalist that his "is probably the least reductionist theory of the mind's evolution one could hope for... It doesn't reduce psychology to biology, but sees psychology as a driving force in biological evolution." (Smith, 2000: 7s). But, like the Baldwin effect, sexual selection is no less biological than any other selection process and the sexual preferences that drive it are material biological products like any other. The only Darwinian account of mind I can imagine that could reasonably be described as more ‘reductionistic’ than Miller’s would be Thomas Henry Huxley’s epiphenomenalism, according to which minds are ineffectual side-effects of the brains that have been produced by evolution (Huxley, 1896). But if epiphenomenalism was ever a live option for Darwinists[6], it has not been so for many decades. The idea that the Baldwin effect will save the mind from evolutionary irrelevance is a confused solution to a non-existent problem.

The second motive for the continuing interest in the Baldwin effect is both more sensible and more important, since it is the motive that has drawn many significant biologists to consider the effect. Ever since Weismann asserted that the germ plasm is passed on from generation to generation unaffected by the bodies that house it, many well known causes of individual variation have been excluded from playing a role in evolution, as opposed to individual development. Developmental biologists, and developmental psychologists like Baldwin, have had to accept that only phenotypic differences caused by genetic changes can provide the raw material for evolution. Furthermore, since natural selection acts only on the genes, an understanding of evolutionary dynamics seems to require only an understanding of selection and an understanding of genetic heredity. Phenotypic differences are selected and the genes responsible for those differences are differentially replicated. Developmental biology and psychology may be interesting in their own rights, but they are not important topics for a student of evolution. Richard Dawkins makes this point in a well-known passage:

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[6] Huxley himself may not have held the view usually attributed to him (Campbell, 2001).

> “when we are talking about development it is appropriate to emphasise non-genetic as well as genetic factors. But when we are talking about units of selection a different emphasis is called for, an emphasis on the properties of replicators... The special status of genetic factors is deserved for one reason only: genetic factors replicate themselves, blemishes and all, but non-genetic factors do not.” (Dawkins, 1982: 98-9)

Insert Figure 1 about here.

The view that organisms inherit nothing but their genes is often explained using a diagram distantly related to the cell-lineage diagrams that Weismann used to explain his doctrine of the continuity of the germ plasm (Griesemer & Wimsatt, 1989). In the familiar diagram of ‘molecular weismannism’ there is a causal arrow from the genes of one generation to those of the next and a causal arrow from the genes of each generation to the phenotype of that generation. There is, however, no causal arrow from the phenotype of one generation to the phenotype of the next. The diagram is so familiar that it is hard to look at it with a naive eye. If one were to do so, however, it would pose an immediate puzzle. Everyone knows that in many species parents feed and nurture their offspring and that differences in feeding and nurturing lead to differences in the phenotype. Every biologist knows that parents pass on DNA methylation patterns and maternal DNA products in the cytoplasm of the egg, not to mention membranes, microtubule organizing centers and many other essential features of a functioning cell that cannot be generated *de novo* by a set of naked chromosomes. Many of these features are sources of significant variation in the offspring's phenotype. What has happened to the causal arrows representing these processes? They are omitted from the diagram because they are taken to be real *developmental* phenomena but not real *evolutionary* phenomena. These causal influences may have an effect on the phenotype of the next generation, but they will not endure for long enough to play a significant role in evolution. John Maynard Smith has made this point in a characteristically forthright manner:

> "Differences due to nature are likely to be inherited whereas those due to nurture are not; evolutionary changes are changes in nature, not nurture." (Maynard Smith, 2000: 189)

The Baldwin effect has continued to excite interest, I suggest, because it allows students of development to draw evolutionary implications from their developmental studies. If acquired traits can become hereditary then knowledge of how such traits are acquired in development is relevant to the construction of evolutionary scenarios. Furthermore, understanding evolutionary dynamics will involve understanding development as well as selection and genetic heredity. But the Baldwin effect is neither necessary nor sufficient to make development evolutionarily relevant. It is not *sufficient* because, as Simpson argued, even if it occurs there is no reason to suppose that it is ubiquitous. The conventional neo-Darwinian approach of which Simpson was an advocate would probably remain a good approximation to the truth and the Baldwin effect would be a mere footnote to that theory (Simpson, 1953). The Baldwin effect is not *necessary* to establish the relevance of developmental to evolutionary studies for two reasons. First, as

I will argue in the next section, there is good reason to think that epigenetic inheritance is an important factor in evolution even if the phenotypic differences thus transmitted never become incorporated in the germ line. Baldwin's 'social heredity', of course, was a form of epigenetic inheritance. Second, as I discuss in section six, one way in which parents influence their offspring is by constructing the niche that the offspring will occupy. Niche-construction fundamentally influences evolutionary dynamics because it implies that organisms are not so much adapted to their niches as coevolved with them (Brandon & Antonovics, 1996; Lewontin, 1982, 1983). Baldwin, anticipated this idea, in a primitive way, in his discussion of how social heredity could 'set the direction of phylogenetic progress' without the operation of the Baldwin effect.

5. Three ways to Marginalize Epigenetic Inheritance

The first way to marginalize epigenetic inheritance is, as discussed above, to assume that it is too unstable and fluctuating to be the basis of cumulative change by natural selection. Since it is cumulative selection, rather than one-step selection, that produces complex adaptation this would seem to relegate epigenetic inheritance to a minor role at best. Kim Sterelny has suggested that this is likely to be particularly true of cultural inheritance: Baldwin's social heredity may exist, but it does not lead to adaptations (Sterelny, 2001). This deflationary view of cultural inheritance may quite possibly be correct, but our current state of knowledge about the longevity of cultural traditions in animals and the degree to which they have involved cumulative changes is too inadequate to allow any substantial conclusion to be drawn. There is considerable cultural variation in tool use, grooming, and courtship behaviors between populations of wild chimpanzees (Whiten et al., 1999). However, there is little evidence that these variations have persisted for long periods of time, nor that they are a product of cumulative cultural change. It is possible that sensitive periods and social and ecological scaffolding may facilitate reliable cultural inheritance. One example is the reliability of human linguistic inheritance, which is supported by a host of such psychological adaptations. Language-like features in other species may be supported in the same way and thus exhibit reliable cultural inheritance. A study on chaffinches in the Atlantic Islands by found substantial congruence between a tree based on morphology and a tree constructed from song syllables, indicating a common evolutionary history going back 1 to 2 million years (Lynch & Baker, 1986). A study on the possible effect of cultural selection on mtDNA diversity in whales suggests that cultural inheritance has exhibited considerable longevity and fidelity in those species with matrilineal social systems. The results of computer simulations indicate that, assuming a 10% reproductive advantage is culturally transmitted down maternal lineages, it would take over 200 generations to produce the observed tenfold reduction in mtDNA diversity observed in lineages with matrilineal social systems (Whitehead, 1998). We know of at least one case in which a process of social heredity in insects has persisted for long enough to create an adaptation. The morphology of queens and the colony structure of the fire ant *Solenposis invicta* differ radically between different lineages because of stably replicated pheremonal nest 'cultures' (Keller & Ross, 1993). Some lineages have nests with several small queens, whilst others have nests with a single large queen. Moving eggs from one colony type to another reveals that a queen raised in a colony with a particular ‘culture’ will develop the body type appropriate to her adopted culture and found a new colony with the same culture. This - pheremone transmitted - difference between colony types has persisted long enough for genes affecting growth rates in queens to diverge as each lineage adapts its growth rate to the demands of either the monogynous or polygynous lifestyle. These cases and others like them suggest that the ability of cultural inheritance - and of epigenetic inheritance more generally - to reliably reproduce features over evolutionarily significant periods of time is an open empirical question and one whose investigation is overdue.

But even if epigenetic inheritance sometimes exhibits high copying fidelity over long timespans, this may not mean that it can play a similar evolutionary role to genetic inheritance, generating the kind of variation on which natural selection can feed to produce complex adaptations. In *The Major Transitions in Evolution*, John Maynard-Smith and Eörs Szathmáry assign a vital role to membrane heredity and the inheritance of endosymbionts as sources of the evolutionary innovations in cell-compartmentalization and cell organelles that created the fundamentally different types of organism we see today - the various types of bacteria and the animals, plants and fungi (Maynard Smith & Szathmary, 1995). These epigenetic inheritance systems play a major role in Maynard Smith and Szathmáry's account of evolution but, they argue, it is not a role that puts these systems on a par with the genes. The genetic inheritance system and cultural transmission in humans are the only two systems that display what they call 'unlimited heredity', the form of heredity that makes possible the evolution of complex adaptation. Most inheritance systems, they argue, can only mutate between a limited number of heritable states which can be specified in advance. Habitat imprinting, for example, can only lead organisms to choose different habitats. DNA methylation can only choose whether existing genes will be switched on or off. The genome and language, however, both have a recursive syntactic structure. Their basic constituents can be put together in many different combinations and these combinations can be of any length. Hence these inheritance systems have an unlimited number of possible heritable states.

The distinction between limited and unlimited heredity systems is an interesting one, and may provide insights into one of the key innovations - an inheritance system with this sort of recursive structure - that enabled the diversification of early life into the vast range of forms we see today. However, it is possible to take issue with the idea that unlimited heredity is a function of the genes and unlimited heredity a function of methylation, membranes and other non-genetic heritable developmental resources. A developmental systems interpretation, according to which the capacity for unlimited heredity is a property of the whole matrix of material resources, is equally defensible (Gray, 2001; Griffiths, In Press September 2001). The way Maynard Smith and Szathmáry interpret the limited/unlimited heredity distinction involves, in effect, partitioning the total number of developmental outcomes that can be generated by a developmental system between the various inheritance systems that make up that system. The number of outcomes allotted to an inheritance system measures its ‘limitedness’ and the genetic inheritance system is allotted massively more outcomes than any other system. To derive this result, Maynard Smith and Szathmáry assume that the number of permutations of DNA codons is the relevant measure of ‘limitedness’ for the genetic inheritance system and that some corresponding measure of the number of permutations of physical parts is the appropriate measure for other inheritance systems. But this is not the only way to partition the developmental outcomes amongst the inheritance systems, nor is it obviously the right way. For any one inheritance system, the range of physical changes that count as evolutionary changes is restricted to those that can be made use of by the rest of the developmental system. This point can be easily grasped by looking at the syntactic structure of human languages - the analogue to which Maynard Smith and Szathmáry are appealing. Not all physical differences between syntactic objects are *syntactic* differences. Differences in handwriting or in the accent with which one speaks, for example, are not syntactic differences. Syntactic differences are those physical differences that are treated as differences by the broader system for which this set of physical objects constitutes a set of linguistic inscriptions. The main lesson of the major evolutionary transitions that are the focus of Maynard Smith and Szathmáry's work is that evolution creates entirely new kinds of developmental systems that massively expand the possible interpretations of existing developmental resources, including genes. Think how many evolutionary possibilities can be caused by a base pair substitution in the DNA of a eukaryote cell that could not be caused by those same substitutions in a prokaryote cell. As Maynard Smith and Szathmary themselves describe, that vast swathe of evolutionary possibilities came into existence through evolutionary change mediated by a limited heredity system. Membranes cannot be constructed without an existing membrane template into which to insert newly synthesized proteins. Hence, major changes to the partitioning of the cell require variation to arise through the membrane heredity system, not through mutations of the DNA. In effect, the measure of 'limitedness' that Maynard Smith and Szathmáry adopt allots to the genetic inheritance system all the outcomes that can be generated by making changes to that system across the full range of possibilities for the other systems while allocating to the other systems only the number of outcomes they could produce given one possible genome. That is why it is not an appropriate measure. It would be no less reasonable to allocate to the genetic inheritance system only the range of outcomes it could generate given one state of the other inheritance systems and to allocate to some other heredity system all the outcomes it could generate given the whole range of possible genomes. What I want to suggest, however, is not that we should engage in such reverse discrimination, but that we should recognize that ‘unlimitedness’ is a property of a developmental system, not of any one of its physical components.

This conclusion can be reinforced by an independent argument. The potential of differences in genes, language and perhaps pheremones in social insects to cause a large number of phenotypic outcomes stems from the fact that these differences 'mean something' to the rest of the developmental system. If the rest of the system surrounding the genes were such that indefinitely many base-pair combinations collapsed into only a few developmental outcomes, then the genetic inheritance system would not be unlimited. It is not hard to imagine cellular machinery with this result - the existing genetic code is substantially redundant in just this way, with several codons corresponding to the same amino acids. Hence, the unlimited nature of the genetic inheritance system is more accurately seen as a property of the developmental system as a whole and not of the genome in isolation.

A third way to marginalize epigenetic inheritance is to argue that only genes have the capacity to generate a fine-grained response to selection and thus to give rise to adaptation. Many epigenetic inheritance systems, such as habitat imprinting or the inheritance of gut endosymbionts, seem to change state in a way that corresponds not to the genetic micro-mutations that conventional neo-Darwinians take to be the fuel of natural selection, but rather to macromutations. Like genetically induced macromutations, single changes in these inheritance systems produce large changes in the phenotype, such as a whole new habitat or a whole new symbiont lineage. It is widely believed that this kind of discontinuous change cannot give rise to adaptation. However, as Russell Gray has pointed out, this argument implicitly assumes that epigenetic inheritance systems must have evolutionary potential separately from one another and that only discrete, as opposed to continuous, variations count as alternative states of an inheritance system. Gray argues that:

> “Extragenetic changes can also be piecemeal and incremental. Just as natural selection can favor combinations of genes at different loci, so selection may favor combinations of endosymbionts. Quantitative variations in cytoplasmic factors, nest design, and habitat preferences could also all be passed on extragenetically. Thus, although combinations of these factors are not unlimited, they can be quite large enough to allow a fine-grained response to selection.” (Gray, 2001: 197).

I have considered three arguments designed to show that epigenetic inheritance has only a marginal role in evolution. The first, the claim that epigenetic inheritance is too unstable to allow cumulative selection of its products, was inconclusive, pointing only to the need for empirical investigation of the actual properties of epigenetic inheritance systems. The other two arguments simply devised inappropriate criteria against which to assess epigenetic inheritance, requiring that it have a significant evolutionary role not in the actual world, in which epigenetic inheritance systems exist as part of a larger developmental system, but in imaginary scenarios in which evolution must proceed using one, decoupled epigenetic inheritance system at a time. These two arguments may show that epigenetic inheritance systems could not have evolutionary significance in the absence of the genetic inheritance system, but that is not at issue. The real evolutionary significance of epigenetic inheritance remains to be determined.

6. Niche Construction and Evolution

Richard Lewontin’s critique of the 'lock and key' model of adaptation is well known (Lewontin, 1982, 1983). In that metaphorical conception of natural selection, adaptations are solutions (keys) to the problems posed by the environment (locks). Organisms are adapted to their ways of life because they have evolved to fit those ways of life. In place of this traditional metaphor Lewontin suggested a metaphor of construction. Organisms and their ecological niches are co-constructing and co-defining. Organism's both physically shape their environments (co-construction) and determine which factors in the external environment are relevant to their evolution (co-definition) thus assembling some subset of the biotic and abiotic factors in their physical environment into a niche. Organisms are adapted to their ways of life because organisms and their way of life were constructed for (and by) one another.

The most detailed attempt to develop the metaphor of construction into a program of actual biological research is that of F. J. Odling-Smee and his collaborators (Laland, Odling-Smee, & Feldman, 2001; Odling-Smee, 1988, 1996; Odling-Smee, Laland, & Feldman, 1996). The current prominence of the term 'niche-construction' is due to this group. Odling-Smee's treatment differs significantly from Lewontin's is that he is concerned not to represent the organism-environment system as a closed system. It is well known that in Australian eucalypt forests, which require frequent bushfires to maintain themselves and where the trees have many features that make bushfires more prevalent, the trees themselves have co-evolved with the bush-fire prone landscape in which they thrive. Nevertheless, the evolution of this organism-environment system was exogenously driven by the progressive drying of the Australian continent. Organisms do, of course, feel the impact of changes in the environment in it's traditional sense of their total biotic and abiotic surroundings, but they experience these impacts via the environment as it appears in relation to them, and thus different lineages experience 'the same changes' quite differently. Odling-Smee tries to respect this situation by assigning separate roles to the environment of a particular lineage of organisms and what he calls the 'universal physical environment'. The former, organism-referent description of the environment is the source of evolutionary pressures on that organism, and the organism is the source of niche-constructing pressures on that environment. The latter, universal physical environment is a source of exogenous change in the organism's environment.

Robert Brandon's theory of the role of the environment in adaptation is a useful complement to Lewontin and Odling-Smee's ideas. Brandon distinguishes not two but three different senses of 'environment' (Brandon, 1990). His 'external environment' corresponds to Odling-Smee's universal, physical environment. All organisms in a particular region of space and time share one external environment. The 'ecological environment', however, must be described with reference to a particular evolving lineage. It consists of those environmental parameters whose value affects the reproductive output of members of the lineage. The idea that only these parameters are part of the organism’s niche corresponds to Lewontin’s claim that organisms and niches are co-defining. Finally, the 'selective environment' is that part of the ecological environment which *differentially* affects the reproductive output of variant forms in the evolving lineage. It is in this last sense of ‘environment’ that the environment contains the sources of adaptive evolutionary pressures on the lineage. Brandon has used these ideas in his own exploration of organism-environment co-evolution (Brandon & Antonovics, 1996). Organisms modify the selective and ecological environments in numerous ways that can potentially influence their evolution. Only some of these modifications of the selective and ecological environments also constitute modifications of the external environment (actual co-construction rather than co-definition) but whether they do so is unimportant when determining their role in the future evolution of the organism-environment system. This is why the simple notion of the ‘external’ or ‘universal physical’ environment is inadequate for understanding organism-environment relationships. Many changes in the external environment do not constitute change from the point of view of the organism and, conversely, an organism can transform its environment without actually changing the universal physical environment (by becoming arboreal or troglobytic, for example). It is what Brandon calls the ecological environment and Lewontin calls an organism-referent description of the environment that defines what counts as a 'change' in the environment for that organism.

The phenomena of epigenetic inheritance and niche-construction are closely related. Both the co-constructing and co-defining relationships between organisms and their environments are frequently mediated by epigenetic inheritance. Inheritance mechanisms such as habitat and host imprinting create associations between an evolving lineage and one aspect rather than another of the external environment, defining an ecological environment out of that wider set of available environmental parameters. The actual, physical construction of aspects of the niche is an epigenetic inheritance mechanism in its own right: the inheritance by the offspring generation of modifications made to the environment by the parental generation. The recognition of the importance of niche-construction in evolution thus reinforces the conclusions of sections four and five, that no Baldwin effects are necessary to allow causal relationships between parent and offspring other than those through the germ line to be of evolutionary significance

7. Conclusion

I have suggested that the role of the Baldwin effect in Baldwin’s own biological thought was as an addendum to a more general theory of social heredity and its possible impact on biological heredity. Baldwin conceived social heredity as an alternative, parallel causal channel creating resemblance between parent and offspring - what we would now call an epigenetic inheritance system. He also understood that social heredity could alter the ecological forces impinging on the organism as a whole, and thus affect the fate of the biological hereditary material - an idea now referred to as niche construction. Even in his period of greatest enthusiasm for the Baldwin effect he saw that process as only one way in which social heredity could affect the future of biological heredity. The other way in which this could occur - niche-construction - is there right alongside the Baldwin effect in ‘A New Factor in Evolution’. In a limited way and in the context of a very dated, Edwardian, progressive conception of the evolutionary process as a whole, Baldwin anticipated these contemporary ‘new factors’ in evolution.

I have suggested that biologists have continued to show interest in the Baldwin effect because of the conviction that unless epigenetic inheritance can leave traces in the DNA it cannot make a significant contribution on an evolutionary timescale. But I have argued that this conviction is mistaken and that the significance of epigenetic inheritance is, at the very least, an open empirical question (see also Moore, this volume). Baldwin effects are very likely not necessary for epigenetic inheritance and niche construction to play a significant role in evolution. Nor would the existence of Baldwin effects guarantee those two processes a significant role. If Baldwin effects occur there is, as Simpson argued, no reason to suppose that they are a major mechanism of evolutionary change. Baldwin effects are thus of limited interest and should not be allowed to distract attention from the more general significance of epigenetic inheritance and niche construction.

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Figure 1. A visual representation of the idea that organisms inherit nothing but genes from their parents, a view often described as ‘molecular weismannism’.