

Piaget's Paradox: Adaptation, Evolution, and Agency

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Abstract

Piaget's *Behaviour and Evolution* (1976) sought to reconcile the view that organismal adaptiveness – in the form of equilibration – could contribute to human behavioural, cognitive, and epistemic evolution with the prevailing evolutionary orthodoxy of the time. He was particularly concerned to demonstrate that human behaviour, cognition, and knowledge acquisition could be drivers of human evolution. Piaget hypothesised constructive role for organisms in evolution was significantly at variance with the prevailing modern synthesis orthodoxy of his time (and ours). He looked to Conrad Hal Waddington's genetic assimilation as a model for how equilibration could generate evolutionary novelties which become fixed by subsequent evolution. I make two claims. Firstly, that Piaget's appeal to Waddington fails to reconcile his views of human evolution with the modern synthesis. Secondly, the newly emerging agential conception of evolution, in which the purposive activities of organisms are the principal causes of evolution, offers strong support to Piaget's model of "organisational" evolution.

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Introduction

One of the enduring themes of Piaget's work on psychological development, behaviour, epistemology, and organismal evolution was the need he saw for continuity. Piaget considered that human knowledge, cognition, and behaviour evolve, and they do so through the same processes by which physiology and anatomy evolve (Burman, 2019; Messerly, 2009). He sought an account that unified evolution in all these domains. Piaget saw striking parallels between biological organisms in physical settings and human minds in epistemological settings. "Eventually his theory and the evidence led him to propose that there are functional invariants – organisation, adaptation, assimilation, accommodation, and equilibration – that exist in both realms" (Messerly, 2009: 94). These functional invariants, observable across development, behaviour, and knowledge acquisition arise out of the adaptiveness of organisms. Organisms are complex adaptive systems constantly in dynamic commerce with their conditions of existence (Burman, 2013), and this fundamental feature of organisms, Piaget surmised, is crucial to the understanding of all aspects of evolution. He understood that this emphasis on the adaptive activities of organisms as the principal driving force put his conception of the process of evolution far beyond the pale of the orthodox evolutionary biology of his time. In his *Behaviour and Evolution* (1976), he sought a reconciliation, drawing heavily on the later works of developmental geneticist, C.H. Waddington.

I argue for two claims here. The first is that Piaget's attempted reconciliation fails. Waddington offers no solution to Piaget's problem of assimilating human behaviour, cognition, epistemology into the evolutionary thinking of his time. The second is that this failure notwithstanding, Piaget's project should not be abandoned. The intervening years in evolutionary biology and its philosophy gesture towards a nascent organism-centred evolutionary biology that has the potential to cast Piaget's project in a new, positive light. Piaget's conception of evolution finds a congenial home in a newly emerging alternative to the modern synthesis, the agential view of evolution. Whereas, the modern synthesis precludes Piaget's conception of behavioural, cognitive, and epistemic evolution, the agential view renders what Piaget called his "hazardous" proposal plausible, tractable, and empirically testable.

Piaget's Paradox

In *Behaviour and Evolution* (1976), Piaget attempts to carve out a special contributory role for human behaviour in evolution. Human behaviour, Piaget insists, clearly generates novel, adaptive responses in our interactions with the world, and these, one might suppose, can become objects of evolutionary change. But the prevailing "neo-Darwinian" approach to evolution at the time offered no account of how it could. This situation Piaget finds profoundly paradoxical. "...the net effect of neo-Darwinism's ever-expanding influence on the one hand and the considerable headway made by ethology on the other one hand has been an extremely paradoxical situation, which needs to be dealt with in some detail" (Piaget, 1976: 24). The problem, as Piaget sees it, is that "the neo-Darwinist position attribute[s] an evolutionary role to behaviour solely at the level of selection, and not at the level of the actual formation of hereditary variations" (1976: 25). That is to say, neo-Darwinism allows us to say that when novel behaviours become inheritable, and vary within a population, they can be objects of selection, but it recognises no processes by which behaviours could become evolvable¹. If neo-Darwinism could offer no such account, Lamarckism surely could. But Piaget was aware that Lamarckism had been so thoroughly discredited at the time that whatever solution he devised to his paradox must find some intermediate ground between them. Piaget was not alone in seeking a via media between Darwinism and Lamarckism. As we shall see,

¹Except of course random genetic mutation.

others – notably Baldwin, Conwy Morris, and Osborn – had tried. But Piaget's attempt uniquely prefigures more recent advances in evolutionary thinking.

Equilibration

Piaget defined behaviour as: "activity directed by organisms towards the outside world in order to change conditions therein or to change their own situation in relation to these surroundings" (Piaget, 1976: ix). He is in no doubt about its evolutionary importance: "It is of the essence of behavior that it is forever attempting to transcend itself and that it thus supplies evolution with its principal motor" (1976: 139). In this respect, behaviour exemplifies what Piaget took to be a fundamental feature of all living things, their adaptiveness. He held that organisms and their environments constitute a dynamic system in which the organism responds to its circumstances, thereby creating new circumstances, in what Piaget called "circular reaction" (Burman, 2013).

Piaget was convinced that organisms, as dynamical complex systems, provided the principal source of the variation that constitutes the raw material of evolution. Evolution begins with "equilibration." Equilibration involves the capacity of organisms to improve their fit to their conditions of existence, by altering themselves or their conditions. Chapman (1992) summarises equilibration as follows:

1. Every organism tends to preserve itself.
2. Each organism modifies its constituent parts in response to environmental influences, which in turn brings the parts into conflict with the whole.
3. The whole minimises this internal conflict by modifying itself.
4. A stable equilibrium develops in which parts and wholes are mutually preserved.

According to Boom, "Equilibration is the tendency of the subject to develop increasing control over experience" (Boom, 2009: 132–133). This capacity of organisms to accommodate, assimilate, and equilibrate is the source of those characters that evolution entrenches and promotes. "It thus appears that evolutionary causation is found in the relationship between the organism's openness to its environment and its organizational properties" (Messlerly, 2009: 100). Because evolution is driven by organismal adaptedness, its direction is biased. "Not only did he posit that the same general constructive process is responsible for both evolutionary and developmental change, but he also suggested that chance plays only a marginal causal role" (Burman, 2019: 289). Piaget dubbed this conception of evolution "organisational" and contrasted it explicitly

with the prevailing “variational” approach prominent at the time (Bickhard, 1988). The core precepts of “variational” evolution were given their definitive articulation by Richard Dawkins, in the very same year that *Behaviour and Evolution* was published.

Evolution is the external and visible manifestation of the survival of alternative replicators. . . . Genes are replicators; organisms. . . are best not regarded as replicators; they are vehicles in which replicators travel about. Replicator selection is the process by which some replicators survive at the expense of others (Dawkins, 1976: 82).

Evolution is principally a phenomenon of genes, and only derivatively of organisms. The variation on which evolution relies is not initiated by organisms, but is a brute chemical given (Walsh, 2023). Arvid Ågren has provided a recent update.

By this reasoning, organisms are nothing but temporary occurrences – present in one generation, gone in the next. And, as a consequence, organisms cannot be the ultimate beneficiary in evolutionary explanations.

Instead, this role is filled by the gene. Genes are considered immortal and they pass on their intact structure from generation to generation. . . . natural selection is conceptualized as a struggle between genes, usually through the effects they have on organisms, for replication and transmission to the next generation (Ågren, 2021: 2).

He later affirms: “The gene’s eye view has been at the centre of evolutionary debates for the past half-century” (Ågren, 2023).

The contrasts between the variational and organisational conceptions of evolution are stark. According to the variational approach, evolution is (i) gene-centred, (ii) ineluctably chancy, and (iii) biased only by the external demands of the environment. On Piaget’s organisational account, evolution is (i’) organism-centred, (ii’) non-chancy, and (iii’) biased by the adaptive capacities of individual organisms.

And yet, as Piaget insisted, there is evidence for his account of organism-led evolution. His own training as a biologist prepared him for this. Piaget’s early biological works centred around environmentally triggered variation in *Limnaea stagnalis*, a freshwater snail. *L. stagnalis* has two morphologies, an elongated spiral form and a low-spired, globular form. The elongated shell develops in slow-moving water, while the globular morph develops in fast-moving streams. A snail with a low, rounded shell is less prone to being swept away in fast flowing water. So, the adaptation that *Limnaea* sp. morphology shows is clearly adaptive. As Piaget was aware, this adaptive form originates within organisms during their lifetimes, and yet, over time, can become

intergenerationally stable. Pure breeding lines of either morph can be established in which the morphology emulates the Mendelian patterns of inheritance. Offspring of heavily selected low-rounded morphs develop the low-rounded morph no matter what environment they are raised in (Messerly, 2009). This looked, not unreasonably to Piaget, like a case in which individual organisms had responded adaptively to equilibrate with their setting, and then assimilated these adaptive changes as inheritable traits.

While this phenomenon appeared to be unexplainable by the precepts of gene-centred, variational evolution, it looked superficially like a case of the inheritance of acquired characters of the sort often associated with Lamarck. Piaget was fully aware that, however, Lamarckian explanations were at the time beyond the pale. So, he sought a way to reconcile this evidently Lamarckian phenomenon with the prevailing gene-centred view on evolution. In seeking this middle way, he looked to Waddington’s (1957) *Strategy of the Genes*.

Piaget, Waddington (and Baldwin, Lloyd Morgan, and Osborn)

The problem that Piaget faced bore similarities to a proposal made in 1896 by the psychologist James Mark Baldwin (and virtually simultaneously by Conwy Lloyd Morgan, and Henry Fairfield Osborn). Baldwin (Baldwin, 1896) proposed a “new factor” in evolution by which learnt, cognitive and social adaptations could eventually become instinctual, and robustly inherited (Depew, 2003). Piaget, was strongly influenced by Baldwin’s views (Burman, 2013). The so-called “Baldwin effect” (Simpson, 1953) fared poorly in the face of the growing dominance of the modern synthesis. The common complaint was that Baldwin offered no mechanism by which a genuinely novel trait, initiated by learning or acquired through behaviour or cultural transmission, could by repeated rounds of selection become genuinely inherited. One of the founders of the Synthesis, Ernst Mayr, suggested “discarding this concept altogether” (Mayr, 1963: 611). Mayr argues that those novelties that are simply the expression of a mutation or a latent genetic disposition should be treated in the same way as any other genetic trait. There is nothing new or surprising in these cases. Mayr insists that the alternative, that these traits are genuinely novel and *not* underwritten by genes, should be dismissed as a misguided lapse into Lamarckism (Depew, 2003). According to Mayr, the middle ground between Lamarckism and neo-Darwinism that Piaget wishes to

stake out simply does not exist. Piaget's claims that behaviour, learning, and the acquisition of knowledge are crucial drivers of evolution would suffer the same fate as the Baldwin effect were there no mechanism by which those traits initiated through organismal adaptiveness (equilibration) could be genuinely inherited.

In this context, Piaget turned to Conrad Hal Waddington. Waddington's visit to Geneva in 1964 greatly excited Piaget (Burman, 2013). He drew heavily on Waddington's work, explicitly so in *Behaviour and Evolution* (1976). In particular, Waddington's conception of genetic assimilation suggested a possible mechanism by which organisational evolution could be made consistent with the prevailing modern synthesis approach to evolution².

Waddington's problem was that of explaining the appearance and eventual inheritance of phenocopies. A phenocopy, in Waddington's sense, is an environmentally induced, uninherited trait. Waddington demonstrated that through repeated iterations of environmental stress and selective breeding, phenocopies could become robustly inherited even in the absence of the environmental stresses that initiate them³.

Waddington subjected his populations of *Drosophila melanogaster* eggs to extreme environmental conditions far beyond those encountered in their natural setting. In one treatment (Waddington, 1953), developing zygotes were exposed to heat shock, which produced in some individuals the crossveinless phenocopy. In another experiment (Waddington, 1956), zygotes were made to develop in excess levels of ether. The result is the dramatic "bithorax" phenocopy, in which these flies develop two thoracic segments. Selective breeding of the phenocopy mutants and repeated exposure to environmental extremes yielded the result that these phenocopies could eventually become robustly inheritable, and furthermore be induced to develop even in the absence of the extreme environmental trigger⁴. As Waddington put it: "...selection for the ability to respond to an environmental stimulus has built a strain in which the abnormal phenotype comes to be

produced in the absence of any abnormal environment" (Waddington, 1953: 123). Waddington's experiments appear to demonstrate that environmentally induced novelties that arise de novo within individual organisms can become reliably inherited. Waddington dubbed the process by which phenocopies become robustly inherited "genetic assimilation."

Waddington (1957) introduced a powerful image to explain genetic assimilation, the epigenetic landscape. The epigenetic landscape depicts the development of a phenotype as a ball rolling down an inclined surface. The contours of the epigenetic landscape – or the "chreode" in Waddington's terminology – guide the developing character along its path. The landscape contains a series of bifurcating valleys. When the developing trait meets a bifurcation point, small triggers propel it to one channel or the other. In this way, traits (cells, tissues, etc.) differentiate. The valleys of the epigenetic landscape have variably steep banks. Near the top, the valleys are shallow, representing the potential for a feature in its early stages to develop into wide array of structures, tissues, or cell types. Towards the bottom, the banks are steep. Here, late in the trajectory, development is robust, capable of withstanding a range of perturbations. Any disturbance, genetic or environmental, may deflect the developing feature up the side of the bank, but it will be guided by the steep bank back to the bottom of the valley. This steepening of the valleys represents the progressive robustness of development, something Waddington calls "canalisation." Crucially, the structure of the epigenetic landscape is undergirded by the interactions between genes (Waddington coined the term "epigenetic" for these interactions).

Waddington used the epigenetic landscape to explain the initial appearance and eventual genetic assimilation of phenocopies. Under conditions of extreme environmental stress, he surmised, the trait or structure is propelled from the valleys of the landscape early in development. In these "uncharted" regions of the landscape, new phenotypes (phenocopies) can occur. Subsequent iterations of breeding and selection favour individuals more reliably capable of producing the phenocopy. In effect, selective breeding selects for a new epigenetic landscape, one in which the phenocopy develops its own valley; it becomes canalised.

Waddington's model of genetic assimilation is brilliant, and prescient. It anticipates the massive expansion of recent research in epigenetics (Noble, 2015). It is hardly surprising that it excited Piaget. He saw in it a possible solution to his paradox of behaviour, not to mention the problem of the Baldwin effect. It is clear that

²Waddington plays a complicated role in this narrative. While Waddington's early writings situate him firmly in the organicist tradition (Peterson, 2016), his *Strategy of the Genes* is explicitly an attempt to reconcile his views on development and inheritance with the prevailing modern synthesis orthodoxy. Ironically, epigenetics, the concept that Waddington's coined in effecting this reconciliation has inspired many of the most rigorous challenges to the modern synthesis (Noble, 2015). I thank a referee from this journal for pointing out this complexity.

³A referee from this journal points out to me that Waddington credits Piaget as being the first to demonstrate that the evolutionary fixation of phenocopies can occur in the wild.

⁴Crossveinless became practically fixed in non-heat shock environments in 20 generations (Waddington, 1953). Bithorax was inheritable in 70% of individuals on non-stressed environments after 18 generations (Waddington, 1956).

Waddington, like Piaget, thought he had reconciled the evident inheritance of “acquired characters” with modern synthesis evolutionary biology, without falling prey to Lamarckism. He claimed that in articulating genetic assimilation, no “new fundamental genetic principles have been disclosed” (1975: 92). Rather, “[t]he theory of genetic assimilation, and the practical demonstration that the process can occur, . . . provides a new way of accounting for all those evolutionary facts for which, in the past, some authors were tempted to advance a “Lamarckian” explanation (1975: 92). According to Piaget’s version of genetic assimilation, novel traits introduced by equilibration eventually become canalised through iterated selection. But it is not entirely clear that Waddington’s genetic assimilation model is the solution that Piaget requires, for reasons suggested by Mayr (above). To understand why Waddington’s genetic assimilation model does not provide a solution to Piaget’s paradox, we need to investigate in a little more detail the evolutionary orthodoxy that they both encountered. I shall call it the “two spaces and a barrier” view (following Walsh, 2014).

Spaces and Barriers

Twentieth century evolutionary thinking seems to have grown up around two guiding principles, the centrality of the gene and an animadversion to Lamarck. The antipathy to Lamarck set in long before the establishment of the gene concept. Indeed, it is reasonable to suppose that the anti-Lamarckian sentiment made possible the centrality of the gene concept. The reason is that whereas Lamarckism posits the organism as both the source of novelties and the locus of inheritance, gene centrism locates these phenomena in the activities of genes. Genes constitute an isolated realm of novelty, inheritance, and developmental information, completely sealed off – sequestered – from the changes that occur to individual organisms during their lifetimes.

Auguste Weissman, and avid early German supporter of Darwinism, was one of the first to adopt this strategy of sequestration, long before the discovery of the gene. In exquisitely detailed embryological experiments, Weismann demonstrated that there is an early separation between the germ plasm and the somatoplasm. The cells of the somatoplasm go on to develop and differentiate into the adult form. The germ plasm is the site of gametogenesis. Crucially, only germ plasm materials are passed on in reproduction. The germ line is sequestered from the somatoplasm, isolated from changes that affect an organism’s form during its development. So, Weis-

mann surmised, changes in the somatoplasm – acquired characters – could not be inherited. The Weismann doctrine secures the stricture against Lamarckism, and in the process, it constructs two discrete spaces – germ line and somatoplasm – with a barrier between them. One of these spaces takes theoretical precedence. Only changes in the germ line can be inherited; so, only they can be genuinely evolutionary changes.

The Weismann doctrine is limited at best. The sequestration of the germ line occurs only in metazoans – and even then, not in all⁵ – and yet the stricture against the inheritance of acquired characters is thought to apply universally to evolution⁶. A more plausibly general version of the two spaces and a barrier view arose with the central dogma of molecular biology. The central dogma, coincidentally was first articulated in the year in which Waddington (1957) published *The Strategy of the Genes*.

Crick (1957) initially formulates it thus: “The Central Dogma: ‘Once information has got into a protein it can’t get out again.’” He later offers a revision: “The central dogma of molecular biology deals with the detailed residue-by-residue transfer of sequential information. It states that such information cannot be transferred back from protein to either protein or nucleic acid” (Crick, 1970). It isn’t clear what the central dogma really tells us about evolution; it is essentially a claim about protein synthesis. But it appears, like the Weismann doctrine, to suggest that the stuff of inheritance (DNA) is unchanged by the processes by which organismal phenotypes are built (in this case, transcription). In doing so, it reinforces, and greatly expands the scope of, the two spaces and a barrier view of evolution.

It is worth pointing out that strictly speaking, neither the Weismann doctrine nor the central dogma precludes the inheritance and evolution of acquired characters. For that matter, neither disqualifies the purposiveness of organisms from participating in evolution. One could consistently hold to both the Weismann doctrine and the central dogma and still countenance additional non-genetic modes of inheritance, and non-genetic sources of novelty. So, Piaget’s organisational evolution could in principle co-exist happily with Weismannism and the central dogma. The exclusion of Piaget’s mode of “organisational” evolution requires much more stringent

⁵ I thank a reviewer from this journal for pointing this limitation out.

⁶ I thank a referee from this journal for pointing out that sequestration of the germ line is not universal among metazoans. Besides, unbeknownst to Weismann, it is false on empirical grounds. As later became apparent, germ line cells can acquire characteristics and transmit them to offspring (Lacal and Ventura, 2018).

conditions. In the case of the modern synthesis version of evolution, these strictures are realised in the explicit redefinition of inheritance, and indeed evolution.

Genotype Space and Phenotype Space

The two spaces and a barrier conception of evolution receives its definitive formulation in the modern synthesis theory of evolution. The modern synthesis theory describes evolution by means of two abstract 'spaces': phenotype space and genotype space (Lewontin, 1974). Phenotype space comprises all the possible biological forms and possible changes in form. Genotype space comprises the frequencies of gene types in a population and their possible changes. There is a division of labour between these spaces that roughly corresponds to the difference between explanans and explanandum; genotype space is the explanans and phenotype space is the explanandum. What we want our evolutionary theory to explain – the changes in biological form over time, the adaptedness and diversity of organisms – are phenomena of phenotype space. The modern synthesis explains these by appeal to processes defined over genotype space.

Here is how it works. For evolution to happen, four things must happen: (i) offspring must resemble their parents (inheritance); (ii) organisms must grow and differentiate between inception and reproduction (and beyond) (development); (iii) there must be a source of novel characters (innovation); and (iv) a process that introduces a bias to evolutionary change (adaptation). The modern synthesis casts each of these as a process belonging to genotype space. Inheritance is the replication and transmission of genes. Development is the expression of a program encoded in genes. Innovation is random genetic mutation (and genetic recombination). Adaptive bias is the differential retention of genes in a population due to natural selection. When the four component processes occur together, evolution moves a population through genotype space. These changes are reflected in changes in form, in phenotype space. To be sure, not all changes in genotype space are registered as changes in phenotype space (Kimura, 1973) and not all changes in phenotype space are registered as changes in genotype space (e.g., Flynn effect, changes in BMI over generations). The primacy of genotype space is underscored by the fact that changes count as evolutionary if and only if they are registered in genotype space. Neutral evolution – genotype space change without accompanying phenotype space change – is after all, considered to be evolution; the Flynn effect, in which there is change in phenotype space (IQ) over generations without changes in genotype space, isn't.

According to the modern synthesis, the component process of evolution – inheritance, development, innovation, and bias – are quasi-autonomous. Each can occur in the absence of the others. But adaptive evolution only occurs if they all do. Moreover, each process is more or less unaffected by the others; they are “quasi-autonomous” (Uller and Helenterä, 2019). Adaptive bias does not affect inheritance or development. Development for its part introduces no novel features or adaptive biases. Mutation, from which all evolutionary innovations arise, is adaptively unbiased.

The separation of genotype space from phenotype, the gene-centred redefinition of evolutionary processes, and the stipulation that only changes that register as changes in genotype space qualify as evolutionary, succeed in effecting the alienation of organisms from evolution that the Weismann barrier and the central dogma only partially succeeded in doing. It is important to understand that these are theoretical commitments of the modern synthesis, postulated and stipulated, rather than empirically observed.

Proximate and Ultimate

The implications of this separation of genotype space and phenotype space were drawn explicitly by Ernst Mayr (1961, 1982). Mayr noted that there are two kinds of questions that biologists frequently ask, “how” and “why”: “how do hearts appear in individual chordates” and “why do chordates have hearts?”. The second type of question appears to call for a teleological explanation, one that cites the contribution that a structure plays to the purposes of an individual organism. But Mayr, in keeping with modern synthesis orthodoxy, believes that organismal purposes cannot explain evolutionary change. So, he offers us a translation. “Why” questions, he argues, implicitly call for an explanation of the prominence of a trait type in a population. These explanations advert to what Mayr calls “ultimate causes.” Ultimate causes are evolutionary processes – processes that operate over populations in evolutionary time. Most prominently, these include the transmission of genetic material, the differential selection of genetic variants (and genetic drift). “How” questions, for their part, are implicitly appeals to explain the appearance of trait tokens within individual organisms. These explanations appeal to what Mayr calls “proximate” causes, those biological processes that take place within organisms during their lifetimes. Proximate causes, according to Mayr, are not causes of evolutionary change⁷.

⁷The distinction is vigorously disputed by Laland et al. (2011).

Assimilation versus Equilibration

This is the evolutionary orthodoxy that Piaget and Waddington both encountered. There are obvious commonalities to the problems that faced them both; each had to reconcile the two spaces and a barrier picture of evolution with what appeared to be the inheritance of characters acquired within an individual's lifetime. That is to say, each had to demonstrate how "proximate causes" can become evolutionary causes. Indeed, as Piaget (1976) himself points out, Waddington (1975) proffered genetic assimilation as an account of the heritable adaptive responses of *Limnaea sp.* that so inspired Piaget⁸. Yet, these superficial similarities notwithstanding, the problems that face Waddington and Piaget are quite divergent. Genetic assimilation is not equilibration. Consequently, it is not at all clear that Waddington's solution applies to Piaget's problem.

Waddington's solution is explicitly an attempt to reconcile the phenomenon of the phenocopy with the modern synthesis picture. His model is one in which the epigenetic constraints upon the capacity of genes to express a particular phenotype (e.g., crossveinless or bithorax) are removed by the introduction of a severe perturbation. The new phenotype can be interpreted not so much as a genuine evolutionary novelty but as a latent trait whose expression had previously been suppressed. Not only were Waddington's phenocopies not novelties, they were not obviously adaptations either. As far as anyone could tell from Waddington's experiments, the new traits were not adaptive, except in the highly contrived surroundings of Waddington's selective breeding experiments. Nor did they arise from developmental – much less behavioural or cognitive – adaptation or equilibration. So far as Waddington's canalisation model tells us, there is nothing about the adaptive plasticity of organisms, their ability to equilibrate, that contributes to evolution.

With the two spaces and the distinctions between proximate and ultimate causes in place, it becomes easy to see why Waddington's genetic assimilation is not a solution to Piaget's paradox. Waddington finds a way to describe the origin and fixation of phenocopies in genotype space terms. In doing so, he cites ultimate processes of gene selection and recombination, the expression of a program encoded in genes. In a sense, Waddington was able to bypass proximate causes; his genetic assimilation model is consonant with the modern synthesis doctrine that all evolutionary causes are ultimate causes. A solution to Piaget's paradox, however, requires us to describe the origin and fixation of traits in terms of the proximate processes of

organismal adaptation, equilibration, learning, etc. There seems little prospect of a genuine reconciliation between Piaget's conception of organisational evolution and the modern synthesis.

Challenging the Modern Synthesis

The very "two spaces and a barrier" picture that precludes Piaget's organisational evolution has recently come under severe scrutiny. Routinely, its challengers charge that the modern Synthesis needs to be radically overhauled (Laland et al., 2014), extended (Pigliucci & Müller, 2010), or even jettisoned completely: "Darwinism in its current scientific incarnation has pretty much reached the end of its rope" (Depew & Weber, 2011: 90). The challenges fall into two general categories. (i) The modern synthesis conception of the component processes of evolution is empirically inadequate. (ii) The component processes of evolution are not related in the way that the modern synthesis supposes them to be (Burman, 2019; Laland et al., 2015; Pigliucci & Müller, 2010; Uller & Helanterä, 2019). The challenges are numerous and diverse. Taken together, they serve to weaken the dominant orthodox modern synthesis "two spaces and a barrier" position. At the same time, they suggest an alternative view of evolution, one much more congenial to Piaget. A brief survey of these challenges will help motivate the alternative.

Empirical Inadequacy

A diverse and voluminous body of recent research suggests that there is more to inheritance, development, innovation, and adaptive bias than is captured by the strictly gene-centred processes of the modern synthesis.

Inheritance

Perhaps the most commonly noted empirical deficiency of the modern synthesis lies in its account of inheritance. Critics point out that if a theory of inheritance is to offer an explanation of the pattern of resemblance and difference between lineages across generations, then the mere transmission of genes is completely inadequate. There are many different extragenetic processes involved (Jablonka & Avital, 2006; Jablonka & Lamb, 2010; Jablonka, 2017). Epigenetic inheritance provides a vivid case in point. Epigenetic marks are applied to DNA and to chromatin by a cell in response to an organism's (or the cell's) endocrine, immune, nutritional, or stress conditions⁹. These marks

⁸I thank a referee from this journal for pointing out this connection.

⁹For a discussion of some transgenerational consequences of maternally induced immune activation, see Pollok and Weber-Stadlbauer (2020).

are transgenerationally stable (Lacal & Ventura, 2018; Weiner & Katz, 2021), lasting sometimes (for example, in *C. elegans*) for up to 40 generations without genetic change (Heard & Martienssen, 2014)¹⁰. Epigenetic marks regulate gene expression in ways that frequently constitute adaptive responses that persist for generations (Herman et al., 2017). By any reasonable account of inheritance, the transmission of epigenetic marks ought to qualify. Yet, epigenetic marks are acquired characters, many of which originate in the adaptive response of organisms (Ferguson-Smith, 2011).

Once epigenetic processes are permitted to contribute to inheritance, there is little principled objection to the inclusion of other extragenetic processes too. Organisms pass on their immunological properties; “. . .the transfer of parental immunological experience to enhance the offspring immune defence is present in both vertebrates and invertebrates, and can be inherited for multiple generations” (Linn & Spagopoulou, 2018: 205). And much more: they pass on their endocrine adaptations (Zhang & Ho, 2011); their microbiota (Grieneisen, 2021).

Organisms also construct and maintain the environments necessary for their offspring to resemble them (Odling-Smee et al., 2003). In short, the resources of inheritance are spread throughout the gene-epigene-development-environment system.

The existence of non-genetic modes of inheritance is increasingly recognised in mainstream biology.

The weight of theory and empirical evidence indicates that non-genetic inheritance is a potent factor in evolution that can engender outcomes unanticipated under the Mendelian-genetic model (Bonduriansky & Day, 2009: 103)¹¹.

Once extragenetic processes are acknowledged to participate in the process of inheritance, it appears that the modern synthesis account of inheritance as replicator transmission is woefully inadequate (Jablonka & Lamb, 2010).

Replication underdetermines persistence of form. . . . genetic and epigenetic inheritance systems constitute complementary mechanisms of adaptation to an environment whose many changes occur along different time scales (Danchin et al., 2019: 5).

There is more to inheritance than the transmission of replicators. This is to say that the modern synthesis representation of inheritance exclusively in genotype space is inadequate.

¹⁰For in-depth discussion of the evolutionary significance (or otherwise) of epigenetic inheritance, see Gapp (2020); Sarkies (2020). See, especially Jablonka (2017).

¹¹Admittedly, Bonduriansky and Day (2018) seek to secure special theoretical privilege for genetic inheritance in explaining evolutionary dynamics.

Development

It is widely recognised that organismal development is highly plastic, robust, and context-sensitive: “to develop is to interact with the environment and to be responsive to context” (Moczek, 2023: 4). The context-sensitive responsiveness of development facilitates trait integration, and the coordination of developing systems. That, in turn, requires a broad developmental repertoire, a range of possible responses that the developing organism can mount in response to unpredictable circumstances, and a concomitant capacity of an organism to enlist its developmental repertoire in context-appropriate ways. The most striking feature of evolutionary developmental biology of the last quarter century is the discovery of the ways in which genotype radically underdetermines phenotype. Proper organismal development is under the control of all manner of extragenetic processes (Gilbert & Epel, 2015; West Eberhard, 2003). This has been repeatedly demonstrated by evolutionary developmental biology, ecological developmental biology (Gilbert & Epel, 2015; Sultan, 2015), by developmental systems theory (Oyama et al., 2000), niche construction theory (Odling Smee et al., 2003), and the plasticity of development¹².

Organismal development is highly adaptively plastic (Gilbert & Epel, 2015; Pfennig, 2004; Sultan, 2015). Developmental plasticity confers on organisms a developmental robustness, the capacity to respond to genetic and extragenetic perturbations in ways that secure the proper development of phenotypes¹³. Organisms of the same sort reliably develop their typical phenotypes not because of genetic and environmental similarity but despite the vagaries of each (Wagner, 2011). This developmentally induced or constrained variation can be a major contributor to phenotypic evolution (Uller et al., 2018; Hu et al., 2020; Jablonka, 2017). The same plasticity also confers on organisms the capacity to produce alternative phenotypes that suit their conditions (Newman, 2022). The various ways in which developmental plasticity might contribute to adaptive evolution are well rehearsed (Ciliberti et al., 2007; Kirschner and Gerhard, 2010; Pfennig et al., 2010; Wagner, 2012; West Eberhard, 2003)¹⁴.

Adaptive Bias

Phenotypic variation is generated by the processes of development, with some variants arising more readily than others – a phenomenon known as “developmental bias”

¹²On developmental plasticity, see also (Badeaux and Shi, 2014; Mitchell et al., 2018).

¹³Levis and Pfennig (2020) survey the evidence for “plasticity-led evolution.”

¹⁴See, for example, Kirschner and Gerhard (2010), Wagner (2012), West Eberhard (2003), Pfennig et al. (2010).

(Uller et al., 2018)¹⁵. Developmental bias is manifest as the production of novel forms (Hu et al., 2020; Uller et al., 2018; Salazar-Ciudad, 2021). Furthermore, epigenetic mechanisms drive – bias – the adaptedness of evolution (Badeaux & Shi, 2014; Weiner & Katz, 2021). The phenomenon of “plasticity-led evolution” is increasingly being recognised as a source of adaptive bias (Hu et al., 2020; Laland et al., 2019; Levis & Pfennig, 2020; Uller et al., 2018, 2020; Salazar-Ciudad, 2021). The leading idea here is that the capacity of individual organisms to respond adaptively to their circumstances imparts an adaptive bias to the evolution of populations. It is worth noting that this adaptive developmental bias simply an instance adaptive bias that Piaget observed in *Limnaea* snails.

Novelty

According to the modern synthesis perspective, all evolutionary novelties are introduced by mutations, and mutations are random. But even if mutations are random, it does not follow that their phenotypic effects are (Nuño de la Rosa & Villegas, 2022). Besides, it is becoming increasingly apparent that evolutionarily significant (and heritable) novelties do not require mutations (West Eberhard, 2003). As stable (inherited) variants that contribute differentially to survival and reproduction, they can be subject to natural selection across multiple generations. And yet, they may require no new genes (Uller et al., 2018; Hu et al., 2020; Jablonka, 2017).

As we have seen, the plasticity of development confers on organisms a broad phenotypic repertoire (West Eberhard, 2003) which in turns allows organisms to innovate, to produce new stable phenotypes in new conditions (Ledón-Rettig et al., 2008). Developmentally induced or constrained variation can be a major contributor to phenotypic evolution.

Much novelty in evolution thus appears to be possible without the need to evolve novel genes, pathways, or cell types. Exactly why, how, and when evolutionary innovations occur and unfold the way they do has thus mostly eluded conventional molecular-, population-, and quantitative genetic approaches towards understanding the evolutionary process (Sultan et al., 2022: 4).

Fractionation

One significant implication of the two spaces and a barrier view of evolution is that each of the component processes of evolution has its own proprietary genetic cause. Crucially, these processes are to a great degree independent of one another. According to the orthodox

view, the introduction of novelties, as we have seen, is exclusively the domain of mutation. It is not affected by development, or inheritance, nor does it introduce an adaptive bias. Development neither introduces novelties, nor adaptive bias. The process of inheritance – being the simple transmission of genes – itself does not bias what is inherited.

As we have also seen, recent work in evolutionary developmental biology and related fields renders this fractionated quasi-independence implausible. Rather, recent evolutionary biology suggests that the component processes of evolution are holistically related (Walsh, 2023). This holism suggests that one and the same process may be the cause of any or all of the component processes of evolution at the same time. Development may simultaneously introduce novelties, impart an adaptive bias to evolution, and secure transgenerational stability. Epigenetic marking may be at once an adaptive response, the introduction of an evolutionary novelty, and a transgenerationally stable character. An organism’s interaction with its environment may likewise generate an adaptive bias, secure the transgenerational stability of characters and promote developmental stability all at once. There is little reason, short of modern synthesis dogma, to suppose that each of the component processes of evolution has its own discrete, proprietary gene-based process, much less that they are independent of one another.

Together, this battery of challenges to the modern synthesis amount to the charge that the processes operating within and defined over genotype space are insufficient to capture the evolutionary changes in phenotype space. But it is change in phenotype space, that as Richard Lewontin reminds us, “we are trying to explain in the first place.” Certainly, there are sufficient grounds for exploring an alternative to the modern synthesis of two spaces and a barrier model.

The Agential Perspective

Neither Piaget nor Waddington could have foreseen how the conceptual landscape was to change. In particular, neither could have anticipated how empirical findings of the early 21st century would undermine the foundation of the modern synthesis. In view of the evident deficiencies of the orthodox modern synthesis view, biologists and philosophers of biology are advocating for alternatives; there are a number of suggestions concerning expansions of or alternatives to the modern synthesis picture. There is a range of proposed extensions,

¹⁵For extended discussions of developmental bias, see: Laland et al. (2019), Uller et al. (2021), Levis and Pfennig (2020).

expansions, and alternatives (Laland et al., 2014, 2015; Pigliucci, 2009; Pigliucci & Müller, 2012). One loose assemblage of views goes under the rubric the extended evolutionary synthesis¹⁶. I choose to highlight one alternative here, the agential view. Although the agential view has only recently emerged it holds out the prospect of a genuine solution to Piaget's paradox.

Philosophers and biologists have recently suggested that the deficiencies of orthodox evolutionary thinking originate from a single source. The modern synthesis omits a particularly significant and defining feature of organisms, *viz.* their agency. Some contend that thinking of organisms as agents yields a new perspective on evolution, one significantly at variance with standard modern synthesis gene-centred approaches¹⁷. The guiding insight of the agential perspective is that, as Richard Lewontin put it: "the organism cannot be regarded as simply the passive object of autonomous internal and external forces; it is also the subject of its own evolution" (Lewontin, 1985: 89).

On this view, organisms enact evolution (Thompson, 2007; Walsh, 2023). The idea of organisms enacting evolution is reminiscent of Piaget's concept of "organisational evolution", in which organisms have the capacity to adapt themselves to their conditions of existence, by marshaling their genetic, extragenetic, environmental, and behavioural resources. This responsiveness of organisms in turn provides the source of novelty, persistence, and adaptive bias needed for evolution. The same processes that empower organisms to adapt also secure the transgenerational stability of these forms. In this way, the traits secured or originated by an organism's purposive pursuit of its own goals – its biology – creates the conditions required for evolution. In exploiting the plasticity of their development, in originating novel forms, in securing their inheritance, organisms are acting as agents.

Agents, unlike like run of the mill objects, experience their conditions of existence as affordances (Walsh, 2012). An affordance is a property of the interaction between an organism and its conditions (Gibson, 1979).

The affordances of the environment are what it offers the animal, what it provides or furnishes, for good or ill. . . I mean by it something that refers to both the environment and the animal. . . It implies the complementarity of the animal and the environment (Gibson, 1979: 127).

¹⁶The implications of the EES for Piaget's thought are expertly surveyed by Burman (2019).

¹⁷For discussion of the agential perspective, see Jaeger (2021), Newman (2023), Nadolski & Moczek (2023), Sultan et al. (2022), Thompson (2007), Uller (2023), Varela Thompson & Rosch (1991), Walsh (2012, 2015, 2023).

That is to say, an organism does not experience its conditions merely as causes of its responses, but as circumstances that are relevant to the pursuit of its goals. An agent copes with its affordances – exploiting or ameliorating them – by marshaling and directing its own repertoire of available responses towards the attainment of its goals. There is a dynamic reciprocity between an organism and its affordances. In responding to its affordances, an organism creates new opportunities for action, new challenges to the pursuit of its goals. This adaptive, purposive coping, adjusting, innovating that is the very nature of organisms is also the driver of adaptive evolution.

The agential perspective offers an alternative conception of the component processes of evolution, and their interrelatedness. Crucially, it is not bound by the modern synthesis gene-centred definitions of the component processes of evolution. Inheritance, far from being the transmission of replicators as the modern synthesis insists, is the pattern of resemblance and difference between lineages across generations, howsoever that pattern is secured. Development, for its part, is not the expression of a program encoded in genes; it comprises the complete suite of genetic, epigenetic, and environmental influences that go into the growth and differentiation of an organism from inception to (at least) reproduction. Novelty is not restricted to genetic mutation; it is constituted by any new transgenerationally stable character, by whatever means it arises. Likewise, adaptively biased population change is more than the differential retention of genes. It is the change in phenotypic adaptedness in a population, however it is secured.

Moreover, according to the agency perspective, there is no reason to suppose that the component processes of evolution are quasi-independent. One and the same capacity of organisms – e.g., developmental plasticity, epigenetic marking, niche construction, immunological, endocrine, or microbial transmission, behaviour, learning – can cause any combination of the component processes of evolution at once. The unifying feature of all these processes is agency of organisms. When organisms exert their adaptive agency, the adaptive evolution of populations ensues.

The agency perspective predicts that the purposive activities of organisms should be seen to participate in each of the component processes of evolution. This, as we have seen above, is strongly borne out empirically. The goal-directed purposiveness of organisms appears, for instance, to be essential to the explanation of the origin of evolutionary novelties. Even those novelties that arise from random genetic mutations are subject to the

adaptive responses of organismal development (Nuño de la Rosa & Villegas, 2022). These novelties, moreover, are not adaptively neutral; they are frequently adaptively biased (Hu et al., 2020; Uller et al., 2020). Likewise, the source of the adaptiveness of adaptive change appears to reside in the purposive activities of organisms. The contribution of organisms in development, innovation, and in securing the intergenerational stability of form is a significant source of adaptive bias in evolution (Hu et al., 2020; Laland et al., 2019; Levis & Pfennig, 2020; Salazar-Ciudad, 2021; Uller et al., 2018, 2020, 2021). Adaptive evolutionary change involves and requires the participation of organisms as complex adaptive systems, creating and responding to their affordances.

Crucially, the agency perspective inverts the presumptive priority of genotype space over phenotype space. On the agential view, evolutionary dynamics takes place exclusively in phenotype space, and is caused by the activities of organisms, their plastic development, their adaptive responses to their circumstances. Genotype space dynamics – the change in the frequency of genotypes over time – is a highly abstracted, idealised reflection of the evolutionary processes occurring in phenotype space (Walsh & Rupik, 2023). It is part of an abstract theoretical tool for describing and quantifying the dynamics of populations over time (Walsh et al., 2017). One implication of this abstract conception of genotype space is that, in the manner of all abstract scientific models, it is only a partial representation of evolutionary dynamics. It is highly likely that there are genuinely evolutionary changes that do not register as changes in gene frequency. Evolutionary changes that arise from adaptive novelties, behaviour, niche construction, epigenetic response, and inheritance, that do not require changes in underlying genes, may leave no trace in genotype space.

In effect, the agential perspective is a repudiation of the two spaces and a barrier conception of evolution encoded in the modern synthesis. There is no distinction between evolutionary processes and biological processes, as Mayr's proximate/ultimate distinction requires (Laland et al., 2015). Rather, evolution is the consequence of organismal biology extended over time. The causes of evolution are all biological; they reside almost exclusively in phenotype space. The evolutionary perspective thus helps explain why the gene-centred modern synthesis account of evolution should be so empirically inadequate. It looks for the causes of evolution in the wrong place. The causes of evolution are not to be found in the statistical principles of population change; they are to be found in the lives and deaths of individual organisms (Walsh, 2019).

The agency perspective has precursors. In chapter 3 of the *Origin*, Darwin asks: "How have all those exquisite adaptations of one part of the organization to another part, and to the conditions of life, and of one distinct organic being to another being, been perfected?" (1859 [1968]: 114). His answer adverts to the purposive activities of organisms, the 'struggle for life'. "All these results . . . follow inevitably from the struggle for existence." Struggling, 'in the large and metaphorical sense' in which Darwin intended, is simply the organism's goal-directed pursuit of its way of life. According to the view that the agential perspective shares with Darwin, the purposive activities of organisms are the principal causes of adaptive evolution.

Significantly, for our purposes, the role of agency in evolution is strongly reminiscent of Piaget's claims about the motivating force of behaviour. The capacity of organisms as agents to respond to their affordances bears a striking similarity to Piaget's concept of equilibration. The reciprocal, co-constituting relationship between organisms and their affordances is strongly reminiscent of Piaget's conception of the "circular reaction" between organisms and their environments (Burman, 2013). This is particularly evident in Piaget's account of the role of behaviour in evolution. He defined behaviour as an "activity directed by organisms towards the outside world in order to change conditions therein or to change their own situation in relation to these surroundings" (Piaget, 1976: ix). As recent biology demonstrates equilibration can also be seen in the plasticity of development, the adaptive epigenetic marking, niche construction. He goes on to suggest that this equilibration must be a principal cause of evolution. As we saw assert, above, equilibration "supplies evolution with its principal motor" (Piaget, 1976: 139). This claim is strikingly consonant with the idea that the purposive agency of organisms is the principal cause of adaptive evolution.

In further convergence with the agency perspective, and in contrast to the modern synthesis, Piaget denied that evolution is ineluctably chancy. The agency perspective explains why. Organisms exert a degree of influence over the amount, kind, and degree of variation in a population. Evolutionary novelties are not random with respect to the survival and/or reproduction of the individuals they arise in. They are biased by the capacity of organisms to respond to their circumstances in ways that promote their own survival. The recent extensive work on developmental bias, and plasticity-driven evolution (cited above), convincingly bears out the non-chancy nature of evolutionary change. The adaptiveness of evolutionary change is inherent in the purposiveness of organisms (Newman, 2022).

This non-random nature of evolutionary dynamics was anticipated by Piaget, but discounted by the modern synthesis.

The agential perspective on evolution is only beginning to receive serious attention from biologists as a genuine, plausible, empirically tractable alternative to the modern synthesis (Jäger, 2021; Nadolski & Moczeck, 2023; Sultan et al., 2022; Uller, 2023; Walsh, 2015). But already at this early stage of its development, it is clear that it bears striking resemblances to Piaget's conception of "organisational" evolution. We saw, above, that Piaget's conception of organisational evolution has three principal features. To recap: for Piaget evolution is (i) organism-centred rather than gene-centred, (ii) driven by the purposive, adaptive abilities of organisms, (iii) directed rather than random. These features are all reflected indeed, explained, by the agential conception of evolution.

Conclusion: Piaget's Error

At the time of the publication of *Behaviour and Evolution* (1976), Piaget's quest for continuity between the domains of human psychological development, behaviour, epistemology, and organismal evolution seemed a forlorn hope. The prevailing "variational" evolutionary biology of the time was predicated on the notion that the processes of evolution are wholly distinct and discrete from those that occur within the lives of individual organisms. Variational evolution as (correctly) described by Piaget is gene-driven, mechanistic, and ineluctably chancy. He clearly saw that his project of unifying behaviour and evolution would require an alternative conception of evolution, one he dubbed "organisational." By contrast, organisational evolution (his "hazardous conjecture") is driven by the adaptiveness of organisms, as seen in their "equilibration." Equilibration is manifest not only in behaviour but also in human cognition, and knowledge acquisition. Organisational evolution requires inter alia that characters that arise in the lifetimes of individual organisms as a result of their adaptive responses to their circumstances can become inheritable and selectable.

These claims must have appeared highly implausible to the orthodox modern synthesis thinking of the time. Nevertheless, the modern synthesis was the only reputable account of evolution available to Piaget. Small wonder, then, that he sought some form of reconciliation. In *Behaviour and Evolution*, Piaget exploited Waddington's genetic assimilation. Genetic assimilation is Waddington's own attempts to render the phenomenon of the phenocopy consistent with modern synthesis evolution.

Waddington's reconciliation works for Waddington's purposes, but it does not provide Piaget with what he needs. Genetic assimilation does not provide a means by which equilibration can drive evolution. It does not require the adaptiveness of organisms to be the principal source of evolutionary novelties, inheritance of characters, and the adaptive bias of evolutionary change in the way that Piaget's "organisational" evolution does. So, while genetic assimilation is consistent with the modern synthesis, Piaget's "organisational" evolution is not.

In the intervening years, the theoretical landscape has altered so radically that a reassessment of Piaget's "organisational" evolution is now warranted. A growing number of evolutionary biologists no longer see the need to pay obeisance to the modern synthesis. In particular, in the past 10 years or so an alternative conception of evolution has begun to emerge, to be articulated, and to gather theoretical and empirical support. The agential view holds that adaptive evolution is a consequence of the activities of organisms as adaptive, self-building, self-regulating purposive entities. Organisms uniquely have the capacity to exploit and mitigate their affordances in the pursuit of their goals of survival and reproduction. The agential capacities of organisms confer on them an enormous degree of robustness and plasticity. These features empower organisms to secure their own successful development, to originate novelties, to secure the conditions for the transgenerational constancy of form, and to bias the process of evolution. Adaptive evolution is inherent in the capacities of organisms, not dictated by the vagaries of chance. In short, on this view, agency is the principal cause of adaptive evolution. In depicting evolution in this way, the agency perspective completely repudiates the two spaces and a barrier conception of evolution that became entrenched in modern synthesis thinking. Moreover, it denies the distinction between the biological processes and evolutionary processes encoded in Mayr's proximate/ultimate distinction. Evolution is simply biology extended over generations.

While the agential perspective is a repudiation of modern synthesis evolution, it seems to suggest a rehabilitation of Piaget's "organisational" evolution. Piaget's organisational evolution is wholly consistent with the agential view. Piaget's conception of the role of behaviour, cognition, and human knowledge acquisition, far from being a failed extension of the modern synthesis theory of evolution should rightly be considered a prescient anticipation of this newly emerging agential conception of evolution. I'm not suggesting that either the agential view or "organisational" evolution was framed with the other in mind. Rather, my claim is that there is a serendipitous

congruity between them. Consequently, the considerations that count in favour of the agency view also warrant a re-evaluation of Piaget's "organisational" evolution.

Piaget's "hazardous conjecture" is that organismal purposiveness, in the form of "equilibration" in all its facets, is a driving force of evolution. The phenomenon of equilibration encompasses organismal development but also extends to behaviour, learning, and knowledge acquisition. Piaget's radical hypothesis, then, amounts to the claim that these features of human behaviour and cognition, evolve and contribute to human evolution in the same way that any other feature of human biology does. At the time this hypothesis was framed, it was so out of keeping with evolutionary thinking that there seemed to be no way to subject these claims to empirical test, and little reason to do so. Piaget's model of behavioural, cognitive, and epistemic evolution may be inconceivable from the perspective of the modern synthesis, but I have been suggesting here that it is much more congenial to the newly emerging agential alternative. The agential perspective on evolution is far from attaining widespread support in current evolutionary biology. It is nascent and largely underdeveloped as a comprehensive competitor to the modern synthesis. But it already has a great deal of empirical support. It largely avoids the empirical inadequacies of the modern synthesis. It further provides a framework for unifying the battery of otherwise disparate challenges to the modern synthesis. One implication of the agential perspective is that it transforms Piaget's bold "hazardous" conjecture about human behavioural, cognitive, and epistemic evolution from a conceptual impossibility into a live empirical issue.

References

- Ågren, A. (2021). *The gene's-eye view of evolution*. Oxford University Press.
- Ågren, A. (2023). Genes and organisms in the legacy of the modern synthesis. In T.E. Dickins, & J.A. Dickins (Eds), *Evolutionary Biology: Contemporary and Historical Reflections Upon Core Theory*. (pp. 555–568). Springer.
- Badeaux, A., & Shi, Y. (2013). Emerging roles for chromatin as a signal integration and storage platform. *Nature Reviews Molecular Cell Biology*, 14(4), 211–224. <https://doi.org/10.1038/nrm3545>
- Baldwin, J. M. (1896). A new factor in evolution. *American Naturalist*, 30(June), 354, 422, 441–451, 536–553. <https://doi.org/10.1086/276408>
- Bickhard, M. (1988). Piaget on Variation and Selection Models: Structuralism, Logical Necessity, and Interactivism. *Human Development*, 31(5), 274–312. <https://doi.org/10.1159/000275815>
- Bonduriansky, R., & Day, T. (2009). Nongenetic inheritance and its evolutionary implications. *Annual Review of Ecology, Evolution, and Systematics*, 40(1), 103–125. <https://doi.org/10.1146/annurev.ecolsys.39.110707.173441>
- Boom, J. (2009). In U. Müller, J. I. M. Carpendale, & L. Smith (Ed), *Cambridge Companion to Piaget*. (pp. 132–149). Cambridge University Press.
- Burman, J. T. (2013). Updating the Baldwin effect. *New Ideas in Psychology*, 26(6), 751–772. <https://doi.org/10.1177/0959354316672595>
- Burman, J. T. (2019). Development. In R.J. W. SteinbergPickren (Ed), *The Cambridge Handbook of the Intellectual History of Psychology*. (pp. 287–317). Cambridge University Press.
- Chapman, M. (1992). Equilibrium and the Dialectics of Organization. In H. Beilin, & P. Pufall (Eds), *Piaget's Theory: Prospects and possibilities*. (pp. 39–60). Lawrence Erlbaum Associates.
- Ciliberti, S., Martin, O. C., & Wagner, A. (2007). Innovation and robustness in complex regulatory gene networks. *PNAS*, 104(34), 13591–13596. <https://doi.org/10.1073/pnas.0705396104>
- Crick F.J. (1957). Nucleic acids. *Scientific American*, 197(3):188–200. <https://doi.org/10.1038/scientificamerican0957-188>
- Crick, F.J. (1970). The Central Dogma of Molecular Biology. *Nature*, 227(5258), 561–563. <https://doi.org/10.1038/227561a0>
- Danchin, E., Pocheville, A., & Huneman, P. (2019). Early-in-life effects and heredity: Reconciling neo-Darwinism with neo-Lamarckism under the banner of the inclusive evolutionary synthesis. *Philosophical Transactions of the Royal Society B*, 374(1770), 20180113. <https://doi.org/10.1098/rstb.2018.0113>

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- Darwin, C. (1859/1968). *The Origin of Species*. Penguin.
- Dawkins, R. (1976). *The Selfish Gene*. Oxford: Oxford University Press.
- Depew, D. (2003). Baldwin and his many Effects. In D. Depew, & B. Weber (Eds), *Evolution and Learning: Baldwin reconsidered*. MIT Press.
- Depew, D. (2011). Adaptation as a Process: The Future of Darwinism and the Legacy of Theodosius Dobzhansky. *Studies in the History of Biology and the Biomedical Sciences*, 42(89–98).
- Ferguson-Smith, A.C. (2011). Genomic imprinting: the emergence of an epigenetic paradigm. *Nature Review Genetics*, 12(8), 565–575. <https://doi.org/10.1038/nrg3032>
- Gapp, K. (2020). Unconventional forms of inheritance. *Seminars in Cell and Developmental Biology*, 97, 84–85. <https://doi.org/10.1016/j.semcdb.2019.08.003>
- Gibson, J.J. (1979). *The Ecological Approach to Visual Perception*. Boston: Houghton Mifflin.
- Gilbert, S. F., & Epel, D. (2015). *Ecological developmental biology: Integrating epigenetics, medicine, and evolution*. (2nd ed., p. 576). Sinauer Associates, Inc.
- Grieneisen, L., Dasari, M., Gould, T. J., Björk, J. R., Greiner, J.-C., Yotova, V., et al. (2021). Gut microbiome heritability is nearly universal but environmentally contingent. *Science*, 373(6551), 181–186. <https://doi.org/10.1126/science.aba5483>
- Heard, E., & Martienssen, R.A. (2014). Transgenerational Epigenetic Inheritance: myths and mechanisms. *Cell*, 157(1), 95–109. <https://doi.org/10.1016/j.cell.2014.02.045>
- Herman, J. J., Sultan, S., Horgan-Kybelski, T., & Riggs, C. (2017). Adaptive transgenerational plasticity in an annual plant: Grandparental and parental drought stress enhance performance of seedlings in dry soil. *Integrative and Comparative Biology*, 52(1), 77–88. <https://doi.org/10.1093/icb/ics041>
- Hu, T., Linz, D., Parker, E.S., & Moczek, A. (2020). Developmental bias in horned dung beetles and its contributions to innovation, adaptation, and resilience. *Evolution & Development*, 1-2(165–180).
- Jablonka, E. (2017). The evolutionary implications of epigenetic inheritance. *Interface Focus*.
- Jablonka, E., & Avital, E. (2006). Animal innovation: The origins and effects of new learned behaviours. *Biology & Philosophy*, 21(1), 135–141. <https://doi.org/10.1007/s10539-004-7106-3>
- Jablonka, E., & Lamb, M. (2010). Transgenerational Epigenetic Inheritance. In M. Pigliucci, & G. Müller (Eds), *Evolution: The Extended Synthesis*. (pp. 137–174). MIT Press.
- Jäger, J. (2021). The fourth perspective: Evolution and organismal agency. *OSF Preprints*. <https://doi.org/10.31219/osf.io/2g7fh>
- Kimura, M. (1973). *The neutral theory of evolution*. Cambridge: Cambridge University Press.
- Kirschner, M., & Gerhard, J.C. (2010). Facilitated Variation. In M. Pigliucci, & G.B. Muller (Eds), *The Extended Synthesis*. (pp. 253–280). MIT Press.
- Lacal, I., & Ventura, R. (2018). Epigenetic Inheritance: Concepts, Mechanisms and Perspectives. *Frontiers in Molecular Neuroscience*, 11, 292. <https://doi.org/10.3389/fnmol.2018.00292>
- Laland, K. N., Uller, T., Feldman, M., Sterelny, K., Müller, G. B., Moczek, A., et al. (2014). Does evolutionary theory need a rethink? (Yes, urgently.). *Nature*, 514(7521), 161–164. <https://doi.org/10.1038/514161a>
- Laland, K. N., Uller, T., Feldman, M., Sterelny, K., Müller, G. B., Moczek, A. P., et al. (2015). The extended evolutionary synthesis: Its structure, assumptions and predictions. *Proceedings of the Royal Society*, 282, 1813. <https://doi.org/10.1098/rspb.2015.1019>
- Ledón-Rettig, C. C., Pfennig, D. W., & Nascone-Yoder, N. (2008). Ancestral variation and the potential for genetic accommodation in larval amphibians: Implications for the evolution of novel feeding strategies. *Evolution & Development*, 10(3), 316–325. <https://doi.org/10.1111/j.1525-142X.2008.00240.x>
- Levis, N., & Pfennig, D. (2020). Plasticity-led evolution: A survey of developmental mechanisms and empirical tests. *Evolution & Development*, 22(1-2), 71–87. <https://doi.org/10.1111/ede.12309>
- Lewontin, R. C. (1974). *The genetic basis of evolutionary change*. Columbia University Press.
- Lewontin, R. C. (1985). The organism as subject and object of evolution. In R. Levins, & R. C. Lewontin (Eds), *The dialectical biologist*. (pp. 85–106). Harvard University Press.
- Linn, M., & Spagopoulou, F. (2018). Evolutionary consequences of epigenetic inheritance. *Hereditas*, 121(3), 205–209. <https://doi.org/10.1038/s41437-018-0113-y>
- Mayr, E. (1963). *Animal Species and Evolution*. Harvard University Press.
- Mayr, E. (1982). *The Growth of Biological Thought*. Harvard University Press.
- Messerly, J.G. (2009). Piaget's biology. In U. Y. Müller, J. I. M. Carpendale, & L. Smith (Eds.), *The Cambridge Companion to Piaget* (pp. 94–109). Cambridge: Cambridge University Press.
- Mitchell, T. S., Janzen, F. J., & Warner, D. A. (2018). Quantifying the effects of embryonic phenotypic plasticity on adult phenotypes in reptiles: A review of current knowledge and major gaps. *Journal of Experimental Zoology Part A*, 329(4-5), 203–214. <https://doi.org/10.1002/jez.2187>
- Moczek, A. (2023). When the end modifies its means: the origins of novelty and the evolution of innovation. *Biological Journal of the Linnean Society*, 139(4), 433–440. <https://doi.org/10.1093/biolinnean/blac061>
- Nadolski, E., & Moczek, A. (2023). Promises and limits of an agency perspective in evolutionary developmental biology. *Evolution & Development*.
- Newman, S. (2022). Inherency and agency in the origin and evolution of biological functions. *Biological Journal of the Linnean Society*.
- Newman, S. (2023). Form, Function, Agency: Sources of Natural Purpose in Animal Evolution. In P. Corning, P. Stuart Kauffman, D. Noble, J. Shapiro, & R. Vane-Wright (Eds), *Evolution 'on Purpose': Teleonomy in Living Systems*. MIT Press.
- Noble, D. (2015). Conrad Waddington and the origin of epigenetics. *Journal of Experimental Biology*, 218(6), 816–818. <https://doi.org/10.1242/jeb.120071>
- Nuño de la Rosa, & C. Villegas (2022). Chances and Propensities in Evo-Devo. *The British Journal for the Philosophy of Science*, 73(2), 533. <https://doi.org/10.1093/bjps/axz048>
- Odling Smee, F.J., Leland, K., & Feldman, M. (2003). *Niche construction: The neglected process in evolution*. Princeton, N.J.: Princeton University Press.
- Oyama, S., Griffiths, P. E., & Gray, R. D. (2000). *Cycles of Contingency*. Cambridge, M.A.: MIT Press.
- Peterson, E. (2016). *The Life Organic: The theoretical biology club and the roots of epigenetics*. Pittsburgh University Press.
- Pfennig, D. (2004). *Phenotypic plasticity and evolution*. CRC Press.
- Pfennig, D.W., Wund, M.A., Schlichting, C., Snell-Rood, E.C., Cruikshank, T., & Schlichting, C., et al (2010). Phenotypic Plasticity's Impacts on Diversification and Speciation. *Trends in Ecology and Evolution*, 25(8), 459–467. <https://doi.org/10.1016/j.tree.2010.05.006>
- Piaget, J. (1976). *Behaviour and Evolution*. Routledge and Kegan Paul. Trans. D. Nicholson-Smith.
- Pigliucci, M. (2009). An Extended Synthesis for Evolutionary Biology. *Annals of the New York Academy of Sciences*, 1168, 218–228. <https://doi.org/10.1111/j.1749-6632.2009.04578.x>
- Pigliucci, M., & Muller, G. (Eds), (2010). *Evolution: The Extended Synthesis*. MIT Press.
- Pollok, D., & Weber-Stadlbauer, U. (2020). Transgenerational consequences of maternal immune activation. *Seminars in Cell and Developmental Biology*, 97, 181–188. <https://doi.org/10.1016/j.semcdb.2019.06.006>
- Salazar-Ciudad, I. (2021). Why call it developmental bias when it is just development? *Biology Direct*, 16(1), 3. <https://doi.org/10.1186/s13062-020-00289-w>
- Sarkies, P. (2020). Molecular mechanisms of epigenetic inheritance: possible evolutionary implications. *Seminars in Cell and Developmental Biology*, 97, 106–115. <https://doi.org/10.1016/j.semcdb.2019.06.005>
- Simpson, G. G. (1953). The Baldwin effect. *Evolution*, 7(2), 110–117. <https://doi.org/10.1111/j.1558-5646.1953.tb00069.x>
- Sultan, S. E. (2015). *Organism and environment: Ecological development, niche construction and adaptation*. Oxford University Press.

- Sultan, S. E., Moczek, A., & Walsh, D. (2022). Bridging the explanatory gaps: what can we learn from a biological agency perspective?. *BioEssays*, 44(1), 2100185. <https://doi.org/10.1002/bies.202100185>
- Thompson, E. (2007). *Mind and World*. Oxford: Oxford University Press.
- Uller, T. (2023). Agency, goal orientation, and evolutionary explanations. In P. Corning, P. Stuart Kauffman, D. Noble, J. Shapiro, & R. Vane-Wright (Eds), *Evolution "on Purpose": Teleonomy in Living Systems*. MIT Press.
- Uller, T., & Helanterä, H. (2019). Niche construction and conceptual change in evolutionary biology. *The British Journal for the Philosophy of Science*, 70(2), 351–375. <https://doi.org/10.1093/bjps/axx050>
- Uller, T., Moczek, A., Watson, R. A., Brakefield, P., & Laland, K. (2018). Developmental Bias and Evolution: A Regulatory Network Perspective. *Genetics*, 209(4). <https://doi.org/10.1534/genetics.118.300995>
- Waddington, C. H. (1956). The Genetic Assimilation of the bithorax phenotype. *Evolution*, 10, 1-X.
- Waddington, C. H. (1953). Genetic Assimilation of an acquired character. *Evolution*, 7(2), 118–126.
- Waddington, C. H. (1957). *The Strategy of the Genes*. Routledge.
- Waddington, C. H. (1975). Genetic assimilation in *Limnaea*. In *The evolution of an evolutionist*. (pp. 92–95). Cornell University Press.
- Wagner, A. (2012). The Role of Robustness in Phenotypic Adaptation and Innovation. *Proceedings of the Royal Society B*, 279(1732), 1249–1258. <https://doi.org/10.1098/rspb.2011.2293>
- Walsh, D. M. (2014). The Negotiated Organism: Inheritance, Development and the Method of Difference. *Biological Journal of the Linnean Society*, 112(2), 295–30. <https://doi.org/10.1111/bij.12118>
- Walsh, D. M. (2015). *Organisms, agency, and evolution*. Cambridge University Press.
- Walsh, D.M. (2023). Evolutionary foundationalism and the myth of the chemical given. In P. Corning, P. Stuart Kauffman, D. Noble, J. Shapiro, & R. Vane-Wright (Eds), *Evolution "on Purpose": Teleonomy in Living Systems*. MIT Press.
- Walsh, D.M., & Rupik, G. (2023). The agency perspective: Counter-mapping the modern Synthesis. *Evolution & Development* (forthcoming).
- Walsh, D.M., Ariew, A., & Matthen, M. (2017). Four Pillars of Statisticalism. *Philosophy, Theory and Practice in Biology*, 1(1201), 1–17. <https://doi.org/10.3998/ptb.6959004.0009.001>
- Weiner, A. K. M., & Katz, L. A. (2021). Epigenetics as driver of adaptation and diversification in microbial eukaryotes. *Frontiers in Genetics*, 12, 642220. <https://doi.org/10.3389/fgene.2021.642220>
- West Eberhard, M. J. (2003). *Developmental plasticity and evolution*. Oxford University Press.
- Zhang, X. & Ho, S.-M. (2011). Epigenetics meets endocrinology. *Journal of Molecular Endocrinology*, 46(1), R11–R32. <https://doi.org/10.1677/jme-10-0053>