Conflation and refutation

A review of Uller, T. & Laland, K.N. (Eds.) (2019.) *Evolutionary Causation: Biological and Philosophical Reflections*. MIT Press. 352 pages. ISBN: 978-0-262-03992-5. $60.00/£50.00.

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1. Introduction

Evolutionary Causation, the new edited book from Tobias Uller and Kevin Laland (Uller and Laland, 2019) should be seen as a positive contribution to those seeking an Extended Evolutionary Synthesis (EES). The ambition for an EES has emerged most vociferously over the past 15 years, but its antecedents stretch back to the key work in the 1970s of Gould, Lewontin and their colleagues. At root arguments for an EES are arguments about how the Modern Synthesis (MS) in evolutionary biology has been found wanting. Much of this discontent has been to do with theoreticians rethinking concepts of adaptation, inheritance and development (Jablonka and Lamb, 2006; Huneman and Walsh, 2017) as well as variation and macroevolution (Pigliucci and Muller, 2010). This book continues this work, but focuses on analysis of the central concept of causation within evolution.

2. What is the MS?

Elsewhere I have defined the MS as a framework for generating hypotheses ((Dickins and Dickins, 2018) and see Otsula, chapter 12). One way to characterize this framework is through its history. A standard view is that the MS emerged from the 1930s through the 1960s as a synthesis between the Darwinian theory of evolution and the Mendelian theory of inheritance, mediated by the advent of population genetics (Provine, 2001).

Whilst it increasingly clear that this gloss fails to capture the messy and to some extent unresolved details of the development of thought within evolutionary biology (Delisle, 2009, 2011) it captures an ideal philosophical structure. The MS is a response to something entailed by Darwinian evolution. Thus, Darwin's contribution was not only the concept of natural selection, but also a definition of what *evolution* is. Evolution is a kind of change that relies upon variation, competition and inheritance (Lewontin, 1970). Darwin produced this definition whilst trying to account for the diversity of life. In this formulation, natural selection is the (statistical) outcome of these components operating over time. Time is present as inheritance implies generational turnover, competition is interaction, and initial variation must precede novelty: these are all events.

It is important to understand that *evolution* is the label we attach to the outcome of the interaction of these components; evolution is not an essence in want of a definition (Popper, 1945). Any system with these components can evolve as it enables a form of selection. That means any such system can change in terms of the proportional representation of its individual members, just so long as there is variation, competition and inheritance (Webb, 2011). Since Darwin, the MS has recognized sources other than natural selection for evolution, understood as a population concept. Thus the principal contribution of Darwin might be seen as a focus upon population thinking.

Selection does not speak to the sources of variation or competition, and nor does it address the causes of inheritance. But any explanation of evolution, within a system, would reasonably seek accounts of these matters. Given that evolution can happen in *any* system that exhibits those properties multiple causes of variation, competition and inheritance will exist in the world. But Darwin's own focus was upon biological systems, and in particular in populations defined at the species level; hence *natural* selection. Darwin was unable to deliver a total theory of the causes of the components of evolution in biological systems but the effort to do so continued as the discipline of evolutionary biology thrived.

Uller and Laland set the agenda accordingly:

> (E)volutionary biology, a discipline charged with providing historical accounts of the properties of living beings, as well as an understanding of the processes that explain the origin of those properties. Familiar phenomena that demand an evolutionary explanation include the fit of form and function (adaptation) and the evolution of reproductive isolation (speciation), but also many others, including the origin of novelty and the organization of biological systems, including genomes and ecosystems. (p.1)

They characterize the discipline of evolutionary biology as a loosely organized endeavour with the process of evolution by natural selection as the common thread. But the biological reality that delivers variation, differential survival and inheritance, and therefore enables natural selection, is incredibly complex. Their central claim is that evolutionary processes 'encompass causation at different levels of biological organization, from genes to ecosystems, and at different timescales" (p.1).

Uller and Laland clearly see the discipline of evolutionary biology as one that seeks the causes of evolution's components. For ease of exposition one might readily state that evolutionary biology aims to understand the causes of evolution. But, in this form we elide the causes of the components with the definition of what evolution is. Variation, competition and inheritance must come into alignment for evolution to occur, but each can exist prior to that alignment. To reiterate, Darwinian *evolution* is not a cause, but rather an outcome. The dynamics of such specific change are in want of fulsome explanation and that will become an historical account, where abstract historical transitions can be modelled. Uller and Laland are clear on this and argue that these complexities lead to modelling solutions that must leave out much causal detail in order to capture a biological system. They caution that the decision about what to leave out is most important.

What is left out, or has been removed, has been a core concern for those advancing the EES. In part this is due to a reliance upon the historical analysis of the MS provided by Gould (Gould, 2002). In his version, the discipline of evolutionary biology increasingly narrowed its view from the 1930s as Larmarckism, orthogenesis, saltationism and group selection were removed from the core theory of evolutionary biology. This led to a focus upon natural selection and adaptation.

> Evolutionary biology today has to incorporate soft inheritance, saltational changes due to systemic mutations, and various types of genetic exchange and cooperation. These all challenge the assumptions of the Modern Synthesis. We believe that rather than trying to continue to work within the framework of a Synthesis that was made in the middle of the last century, we now need a new type of evolutionary theory, one that acknowledges Darwinian, Lamarckian and saltational processes. ((Jablonka and Lamb, 2008): 394)

Whilst there is a clear irony in exhorting scholars to abandon an outmoded mid 20th century framework in favour of an 18th century one, the thrust of this new

book, and many other EES writings, is that recent findings in biology give us reason to rethink the causal role of acquired characteristics, agency, and development.

3. Details

My claim is that evolutionary biology is an on-going project that looks to account for the emergence of the relevant antecedent conditions for evolution to occur. All of the contributors to this book would agree with this. Furthermore, the MS is a framework that facilitates this effort. It is the adequacy of this framework that is in question.

Evolutionary Causation clearly adheres to a Gouldian perspective, and focuses upon the introduction of population thinking, gene-centrism and the distinction between proximate and ultimate causation as the major causal contributions of the MS. These introductions are thought to have excluded, or removed, developmental and individual-level considerations in the MS.

In Chapter 11, Walsh claims that the debate between MS and EES scholars rests upon the removal of the individual from evolutionary biology by population thinking. His argument is straightforward. Darwin's insight was that individuals' struggle for life led to changes in lineages due to arrivals and departures within a population. For Walsh this is a true cause of evolution.

> (T)here is one level of causation; all the causes of evolution are causes of arrival and departure (the 'struggle for life'). Yet, there are two discernible levels of effect. There are effects on individual organisms (first order effects), and there are effects on the distribution of abstract trait types in a population (higher order effects). Consequently, there are two wholly distinct kinds of evolutionary explanation. (p.238).

First order explanations lack generality as they merely give detailed accounts of a current situation. Higher order explanations resolve this as they capture available generality as an effect of a certain kind. In evolution:

> The first order cause explanation describes the ways in which biological, ecological processes impinged on the lives, deaths, and reproductions of individual organisms, and how the aggregate of these in turn was realized (in) changes in trait structure... This... has its limitations. It does not tell us, for example, how sensitive the change in trait structure is to variations in individual causes of living and dying. For that we need to offer the sort of higher order effect explanation we find in Modern Synthesis population thinking. (p.234)

This last is achieved by seeing the population as an ensemble of abstract traits - Fisher's solution - these traits then get fitness parameters to measure relative growth rates enabling us to explain relative frequencies. Walsh sees these as self contained, complete explanations that are complementary and non-competing.

Up to this point Walsh is in accord with the view of evolutionary biology as a discipline concerned with the antecedent conditions for evolution. However, he moves on to discuss Mayr's rendition of the proximate-ultimate distinction (PUD) (Mayr, 1961). The outcome of which appears to be that "'proximate causes,' those individual-level processes that cause organisms severally to have the traits they have, do not appear in evolutionary explanations." (p.239). Walsh then endorses an earlier statement of Laland's (Laland *et al.*, 2011) that proximate mechanisms both shape and respond to selection.

This argument is a central piece of the EES position. Uller and Laland (Chapter 1) relate PUD to Mayr's strong commitment to population thinking rather than what they call *transformational* or developmental *explanations* in evolutionary biology

(Sober, 1984). Transformational accounts are aggregation accounts of a population, looking at individual differences one at a time to account for population structure; this is contrasted with *variational* explanations which rely upon capacities that get you into a population, i.e. selection based accounts. As Sober points out:

> Lamarck's theory was developmental. It explained the evolution of species by laying down a sequence of stages through which life forms are constrained to pass. Species evolve because the organisms in them are gradually modified. In contrast, Darwin's theory of the evolution of species is not developmental. Darwin explained change in a species by a mechanism that permits (and.. even requires) stasis in organisms. In addition, the Darwinian paradigm views evolution as opportunistic, not preprogrammed. Selectional theories and developmental theories have fundamentally different explanatory structures. (pp. 148-149)

The standard reading of Mayr is that he saw developmental processes as proximate. Mayr (1961), in an example drawn from migration, precisely says that the genetic disposition of a bird is an ultimate cause, a disposition introduced by natural selection. But this is not to say that the operation of the genes, and their role in growth and development, is anything other than proximate. Thus Mayr can be seen as arguing that the population of birds (a migrating species) is made up of individuals with disposition to migrate due to selection. Evolution has occurred as a change from an ancestral state of only a few birds with this disposition to a majority share. Nonetheless, the causes of individual variation on this disposition are not without interest, they are just logically separable (Dickins and Barton, 2012). Given this, Sober's dichotomy strikes me more as emphasis than insight. These are two parts of evolutionary biology, but not two parts of *evolution*.

Uller and Laland would disagree with this last statement. Their claim is that PUD has been used to carve a consensus about what constitutes satisfactory evolutionary explanations (historical and functional accounts) and which do not (mechanistic and developmental accounts). Their strong feeling is that PUD makes "all of the sustained directionality in evolution (come) from fitness differences between genotypes, or natural selection" (p.3). For Uller and Laland PUD may be inadvertently ruling out legitimate alternative evolutionary explanations including the role of developmental processes. These processes, they claim, have been seen as irrelevant for explanations of phenotypic evolution. But, if we were Mayr and supportive colleagues, we would say phenotypes do not evolve, populations do.

How are we to make sense of this? Uller and Laland use an example of social learning in killer whales enabling fit to local feeding ecologies. Behavioural plasticity and social learning enable what they term *adaptive directionality*, challenging the view of natural selection as exclusive in this domain: in effect, behaviour is directed to a solution to a locally contingent problem, and this affects what selection has to operate over. This idea is close to orthogenesis, but the direction of evolution is affected by the action of individuals as in niche construction (Chapter 7). This does not imply hidden, or intrinsic design in evolution. In this example, different local contingencies might lead to different solutions. Uller and Laland locate accounts like this within *transformational* explanations and claim that a *variational* account would not invoke environmental influence on how traits originate and are inherited. This example nicely demonstrates the laser focus that the EES has upon the phenotype. Their question is not "is this population, so characterized, a product of the kind of change labelled *evolution*?" Rather, it is "what are the causes of individual trait variation?" Because they know that trait variation can play into evolution, in the presence of competition and inheritance, there is an awareness of possible,

downstream evolutionary effects. But, the EES is apt to conflate these two kinds of process.

Not only is the killer whale an example of conflation it is also an example of a missing level in the parsing of explanatory tasks. The example relies on social learning to introduce trait variation enabling adaptation to a local ecology. What is not asked, and what would occur to those pursuing a *variational* account, is for an account of the selection for social learning. Any such account would undoubtedly include discussion of relevant ecological variability and the role of data led tracking solutions for robustness. Thus the authors refer to phenomena that can be accommodated within standard selection models. Uller and Laland address this criticism, and suggest this view is a reformulation of what requires an evolutionary explanation and they suggest three problems with it:

1) It appears to deliver an incomplete explanation, as it does not drill into specific historical contingencies for specific populations of killer whales.

2) Strict exclusion of proximate causes "appears to confer on genes causal and informational privilege in development" (p.5) and misses that genes have their effects through development.

3) The reformulation relies on the idea that variation, differential fitness and heredity are autonomous processes.

The accusation of conflation may seem churlish in light of this riposte. Surely the manner in which PUD is used in the MS is merely an epistemic strategy? Perhaps an overly strong commitment to that strategy could cause scientists to miss ontological detail and at least impoverish their understanding of the natural world. After all, the mess of nature is a dynamic swirl of development and population changes emerging from a multitude of interactions up and down the biological hierarchy. Uller and Laland try to capture this idea by advocating an entangled model that they do not really explain (p.6). That PUD is an epistemic strategy is not a criticism, but it should be noted that it is drawn from an ontological point about what *evolution* is. Development simply does not fit that category, but, of course, both belong to the set labelled *change*.

The reference to privileging of the gene is to another aspect of the MS, its purported gene-centrism. Dayan and colleagues (Chapter 5) declare that understanding "the origin of biological variation is one of the principal goals of biology" (p.81). The MS was directed at this task but not only focused upon the gene as the "sole means of inheritance but also as the primary determinant of phenotypes themselves" (p.81). This has led to evolution being studied in terms of genetic variation but according to the authors we should really be interested in the evolution of traits (note, again, this use of evolution as a concept of emergence).

Dayan et al. claim that the MS linked genes and traits in a one-to-one, and linear fashion, and as deterministic genotype-to-phenotype mapping. This is contrasted with phenotypic plasticity, a live topic in recent years and they "suggest that we are on the cusp of integrating these diverse factors into a framework that predicts how environmentally induced variation interacts with genetic variation to influence traits and evolutionary trajectories" (p.81).

For Dayan et al., the idea of a direct relationship between genotype and phenotype, or a genetic blueprint, is central to the MS. This is often presented as a simplifying heuristic - the genotype-to-phenotype map – which they see as naively giving causal priority to natural selection among genetic variants in

research. Thus the gene is privileged. Yet, somewhat confusingly, they then discuss a few key population and quantitative genetic studies that make mapping assumptions and claim this as a useful heuristic, but one that does not capture the complexity of nature. This seems to be an odd criticism as heuristics are specifically designed to avoid complexity and to deliver a utility efficiently. GWAS work revealing underwhelming heritability estimates is focused upon along with a number of interpretations of missing heritability - but many of these possible explanations are in fact genetic (p.83) as well as epistatic and epigenetic, leading to the suggestion that the genotype-to-phenotype map may be too complex or dynamic to be of much use. This may be a just comment about some models, but it is only a comment about models, not about reality. Of course, what this leads to is the need to think about the role of the environment and gene by environment interactions. "The consequences of GxE are twofold. First, by providing the variation for plasticity on which selection can act, GxE permits the evolution of plasticity itself ... Perhaps more importantly, the existence of GxE means that genetic variation is expressed differently across environmental contexts." (p.86).

So, just what does "gene centric" or "gene centrism" mean? All of the commentary from Dayan et al. makes clear that there are things interacting with genes, augmenting gene action etc. but all of those processes only make sense in light of the gene. Genes are fundamental constraints, in that they are essential components, even in these non-linear models. What is relevant is some sort of genetic activity that arises from the base sequence, and can be modified via mutation – which in fact make mutations more fundamental for a variational account. Moreover, the idea that genes are a part of a developmental process has never been denied within the MS, and Dayan et al. conspicuously fail to derive a citation to counter this. Indeed, perhaps the most notable gene-centrist, Dawkins, directly advocates the role of genes as catalysts in his seminal book ((Dawkins, 1989) p.240). In my view, the use of the term privilege is always a red flag - it might be better read as explanatory priority, which is an epistemic constraint rather than an ontological commitment. But again this is an epistemological strategy born of an ontological commitment. What this means is that there is genetic primacy in some causal developmental chain, but this does not make genes a sufficient condition for the final phenotype, only necessary. Metaphorically, we might see the genotype as the geological layers beneath a detailed map with layers for terrain, and supported ecosystems etc. There is causality here, and there is the opportunity for abstract models to make predictive jumps across layers.

All this to one side, the focus for Dayan et al. has again been on the emergence of the phenotype, not upon evolution. Their interest in plasticity is not necessarily a challenge to the MS. To this end, the authors discuss the standard robustness interpretation of plasticity as a data led response to environmental change that enables the preservation of the genome (Meyers and Bull, 2002), but they point out that phenotypic plasticity might mediate the relationship between selection and genetic variation. But surely there is no contradiction here? The idea that plasticity might buffer the genome against change is a mediation between selection and the genome that would allow directional selection assuming plasticity mechanisms were susceptible to mutation. In other words, buffering does not have to be total and it would be odd if it were. Rather buffering introduces a useful time lag, but not a total exclusion for more systemic change. Dayan et al. understand this and also the role of this in changing population dynamics and the outcomes of evolution.

This leads Dayan et al. to genetic accommodation where phenotypes are leaders and genes are followers (West-Eberhard, 2003). This is described in standard MS

terms – so the choices are that either a mutation leads to a new phenotype, or a novel phenotype is delivered by previously unexpressed plastic response, and then selection refines all this by operating on various genetic loci. The speed of evolution can be rapid as plasticity can enable an alternative phenotype to emerge in multiple individuals in the population and that gives more options for selection to operate over. This is all packaged as the environment playing a role in both the production of and selection on a trait. But this is not in fact so because plasticity is dependent, in a causal way, upon underlying genes that in turn can deliver alternative and (in this instance) novel phenotypes. That plasticity is conditionally expressed under some probability distribution (Nettle and Bateson, 2015). When those conditions are met, the alternative phenotype is expressed, but it is not solely caused by those conditions, but rather it awaits those conditions. None of this is in contradiction to neutral evolution theories that allow for genetic robustness but also the chance of phenotypically delivered utility in future, changed environments, which is often packaged as evolvability (Wagner, 2005, 2008; Pigliucci, 2008). Evolvability simply means that there is a possibility of later selection and evolution.

A key thing to take home here is that if you choose not to think about a possible role for selection or to address any patterning in phenotypic plasticity, then phenotypic response to the environment is irregular and non-law like. There is no real data available to support this hypothesis, but one could choose to believe that this is because it has not yet been collected. You could even see this as a limit of our current scientific methods. At this stage, such ideas are close to skyhooks (Dennett, 1995). Skyhooks are concepts, usually hypothesized mechanisms that do not build on lower levels of organization, but rather impose complexity from on high. As such they are profoundly anti-reductionist but also leave us with miraculous questions to resolve about their origins.

In Chapter 6, Sultan aims to re-evaluate the idea of genetic control as a causal principle. She tells us that developmental outcomes are shaped by multiple types of information and states that "phenotypes emerge from the real-time regulatory interactions of the evolved genotype with transient environmental and/or epigenetic influences that occur at timescales from within a generation to several of many generations" (p.117). This reveals two things. First, the genotype has a data role that can be regulated by other inputs. This suggests that the genotype provides an information context for environmental and other inputs. Programs operate to deliver this kind of functionality, and yet Sultan is eager to move away from the view of reaction norms as a part of developmental programs. Second, no effort is made to think about why the timescale variation is in place within these mechanisms. She sees all of this delivering distributed control of the emerging phenotype.

Sultan sees this rich developmental context as decoupling phenotypic outcomes from specific allelic differences and altering selection (p.119). I think what is meant is that the allelic variation is not directly visible in all circumstances because it is expressed differently under different conditions etc. This will impact upon selection dynamics for the genotype but it is hardly a revelation and it is hardly a challenge to the genetic program idea. In drawing to a conclusion Sultan comments:

> Now the key challenge is to understand how the stable, continuous DNA information stream that most robustly tracks evolutionary lineage and diversification is altered by selective evolution, when that DNA stream comprises only a *partial, context-dependent component* of selective causation - that is, when fitness variation reflects genotype-environment entanglement rather than genetic differences alone. (p. 119)

This sounds revolutionary but it is not. All Sultan has noted really is that the expression of the phenotype involves multiple causes, which all evolutionary biologists have always thought, and that this adds noise to *GxE* speculations. The gene is still a necessary and context-setting condition for phenotypic development. Development is patterned as a result of this, but also because natural selection would not lead to truly random developmental outcomes, and *evolution* is as *evolution* has been since Darwin defined it.

4. Conclusion

Uller and Laland begin *Evolutionary Causation* with a caution about models. Models can be understood as connecting theory to phenomena (De Regt, 2017). As such models are derived from theoretical principles but are abstract representations exploiting similarities with the real world (Giere, 2004). Models bring phenomena into theory and they are a mechanism of explanation. When this is done, according to both De Regt and Giere, we have gained scientific understanding of the phenomenon at hand. In many ways, Uller and Laland are asserting that natural selection is a principal theoretical contribution of evolutionary biology, which can be used to generate models at various different levels of biological organization. But, this contribution can be understood differently within distinct theoretical frameworks as a consequence of the kind of causation invoked. By implication, the argument is that the MS was a partial modelling effort that excluded various aspects of the biological totality. The further implication is that the concepts of causation developed within the MS modelling effort were also partial, and perhaps wrong.

The MS is indeed a framework to deliver models. Its core conceit is the definition of evolution given by Darwin, and it has relied upon natural selection to generate hypotheses. But the MS has not been unaware of other ways to produce patterned population change, and drift is always to be considered for example. What natural selection does is enable the construction of falsifiable hypotheses about particular biological systems. As such, the MS might be seen as a viable research program, following Lakatos. As a viable research program, new discoveries about the origin of variation, or competition or inheritance are to be dealt with – do they challenge the hard-core axioms of the program, or can they be accommodated, even with tweaks to the protective belt of day-to-day empirical work? In order to challenge new predictions must be made.

I think the empirical work referenced by the EES, and in *Evolutionary Causation*, presents useful, protective belt challenges to the research program of the MS. But these challenges are really around quibbles with regard to specific models. Thus the EES are, in effect, simply arguing for lists of different constraints and considerations in the abstractions made in order to claim greater or more comprehensive coverage. That quickly becomes a moot point because the ambitions of the modellers will vary. Nonetheless the MS should welcome a fully worked theory of the emergence of the phenotype, of variation and of inheritance and so all of this work needs to be considered. What is not presented is a real challenge to the core axioms of the MS. One possible reason for this is that, to date, the EES has relied on partial histories of the transitions in evolutionary biology, and claims made about only a handful of contributors. This amounts to an inductive attempt to characterize a framework from a small sample of studies. History and philosophy of science has been wise to this problem for some time (De Regt, 2017). This approach is unlikely to make full contact with the discipline as practiced, nor the many available interpretations of the literature. Indeed, one might wonder whether the disciplinary backgrounds and task demands of the contributing authors determines where they choose to focus in the entangled mess of nature.

Whilst I clearly take a critical view of the EES I do think the arguments they present have a role to play. It is good practice to stop, every now and then, and to inspect one's own core assumptions; and, it is certainly good practice to look to new findings in biology and be certain sure they make sense in light of extant frameworks because at some point they may not. The contributors to this book will make any reader do this. But ultimately the EES seems focused upon a variety of complex feedback effects in evolution that are predominantly the realm of ecology. Those effects are important and require parsing and explaining. The EES provides no new tools for doing this, nor any novel predictions, and the discussion of causation in this book merely conflates the phenomena with explanation. My suspicion is that true and productive challenges to evolutionary biology will arise from efforts directed toward the origins of life itself, and constraints upon this afforded by physics.

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