Is non-genetic inheritance just a proximate mechanism? A corroboration of the Extended Evolutionary Synthesis

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Abstract

What role does non-genetic inheritance play in evolution? In recent work we have independently and collectively argued that the existence and scope of non-genetic inheritance systems, including epigenetic inheritance, niche construction/ecological inheritance, and cultural inheritance - alongside certain other theory revisions - necessitates an extension to the neo-Darwinian Modern Synthesis (MS) in the form of an Extended Evolutionary Synthesis (EES). However, this argument has been challenged on the grounds that non-genetic inheritance systems are exclusively proximate mechanisms that serve the ultimate function of calibrating organisms to stochastic environments. In this paper we defend our claims, pointing out that critics of the EES (i) conflate non-genetic inheritance with early 20th century notions of soft inheritance; (ii) misunderstand the nature of the EES in relation to the MS; (iii) confuse individual phenotypic plasticity with trans-generational non-genetic inheritance; (iv) fail to address the extensive theoretical and empirical literature which shows that non-genetic inheritance can generate novel targets for selection, create new genetic equilibria that would not exist in the absence of non-genetic inheritance, and generate phenotypic variation that is independent of genetic variation; (v) artificially limit ultimate explanations for traits to gene-based selection, which is unsatisfactory for phenotypic traits that originate and spread via non-genetic inheritance systems; and (vi) fail to provide an explanation for biological organisation. We conclude by noting ways in which we feel that an overly gene-centric theory of evolution is hindering progress in biology and other sciences.

Keywords: biological organisation, cultural evolution, epigenetic inheritance, Extended Evolutionary Synthesis, Modern Synthesis, niche construction, non-genetic inheritance

1. Introduction

What role does non-genetic inheritance play in evolution? By 'non-genetic inheritance' we mean the transmission of information across multiple generations of individuals through a mechanism other than DNA replication, such as cultural inheritance via social learning (e.g. imitation or language), epigenetic inheritance via epigenetic marks (e.g. methylation patterns of genes), or ecological/niche inheritance via the environment. In previous work (Danchin et al. 2011; Jablonka and Lamb 2005; Odling Smee et al. 2003; Pigliucci and Müller 2010), we have argued that the existence and scope of non-genetic inheritance across a range of taxa - together with findings in evo-devo and other disciplines - requires a radical revision of the Modern Synthesis (Huxley 1942) (henceforth MS; also known as Neo-Darwinism), in which evolution is defined as changes in gene frequencies resulting from genetic drift, mutation, gene flow and natural selection of genes. We have called instead for an Extended Evolutionary Synthesis (Pigliucci and Müller 2010) (henceforth EES), in which phenotypic change and adaptation can result from both genetic and non-genetic inheritance (Danchin et al. 2011; Danchin and Wagner 2010; see also Bonduriansky and Day 2009; Mameli 2004).

Our calls have not gone unchallenged. We focus here on perhaps the most explicit critique of our work by T. Dickins and colleagues (Dickins and Barton in press; Dickins and Dickins 2007; Dickins and Dickins 2008; Dickins and Rahman 2012; Scott-Phillips et al. 2011), although similar criticisms have been made by others (Dawkins 2004; Haig 2007). Dickins and colleagues' argument, which they apply equally to humans (Scott-Phillips et al. 2011) and non-human species (Dickins and Rahman 2012), is that non-

genetic inheritance systems are exclusively proximate mechanisms that evolved for the ultimate function of calibrating organisms to environmental stochasticity. They maintain that ultimate 'why' questions – questions concerning why particular traits are favoured, and the existence of adaptations that exhibit apparent design – can only be answered at the level of natural selection acting on genetic variation. Consequently, they argue that the existence of non-genetic inheritance "poses no challenge to the explanatory and conceptual resources of the MS, which are sufficient" (Dickins and Rahman 2012, p.2913). Furthermore, by allegedly confusing proximate and ultimate causes in this way, we are charged with "hinder[ing] scientific progress" (Scott-Phillips et al. 2011, p.39) by perpetuating confusion and causing wasted effort.

In this commentary we seek to clarify and defend our position. For ease of exposition we focus our response on the most recent and most explicit critique of the EES published by Dickins and Rahman (2012, henceforth D&R). We first clarify the status of the EES in relation to the MS. We then show that D&R fail to address the fundamental point that transgenerational non-genetic inheritance can significantly transform evolutionary dynamics by generating novel targets for selection, affecting the rate and manner of information transmission across generations, and creating new genetic equilibria that would not exist in the absence of non-genetic inheritance. We then argue that D&R's use of the 'ultimate-proximate' distinction is unhelpful and unproductive in this debate, and that the EES is necessary to fully understand biological organisation. We conclude by defending our work against the charge that it is hindering scientific progress.

2. Scope and status of the EES

What exactly are we claiming when we argue that the MS needs to be extended? D&R, like other critics, relate the EES to the early 20th century notion of 'soft inheritance'. This term, which they attribute to Mayr, is defined by D&R as "the inheritance of variations that are the result of non-genetic effects" (D&R, p.2913). This is incorrect. In fact, Mayr defined soft inheritance as "the belief in a gradual change of the *genetic material itself*, either by use or disuse, or by some internal progressive tendencies, or through the direct effect of the environment" (Mayr and Provine 1980, p.15). Soft inheritance, as originally defined by Mayr, therefore involves direct changes to DNA sequences. In contrast, the non-genetic inheritance systems that we argue are evolutionarily important, such as epigenetic inheritance or cultural transmission, do *not* involve direct changes in DNA sequences. There is no suggestion, for example, that culturally transmitted religious beliefs change DNA sequences. Epigenetic inheritance (like cultural transmission) is *defined* as change that occurs independently of changes in the DNA sequence. The issue of directed changes to DNA is a separate and fascinating issue (Shapiro 2011), but is logically distinct to non-genetic inheritance. To reiterate, the contemporary debate over the role of non-genetic inheritance in evolution is *not* the same as the rejection of soft inheritance prior to the MS (Bonduriansky 2012), and it is unhelpful to conflate the two.

Another source of confusion is over the status of the EES in relation to the MS. D&R distinguish between 'general' evolutionary theory, which "captures the basic Darwinian dynamics of variation, inheritance, competition and selection" (D&R, p.2915) but is mechanism-neutral with respect to how these dynamics operate, and 'special' theories

131 such as the MS, which specifies mechanisms by which, for example, variation arises
132 (undirected genetic mutation and recombination) and inheritance occurs (Mendelian
133 genetic inheritance)[1]. D&R argue that the EES is a general theory and hence cannot
134 challenge the MS. This is again mistaken: the EES is intended as a special theory that
135 extends and replaces the MS. We have argued (Danchin et al. 2011; Jablonka and Lamb
136 2005; Odling Smee et al. 2003; Pigliucci and Müller 2010) that the specialized
137 assumptions of the MS, such as, natural selection, recombination and undirected genetic
138 mutation, are not sufficient to explain the adaptive dynamics of evolution, and must be
139 expanded to include a suite of additional developmental, epigenetic, behavioural and
140 cultural processes. To argue that the EES fails to challenge the MS because it is "not the
141 same order of account as that of the MS" (D&R, p.2915) is incorrect.
142
143 **3. Is non-genetic inheritance just a proximate calibration mechanism?**
144 D&R's central argument is that non-genetic inheritance functions to calibrate organisms
145 to environmental stochasticity, thus remaining under ultimate genetic control. In support
146 of this notion of 'genetic control' they cite human twin studies purporting to show the
147 heritability of epigenetic marks, and discuss two examples in rats, one in which maternal
148 licking of pups alters those pups' subsequent parental behaviour and stress responses via
149 epigenetic changes in offspring neural circuits (Champagne 2008), and another involving

[1] We note that while D&R attribute the distinction between general and specific evolutionary theories to Webb (2011), it is much older. Lewontin (1970), for example, clearly spelled out the general aspects of Darwinian evolution (variation, inheritance and differential fitness), and explained how genetic evolution is but one specific theory that fulfills these criteria. It is curious that Webb (2011) cites no references in his paper, neither Lewontin (1970) nor any of the large subsequent literature that has built on Lewontin's distinction.

150 learning biases such that rats are more likely to associate nausea with tastes rather than
151 other sensory stimuli (the 'Garcia effect': Garcia et al. 1955).
152
153 There are several problems with this argument. First, D&R repeatedly conflate non-
154 inherited individual phenotypic plasticity with transgenerational phenotypic plasticity that
155 is transmitted to subsequent generations via non-genetic inheritance, and thus fail to
156 address our arguments for the importance of the latter in driving evolutionary dynamics.
157 Phenotypic plasticity occurs when phenotypes vary in response to environmental
158 variability in the absence of corresponding DNA variation, and such direct proximate
159 responses may entail epigenetic or individual learning mechanisms. Non-genetic
160 inheritance, in contrast, occurs when variable information that is unrelated to DNA
161 sequence variation is transmitted *across successive generations of individuals*, such as
162 occurs with epigenetic inheritance and cultural transmission / social learning. D&R fail to
163 recognise this distinction. Taste aversion in rats, for example, concerns individual
164 phenotypic plasticity, with individual rats' food preferences shifting, within genetically
165 specified limits, in response to foods experienced within their lifetimes. There is no trans-
166 generational inheritance in this example as D&R present it, therefore it has no bearing on
167 the EES debate. Furthermore, D&R appear to then conflate cultural transmission and
168 individual learning in general ("even cultural learning processes are situated within
169 individuals", D&R, p.2918), seemingly subsuming all cultural learning/transmission into
170 individual learning. This entirely misses the point, and is empirically untenable:
171 individual learning alone cannot lead to transgenerational cultural inheritance, and there
172 is extensive evidence that cultural transmission can drive behavioural distributions away

from individual preferences (Boyd and Richerson 1985; Galef and Laland 2005; Mery et al. 2009), including taste preferences in rats (Laland and Plotkin 1990). Cultural transmission is observed across a diverse range of species (Galef and Laland 2005), and in humans allows the accumulation of vast amounts of information over successive generations independently of genetic variation (Richerson and Boyd 2005).

A similar point can be made for epigenetic inheritance. Contrary to D&R's claim that "the potential for epigenetic transgenerational inheritance appears limited" (p.2916), there is abundant and accruing evidence for chromatin- or RNA-mediated cellular inheritance of epigenetic variations over multiple generations, independent of DNA variation (Jablonka 2012; Jablonka and Raz 2009). The most extensive studies have been conducted in plants (Schmitz et al. 2011), and similar transgenerational effects have been documented in nematodes, yeast, insects and recently mammals (Jablonka 2012; Jablonka and Raz 2009). Compared to these breeding experiments, the human twin studies cited by D&R are only an indirect means of assessing the degree to which epigenetic variation matches genetic variation, yet even they demonstrate that, to quote what D&R themselves describe as the largest study to date, "epigenetic profiles are not fully determined by DNA sequence" (Kaminsky et al. 2009, p.242). While it is trivially true that the mechanisms underlying epigenetic inheritance and cultural transmission must be genetically influenced (just as it is trivially true that the mechanisms of the MS, such as DNA replication or recombination via meiosis, are genetically influenced), this often diffuse influence is a long way from the complete genetic control portrayed by D&R.

Turning back to D&R's argument, the notion that individual phenotypic plasticity (e.g. individual learning or epigenetic variation) functions to calibrate organisms to stochastic environments is interesting, but not new. Campbell (1960) and Lorenz (1969) discussed learning in these terms decades ago, and since then numerous formal models have explored how phenotypic plasticity can evolve in response to varying rates and forms of environmental stochasticity, for both epigenetic (Lachmann and Jablonka 1996) and learning processes (Boyd and Richerson 1985; Aoki et al. 2005). These models indicate that epigenetic or learning-based phenotypic plasticity can readily evolve when environments change too rapidly for genetic evolution to track directly, that is, when environments change within an individual's lifetime (what Lorenz (1969) called 'generational deadtime') or slightly longer, such that natural selection acting over multiple generations cannot adequately respond.

Yet D&R do not appreciate the main implication of this notion of calibration for their argument about genetic control. If the function of phenotypic plasticity is to track environmental change that cannot be anticipated by genes, then there simply *must* be a partial decoupling between genes and phenotypic plasticity, otherwise the latter would never have evolved. This applies even more to transgenerational non-genetic inheritance. Once information can be inherited non-genetically, it can significantly transform evolutionary dynamics through reciprocal feedback between the different inheritance systems. This goes far beyond mere proximate 'calibration'. Gene-culture coevolution is the best-understood example, having been subject to formal theoretical modelling for nearly 40 years (since Cavalli-Sforza and Feldman 1973). D&R are incorrect to say that

these studies "model cultural change as if it were directly tied to genetic variation" (D&R, p.2917); phenotypes are modelled as the product of both genetic and cultural inheritance, which are assumed to be at least partially independent (yet interacting). These models show that cultural inheritance can modify selection contexts and drive genetic evolution to new stable equilibria that would not have existed in the absence of cultural inheritance (Laland et al. 2010; Boyd and Richerson 1985). Evidence from molecular genetics and archaeology supports these predictions in several cases, such as the spread of lactose tolerance alleles in populations that possess culturally transmitted dairy farming practices or the spread of sickle cell alleles in response to increased malaria from culturally transmitted yam cultivation (Laland et al. 2010). Gene-culture coevolution is not just restricted to agriculture-related change. Laland, Kumm and Feldman (1995) showed that culturally transmitted practices such as female-biased infanticide and female-biased abortion can significantly and permanently alter the genetically-specified primary sex ratio, while Mesoudi and Laland (2007) showed that culturally transmitted beliefs in partible paternity (that children can have more than one 'biological' father, as is commonly believed in many traditional South American societies) can drive human mating systems to different equilibria compared to the purely genetic evolution of human mating behaviour. Recent models suggest similar coevolutionary dynamics between genetic and epigenetic inheritance (Day and Bonduriansky 2011), and models that have incorporated epigenetic inheritance into classical population genetic models show that the dynamics of populations are profoundly influenced by heritable epigenetic variations (Geoghegan and Spencer 2011). The process of niche construction (Odling Smee et al. 2003), whereby organisms modify their

selective environments, adds further complexity by transforming selection acting on descendant populations. The "stochastic environment" discussed by D&R is not a fixed, external entity to which genetic evolution adapts populations, it itself constitutes an inheritance system (ecological inheritance) that can generate novel, consistent and directional selection on genes. D&R completely ignore this extensive theoretical and empirical literature on the interaction between multiple inheritance systems.

4. Beyond "genetic=ultimate, non-genetic=proximate"

At the heart of the disagreement, we think, is D&R's dogmatic insistence that ultimate "why" questions can only be answered in terms of the natural selection of genes, with everything ontogenetic treated as solely a proximately causal process (see also Scott-Phillips et al. 2011; Dickins and Barton in press). While this may have been a useful heuristic at the formation of the MS in the context of debates over soft inheritance (which, as noted above, is quite different to non-genetic inheritance), the weight of evidence for the causal role of non-genetic inheritance in evolution now invalidates the simple equating of 'ultimate causation=gene-based selection', and strongly implies reciprocal causation rather than the unidirectional causality assumed by D&R (Laland et al. 2011). The question "why do different human groups vary in their genetic propensity to drink milk", for example, seems impossible to answer without appealing to culturally transmitted farming practices. It is difficult to see the latter as merely "proximate" given that cultural evolution is driving changes in gene frequencies (Laland et al. 2010; Gerbault et al. 2011). Researchers cannot simply take the selection pressures on adult lactose absorption alleles as a given, pre-established and fixed feature of the environment,

as they are changing dynamically as the cultural practice and favoured genotype coevolve. Or to take another example, the question "why do people in England predominantly speak English, and people in France mostly speak French?" seems impossible to answer in terms of changes in gene frequencies, given that linguistic variation is independent of genetic variation (there are no genes for speaking French, for example). Instead, this question would have to be addressed in terms of the cultural evolution and diversification of the Indo-European language family over the last few thousand years through cultural equivalents of mutation (copying errors) and selection (see Pagel 2009), as well as sociolinguistic processes that have no obvious parallel in genetic evolution (see Labov 2001), and which can be addressed using similar phylogenetic methods to those used to reconstruct genetic evolutionary relationships (e.g. Gray and Atkinson 2003; Bouckaert et al. 2012). This type of question is not limited to humans, of course, and one could ask similar questions about why, say, one population of great tits know how to break the foil of milk bottle caps and another population does not (Lefebvre 1995), or why one population of chimpanzees uses tools to crack nuts and another does not (Whiten et al. 1999), both of which are best explained as cultural variation resulting from cultural transmission rather than genetic variation resulting from genetic inheritance[2]. One might label these cultural dynamics as all 'proximate', as Dickins and Barton (in press) do, but they surely concern ultimate 'why' questions: why particular phenotypic traits (e.g. drinking cows' milk, speaking English, nut-cracking)

[2] Natural selection can also act on cultural or epigenetic variation, such as when differential birth rates affect the spread of different religions (Hout et al. 2001) or epigenetic variants that promote tameness are selected during domestication, as suggested by artificial selection experiments in silver foxes (Jablonka and Raz 2009).

285 emerge and spread amongst different populations, and the appearance of complex design
286 features[3]. In the case of language, it is increasingly recognised that cultural transmission
287 can generate in language complex design features (the cultural equivalent of adaptation in
288 biological evolution; see Kirby, Cornish and Smith 2008), something that the proximate-
289 ultimate causation distinction hinders researchers from appreciating (Laland et al. 2011).
290 By abandoning an artificial 'ultimate=genetic' definition, such cultural dynamics can be
291 appropriately seen as drivers of phenotypic variation. The same applies to
292 developmentally-induced, epigenetically inherited variation and niche-constructed
293 environments.
294
295 **5. Biological organisation**
296 We find curious D&R's argument that "advocates of the EES consistently fail to
297 understand biological organization and its provenance" (p.2917). In our view, the MS
298 was founded on tenets that, while useful heuristics for advancing biological theory at that

[3] Dickins and Barton (in press) maintain that all such cultural dynamics (such as language change) should be seen as proximate rather than ultimate causes. They equate cultural evolution with 'historical accounts', which "are not in any sense default ultimate accounts", because (i) historical/cultural dynamics are governed by ultimate genetic causes at some higher level of organisation, and (ii) there is no adequately worked-out theory of cultural evolution that provides an equivalent level of explanatory power to genetic evolution. We disagree. Regarding point (ii), decades of empirical and theoretical work in cultural evolution has identified numerous learning biases that can explain specific behavioural patterns (Mesoudi 2011; Richerson and Boyd 2005), including frequency-dependent (e.g. conformist or anti-conformist) biases and model-based biases such as prestige or success bias. Regarding point (i), as we argued in section 3, the fact that cultural learning biases may have a genetic origin does not imply that the behaviour that results from cultural dynamics is under direct genetic control. Hence our claim that these cultural dynamics are often more appropriately seen as ultimate, rather than (or as well as) proximate, causes of behaviour.

299 time, are now known to be anachronistic. These tenets include the legitimacy of
300 neglecting developmental processes thereby allowing evolution to be studied through
301 population genetics alone, and a focus on a single level of ultimate causation. These
302 tenets fail to fully address biological organization, and the EES arose precisely in
303 response to this deficiency. All of the key components of the EES (evo-devo, epigenetics,
304 multilevel selection, niche construction, cultural evolution, etc.) address the issue of
305 interaction between levels of organization as well as the origin and fixation of specific
306 forms of organization at each of these levels, from the genetic to the cultural (see, for
307 example, Müller (2007) for evo-devo, Odling-Smee et al. (2003) for niche construction,
308 or Boyd & Richerson (1985) for gene-culture coevolution). Such work emphasises key
309 concepts such as modularity (Müller 2007) or nested hierarchies of inheritance systems
310 (Odling Smee et al. 2003) that are entirely absent in the MS. If the problem of
311 organization is phrased in the characteristic manner of the MS, i.e. reducing organization
312 to the coordinating function of genes, it is not surprising that critics such as D&R are
313 disappointed by the EES. But this bypasses the true features of complex phenotypic
314 organization in organismal evolution. We argue, in contrast, that it is the EES that
315 concentrates on the provenance of organization, by including the comprehensive
316 organizing properties of development, inheritance, behaviour, and culture.
317

318 **6. Progress in the evolutionary sciences**

319 We believe that an exclusive focus on gene-based selection as the sole ultimate cause of
320 evolutionary design is hindering progress in the evolutionary sciences. Incorporating non-
321 genetic inheritance into heritability studies can potentially solve the so-called ‘missing-

heritability' problem (Danchin et al. 2011; Furrow et al. 2011), and explain the spread of novel alleles and maladaptive behaviour (Laland et al. 2010). The human behavioural and social sciences, in particular, have been highly critical of gene-based approaches to the study of human behaviour such as sociobiology or, more recently, evolutionary psychology (Layton 2010; Kendal 2012). This is largely because the phenomena that social/behavioural scientists study - the cultural dynamics of languages, technology, religious beliefs, socio-political institutions and so on - are not under direct genetic control, and can only be explained as cultural adaptations that arise through cultural evolution (Mesoudi 2011; Boyd et al. 2011). Similarly, a gene-based approach may not be appropriate in medical research on supposedly genetic human neurological disorders such as epilepsy or autism, as the inclusive heritability of such disorders may incorporate significant non-genetic components (Ben-Ari and Spitzer 2010; Ben-Ari 2008; Furrow et al. 2011). An evolutionary theory that encompasses multiple interacting inheritance systems and the interactions between them is far more compatible with socio-cultural phenomena, in both humans and non-human species alike, than a gene-centric evolutionary theory (Danchin et al. 2004; Layton 2010; Kendal 2012). We encourage D&R, and evolutionary biologists in general, to abandon the dogma of the MS and adopt a more nuanced, multifaceted theory of evolution.

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