

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21

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

Published in *Biological Theory* 7(3) 189-195.

Mesoudi, A.*¹, Blanchet, S.^{2,3}, Charmantier, A.⁴, Danchin, E.^{3,5}, Fogarty, L.⁶, Jablonka,
E.⁷, Laland, K.N.⁸, Morgan T.J.H.⁸, Müller, G.B.⁹, Odling-Smee, F. J.¹⁰, Pujol, B.^{3,5}

Word count: 5107

*Corresponding author: a.a.mesoudi@durham.ac.uk

Authors' note: apart from the first / corresponding author, all other authors are ordered
alphabetically

21

Author affiliations:

22

¹Department of Anthropology, Durham University, Dawson Building, South Road,

23

Durham DH1 3LE, UK

24

²CNRS; Station d'Écologie Expérimentale du CNRS à Moulis, USR2936, Moulis, 09200

25

Saint-Girons, France.

26

³CNRS, UPS, ENFA; EDB (Laboratoire Evolution & Diversité Biologique); UMR5174;

27

118 route de Narbonne, F-31062 Toulouse, France.

28

⁴CNRS; Centre d'Écologie Fonctionnelle et Évolutive, UMR5175, Campus CNRS, 1919

29

Route de Mende, 34293 Montpellier cedex 5, France.

30

⁵Université Paul Sabatier; EDB; UMR5174; F-31062 Toulouse, France.

31

⁶Department of Biology, Stanford University, Stanford, CA 9430, USA.

32

⁷The Cohn Institute for the History and Philosophy of Science and Ideas, Tel-Aviv

33

University, Tel-Aviv 69978, Israel.

34

⁸School of Biology, University of St. Andrews, Bute Medical Building, Queen's Terrace,

35

St. Andrews, Fife, Scotland KY16 9TS, UK.

36

⁹Department of Theoretical Biology, University of Vienna, Althanstrasse 14, A-1090

37

Vienna, Austria.

38

¹⁰Mansfield College, University of Oxford, Oxford OX1 3TF, UK.

39

39

Abstract

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

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

62

62 **1. Introduction**

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

78

79 Our calls have not gone unchallenged. We focus here on perhaps the most explicit
80 critique of our work by T. Dickins and colleagues (Dickins and Barton in press; Dickins
81 and Dickins 2007; Dickins and Dickins 2008; Dickins and Rahman 2012; Scott-Phillips
82 et al. 2011), although similar criticisms have been made by others (Dawkins 2004; Haig
83 2007). Dickins and colleagues’ argument, which they apply equally to humans (Scott-
84 Phillips et al. 2011) and non-human species (Dickins and Rahman 2012), is that non-

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

95

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

107

108 **2. Scope and status of the EES**

109 What exactly are we claiming when we argue that the MS needs to be extended? D&R,
110 like other critics, relate the EES to the early 20th century notion of ‘soft inheritance’. This
111 term, which they attribute to Mayr, is defined by D&R as “the inheritance of variations
112 that are the result of non-genetic effects” (D&R, p.2913). This is incorrect. In fact, Mayr
113 defined soft inheritance as “the belief in a gradual change of the *genetic material itself*,
114 either by use or disuse, or by some internal progressive tendencies, or through the direct
115 effect of the environment” (Mayr and Provine 1980, p.15). Soft inheritance, as originally
116 defined by Mayr, therefore involves direct changes to DNA sequences. In contrast, the
117 non-genetic inheritance systems that we argue are evolutionarily important, such as
118 epigenetic inheritance or cultural transmission, do *not* involve direct changes in DNA
119 sequences. There is no suggestion, for example, that culturally transmitted religious
120 beliefs change DNA sequences. Epigenetic inheritance (like cultural transmission) is
121 *defined* as change that occurs independently of changes in the DNA sequence. The issue
122 of directed changes to DNA is a separate and fascinating issue (Shapiro 2011), but is
123 logically distinct to non-genetic inheritance. To reiterate, the contemporary debate over
124 the role of non-genetic inheritance in evolution is *not* the same as the rejection of soft
125 inheritance prior to the MS (Bonduriansky 2012), and it is unhelpful to conflate the two.
126

127 Another source of confusion is over the status of the EES in relation to the MS. D&R
128 distinguish between ‘general’ evolutionary theory, which “captures the basic Darwinian
129 dynamics of variation, inheritance, competition and selection” (D&R, p.2915) but is
130 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

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

178

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

195

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

208

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

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

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

248

249 **4. Beyond “genetic=ultimate, non-genetic=proximate”**

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

265 as they are changing dynamically as the cultural practice and favoured genotype
266 coevolve. Or to take another example, the question “why do people in England
267 predominantly speak English, and people in France mostly speak French?” seems
268 impossible to answer in terms of changes in gene frequencies, given that linguistic
269 variation is independent of genetic variation (there are no genes for speaking French, for
270 example). Instead, this question would have to be addressed in terms of the cultural
271 evolution and diversification of the Indo-European language family over the last few
272 thousand years through cultural equivalents of mutation (copying errors) and selection
273 (see Pagel 2009), as well as sociolinguistic processes that have no obvious parallel in
274 genetic evolution (see Labov 2001), and which can be addressed using similar
275 phylogenetic methods to those used to reconstruct genetic evolutionary relationships (e.g.
276 Gray and Atkinson 2003; Bouckaert et al. 2012). This type of question is not limited to
277 humans, of course, and one could ask similar questions about why, say, one population of
278 great tits know how to break the foil of milk bottle caps and another population does not
279 (Lefebvre 1995), or why one population of chimpanzees uses tools to crack nuts and
280 another does not (Whiten et al. 1999), both of which are best explained as cultural
281 variation resulting from cultural transmission rather than genetic variation resulting from
282 genetic inheritance². One might label these cultural dynamics as all ‘proximate’, as
283 Dickins and Barton (in press) do, but they surely concern ultimate ‘why’ questions: why
284 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 features³. 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-

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

340

341 **REFERENCES**

342 Aoki K, Wakano JY, Feldman MW (2005) The emergence of social learning in a
343 temporally changing environment: A theoretical model. *Curr Anthropol* 46:334-340

- 344 Ben-Ari Y (2008) Neuro-archaeology: Pre-symptomatic architecture and signature of
345 neurological disorders. *Trends Neurosci* 31:626-636
- 346 Ben-Ari Y, Spitzer NC (2010) Phenotypic checkpoints regulate neuronal development.
347 *Trends Neurosci* 33:485-492
- 348 Bonduriansky R (2012) Rethinking heredity, again. *Trends Ecol Evol* 27:330-336
- 349 Bonduriansky R, Day T (2009) Nongenetic inheritance and its evolutionary implications.
350 *Ann Rev Ecol Evol Syst* 40:103-125
- 351 Bouckaert R, Lemey P, Dunn M, Greenhill SJ, Alekseyenko AV, Drummond AJ, Gray
352 RD, Suchard MA, Atkinson QD (2012) Mapping the origins and expansion of the
353 Indo-European language family. *Science* 337:957-960
- 354 Boyd R, Richerson PJ (1985) *Culture and the evolutionary process*. University of
355 Chicago Press, Chicago, IL
- 356 Boyd R, Richerson PJ, Henrich J (2011) The cultural niche: Why social learning is
357 essential for human adaptation. *PNAS* 108:10918-10925
- 358 Campbell DT (1960) Blind variation and selective retentions in creative thought as in
359 other knowledge processes. *Psychol Rev* 67:380-400
- 360 Cavalli-Sforza LL, Feldman MW (1973) Cultural versus biological inheritance:
361 phenotypic transmission from parents to children. *Am J Hum Genet* 25:618-637

- 362 Champagne FA (2008) Epigenetic mechanisms and the transgenerational effects of
363 maternal care. *Front Neuroendocrin* 29:386-397
- 364 Danchin E, Charmantier A, Champagne FA, Mesoudi A, Pujol B, Blanchet S (2011)
365 Beyond DNA: integrating inclusive inheritance into an extended theory of evolution.
366 *Nature Rev Genet* 12:475-486
- 367 Danchin E, Giraldeau LA, Valone TJ, Wagner RH (2004) Public information: From nosy
368 neighbors to cultural evolution. *Science* 305:487-491
- 369 Danchin D, Wagner RH (2010) Inclusive heritability: Combining genetic and non-genetic
370 information to study animal behavior and culture. *Oikos* 119:210-218
- 371 Dawkins R (2004) Extended phenotype—but not too extended. A reply to Laland, Turner
372 and Jablonka. *Biol Phil* 19:377-396
- 373 Day T, Bonduriansky R (2011) A unified approach to the evolutionary consequences of
374 genetic and nongenetic inheritance. *Am Nat* 178:E18-E36
- 375 Dickins TE, Barton RA (in press) Reciprocal causation and the proximate-ultimate
376 distinction. *Biol Phil*.
- 377 Dickins TE, Dickins BJA (2007) Designed calibration: Naturally selected flexibility, not
378 non-genetic inheritance. *Behav Brain Sci* 30:368-369
- 379 Dickins TE, Dickins BJA (2008) Mother Nature's tolerant ways: why non-genetic
380 inheritance has nothing to do with evolution. *New Ideas Psychol* 26:41-54

- 381 Dickins TE, Rahman Q (2012) The extended evolutionary synthesis and the role of soft
382 inheritance in evolution. *Proc R Soc B* 279:2913-2921
- 383 Furrow RE, Christiansen FB, Feldman MW (2011) Environment-sensitive epigenetics
384 and the heritability of complex diseases. *Genetics* 189:1377-1387
- 385 Galef BG, Laland KN (2005) Social learning in animals: Empirical studies and
386 theoretical models. *BioScience* 55:489-499
- 387 Garcia J, Kimeldorf DJ, Koelling RA (1955) Conditioned aversion to saccharin resulting
388 from exposure to gamma radiation. *Science* 122:157-158
- 389 Geoghegan JL, Spencer HG (2012) Population-epigenetic models of selection. *Theor*
390 *Popul Biol* 81:232-242
- 391 Gerbault P, Liebert A, Itan Y, Powell A, Currat M, Burger J, Swallow DM, Thomas MG
392 (2011) Evolution of lactase persistence: an example of human niche construction. *Phil*
393 *Trans R Soc B* 366:863-877
- 394 Gray RD, Atkinson QD (2003) Language-tree divergence times support the Anatolian
395 theory of Indo-European origin. *Nature* 426:435-439
- 396 Haig D (2007) Weismann rules! OK? Epigenetics and the Lamarckian temptation. *Biol*
397 *Phil* 22:415-428
- 398 Hout M, Greeley A, Wilde MJ (2001) The demographic imperative in religious change in
399 the United States. *Am J Sociol* 107:468-500

- 400 Huxley JS (1942) *Evolution, the modern synthesis*. Allen & Unwin, London
- 401 Jablonka E (2012) *Epigenetic inheritance and plasticity: The responsive germline*. *Prog*
402 *Biophys Mol Biol*
- 403 Jablonka E, Lamb MJ (2005) *Evolution in four dimensions*. MIT Press, Cambridge, MA
- 404 Jablonka E, Raz G (2009) Transgenerational epigenetic inheritance: prevalence,
405 mechanisms, and implications for the study of heredity and evolution. *Q Rev Biol*
406 84:131-176
- 407 Kaminsky ZA, Tang T, Wang SC, Ptak C, Oh GHT, Wong AHC, Feldcamp LA, Virtanen
408 C, Halfvarson J, Tysk C (2009) DNA methylation profiles in monozygotic and
409 dizygotic twins. *Nature Genet* 41:240-245
- 410 Kendal JR (2012) *Cultural niche construction and human learning environments:*
411 *investigating sociocultural perspectives*. *Biol Theor* 6:241-250
- 412 Kirby S, Cornish H, Smith K (2008) *Cumulative cultural evolution in the laboratory: An*
413 *experimental approach to the origins of structure in human language*. *PNAS*
414 105:10681-10686
- 415 Labov W (2001) *Principles of linguistic change (II): Social factors*. Blackwell, Malden,
416 MA
- 417 Lachmann M, Jablonka E (1996) *The inheritance of phenotypes: an adaptation to*
418 *fluctuating environments*. *J Theor Biol* 181:1-9

- 419 Laland KN, Kumm J, Feldman MW (1995) Gene-culture coevolutionary theory. *Curr*
420 *Anthropol* 36:131-156
- 421 Laland KN, Odling-Smee J, Myles S (2010) How culture shaped the human genome:
422 bringing genetics and the human sciences together. *Nature Rev Genet* 11:137-148
- 423 Laland KN, Plotkin HC (1990) Social learning and social transmission of foraging
424 information in Norway rats (*Rattus norvegicus*). *Anim Learn Behav* 18:246-251
- 425 Laland KN, Sterelny K, Odling-Smee J, Hoppitt W, Uller T (2011) Cause and effect in
426 biology revisited: Is Mayr's proximate-ultimate dichotomy still useful? *Science*
427 334:1512-1516
- 428 Layton R (2010) Why social scientists don't like Darwin and what can be done about it. *J*
429 *Evol Psychol* 8:139-152
- 430 Lefebvre L (1995) The opening of milk bottles by birds: Evidence for accelerating
431 learning rates, but against the wave-of-advance model of cultural transmission. *Behav*
432 *Process* 34:43-53
- 433 Lewontin RC (1970) The units of selection. *Annu Rev Ecol Syst* 1:1-18.
- 434 Lorenz K (1969) Innate bases of learning. In: Pribram K (ed) *On the biology of learning*.
435 Harcourt, New York, pp 13-91
- 436 Mameli M (2004) Nongenetic selection and nongenetic inheritance. *Brit J Phil Sci* 55:35-
437 71

- 438 Mayr E, Provine W (eds) (1980) *The evolutionary synthesis*. Harvard University Press,
439 Cambridge, MA
- 440 Mery F, Varela SAM, Danchin E, Blanchet S, Parejo D, Coolen I, Wagner RH (2009)
441 Public versus personal information for mate copying in an invertebrate. *Current*
442 *Biology* 19:730-734
- 443 Mesoudi A (2011) *Cultural evolution: How Darwinian theory can explain human culture*
444 *and synthesize the social sciences*. University of Chicago Press, Chicago, IL
- 445 Mesoudi A, Laland KN (2007) Culturally transmitted paternity beliefs and the evolution
446 of human mating behaviour. *Proc R Soc B* 274:1273-1278
- 447 Müller GB (2007) Evo-devo: extending the evolutionary synthesis. *Nature Rev Genet*
448 8:943-949
- 449 Odling Smee FJ, Laland KN, Feldman M (2003) *Niche construction*. Princeton
450 University Press, Princeton, NJ
- 451 Pagel M (2009) Human language as a culturally transmitted replicator. *Nature Rev Genet*
452 10:405-415
- 453 Pigliucci M, Müller GB (2010) *Evolution: the extended synthesis*. MIT Press,
454 Cambridge, MA
- 455 Richerson PJ, Boyd R (2005) *Not by genes alone*. University of Chicago Press, Chicago

- 456 Schmitz RJ, Schultz MD, Lewsey MG, O'Malley RC, Urich MA, Libiger O, Schork NJ,
457 Ecker JR (2011) Transgenerational epigenetic instability is a source of novel
458 methylation variants. *Science* 334:369-373
- 459 Scott-Phillips TC, Dickins TE, West SA (2011) Evolutionary theory and the ultimate–
460 proximate distinction in the human behavioral sciences. *Perspect Psychol Sci* 6:38-47
- 461 Shapiro JA (2011) *Evolution: a view from the 21st century*. FTPress Science.
- 462 Whiten A, Goodall J, McGrew WC, Nishida T, Reynolds V, Sugiyama Y, Tutin CEG,
463 Wrangham RW, Boesch C (1999) Cultures in chimpanzees. *Nature* 399:682-685