Biol Theory
DOI 10.1007/s13752-013-0091-5

LONG ARTICLE

Is Non-genetic Inheritance Just a Proximate Mechanism? A Corroboration of the Extended Evolutionary Synthesis

Alex Mesoudi · Simon Blanchet · Anne Charmantier · Étienne Danchin · Laurel Fogarty · Eva Jablonka · Kevin N. Laland · Thomas J. H. Morgan · Gerd B. Müller · F. John Odling-Smee · Benoît Pujol

Received: 10 December 2012 / Accepted: 29 January 2013

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 (1) conflate non-genetic inheritance with early 20th-century notions of soft inheritance; (2) misunderstand the nature of the EES in relation to the MS; (3) confuse individual phenotypic plasticity with trans-generational non-genetic inheritance; (4) 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; (5) 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 (6) fail to provide an explanation for biological organization. We

A. Mesoudi (✉)
Department of Anthropology, Durham University, Durham, UK
e-mail: a.a.mesoudi@durham.ac.uk

S. Blanchet
CNRS; Station d'Écologie Expérimentale du CNRS à Moulis, 09200 Moulis, France
e-mail: simon.blanchet@ecoex-moulis.cnrs.fr

S. Blanchet · É. Danchin · B. Pujol
EDB (Laboratoire Évolution & Diversité Biologique), CNRS, UPS, ENFA, UMR 5174, 31062 Toulouse, France
e-mail: etienne.danchin@univ-tlse3.fr

B. Pujol
e-mail: benoit.pujol@univ-tlse3.fr

A. Charmantier
CNRS; Centre d'Écologie Fonctionnelle et Évolutive, UMR 5175, Montpellier, France
e-mail: anne.charmantier@cefe.cnrs.fr

L. Fogarty
Department of Biology, Stanford University, Stanford, CA, USA
e-mail: lfogarty@stanford.edu

E. Jablonka
The Cohn Institute for the History and Philosophy of Science and Ideas, Tel-Aviv University, Tel-Aviv, Israel
e-mail: jablonka@post.tau.ac.il

K. N. Laland · T. J. H. Morgan
School of Biology, University of St. Andrews, St. Andrews, Fife, Scotland, UK
e-mail: knl1@st-andrews.ac.uk

T. J. H. Morgan
e-mail: tjhm3@st-andrews.ac.uk

G. B. Müller
Department of Theoretical Biology, University of Vienna, Vienna, Austria
e-mail: gerhard.mueller@univie.ac.at

F. J. Odling-Smee
Mansfield College, University of Oxford, Oxford, UK
e-mail: john.odling-smee@mansfield.ox.ac.uk

Published online: 13 February 2013

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 organization · Cultural evolution · Epigenetic inheritance · Extended Evolutionary Synthesis · Modern Synthesis · Niche construction · Non-genetic inheritance

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 EvoDevo 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 2012 in press; Dickins and Dickins 2007, 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 favored, 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 organization. We conclude by defending our work against the charge that it is hindering scientific progress.

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. 2,913). 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. 2,915) but is mechanism-neutral with respect to how these dynamics operate, and "special" theories such as the MS, which specifies mechanisms by which, for example, variation arises (undirected genetic mutation and recombination) and inheritance occurs (Mendelian genetic inheritance).[1] D&R argue that the EES is a general theory and hence cannot challenge the MS. This is again mistaken: the EES is intended as a special theory that extends and replaces the MS. We have argued (Danchin et al. 2011; Jablonka and Lamb 2005; Odling Smee et al. 2003; Pigliucci and Müller 2010) that the specialized assumptions of the MS, such as natural selection, recombination, and undirected genetic mutation, are not sufficient to explain the adaptive dynamics of evolution, and must be expanded to include a suite of additional developmental, epigenetic, behavioral, and cultural processes. To argue that the EES fails to challenge the MS because it is "not the same order of account as that of the MS" (D&R, p. 2,915) is incorrect.

Is Non-genetic Inheritance Just a Proximate Calibration Mechanism?

D&R's central argument is that non-genetic inheritance functions to calibrate organisms to environmental stochasticity, thus remaining under ultimate genetic control. In support of this notion of "genetic control" they cite human twin studies purporting to show the heritability of epigenetic marks, and discuss two examples in rats, one in which maternal licking of pups alters those pups' subsequent parental behavior and stress responses via epigenetic changes in offspring neural circuits (Champagne 2008), and another involving learning biases such that rats are more likely to associate nausea with tastes rather than other sensory stimuli (the "Garcia effect"; Garcia et al. 1955).

There are several problems with this argument. First, D&R repeatedly conflate non-inherited individual phenotypic plasticity with transgenerational phenotypic plasticity that is transmitted to subsequent generations via non-genetic inheritance, and thus fail to address our arguments for the importance of the latter in driving evolutionary dynamics. Phenotypic plasticity occurs when phenotypes vary in response to environmental variability in the absence of corresponding DNA variation, and such direct proximate responses may entail epigenetic or individual learning mechanisms. Non-genetic inheritance, in contrast, occurs when variable information that is unrelated to DNA sequence variation is transmitted *across successive generations of individuals*, such as occurs with epigenetic inheritance and cultural transmission/social learning. D&R fail to recognize this distinction. Taste aversion in rats, for example, concerns individual phenotypic plasticity, with individual rats' food preferences shifting, within genetically specified limits, in response to foods experienced within their lifetimes. There is no trans-generational inheritance in this example as D&R present it, therefore it has no bearing on the EES debate. Furthermore, D&R appear to then conflate cultural transmission and individual learning in general ("even cultural learning processes are situated within individuals"; D&R, p. 2,918), seemingly subsuming all cultural learning/transmission into individual learning. This entirely misses the point, and is empirically untenable: individual learning alone cannot lead to trans-generational cultural inheritance, and there is extensive evidence that cultural transmission can drive behavioral 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. 2,916), 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

[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.

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 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 modeling 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. 2,917); phenotypes are modeled 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 et al. (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 behavior. 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 2012). 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.

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 2012 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 favored 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 knows 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 2012 (in press) do, but they surely concern ultimate "why" questions: why particular phenotypic traits (e.g., drinking cows' milk, speaking English, nut-cracking) emerge and spread amongst different populations, and the appearance of complex design features.[3] In the case of language, it is increasingly recognized that cultural transmission can generate in language complex design features (the cultural equivalent of adaptation in biological evolution; see Kirby et al. 2008), something that the proximate-ultimate causation distinction hinders researchers from appreciating (Laland et al. 2011). By abandoning an artificial "ultimate = genetic" definition, such cultural dynamics can be appropriately seen as drivers of phenotypic variation. The same applies to developmentally induced, epigenetically inherited variation and niche-constructed environments.

Biological Organization

We find curious D&R's argument that "advocates of the EES consistently fail to understand biological organization and its provenance" (p. 2,917). In our view, the MS was founded on tenets that, while useful heuristics for advancing biological theory at that time, are now known to be anachronistic. These tenets include the legitimacy of neglecting developmental processes thereby allowing evolution to be studied through population genetics alone, and a focus on a single level of ultimate causation. These tenets fail to fully address biological organization, and the EES arose precisely in response to this deficiency. All of the key components of the EES (EvoDevo, epigenetics, multilevel selection, niche construction, cultural evolution, etc.) address the issue of interaction between levels of organization as well as the origin and fixation of specific forms of organization at each of these levels, from the genetic to the cultural (see, e.g., Müller (2007) for Evo-Devo, Odling Smee et al. (2003) for niche construction, or Boyd and Richerson (1985) for gene-culture coevolution). Such work emphasizes key concepts such as modularity (Müller 2007) or nested hierarchies of inheritance systems (Odling Smee et al. 2003) that are entirely absent in the MS. If the problem of organization is phrased in the characteristic manner of the MS, i.e., reducing organization to the coordinating function of genes, it is not surprising that critics such as D&R are disappointed by the EES. But this bypasses the true features of complex phenotypic organization in organismal evolution. We argue, in contrast, that it is the EES that concentrates on the provenance of organization, by including the comprehensive organizing properties of development, inheritance, behavior, and culture.

[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).

[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 (1) historical/cultural dynamics are governed by ultimate genetic causes at some higher level of organization, and (2) 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 (2), decades of empirical and theoretical work in cultural evolution has identified numerous learning biases that can explain specific behavioral 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 (1), as we argued above, the fact that cultural learning biases may have a genetic origin does not imply that the behavior 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 behavior.

Progress in the Evolutionary Sciences

We believe that an exclusive focus on gene-based selection as the sole ultimate cause of evolutionary design is hindering progress in the evolutionary sciences. Incorporating non-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 behavior (Laland et al. 2010). The human behavioral and social sciences, in particular, have been highly critical of gene-based approaches to the study of human behavior such as sociobiology or, more recently, evolutionary psychology (Layton 2010; Kendal 2012). This is largely because the phenomena that social/behavioral 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 (Boyd et al. 2011; Mesoudi 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 2008; Ben-Ari and Spitzer 2010; Furrow et al. 2011). An evolutionary theory that encompasses multiple interacting inheritance systems and the interactions between them is far more compatible with sociocultural phenomena, in both humans and non-human species alike, than a gene-centric evolutionary theory (Danchin et al. 2004; Kendal 2012; Layton 2010). 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.

References

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

Ben-Ari Y (2008) Neuro-archaeology: pre-symptomatic architecture and signature of neurological disorders. Trends Neurosci 31: 626–636

Ben-Ari Y, Spitzer NC (2010) Phenotypic checkpoints regulate neuronal development. Trends Neurosci 33:485–492

Bonduriansky R (2012) Rethinking heredity, again. Trends Ecol Evol 27:330–336

Bonduriansky R, Day T (2009) Non-genetic inheritance and its evolutionary implications. Ann Rev Ecol Evol Syst 40:103–125

Bouckaert R, Lemey P, Dunn M, Greenhill SJ, Alekseyenko AV, Drummond AJ, Gray RD, Suchard MA, Atkinson QD (2012) Mapping the origins and expansion of the Indo-European language family. Science 337:957–960

Boyd R, Richerson PJ (1985) Culture and the evolutionary process. University of Chicago Press, Chicago

Boyd R, Richerson PJ, Henrich J (2011) The cultural niche: why social learning is essential for human adaptation. PNAS 108:10918–10925

Campbell DT (1960) Blind variation and selective retentions in creative thought as in other knowledge processes. Psychol Rev 67:380–400

Cavalli-Sforza LL, Feldman MW (1973) Cultural versus biological inheritance: phenotypic transmission from parents to children. Am J Hum Genet 25:618–637

Champagne FA (2008) Epigenetic mechanisms and the transgenerational effects of maternal care. Front Neuroendocr 29:386–397

Danchin D, Wagner RH (2010) Inclusive heritability: combining genetic and non-genetic information to study animal behavior and culture. Oikos 119:210–218

Danchin E, Giraldeau LA, Valone TJ, Wagner RH (2004) Public information: from nosy neighbors to cultural evolution. Science 305:487–491

Danchin E, Charmantier A, Champagne FA, Mesoudi A, Pujol B, Blanchet S (2011) Beyond DNA: integrating inclusive inheritance into an extended theory of evolution. Nat Rev Genet 12:475–486

Dawkins R (2004) Extended phenotype–but not too extended. A reply to Laland, Turner and Jablonka. Biol Phil 19:377–396

Day T, Bonduriansky R (2011) A unified approach to the evolutionary consequences of genetic and non-genetic inheritance. Am Nat 178:E18–E36

Dickins TE, Barton RA (2012) Reciprocal causation and the proximate–ultimate distinction. Biol Phil (in press)

Dickins TE, Dickins BJA (2007) Designed calibration: naturally selected flexibility, not non-genetic inheritance. Behav Brain Sci 30:368–369

Dickins TE, Dickins BJA (2008) Mother Nature's tolerant ways: why non-genetic inheritance has nothing to do with evolution. New Ideas Psychol 26:41–54

Dickins TE, Rahman Q (2012) The extended evolutionary synthesis and the role of soft inheritance in evolution. Proc R Soc B 279:2913–2921

Furrow RE, Christiansen FB, Feldman MW (2011) Environment-sensitive epigenetics and the heritability of complex diseases. Genetics 189:1377–1387

Galef BG, Laland KN (2005) Social learning in animals: empirical studies and theoretical models. Bioscience 55:489–499

Garcia J, Kimeldorf DJ, Koelling RA (1955) Conditioned aversion to saccharin resulting from exposure to gamma radiation. Science 122:157–158

Geoghegan JL, Spencer HG (2012) Population-epigenetic models of selection. Theor Popul Biol 81:232–242

Gerbault P, Liebert A, Itan Y, Powell A, Currat M, Burger J, Swallow DM, Thomas MG (2011) Evolution of lactase persistence: an example of human niche construction. Phil Trans R Soc B 366:863–877

Gray RD, Atkinson QD (2003) Language-tree divergence times support the Anatolian theory of Indo-European origin. Nature 426:435–439

Haig D (2007) Weismann rules! OK? epigenetics and the lamarckian temptation. Biol Phil 22:415–428

Hout M, Greeley A, Wilde MJ (2001) The demographic imperative in religious change in the United States. Am J Sociol 107:468–500

Huxley JS (1942) Evolution, the modern synthesis. Allen & Unwin, London

Jablonka E (2012) Epigenetic inheritance and plasticity: the responsive germline. Prog Biophys Mol Biol. http://dx.doi.org/10.1016/j.pbiomolbio.2012.08.014

Jablonka E, Lamb MJ (2005) Evolution in four dimensions. MIT Press, Cambridge

Jablonka E, Raz G (2009) Transgenerational epigenetic inheritance: prevalence, mechanisms, and implications for the study of heredity and evolution. Q Rev Biol 84:131–176

Kaminsky ZA, Tang T, Wang SC, Ptak C, Oh GHT, Wong AHC, Feldcamp LA, Virtanen C, Halfvarson J, Tysk C (2009) DNA methylation profiles in monozygotic and dizygotic twins. Nat Genet 41:240–245

Kendal JR (2012) Cultural niche construction and human learning environments: investigating sociocultural perspectives. Biol Theor 6:241–250

Kirby S, Cornish H, Smith K (2008) Cumulative cultural evolution in the laboratory: an experimental approach to the origins of structure in human language. PNAS 105:10681–10686

Labov W (2001) Principles of linguistic change (II): social factors. Blackwell, Malden

Lachmann M, Jablonka E (1996) The inheritance of phenotypes: an adaptation to fluctuating environments. J Theor Biol 181:1–9

Laland KN, Plotkin HC (1990) Social learning and social transmission of foraging information in Norway rats (*Rattus norvegicus*). Anim Learn Behav 18:246–251

Laland KN, Kumm J, Feldman MW (1995) Gene-culture coevolutionary theory. Curr Anthropol 36:131–156

Laland KN, Odling-Smee J, Myles S (2010) How culture shaped the human genome: bringing genetics and the human sciences together. Nat Rev Genet 11:137–148

Laland KN, Sterelny K, Odling-Smee J, Hoppitt W, Uller T (2011) Cause and effect in biology revisited: is Mayr's proximate–ultimate dichotomy still useful? Science 334:1512–1516

Layton R (2010) Why social scientists don't like Darwin and what can be done about it. J Evol Psychol 8:139–152

Lefebvre L (1995) The opening of milk bottles by birds: evidence for accelerating learning rates, but against the wave-of-advance model of cultural transmission. Behav Process 34:43–53

Lewontin RC (1970) The units of selection. Annu Rev Ecol Syst 1:1–18

Lorenz K (1969) Innate bases of learning. In: Pribram K (ed) On the biology of learning. Harcourt, New York, pp 13–91

Mameli M (2004) Nongenetic selection and nongenetic inheritance. Brit J Phil Sci 55:35–71

Mayr E, Provine W (eds) (1980) The evolutionary synthesis. Harvard University Press, Cambridge

Mery F, Varela SAM, Danchin E, Blanchet S, Parejo D, Coolen I, Wagner RH (2009) Public versus personal information for mate copying in an invertebrate. Curr Biol 19:730–734

Mesoudi A (2011) Cultural evolution: how Darwinian theory can explain human culture and synthesize the social sciences. University of Chicago Press, Chicago

Mesoudi A, Laland KN (2007) Culturally transmitted paternity beliefs and the evolution of human mating behaviour. Proc R Soc B 274:1273–1278

Müller GB (2007) Evo-devo: extending the evolutionary synthesis. Nat Rev Genet 8:943–949

Odling Smee FJ, Laland KN, Feldman M (2003) Niche construction. Princeton University Press, Princeton

Pagel M (2009) Human language as a culturally transmitted replicator. Nat Rev Genet 10:405–415

Pigliucci M, Müller GB (2010) Evolution: the extended synthesis. MIT Press, Cambridge

Richerson PJ, Boyd R (2005) Not by genes alone. University of Chicago Press, Chicago

Schmitz RJ, Schultz MD, Lewsey MG, O'Malley RC, Urich MA, Libiger O, Schork NJ, Ecker JR (2011) Transgenerational epigenetic instability is a source of novel methylation variants. Science 334:369–373

Scott-Phillips TC, Dickins TE, West SA (2011) Evolutionary theory and the ultimate–proximate distinction in the human behavioral sciences. Perspect Psychol Sci 6:38–47

Shapiro JA (2011) Evolution: a view from the 21st century. FT Press Science, Upper Saddle River, NJ

Whiten A, Goodall J, McGrew WC, Nishida T, Reynolds V, Sugiyama Y, Tutin CEG, Wrangham RW, Boesch C (1999) Cultures in chimpanzees. Nature 399:682–685