On reciprocal causation in the evolutionary process

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Abstract:

Recent calls for a revision the standard evolutionary theory (ST) are based on arguments about the reciprocal causation of evolutionary phenomena. Reciprocal causation means that cause-effect relationships are obscured, as a cause could later become an effect and *vice versa*. Such dynamic cause-effect relationships raises questions about the distinction between proximate and ultimate causes, as originally formulated by Ernst Mayr. They have also motivated some biologists and philosophers to argue for an Extended Evolutionary Synthesis (EES). Such an EES will supposedly replace the Modern Synthesis (MS), with its claimed focus on unidirectional causation. I critically examine this conjecture by the proponents of the EES, and conclude – on the contrary – that reciprocal causation has long been recognized as important in ST and in the MS tradition. Numerous empirical examples of reciprocal causation in the form of positive and negative feedbacks now exists from both natural and laboratory systems. Reciprocal causation has been explicitly incorporated in mathematical models of coevolutionary arms races, frequency-dependent selection and sexual selection. Such feedbacks were already recognized by Richard Levins and Richard Lewontin, long before the call for an EES and the associated concept of niche construction. Reciprocal causation and feedbacks is therefore one of the few contributions of dialectical thinking and Marxist philosophy in evolutionary theory, and should be recognized as such. While reciprocal causation have helped us to understand many evolutionary processes, I caution against its extension to heredity and directed development if such an extension involves futile attempts to restore Lamarckian or "soft inheritance".

Introduction

About a year ago – in January 2016 – the great population biologist Richard Levins passed away (Mehta 2016). Scientifically speaking, Levins was mainly known for his pioneering models about the evolution of genetic variation and adaptive plasticity in changing environments (Levins 1968). But Levins was also a political activist, life-long committed communist and Marxist (Maynard Smith 1988). Together with his political and scientific ally at Harvard University – population geneticist Richard Lewontin – Levins published a partly controversial book in 1985 entitled *The Dialectical Biologist* (Levins and Lewontin 1985). In this book, Levins and Lewontin advocated the use of the dialectical method – as developed by German socialists and philosophers Karl Marx and Friedrich Engels – and applied dialectical thinking to ecology and evolutionary biology (Levins and Lewontin 1985). Although the two Marxists Levins and Lewontin were highly critical of what they called "Cartesian reductionism" which they argued dominated Western science in general and evolutionary biology in particular, they did not deny the success of this traditional research approach. However, they suggested that the dialectical method should complement the traditional reductionist approach (Levins and Lewontin 1985). Given that this is actually a fairly modest claim, it is somewhat surprising that this book was initially met with such an extreme hostility from a large part of the community of evolutionary biologists.

One notable exception to this negative reception of their book was Levins and Lewontin's friend and colleague, the British evolutionary biologist John Maynard Smith, who published a review of the book in *London Review of Books* (Maynard Smith 1988). Maynard Smith was himself a former member of the Communist Party of Great Britain (CPGP) and had thus a background in Marxist philosophy. In his critical review, Maynard Smith did not hesitate to admit that Levins and Lewontin's dialectical method had some scientific utility (Maynard Smith 1988). He also

praised Levins work as one of the best examples of how dialectical thinking could provide scientific insights about ecological phenomena and evolutionary processes beyond Cartesian reductionism (Maynard Smith 1988). Maynard Smith's largely sympathetical although not entirely uncritical review contrasts sharply with the hostility against the book and its authors from other parts of the evolutionary biology community in the US. In retrospect and with a knowledge of subsequent history – most notably the fall of the Berlin Wall in 1989 and the collapse of the Soviet Union in 1991 – the extremely negative reactions to *The Dialectical Biologist* could perhaps be interpreted as an effect of the general political climate in the US and the ongoing Cold War. Also, many evolutionary biologists in the US were probably not aware about the crucial difference between Stalinism as an official state ideology in the Soviet Union and Eastern Europe, and the more critical intellectual Marxist analytical tradition in Western Europe.

One of the most famous chapters of *The Dialectical Biologist* has the title "The Organism as the Subject and Object of Evolution". In this chapter, the authors build upon some earlier foundational work by Lewontin (Lewontin 1983) and some very general coupled differential equations to explore the relationship between organism (***O***) and environment (***E***). They show that organisms are not only passive *objects* of the external environment which suffer from the force of natural selection, but the organisms are also active *subjects*, who actively modify their environments, often towards their own advantage. Thus, a fit between ***O*** and ***E*** can in principle be achieved in two different ways (although not mutually exclusive); either natural selection modifies ***O*** to fit ***E***, or ***O*** modifies ***E*** to its own advantage (Okasha 2005). One empirical example of this is thermoregulation behaviours in ectothermic animals like reptiles and insects. In *Anolis*-lizards, for instance, it has been shown that because of adaptive behavioural thermoregulation, animals can "buffer" themselves against harsh thermal environments (e. g.

too cold environments) by actively searching for warmer places, thereby partly counteracting selection for improved thermal physiology (Huey et al. 2003). The main point is that there is a reciprocal feedback between ***O*** and ***E***: ***E*** influences ***O*** through the process of natural selection, but ***O*** can also influence ***E*** through niche construction (Odling-Smee et al. 2003; Okasha 2005). For the consistence of terminology I shall call such feedbacks between ***O*** and ***E*** for *reciprocal causation* for the rest of this article, following the terminology by Laland and colleagues (Laland et al. 2015), although I note that Laland (2004) has also called this "cyclical causation" in one of his previous papers (Laland 2004; Dawkins 2004).

Naturalists and field biologists have long been aware that organisms are not only passive objects of selection, but can actually modify their environments or use adaptive habitat selection to maximize fitness (Huey et al. 2003), so in that sense Levin's and Lewontin's main contribution was to formalize what many already knew. However, it took a couple of more decades until Levin's and Lewontin's ideas attracted more interest from modellers. In 2003, Odling-Smee, Laland and Feldman published a book called *Niche Construction – the neglected process in evolution* (Odling-Smee et al. 2003). Building on the original foundations laid by Levins and Lewontin, they further developed the mathematical models of coupled differential equations between ***O*** and ***E***, and argued that niche construction deserve increased attention from evolutionary biologists, as it might potentially be considered a process of equal importance as natural selection on organisms. While many evolutionary biologists would probably agree that niche construction and associated phenomena (including reciprocal causation) are indeed interesting and important, Odling-Smee et al. (2003) were also criticized for overstretching the domain of niche construction (Brodie, III 2005; Dawkins 2004; Okasha 2005). Interestingly, Odling-Smee et al. (2003) cite Levins and Lewontin (1983) at only one page in the beginning of their volume, and neither of the terms "dialectics" or "Marxism" appear in their index. This

is quite a remarkable omission, considering the intellectual and scientific roots of niche construction and the crucial contributions by Levins and Lewontin. It is as if the Cold War was still ongoing in 2003, when *Niche Construction* was published. This is unfortunate, as even the otherwise skeptical John Maynard Smith was not afraid of admitting the fruitful contribution of some aspects of Marxist philosophy to evolutionary theory (Maynard Smith 1988; Maynard Smith 2001).

Niche construction and reciprocal causation have recently been used as arguments in calls for an Extended Evolutionary Synthesis (EES), to replace the Modern Synthesis (MS), sometimes also called Standard Theory (ST)(Laland et al. 2015). These calls have, however, also met several criticisms (Welch 2016). Among the criticisms that have been raised against the EES are that reciprocal causation is already well-recognized in several subfields of evolutionary biology, that it is already incorporated in standard evolutionary theory and that so-called "soft inheritance" is unlikely to be important in evolution (Brodie 2005; Dickins and Rahman 2012; Welch 2016). Here, I discuss this further with a focus on the role of reciprocal causation in the evolutionary process. I show that reciprocal causation features commonly in both empirical investigations and in theoretical models of both ecology and evolution, although it is seldom explicitly framed as such or couched in terms of niche construction. The main challenge is therefore mainly empirical; namely to use existing analytical, statistical and mathematical tools to analyze reciprocal causation and spread knowledge about these tools to other subfields, rather than calling for a major revision of evolutionary theory, or establish an EES.

Reciprocal causation: frequency-dependence, eco-evolutionary dynamics and co-evolution

Brodie (2005) in his review of *Niche construction* criticized the Odling-Smee et al. (2003) for painting a biased and misleading view of how evolutionary biologists study selection and its consequences:

> *"The authors work hard to convince the reader that niche construction is a new ''extended theory of evolution'' that is a ''co-contributor, with natural selection, to the evolutionary process itself'' (p. 370). This argument is based on the somewhat disingenuous contention that evolutionary biologists view natural selection as an abiotic entity that is not influenced or changed by living organisms, and that ''adaptation is conventionally seen as a process by which natural selection shapes organisms to fi pre-established environmental 'templates''' (Laland et al. 2004). This straw man is weakened by the long list of similar ideas that the authors themselves describe, from frequency-dependent selection, to coevolution, to cultural inheritance, to maternal effects. Each of these ideas (and many others) points to a general appreciation that selection is a dynamic process that changes as organisms evolve and interact with their environments. The basic tenets of niche construction can be traced back at least as far as Fisher (1930). The oft-misunderstood fundamental theorem apparently included the assumption that growing populations are expected to degrade their environments so that the positive effects of genetic increases in fitness combine with negative feedback on environmental variation for fitness (Frank and Slatkin 1992). The net result in Fisher's view was that selection for increased fitness would not lead to any observable change in population mean fitness because evolving organisms modify their environments. The more active sense of engineering an organism's own selection was captured early on by Mayr's (1963) notion that behavior leads the evolution of morphology, ecology, and species differences. Through behavioral plasticity, organisms might shift niches, change diets, and move to new habitats, thereby changing selection so that ''other adaptations to the new niche. . . are acquired secondarily'' (Mayr 1963, p. 604). The basic premise that organisms interact with selection through a dual-direction causal arrow is not particularly novel or earth-shattering."*

From the perspective of an empirical field-oriented evolutionary biologist, I very much agree with Brodie's characterization of the ST and the MS above. Here, I would like to add a few more examples to his list of well-studied phenomena and processes which exemplify reciprocal causation. First, negative frequency-dependent selection (NFDS) is a well-recognized evolutionary process that involves reciprocal causation, and which was known already by the founding fathers of the MS and the early mathematical population geneticists. NFDS was explicitly incorporated in Fisher's model for sex ratio evolution (Fisher 1930) and investigated in depth by Sewall Wright in terms of its role in maintaining genetic polymorphisms (Wright 1969). Later, NFDS became popular also in behavioural ecology, through the use of evolutionary game theory (Maynard Smith 1982). Empirically, NFDS has been identified and

studied in several field and laboratory systems, and it is a healthy and dynamic research field that has grown out of the MS (Sinervo and Lively 1996; Sinervo et al. 2000; Svensson et al. 2005; Neff and Svensson 2013; Zhang et al. 2013; Le Rouzic et al. 2015). The importance of NFDS is by no means restricted to its wellknown role in maintaining genetic polymorphisms within local populations, but it can also affect population performance such as stability, productivity or extinction risk (Takahashi et al. 2014). Negative frequency-dependence might also be an important process in community ecology, where it can preserve biodiversity through rare-species advantages (Wills et al. 2006). NFDS is an example of a negative feedback loop in which a genotype's fitness is negatively regulated by its own frequency; the genotype can thus be said to "construct" its local selective environment (Brandon 1990; Fig. 1A)**.** Therefore, NFDS is indeed a prime example of a process exemplifying reciprocal causation, and it has been long been recognized as important in standard evolutionary theory. Finally, Fisher's Fundamental Theorem (as mentioned by Brodie 2005) does also contain a strong element of negative frequency-dependence and density-dependence through the effects of the deterioration of the environment that follows after an efficient, aggressive or highly exploitative genotype have spread in a local population and starts to encounter and interact increasingly with itself (Frank and Slatkin 1992).

However, also positive frequency-dependent selection (PFDS) is an important evolutionary process that exemplifies reciprocal causality (Fig. 1B). Under PFDS, a genotype's fitness will increase as it becomes more common. PFDS leads to the loss of polymorphism and the fixation of locally common genotypes, in contrast to NFDS which plays a conservative role in population divergence (Svensson et al. 2005; cf. Fig. 1A vs. B). One classical example where PFDS plays an important role in evolution is Müllerian mimicry in *Heliconus*-butterflies, where locally common warning colouration patterns are favoured due to predator learning (Chouteau

et al. 2016). Another example comes from classical models of sexual selection through female choice, in particular the Fisher-Lande-Kirkpatrick model (Fisher 1930; Lande 1981; Kirkpatrick 1982; Prum 2010). In this model, there is genetic variation in both female preferences for a male secondary sexual trait and the male trait itself. Female choice and non-random mating leads to the buildup of linkage disequilibrium (LD) between female preference alleles and male trait alleles, and by extension a genetic correlation between these disparate traits, even if they are governed by separate sets of loci (Kirkpatrick 1982). Provided that this genetic correlation becomes of sufficiently high magnitude, a "runaway" process can get started whereby PFDS operates on the population and leads to fixation of certain male-expressed trait alleles and the corresponding female preference alleles (Prum 2010). The important message here is that selection becomes self-reinforcing: given set of preferences, male-expressed trait-alleles spread through the synergistic effects between PFDS and the LD that was generated by the female preference. Thus, not only do the traits of males and females evolve, but so does the selective environment itself – an excellent example of reciprocal causation and feedback between organism and environment (Levins and Lewontin 1985). Recently, positive frequency-dependence has also been suggested to be important in maintaining diversity of ecologically equivalent species (e. g. those formed by sexual selection alone) on regional scales (M'Gonigle et al. 2012), and it might also play an important role in community ecology through priority effects (De Meester et al. 2016). Even more generally, positive feedbacks and runaway processes have been suggested to be important in human social evolution in coalition formation and cooperative networks (Gavrilets et al. 2008) and in ecosystem ecology and climate science (Malm 2007). Positive and negative feedbacks is thus a fact of life.

Reciprocal causation has also a key role in the field of "eco-evolutionary dynamics" (Schoener 2011; Hendry 2016), where the focus are the bidirectional feedbacks between ecological (e. g.

population dynamics) and evolutionary processes (e. g. genetic change within populations). Eco-evolutionary dynamics is expected when ecological and evolutionary time scales converge, such as in the case of rapid evolution, e. g. due to human-induced environmental changes (Hendry et al. 2017). Under such scenarios does not only ecological change affect genetic change, but also *vice versa*: genetic changes can feed back in to ecology and influence population dynamics (Sinervo et al. 2000; Yoshida et al. 2003). Note that this breakdown of the separation between ecological and evolutionary time scales discovered by empiricists is a challenge to certain modelling approaches, such as Adaptive Dynamics (AD), where a strict separation between ecological and evolutionary processes forms a core assumption (Dieckmann and Doebeli 1999; Waxman and Gavrilets 2005). This dynamic feedback between ecology and evolution will without doubt continue to be explored in the future, and shows that reciprocal causation forms a key part of a rapidly growing research field that has developed independently from niche construction. Interestingly, Schoener (2011) called eco-evolutionary dynamics for "the newest synthesis" between ecology and evolution. Schoener's call for this new synthesis was independent of Pigliucci, Laland and colleagues (Pigliucci 2007; Pigliucci and Müller 2010; Laland et al. 2015). In my opinion, and from a strict empirical viewpoint, eco-evolutionary dynamics have contributed more towards the development of a concrete empirical research program than has the EES sofar. I also agree with Welch (2016), that there is an unfortunate tendency in evolutionary biology to repeatedly use the richness of understudied and underappreciated phenomena and processes in our field as arguments for the urgent need of reform and to proclaim the arrival of new syntheses. It is worth emphasizing that there are also several other, more or less parallel attempts to call for new evolutionary synthesis, which are either based on conceptual considerations (Arnold 2014) or based on the rapid development of methods (Losos et al. 2013) that are considerably more modest in their scope than the EES.

Reciprocal causation also forms a key component in studies of co-evolution, either within or between species, such as enemy-victim interactions (Fig. 1C). For instance, under interlocus contest evolution (ICE) between male and female over mating rates (i. e. sexual conflict), males evolve traits that increase their mating success, whereas females evolve resistance towards excessive male mating harassment (Rice and Holland 1997). Under certain conditions, such antagonistic male-female sexual interactions can lead to intraspecific co-evolutionary arms races (Rice 1996) and even speciation (Gavrilets 2000). Females can also evolve either resistance or tolerance to male mating harassment (Arnqvist and Rowe 2005; Gosden and Svensson 2009; Svensson and Raberg 2010), and such sexual antagonism can also, as an alternative outcome, result in the evolution of male and female genetic clustering or polymorphisms (Gavrilets and Waxman 2002; Svensson et al. 2009; Karlsson et al. 2013; Karlsson et al. 2014; Le Rouzic et al. 2015). These antagonistic male-female interactions sometimes result in "Red Queen"-dynamics and co-evolutionary feedbacks between male and female fitness interests (Rice and Holland 1997). This exemplifies how reciprocal causation is deeply embedded in the empirical research traditions of evolutionary genetics and evolutionary ecology. Reciprocal causation also forms the core of theories of interspecific coevolution, such as in the idea of geographic coevolutionary selection mosaics across the landscape of interacting species (Gomulkiewicz et al. 2000; Nuismer et al. 2000; Thompson 2005). As in the case of frequency-dependent selection and eco-evolutionary dynamics, appreciation of reciprocal causation is the norm rather than the exception among active empiricists in the field. Few evolutionary ecologists today would therefore defend the simple unidirectional causality that Laland et al. (2015) associate with the MS and ST.

Reciprocal causation is also explicitly incorporated in models and in empirical investigations in the evolutionary quantitative genetics tradition, where researchers are using the statistical

methods that were developed by Russel Lande and Stevan Arnold (Lande and Arnold 1983; Endler 1986). Examples of such studies incorporating reciprocal causality include indirect genetic effects in social evolution (Moore et al. 1998; Wolf et al. 2001), the evolutionary dynamics of maternal effects (Kirkpatrick and Lande 1989; Wade 1998) and analyses of how interspecific interactions shape selection pressures on phenotypic traits (Ridenhour 2005). Empirical selection studies nowadays are not only aiming to quantify selection differentials and selection gradients, but researchers actively strive to understand the ecological causes of selection, whether those causes are predators, intra- or interspecific competitors (Wade and Kalisz 1990; Svensson and Sinervo 2000; Svensson and Friberg 2007; Calsbeek and Smith 2007; Calsbeek et al. 2012; Kuchta and Svensson 2014). It is a very long time ago since evolutionary ecologists were simply satisfied by having quantified selection; nowadays they are busy understanding the causes of selection and few journals in evolutionary biology publish studies simply presenting selection coefficients without any ecological context (and rightly so).

Analytical and empirical tools for studying reciprocal causation

As I have discussed and exemplified above, reciprocal causation is hardly controversial among evolutionary biologists and widely recognized to be important in several subfields in evolutionary biology, including frequency-dependent selection, eco-evolutionary dynamics, co-evolution and evolutionary quantitative genetics. Moreover, reciprocal causality has been recognized as important for several decades and well before the formalization of niche construction (Odling-Smee et al. 2003) and more recent calls for an EES (Laland et al. 2015). Very few empirical and theoretical evolutionary biologists today adhere to the simple unidirectional causality that Laland et al. (2015) have recently criticized. It is therefore somewhat questionable whether the MS and ST have been fairly characterized by Laland and

coworkers. I think not. Even Ernst Mayr himself expressed a more dynamical view of causality in other contexts and publications than he did in his distinction between proximate and ultimate causes (Laland et al. 2011). Mayr's views of the role of behaviour as a "pace maker" in evolution (Mayr 1963; cited by Brodie 2005), strikes me as being much more sophisticated than the picture of his strict views on unidirectional causality that has been described by Laland et al (2011). Mayr's view of a crucial role of behaviour in the evolutionary process is clearly compatible with feedbacks between the organism and its environment. His surprisingly early insights on the issue has clear similarities with similar views expressed several decades later by West-Eberhard, Levins and Lewontin (West-Eberhard 1983; Levins and Lewontin 1985).

If reciprocal causation is then so widely recognized – at least in several key fields – why then is it not more studied? The answer is probably that there are logistical and empirical challenges, and not all researchers are aware of suitable tools. Progress in the field of evolutionary biology is perhaps more often limited to methods these days than to lack of conceptual insights. It therefore becomes more urgent to communicate between subfields so that researchers become aware of which analytical and empirical tools that are aready available, but which are underutilized. I therefore agree fully with Laland et al. (Laland et al. 2013) that different subfields in biology should become better integrated, but I doubt that such integration will need or necessarily be facilitated by the adoption of an EES, at least not in its current rather vague form. Rather, the main motivation for fostering integration between different fields in biology is that statistical, mathematical and other analytical tools suitable for studying reciprocal causation are underutilized in some subfields, and scientific communication would facilitate their spread.

One such tool that is clearly underutilized in many areas of evolutionary biology and which is

excellently suited to analyze direct and indirect effects is path analysis and structural equation modelling (SEM)(Shipley 2002). Although it has been advocated as a suitable tool in selection analyses on phenotypic traits (Kingsolver and Schemske 1991), path analyses of selection are still relatively few (Sinervo and DeNardo 1996). This is unfortunate, as path analyses and its complementary statistical tool SEM is an excellent tool to incorporate information about how organismal traits are influenced by local social, biotic and abiotic environments, and investigate how traits and selective environments in turn affect fitness (Svensson et al. 2001; Gosden and Svensson 2009). Moreover, path analysis can also be combined with experimental manipulations – either of traits, local selective environments, or both (Sinervo and Basolo 1996; Svensson and Sinervo 2000). Integrative studies combining path analysis, analysis of causation and experimental manipulations will increase our knowledge of organism-environment feedbacks and their role in the evolutionary process (Svensson et al. 2002). Empirical information from covariance or correlation matrices can be translated in to causal quantitative models, whereby SEM provides a powerful tool to evaluate the fit of various alternative models (Shipley 2002).

Another underutilized tool to study reciprocal causation in the evolutionary process is time-series analysis (Le Rouzic et al. 2015). Time-series analysis have perhaps been more used by ecologists interested in population dynamics than by evolutionary biologists, but it holds great promise as a tool to infer the processes driving ecological and genetic dynamics of interacting genotypes within species (Moorcroft et al. 1996; Pemberton et al. 1998; Sinervo et al. 2000; Le Rouzic et al. 2015) or in between-species interactions (Yoshida et al. 2003), especially if it is combined with experimental manipulations to verify causal agents of selection (Wade and Kalisz 1990; Svensson and Sinervo 2000). I anticipate that evolutionary time-series analysis will become an important tool in future studies dealing with eco-evolutionary dynamics, intra-

or interspecific co-evolutionary processes in natural populations (Le Rouzic et al. 2015; Hendry 2016).

Other promising research approaches to investigate reciprocal causation and dynamic feedbacks between organisms and their local environments include studies of non-random dispersal with respect to phenotype or genotype (Edelaar et al. 2008; Eroukhmanoff et al. 2011) and its consequences for matching habitat choice (Edelaar and Bolnick 2012), quantatitive studies on the dynamics of niche evolution using phylogenetic comparative methods (Wiens 2011; Wiens et al. 2011) and experimental field studies on how animals use regulatory behaviours to maintain physiological homeostasis (Huey et al. 2003). Taken together, there is therefore a rich diversity of empirical and analytical tools available to evolutionary biologists who are seriously interested in understanding how reciprocal causation and dynamic feedbacks between ecological and evolutionary processes influence organisms, from individuals to populations, species and higher taxa.

A cautionary note regarding reciprocal causation in heredity and development

As I have shown in this article, reciprocal causation is already widely recognized in several fields, particularly those at the interface between ecology and evolution, in contrast to recent claims by Laland et al. (2015). Laland et al. (2015) do of course not deny the existence of such previous studies but they argue that:

"However, reciprocal causation has generally been restricted to certain domains (largely to direct interactions between organisms), while many existing analyses of evolution, habit- or frequency-dependent selection are conducted at a level (e. g. genetic, demographic) that removes any consideration of ontogeny. Such studied do capture a core structural feature of reciprocal causation in evolution – namely, selective feedback – but typically

fail to recognize that developmental processes can both initiate and co-direct evolutionary outcomes" (p. 7. Laland et al. 2015).

Thus, Laland et al. (2015) admit that reciprocal causation is and has often been studied by evolutionary biologists, but they argue that ontogeny and development should be incorporated in such analyses. I hardly disagree here, and I think incorporating the role of development and ontogeny in studies of (say) frequency-dependent selection, eco-evolutionary dynamics, co-evolution and analyses of selection is likely to yield many novel and important insights. However, the reason that development has not been incorporated in that many previous studies in this field is not that the researchers in question rely on an outdated and simple view of unidirectional causation, as implied by Laland et al. (2015). The reason is more likely a practical one: it is complicated and challenging empirically to understand and study reciprocal causation even at single ontogenetic level, i. e. among adults. I therefore disagree with Laland et al. (2011; 2013) when they imply that the lack of consideration of development in past studies is due to the lasting legacy of Ernst Mayr's proximate-ultimate dichotomy, and their suggestion that evolutionary biologists in general still adhere to an outdated view of unidirectional inheritance. Rather, the lack of studies of this kind reflect legitimate and difficult empirical challenges and I am not convinced that the EES-framework alone can solve these problems, unless some more concrete novel methodological or analytical tools are provided.

Moreover, is also not entirely correct to claim that evolutionary geneticists and evolutionary ecologists have not paid attention to the interplay between ontogeny and selection. For instance, researchers have modelled and investigated how selection pressures change both in magnitude and sign during the organism's life cycle (Schluter et al. 1991; Barrett et al. 2008). There is also much interest and ongoing theoretical and empirical research aiming to integrate and model the interaction between interlocus sexual conflict at the adult stage over the reproductive interests

of males and females, with intralocus sexual conflict experienced earlier in ontogeny (Rice and Chippindale 2001; Chippindale et al. 2001; Barson et al. 2015; Pennell et al. 2016). Finally, there is increased appreciation of how alternative reproductive strategies shape ontogenetic trajectories, and how the same ontogenetic trajectories in turn affect adult phenotypes (Neff and Svensson 2013).

Laland et al. (2015) wish to extend the domain of reciprocal causation from the interaction between ecological and evolutionary processes (as discussed in this article) to the domain of organismal development, what they call "constructive development". I will not dwell too deeply in to this here, due to space limitations, except that I note that there is of course no *a priori* reason why reciprocal causation and dialectical thinking should not be possible to apply also to development. However, constructive development is also perhaps the aspect of the EES that is most controversial and which has sofar been met with most resistance. This resistance is partly understandable and justified, on historical grounds. The architects of the MS had to work hard to get rid of popular evolutionary mechanisms of inheritance and evolution that in hindsight have clearly turned out to be wrong, including orthogenesis, vitalism, saltationism and Lamarckian inheritance (Smocovitis 1996; Mayr and Provine 1998). The increasing interest in epigenetic inheritance is certainly justified and will most likely lead to new empirical insights. Clear cases of epigenetic inheritance now exists (Dias and Ressler 2014), but it is now mainly an empirical issue to understand the importance of such effects and how widespread they are. However, claims that epigenetic inheritance can play a major role in explaining macroevolutionary phenomena (Pigliucci and Murren 2003) or that such epigenetic inheritance would imply a major role for Lamarckian inheritance in evolution has been justifiably criticized as speculative and without empirical footage (West-Eberhard 2007; Dickins and Rahman 2012). Neither Lamarck nor Lysenko are likely to be vindicated, in spite of the wishful thinking

of those who might think otherwise (Jablonka and Lamb 2005; Haig 2007).

Maynard Smith (1988; 2001) who certainly admitted that Marxist philosophy and dialectical thinking could have a constructive influence on evolutionary theory cautioned against overstretching dialectics to heredity and development. His cautionary point should also be taken seriously by those today who argue for constructive development, including Laland et al. (2015). Few evolutionary biologists would claim that the genotype-phenotype map is linear or would deny that genes, environments and genotype-environment-interaction jointly influence the development of phenotypes. The ideas of cultural evolution and various forms of non-genetic inheritance (including ecological inheritance) can and does play some role in between-generation change of phenotypes is also gaining some acceptance. However, it is considerably more controversial to argue for reciprocal causation in the sense that environment would causally influence genetic inheritance, i. e. a revival of acquired genetic inheritance and neo-Lamarckism. The idea that genes are unaffected by environments and that the germ line is separated from the soma may seem undialectical, but it is a fact of life. Strong evidence would be required for any claims – made by EES proponents or others - that the Weismannian germline-soma separation is not valid anymore, that the so-called "Central Dogma" of molecular genetics does not hold up (Maynard Smith 1988; 2001) or that soft inheritance plays a major role in evolution (Haig 2007; Dickins and Rahman 2012). Thus, while most evolutionary biologists would have little problems in accepting reciprocal causality in ecological and evolutionary interactions between organisms and their selective environments, few are prepared to accept reciprocal causality in the domains of directed development and genetics, unless strong empirical evidence is presented for such claims.

Conclusions

Reciprocal causation is frequent in many studies of evolutionary processes, particularly those involving interactions between organisms, both within or between species. Research on reciprocal causation has a long tradition in evolutionary biology, contrary to recent claims by proponents for the EES. Reciprocal causation was studied well before the recent calls for an EES, although not explicitly under the umbrellas of niche construction. Evolutionary biologists today – particularly those working at the interface between ecology and evolution seldom have such a simplified view of unidirectional causation as claimed by Laland et al. (2015), and reciprocal causation is already part of the conceptual framework of many empirical biologists. Apart from the subfields I have discussed in this article, there are also several other relatively new and emerging areas where reciprocal causation is deeply embedded. For instance, non-random dispersal of phenotypes and matching habitat preferences (Edelaar et al. 2008; Edelaar and Bolnick 2012; Eroukhmanoff et al. 2011) is increasingly appreciated as being important, and shows that organisms are not solely passive objects of evolution, but evolutionary subjects in their own right, with some degree of independence (cf. Levins and Lewontin 1985). Similarly, niche conservatism (Wiens et al. 2010) is common in many organisms, meaning that organisms actively track habitats where their fitness is maximized, rather than passively evolving *in situ*. Such niche conservatism has many interesting evolutionary consequences for speciation (Wiens 2004), thermal adaptation (Svensson 2012) and thermoregulatory behaviours (Huey et al. 2003).

Evolutionary biology is a rich and diverse discipline that spans many levels of biological organization and taxonomic groups and which covers many different types of questions. This richness and diversity of our discipline can be viewed as a strength, but also comes with a cost:

it is relatively easy to find areas where more research would be needed and topics that have been relatively little explored (Welch 2016). The existence of such knowledge gaps is presumably the primary science-sociological explanation for why calls for major revision of evolutionary theory or attempts to formulate new syntheses appear with regular intervals (Welch 2016). We have seen several more or less independent attempts to formulate new syntheses only during the last decade (Pigliucci 2007; Pigliucci and Müller 2010; Schoener 2011; Losos et al. 2013; Arnold 2014; Laland et al. 2015). These attempts were preceeded by several other calls in the past (Gould 1980). However, as I have argued elsewhere, new syntheses do not automatically establish themselves in the evolutionary research community because they are being declared to be here, or because some biologists think that they are warranted (Svensson and Calsbeek 2012a). Rather, new syntheses become established only if they provide some new analytical, experimental, mathematical or statistical tools that moves the research field forward. The MS was never such a monolithic research paradigm as often portrayed by some of its critics (Jablonka and Lamb 2005; Laland et al. 2015). Rather, it was a loose, albeit largely successful framework and attempt to unify the very heterogeneous different branches of biology (Smocovitis 1996; Mayr and Provine 1998). The MS contained several very conflicting perspectives on evolutionary biology, both between different branches of population genetics (Provine 1986; Frank and Slatkin 1992; Coyne et al. 2000; Wade and Goodnight 1998) and between researchers focusing on micro- vs. macroevolution (Eldredge and Gould 1972; Charlesworth et al. 1982; Futuyma 2015). Past and ongoing debates about the role of selection versus neutralism in explaining genetic variation (Lewontin 1974; Gillespie 1991) and the role of population structure, genetic drift and mass-selection in large panmictic populations (Coyne et al. 2000; Wade and Goodnight 1998) all illustrate that the MS has been continually evolving and adapting as a flexible minimum platform that has survived several replacement attempts (Smocovitis 1996; Svensson and Calsbeek 2012b). The MS will therefore

most likely probably continue to evolve and slowly adapt also in the future (Arnold 2014).

The perspective put forward in this article is largely an empiricist one. While I disagree with Laland et al. (2011;2015) that reciprocal causality is that neglected in evolutionary biology, I fully agree with them that it should become more widely appreciated, recognized and studied empirically and theoretically. The study of reciprocal causation need to move beyond rhetoric and position papers and become operational. The insights that organisms construct their own environments and appreciation of organism-environment feedbacks as well as the discussion whether there exist empty niches (or not) are all interesting but needs to be translated to a rigorous empirical research program, for instance, by utilizing the analytical tools I have discussed in this article. The widespread existence of reciprocal causation should not be taken as an argument that the study of cause-effect relationships are empirically impossible, but should rather motivate researchers to dissect long causal chains in to smaller operational study units with the goal to better understand the evolutionary process. Of course, we need to appreciate the crucial difference between the ecological and selective environment (Levins and Lewontin 1985; Brandon 1990), but this challenge should not hinder the development of operational tools and to use existing analytical methods, such as path analysis and time-series analysis.

Reciprocal causation is already deeply embedded in many – perhaps the majority – of evolutionary processes, and should therefore be a natural and major research focus. Although broader appreciation of the role of reciprocal causation is unlikely to lead to a new paradigm shift (Laland et al. 2013) and is probably not a sufficient reason to call for major revision of evolutionary theory (Laland et al. 2015), reciprocal causation is nevertheless a good example of how Marxist philosophy and dialectical thinking have had a positive influence on the

development of our field. We should therefore fully embrace the concept of reciprocal causality and acknowledge the important contributions of Levins and Lewontin in their important book (Levins and Lewontin 1985).

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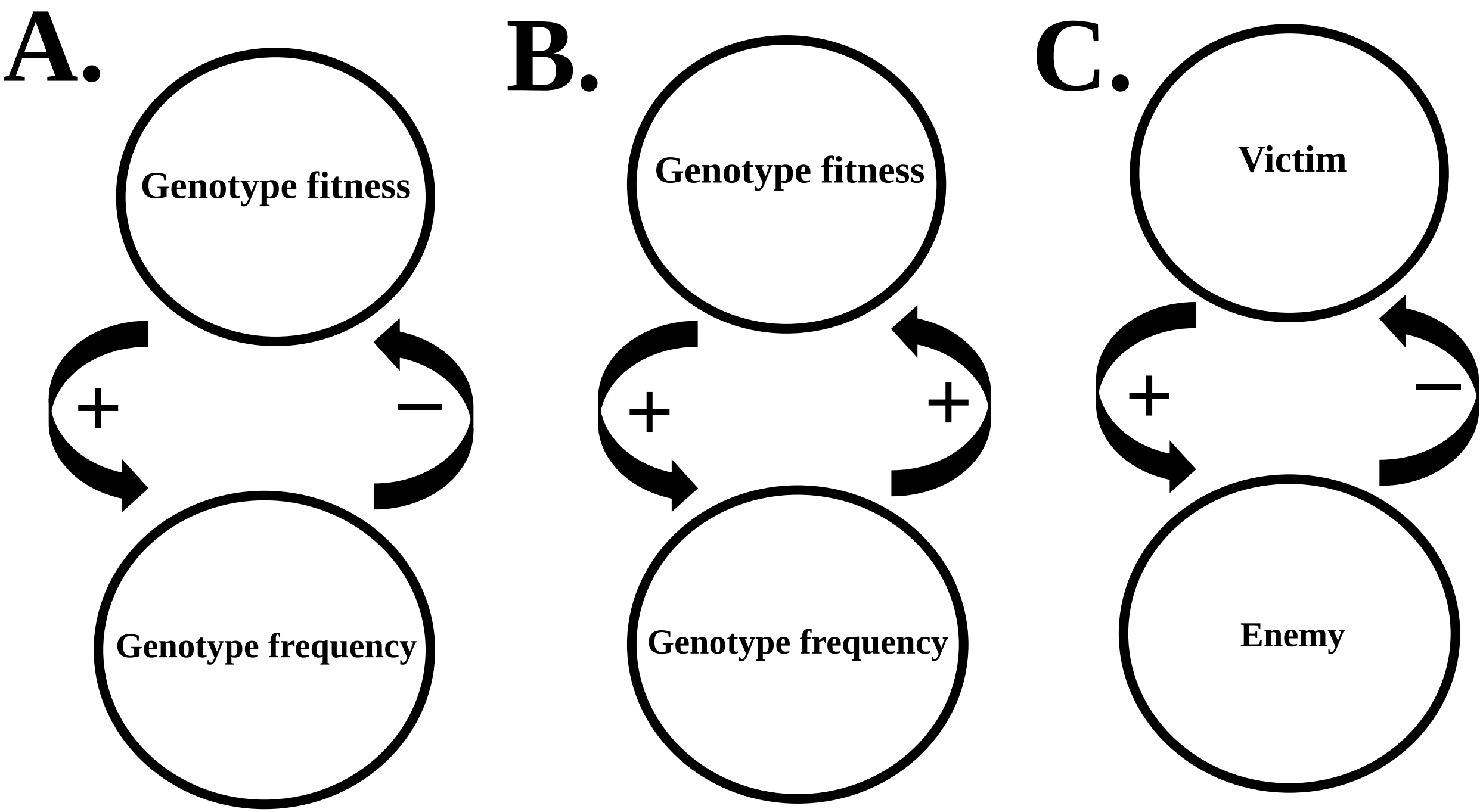
Legend to figure

Figure 1. Three examples of reciprocal causation and feedbacks in the evolutionary process. **A.** Negative frequency-dependent selection (NFDS), exemplifies negative (regulatory) feedback between genotype frequency and fitness. As a genotype increases in frequency, its fitness declines, leading to the preservation of genetic diversity and genetic polymorphisms locally. The genotype thus "constructs" its own selective environment by regulating its own fitness, and the selection coefficient on the genotype changes dynamically with changing frequency. **B.** Positive frequency-dependent selection (PFDS), exemplifies positive feedback between a genotype's frequency and its fitness, leading to fixation of the most common phenotype. **C.** Enemy-victim coevolution is an example of a negative feedback, that can either lead to stable equilibria or co-evolutonary cycles (e. g. "Red Queen" evolutionary dynamics). The enemy and the victim can belong to different species (e. g. predators or prey, parasites or hosts) or the same species (e. g. males and females).


A.
Genotype fitness
+
−
Genotype frequency
B.
Genotype fitness
+
+
Genotype frequency
C.
Victim
+
−
Enemy