Chapter 23: Mechanisms in Evolutionary Biology

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I. Introduction

In 1961, renowned Harvard biologist Ernst Mayr asked himself why the warbler he had been observing on the grounds of his New Hampshire summer home began its southward migration on the night of the 25th of August. The answer, he came to realize, was not a simple one. There were ecological causes: being an insect-eater, the warbler would starve in the New Hampshire winter. There were physiological causes: the warbler had an intrinsic capacity to sense the dwindling number of daylight hours. And there were genetic causes: the warbler's special sensitivity to environmental stimuli indicating the approach of colder climate was programmed into its very DNA. But on Mayr's view, these myriad reasons for the warbler's migration were really just of two kinds: the *proximate causes* dealing with the physiology of the warbler as it related to the photoperiodicity and air conditions in its environment, and the *ultimate causes* dealing with the bird's evolutionary history, the way its genetic constitution had been molded by natural selection over many thousands of generations (Mayr 1961).

Well beyond understanding the migration habits of the common warbler, Mayr's distinction between proximate and ultimate causes was to serve as grounds for demarcating the distinct explanatory magisteria of two different kinds of biology. On the one hand, *functional biology* seeks to understand proximate causes: "the functional biologist is vitally concerned with the operation and interaction of structural elements, from molecules up to organs and whole individuals. His ever-repeated question is 'How?'" (Mayr 1961, 1502). *Evolutionary biology*, on the other hand, lives squarely in the domain of ultimate causes:

> The animal or plant or micro-organism… [the evolutionary biologist] is working with is but a link in an evolutionary chain of changing forms, none of which has any permanent validity. There is hardly any structure or function in an organism that can be fully understood unless it is studied against this historical background. (Mayr 1961, 1502)

Rather than asking 'How?', the evolutionary biologist's perpetual question is 'Why?'.

Mayr's ideas regarding functional vs. evolutionary biology have been the subject of renewed interest in the philosophy of biology literature. Some have argued that they have significant flaws (Laland et al. 2011, 2013, Callicot 2013) while others have attempted to support the spirit, if not the letter, of Mayr's conclusions (Ariew 2003, Scholl and Pigliucci 2014). I will not weigh in on the specifics of this ongoing debate, but I bring up Mayr's framework because it represents an influential and pervasive way of thinking about the explanatory scope of evolutionary biology—one that, if sound, has important implications for the role of mechanistic philosophy of science in evolutionary biology. Consider, for example, the following recent characterization of evolutionary biology from Futuyma's foundational text, *Evolution*,

> Evolutionary biology is a more historical science than most other biological disciplines, for it seeks to determine what the history of life has been and what has caused those historical events. It complements studies of the PROXIMATE CAUSES (immediate, mechanical causes) of biological phenomena—the subject of cell biology, neurobiology, and many other biological disciplines—with an analysis of the ULTIMATE CAUSES (the historical causes, especially the action of natural selection) of those phenomena. (Futuyma 2013, 13).

As seen here, evolutionary biology is still today conceived of as an essentially historical science devoted to understanding ultimate causes. And it is still conceived of as distinct from fields like cell biology, molecular biology, developmental biology, or genetics whose job is to delineate immediate, mechanical, proximate causes.

What does any of this have to do with the philosophy of mechanisms and its role in evolutionary biology? Here is the rub. Implicit in both Mayr's and Futuyma's ideas are the following two claims:

(i) Evolutionary biology takes ultimate rather than proximate causes as its subject matter.

and

(ii) Mechanistic causes are proximate rather than ultimate.

But if we accept (i) and (ii), it would seem to follow that

(iii) Mechanistic causes are not the subject matter of evolutionary biology.

Ergo, we might extrapolate,

(iv) *The mechanistic approach to philosophy of science has nothing* (at least directly) *to offer the study of evolution.*

The purpose of this chapter is to explore whether these conclusions are warranted -- that is, to explore whether, and to what extent, the mechanistic philosophical framework has any purchase in evolutionary biology.

There are a number of ways we might go about this. One way would be to question whether there *are* any immediate mechanistic causes driving evolution. Is natural selection aptly understood as a mechanism? Is drift? Mutation? Let us refer to this as the *metaphysical question*:

(MQ) Are any of the primary causal drivers of evolution aptly understood as mechanisms?

If any of primary causal drivers of evolution are aptly understood as mechanisms, and the study of the primary causes of evolution is within the subject matter of evolutionary biology, then it

would seem that—contra (iii)—there *is* a straight-forward sense in which evolutionary biology should take mechanistic causes as (at least partly constitutive of) its subject matter.

Beyond the metaphysical question, however, there is also an important *epistemological question*:

> (EQ) When applied to evolutionary biology, does the mechanistic philosophical framework help add to our understanding of evolution?

The answer to this question, I suggest, may be quite independent from the first.[1] Regardless of whether there are any mechanisms of evolution in the robust metaphysical sense, it might still be the case that the mechanistic philosophical framework can offer some important explanatory, pragmatic, or otherwise strategic resources that, when applied to the field of evolutionary biology, help *illuminate* the phenomenon of evolution. If so, then—contra (iv)—there may indeed be a valuable role for the philosophy of mechanisms in the study of evolution.

In what follows, I will consider responses to both the metaphysical and epistemological questions. In section 2, I briefly lay out some of the central metaphysical features of mechanisms as they have been characterized in recent literature. In section 3, I examine whether these features of mechanisms are aptly understood as being instantiated by three of the primary causal drivers of evolution: natural selection, drift, and mutation. In section 4, I expound a few of the strategic roles played by mechanistic thinking and hint at the ways they might apply to the field of evolutionary biology regardless of whether there turn out to be any mechanisms driving evolution. I conclude, in section 5, that—on *at least some* philosophical characterizations of mechanisms, and with regard *to at least some* of the central processes of evolutionary biology—there is room for affirmative answers to both the metaphysical and epistemological questions. And thus, there may indeed be an important place for mechanisms in evolutionary biology.

II. Central Metaphysical Features of Mechanisms

In this section, I highlight five central metaphysical features of mechanisms: components, operations, organization, function, and regularity.[2] Many of these aspects of mechanisms have been discussed by other authors in this volume (See especially Chapters 7, 8, 9, 10, 12), so I will not spend much time here. The point is to say just enough about what makes mechanisms the kinds of things that they are in order to be able to arbitrate the question of whether natural selection, drift, or mutation might qualify.

F1 - components: Mechanisms have *components* or *constitutive parts*. Machamer, Darden, and Craver (MDC) call these "entities" and claim that, along with their "activities", they form the two aspects of the "dualist ontology" of mechanisms (MDC 2000, 3). Stuart Glennan refers to these as "parts" (Glennan 1996, 52; 2002, S344), Bechtel and Abrahamsen as "component parts" (Bechtel and Abrahamsen 2005, 47); (see Chapter 9 for further discussion about mechanism components).

F2 - operations: Mechanisms' component parts perform *operations*; they *do* things. In MDC's terms, these are the "activities" performed by the entities—the second aspect of mechanisms' dualist ontology (MDC 2000, 3). Glennan characterizes this aspect of mechanisms as the "interactions" between parts (Glennan 1996, 52; 2002, S344), and Bechtel and Abrahamson as "component operations" (Bechtel and Abrahamsen 2005, 47).

F3 - organization: Mechanisms must be—in some sense—*organized*. For MDC, this means that mechanisms have "start-up" and "termination" conditions, and that mechanism's entities must be (1) located (2) structured, (3) oriented; and a mechanism's activities must have (4) temporal order (5) rate, (6) duration (MDC 2000, 3). Bechtel and Abrahamsen speak of a mechanism as a

"structure" performing a task by virtue of its component parts, operations, and their "organization" (Bechtel and Abrahamsen 2005, 47). And Glennan speaks of mechanisms as "complex systems" underlying a given phenomenon (Glennan 1996, 52; 2002, S344).

Unlike the merely nominal disagreements mentioned in (F1) and (F2), however, there appear to be substantive differences in the organizational requirements placed on mechanisms between contemporary mechanists. Bechtel and Abrahamsen (2009, 2010, 2013) argue that the MDC organizational requirements are untenable if we wish to characterize feedback mechanisms (e.g., the mechanisms responsible for circadian rhythms) or for mechanisms for the maintenance of equilibrium (e.g., regulatory mechanisms)—as these phenomena have no clear start or termination conditions. In order to accommodate single causal chains as mechanisms (e.g., the 1980 death of French literary critic Roland Barthes when he was struck by a laundry truck while crossing a Paris street), Glennan goes even further towards loosening the organizational requirement by arguing for a notion of "ephemeral mechanism" according to which the configuration of a mechanism's parts may be "short-lived" and "non-stable" (Glennan 2010, 260).

F4 - function: Mechanisms have to be *set up to do something*; they must carry out a *function.* Glennan (1996) points out that "one cannot even identify a mechanism without saying what it is that the mechanism does" (Glennan 1996, 52). For MDC, mechanisms have a function "to the extent that the activity of a mechanism as a whole contributes to something in the context that is taken to be antecedently important" (MDC 2000, 6). And Bechtel and Abrahamsen state explicitly that "a mechanism is a structure performing a function" (Bechtel and Abrahamsen 2005, 47).

Once again, however, there appear to be substantive disagreements about how to understand mechanistic function. As in the more general debate about functions in biology, there seem to be roughly two camps: proponents of *normative* notions of function (e.g., Millikan's (1989) and Neander's (1991) notion of "proper function"—notably defended in the context of mechanisms by Garson (2011)) vs. *non-normative* causal notions of function (e.g., Cummin's (1975) idea of "causal-role function"—notably applied to mechanisms by Craver (2001)). (See Chapter 8 for a detailed discussion of mechanistic function).

F5 - regularity: Mechanisms carry out their function in a *regular* fashion. For MDC, the entities and activities constitutive of a given mechanism "work always or for the most part in the same way under the same conditions" (MDC 2000, 3). Bechtel and Abrahamsen (2005) say that mechanisms are structures responsible for a given "phenomenon"—where 'phenomenon' is here understood in the regularist sense advocated by Bogen and Woodward (1988).

As with (F3) and (F4), there is again substantive disagreement between mechanists about the degree to which regularity should be conceived of as a metaphysical prerequisite for mechanisms. Machamer (2004) as well as Bogen (2005) have argued that regularity should be stricken from the MDC characterization of mechanism on the grounds that many of the phenomena targeted for mechanistic explanation (e.g., especially synaptic transmission) fail to achieve termination conditions much more often then they succeed. DesAutels (2011, 2015) argues that neither a fully regularist nor a fully irregularist characterization of mechanism is tenable, and a notion of stochastic mechanism must be developed. Holly Andersen (2012) defends a version of the MDC regularity requirement but develops a helpful taxonomy according to which mechanistic regularity comes in a variety of strengths and may be located in a variety of

loci within a given mechanism (see Chapters 11, 12, and 13 for further exposition of some of the issues surrounding mechanistic regularity).

III. Do the Primary Causal Drivers of Evolution Instantiate (F1) – (F5)?

Having now laid out the above central metaphysical features of mechanisms (F1) – (F5) as well as having drawn attention to a few places where contemporary mechanists disagree on the nature and strength of these various requirements, we can get on with exploring whether they are instantiated by some of the primary causal drivers of evolution: natural selection, drift, and mutation.

Natural selection: Provided there is heritable variation of fitness-relative traits among a population as well as competition for limited resources, nature tends to preserve those characteristics that afford their possessors the greatest chance to survive and reproduce, and it tends to reject those that do not. The result is that, over time, species become increasingly well-matched to their respective environments—or else they go extinct. In its basic form, this is *natural selection*. Understood in this way, does natural selection instantiate (F1) – (F5)?

Regarding (F1) and (F2), I suggest that natural selection fares pretty well. Consider Darwin's finches as a paradigmatic case. In this example, natural selection seems clearly made up of *components*: finches with differing beak-lengths and seeds that are harder or easier to forage depending on beak-shape. And these componential entities undertake *operations*: foraging for seeds, differential reproduction, and predator avoidance among fitter finches. Barring general skepticism about the nature of part-whole relations—regarding which mechanistic mereology would hardly seem to be a special case—it seems entirely reasonable to conceive of natural selection as being composed of active entities.

Regarding (F3) – (F5), the story is more complicated. Whether natural selection is taken to meet the organization criterion set forth in (F3) depends on how strict we take the requirement to be. On the original MDC characterization of mechanism according to which mechanisms are to have definitive set-up/start and termination conditions, natural selection seems to fall short.[3] When would we say of Darwin's finches that the mechanism of natural selection begins or ends? It seems that any attempt to delineate such temporal boundaries would be arbitrary or ad hoc. Furthermore, unlike man-made mechanisms like clocks or toasters, there is not an obvious physical boundary around the mechanism of natural selection—which makes it hard to determine what its internal structure might be. That said, on more lenient versions of the organizational requirement, there do not seem to be any obvious organizational impediments for natural selection. If, like Bechtel and Abrahamson (2009, 2010, 2013), we allow for feedback mechanisms or mechanisms for the maintenance of equilibrium, neither of which have obvious start or termination conditions, then it seems we should not disallow natural selection from being a mechanism on the grounds that it lacks such conditions. Or if, like Illari and Williamson (2010), we cash out mechanistic organization in terms of functional individuation, then natural selection meets the organizational requirement just as well as paradigmatic mechanisms from molecular biology (e.g., protein synthesis). Or if, like Glennan (2010), we allow for any single causal chain to be a short-lived, non-stable, ephemeral mechanism, then it is even easier to see that natural selection passes muster.

What about (F4)? Does natural selection have a function? Is it set up to do something? Once again, these questions do not have straightforward answers. Whether there is sense to be made of teleology in evolution is an issue with a long and complicated history.[4] That said, even if we agree (as most do) that there is not teleological directionality to evolution—no end-goal it

sets out to achieve—there may still be a sense in which natural selection has a function. Namely, *it is that which brings about adaptation*. And if we take on something like the causal-role understanding of biological function (a la Cummins (1975) and Craver (2001)), and grant that natural selection is that which plays the causal role of bringing about adaptation, then natural selection might well satisfy (F4)[5].

So how about (F5)? Does natural selection evince the regularity that is required for being a mechanism? Once again, whether we answer this in the affirmative depends on how one understands mechanistic regularity. If one requires fully deterministic output conditions from a process in order for it to count as a mechanism, then natural selection surely does not pass. For as Gould (1990) famously argued, if the tape of evolution were played back again-and-again, it would never turn out the same way twice. In this way, natural selection should be understood as probabilistic rather than deterministic. And Skipper and Millstein (2005) have argued that this probabilistic character of natural selection precludes it from meeting the regularity requirements set forth by MDC (2000). On the other hand, failure to behave fully-deterministically does not preclude all notions of mechanistic regularity from being met. Barros (2008) argues that natural selection is a biased stochastic mechanism: one that is regular in that it succeeds in producing predicted outcomes over 50% of the time. DesAutels (2016) argues that, when we distinguish between process vs. product, internal vs external, and abstract vs. concrete regularity, then natural selection escapes the Skipper and Millstein regularity critique just fine.

Drift: Drift happens when a population changes over time—where these changes are *not* the result of natural selection.[6] Consider, for example, a population of snails half of which are pink, and half are which are yellow.[7] Imagine further that the yellow snails are twice as fit as the pink ones because of their greater resistance to the sun. Scientists observing these snails expect,

therefore, that the population of yellow to pink snails should increase from 1/2 of the population to 2/3 in the subsequent generation. However, suppose that after observing these snails for one generation, they find that the population of yellow snails actually decreases from 1/2 to 2/5 of the snail population. Because this change in the population is not due to natural selection (selection would have increased the relative proportion of yellow snails), they attribute this unexpected result to drift. Understood in this way, how does drift fare with regard to (F1) – (F5)?

Regarding (F1) and (F2), I suggest it remains reasonable to consider drift as comprising both components and operations. In our above example, there are pink and yellow snails engaged in usual survival and reproductive activities. There are also whatever entities and activities were responsible for the unexpected decrease in the population of yellow snails—perhaps a dangerous fungus to which only the yellow snails were vulnerable. So once again, modulo general mereological skepticism, there seems to be little problem with conceiving of this instance of drift as being composed of active entities.

Unfortunately, the story regarding (F3) – (F5) is even more difficult than it was with natural selection. For instance, it seems particularly difficult to conceive of a sense in which drift is *organized*—or that it has any particular *structure*. At least with natural selection, there are some commonalities between the sorts of relationships that occur among its participants (e.g., competition for limited resources or differential reproduction favoring fitter members of the population), and the participants must meet certain preconditions in order for natural selection to occur (e.g., variation in the population and heritability of these variations). On the other hand, because drift is an umbrella term catching all non-selective instances of population change over time, it seems much more difficult to delineate any common characteristics shared among all of its instances. And this, I suggest, makes it exceedingly difficult to understand drift as meeting

any kind of organizational constraint. That said, there may still be some sense to be made of drift as a *causal* process.[8] (The decrease in population size of the yellow snails, though not caused by natural selection, was surely caused). And if we follow Glennan (2010) in allowing for a conception of ephemeral mechanism that covers all instances of single causal chains, then it may well be that we could conceive of instances of drift as ephemeral mechanisms: i.e., mechanisms that are "short-lived" and "non-stable" like the one responsible for the death of Roland Barthes.

But does drift have a *function*? Is there something that drift is *set up to do*? Once again, this is tough to answer affirmatively. Unlike with natural selection which (at least on a causal-role notion of function) can be understood as that which brings about adaptation, drift does not seem to do any specific thing; it does not serve any particular unified causal role. Unless we allow for the function of drift to be the fixation of a given trait absent selection (a function that risks vacuity given the very definition of drift), it seems drift cannot meet (F4).

Which leaves (F5): *regularity*. Is there any sense to be made of drift operating regularly? Here, I suggest that the prospects are once again somewhat dim. Due to the aforementioned nature of drift as a catch-all for every instance of non-selective population change, it is near impossible to conceive of it as operating in a regular fashion. A population might drift because of disease, human encroachment, natural disasters, or just plain bad luck. And if mechanistic regularity requires, as MDC suggest, that mechanisms operate "always or for the most part in the same way under the same conditions," then we might think that drift appears to fall woefully short. That said, there may be more space for understanding drift as operating regularly than all of this might portend. For one thing, drift (at least according to many biologists) *tends to occur more often among smaller populations*. So in this sense, there are at least some conditions we can specify under which drift is more likely to occur. And if such conditions can be specified,

then there may at least be a sense in which drift operates more regularly in some circumstances than others.

Mutation: Deoxyribonucleic acid (DNA) is composed of two polymers made up of nucleotides and a backbone of sugars and phosphate groups—all organized into the shape of a double helix. DNA replication begins when the parent molecule gets unzipped as the hydrogen bonds between the base pairs are broken. Once separated, the sequence of bases on each of the unzipped strands becomes a template for the insertion of a complementary set of bases. Deoxynucleoside triphosphates assemble the new strands in the order that complements the order of bases on the strand serving as the template. When the process is complete, two DNA molecules have been formed identical to each other and to the parent molecule. However, there are several ways that the process of DNA replication can (and does) go wrong—resulting in *mutation*. One such way is when a base is changed by the repositioning of a hydrogen atom, altering the hydrogen bonding pattern of that base, and resulting in incorrect base pairing during replication. Another way is when there is a loss of a purine base (A or G) to form an apurinic site (AP site). There can also be denaturation of the new strand from the template during replication, followed by renaturation in a different spot. This can lead to insertions or deletions.

Despite the fact that mutations are essentially *mistakes* in DNA replication, it is a good thing they occur. Without them, there would be no evolution. Mutations are the source of the variation on which natural selection acts. As such, mutation is an indisputable causal driver of evolution. So how does mutation fare with regard to (F1) – (F5)?

Here, again, there are no serious obstacles to meeting (F1) or (F2): we have component entities: DNA, a purine base, individual molecules, etc., and we have operations: unzipping, separating, inserting, etc.

Regarding (F3) – (F5), the situation with mutation seems a good measure improved from that of both natural selection and drift. Mutation appears to have plausible start and set-up conditions: the presence of DNA in a living organism and the initiation of hydrogen bond separation. And it has reasonable termination conditions: the existence of a mutated strand of DNA. Additionally, unlike either natural selection or drift, the process responsible for mutation has a readily discernable molecular structure common among all of its instances. Furthermore, much like natural selection, mutation can easily be seen to carry out a unified causal-role function: it is that which brings about genetic variation at the population level as a result of copying errors at the individual level. Finally, more-so than either selection or drift, mutation seems to occur in a regular fashion. It is well-known, for example, that errors during nucleotide substitution are biased and regularly occur more often at certain locations on the chromosome.

So how should we sum up the metaphysical question? To what extent is it the case that these primary causal drivers of evolution (natural selection, drift, and mutation) are aptly understood as mechanisms? What we have just seen is that the answer to the metaphysical question is complicated. Among the primary causal drivers of evolution, whether they are aptly understood as meeting (F1) – (F5) depends crucially on how liberal we understand these requirements to be. With strict enough constraints on organization, function, and regularity, it may well be that none of these processes end up counting as mechanisms. On highly liberal versions of these constraints, they all may well qualify. So the painfully residual question, then, is: *how liberal should we understand these metaphysical requirements to be*? The answer to *this* question, I am afraid I cannot offer in this short essay. But I will say this. Liberalized metaphysical requirements on mechanisms have the advantage of allowing more physical processes to be understood as mechanisms, and this may have several pragmatic benefits (to be

briefly expounded briefly in the following section). However, highly liberalized metaphysical requirements have drawbacks as well. Namely, the more physical processes we allow to count as mechanisms, the less interesting and distinctive mechanistic explanations will end up being. As with any tradeoff between theoretic virtues, the right balance can only be arbitrated by extra-theoretic values.

IV. Does the Mechanistic Framework add to our Understanding of Evolution?

Let us turn our attention to the epistemological question. That is, let us consider whether, *regardless* of whether any of the primary causal drivers of evolution are aptly understood as mechanisms, the mechanistic philosophical framework might help add to our understanding of evolution.

But how could this possibly be? How could mechanistic philosophy of science help illuminate the study of evolution even if there are no mechanisms driving it? In pondering this question, it will be helpful to consider the notion of "strategic mechanism" recently developed by Levy (2013). According to Levy, one way of approaching the philosophy of mechanisms is to conceive of it as constituting a *strategy*: "a way of doing science, a framework for representing and reasoning about complex systems" (Levy 2013, 105). Regardless of the metaphysical status of mechanisms out there in the world, the idea is that the mechanist "sees mechanistic methods as having particular cognitive and epistemic features" (ibid, 105) and maintains that "certain phenomena are best handled mechanistically" (ibid, 100).

In keeping with Levy, I suggest that there are at least three significant strategic roles that mechanisms might play in our understanding of science regardless of their metaphysical status: *reductionist explanation*, *manipulation*, and *discovery*. And when applied to the field of

evolutionary biology, it seems to me that each of these strategic roles has the potential for adding to our understanding of evolution.

The strategic role of reductionist explanation is emphasized most convincingly by Bechtel and Richardson (1993) in their discussion of "complex localization." They write, "Complex localization requires a decomposition of systemic tasks into subtasks, localizing each of these in a distinct component. Showing how systemic functions are, or at least could be, a consequence of these subtasks is an important element in a fully mechanistic explanation" (Bechtel and Richardson, 125). Thinking about nature mechanistically leads naturally to explanation by reduction and decomposition. When thinking mechanistically, we can more readily come to an understanding of *why* some phenomenon occurs by reducing it to its component parts and operations and by showing *how* the phenomena in question is brought about these parts and operations. Evolution is no exception. To understand why some trait (or individual with a given trait) was selected for, it seems a perfectly good strategy to decompose the larger ecological system in which the individual is a member into its component parts and operations—and in doing so—gain insight as to the advantages the trait confers. A similar story can be told of the strategic role of mechanisms for manipulation. When we model an instance of selection, drift, or mutation as a mechanism (often this is done using mechanism schemata made up of boxes and arrows representing causal relationships), it may become more salient where to intervene on the system in question in order to test various hypotheses about the nature of these causal interactions.[9] Finally, as emphasized in their recent book, Craver and Darden (2013) convincingly show that mechanistic thinking has tremendous benefits viz. scientific discovery: by progressively filling in the black boxes in our incomplete mechanism schemata, scientists are supplied with an invaluable heuristic for moving from *how-possibly* explanations to *how-actually*

explanations of the natural world. This benefit seems especially apropos in the study of evolution where so many of our discoveries (e.g., fossils of intermediate forms like *Archaeopteryx* or *Tiktaalik*) originated as the product of speculative hypotheses.

Of course, it would require much more argument to establish that these strategic advantages of mechanistic thinking *actually do* pay off in the field of evolutionary biology. However, since I cannot undertake this work here in any detail, I am content for the time being to establish that an affirmative answer to the epistemological question has a degree of plausibility. And this plausibility remains even if no affirmative answer can be given to the metaphysical question. Levy's notion of strategic mechanism helps to do just that.

V. Conclusion

In this chapter, I have explored answers to both the metaphysical and epistemological questions regarding the status of mechanisms in evolutionary biology. Regarding the metaphysical question, I briefly laid out some of the central metaphysical features of mechanisms and examined whether they are aptly understood as being instantiated by a three of the primary causal drivers of evolution: natural selection, drift, and mutation. Provided we adopt relatively lenient versions of these constraints, I suggest the answer is yes. Regarding the epistemological question, I briefly expounded Levy's notion of strategic mechanism and hinted at ways the strategic benefits of mechanistic thinking might apply to the field of evolutionary biology regardless of whether there turn out to be any mechanisms driving evolution. I conclude, therefore, that Mayr and Futuyma may have been a bit hasty in their implicit claim that evolutionary biology be confined to the realm of ultimate causes at the exclusion of mechanistic ones. Contrary to this view, I have shown that—on *at least some* philosophical characterizations of mechanisms, and with regard *to at least some* of the central processes of evolutionary

biology—there is room for affirmative answers to both the metaphysical and epistemological questions. And thus, there may indeed be an important place for mechanisms in evolutionary biology.

Notes:

[1] The distinction between metaphysical and epistemological questions regarding the appropriateness of mechanistic thinking as applied to a given domain echoes one made by Glennan and Illari in Chapter 7 of this volume.

[2] See Illari and Williamson (2012) and Chapter 1 for arguments that these requirements can be expressed by a common conception of mechanism that applies widely across the sciences.

[3] See Skipper and Millstein (2005) for an argument to this effect.

[4] See Ariew (2007) for a nice survey of some of these issues.

[5] Not everyone agrees that natural selection is a causal process (cf, especially Matthen and Ariew 2002). Following Millstein (2006) and more recently Ramsey (forthcoming), I presume that natural selection can be given a causalist interpretation.

[6] More detailed philosophical analyses of drift vary greatly. Some argue that drift cannot be distinguished from selection and so cannot be conceived of as a distinct evolutionary process (for instance, Matthen and Ariew 2002, Matthen 2009). And there are some (for example Millstein 2002, 2008) who argue that drift is distinct from selection by virtue of the kind of process it is (indiscriminate as opposed to discriminate sampling of a population).

[7] Example comes from Roberta Millstein (1996, S15)

[8] As with natural selection, there are some who deny that drift is causal. See especially Lange (2013) as an example.

[9] See especially Craver (2007) for a detailed exposition of this advantage.

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