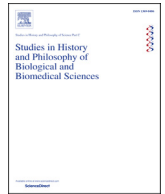Dear Author,

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Natural selection and mechanistic regularity

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A B S T R A C T

In this article, I address the question of whether natural selection operates regularly enough to qualify as a mechanism of the sort characterized by Machamer, Darden, and Craver (2000). Contrary to an influential critique by Skipper and Millstein (2005), I argue that natural selection can be seen to be regular enough to qualify as an MDC mechanism just fine—as long as we pay careful attention to some important distinctions regarding mechanistic regularity and abstraction. Specifically, I suggest that when we distinguish between process vs. product regularity, mechanism-internal vs. mechanism-external sources of irregularity, and abstract vs. concrete regularity, we can see that natural selection is only irregular in senses that are unthreatening to its status as an MDC mechanism.

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1. Introduction

Nature tends to preserve those traits that afford their possessors the greater chance to survive and reproduce, and it tends to reject those that do not. The result is that species become increasingly matched to their respective environments; they become exquisitely adapted over time. In its most basic form, this is natural selection.

For natural selection to occur, Darwin argued that three, and only three, conditions must obtain. (1) There must be *variation* among members of a population (e.g., some wolf pups are born with faster running abilities than others); (2) These variations must be *heritable* (e.g., those faster wolf pups will tend to have faster offspring); and (3) There must be, what he termed, a 'struggle for existence' (e.g., there must be more wolf pups in a given generation than can survive).[1] Given, these three preconditions, it follows that those individuals with advantageous variations will tend to survive and reproduce while those with deleterious ones will tend to die off.

As helpful as the above basic characterization of natural selection is, it leaves open the answer to a question that has long interested philosophers of biology: what kind of a thing is natural selection? How should we represent what sort of process it is?

Indeed, Darwin himself seems to have been unsure about the answer to this question. In *the Origin of Species*, He referred to natural selection in a myriad of ways: an "action" (1859/1964, 90, 108, 129, 133, 211), a "doctrine" (5, 95), a "means" (6, 246), a "power" (43, 109, 205, 238, 454), a "theory" (237, 245, 281, 320, 325, 338, 345, 460, 462, 472, 474, 478), a "principle" (80, 95, 116, 127, 188, 206, 239, 475), and a "process" (93, 104, 109, 179, 203, 235, 280, 350).[2] In part due to Darwin's own apparent indecision in the *Origin,* contemporary philosophers of biology have been hard at work arguing for a more precise understanding of what kind of a process natural selection actually is. Some have argued that natural selection is best understood as a *force* (Sober, 1984; Stephens, 2004, 2010); some that it is a *purely statistical trend* manifesting in natural histories (Matthen & Ariew, 2002, 2009; Walsh, 2004); some that it results from *causal processes operating at the individual level* (Glennan, 2009); some that natural selection is a *causal process, but one that necessarily operates at the population level* (Millstein, 2013), and some that it is a multi-staged *mechanism characterizable on both the individual and population level* (Barros, 2008). Each of these positions has outspoken critics.

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[1] There is debate as to whether (3) is really required for natural selection. But nothing I go on to say turns on this debate.

[2] See Havstad (2011) for the source of this research.

In what follows, I hope to show that there are some good reasons for understanding natural selection as a mechanism of the sort characterized by Machamer, Darden, and Craver (2000) (henceforth 'MDC mechanism'). And by appealing to some important and heretofore unrecognized distinctions regarding mechanistic regularity and abstraction, natural selection can be seen to escape at least one of the serious problems set forth against it counting as such: that it fails to operate regularly enough.

I will proceed in the following steps. In Section 2, I offer some prima facie reasons for understanding natural selection as a mechanism. In Section 3, I outline one influential argument against natural selection as an MDC mechanism: that it fails to meet the regularity requirement set forth in the MDC characterization of mechanism. In Section 4, I draw three distinctions regarding mechanistic regularity and show that natural selection only fails to be regular in ways that should be seen as unthreatening to its status as an MDC mechanism. First, I distinguish between process and product regularity and argue that the Skipper and Millstein critique only shows natural selection to be product irregular not process irregular, but there are good reasons for thinking that process regularity should matter more for MDC mechanisms. Second, I distinguish between mechanism-internal and mechanism-external sources of irregularity and argue that the sources of the irregularities associated with natural selection constitute unthreatening mechanism-external sources of irregularity. And third, I distinguish between abstract and concrete regularity and show that how regularly we conceive of natural selection depends crucially on the degree of abstraction we employ to schematize it. When schematized in a highly abstract manner, I contend, natural selection can be seen to operate regularly enough for certain legitimate explanatory contexts. I conclude in Section 5 by suggesting that this debate evinces an important general point about grounding mechanistic explanations in a complex, contingent biological world.

2. Prima facie reasons for natural selection as an MDC mechanism

The search for mechanisms pervades the life sciences. Examples abound. Endocrinologists search for the mechanisms by which specialized cells, tissues, or organs transport hormones throughout the body (Mizoguchi, Kamimura, Kiuchi, & Kataoka, 2015); geneticists search for gene silencing mechanisms that play important roles in suppressing genes that are required in only certain contexts (Kim, Ma, & Cerutti, 2015); plant scientists study the mechanisms by which plants signal the presence of microbial pathogens (Wu, Shan, & He, 2014). There are thousands more examples.

Recently, much work in the philosophy of science has been devoted to understanding *what* exactly it is that scientists look for when they search for mechanisms and *how* these mechanisms are meant to function in scientific reasoning. One now widely accepted philosophical characterization of mechanism was put forward in Machamer, Darden, and Craver's oft cited paper "Thinking about Mechanisms" (Machamer et al., 2000).

MDC: Mechanisms are entities and activities organized such that they are productive of regular changes from start or set-up to finish or termination conditions. (Machamer et al., 2000, 3)[3]

On this characterization of mechanism, the beating heart clearly qualifies as a mechanism. It is composed of entities (aorta, ventricles, arteries, and so on) and activities (beating, pumping etc.) that are organized to produce regular changes (blood circulation) from the start of an animal's life to its end. This analysis applies equally well, mechanists have argued, to the molecular mechanisms of DNA replication and protein synthesis (Darden, 2006, 2008; Darden & Craver, 2013) as it does to the processes of synaptic transmission in the brain (Andersen, 2012; Bogen, 2005; Craver, 2007) as it does to the ubiquitous maintenance of circadian rhythms in all living beings (Bechtel & Abrahamsen, 2013[4]).

It would be nice if natural selection could also be characterized as an MDC mechanism. As is now widely recognized, mechanistic explanation—at least in the life sciences—affords several important advantages over the once-received deductive-nomological (D-N) account of scientific explanation.[5] On the D-N model, scientific explanation proceeds by identifying at least one law of nature, specifying the explanandum event's precise initial conditions, and showing that, given the laws and the initial conditions, the event had to have occurred. Mechanistic explanation, on the other hand, explains a given event by describing the causal mechanism that produced it.[6] There are several advantages of the latter over the former. Mechanisms, unlike laws of nature, are comfortably understood as physically existing in the world, so are more easily investigable by empirical science. Furthermore, generalized mechanism schemas allow us to explain more than the specific explanandum instance under examination; mechanisms support generalizations and ampliative inference in scientific explanation much the same way that laws once did. However, unlike with laws-based accounts, the mechanisms underlying these generalizations and ampliative inferences need not be exceptionless, necessary, or contain universals. Mechanistic explanation also matches intuitions, originally highlighted by Salmon,[7] that giving a scientific explanation must involve laying bare the causal structure of the world: locating a puzzling phenomenon in its causal nexus. And, as a matter of coherence with current practice, biologists *actually do* engage in searching for and describing mechanisms.

Prima facie, natural selection seems amenable to a mechanistic analysis. There are several reasons for this. For one, central to the notion of mechanism is the idea that mechanisms, among other things, are *set up for something*; mechanisms are productive of some general *phenomenon*. This feature of mechanisms is highlighted in many of Stuart Glennan's early works. In his 1996 article, "Mechanism and the Nature of Causation", Glennan points out the "one cannot even identify a mechanism without saying what it is that the mechanism does" (Glennan, 1996, 52). Put another way, whatever else a mechanism is, it at least needs to have a function; it needs to be set up to do something. Here, it seems natural selection fares quite well. Quite clearly, natural selection is a system *for* something: it is that which brings about adaptation.[8]

Another central feature of mechanisms is that they support reductionist explanation. That is, one of the reasons why mechanisms are so explanatorily useful is that they can be decomposed to their component parts and operations, and by doing so, crucial

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[3] Similar characterizations have been put forward by Glennan (1996, 2002) and Bechtel (2006), but MDC's characterization has received the most attention in the literature—so theirs is the one on which I focus my discussion.

[4] It should be noted that Bechtel and Abrahamsen criticize the MDC characterization for implying that mechanisms must be linear. The circadian example is cyclic, with feedback. However, more recent developments in the mechanisms literature have amended the MDC characterization to better suit feedback mechanisms (cf. Craver & Darden, 2013).

[5] Cf. Hempel (1942, 1965).

[6] See Bechtel & Abrahamsen (2005) and Craver (2007) for detailed treatments of the nature and norms of mechanistic explanation.

[7] Cf. Salmon (1984).

[8] In making this point, I don't mean to attribute any problematic teleology to natural selection. It isn't that natural selection is directed towards any specific goal (e.g., to approach perfection or the like). Rather, I mean only to draw attention to the fact that natural selection has a *function* (in the Cummins [1975] sense of causal-role function). It is that which brings about adaptation.

insight into the *why* the phenomenon in question regularly occurs can be achieved.[9] This feature of mechanistic explanation gets emphasized in Bechtel and Richardson's seminal 1993 book, *Discovering Complexity: Decomposition and Localization as Strategies in Scientific Research.* In their discussion of (what they call) 'complex localization', Bechtel and Richardson 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 & Richardson, 1993, 125). At first glance, there is a clear sense in which natural selection fits into this reductionistic explanatory framework: it seems decomposable into its component parts as well as the tasks these parts perform. Natural selection, we might think, is composed of entities (e.g., populations of organisms with varying traits and a critical environmental factor) and activities (e.g., interaction of organisms with the environment and differential reproduction). In this respect, natural selection fits in quite well with the reductionistic approach to scientific explanation afforded by the mechanistic approach.

3. The regularity critique

Despite these prima facie reasons for understanding natural selection as an MDC mechanism, some authors have resisted such a characterization. Among its primary opponents are Skipper and Millstein (2005) and Havstad (2011).[10] These authors appeal to a number of reasons why the MDC characterization of mechanism fails to 'get at' natural selection. In order to achieve sufficient depth of analysis, however, I focus on just one of these arguments: the part of Skipper and Millstein's critique in which they argue that natural selection fails to be *regular* in the way the MDC require.[11]

On the MDC characterization of mechanism, recall, the constituent entities and activities of a mechanism must be organized such that they are productive of *regular* changes from start or set-up to finish or termination conditions. But what does this mean? Unfortunately, MDC do not say much about how we should understand this appeal to regularity. The only drop of clarification they supply is that "[m]echanisms are regular in that they work always or for the most part in the same way under the same conditions" (MDC, 3). As we will see, this remark can be interpreted in a number of ways. But as Skipper and Millstein see it, no matter how we interpret the regularity condition, it makes no sense to think of natural selection as meeting it. They write, "With respect to regularity on MDC's account, recall that '[m]echanisms are regular in that they work always or for the most part in the same way under the same conditions.' However, we see natural selection as probabilistic" (Skipper & Millstein, 2005, 342). On Skipper and Millstein's view, the probabilistic nature of natural selection precludes it from meeting the regularity requirement set forth by MDC.

To motivate this claim, Skipper and Millstein ask us to consider Darwin's finches. We are to suppose there is a collection of one hundred equal-sized populations of finches with the same distribution of beak lengths, all located in the same environment. We are to suppose, further, that these finches engage in their usual survival and reproduction activities. Skipper and Millstein ask us then to imagine examining the distributions of beak length in the subsequent generation. They write,

> In this thought experiment, we would not expect the same distributions of beak length in each of the one hundred populations. In some of the populations longer beaks may prevail, in others, shorter beaks may prevail, and in some populations the distribution may be roughly equal. This is due to the fact that the 'petty influences' (Galton's phrase) on each of the populations is different. As a result, the finches that are the fittest may not in fact have the greatest representation in the subsequent generation, due to the vagaries of disease, predators, lack of success in finding food, or simply being 'in the wrong place at the wrong time'. (Skipper & Millstein, 2005, 343)

This thought experiment is supposed to prompt the reader's intuition that natural selection does not work always or for the most part in the same way. There is something inherently probabilistic about natural selection. In a collection of 100 populations of finches with identically distributed beak lengths—all of which engage in their normal survival and reproduction activities—we simply should not expect the evolutionary outcomes of each of these populations to be identical in the subsequent generation. Why?— because there are just too many factors involved in deciding the eventual evolutionary outcome (e.g., disease, predation, or bad luck) that might go slightly differently in each respective evolutionary iteration. Indeed, it would be something of a miracle if all of these 100 populations ended up with identical beak-length distributions after only one generation. The fact that there is virtually no way these populations would end up with identical beak length distributions means that natural selection simply *does not* operate with machine-like regularity. To use MDC's terminology, natural selection *does not* work always or for the most part in the same way. Rather, it operates probabilistically.

Skipper and Millstein do admit that "…it may be true that given *identical* conditions in each of the one hundred populations we would get exactly the same distribution in each population…" (Skipper & Millstein, 2005, 343). But they reply to this counter-suggestion in two ways. First, citing Brandon and Carson (1996), they allow that genuine indeterminism might be at play (presumably at the level of spontaneous mutation), such that we might get different distributions even with identical starting conditions. And second, they suggest that even if there is no genuine indeterminism, "…it is hard to believe that this [regularity given identical conditions] is what MDC have in mind given their concern to describe mechanism schemata (the operation of the Na+ channel) that can be instantiated in particular cases (e.g., the depolarization of a specific nerve cell)" (Skipper & Millstein, 2005, 343). The point here seems to be that, whatever MDC might have meant when they required of mechanisms that they 'work always or for the most part in the same way under the same conditions', it must not have been merely that mechanisms do the same thing given *identical* background conditions. Indeed, mechanism schemata (e.g., membrane depolarization and protein synthesis) are supposed to capture a generic regularity that covers all of the relevant instances of these schemata. And of course these instances would be situated in differing background conditions.

Thus, they conclude,

> The bottom line is that natural selection is not regular in the way that MDC require (presumably because natural selection is not regular in the way the mechanisms that MDC discuss are, such

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[9] Many in the mechanisms literature emphasize the additional benefit that explanation via the identification of mechanisms also supplies information about how and where to *intervene* on a system (see especially Waskan [2008] for this point). As we will see, however, the extent to which this is the case depends on the degree of abstraction employed when schematizing the mechanism. The less abstract a mechanism schema is, the more detail will be supplied on how and where to intervene.

[10] Aspects of each of these authors' argument are also echoed in Kalkman (2015).

[11] In addition to failing to meet the regularity requirement, Skipper and Millstein also deem natural selection not to have met the organization and productive continuity criteria set forth by MDC. Because I intend my discussion of the regularity critique as it applies to natural selection as an illustration of a deeper point about the prospects of explaining probabilistic phenomena mechanistically, I limit my discussion to regularity.

as DNA transcription and protein synthesis). (Skipper & Millstein, 2005, 342−343)

In argument form, the regularity critique offered by Skipper and Millstein looks like this.

> P1. MDC requires that mechanisms behave regularly (i.e., they 'work always or for the most part in the same way under the same conditions').
> P2. But natural selection operates probabilistically—where this can be couched either in terms of:
> (i) 'petty influences' which we should expect to differ across evolutionary iterations or
> (ii) genuine indeterminism operating at the molecular level
> P3. Given either disjunct of (P2), natural selection cannot meet the regularity requirement set forth by MDC.
> C1. Therefore, natural selection cannot be an MDC mechanism.

Let us call this argument the *Skipper and Millstein (S-M) Regularity Critique*.

4. Dismantling the S-M regularity critique

As straight-forward as it seems, I am going to show this argument ultimately fails.

Here is my general strategy. I will draw three distinctions regarding mechanistic regularity and show that natural selection only fails to be regular in ways that are unthreatening to its status as an MDC mechanism. First, I distinguish between process and product regularity and argue that the S-M Regularity Critique only shows natural selection to be product irregular not process irregular, but it is process regularity that should matter more for MDC mechanisms. Second, I distinguish between mechanism-internal and mechanism external sources of irregularity and argue that (i) the 'petty influences' cited by Skipper and Millstein constitute an unthreatening mechanism-external source of irregularity, and (ii) random mutations, if there are any, merely constitute unthreatening stochastic inputs or background conditions to the mechanism. And third, I argue that (irrespective of the determinism issue) how regularly we conceive of natural selection depends crucially on the degree of abstraction we employ to schematize it. And when schematized in a highly abstract manner, natural selection can be seen to operate regularly enough for certain legitimate explanatory contexts.

4.1. Process vs. product regularity

When we say of a particular mechanism that it behaves regularly, we might mean one of two things. First, we might mean that the constituent entities and activities of a mechanism behave in roughly the same way each time the mechanism operates. Or we might mean that the output of a mechanism is roughly the same each time it operates.[12] The first sense, I call *process regularity*. And the second, *product regularity*.

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[12] Drawing from Glennan (1992) and Barros (2008, 311) makes a similar distinction in his own response to Skipper and Millstein. However, since his argument for natural selection as a mechanism turns on whether the outcomes of natural selection can be predicted with a higher than 50% rate of accuracy, he limits his discussion to the regularity of outputs. I think this approach is mistaken. First, I take the reasons for requiring mechanisms to be regular to have less to do with the ability to make successful predictions and more to do with whether a mechanism can support ampliative inference in explanation. On the latter desideratum, mechanisms with some degree of process regularity does just fine. But even if this were not the case, I am not as convinced as Barros is that the outcomes of evolution can be predicted as accurately as he suggests.

Imagine a lottery ball machine like the ones you have seen on television after the evening news. Here we have a machine that does the same thing each time it operates: it pumps air into a big, clear, globe thereby jostling the numbered lottery balls until it is time for them to be spit out and read aloud by the lottery official. But despite working the same way every time it gets used, it produces numerical results with no discernable pattern. Indeed, that is its purpose: to produce random numbers. Now contrast the lottery ball machine to one of those self-operating vacuum cleaner devices. These vacuum robots, provided they are not impeded, produce the same outcome each time they operate (e.g., a floor free of dust-bunnies), but to do so, they follow a very different path each time. Like an ant in search of food, the vacuum robot never follows the same precise route. These are simplistic, idealized examples to illustrate the following basic distinction. Sometimes mechanisms operate regularly in the sense of regularly producing the same outcome albeit via operating differently each time; these mechanisms are product regular.[13] And sometimes mechanisms operate regularly in the sense that they do the same thing, albeit in a way that produces different outcomes each time; these mechanisms are process regular.

I suggest that, when this distinction is made explicit, the S-M Regularity Critique loses much of its force. Here is my argument.

> P5. Skipper and Millstein's finch example only shows natural selection to be product irregular, not process irregular.
> P6. But product irregularity alone is unthreatening as to whether a causal process qualifies as an MDC mechanism.
> C2. So, Skipper and Millstein's finch example only shows natural selection to be irregular in a way that is unthreatening to its status as an MDC mechanism.

Both (P5) and (P6) need support to show that (C2) follows.

So let us turn to (P5). I submit that, while Skipper and Millstein's finch example might show that natural selection is product irregular, it far from shows that it is process irregular. Recall that, according to Skipper and Millstein's finch example, we are to imagine 100 populations of finches each participating in their normal survival and reproduction activities. With these conditions in place, they write, "We would not expect the same distributions of beak length in each of the one hundred populations. In some of the populations longer beaks may prevail, in others, shorter beaks may prevail, and in some populations the distribution may be roughly equal" (Skipper & Millstein, 2005, 420). There are two important features to note in their thought experiment: (i) the survival and reproductive activities of the finches *are held fixed*. And (ii) it is the distribution of beak lengths *at the end of each evolutionary iteration* that we should expect to vary. Even if we take their example at face value, it only shows that the precise *outcomes* of natural selection operating on each population of finches will vary across evolutionary iterations. It does not show that the processes of natural selection work differently each time. Indeed, on their own example, the constituent entities and activities of the selection process (e.g., same finches and same survival and reproductive activities) are held fixed. So Skipper and Millstein's finch example only shows that natural selection is product irregular, not process irregular.

Of course, it remains to be shown whether any sense can be made of the claim that the processes of natural selection *are* the same across its instances. Whether this can be done depends on

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[13] This notion of product regularity absent process regularity is similar to the phenomenon of robustness as it applies to biological outcomes. See Brigandt (2015) for a detailed discussion of how mechanistic explanation relates to biological robustness.

several complexities having to do with typing and abstraction which I take up in a later section. For now, however, I am content to have shown that Skipper and Millstein's finch example only indicates that natural selection is product irregular not process irregular: (P5).

Which brings us to (P6). Why think that mere product irregularity is unthreatening to MDC mechanisms? To begin to answer this, recall that MDC initially formulated their account of mechanism explicitly to target molecular biology. They write, "Our goal is to sketch a mechanistic approach for analyzing neurobiology and molecular biology…" (Machamer et al., 2000, 2). As their central example from molecular biology, MDC focus on protein synthesis. So let us see if protein synthesis is more aptly characterized as product or process regular. If protein synthesis is more aptly characterized as process regular rather than product regular, then this would be significant support for the claim that process regularity is the kind of regularity that matters more for MDC, and therefore, that product irregularity alone should not be taken to be threatening to the mechanistic status of a causal process. At the very least, it would undermine Skipper and Millstein's conclusion that "natural selection is not regular in the way the mechanisms that MDC discuss are, such as DNA transcription and protein synthesis" (Skipper & Millstein, 2005, 342–343).

Consider the protein synthesis mechanism. Protein synthesis takes place when DNA is transcribed into messenger RNA, which is translated into protein (see Fig. 1).

The process of protein synthesis fits nicely within the mechanistic framework. It is composed of entities: DNA (deoxyribonucleic acid in the shape of a double helix), RNA polymerase (a core enzyme), and mRNA (ribonucleic acid, which carries information to the protein synthesis machinery of the cell). And it has activities associated with it: *binding* of RNA polymerase to the DNA's promoter sequence, and *elongation* (when the RNA polymerase traverses the template DNA strand, using base pairing complementarity with the DNA template to create an RNA copy). Furthermore, protein synthesis has set-up conditions (e.g., the presence of DNA in a living organism), start-up conditions (e.g., initiation of transcription), and termination conditions (e.g., the successful synthesis of a brand new protein). What is important for the purposes of this discussion, however, is this: *protein synthesis is more aptly considered process regular rather than product regular.* The reason is simple. Although protein synthesis always includes the same processes (e.g., transcription from DNA to messenger RNA and ordering of the amino acids in the polypeptide chain), the proteins synthesized by these processes—the *output* of the protein synthesis mechanism—vary significantly depending on where the proteins are in the body and what they do. Using the very same processes of transcription, and translation, our bodies synthesize contractile proteins for contracting muscles, enzymatic proteins for catalyzing biochemical reactions, defensive proteins for fighting disease, hormonal proteins that act as messengers, storage proteins for storing amino acids and metal ions needed in the body, and the
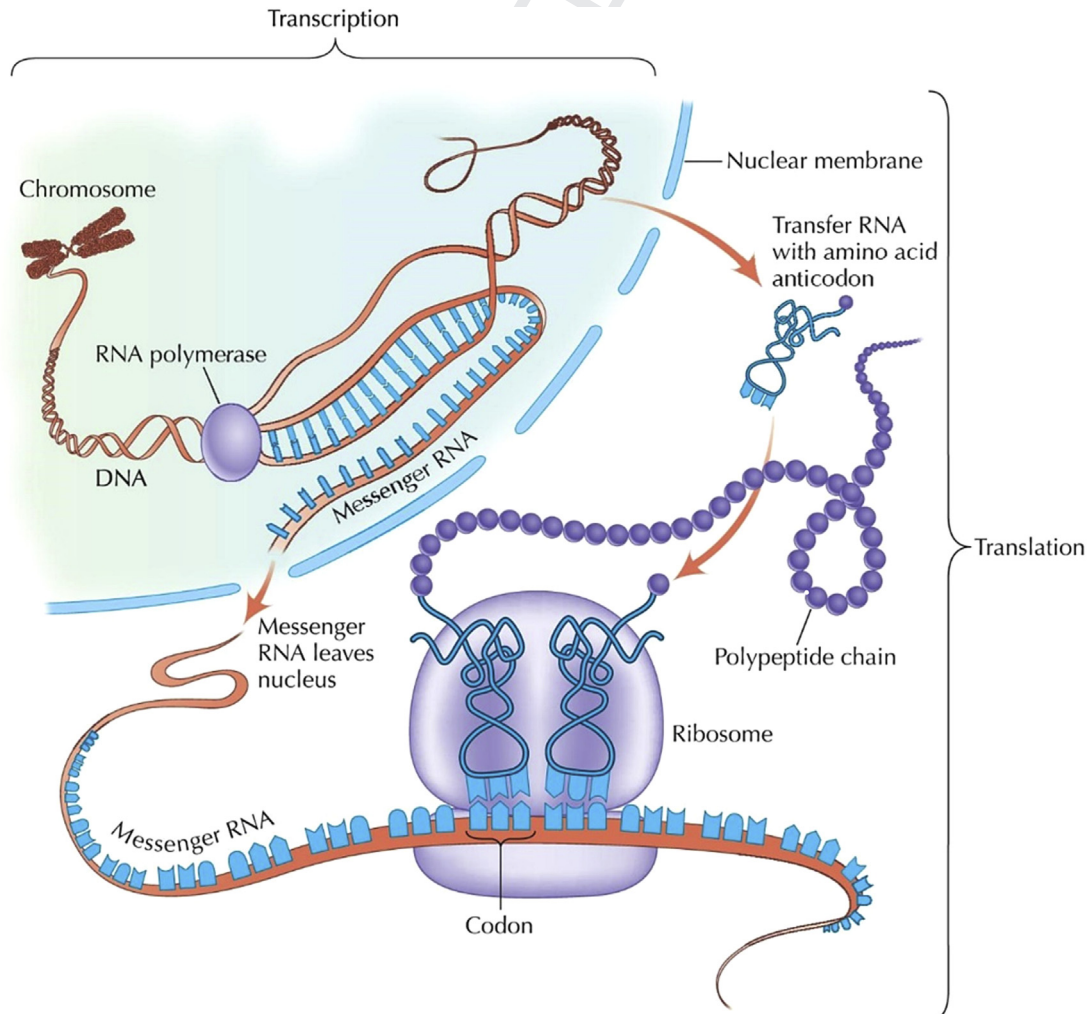

Fig. 1. Protein synthesis schema (from Barton, Briggs, Eisen, Goldstein, & Patel, 2007, 50; reproduced by permission).

list goes on. Here is the upshot. Although the processes of protein synthesis 'work always or for the most part in the same way', the output of protein synthesis differs greatly.[14] And since protein synthesis is one of MDC's paradigm targets for mechanistic analysis, it is reasonable to conclude that process regularity is what matters when it comes to qualifying as an MDC mechanism; product irregularity alone is unthreatening. Hence, (P6).

So putting premises (P5) and (P6) together, we can see that Skipper and Millstein's finch example only shows natural selection to be irregular in a way that is unthreatening to its status as an MDC mechanism: (C2).

4.2. Internal vs. external sources of irregularity

In this section, I draw another distinction regarding mechanistic regularity that, when appreciated, shows a different weakness of the S-M Regularity critique.

When a particular mechanism fails to behave regularly, there are two kinds of ways this can occur: because of inhibitory sources either *internal* or *external* to the mechanism.[15] Think for a moment about your household toaster. Suppose your toaster fails to regularly produce adequate toast because, on many of the occasions that you use it, you are also using so many other appliances in your kitchen that you blow a fuse. This would be a mechanism-external source of irregularity. The reason your toaster fails to achieve its output conditions in a regular fashion is not due to anything *within* the mechanism; it is due, rather, to inhibitory conditions in its surrounding. If, on the other hand, your toaster fails to produce toast because there is a faulty connection in its wiring, this would be an internal source of irregularity.

This distinction centers around the notion that certain entities and activities are internal to a mechanism vs. external to it. As such, something needs to be said about how we are meant to draw boundaries around mechanisms. When it comes to drawing

boundaries around mechanisms, however, things are more complicated in the biological world than they are in the realm of man-made machines such as toasters. As Craver puts it,

> […] machines often have easily identifiable parts contained within well-defined boundaries. We look into a clock and readily identify the pendulum, the counterweights, its ratchets and gears. The parts of neural mechanisms are in many cases not so visible, not so readily distinguished from their surroundings; in some cases, they are widely distributed and dynamically connected, defying any attempts to localize functions to particular parts. (Craver, 2007, 4).

Mechanisms operating in the natural world often defy simplistic machine-like analogies. They often do not come in easily identifiable packages. And definitive boundaries are often hard to draw around them. However, this does not mean that there is no way to individuate biological mechanisms. A mechanism is composed of just those entities and activities that, when organized in the relevant way, produce the phenomenon one is interested in explaining. As Craver points out, if one is interested in explaining the functional capacity of a car to drive, one appeals to its engine (which can itself be decomposed into its constituent mechanisms). But one does not appeal to the car's hubcap to do this explanatory work; the car would still drive without a hubcap. In other words, entities and activities are constitutive of a given mechanism just in case the mechanism could not serve its function without them. These constitutive entities and activities are internal to the mechanism. All others are external to it.

So having become clearer about mechanism boundaries and individuation, let us return to this idea of mechanism-internal vs. mechanism-external sources of irregularity. Consider a few illustrative biological mechanisms with sources of irregularity of both of these sorts operating in the natural world. Synaptic transmission can be disrupted by a variety of factors outside of the synaptic mechanism itself. In Alzheimer's disease, patients have an abnormal aggregation of a microtubule-binding protein called 'tau'. These proteins form long, thin polymers that wind around one another to form what are called *neurofibrillary tangles* which accumulate in neuronal cell bodies, dendrites, and axons. These tangles, when present, interfere with axonal transport of electrical signals in the brain (Kandel, Schwartz, Jessell, Siegelbaum, & Hudspeth, 2013, 78). This is an example of mechanistic irregularity due to inhibitory features (e.g., neurofibrillary tangles) *outside* the mechanism: the tangles are not required for the ability of the mechanism to carry out its function. However, synaptic transmission can also fail to occur due to problems *inside* the mechanism. Normal conduction of nerve signals in the brain can be disrupted, for example, by defects in myelin proteins—proteins that insulate the very axons that carry electrical signals. This can result in serious disturbances in sensory and motor function (Kandel et al., 2013, 91). Since axons are among the entities without which the synaptic transmission mechanism would be unable to serve its function, this should be seen as a mechanism-internal source of irregularity. Another example is DNA replication. DNA can fail to replicate fidelously because its purine base (internal to the mechanism) is changed by the repositioning of a hydrogen atom, altering the hydrogen bonding pattern of that base resulting in incorrect base pairing during replication. And since a particular purine base for a particular strand of DNA is internal to the DNA replication mechanism, this should be seen as a mechanism-internal source of irregularity. However, there may also be instances where DNA replication fails to occur perfectly due to outside inhibitory forces (e.g., epigenetic methylation).

I suggest that the sort of irregularity attributed to natural selection by Skipper and Millstein is more aptly understood as having

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[14] An objection might arise here. If we look close enough at the processes giving rise to these different proteins, the objector might suggest, we would see that in fact the processes involved in producing different proteins are just as different as the proteins that they produce. If they were the *exact same* processes, then they would contain the same entities and activities, and would thereby *have to* end up producing the same proteins. Which is to say, it simply is not the case that the very same processes constituent of the protein synthesis mechanism lead to variable protein outputs. When there is variance in the mechanism output, there must also be variance in the processes producing these outputs.

My first response to this objection is to agree that differing proteins require, in some sense, different processes to have produced them. However, I would suggest (as I will go on to do in Section 4.2) that these differences might be accounted for by appeal to the variance in *inputs* required for synthesizing the different proteins. So while the protein synthesis mechanism might operate on different segments of DNA (e.g., to produce enzymatic proteins vs. hormonal proteins), the processes (e.g., translation and transcription) working on those differing segments may well still be the same. And if the processes working on these varied inputs are still the same, then protein synthesis still counts as an instance of process regularity in the absence of product regularity. My second line of response is to point out that issues about sameness across processes are indelibly tied up in issues about typing (e.g., of processes and products) as well as abstraction (i.e., the amount of detail included in our descriptions of these processes and products). If we are talking about a type of process characterized at a high level of abstraction, these processes may well count as the same. At a lower level of abstraction, they may not. I deal with this issue in more detail in Section 4.3.

[15] Pemberton & Cartwright (2014) make a similar observation when they acknowledge that how law-like a nomological machine behaves can be sensitive to "factors external to the start arrangement of the machine" (Pemberton & Cartwright, 2014, 1749). In her discussion of mechanistic regularity as it relates to ecology, Holly Andersen (2012) also seems to acknowledge this distinction: "Ecologists may know the start-up conditions for ecological succession in a given ecosystem, involving disturbances to habitat or creation of new habitat. Yet not all start-up conditions lead to the termination conditions of climax communities, because there are a range of external factors governed by chance that could prevent the mechanism from working" (Andersen, 2012, 422).

a mechanism-external source rather than a mechanism-internal one. And this is important because I also contend that mechanism-external sources of irregularity should not be seen as threatening to a causal process's status as an MDC mechanism. Here is my argument.

P7. Skipper and Millstein's finch example is meant to show that natural selection fails to behave regularly because of:
 (i) 'petty influences' which we should expect to differ across evolutionary iterations or
(ii) genuine indeterminism operating at the molecular level
P8. Both (i) and (ii) constitute mechanism-external sources of irregularity.
P9. However, irregularity entirely due to mechanism-external inhibiting factors should not be seen to threaten a purported mechanism from qualifying as an MDC mechanism.
C3. So, Skipper and Millstein's finch thought experiment should not be seen to threaten an understanding of natural selection as being regular enough to qualify as an MDC mechanism.

(P7) is just a restatement of the second premise of the S-M Regularity Critique summarized in Section 3. But (P8) and (P9) require support to show that C3 follows.

What makes petty influences and random mutations mechanism-external sources of irregularity as (P8) suggests they are? Once again, drawing the boundaries around a purported natural selection mechanism is not as simple as with clocks and toasters. But, nevertheless, we can get a sense of which are the entities and activities without which natural selection would be unable to serve its function. And in doing so, we can get a sense of which are the entities and activities that are internal to the natural selection mechanism. Least controversially, a given natural selection mechanism requires a population of organisms with variation in fitness-relative traits. In our example, these are the finches with varying beak-lengths. But for natural selection to occur, there must also be some kind of critical environmental factor. In the case of the finches, this is usually presented as seeds with differing husks and seeds which are located in places such that certain of the finches' beaks are better suited to forage for them, and certain of the finches' beaks are worse suited for foraging for them. Outside of this critical environmental factor, the rest of the environment, I submit, should not be taken to be constitutive of the token natural selection mechanism. Like the car's hubcap, the details of the non-critical environmental features are not required for natural selection to execute its function. As Darwin demonstrated, all that is required for natural selection to produce exquisite adaptations is for there to be variation among a population, heritability of fitness-relative traits, and struggle for existence (which we can extend to engagement with a critical environmental factor). All else is external to the mechanism. So, on this line of reasoning, with the exception of the critical environmental factor, the rest of the finches' environment should not be seen to be constitutive of the token natural selection mechanism. However, and this is key, the 'petty influences' cited by Skipper and Millstein as the source of irregularity in the finch example belong in just this class of non-critical environmental features which are not constitutive of the token mechanism. If certain finches die because of disease, predators, bad luck, or just being in the wrong place at the wrong time, these are the result of non-critical features of the finches' environment. So these petty influences are mechanism-external sources of irregularity.

A similar, but slightly different, story can be told about (so-called) random mutation. Errors made during DNA replication which are the result of spontaneous molecular processes, no doubt, play a vital role in natural selection. Indeed, they constitute the source of the variation on which natural selection operates. However, I contend that this random mutation (if it really is random) is best conceived of as a stochastic input or background condition to the natural selection mechanism. To illustrate this notion of stochastic input, remember your toaster again. One reason it might fail to perfectly produce identical toast each time it operates is that on some mornings you feed it plain white bread, while on other mornings you feed it a bagel. Although there is a sense in which the resulting toast is irregular, it is not due to any irregularity internal to the toaster mechanism; it is merely the result of having been fed different inputs. This is the same situation with random mutation. While random mutation itself may be conceived of as irregular in a certain sense, *what natural selection does with it may not be.* Once random mutations occur, and some finches are born with slightly longer or slightly shorter beaks, natural selection will act of those variations in a regular way. Namely, it will tend to preserve the finches whose beaks are best suited to reach the seeds. The bottom line is that, whether a particular mutation occurs in a particular finch may be inherently probabilistic (even random), but what natural selection does with it may follow a regular pattern.

So if the source of the irregularity attributed to natural selection is conceived as (i) Galton's petty influences, these are non-critical features of the natural selection mechanism's environment and are thus mechanism-external. And if the source of irregularity is conceived as (ii), random mutation, this is merely a stochastic input to the mechanism, so is also mechanism-external. Hence (P8).

But what about (P9)? Why think that mechanism-external sources of irregularity should not be seen to inhibit a process from qualifying as an MDC mechanism? Let us think about toasters again. Suppose, as before, that my toaster fails to regularly produce adequate toast because, on many of the occasions that I use it, I am also using so many other appliances in my kitchen that I blow a fuse. Let us ask whether a failure of regularity of this sort should be seen to undermine the toaster's status as a mechanism. I do not see any reason why it should. Here is the general point. Even the most regular mechanism can be made to fail to produce its outcome if it is inhibited externally. Synaptic transmission, protein synthesis, DNA replication, all considered highly regular biological mechanisms, can be made to fail to occur by the right kind of inhibitory interference (e.g., if a test subject is killed). What matters for the mechanistic explanatory framework is that mechanisms function with some degree of regularity—*when free from inhibitory influence.* The only point I wish to emphasize is that sensitivity to external sources of irregularity should not, by itself, be seen to threaten a process should count as a mechanism. And if this is so, then external irregularity should not, by itself, constitute a reason for rejecting a process from counting as an MDC mechanism.

Here is the upshot. According to the S-M regularity critique, natural selection fails to be regular because of either Galton's petty influences or because of random mutation. However, both Galton's petty influences and random mutation are mechanism-external sources of irregularity. But mechanism-external sources of irregularity alone should not be considered threatening to the status of a causal process as an MDC mechanism. So the S-M Regularity Critique, once again, fails to demonstrate that natural selection is not regular enough to be an MDC mechanism.

4.3. Abstract vs. concrete regularity

There is one more distinction to make which, when appreciated, shows yet another way to dismantle the S-M regularity Critique. A mechanism might be thought to behave regularly in an abstract sense or in a concrete sense. To explain what this means, however, we first need to think about mechanism types versus tokens. And then we need to think about abstraction.

Like all natural kind concepts, mechanisms come in types and tokens. Andersen (2012) does a particularly nice job of illustrating this. Here is what she says,

> [T]he term "mechanism" may apply to either a type or a token. On one hand, the term can be used to pick out a single individual causal chain in the world. When a particular neuron fires on a given occasion, a mechanism led to that firing. On the other hand, the term is often used to indicate a type of causal chain, one that could recur on multiple instances: when a neuroscience textbook describes the mechanism for neuron firing, it does not describe a single instance, but rather a type of causal chain that presumably occurs on many occasions. In this way, mechanisms can explain both what happens on a single occasion, as well as what happens on all the occasions on which a neuron fires due to this mechanism. (Andersen, 2012, 417)[16]

As Andersen rightly points out, scientists explain by appeal to both individual, actualized mechanisms (i.e., mechanism tokens) and general representations of mechanisms (i.e., mechanism types). When appealing to the latter, a mechanism type often gets depicted as a *schema*—"a truncated abstract description" of a mechanism type in which entities are often depicted in boxes and their activities depicted as arrows (MDC, 15). These mechanism-type schemas vary in their *degree of abstraction*. That is, mechanism-type schemas vary in how much detail they include. The more abstract a mechanism schema is, the more detail it leaves out. Here is an example. The protein synthesis mechanism can be schematized, on the one hand, like this:

Or, as we saw in Section 4.1, it can be schematized with a much lower degree of abstraction (see Fig. 1). Both Figs. 1 and 2 are schematic representations of the mechanism-type of protein synthesis. Both are generalized pictorial representations rather than illustrations of a particular protein synthesis mechanism operating in the world. However, Fig. 2 has a much higher degree of abstraction; it leaves out much more detail. Fig. 1, on the other hand, has a much lower degree of abstraction: it contains quite a bit of the detail left out by the first.

But how does any of this relate to mechanistic regularity? The answer I suggest here is that there is a couple of interesting correlations between how abstractly a mechanism is schematized and how regularly it can be understood to behave. I describe two such correlations below.

Given:

(a) the existence of a large number of working mechanism tokens with
(b) at least some features in common and
(c) a high degree of variance in the specific make-up of the features schematized,

CR1: the more abstractly a mechanism-type is schematized, the more of these mechanism tokens instantiate it—that is, the more of these mechanism tokens work in the way specified in the abstract mechanism schema.

CR2: the less abstractly a mechanism-type is schematized, the more it will be the case that the instances it covers behave in exactly that way.

To illustrate these correlations, let us think one more time about toasters. There are toaster tokens, like the one that actually sits on your kitchen counter. There is also a toaster type, a generalized abstraction of a toaster's entities and activities. Like protein

TRANSCRIPTION   TRANSLATION

DNA ⟶ RNA ⟶ Protein

Fig. 2. Watson's central dogma diagram (Redrawn, based on Watson, 1965).

synthesis, toaster types can be represented with higher or lower degrees of abstraction. A toaster type with a low degree of abstraction would include many details (e.g., how many toast slots, how many toast settings, what kind of materials used, etc.). A toaster type with a high degree of abstraction would leave these details out—opting instead only to depict the bare-bones necessary features something needs to be a toaster. Regarding (CR1), the point I wish to emphasize here is that, (a) as long as there are a large number of working toaster mechanisms, (b) these mechanisms have at least some features in common, and (c) there is a high degree of variance in the specific make-up of the schematized features, *the more abstractly a mechanism type is schematized, the more of these mechanism tokens it will cover*. In other words, the more abstract a toaster type, the more toaster tokens will be subsumed under it. This, I suggest, is a new kind of regularity. Let us call it: *abstract regularity*. A highly abstract schematization of a toaster represents a kind of toaster regularly in this sense because (provided the schematization is abstract enough) *every single toaster that exists operates in the way specified by the schema*.

On the other hand, there is a sense in which less abstract mechanism schemas represent a different kind of regularity. As described in (CR2), the less abstract a mechanism schema is (i.e., the more detail it includes), the more it will be the case that the mechanisms instantiating it operate in exactly in that fashion. There just will not be many of them. Call this kind of regularity: *concrete regularity*.

As applied to NS, on the one hand, we can give a characterization of the mechanism of natural selection as a specific, actualized instance of natural selection at work (e.g., the actual sequence of entities and activities leading to the varying beak-lengths of Darwin's finches). And, on the other hand, we can describe NS as a mechanism type. Using Skipper and Millstein's own diagram together with its filling instructions, natural selection as a mechanism type might look like this:

Filling instructions:

I. Initial conditions
1. A population of *O*s exist.
2. *O*s vary according to forms of *T*, which are heritable.
3. *O*s are in environment *E* with critical factor *F*.
II. Interaction
1. *O*s in virtue of the varying forms of *T* interact differently with environment *E*.
2. Critical factor *F* affects that interaction.
3. This may lead to
III. Effects (1)
1. differential survival rates of *O*s across forms of *T* in *E*.
2. This may lead to
IV. Effects (2)
1. differential reproductive rates of *O*s across forms of *T* in *E*.
2. This may lead to
V. Effects (3)
1. differential representation in the population of *O*s across forms of *T* in *E*.
2. This may lead to
VI. Effects (4)
1. the predominance of *O*s with a certain form of *T* over other forms of *T* in *E*.

2. This may lead to

VII. Effects (5)

1. adaptation of the lineage with respect to *T* in *E*.

Filling instructions:

'*O*' is to be replaced by the name of some organism

'*T*' is to be replaced by some determinable organismic trait

'*E*' is to be replaced by the description of the environment of '*O*'

'*F*' is to be replaced by the description of a critical factor in '*E*'

As we shall see, whether we characterize natural selection as a token or a type will matter crucially for how regularly it is seen to behave.

But before showing why the token/type distinction is important, there is one more idea, the explication of which will help tremendously in addressing the remaining issues for natural selection as a mechanism: the role of abstraction in mechanistic explanation. In a recent article entitled, "Abstraction and the Organization of Mechanisms" (2013), Levy and Bechtel make the case that certain mechanistic explanations are successful by virtue of the fact that the mechanism(s) referred to in these explanations are allowed to remain abstract. They argue convincingly that, in the case of at least some of the phenomena targeted for mechanistic explanation, "It is always possible and, we argue, often desirable to overlook the more concrete aspects of a system and represent its organization abstractly as a set of interconnections among its elements." (Levy & Bechtel, 2013, 255). I take their main idea to be this: when attempting to explain highly dynamic mechanistic systems with many interconnected parts, it is often beneficial to leave out some of the details. The reason is, as they put it, "The concrete relations that are pertinent to organization differ across different systems." But, and this is key, sometimes detail-poor explanatory models enable us to "track those features of the system that make a difference to the behavior being explained" (Levy & Bechtel, 2013, 256).

Levy and Bechtel, in this article, point to a peculiar feature of scientific explanation, one that has been recognized before. Think for a moment about an example famously articulated by Hillary Putnam (1975) in which we are trying to explain why a one-inch square peg will not fit into a circular hole with a one-inch diameter. We might explain this by appeal to a detail-rich account of why the specific micro-physical properties of *this particular peg* impede its ability to pass through the molecular make-up of *this particular hole*. But the more explanatorily satisfying answer here, Putnam points out, might actually be an abstract one having to do with the generic properties of squareness and roundness of the peg and hole as such. In giving such an explanation we can track the features of the system (e.g., the squareness of the peg and roundness of the hole) that make a difference to the behavior we are interested in explaining. And those are not necessarily the micro-physical properties. The moral of the story here is meant to be that the best explanation is not always the most detailed; sometimes explanations are good because they are abstract.

So how do the concepts of token vs. type mechanisms and degrees of abstraction help us to address the S-M Regularity Critique? My answer lies in the following argument.

P10. The degree of regularity of a particular mechanism is sensitive to two aspects of how the mechanism is represented: (1) whether it is characterized as a *type* rather than a *token*, and if characterized as a type, (2) its *degree of abstraction*.

P11. When conceived as a type with a relatively high degree of abstraction, natural selection should be understood as behaving
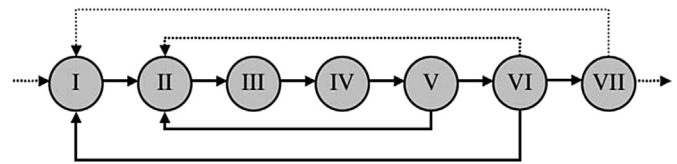

Fig. 3. Abstract natural selection schema (From Skipper & Millstein, 2005, 330; reproduced by permission).

'always or for the most part in the same way under the same conditions' thereby fulfilling the MDC regularity requirement.

P12. There are legitimate explanatory contexts (a la Putnam's pegs) in which natural selection should be characterized as a mechanism type with a high degree of abstraction.

C4. So, contra the conclusion of the S-M Regularity Critique, there are legitimate explanatory contexts in which natural selection qualifies as regular enough to meet the MDC requirement.

I have already motivated (P10). But (P11)–(P12) need more support to show that (C4) follows.

Starting with (P11), let us apply my characterization of abstract regularity to natural selection. Like the aforementioned toaster tokens, actualized instances of natural selection (e.g., the actual selection history for Darwin's finches) will be constituted by specific entities (e.g., population of finches) engaging in specific activities (e.g., foraging for seeds) in specific environmental surroundings (e.g., the Galapagos Islands). This concrete description of natural selection as a token may only qualify as regular in the sense described by CR2. And on this measure, Skipper and Millstein seem right that natural selection does not work always or for the most part in this fashion. However, when idealized as an abstract mechanism type, natural selection can be depicted as a general kind of process (e.g., Fig. 3 above). And when represented with this high degree of abstraction, it overcomes the individual differences between its actualized token instances. Represented like this, natural selection does operate the same way every time: (P11).[17]

Furthermore, I suggest that there are legitimate explanatory contexts in which natural selection as an abstract mechanism type does real explanatory work—work that mere tokens of natural selection cannot do. As with Putnam's pegs, there are instances where we are not interested in the specifics of why a particular population has ended up with the morphological make-up that it does. But rather, we might be interested in why, in general, low-spired snail shells prevail in tidal pool regions across the globe. Here we would not want to appeal to a specific population of snails/crabs. We would instead want to depict the natural selection type as it applies to snail predation across a variety of specific tidal pool

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[17] An objection might arise here regarding just how abstract a schema of natural selection would need to be in order to overcome the individual differences at the token level. Havstad (2011) argues that, in order to achieve this, we would arrive at a schema so abstract it could no longer differentiate natural selection from general selection.

By way of response to this objection, I would first agree that, characterized at its highest degree of abstraction, it may well be the case that our natural selection schema fails to pick out only instances of natural selection as opposed to general selection. However, I would suggest, it is not clear why this is a problem. It depends on what I am trying to explain. If my explanatory goal is to figure out precisely what differentiates natural selection from other kinds of selection processes, then this highly abstracted schema will not do much good. But suppose I want to know whether struggle for existence is a necessary component of natural selection. Then it may do just fine to have an abstract schema that depicts features that all instances of natural selection share (even if it fails to depict the sufficient conditions for membership in the natural selection type).

regions. We may even have legitimate questions about what instances of selection share in common at its *most* abstract level—in which case an answer would be to appeal to the highest degree of abstraction: (P12).

Putting these premises together, we can now see that, contra Skipper and Millstein, there are legitimate explanatory contexts in which natural selection qualifies as regular enough to meet the MDC regularity requirement: (C4).

5. Conclusion(s)

In this paper, I have explored whether natural selection fails to operate regularly enough to be an MDC mechanism. I have argued that it does not. To do so, I first offered prima facie reasons for why we might want to think about natural selection in mechanistic terms. I then summarized Skipper and Millstein's regularity critique against natural selection as an MDC mechanism. To counter this critique, I then drew three distinctions regarding mechanistic regularity and showed that natural selection only fails to be regular in ways that should be seen as unthreatening to its status as an MDC mechanism. First, I distinguished between process and product regularity and argued that the Skipper and Millstein critique only shows natural selection to be product irregular not process irregular, but it is process regularity that should matter more for MDC. Second, I distinguished between mechanism-internal and mechanism-external sources of irregularity and argued that the sources of the irregularities associated with natural selection constitute an unthreatening mechanism-external sources of irregularity. And third, I distinguished between abstract and concrete regularity and showed that how regularly we conceive of natural selection depends crucially on the degree of abstraction we employ to schematize it. When schematized in a highly abstract manner, I suggested, natural selection can be seen to operate quite regularly.

Beyond the specifics of these arguments, however, there are some more general conclusions to draw. Following Beatty (1995), one might take the biological world to be riddled with contingency: too complex to formulate laws governing it. One might object to a mechanistic approach to explaining the living world for similar reasons: when it comes to biological phenomena, we do not have something that looks like clockwork. We have a teeming, seething, mess. Sensitive to this exception-ridden, highly complex nature of the living world, proponents of the mechanistic approach either soften their regularity requirement (e.g., MDC's now oft-quoted "mechanisms are regular in that they work always or for the most part in the same way") or they get rid of it altogether (e.g., Bogen, 2005; Glennan 2011; Machamer, 2004). What I take the arguments in this paper to have shown is that proponents of MDC mechanisms can do better than this; they can be more nuanced in how they respond to problems regarding irregularity; and indeed careful attention to these arguments can provide crucial resources for understanding how appealing to mechanisms can explain probabilistic phenomena in the natural world.

So rather than a mere attempt to vindicate the use of the word 'mechanism' to describe natural selection, I take these arguments to demonstrate a general strategy for thinking about appealing to mechanisms to explain stochastic biological phenomena in the contingent natural world. The central features of this strategy might be summarized as follows:

○ Be aware of the nature and source of the irregularities that riddle your causal process of interest. Not all irregularity is equally damning to a process's mechanistic status.
○ Be aware that irregularity takes different forms, some of which do better than others at supporting generalizations and ampliative inference in science.

○ Understand that, whether a causal process behaves regularly enough to count as a mechanism, depends crucially on the degree of abstraction used in its representation.

Acknowledgments

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