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Stranger in a strange land

An optimal-environments account of evolutionary mismatch

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Abstract In evolutionary medicine, researchers characterize some outcomes as evolutionary mismatch. Mismatch problems arise as the result of organisms living in environments to which they are poorly adapted, typically as the result of some rapid environmental change. Depression, anxiety, obesity, myopia, insomnia, breast cancer, dental problems, and numerous other negative health outcomes have all been characterized as mismatch problems. The exact nature of evolutionary mismatch itself is unclear, however. This leads to a lack of clarity about the sorts of problems that evolutionary mismatch can actually explain. Resolving this challenge is important not only for the evolutionary health literature, but also because the notion of evolutionary mismatch involves central concepts in evolutionary biology: fitness, evolution in changing environments, and so forth.

In this paper, I examine two characterizations of mismatch currently in the literature. I propose that we conceptualize mismatch as a relation between an optimal environment and an actual environment. Given an organism and its particular physiology, the optimal environment is the environment in which the organism's fitness is maximized: in other words, the optimal environment is that in which the organism's fitness is as high as it can possibly be. The actual environment is the environment in which the organism actually finds itself. To the extent that there is a discordance between the organism's actual and optimal environments, there is an evolutionary mismatch. In the paper, I show that this account of mismatch gives us the right result when other accounts fail, and provides useful targets for investigation.

Keywords Evolutionary mismatch · Developmental mismatch · Evolution · Biology · Mismatch · Adaptation · Environment

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1 Introduction

In the fields of evolutionary psychology and evolutionary medicine, evolutionary explanations for health outcomes characterize some negative outcomes as mismatch problems. Roughly, mismatch problems are those problems which arise as the result of an organism living in an environment to which it is poorly adapted, typically as the result of some rapid environmental change or migration. Depression, anxiety, obesity, myopia, insomnia, breast cancer, dental problems, and numerous other negative health outcomes have all been characterized as mismatch problems at various times. These sorts of outcomes are often referred to as evolutionary mismatch.

However, relatively little work[1] has been done to define the concept of evolutionary mismatch. Currently, the mismatch concept appears to be characterized in only vague terms, with characterizations often relying on biological examples to do the heavy lifting. Resolving this vagueness is a necessary step in untangling the claims being made.

In the next section of the paper, I will discuss the concept of evolutionary mismatch a bit more as it appears in the empirical literature. I do this to help the reader get some grip on the biological phenomena of interest, and to provide some motivation for the project. As noted in Lloyd et al. (2011), evolutionary mismatch (or something like it) seems to have implications for evolutionary biology as a whole. Although the concept has explicitly been used only in a limited body of research, the idea of fit to environment and the causal impact of environmental change on evolutionary change are objects of central interest to biologists.

In the third section of the paper, I will briefly consider both accounts of evolutionary mismatch given in the philosophical literature to date (Lloyd et al. (2011) and Cofnas (2016)), and suggest that both approaches are helpful but can be profitably revised.

In the fourth section of the paper, I develop the primary account, which argues that evolutionary mismatch should be understood as a discordance between an organism's optimal environment (given its physiology) and the organism's actual environment. To the extent that the organism has lower fitness in its actual environment than in its optimal environment, there is an evolutionary mismatch.

In the fifth section of the paper, I motivate the primary account and show that the conceptual moves are plausible and that they cohere with existing scientific literature on mismatch.

In the final section of the paper, I consider some objections. In the objections, I also consider broader questions of the scientific relevance of the view.

[1] In the philosophical literature to date, the only detailed attempts of which I am aware are Lloyd et al. (2011) and Cofnas (2016). Garson (2015) discusses a certain kind of mismatch but does not give a general characterization.

2 What is evolutionary mismatch?

To get a handle on the basic idea of mismatch, we need to understand one of the key components of alleged mismatches: the ancestral environment, also known as the Environment of Evolutionary Adaptedness (EEA).[2] The EEA is the environment to which a given population is primarily adapted, the selection pressures of which have driven much of the population's evolution. Mismatch theorists tend to argue that *Homo sapiens* has an EEA, frequently identified as the African savannah approximately 50,000 years ago and earlier.[3] Having spent dozens of millennia evolving in response to the particular selection pressures of foraging life on the savannah and relatively little time evolving since leaving Africa (and leaving foraging), *Homo sapiens* is still primarily adapted to that environment.

Contemporary environments, on the other hand, tend—particularly in the developed world—to be very different from a foraging life, in all sorts of plausibly selection-relevant ways: modern diets are more palatable and calories are easier to obtain; day-to-day life is more sedentary; women have more menstrual cycles; social relationships are more diffuse; and so forth.[4]

A mismatch arises when ancient genes—genes which evolved to meet the challenges of the EEA—run up against novel situations in contemporary environments to which those genes are not well-adapted. The mismatch, it is alleged, sometimes results in deleterious consequences for adaptedness (i.e. reproductive fitness) or health.[5] As Cosmides and Tooby (1997) put it, "...our modern skulls house a Stone Age mind."

This sort of mismatch comes in three parts: the EEA, the contemporary environment, and the negative outcome due to the contemporary environment. (To be clear, again: this is just a sketch of the idea. My intent is only to give the reader an impression of the idea in play.) In order to fill out the picture a bit,

[2]Credit for coining the term is typically given to Bowlby (1982), cf. Barr (1999), Tooby and Cosmides (2005), Gluckman and Hanson (2006), Taylor (2015). A related term is 'ancestral environment', preferred by Lloyd et al. (2011) but treated as roughly equivalent to EEA. I am using the established EEA terminology for brevity, and I think that even mismatch theorists who eschew the particular terminology will recognize its theoretical analogues in their own accounts. The EEA is really a cluster of related concepts and gets used in various ways—Lloyd et al. (2011), for example, describe traits rather than populations as having 'ancestral environments'—but all I need at the moment is for the reader to understand roughly what the EEA is. Those already familiar with the term should take my use of EEA as a gesture at the phenomenon of interest rather than a commitment to the particular claims of Tooby and Cosmides's evolutionary psychology. Thanks to an anonymous reviewer for this point.

[3]See, e.g., Lindeberg (2010) 30. As I said in footnote 2, for the sake of brevity I am eliding the full set of views here: some theorists prefer to think of each trait or mechanism as having a specific EEA.

[4]See, e.g., Nesse and Williams (1994), Trevathan (1999), Tooby and Cosmides (2005), Gluckman and Hanson (2006), Keller and Nesse (2006), Gluckman et al. (2009), Lindeberg (2010), Lieberman (2013), and Aktipis et al. (2015).

[5]Whether we are being given a story about fitness outcomes or health outcomes can sometimes be a bit fuzzy. More on this in section 6.2 on page 16.

I will give two examples of mismatch hypotheses which have been proposed in the evolutionary health literature: scurvy and myopia.

Vitamin C (ascorbic acid) is an essential nutrient for *Homo sapiens*, playing a role in wound healing and immune function, among other important functions. Though most mammals are capable of synthesizing vitamin C, this ability appears to have been lost in the haplorhine lineage—the tarsiers, monkeys, and apes (Pollock and Mullin (1987)). Consequently, humans must obtain sufficient vitamin C through diet. Scurvy is a disease resulting from a lack of adequate vitamin C, and is particularly well-known for historically afflicting sailors on long ocean voyages, who typically had extremely limited access to vitamin C-rich fruits and vegetables. In the EEA, however, humans presumably had extensive access to fruits and vegetables, providing abundant vitamin C. (Eaton and Konner (1985) estimate that contemporary hunter-gatherers have a daily vitamin C intake of 392.3 mg/day, approximately nine times the U.S. government-recommended daily minimum at the time of the study. Konner and Eaton (2010) updates these numbers to approximately 500 mg/day and approximately five times, respectively.) The mismatch arises when an organism from a species which evolved in a vitamin C-rich environment, and which requires vitamin C to live, is placed in a vitamin C-poor environment. In other words, humans are adapted to the selection pressures of an environment in which vitamin C can be obtained through adequate nutrition—not to those of an environment in which vitamin C cannot be so obtained.[6]

Another hypothesized, though controversial, example: humans evolved spending most of their time outside, using their eyes to distinguish objects near and far. In childhood, continual work at short distances (particularly close-in work like reading) does not allow the eye to develop properly, resulting in a longer eye which is not able to focus light from distant objects to form sharp images. In other words, the human eye evolved in outside environments where objects at both close and far distances were regularly available to act as stimuli in optical development. In contemporary environments, focus at distance is less common, resulting in myopia among humans already prone to it. The mismatch here, then, is the lack of necessary stimuli during development—stimuli provided by the EEA, but not by the contemporary environment.[7]

The mismatch concept has utility beyond these human examples. Lloyd et al. (2011) describe mismatch as "central to evolutionary theory." Examples in the rest of the paper should illustrate this, and I will say more to underscore this point in the conclusion.

[6]For more discussion of this example, see Gluckman et al. (2009).

[7]For more extensive discussion of this highly simplified example and related hypotheses, see Nesse and Williams (1994); Gluckman et al. (2009); Lindeberg (2010); Lieberman (2013). By contrast, Tideman et al. (2016) found that myopia had a stronger association with serum vitamin D levels than with time spent outdoors, indicating that low vitamin D may be implicated in myopia. Pan et al. (2017) suggest that the evidence supporting the vitamin D hypothesis is still insufficiently strong, however, and argue that the current state of the evidence favors time spent outdoors as a more likely protective factor than higher serum vitamin D. I use the simplified example above in part because it is more popular in the evolutionary health literature.

3 Previous mismatch accounts

Evolutionary mismatch has thus far received relatively little focused philosophical attention. Although some particular mismatch claims have been explored,[8] the basic conceptual framework of evolutionary mismatch has yet to be fully elucidated. In this section, I will briefly discuss the two philosophical papers (one unpublished) which have attempted to describe mismatch in substantial detail.[9] I will outline some worries for each approach *if they are taken to be exhaustive accounts of evolutionary mismatch.* I think both accounts shed important light on the forms that evolutionary mismatch can take, however, and I see my work here as an attempt to build on theirs rather than to overthrow them.

3.1 Lloyd et. al's mismatch primer

After a 2011 pair of workshops on evolutionary mismatch put on by the Evolution Institute and the National Evolutionary Synthesis Center, Lloyd et al. wrote an introduction to an intended (but never published) special issue of *Evolutionary Applications* focused on mismatch.[10] The paper is intended to serve as a primer on evolutionary mismatch, introducing key concepts, discussing biological cases, and laying out the theoretical importance of conceptual work on mismatch. Lloyd et al. (2011) say that evolutionary mismatch "can be defined as a negative consequence that results from a trait that evolved in one environment being placed in another environment." On this account, mismatch is an *outcome* caused by a given trait's lack of fit to an environment relative to the trait's ancestral environment. Importantly, Lloyd et al. state that ancestral environments "must be defined separately for each trait." Thus, a particular trait may have negative consequences in a new environment (E_2) relative to its EEA (E_1). Lloyd et al. use the example of RubisCO (ribulose bisphosphate carboxylase/oxygenase), an ancient enzyme used by most autotrophs in carbon fixation (Tabita et al. (2007)). (I add more detail to their example here but I believe I preserve its spirit.) To function properly, however, RubisCO requires a minimal concentration of CO_2 in the atmosphere. When the concentration of CO_2 drops too low, RubisCO can no longer fix carbon for the organism. (Nisbet et al. (2007)) Thus, Lloyd et al. contend, in any oxygen-rich environment RubisCO will result in a mismatch. In cyanobacteria, for example, this (putative) mismatch is ameliorated by pathways which transport CO_2 to the carboxysome, "a microcompartment containing RubisCO in

[8] See, e.g., Buller (2006).

[9] Garson (2015) gives a sketch of evolutionary mismatch as well, but I take the scope of the account to be a particular kind of mismatch. Thus, I will treat it here as subsumed under Cofnas (2016), due in part to the latter's use of the former. My thanks to Justin Garson for calling my attention to his work here.

[10] The paper is available at the website of The Evolution Institute at https://new.evolution-institute.org/wp-content/uploads/2015/08/Mismatch-Sept-24-2011.pdf.

which elevated CO_2 is sequestered." (Nisbet et al. (2007)) Lloyd et al. say that in some cases, like RubisCO, mismatches "can become permanent features of life" which are compensated for rather than eliminated by further evolution.

Although it makes an important contribution to philosophical work on mismatch, the paper takes the (in my view) surprising position that it will typically make sense to talk about particular traits causing mismatch in some environments, with that mismatch being compensated for by the emergence of new traits. Let us look at the RubisCO case again: on the view in Lloyd et al. (2011), RubisCO causes a mismatch in all high-oxygen environments regardless of additional traits which may alleviate the always-present mismatch. It is unclear to me, however, why we ought to carve up the traits this way: why should the traits be seen as atomic rather than compound? In other words: rather than saying that RubisCO (R) causes a mismatch and compensating traits (C) do not, perhaps the trait $R \wedge \neg C$ causes a mismatch but the trait $R \wedge C$ does not. Organisms' traits rarely exist in pure $A/\neg A$ isolation: there are costs and benefits to suites of traits taken as wholes rather than merely to atomic traits. Thus, the view advanced by Lloyd et al. seems (at least as they use it in the RubisCO case) to suggest that traits can involve mismatches even when they are part of fitness-enhancing wholes. (Similar to the worry about excessive atomization in Lloyd (2015), it is also unclear to me that RubisCO can be treated as biologically distinct from other cellular mechanisms in the way Lloyd et al. describe.)

Now, Lloyd et al. acknowledge the challenges inherent to individuating traits, and they might point to a number of ways to identify RubisCO as a distinct trait: its function, the specific genes which code for it, and so forth. In this case, however, I worry that individuating RubisCO like this might serve to obscure more than clarify. As noted earlier, RubisCO *still serves a fitness-enhancing role* when the organism is in a low-CO_2 environment: it allows the organism to fix carbon. Some organisms which existed with a $\neg R$ mutation would presumably experience drastically reduced fitness, which is surely a negative outcome of interest. Yet this implies (by hypothesis) that $\neg R$ would entail a mismatch, just as R (by their stipulation). I find this result counterintuitive. The response here might be to point out that R is a trait which evolved in a low-oxygen environment, and so it can lead to a mismatch when it exists in a high-oxygen environment; $\neg R$ is a novel trait and so environmental change for a $\neg R$ organism cannot result in a mismatch, however profound the negative consequences for fitness. If that is the case, they might argue that even though both R and $\neg R$ result in fitness harms, only R has the history necessary for it to 'count' as a mismatch. I worry that this only exacerbates the problem, however, as it suggests that an evolutionary mismatch cannot result as the emergence of a new trait with poor 'fit' to the current environment. (Indeed, they implicitly exclude this possibility.) I find it quite plausible to think that there is a mismatch when a novel trait is maladaptive in the current environment—at least if said trait would be neutral or adaptive in some environments. I would consider it a theoretical virtue for an account of mismatch to be able to handle a broader range of ways that

organisms with certain traits can have poor fit to their environment. If the reader's intuition runs toward the restriction of a specific evolutionary history of the mismatch-producing trait, then they may find little here to worry them about the account in Lloyd et al. (2011) and thus little reason to prefer my view to theirs. Indeed, that intuition may even militate against the account I develop here. I discuss this more in the overbroadness objection in 6.4 on page 18.

The motivation in Lloyd et al. (2011) for thinking of RubisCO this way may be this: "Human efforts to ameliorate mismatches to our current environment sometimes bear an intriguing resemblance to the traits that evolved to ameliorate the effects of oxygen on RubisCO." I am sympathetic to this point, but I think it is unnecessary to insist on a mismatch persisting after the fitness effects have been ameliorated. Consequently, I think their view includes some non-mismatch cases (e.g., plausibly, RubisCO) and excludes some cases of mismatch (e.g. the $\neg R$ organism).

It is worth noting here that I have treated the Lloyd et al. picture of mismatch as fitness-focused, though they actually allow for the "negative consequence" of interest to be a negative fitness *or* health effect. This distinction is important to track, in that changes in health do not inevitably entail changes in fitness: we might think, here, of diseases which appear so late in life that their fitness effects are negligible. The evolutionary health literature explicitly acknowledges that selection works on fitness and thus may not select against traits which harm health but not fitness (cf. Nesse and Williams (1994): 235-236). Sometimes, however (as I said in footnote 5), mismatch talk elides health and fitness effects. To keep the phenomena in sharper focus, I have chosen to treat mismatch as a fitness issue. I discuss this further in the welfare objection in section 6.2 on page 16.

With all that said, I want to emphasize that although I differ with Lloyd et al. over their particular definition of mismatch, the paper includes many keen insights: the centrality of mismatch-sounding themes to evolutionary biology, the importance of successfully individuating the traits and environments of interest, and the utility of the mismatch concept outside of the human cases which seem to be the focus of most of the literature on mismatch.

3.2 Cofnas's teleofunctional account

The other paper, Cofnas (2016), defines evolutionary mismatch as "deviations in the environment that render biological traits unable, or impaired in their ability, to produce their selected effects." Here, Cofnas draws on an account of proper function of a trait characterized in terms of the adaptive function of the trait. ('Proper function' can refer to Neanderian proper function, or Millikanian direct proper function or Millikanian invariant derived proper function.) An environment which deviates from an organism's ancestral environment such that in the former, an adaptive trait cannot carry out its proper function, is an environment which is evolutionarily novel. Novel environments (in this sense)

are mismatched to ancestral environments. In short, mismatch obtains when an environmental change relative to the ancestral environment causes a trait to be inhibited from fulfilling its proper function. (As mentioned earlier, I take Cofnas to draw in part from the partial characterization in Garson (2015) of mismatch as a psychological adaptation which is maladaptive in the novel environment, and so I will focus my attention on the teleofunctional account.)

Adaptations allow organisms to develop and also to interact with their environments in ways which improve their fitness. This gives us the teleofunctional account's taxonomy of mismatch types: (1) underdevelopment-inducing (e.g. myopia), (2) ineffectiveness-inducing (e.g. peppered moths in a polluted environment), (3) misrepresentation-inducing (e.g. supernormal stimuli), and (4) misresponse-inducing (e.g. apartment-living cats pouncing on shadows).

One advantage of Cofnas's view is its consonance with the adaptationism which permeates much of the evolutionary health literature, much of it quite explicit.[11] Cofnas also provides a very helpful taxonomy of different sorts of mismatch which is, I think, a major contribution to the body of work on mismatch. I worry, however, that Cofnas may leave out a putative case of mismatch which can be found in the literature.

Consider:

> **Island Prey**: we will postulate an island population P of organisms which has no predators at t_1. Members of P have no predator-avoidance adaptations: the organism's lineage long ago lost such adaptations through drift, so now the organism is not equipped to avoid predation. Suppose that at t_2 a predator finds its way to the island and begins preying with great gusto on the predation-naïve population, which is essentially helpless: facing no selection for predator-avoidance traits, organisms within the population are simply oblivious.[12]

It is important to note that in Island Prey, by stipulation, the lack of predator-avoidance traits in P are not an adaptation to a predator-free environment: there is no selection *against* such traits; they were simply not maintained in the population over time. The environment has changed and the organisms are poorly-adapted to the change, and yet it seems that no particular trait is failing to perform its proper function—unless the idea is that the predator's jaws prevent the spinal column for performing its proper function, which does not quite seem to be the sort of mismatch Cofnas is interested in (since the same thing can be said any time a lion takes a gazelle, which is surely not an evolutionarily novel situation—mismatch only occurring in situations of evolutionary novelty, per Cofnas).

Moreover, even if there are variants in P which respond properly to the predator, these predator-avoidance traits are not adaptations because they are (again, by stipulation) not the result of an evolutionary history of selection for

[11] This general adaptationism has been criticized in, e.g., Valles (2012) and Cournoyea (2013), and I will not engage with that worry here.

[12] The discussion here is inspired by discussion in Sih et al. (2010) and Sih (2013) of evolutionary mismatch caused by novel predators.

the traits: they are simply variants which become advantageous after an environmental change. For both variants, however—avoidant and non-avoidant—there is (seemingly) no evolutionary mismatch because there is no inhibition of traits performing their proper functions. I take it that a commonsense view of mismatch would diagnose a mismatch for the non-avoidant variants (relative to the avoidant variants, at least). If our view of mismatch would exclude diagnosis of such cases, our view may require some work.

I take it that I have shown there is a meaningful challenge for the teleofunctional account of evolutionary mismatch. I think, however, that Cofnas has done outstanding work in unpacking many forms of organism-relevant environmental change. Thinking about mismatch in terms of function, as he does (and as in Garson (2015)), should furnish a number of interesting objects of investigation.

4 An optimal-environments account of evolutionary mismatch

4.1 Predictive Adaptive Responses and developmental mismatch

Gluckman et al. (2005) propose the existence of a category of phenomena which they call Predictive Adaptive Responses (PARs). PARs comprise those adaptive responses of organisms which reflect phenotypic plasticity with delayed benefits. By way of example, they discuss the effect of nutritional stress on humans *in utero*, famously observed as a result of the Dutch famine in the winter of 1944-1945.

At the end of World War II, food rationing in the Netherlands became severe, with official rations dropping from 1800 kcal/day in late 1943 to 1400 kcal/day in October 1944, falling below 1000 kcal/day in November 1944. Daily rations then fluctuated between 400 and 800 kcal/day until April 1945, and then rose to over 2000 kcal/day by June 1945. A half-century later, surviving Dutch adults who had been born from November 1943 through February 1947 were examined for obesity, glucose tolerance, and other markers of metabolic impairment. Those adults born during or shortly after the famine—particularly those who were in mid- or late-gestation during the famine—displayed markedly worse glucose tolerance and insulin resistance than their counterparts born significantly earlier or significantly later.[13] In other words: adults who had been *in utero* while their mothers were under significant nutritional stress were more likely, later in life, to suffer from various forms of metabolic dysfunction.

Along with insulin resistance and impaired glucose tolerance, Gluckman et al. (2005) note that maternal nutritional stress can also lead to increased adiposity, reduced muscle mass, and a more compact body shape. They propose that the fetus "learns" that its environment is calorie-poor, and thus

[13]See e.g. Ravelli et al. (1998) for more discussion of this incident and the specific results. See Hales and Barker (2013) for further discussion of the so-called "thrifty phenotype hypothesis".

alters metabolic development to conserve energetic resources throughout life. On this hypothesis, the fetus predicts a calorie-poor environment and uses its inherent phenotypic plasticity to respond to the prediction. Gluckman et al. (2005) and Gluckman et al. (2011) both argue that when this prediction turns out to be incorrect—i.e. when the environment turns out to be calorie-rich—a developmental mismatch may arise which causes the impaired glucose tolerance and insulin insensitivity already mentioned. The fetus has adapted developmentally to anticipated nutritional stress, and nutritional surplus may be more likely to cause obesity, type 2 diabetes, and other health problems in the adults who experienced nutritional stress *in utero*.

This mismatch, they think, is between the environment predicted by the organism (in this case, the fetus) and the actual environment in which the organism finds itself. In this sort of case, the PAR's interaction with the 'wrong' environment can plausibly result in negative fitness consequences for the organism, while encountering the optimal environment can result in positive fitness consequences. In the next section, I propose extending the idea of 'optimal environment' to generate a broader concept of evolutionary mismatch.

4.2 The main move

The existence of the PAR implies that given a particular organism's physiology, there is an environment in which the PAR is the appropriate response. In other words, there is an environment in which the organism's fitness is increased as a result of the PAR. This can be extended: given a particular organism and its physiology,[14] there is a set of environments where that organism's fitness[15] is maximized, i.e. is higher than in all other possible environments. (I will present a more detailed motivation for this claim in the next section.) I consider these environments the optimal environments for the given organism: in all environments other than these, the organism's fitness is sub-maximal. In the optimal environments, the organism's fitness is higher than in all other environments.[16]

These optimal environments are not necessarily environments free of stressors or competition: perhaps the organism requires stressors for its fitness to be maximized. I make no claim about pleasant temperatures, energetic richness, or any other specific environmental character relevant to fitness. The only vari-

[14]I use 'physiology' to refer collectively to an organism's heritable traits (including phenotypic plasticity), rather than merely to genotype or merely to phenotype.

[15]My view does not depend on a particular analysis of evolutionary/reproductive fitness, and the reader should feel free to insert her preferred account. Although I assume a concept of fitness linked in some way to reproductive success, I see no reason that a concept like that developed in Bouchard (2011) could not work here.

[16]To be clear, my use of the term 'optimal environment' is not intended to refer to optimization in the sense of optimal foraging strategies or other optimality models as discussed in e.g. Orzack and Sober (1994). If anything, the reader should take this sense of 'optimal environment' to be similar to, if not quite identical with, that invoked by Gluckman and Hanson (2006) 17-19, 33 and Levins (1968) 14: an environment in which fitness is maximized.

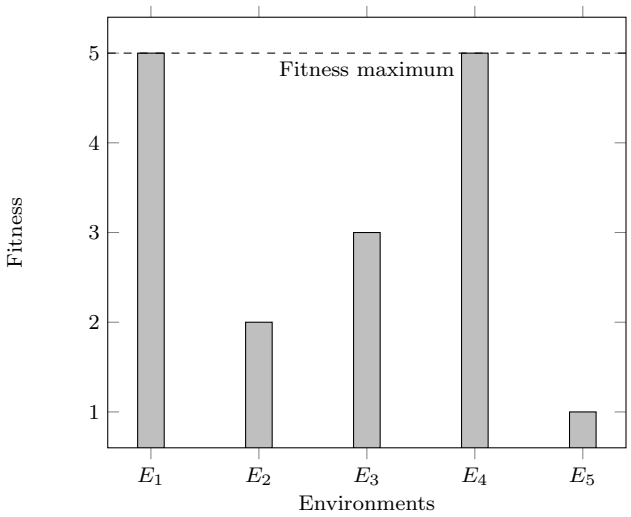

Fig. 1: Mismatch at a glance

able these environments inherently have in common is the given organism's fitness in those environments.

Given the optimal environments (which I will refer to as E_{mf}), we now turn to the organism's actual environment, E_a. If the organism has reduced fitness in E_a relative to E_{mf}, then the two environments are mismatched. In other words, when the organism is less fit in its actual environment than it would be in its optimal environment, evolutionary mismatch obtains. We can see this a bit more clearly in figure 1 on page 11.

Consider figure 1. Given an arbitrary organism with a maximum fitness across all environments of 5 and a range of possible environments (E_1, E_2, etc.): because the organism's fitness is maximized in E_1 and E_4, E_1 and E_4 are its optimal environments. In all other environments, mismatch obtains. (The reader should take 5 to quantify a fitness measure compatible with her preferred account, should she have one.) My central claim:

> **CC**: evolutionary mismatch obtains when an organism O is in an actual environment E_a such that O's fitness is lower than it would be in an optimal environment E_{mf}.

Let us return, now, to the earlier mismatch hypotheses: scurvy and myopia.

On the story I am telling, scurvy arises because the traits of *Homo sapiens* require an abundance of vitamin C in the environment. E_{mf}, then, is an environment rich in sources of vitamin C. In this respect, E_{mf} matches closely with the ancestral environment of humans.[17] If E_a is an environment relatively poor in vitamin C—say, the environment experienced on a long ocean voyage a few centuries past—then there is a mismatch between E_{mf} and E_a resulting in decreased fitness of the organism.

[17] As mentioned above, Eaton and Konner (1985) and Konner and Eaton (2010) can be consulted for estimates of dietary vitamin C in the diets of early humans.

So also with myopia: the ontogeny of the human eye is such that fitness is maximized in an environment with a broad range of focal distances (E_{mf}). When the eye in a young human is subjected to an environment poor in focal stimuli (E_a), the mismatch between these environments can cause nearsightedness. Now, I must acknowledge that we might dispute whether myopia genuinely reduces fitness in the contemporary environment—given that we can accommodate it with prescription lenses, eye surgery, and so forth. If myopia does not reduce fitness, then perhaps it is caused by evolutionary mismatch only in the case of some (perhaps most) humans, and in other cases the environmental change still causes the physiological effect but does not have a fitness effect. If the mismatch theorist is committed to saying that myopia is *always* an evolutionary mismatch (given the same physiological and environmental changes), then my view would sometimes break with the literature. (And my competitors might have an advantage here, as neither the teleofunctional account nor Lloyd et al's account require a fitness effect to diagnose a mismatch.) I think this need not be too worrisome, however, for two reasons. First, myopia is still the *kind* of problem which can result from an evolutionary mismatch, even if not all cases of myopia will result from an evolutionary mismatch. Second, my view of mismatch requires an impact on the organism's total fitness, and so it allows for the environment to compensate for specific negative physiological effects. (I discuss this issue further in section 6.2 below.)

Finally, consider the predation-naïve population I discussed in section 3.2's Island Prey: the teleofunctional account seems to imply that in the absence of a particular trait being inhibited in its proper function, there is seemingly no evolutionary mismatch which results from the invasion of a new predator. On the optimal-environments view developed here, the invasion of a new predator species causes an environmental change which reduces fitness in the naïve organisms, without reference to particular inhibited adaptations. Moreover, any variants in the population which are better at predator-avoidance will probably be in a decreased state of mismatch relative to their naïve conspecifics. Thus, a pretty clear case of evolutionary mismatch seems not to be identified by the teleofunctional account, and is correctly identified by the optimal-environments account.

As I hope is clear from the explicit link I have drawn, this mismatch concept also works when considering the failure of PARs: the traits of humans *in utero* during the Dutch famine required a calorie-poor environment (E_{mf}) which was mismatched to the actual, calorie-rich environment (E_a) they encountered. When PARs succeed in generating an E_{mf} concordant with E_a, there is no mismatch—just as there is no mismatch when an organism is maximally well-adapted to its environment.

The careful reader will note that I have not explicitly integrated the ideas of the EEA or of evolutionary novelty and environmental change, as Cofnas (2016) and Lloyd et al. (2011) do. The explanation for the mismatch between E_{mf} and E_a could comprise recent significant environmental change, but could also comprise the emergence of new variants in a population, where the new variant has an E_{mf} quite different from that of its conspecifics. If E_{mf} for the

new variant more closely (or, for that matter, less closely) resembles E_a, then increased (or decreased) success of the new variant relative to conspecifics may be more easily explicable. Thus, I think that this approach plausibly includes more biological phenomena of scientific interest than a mismatch concept wedded to the EEA as a component. (I explain more below when I discuss the EEA objection in section 6.1.)

5 The argument

5.1 The plausibility of optimal environments

Some might wonder, at this point, whether the notion of an optimal environment is even coherent. I hope to show here that on the account of mismatch I am developing, the reader need only agree with me on some very minor assumptions in order for this analysis of mismatch to marry up with phenomena in the world. The core assumption is this:

> **CA**: For evolutionary mismatch to be possible with respect to a particular organism, there must be at least two environments (E_1 and E_2) such that in E_1 the organism has higher fitness than it does in E_2.

To see how this works: the optimal environment for an organism is just that environment where the organism's fitness is higher than in all other environments. Thus, so long as more than one fitness value is possible for an organism across different possible environments, the environment in which the organism's fitness is highest will be E_{mf}, the optimal environment.[18]

5.2 Coherence with the rest of the literature

I see my account lining up with the rest of the literature on evolutionary mismatch fairly well, in three specific ways: (1) the account reflects two common uses of the term to label particular kinds of relations; (2) the account is able to accommodate developmental ideas of mismatch; (3) the account gives us the right result in some putative cases. (I discuss my most significant departure from the literature in the EEA objection below.)

First: mismatch is sometimes treated as a relation between trait/organism and environment[19] and sometimes treated as a relation between environments[20]. Trying to clarify what mismatch actually is—assuming that it is a relation—requires us to identify the relata. Here, I am essentially treating E_{mf}

[18] As noted in footnote 16, I realize that 'optimal' can be a loaded concept, and so I would like to stress that my use of the term is not intended to imply any theoretical commitments other than what I have explicitly stated here.

[19] Cf. Nesse and Williams (1994); Editorial. (2009); Lindeberg (2010).

[20] Cf. Gluckman et al. (2005); Gluckman and Hanson (2006); Keller and Miller (2006); Gluckman et al. (2011); Jasienska (2013); Cofnas (2016).

as a property of the organism. Thus, on my account, mismatch is a relation between two environments—optimal and actual—where one of the environments is a property of the organism. This view then implies that mismatch is also a relation between the organism and its actual environment. If mismatch is concerned with poor fit between organisms and their environments, then poor fit between the optimal environment and the actual environment still captures the key idea. In other words, mismatch as a relation between environments *and* mismatch as a relation between organism and environment are both captured by the view developed here.

Second: my account draws on an existing model of developmental mismatch to develop an integrated definition of evolutionary mismatch which incorporates the results of both developmental history and evolutionary history—the actual physiology and psychology of the organism—to show how mismatch occurs. When I discussed PARs in 4.1, I pointed to a form of mismatch in the literature—developmental mismatch. Yet developmental mismatch depends in PAR cases not on a mismatch between the EEA and the actual environment, but between the environment 'predicted' by the organism and the organism's actual environment. By making the mismatch concept about an optimal environment rather than *either* the predicted environment or the EEA, I capture the relevant biological features of both kinda of mismatch: reduced fitness relative to the optimal environment. In PARs, the optimal environment is partially a result of the PAR itself; in classic mismatch cases, the optimal environment is a result of evolutionary history. In both cases, however, evolutionary history and developmental history interact such that there is a particular set of optimal environments for the organism in question. In short: this view integrates two important senses of mismatch in the literature.

Third: my account gives us the right result for putative cases of evolutionary mismatch. In the literature, scurvy, myopia, and novel predators are all cases in which mismatch is said to obtain—and my view agrees that they are (on the plausible assumption that all these things tend to inhibit fitness).

Ultimately, the optimal-environments view excludes non-mismatch cases (e.g. RubisCO) which one of the other views implausibly includes, and it includes mismatch cases (e.g. novel predators, poor fit of novel variants) which one of the other views implausibly excludes.

6 Objections

6.1 The EEA objection

The EEA or a similar notion (such as ancestral environment) is central to most accounts of evolutionary mismatch. Thus, a view of mismatch which eschews the EEA does not map well onto how mismatch is actually talked about by researchers. Worse, it is not clear how there is an *evolutionary* mismatch at all if the environmental history of the organism's lineage is not part of the story. This is surely an odd way to talk about evolutionary explanations of fitness

and health outcomes, which is ostensibly a major reason we want to discuss mismatch in the first place!

This worry is less serious than it first appears, because the EEA plays an indirect but clear explanatory role in the optimal environment concept: the EEA explains the physiology of the organism; the physiology of the organism explains the optimal environment. (This is not to say that the EEA is the entire explanation; rather, the EEA forms part of the explanation.) Thus, the actual biological phenomenon of evolutionary mismatch is not a discordance between the EEA and E_a, but the EEA is an important part of the explanation for the optimal environment. Moreover, due to the nature of the EEA, the optimal environment will plausibly share many significant features (e.g. energetic resources or temperature) with the EEA.

The emergence of new variants can have implications for mismatch, as well. Some organisms are variants which may be less fit in their EEA and more fit in a novel environment than most of their conspecifics, and a view of mismatch which privileges the EEA seems likely to run into difficulties explaining this: is mismatch no longer a meaningful category with respect to a new variant? That seems peculiar. On the optimal-environments view, mismatch for the variant is decreased relative to other, older variants in the population. By decentering the EEA, mismatch can still be quantified with respect to new variants (or the organisms which instantiate them).

An additional benefit of my view is that it makes no implicit suggestion of the EEA as Eden: I do not privilege the EEA as a time when adaptation was complete. Adaptation is an ongoing process due to the variable nature of both environments and gene pools. In most environments, even ancestral environments for which an organism has evolved, some check on the population is likely to remain. Because the EEA is often (always?) not the optimal environment for a given organism, the optimal-environments view captures this ongoing adaptive friction.

Further, my account of mismatch helps us to understand what is occurring with invasive populations: the organisms in the population are enjoying dramatically increased fitness in a novel environment; in other words, there is *less* evolutionary mismatch in the novel invaded environment than there was in the population's EEA. A sense of mismatch dependent on the EEA seems obliged to remain silent regarding invasive species.

These considerations—new variants, the ongoing nature of adaptation, and invasive populations—are all specific advantages in favor of decentering the EEA with respect to the mismatch concept.

Some might worry here that I provide too much of an assist to the mismatch theorist: given that they so often rely on claims about the EEA to motivate a mismatch hypothesis, perhaps I should not propose a modification which does not require them to keep the EEA front and center. All I can say to this concern is that I propose to rationally reconstruct the mismatch concept. To the extent that this makes mismatch hypotheses clearer or easier to evaluate, I see my account as successful.

6.2 The welfare objection

As I noted in section 3.1 on page 7, the mismatch literature often describes mismatch as a cause of health problems. For example, Lloyd et al. (2011) suggest in their general characterization of mismatch that mismatch can have a health effect or a fitness effect. As Nesse and Williams (1994) (235-236) say, some diseases which occur late in life (e.g. certain cancers) may well have little or no fitness effect, despite their devastating impact on human health and happiness. If vulnerability to these diseases is only actualized in novel environments, one might think that this is an example of an evolutionary mismatch—even if there is no commensurate health effect. Myopia may be another example of a 'mismatch' with respect to health, but which has no fitness effect in novel, contemporary environments. Li et al. (2018) say that mismatch is "adaptive lag" such that adaptive psychological or physiological mechanisms have not had time to adapt to certain environmental changes, and they explicitly include cases where the adaptive lag affects health but not fitness. (Note the similarity of this view to that defended in Cofnas (2016), discussed above in section 3.2.) Given these examples from the literature, one might worry that I am too hasty to exclude health outcomes as possible cases of mismatch. I treat fitness effects of the right kind as both necessary and sufficient for mismatch to obtain, whereas these cases from the literature seem to treat fitness effects as sufficient but perhaps not necessary. This disagreement with some of the literature may be unappealing.

I will defend both a weaker claim and a stronger claim with respect to this objection.

My weaker claim is that given the obscurity of the current discussion of evolutionary mismatch, it is worth trying to nail down some less-ambiguous features of the phenomenon. Surely extinctions driven by novel environmental factors are paradigm cases of increased mismatch, and as such we should be concerned with fitness effects—a more pressing concern for biologists and ecologists. We may think that there is a way to work out a health-focused characterization of mismatch as well but, given that the ultimate explanations involved are supposed to be evolutionary ones, we ought to start with a fitness characterization. Evolution by natural selection, after all, involves differential fitness—not differential health. Working out the interaction between the health and fitness 'versions' of mismatch might then be a next step (which I do not attempt here).

My stronger claim is that if fitness mismatches and health mismatches will not always co-occur, then we will often be talking about rather different phenomena such that we need to distinguish them. If only fitness mismatch is directly visible to natural selection, then it is not clear how health mismatch is actually *evolutionary*. We might give be able to give an evolutionary explanation for vulnerability to a particular health outcome, but in the absence of a fitness effect the outcome remains evolutionarily neutral. Calling both of these things 'evolutionary mismatch' when only one of them is possibly implicated in selection seems to me an employment of a misleading taxonomy.

To sharpen this point: the rapidity with which environments have changed is supposed to be a primary cause of particular mismatch cases. For example, Sih (2013) use the term "human-induced rapid environmental change," or HIREC, to describe some of the mismatch-relevant changes. Insufficient time to adapt is treated as a major part of the explanation for mismatch in, e.g., Li et al. (2018) discussed above. In the absence of fitness effects, however, there is no adaptation anyway. In other words: we are told that mismatch occurs in part because natural selection occurs more slowly than does environmental change. But mismatch which is restricted to health effects will persist even if the ostensibly novel environment also persists long enough for natural selection to work. Consequently, treating fitness mismatch and health mismatch as variants of the same basic phenomenon strikes me as often likely to obscure rather than clarify—especially if we want a mismatch concept which is viable outside of evolutionary medicine.

6.3 The misfire objection

PAR failure may occasionally make for a hard case with respect to the optimal-environments account of mismatch, particularly as described by Matthewson and Griffiths (2017).[21] The paper identifies four ways of "going wrong" biologically. The fourth way, "heuristic failure", is of particular interest here. A heuristic failure occurs when an organism initiates a particular developmental trajectory which turns out to be 'wrong'. They refer to the type of water flea (*Daphnia*) discussed in Boersma et al. (1998) and Gluckman et al. (2005). *Daphnia* grows a helmet-like shell structure and a tail spike in the presence of predators and ordinarily does not without predators. If a female *Daphnia* develops these structures, her offspring will also develop these structures without respect to predation risk in their own environments. Naturally, this sort of development is costly, and is only appropriate in the presence of predators. As such, developing the helmet and tail spike in the absence of predators reduces fitness relative to the choice *not* to develop—but helmeted, spikey *Daphnia* will not have lower fitness relative to environments with a predation risk (the way adults in utero during the Dutch famine might have lower fitness in calorie-rich environments than calorie-poor environments). In other words, *Daphnia*'s heuristic failure is a failure to employ the optimal strategy for an environment—rather than a case of an environment not being optimal. This failure of fit between development and environment seems, let us say, mismatchy, and perhaps the optimal-environments view is weaker for its failure to diagnose it as mismatch.

The distinction I made suggests my answer: evolutionary mismatch explanations are normally explanations of events *caused by environmental changes*. *Daphnia*'s heuristic failure is not caused by an environmental change, but by a developmental choice (so to speak). Reduction in fitness occurs not relative

[21] Thanks to an anonymous reviewer for drawing my attention to this point.

to environments but relative to developmental trajectories. As such, it is not properly a case of evolutionary mismatch. I suggest, then, that developmental or behavioral choices which are inappropriate be treated as 'misfires'.[22] These misfires would overlap significantly (perhaps entirely) with mismatch type 4 (misresponse-inducing) from Cofnas (2016). Perhaps these misfires would be adaptive in the EEA (or in the predicted environment) but are not the fitness-maximizing choice for the actual environment—or perhaps they might also occur at the local level in the EEA (as when all of *Daphnia*'s local predators die out or migrate, leaving her offspring to develop inappropriately). Note, then, that some examples of misfires will also be examples of evolutionary mismatch and some will not. This cross-classification strikes me as a good reason to distinguish the concepts, and to employ both concepts where appropriate.

6.4 The overbroadness objection

The reader may object that the account is overbroad, and that this sense of mismatch includes too many non-mismatch phenomena. This worry has three prongs: (1) it implies that mismatch obtains for most (perhaps all) organisms; (2) it conflates mismatch with insufficient adaptation without distinguishing the ways insufficient adaptation can occur; (3) it suggests we talk about fitness using a different vocabulary without justifying the additional complexity. As a result of these concerns, the thought might go, the optimal-environments view does not seem to add anything of scientific interest.[23] I engage with each worry in turn.

The first worry—that 'mismatch' (as defined on my view) is universal and, therefore, boring—can take two forms. (1a) suggests that an improved environment can *always* be imagined for any given organism: there is always at least one feature that can be altered to enhance the fitness of the organism—say, an infinitesimal but conceivable increase in energetic resources or mating opportunities. This approach appears to assume that the fitness of all organisms can be improved upon limitlessly, and that for any given organism there is no conceivable upper bound on its fitness. This seems clearly wrong: even if nothing else acts as a physiological limit (which seems unlikely), gamete production seems likely to hit a ceiling in sexual reproducers. In asexual reproducers there are typically limits on lifespan and rate of reproduction. (1b) makes a subtler point: given that few if any organisms find themselves in their optimal environment, mismatch is so pervasive that its presence is unremarkable. I agree: the *mere* presence of mismatch is relatively unremarkable. The degree of mismatch and variations in mismatch, however, can be critically important: the sudden jump in mismatch for the dinosaurs 65 million years ago—the sudden increase in distance between the dinosaurs' optimal and actual environments—helps

[22] My use of the term is inspired by Maner and Kenrick (2010)'s discussion of social anxiety.

[23] I am grateful to an anonymous reviewer for these helpful points.

explain the K-T extinction. Variations in mismatch—where environmental changes cause decreases in mismatch for some organisms, and increases for others—can help explain evolutionary change. As a practical matter, a biologist might not often be explicitly concerned with the question of whether mismatch obtains *at all*, but surely extent of, cause of, and variation in mismatch are all worthy objects of investigation.

The second worry is that this view simply defines mismatch as sub-maximal fitness, when really mismatch ought to be seen as a particular path to sub-maximal fitness—e.g. a functional account like that offered by Cofnas (2016). I think this objection is mistaken. Mismatch on my view does encompass a broad variety of biological phenomena, but it does not require that these be treated as an undifferentiated mass any more than defining fitness as expected reproductive success requires us to treat different fitness measurements as an undifferentiated mass. The ways that mismatch can happen—the plethora of ways, in other words, that organisms' optimal and actual environments can differ—are surely subject to interesting taxonomical work. As I suggested in my discussion of the teleofunctional account above, environmental inhibition of traits' proper functions are sometimes fitness-reducing, which suggests that one interesting kind of mismatch involves such an environmental inhibition. But as I showed, not all mismatches are caused by the inhibition of a proper function. I do not set out to create a mismatch taxonomy in this paper, but the account proposed certainly does not prevent such work.

The third worry suggests that if all genuine mismatch cases are really cases of sub-maximal fitness, we might as well stick to talking about fitness and ignore mismatch entirely. The antecedent here is true, but does not imply the consequent. The optimal-environments view calls our attention to at least two objects of investigation: the maximal fitness of an organism, and the optimal environment. Each of these is ecologically and evolutionarily relevant. The maximal fitness of an organism, if discovered, allows us to identify the ceiling of its likely rate of reproduction (assuming a fitness concept grounded in reproduction). In other words, it allows a researcher to determine the *maximal* ability of an organism to increase its share of the population, which in turn allows for a prediction of changes in biotic communities. The optimal environment, if known, allows ecologists and evolutionists to identify the likely effects of a particular environmental change on the populations and communities they study. This view of mismatch places both of those ideas into a broader framework.

6.5 The objection of peculiar cases

Consider a case:

> **Gamete Harvester**: a group of extremely-well-resourced, morally-troubled scientists decide to prevent evolutionary mismatch for one human male. To do so, they raise Fred from zygote to death, carefully controlling his environmental factors to maximize sperm production

> and quality. Their reproductive technology is such that they successfully ensure that (1) Fred physiologically could not produce a single sperm more over the course of his life, and that (2) every single one of Fred's sperm is used to create a viable embryo, which is then raised to reproductive maturity. Consequently, Fred's descendant contribution to the next generation is many millions of offspring.[24]

Gamete Harvester suggests that the notion of 'optimal environment' as I have defined it can include some very peculiar cases. It may be counterintuitive to think that some highly artificial environments are the optimal environment for organisms. After all, surely including environments like that in the Gamete Harvester suggests that very few organisms ever evade massive mismatch. Perhaps it is wrong to use an account which allows in unnatural cases.

An initial response is immediately available: it seems unlikely that biologists will have any interest in biologically implausible hypothetical cases such as Gamete Harvester. Thus, the (ostensible) failure of the account in such a case is not particularly worrisome: biological concepts need not be compatible with all possible cases. I take it that this is in the spirit of the skepticism towards the "ingenious construction of fictional counterexamples" expressed in Millikan (1989).[25]

Now allow me to attempt some defense in depth, here, on the supposition that some critics will find the Millikan-style move above unappealing. To that end, I offer two further responses, one theoretical and the other practical. The theoretical response is quick: given the huge numbers of gametes produced by human males, why do men not achieve the level of reproductive success in the Gamete Harvester? Well, for the simple reason that they are never in such environments! But were they to be in such environments, then they would have that level of reproductive success. In other words, they would be fitter in the Gamete Harvester environment (though perhaps not happier). The optimal environment for the organism's physiology helps to explain its failure to achieve physiologically maximal reproductive success. (For the sake of the argument I conceded that such an absurd project is possible, but I do not think it is at all obvious that such an intervention could be achieved.)

The practical response draws on our conservation interests: although the EEA of an endangered species will presumably provide some insight on how best to maximize the reproductive success of its population, it seems plausible that simple historic reenactment will sometimes be inadequate. In other words, sometimes human intervention in highly artificial environments (such as zoos) may be necessary to preserve species of interest. The optimal-environments account of evolutionary mismatch thus helps to explain fitness variation across natural and artificial environments. In short: acknowledging that artificial environments may maximize fitness is actually a virtue of my account.

[24] It may be the case that fitness is not actually maximized in this case because certain facts of human physiology make this impossible, or it relies on the wrong fitness concept. I suspect the former is actually true. Nevertheless, I think the case worth discussing for the benefit of those who think otherwise.

[25] Thanks to an anonymous reviewer for suggesting this response.

An interesting case here is that of the Lord Howe Island Phasmid (*Dryococelus australis*), also known as the tree lobster. By the 1960s, the tree lobster was believed to be extinct—apparently as a result of predation by black rats, an invasive species on Lord Howe Island, to which the tree lobsters were endemic. In 2001, a small population of only 24 individuals was discovered living around a bush on (relatively) nearby Ball's Pyramid, an island only a few hundred meters wide. Two male-female pairs were taken from the island. One pair went to Insektus (a private facility in Sydney); the other went to the Melbourne Zoo. Although the Sydney pair reproduced successfully, the population quickly died out. The Melbourne Zoo, however, was successful: the habitat for the initial breeding pair was a climate-controlled glasshouse facility. Eggs (laid into a sand container specifically for that purpose) were then placed in an identical facility, and buried in different substrates to maximize chances of success. As of 2009, the Melbourne Zoo-raised population included 700 insects (and thousands of eggs), and plans are underway to repopulate Lord Howe Island with the tree lobster (Priddel et al. (2003); Honan (2008); Buckley et al. (2009); Carlile et al. (2009)).

The Melbourne Zoo environment was artificial—a peculiar case, in other words—in a number of ways: it was carefully climate-controlled; it was free of predators; it had a staff to administer nutrient solutions to sick tree lobsters (Honan (2008)). Given that the initial male and female brought to the zoo now have hundreds of living individual offspring (compared to a wild population still at similar numbers as when first discovered, per Carlile et al. (2009)), it seems obvious that a tree lobster's fitness will be substantially elevated in the zoo relative to the bush on Ball's Pyramid. Even if the zoo is not quite the tree lobster's optimal environment, I think it is plausible to say that there is a greater degree of mismatch for the tree lobster on Ball's Pyramid than at the Melbourne Zoo. In other words: by reducing the severity of mismatch for the tree lobster, the zoo was able to substantially improve reproductive outcomes and, thus, probability of species survival.

A final point: some of the counterintuitiveness of Gamete Harvester may come from the fact that it is a case of high fitness and low welfare.[26] The case, then, neatly illustrates why we ought to be cautious when treating fitness and health interchangeably—or at least imprecisely. This is an extreme example of how the two can come apart. I think this does not militate strongly in favor of a welfare-centered mismatch account, however: I assume that no actual clinician would recommend a treatment regimen like Gamete Harvester, no matter how valuable she generally found evolutionary mismatch as a clinical concept. For more on this, see section 6.2 above.

6.6 The elephants without tusks objection

Another result which might worry us: on my view, mismatch might stay the same or even be exacerbated for an organism at the same time that its relative

[26] Thanks to an anonymous reviewer for this point.

fitness increases. Consider a variant of elephant which is tuskless ($\neg TE$). Presumably, $\neg TE$'s optimal environment would be an environment where tusks are less essential. It should be easy to see why $\neg TE$ faces some degree of mismatch in the elephant's EEA: tusks aid in defense, in digging, and so forth. Environmental change is occurring, however. Over decades of hunting African elephants for their tusks, poachers appear to have exerted very strong selection pressure against developing tusks, resulting in a sharp increase in the number of elephants without tusks. In other words, selection against tusked elephants (TE) and in favor of $\neg TE$ is ongoing.[27]

In the poaching environment, $\neg TE$ has a higher relative fitness than TE, because poachers do not have a reason to target $\neg TE$. This is perhaps troubling for the optimal-environments account because $\neg TE$ is still in a state of evolutionary mismatch: its absolute fitness[28] (we shall assume) does not vary across poaching and non-poaching environments, and so the extent to which mismatch obtains for $\neg TE$ remains the same. In other words, $\neg TE$ is no less fit when poachers are around than it would be in its EEA—nor is $\neg TE$ more fit in absolute terms. Given that $\neg TE$ is apparently out-competing TE, though, one might think it odd to say that in the poaching environment $\neg TE$ faces evolutionary mismatch.

The worry about changes in relative fitness without a correlated change in mismatch illustrates an advantage for my view: a view of mismatch focused only on relative fitness values will offer no explanation for declining demographic and reproductive success for the population as a whole; it can only explain change in frequencies of particular variants. The optimal-environments view can explain the change in frequencies: because the absolute fitness of TE in the poaching environment crashed (due to the increased mismatch with TE's optimal environment), while the absolute fitness of $\neg TE$ held steady (due to no increase in mismatch), $\neg TE$ trends toward a larger frequency in the population. At the same time, the priority I place on absolute fitness in particular environments allows us to explain why the population as a whole may decline: even though $\neg TE$ has a higher relative fitness than TE, $\neg TE$ is still not in its optimal environment and thus is still not very fit in absolute terms.

In short: by defining mismatch as a difference of absolute fitness in actual versus optimal environments, the optimal environment view can explain both the phenomena that a relative fitness view of evolutionary mismatch can explain, and also some phenomena that it cannot.

6.7 The relative fitness objection

When we tell evolutionary stories, we tell stories of differential reproduction: evolution occurs not merely as a result of how well- (or poorly-) suited an organism is to its environment, but also as a result of how well-suited it is

[27] See Jachmann et al. (1995) and Owens and Owens (2009) for some discussion of this.

[28] Absolute fitness, as opposed to relative fitness.

relative to other members of its population. Change in the frequency of variants over time is not simply caused by the demographic success of a particular variant; it is caused by the relative demographic successes of all variants in the population.

With that in mind, consider:

> **Weird Mismatch**: two organisms, O_1 and O_2 are in an environment E_1 such that O_1 has a fitness of 2 and O_2 has a fitness of 1. Consider another environment, E_2, in which O_1 has a fitness of 4 and O_2 has a fitness of 8. For convenience, I will stipulate that E_2 is the optimal environment (as I have defined it here) for both O_1 and O_2.

By the view I have been developing, there is an evolutionary mismatch for both organisms in E_1. Yet we might think this is odd: while it is true that O_1 has reduced fitness in E_1 relative to E_2, it is also the case that O_1 has higher relative fitness. In E_1, O_1 will tend to increase in its proportion of the population relative to O_2, and yet in the environment where it is less "mismatched", O_1 will tend to decline as a proportion of the population.

One might think that an organism must surely be considered better-matched in an environment where it has a higher relative fitness and will trend towards a larger proportion of the population.[29] Indeed, one might suspect that a notion of mismatch which does not seem to predict evolutionary change properly is not a very useful concept at all.

I consider this the most forceful objection to my characterization, but I think that it can be resolved satisfactorily.

First: as in the examples mentioned in section 2 on page 3, the negative fitness effects of something like scurvy are treated as mismatch due to an environmental change—not 'mismatch' which is actually just lower relative fitness. After all, a sufficiently vitamin C-poor environment may lower the fitness of all members of the population, and it seems plausible that we would want to say that mismatch occurs. The relative fitness objection requires that we only acknowledge mismatch when environmental change induces lowered relative fitness. My read of the literature is that this simply does not marry up well with the way the concept is used: environmental change can cause evolutionary mismatch for all variants within a population. Weird Mismatch is only a problem if we commit to mismatch as reduced relative fitness, which gives us other counterintuitive results. This view of mismatch also does not seem to get us additional clear cases left out by my proposal, and does not seem to handle cases which cannot be handled simply by talking about relative fitness *simpliciter*.

Consider:

> **Extinction**: an island population P of large, flightless birds, long protected from serious predation, is exposed to humans. The birds, lack-

[29] Credit for this specific objection goes to Andrew Sih of UC Davis. Several other concerns raised in this section emerged as a result of our conversation, but not all were directly expressed the way I have raised them here. Any failures of clarity or plausibility are my own.

> ing fear or any other predation-avoidance adaptations, treat the new predators with curiosity or indifference, and thus are easily hunted. Consequently, fitness across all variants within P crashes and it goes extinct.

Like Island Prey in section 3.2, Extinction gives us a story of a population exposed to a radical environmental change which results in across-the-board fitness reductions within P. Were we to insist on a relative fitness view of mismatch, we would be obliged to say that no mismatch occurs in Extinction. I take it that if evolutionary mismatch can be anything in biology, it can be the sort of thing that results in an extinction event. Thus, the relative fitness view gives us the wrong result.

Finally: in the elephants without tusks objection (section 6.6 on page 21), I suggested that change in mismatch levels among the variants of a population—ostensibly as the result of environmental change—probably can be used to explain some kinds of evolutionary change. If so, then it seems the optimal-environments view will still allow us to talk about many of the sorts of biological phenomena for which we might be tempted to use a relative fitness approach to mismatch. Keeping the concepts distinct gives us a different way of thinking about connected phenomena. Further, doing so provides a new target of investigation: the optimal environment.

6.8 The types and tokens objection

Perhaps my focus on individual organisms does not sufficiently reflect biological practice, which is primarily concerned with the differential success of variants (types) rather than the success of particular individuals (tokens). By giving an account of mismatch focused on the individual organism, one might think, I am giving an account of a concept which is unlikely to be fruitful.

I will respond in three ways. First, defining biological concepts at the organism level is certainly done by philosophers of biology: cf. Mills and Beatty (1979) for a well-known example. Second, given the particular ways that mismatch is discussed in the literature (particularly the clinical literature), a focus on individual cases seems to meet the need of those doing research on evolutionary mismatch. Third, I think the optimal-environments account can plausibly be extended to variants in a population, something like this:

> **Variant mismatch**: evolutionary mismatch obtains for a variant V in a population P when V is in an actual environment E_a such that V's fitness is lower than it would be in an optimal environment E_{mf}.

As noted in footnote 16, I take it that this sense of optimal environment is not too different from the sense of optimal environments described in Gluckman and Hanson (2006) and Levins (1962, 1968). A particular variant's optimal environment is that in which its "best performance" (with respect to

fitness) occurs. Thus, extending Levins' view to think about mismatch, evolutionary mismatch obtains for any variant in a population not in that optimal environment. (Presumably this would be characterized as mean mismatch across all organisms with the variant in question, but I am not committed to this approach.)

The argument can run similar to the argument in section 5: if we grant that a particular variant can be fitter in some environments than others, then at least one of those environments will be the optimal environment. If we move to the level of variants, we will also likely have a constrained set of environments to consider (relative to an individual organism's optimal environment set), given the reduced need to consider many individual traits and how they might interact with different environments. This, in turn, should help us get some traction with the challenge of identifying optimal environments in the case of individual organisms. (Indeed, in many cases the variant level may be sufficient for the purposes of the researcher.)

7 Taking stock

In this paper, I have proposed that evolutionary mismatch be conceptualized as a relation between the fitness-maximizing, or optimal, environment for an organism, E_{mf} and the actual environment of the organism, E_a. I have given some reasons to think that this is a plausible concept of evolutionary mismatch. I have shown that the two primary competing views of evolutionary mismatch each count some non-mismatch cases as mismatch cases, and count some mismatch cases as non-mismatch cases. The view that I develop avoids these problems, while being amenable to incorporating the key insights of competing accounts.

The real test for the optimal-environments view will be its fruitfulness. If the view is useful, it will provide new ways to think about questions which lie close to the foundations of evolutionary biology. I have argued that the view gives us specific new targets for investigation—the physiologically maximal fitness of an organism, and the organism's optimal environment—and suggested that these concepts can play clarifying and explanatory roles in evolutionary biology, in ecology and conservation biology, in developmental biology and the study of phenotypic plasticity, and other areas of the life sciences.

Further development of this view will be beneficial. First, I have given a sketch of variant mismatch in the previous section, but it is not yet fully-developed. Conceptual work enabling the study of mismatch at a population level is an obvious next step. Second, I have not laid out a methodology for identifying maximal fitness or optimal environments. Third and finally, here I have treated on mismatch as a fitness phenomenon. Given that many mismatch researchers want to do clinical work with the concept, however, another necessary step will be to elucidate the relationship between health and fitness with respect to evolutionary mismatch. Work remains to be done, but I take

it that I have shown that the optimal-environments view is, at its foundation, plausible and worth developing further.

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