Molecular challenges to adaptationism

Abstract:

We examine how molecular challenges to adaptationism, as recently put into philosophical focus by Sarkar (2015), fare against different types of adaptationist theses that have been distinguished in the philosophical debate on adaptationism. Our aim is to defend a weak form of empirical adaptationism according to which a majority of phenotypic traits at the organismal level are fixed by natural selection. Sarkar (2015) indicates a possible puzzle for this view by claiming that the same arguments that challenge adaptationism at the level of the genome can potentially apply at higher levels. We argue that many of the disputes about the importance of neutrality of molecular evolution as a challenge to the adaptationist thesis stem from unprecise use of terms such as phenotypic traits or complex phenotypes. As an important step towards a solution of this problem, we propose a strategy that tracks how changes at the molecular level can cause phenotypic effects at the organismal level, and identifies as important those phenotypic changes where a new function at the organismal level was introduced. We argue that for such cases, it is justified to conclude that selection was responsible for the fixation of a new trait. While this does not yet vindicate empirical adaptationism until further empirical research is done, it does provides us with the strategy of testing it. It shows how the puzzle can be solved by pointing to cases where new and important functions at the organismal level arise and where it can be clearly shown that selection was strong enough to counteract the effects of drift.

1. Introduction

The debate on adaptationism, while prominent in biology from the late 19th century,[1] attracted philosophical interest after the famous *Spandrels* paper by Stephen Jay Gould and Richard Lewontin (1979). The philosophical debate on adaptationism focused primarily on conceptual and methodological issues such as how evolutionary explanations should look like; how exactly to formulate (and test) the adaptationist thesis; whether constraint hypotheses are genuine rivals to adaptive hypotheses etc. (Orzack & Forber, 2012). Also, it seems that many philosophers embraced Gould and Lewontin's view and rejected adaptationism as a problematic thesis. For instance, Dennet, in *Darwin's Dangerous Idea* (1995, p. 239) writes how the *Spandrels* paper was regarded by philosophers and other humanists as a refutation of adaptationism, while the same is not the case with the majority of biologists. Perhaps this was the reason why the challenge to adaptationism coming from molecular evolutionary biology, especially in the light of the neutral theory of molecular evolution (Kimura, 1983), has not been widely addressed by philosophers.

Those philosophers of biology who did consider themselves adaptationist did not think that the data coming from evolutionary molecular biology goes against their claims because they took adaptationism to be limited to non-molecular traits (Orzack & Sober, 1994). However, recently Sahotra Sarkar (2015) presented a challenge to adaptationism that can be extended to the non-molecular level as well. He argues that the same arguments that challenge adaptationism at the level of the genome can potentially apply at levels higher than that of genome architecture. Sarkar himself admits that this is a puzzle since there is no good reason

[1] Already Darwin in the later editions of the *Origin of species* discussed the relation between natural selection and other evolutionary factors.

to doubt that a significant number of phenotypic traits at the organismal level are results of selection.

In this paper we will examine this so-called genomic challenge to adaptationism, and argue that Sarkar's argument regarding the threat to the adaptationist thesis about the phenotypical traits at the organismal level does not hold. In order to show this, we will consider different versions of adaptationist thesis that are being considered in the philosophical debate on adaptationism: empirical, explanatory, and methodological adaptationism (Godfrey-Smith, 2001). On the backdrop of these views, we will defend a weak version of empirical adaptationism.

Adaptationists sometimes spell out their claim by stating that this concerns *important* phenotypic traits, or *complex* traits, thereby leaving it very vague what exactly is meant by these terms. For this reason we propose a strategy of mapping out how the changes at the molecular level bring about changes in phenotypes at the organismal level. We argue that in those cases where the change at the molecular level brings about a new function at the organismal level, we are, prima facie, justified to conclude that selection will act (or has acted) to fix that trait (or eliminate it).

As an informative case study we use the phenomenon of neofunctionalization *via* gene duplication since it provides us with illustrative examples of how genetically redundant material fixed at the molecular level by neutral forces, can serve as a basis for new functions at the organismal level that are then fixed by natural selection. In this respect, we examine three cases for which it can be established that such scenarios have occurred. These cases do not by any means provide evidence that such events do occur in majority of phenotypic traits at the organismal level, but they do provide us with a good starting point for empirically assessing adaptationist claims. In any case, we believe that they illustrate that Sarkar's (2015)

claim that there is a puzzle involved in explaining how it is possible that a significant number of phenotypic traits were fixed by natural selection, is not an actual puzzle.

2. Challenges to adaptationism: a non-adaptationist hypothesis is a better explanation of genomic and organismal complexity

According to the neutral theory of molecular evolution (Kimura, 1983), most genetic variation at the molecular level is a consequence of mutation and random genetic drift and therefore cannot be explained by invoking natural selection. There are two basic reasons for this claim: first, the stochastic theory of population genetics which is mathematical in nature, and second, molecular genetics where molecular advances have brought more direct insight into molecular evolution (Kimura, 1983). The theory asserts that only a minute fraction of DNA (or RNA) changes are adaptive. Also, most of the intraspecific variability at the molecular level, including protein and DNA polymorphism are neutral, which means that the majority of polymorphic alleles are maintained by the balance between mutational input and random extinction (Kimura, 1989). Most important evidence in support of the neutral theory of molecular evolution is the constancy of the rates of amino acid or nucleotide substitutions per site and the fact that functionally less important molecules evolve faster.

Another important set of data that support the claims of neutral theory are the data regarding the complexity of eukaryotic genomes. Eukaryotes on average have more genes, larger proteins, longer and more elaborate regulatory regions, and unique models of gene expression (Koonin, 2004). Insights into eukaryotic genome architecture has, in Sarkar's (2015) words, created a puzzle since the emergence of their structural and behavioral complexity is hard to explain by invoking natural selection. Firstly, the amount of DNA in genomes of closely related eukaryotic species varies to a substantial degree. Secondly, there is no correlation between this amount and the morphological complexity of a species. Thirdly,

eukaryotes contain much more DNA than seems necessary for the specification of their proteins (Gregory, 2001).

Michael Lynch (2007) argues that many aspects of genomic architecture, gene structure and developmental pathways are difficult to explain without invoking genetic drift and mutation as the main factors. Complexity of eukaryotic genomes is hard to explain in adaptive terms because each addition to a gene increases its vulnerability to mutational inactivation which should lead to elimination under selective pressures. Furthermore, due to relatively small population sizes of multicellular species, they are expected to accumulate gratuitous gene-structural changes without any direct selection for them (Lynch, 2007, pp. 8599-8600).

Eugene Koonin (2004) also talks about the puzzle that along with 'useful' complex features, eukaryote genomes have accumulated many 'selfish' elements which have no function for the organism containing them. He examines two potential interpretations of the evolution of biological complexity and entropy of the genomes: according to one, the increase in complexity is an adaptation, but the mechanisms leading to it are imperfect which led to entropy increase. According to the other, the increase in complexity is a by-product of entropy growth, which is a neutral process. He opts for the Lynch and Conery's (2003) theory that implies the second option by invoking the effective size of the evolving population. It is worth noting that both options might be perceived as a threat to adaptationism at the genomic level (even though the second option represents a more serious problem). From this Koonin (2004) concludes that nonadaptationist explanation is the current null hypothesis on the origin of biological complexity.

Sarkar (2015) summarizes these conclusions in what he calls the core argument against adaptationism:

> P1: The physical properties of DNA and its cellular environment lead to increased genome size and its baroque structure.

P2: Genome size is negatively correlated with population size.

P3: Selection acts against larger genomes.

P4: Small population sizes prevent the elimination of features selected against unless selection is very strong.

C: Genomes increase in size, diversity, and so on and persist even though selection acts against these features. (Sarkar, 2015, pp. 519-520)

Since the aim of this article is to examine neutralism as a threat to adaptationism, we will not examine alternative adaptationist explanations of genome complexity. We will take it as granted that genomic research has so far been successful in corroborating Kimura's neutral theory of molecular evolution. What remains to be seen is whether from this follows that adaptationism cannot hold even at the level of organismal phenotypes. It seems that Sarkar (2015, p. 529) would endorse this implication, since he says that the core argument can be applied beyond the level of genome architecture. However, it must be noted that according to Sarkar this possibility really presents a puzzle since he claims that there is no good reason to doubt that "a significant number of phenotypic features at the organismic level (and probably at higher levels of organization) are results of selection (…)" (Sarkar, 2015, p. 529). Nevertheless, we think that the puzzle can be dispelled. In the next section, we begin to show how that could be done.

3. Does the thesis of neutrality of molecular evolution threaten adaptationism regarding phenotypic traits?

Many adaptationists have discarded the neutrality of molecular evolution as a possible threat to adaptationism since adaptationism is concerned primarily with phenotypic, non-molecular

traits (Orzack & Sober, 1994). However, this view depends on what we take to constitute phenotypic traits. On the standard definition, phenotype comprises observable features of an organism, that are based on the coding of the genotype (Rittner & McCabe, 2004). On the basis of this definition, one might include molecules such as RNA and proteins into organism's phenotype. These molecules are not visible at the organismal level, but are observable and can be thus considered as parts of the phenotype. Since the neutral theory claims that most of the intraspecific variability at the molecular level (including protein and DNA polymorphism) is selectively neutral, it would follow that many phenotypic traits are neutral as well. However, this view would not capture the standard usage of the term phenotypic traits. Even Kimura (1983, pp. 55-97), the author of the neutral theory, does not consider molecular evolution to be concerned with phenotypic traits, and actually marks a contrast between molecular evolutionary rates and phenotypic evolutionary rates.[2]

In order to make the adaptationist view more specific, we will follow Maeso et al. (2012) in distinguishing between different levels of biological organization. They note that phenotype should be considered as a continuum across different scales of biological complexity, but can be divided into three levels for practical reasons: organismal level (individual features such as anatomy, physiology, behavior, etc.); cellular level (cell movements, secretory capacities, morphology, organellar composition, etc.); and molecular level (all observed traits below the cellular level: transcriptome, proteome, biochemical properties, chromatin structure, etc.).

We think that it is important to make these distinctions because in many debates the so-called adaptationists and anti-adaptationists (or pluralists) seem to argue past each other due

[2] When discussing phenotypic evolution Kimura focuses on fossil records, so phenotypic traits in this case refer to morphological characters. In addition, it can be noted that Kimura does not think that the neutral theory denies the role of natural selection in determining the course of adaptive evolution (Kimura, 1989).

to different uses of the term trait, phenotypic trait, complex trait etc.[3] For instance, very often salient features such as wings, limbs or eyes are taken as paradigmatic adaptive traits. Nevertheless, one can argue that we should consider them collections of traits, rather than as single traits. In addition, some adaptationists claim that complex traits must be a result of natural selection, but it is hard to determine how to test that claim if we take it that many factors must have been involved in the evolution of some complex trait (which can also be taken as a set of simpler traits), some of which might have to do with purely physical laws (limitations such as Gould and Lewontin's (1979) example of body plans), some with neutral factors, and some with natural selection.

We will take it that adaptationists standardly have in mind the organismal phenotype when they claim that most phenotypic traits are adaptations (Orzack & Sober, 1994; Maynard Smith, 1978). This means that we will not be concerned with those authors who are adaptationist about the genomic level as well, and offer adaptive stories that go against the neutral theory.[4]

We will argue that in order to identify important phenotypic traits that adaptationism refers to we need to trace how changes at the molecular level cause effects at the organismal level. In cases where this link can be clearly established and where a new function at the organismal level is introduced, it is justified to assume that selection will act to fix (or delete) such a trait. Also, there appears to be plenty of evidence that genomic complexity as a product of neutral evolution creates a precondition for strong selection pressures in evolution of phenotypic traits. In the next section we provide grounding for this claim.

[3] For an informal but relevant debate on this topic, where the issues of clear definition of phenotypic traits are an important part of the discussion, see this blog: http://sandwalk.blogspot.hr/2011/02/dawkins-darwin-drift-and-neutral-theory.html.

[4] For a survey and criticisms of attempts to offer adaptationist stories that would account for the architecture of the human genome and eukaryotic genomes in general see Sarkar (2014).

4. A synergism between nonadaptive evolution at the DNA level and adaptive evolution at the phenotypic level

We follow Lynch (2007, p. 8601) in his view that there is a synergism between nonadaptive evolution at the molecular level and adaptive evolution at the phenotypic level. He argues that from the neutrality of molecular evolution does not follow that we need to abandon the view that many of the external morphological and behavioral manifestations in today's organisms are adaptive. In order to demonstrate how this synergism might work we will examine gene duplication as one of the evolutionary neutral events that is important source of new functions that selection can act upon.

Gene duplication is a major source for genome evolution, and the principal one for eukaryotes (Koonin, 2011).[5] Genomic research has shown that the majority of genes in any genome belong to families of paralogs. Furthermore, it is the main source of functional diversity on the level of the genotype (Lynch & Conery, 2003; Ponting, 2008; Conant & Wolfe, 2008). This is due to the fact that after gene duplication each of the gene copies can evolve independently and acquire a functional novelty.

Susumu Ohno (1970) first proposed that the extra gene copies that are created by duplication are redundant; since the original gene already performs the necessary function, the extra copies are free from selective pressures and can acquire new mutations without being deleted by the purifying selection. This, in turn, allows them to acquire an additional function (whether it is a completely new one, or one of the functions performed by the original gene). This maintenance of duplicate genes can be a product of neutral evolution, or of selection.

[5] However, it should be noted that there are many other mechanisms underlying the origins of new genes other than gene duplication. Novel genes can arise from messenger RNAs of ancestral genes, protein-coding genes metamorphosed into new RNA genes, genomic parasites co-opted as new genes and new protein. Moreover, RNA genes can be composed from previously non-functional sequences (Kaessmann, 2010). Here we take gene duplication as a good example because it is a well-studied source of new genes and potentially new functions at the level of organismal phenotype.

Several models of maintenance of gene duplicates have been proposed, most notably neofunctionalization, subfunctionalization, and increased gene-dosage advantage. In neofunctionalization model, one copy of the gene keeps the original function and is maintained by purifying selection, while the redundant copy is free to evolve and potentially acquire new function (Ohno, 1970; Walsh, 1995; Force, et al., 1999). The copy is free to evolve because after duplication, the copy is freed from purifying selection which leads to the acceleration of evolutionary rate. In case there are some adaptive changes in the new copy, positive selection can act to fix that changes. Thus, the neofunctionalization model invokes positive selection as a mechanism responsible for the fixation of a new function. In the subfunctionalization model, on the other hand, both duplicates accumulate mutations trough drift. Here, the original function can be divided between the two duplicates, each taking part of the original function (Force, et al., 1999; Lynch & Force, 2000). The two new functionally distinct copies are then preserved trough purifying selection. Another model is increased gene-dosage advantage according to which duplication is itself beneficial due to increased amount of gene product, and that is the reason why both duplicates become rapidly fixed (Konrad, Teufel, Grahnen, & Liberles, 2011)

There seems to be an agreement that certain amount of gene duplications have been fixed by positive selection (Romero & Palacios, 1997; Kondrashov, Rogozin, Wolf, & Koonin, 2002; Sebat, et al., 204; Hastings, 2007). Nevertheless, the question remains whether selection plays an important role in fixation of a significant fraction of gene duplications. This is an interesting question in the debate on adaptationism at the molecular level, but it need not have a direct consequences for adaptationism about organismal phenotypic traits. While the majority of molecular changes can be a result of neutral evolution, there is still a possibility that changes that result in consequences at the level of organismal phenotype, are under the strong influence of selection. We take the neofunctionalization model as possibly the most

interesting case because it produces entirely new functions. What remains to be examined is whether the majority of new functions at the level of organismal phenotypes actually are fixed by selection.

We will argue that there are clear cases where we can assume an adaptationist approach to the problem of phenotypic evolution. These are the cases where it can be established that a change at the molecular level had as a consequence a change in the phenotypic level that brought about a new function. In order to demonstrate this, we will use three case studies that illustrate this kind of situation.

5. Neofuntionalization: examples where new organismal phenotypic functions were fixed by positive selection

5.1 The case of color vision

Opsins are light-sensitive proteins in the photoreceptor cells of the retina that mediate the conversion of a photon of light into electrochemical signal. Genes that contain coding sequences for opsins, belong to the same gene family that accrued during our evolutionary past by the gene duplication processes and, then, by functional divergence, resulted in adaptive functional novelties. The trichromatic vision of Old World monkeys and primates represents a functional novelty conferring a high adaptive value on organisms bearing the trait of the gene family containing the corresponding gene duplicates (Golding & Dean, 1998, p. 359). The adaptive value can be inferred by studying the ecological consequences of the color vision acquisition: individual organisms having the functional novelty in question could easily explore and construct completely new ecological niches, which, then, brought about a significant impact on the related ecosystem. The described case suggests an incremental construction of fit between organisms and their environment which leads us to conclude that a selective mechanism is at work (Sterelny, 2006).

The described case of the evolution of trichromatic color vision represents a case where, unlike in most cases, the relationship between the genotypes and phenotypes is straightforward; a very simple change at the molecular level produces an important change at the phenotypic level for which we can be fairly certain that it brought advantage to the organisms bearing it.

Golding and Dean (1998) propose the same approach; in order to infer that an adaptive change took place, next to information on raw sequence and phylogeny, we need phenotypes. Thus, in cases where we can detect a direct link between a change at a molecular level and a change at the phenotypic level (Melin, et al., 2014) and where the change at the phenotypic level brings an incremental construction of fit between organisms and their environment, we are justified to conclude that the positive Darwinian selection is at work. The combination of information on phylogeny, structural information, and information on physiology and ecological conditions at the time of the fixation of this trait allows us to conclude that positive selection is responsible for the fixation of the trait.

5.2 The case of Digestive RNASE1 Genes in Leaf-Eating Monkeys

Pancreatic RNASE genes are a particularly illustrative example of neofunctionalization in leaf eating Columbine monkeys. Columbine monkeys have an important pancreatic enzyme RNASE1 that helps them digest bacterial ribonucleic acid (RNA), an important source of nitrogen. It was demonstrated that after the duplication of RNASE1 gene, the extra copy of the gene (called RNASE1B) mutated and acquired a new function of producing an enzyme that was more efficient in deriving nutrients from bacteria in the foregut (Zhang, Zhang, & Rosenberg, 2002) thereby making monkeys more efficient in extracting energy from leaves. Using molecular analyses and functional assays Zhang et al. have shown that the duplicated RNASE1 genes in two leaf-eating monkey species (Asian *Pygathrix nemaeus* and African *Colobus guereza*,) evolved rapidly under positive selection for improved digestive efficiency,

as a response to the increased demands for the enzyme for digesting bacterial RNA. In addition, it was shown that duplication occurred after the separation of African and Asian Colombines, which leads to conclusion that there were two separate duplication events that were followed by similar selection pressures (Zhang, 2006). Thus, this is also a case where a small functional change at the molecular level following gene duplication brought about new, salient function that increased organisms' fitness.

5.3. Evolution of antifreeze protein

Due to the high salt content, the waters of Arctic and Antarctic can reach -2 degrees Celsius. Fishes living in such an environment need to develop a mechanism that will enable their blood not to freeze in order to survive. Antifreeze proteins in the blood bind to ice crystals and deter the joining of additional water molecules which decreases the temperature of macroscopic ice expansion below the colligative freezing point.

It has been shown that antifreeze proteins (AFPs) arose independently in different polar marine teleost lineages due to strong selection pressures in the late Cenozoic sea-level glaciation (Deng, Deng, Ye, He, & Chen, 2010). Several fish AFPs evolved by duplication from ancestral genes with different functions. For example, type III AFPs of polar zoarcoid fishes are homologous with the small C-terminal domain of sialic acid synthase (SAS), a cytoplasmic enzyme that catalyzes intracellular synthesis of sialic acids from N-acetylmannosamine or Man-NAc-6-phosphate and phosphoenolpyuverate. Type III AFPs are secreted plasma proteins that bind to ice crystals and prevent ice growth. Deng et al. (2010) suggest that enzymatic and antifreeze functions within the same ancestral SAS molecule point

to adaptive conflict that was resolved by gene duplication and neofunctionalization of the copied gene.

The three cases we considered illustrate the situation where a change at the molecular level brings forth a new, salient function for highly complex multi-cellular individual organisms.[6] We argue that in such cases an adaptationist approach to phenotypic evolution (at the organismal level) is, at least prima facie, justified. That is, they actually represent cases where, as required by Sarkar (2015), selection was strong enough to counteract the effects of drift that would follow as a consequence of the relatively small populations.

One might wonder whether these kind of cases occur relatively rarely and whether one is justified in reaching an adaptationist conclusion from examining just a couple of examples. Nevertheless, we believe that this approach at least provides us with the strategy of spelling out the adaptationist thesis that can be put to empirical test. This strategy is sensitive to the hard problem (also acknowledged by Sarkar[7]) of producing precise estimates for population sizes and selection coefficients for historical populations. We agree that it is a problem to establish the action of selection with certainty, but we think that a careful examination of various factors, from the changes on the molecular level to the data on ecology, can provide us with the most reliable results.

In the philosophical debate on adaptationism, there has been many claims regarding the testability of adaptationist thesis, and the specific ways of defining adaptationism. In the next

[6] We acknowledge the fact that in any cases where positive selection fixes traits at the molecular level, there are methods for detecting the act of selection, such as showing that nonsynonymous nucleotide substitutions exceed synonymous nucleotide substitutions during the early stages after duplication. However, we take it that this is not enough to conclusively establish adaptationism at the molecular level, and this is not the adaptationist thesis that we are concerned with. We limit adaptationism to the claim about the evolution of phenotypic traits. In addition, in order to reach the conclusion about the act of selection, further information about environmental conditions and the usefulness of the new evolved function should be taken into consideration.

[7] Sarkar (2015) addresses this issue and acknowledges that the lack of this information makes his argument 'qualitative' instead of quantitative, but still not merely verbal as adaptationist 'just so stories'. We think that our strategy is a good starting point in avoiding the accusation of offering merely verbal accounts of evolution of phenotypic traits. This strategy is still not backed by enough quantitative data, but we take it that it represents a substantial step towards testing the adaptationist hypothesis regarding organismal phenotypic traits. In our case, this consists in tracing how the mechanisms at the molecular level cause organismal phenotypic changes.

section we examine three theses of adaptationism as presented in the philosophical debate and analyze how they fare with respect to the so-called genomic challenge.

6. Philosophical debate on adaptationism: three kinds of adaptationist views

Peter Godfrey-Smith (2001) has identified three main types of adaptationism which became a standard reference point in the philosophical debates on the topic[8]: empirical, explanatory and methodological adaptationism. Empirical adaptationism is the view according to which natural selection is a powerful and ubiquitous force that drives the evolutionary change. According to this view, in order to explain and predict evolutionary phenomena it is enough to focus on the role played by natural selection and ignore other possible causal factors. This view is taken as an empirical claim about the biological world that we should be able to put to test.

Explanatory adaptationism appears to be a weaker claim that stresses the importance of selection for explaining the apparent design of organisms, which is taken to be the most important biological phenomenon. However, explanatory adaptationism leaves open the possibility that natural selection is not the most ubiquitous source of evolutionary change. On the other hand, methodological adaptationism is the claim about scientific methodology; it claims that the best approach for investigating evolutionary processes is to look for features of good adaptation and design. This methodological principle is also compatible with the claim that natural selection is not the most pervasive or even frequent force of evolutionary change.

The thesis of methodological adaptationism will not be of interest in this paper since we are concerned with the possibility of determining the role of selection in the evolution of phenotypic traits. However, methodological adaptationism is a claim that can be taken as

[8] There have also been proposals to divide the adaptationst views in more detail. For instance, Lewens (2009) distinguishes seven types of adaptationism (while acknowledging the main three types, which he then subdivides into distinct subtypes).

valid regardless of the actual role that selection played in evolution. The thesis of adaptationism that is most interesting in the perspective of this paper is the claim that natural selection has been the only important cause of most of the phenotypic traits found in most species (Sober, 1998, str. 72). This is standardly taken to be empirical adaptationism. In this respect, we want to make some clarifications concerning the type of empirical adaptationism that we wish to defend.

We will not enter into the debate on whether adaptationism needs to defend the optimality criterion, as proposed by Orzack and Sober's (Orzack & Sober, 1994) specification of adaptationist claim. According to the optimality criterion, for trait T of an individual in a given population, the claim (O) is true, where (O) states: Natural selection is a sufficient explanation of the evolution of T, and T is locally optimal. We will be concerned with the thesis regarding the ubiquity of natural selection, and not the specific thesis that relies on the claims of optimality. One of the reasons for this is the fact that the optimality criterion seems to be relative to the way we specify what it takes for something to be considered optimal. Also, the genomic challenge to adaptationism as presented by Sarkar (2015) does not enter into the debate on optimality. Already this is enough to qualify our version of adaptationism as *weak*. However, we also further add the criterion (that is often taken for granted in the debate) that the claim about ubiquity of selection applies only to organismal phenotypic traits. Thus, weak adaptationism is a type of empirical adaptationism according to which natural selection is a ubiquitous force that operates at the level of organismal phenotypic traits.

Introduction of this middle position, we believe, clarifies some of the supposed ambiguity in views of the authors, such as Dawkins (1999) and Dennet (1995), who were identified by Godfrey-Smith (2001, pp. 339,340) as being in between empirical and explanatory adaptationism. For example, Godfrey-Smith identifies Dawkins as being explanatory adaptationist and holding a view according to which selection is rare, but occurs often enough

to answer the big evolutionary questions (which is, according to Godfrey-Smith, sufficient for not including Dawkins among empirical adaptationists).

To support the claim that Dawkins is not empirical adaptationist, Godfrey-Smith cites the fact that Dawkins does not seem to have anything invested in the debate between neutralists and adaptationists. Dawkins, for instance, argues that biochemical controversy over neutralism is quite different from the adaptationist controversy since adaptationism is concerned with "(…) whether, given that we are dealing with a phenotypic effect big enough to see and ask questions about, we should assume that it is the product of natural selection." (Dawkins R. , 1999, p. 32) He adds that biochemists' neutral mutations are not mutations at all for those who look at gross morphology, physiology, and behavior. He concludes that "If a whole-organism biologist sees a genetically determined difference among phenotypes, he already knows he cannot be dealing with neutrality in the sense of the modern controversy among biochemical geneticists." (Dawkins R. , 1999, p. 32).

However, as suggested by Godfrey-Smith (2001, pp. 340,341), sometimes Dawkins seems to be making a more ambitious claim about the large amount of biological world that was shaped by natural selection. For instance he refers to the "sheer hugeness" of the phenomenon (Dawkins R. , 1986, p. 15). This would appear to make his view ambiguous between explanatory and empirical adaptationism.

In our view, Dawkins can be taken to be a weak empirical adaptationist since his discussion is clearly limited to organismal phenotypic traits. We take it that this reading better captures Dawkins' claims about adaptations than the thesis of explanatory adaptationism as explained by Godfrey-Smith (2001). Due to the fact that Godfrey-Smith does not make a distinction regarding the levels at which phenotypic traits are considered, he cannot interpret Dawkins as an empirical adaptationist (since, for instance, it is clear that selection is not so ubiquitous at the genomic level). For this reason, he takes Dawkins' adaptationism to mainly

consist in the claim that adaptation and apparent design are the most important biological problems worth considering. However, the problem with this view (explanatory adaptationism) is that it is not empirically testable and merely expresses a personal preference or inclination for one type of biological explanations. While it seems that Dawkins really does endorse this view on the importance of apparent design in evolutionary biology, we believe that this view does not exhaust his adaptationism. Thus, we agree with Godfrey-Smith that explanatory adaptationism is not a testable claim, but disagree with him that Dawkins cannot be interpreted as a weak empirical adaptationist, which is a testable claim.

We take it that Dawkins' emphasis on the importance of phenotypic effects that are "big enough" and that are of interest to "whole-organism biologists" illustrates the need to introduce distinctions between different levels of phenotypic effects, and thereby to narrow the scope of the adaptationist thesis, as we have suggested. Moreover, if we consider Dawkins' view in the light of our strategy of trying to specify very closely what we mean by organismal phenotypic traits (that are products of selection), then we can see that the view does not fall prey to the accusation of untestability.

7. Conclusion

We have examined the so-called genomic challenge to adaptationism and argued that a weak version of empirical adaptationism is not challenged by the results from molecular evolutionary biology, as argued by Sarkar (2015). We defined weak empirical adaptationism as a view according to which majority of phenotypic changes at the organismal level were products of natural selection. Our proposal is that such a view can be tested by careful examination of molecular changes that bring about changes at the phenotypic level. For this purpose, we briefly examined three examples of cases where it can be concluded that

selection fixed the phenotypic trait in question and was strong enough to counteract the effects of drift due to the small population size.

At this point, we do not wish to claim that weak empirical adaptationism is vindicated by the current data. What we contend, nevertheless, is that it is certainly a testable empirical claim. Thus, weak empirical adaptationism as a view about the power of selection in accounting for evolution of phenotypic traits at the organismal level, is not threatened by the data coming from molecular evolutionary biology.

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