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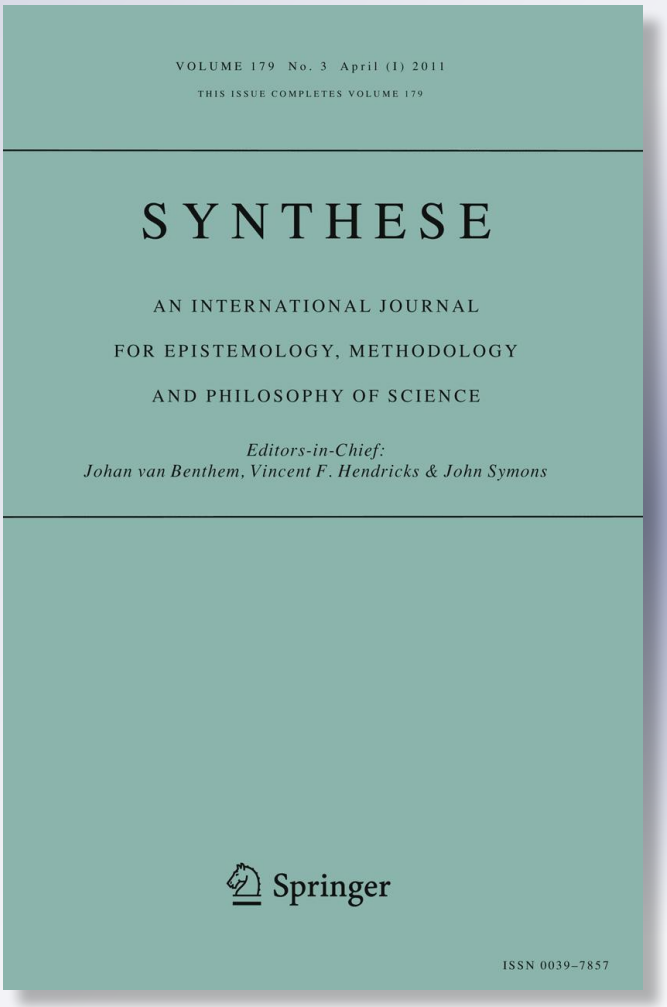

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Replication without replicators

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Abstract According to a once influential view of selection, it consists of repeated cycles of replication and interaction. It has been argued that this view is wrong: replication is not necessary for evolution by natural selection. I analyze the nine most influential arguments for this claim and defend the replication–interaction conception of selection against these objections. In order to do so, however, the replication–interaction conception of selection needs to be modified significantly. My proposal is that replication is not the copying of an entity, the replicator, but the copying of a property. Thus, we can have a replication process without there being a replicator that is being copied.

Keywords Selection · Replication · Interaction · Properties · Entities · Microbial evolution · Clonal evolution

1 Introduction

There are two distinct ways of conceiving of selection processes. According to one, selection is heritable variation of fitness. According to the other, it consists of repeated cycles of replication and interaction. These two models of selection[1] give us very different ways of formulating evolutionary explanations and they even yield different kinds of evolutionary explanations. We need to decide which one to use.

The aim of this paper is to defend the less popular of these two models of selection: the replication–interaction model. But I argue that in order to defend this model of

[1] I will refer to these two ways of conceiving of selection as two *models* of selection, acknowledging that my use of the concept of models is different from the way this term is used in biology.

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selection from a number of influential objections, we need to modify it significantly. But let us first see what these two competing models of selection are.

According to the first model (Lewontin 1970; Maynard Smith 1987), selection should be described as heritable variation of fitness. A typical formulation is the following (see also Lewontin 1970, p. 1; Endler 1986, p. 4; Ridley 1996, pp. 71–72; Godfrey-Smith 2007, p. 515):

> A sufficient mechanism for evolution by natural selection is contained in three propositions:
>
> 1. There is variation in morphological, physiological, or behavioral traits among members of a species (the principle of variation).
> 2. The variation is in part heritable, so that individuals resemble their relations more than they resemble unrelated individuals and, in particular, offspring resemble their parents (the principle of heredity).
> 3. Different variants leave different numbers of offspring either in immediate or remote generations (the principle of differential fitness) (Lewontin 1980, p. 76).[2]

According to the alternative concept, selection consists in repeated cycles of two separate processes. As Ernst Mayr says, "natural selection is actually a two-step process, the first one consisting of the production of genetically different individuals (variation), while the survival and reproductive success of these individuals is determined in the second step, the actual selection process (Mayr 1991, p. 68, see also Mayr 1982, pp. 519–520, 2001, p. 117, 1978): David Hull calls these two steps replication and interaction (Hull 1981, 1988, 2001). He defines selection as:

> The repeated cycles of replication and environmental interaction so structured that environmental interaction causes replication to be differential. (Hull 2001, p. 53)

In turn, Hull defines the unit of replication, the 'replicator' as "an entity that passes on its structure largely intact in successive replications" (Hull 1988, p. 408. Cf. Hull 1980, p. 318, for a slightly different definition). The unit of interaction, 'interactor', on the other hand, is defined as the "entity that interacts as a cohesive whole with its environment in such a way that this interaction causes replication to be differential" (Hull 1988, p. 408. Cf. Hull 1980, p. 318).

This replication–interaction model of selection was introduced as an improvement on the heritable variation of fitness model, and it is supposed to clarify a number of details left implicit therein. More precisely, the replication–interaction model has been thought to help us to understand what is at stake in the units of selection debate: if selection is replication plus interaction, then we should not talk about the units of selection, but rather the units of replication and the units of interaction, which may not be (and in fact most often are not) the same. The thought is that the replication–interaction distinction in itself will not solve this problem, but it is supposed

[2] According to Lewontin, each of these three propositions are necessary for evolution by natural selection (besides being jointly sufficient). See Lewontin (1980, p. 76).

to help us to formulate the problem in such a way that would make it possible to tackle it (see, e.g., Lewontin 1970, p. 7; Brandon 1982, 1988 and especially Lloyd 2001).

In the last decade or so, more and more philosophers have argued against the replication–interaction model. Their main claim is that replication is not necessary for evolution by natural selection or, as I will put briefly, for selection.[3] I will defend the replication–interaction conception of selection against these objections and argue that replication is indeed necessary for selection. In order to do this, however, the replication–interaction conception of selection needs to be modified significantly. If, however, the replication–interaction model, or at least a revised version thereof, no longer faces these objections, then we have good reason to revive it, as it may enjoy some significant explanatory advantages over the heritable variation of fitness model.

But the aim of this paper can be stated more modestly: even if the replication–interaction model did not have any immediate explanatory advantages over the heritable variation of fitness model (although I will argue in the last section that it does), if we manage to formulate a version of the replication–interaction model that is not susceptible to the objections that have been raised against the original version of the replication–interaction model, then we could conclude that this model (or, rather, a version thereof) should not be dismissed as inferior to the heritable variation of fitness model. The real aim of this paper is to put (a version of) the replication–interaction model back on the table so that we can decide which model of selection is the more fruitful to use.

Depending on how we think of the relation between these two ways of thinking about selection, we may not have to decide for one and reject the other. It may be possible to consider them as complementary explanatory schemes. If this is indeed an option, then outlining a plausible version of the replication–interaction model is all the more important.

The structure of the paper is the following. First, I give a characterization of the original version of the replication–interaction model and especially the concept of replication that it uses (Sect. 2) and enumerate the nine most influential objections against it (Sect. 3). After reinterpreting the concept of replication as the replication of properties (rather than entities) (Sects. 4, 5), I point out that the objections against the original replication–interaction model fail to apply if we conceive of replication this way (Sect. 6). Finally, I consider some potential explanatory advantages in favor of the revised model of selection (Sects. 7, 8).

[3] There is an important terminological difference in the way the concept of selection is being used in the literature. Some ask whether replication is necessary for *evolution by natural selection* (Okasha 2007; Godfrey-Smith 2007), others ask whether replication is necessary for *selection* itself (Hull 1988; Neander 1995; Hull 2001; Nanay 2005). I assume that these are two different ways of asking the same question (the question of whether replication is necessary for *evolution by natural selection*) and I'll use the latter formulation, because it's simpler. If the reader prefers the former one, she should read 'evolution by natural selection' instead of 'selection' in what follows.

2 Replication as the copying of replicators

The concept of replicator and that of interactor were originally introduced by Richard Dawkins and David Hull.[4] The distinction between replicators and interactors, as Robert Brandon puts it, "is best seen as a generalization of the traditional genotype–phenotype distinction" (Brandon 1996, p. 125,).[5]

In the familiar case of natural selection, the replicator is the gene, whereas one possible interactor is the organism itself. Genes are passed on and the organism interacts with the environment in such a way that this interaction causes the replication of genes to be differential. In other words, those genes that are responsible for the development of more successful organisms are more likely to replicate.

To go back to Hull's definition of replicator as "an entity that passes on its structure largely intact in successive replications" (Hull 1988, p. 408), we are faced with the question of what "passing on the structure" implies. One of Richard Dawkins' various definitions of replicator shows clearly that 'passing on' or 'copying' need not mean something particularly strong. Dawkins writes: "I define a replicator as anything in the universe of which copies are made" (Dawkins 1982b, p. 83).

John Maynard Smith and Eörs Szathmáry give a more restrictive and more informative definition of replication, which is also quite weak. They claim that any entity can count as a replicator that can come to existence "only if there is a pre-existing structure of the same kind in the vicinity" (Maynard and Szathmary 1995, p. 41). In other words, replicator is "an entity that only arises by division or copying of a pre-existing entity" (Maynard and Szathmary 1995, p. 58).

Intuitively two aspects of "passing on" or "copying" seem necessary: similarity and causal connection. This intuition has been captured by Peter Godfrey-Smith's definition:

> Y is a replicate of X if and only if: (i) X and Y are similar (in some relevant respects), and (ii) X was causally involved in the production of Y in a way responsible for the similarity of Y to X. (Godfrey-Smith 2000, p. 414)

It is important to note that even this notion is very weak (see also Nanay 2002). Godfrey-Smith himself points out that photocopying, for example, is a replication process according to this definition. A way of strengthening the definition of replicator in such a way that photocopying would not qualify as replication is the following. David Hull writes that "in order to function as a replicator, an entity must have structure and be able to pass on this structure in a sequence of replications. If all a gene did was to serve as a template for producing copy after copy of itself without these copies in turn producing additional copies, it could not function as a replicator" (Hull 1988, p. 409,

[4] Dawkins (1976/1989, 1982a, 1982b). Hull (1980, 1988). Dawkins contrasted replicators with vehicles, and the term of interaction was introduced by David Hull.

[5] One could question whether the concepts of replicator should be conceived as the generalisation of the concept of gene or, as Brandon seems to suggest, that of genotype. In what follows, I will assume that the concept of replicator is most commonly interpreted to refer to tokens and not types and therefore is the generalisation of the concept of gene.

see also Aunger 2002, pp. 73–74 and esp. p. 76 for a similar way of thinking about replicators).

The problem with this proposed definition is that it is probably too strong. The concept of replicator, if it is to be understood as the generalization of the concept of gene, should not be taken to be an entity that copies *itself*. The gene, for example, certainly does not make copies of itself (Lewontin 1991, pp. 48–49). Genes are copied by a complicated mechanism. In other words, taken in isolation, genes are not "able to pass on [their] structure in a sequence of replications".

So it seems that most definitions of replicator are so weak that clearly non-biological processes will also count as replication. And it is not clear that there is a way of strengthening these definitions in a plausible manner.[6] Is this a problem? No. The concept of replication is not supposed to be a sufficient, but merely a necessary condition for selection. Many more conditions need to be satisfied before a replication process gives rise to selection. More precisely, it needs to be followed by an interaction process in such a way that this interaction makes the next round of replication differential. Thus, if there are trivial, non-biological cases of replication, like photocopying, that is not a problem: the theory of selection is supposed to tell us which replication processes are biologically interesting: which ones give rise to selection and which ones do not. As we shall see in the next section, there are problems with the definitions of replication I have discussed in this section, but they lie elsewhere.

What is in common between these definitions of replication is that they all take for granted that replication is a copying process, where entities (the replicators) are being copied. These entities (the replicators) are taken to be concrete particulars. I will question this assumption and argue that not entities, but properties are being copied in the replication process. I will argue that we need to redefine replication in this way in order to salvage the replication–interaction model of selection. But before outlining and defending this new account of selection, and a new definition of replication, I will go through the most important objections against the concept of replication and against the replication–interaction model in general.

3 What is wrong with the replication–interaction account of selection?

A number of arguments have been given against the replication–interaction conception of selection. I will consider nine such objections.

The first three aim to point out that the replication–interaction model is not general enough: there are cases of selection where replication is not involved.

(1) *Selection can happen if there is sufficient phenotypic parent–offspring resemblance*. Replication is not needed (Okasha 2007, p. 15).

Suppose that in a population rich individuals are more likely to survive and reproduce (because, say, the vaccination against a widespread and serious illness is very expensive). They also tend to give their money to their offspring, who will then be

[6] Another, very different, way of strengthening the definition of replication was suggested by James Griesemer, who replaces the concept of replication with the more restrictive concept of reproduction. I analyze this concept and Griesemer's move in general as well as the problems it raises in Sect. 6 (7).

richer than other members of the population. There is selection in this population (for being rich): there is heritable variation of fitness. But as being rich is an entirely acquired trait with no genetic component, there is no replication. We have selection without replication.[7]

(2) *Selection can happen if there is comparative parent–offspring resemblance (phenotypic or genotypic)*. Replication, which is supposed to have a very high degree of fidelity, is not necessary.[8]

Peter Godfrey-Smith writes: "It is sufficient for evolution by natural selection (given other conditions) that parent and offspring be *more* similar than randomly chosen individuals of different generations. So any absolute degree of parent/offspring similarity (except 100%) will be sufficient in some contexts and insufficient in others" (Godfrey-Smith 2007, p. 515).

(3) *There are ways of transmitting information (extragenetic inheritance, cultural transmission) that do not count as replication but that are (given other conditions) sufficient for selection* (Okasha 2007, p. 15; Avital and Jablonka 2000, p. 359; Jablonka and Lamb 1995; Richerson and Boyd 2005, Chap. 3).

Samir Okasha summarizes this line of objection: "cultural and behavioural, as well as genetic, inheritance can generate the parent–offspring similarity needed for an evolutionary response to selection. These inheritance channels do not involve particles bequeathing 'structural copies of themselves to succeeding generations'. So evolutionary changes mediated by cultural and behavioural inheritance cannot be described as the differential transmission of replicators" (Okasha 2007, p. 15).

The second group of objections questions the status of replicators and interactors as evolved entities:

(4) *The replication–interaction model takes the notion of replicators and interactors for granted as fully evolved entities*. Thus, under this conception it would not be possible to give a selective explanation for the evolution of replicators and interactors themselves (Griesemer 2000, p. 71; Okasha 2007, p. 16). We may be able to explain the evolution of the DNA molecule in terms of some other replicators (Brandon 1990; Hull 2001), but we cannot explain the evolution of replicators *per se*.

(5) *We can apply the explanatory scheme of natural selection in cases where neither replicators nor interactors were fully evolved*. Replication is defined as a very accurate copying process, but in the early stages of evolution, copying was not at all very accurate (Maynard and Szathmary 1995). Similarly, the unit of interaction is defined as a cohesive whole, but again, there were various stages

[7] Here is an analogous example (taken from real life): Cases in which large mothers have large offspring as a result of laying eggs with larger food reserves are a mainstay of the literature on 'maternal effects' in evolutionary theory (Mousseau and Fox 1998; Uller 2008). These offspring have higher fitness than their smaller rivals. Since this component of offspring fitness is an entirely acquired trait with no genetic component, we have selection without replication, that is, without the copying of replicators.

[8] Dawkins and Hull both admit in their original account that the replication process does not need to have 100% fidelity, but they both emphasize that the degree of fidelity needs to be high.

of evolution where what interacted with the environment (what should be considered interactors) were not at all cohesive wholes (Michod 1999). Thus, the replication–interaction model is not applicable to early stages of evolution.

The third group of objections focuses on developmental considerations:

(6) *The notion of replication ignores development* (Griesemer 2000, pp. 72–74). In the replication–interaction model, interaction follows replication and then a new round of replication follows interaction, but development is missing from this picture. How do we get from replicators to interactors? The replication–interaction model fails to specify this (as even the advocates of the model admit: see Brandon 1990).

Further, an extremely influential argument in the last decade has been that the genetically coded and the developmental part of evolutionary explanations cannot and should not be separated from one another. More precisely, it has been argued that there is no sharp distinction between 'genetic' and 'environmental' developmental causes. As a result, it is a mistake to talk about genes as replicators: it is unclear whether the term replicator is meaningful at all, but if it is, it must denote the entire life-cycle, which makes many features of the original replicator–interactor distinction problematic (see esp. Griffiths and Gray 1994, p. 304. See Griffiths and Gray 1997; Oyama et al. 2001 but see also Sterelny and Kitcher 1988; Sterelny et al. 1996).

(7) *Griesemer's notion of 'reproducers' can do all the theoretical work the notion of replicators can* and it is not problematic from a developmental point of view (Griesemer 1998, p. 194, 2000, pp. 74–76, 2002, p. 105).

The process of reproduction is defined as "multiplication with material overlap of propagules with developmental capacity" (Griesemer 2000, pp. 74–75). Griesemer writes that "the relation between parents and offspring is not merely one of resemblance, but rather is one of material overlap. Offspring are made from parts of the parents, they are not merely similar objects made from wholly distinct materials" (Griesemer 2000, p. 74). His argument for the claim that material overlap is a necessary feature of the parent–offspring relation that is necessary for selection is related to (6) above: the replica needs to be capable of developing into a replicable entity and it is difficult to see how this capacity could be transmitted without any material overlap. Finally, the following two objections are not supposed to be fatal, but are worth mentioning:

(8) *The replication–interaction model involves two types of entities* (replicators, interactors), not one (the heritable variation of fitness Okasha 2007, p. 15). It is important to note that (i) it is not clear how we are supposed to count the types of entities presupposed in the two models and whether it makes sense to compare these numbers and (ii) the number of entities presupposed in an explanation is not a clear indication of its simplicity/complexity.

(9) *The replication–interaction model is a historical paradox*: Darwin did not use these notions (or anything like them) (Gould 2002). One may wonder why this would be a problem: the replication–interaction account is supposed to be a model of selection and not of Darwin's views on selection.

Most of these objections (objections (1)–(7)) are very influential and they are also valid if we consider some of the original definitions of replication. Recent attempts to redefine replication may or may not be able to address some of these objections (see especially Brandon 1990). I will not attempt to defend the original definition or minor modifications thereof. I will argue that if we radically revise the concept of replication and interpret replication as the copying of a property and not of an entity, then these objections lose their appeal.

What I attempt to do in the next three sections is to outline a new account of replication and show that while most of these objections are very powerful with regards to the original conception of replication, they lose their force if we modify this notion the way I suggest. I outline a new conception of replication in the next two sections and then reconsider these objections in Sect. 6.

4 Replication as the copying of properties

My proposal is that replication is not the copying of an entity, the replicator, but the copying of a property.[9] Thus, we can have a replication process without there being a replicator that is being copied.

It is important to clarify what is meant by entities and properties here. The cup in front of me is an entity. It has lots of properties, some interesting, some others less so. Its colour is one property, its shape is another one, etc. Thus, the copying of an entity and the copying of one of the properties of this entity are very different processes. Properties are always properties of entities of course. But it is possible to copy a property of an entity without thereby copying the entity itself.[10]

The definition of replication is then the following (mirroring the definition suggested by Godfrey-Smith 2000, p. 414):

> Property P of object a is a replica of property Q of object b if and only if:
>
> (i) P is similar to Q and
> (ii) Q is causally involved in the production of P in a way responsible for the similarity of P to Q.

Some further care is needed to explicate (i), (ii), as well as the nature of properties in the new definition of replication.

What do we need to assume about the nature of the properties that are being replicated? First, in the discussion of the replication of properties, I will talk about these properties as tropes: abstract particulars that are logically incapable of being present in two different individuals at the same time. Thus, even if P is a perfect replica of Q, P and Q are different properties. But we can easily modify this terminology to

[9] This idea was first introduced but not defended in Nanay (2002, p. 113).

[10] Biologists call the properties of organisms 'traits'. If someone prefers this concept to the concept of properties, she can rephrase my definition of replication as the copying of traits. But as the replicated properties are not necessarily properties of an organism, I will talk about properties, rather than traits, in what follows in order to preserve generality.

fit a conception of properties as universals.[11] Second, the properties that are being replicated can be both intrinsic and relational properties, although I suspect that most biologically interesting cases of replication will involve the former.[12]

To turn to (i), the first thing to note is that the similarity between two properties is notoriously difficult to define. A restriction that could help is that P and Q needs to be properties of the same kind. If Q is a color property, Q must also be a color property. If Q is a shape property, P must also be a shape property.[13]

Second, what degree of similarity is required in order for (i) to be satisfied?[14] 100%? 98%? The answer is clearly not a fixed percentage.

P of a is considered to be similar to Q of b, if the degree of similarity between them is higher than it is between the corresponding properties of two randomly chosen individuals in the population. Thus, (i) could be rephrased as: 'the degree of similarity between P and Q is higher than it is between the corresponding properties of two randomly chosen individuals in the population'.

To turn to (ii), some may find it unclear what 'causal responsibility for similarity' is supposed to be. More precisely, (ii) as it stands commits us to a view of causation according to which properties are causally efficacious. But not everybody endorses such account of causation: Davidson holds that causal relation holds between events and properties play no role in causation (Davidson 1993). There may be independent reasons to doubt the Davidsonian picture (see Heil and Mele 1993) and there may be independent reasons in favour of one or another account of causation according to which property-instances or property-types are causally efficacious (Robb 1997; Nanay 2009a). But those who are reluctant to give up the Davidsonian account of causation should read (ii) as a claim about causal explanation (as Davidson does allow

[11] If we opt for this, then we need to assume that the properties that are being replicated are super-determinates. An old and respectable way of characterizing the relation between properties is the determinable–determinate relation (Johnson 1921; Funkhouser 2006). To use the classic example, being red is determinate of being colored, but determinable of being scarlet. Properties with no further determinates (if there are any) are known as super-determinates (Johnson 1921, p. 185; Armstrong 1961, p. 59). It has been argued that super-determinate properties are the only causally efficacious properties (Armstrong 1997; Mellor 1995; Gillett and Rives 2005; Crane, forthcoming, but see Yablo 1992 for an influential dissenting view). As the properties that are being replicated are supposed to be causally efficacious, I assume that they are super-determinate properties. What is being replicated is the property of being 5.37 inches and not the property of being longer than 5 inches.

[12] There is a thorny question about how to individuate replication processes according to this proposal. If several different properties of an entity get replicated (as in the case of gene-replication or of photocopying), does this mean that we have to posit several different but parallel replication processes? A simpler way of describing the replication in these cases would be to say that the conjunction of these properties is what gets replicated. A consequence of this is that the replication of an entity could also be described as the replication of a (conjunctive) property.

[13] It is not clear that this restriction solves all the problems about the nature of similarity between different properties. After all, it is not clear whether orange is more similar to red or to yellow. In what follows, I assume that there is some kind of ordering of properties within a specific kind of property (such as shape, size or color) that could be used to determine the degree of similarity between properties. One helpful way of conceptualizing the ordering of properties is Austen Clark's (and Quine's) account of 'quality space' (Clark 1993, 2000, see also Nanay 2009b).

[14] Note that the same question arises if we try to clarify what is meant by hereditability: if a trait-type is hereditable, how much do its instances in different generations need to resemble each other?

for properties in causal explanation): Q causally explains the production of P as well as the similarity between P and Q.[15]

An important feature of this definition is that a and b are not necessarily objects of the same kind. Object b may be an apple and object a may be a color photograph of this apple. The color of the photograph can be a replica of the color of the apple under my definition, but this does not mean that the objects themselves are replicas or copies or replicators in the old sense of the word.

A consequence of this is that we need to think differently about the concept of lineage. According to the original version of the replication–interaction model, the primary ontological category of evolutionary phenomena is that of lineages (see Hull 1980, pp. 327–329). A lineage is traditionally defined as "an entity that changes indefinitely through time as a result of replication and interaction" (Hull 1980, p. 327). Hull claims that replicators form a lineage and so do interactors: the replicator-lineage is part of the interactor-lineage. But if we accept the concept of replication I proposed here, then replicators cannot form a lineage, because they do not exist.

Is this a problem for my account? I don't think so. Note that if we accept objection (2) or objection (3) above, then we need to give up the concept of the lineage of replicators anyway. In both (2) and (3), there is selection without there being a lineage of replicators. Further, why should we think that the lineage that is considered to be the primary ontological category of evolutionary phenomena must be the lineage *of replicators*? Hull has a hierarchical conception of lineages, where both replicators and interactors form lineages. As he writes:

> An important characteristic of lineages is that each lower-level lineage is included as part of all subsequent higher-level lineages. Gene-lineages are included physically as part of organism-lineages. Assuming that these organisms in turn form colonies, the relevant organism-lineages are included in colony-lineages, and so on, up to the level of biological species. (Hull 1980, pp. 327–328)

So even if we cannot talk about the lineage of replicators, this does not make the concept of lineage obsolete as, according to Hull, interactors also form lineages. In the examples that are used in objections (2) and (3) above, there is selection, but the parent–offspring resemblance is achieved not by means of replicators but by some other means, say, by epigenetic inheritance. The crucial point is that in these cases we can still talk about lineages (see Nanay, forthcoming, ftnt 8): epigenetic inheritance, for example, leads to lineages, but not to lineages of replicators. As we shall see in Sect. 6, if we accept the definition of replication I propose here, then in cases like

[15] A further potential worry about this concept of causal relevance is this. It is difficult enough to determine what entity is causally responsible for what, but when it comes to the causal relevance of properties, things get even more blurry. How could we keep track of which property of an object is causally responsible for something? It is important that this worry is an epistemic one. If we accept a non-Davidsonian account of causation, then there is always a fact of the matter about what property of an object is causally responsible for something (in fact, this constitutes the main objection to Davidson's account, see Dretske 1989, pp. 1–2; Sosa 1993; McLaughlin 1993; Kim 1993). Further, if the causal relevance of properties is salient enough for these criticisms of Davidson's theory to get off the ground, then our epistemic access to the causal relevance of properties cannot be all that poor.

the ones we encounter in objections (2) and (3), we have selection, but we also have replication and lineages. But we do not have replicators.

With this new definition of replication in hand, it is a good idea to examine whether the most frequently quoted examples of replication (and the most frequently quoted examples of what is not replication) would count as replication under this definition.

The copying of genes, the most central example, will count as replication under this definition, as there are properties of my gene that are replicas of my mother's gene. A number of properties of genes gets replicated but let us focus on the property of having a certain nucleotide-sequence: the nucleotide-sequence of my mother's gene and the nucleotide-sequence of my gene are replicas: (i) they are similar (the degree of similarity between my nucleotide-sequence and my mother's is higher than it is between two randomly chosen nucleotide-sequences in the population) and (ii) the nucleotide-sequence of my mother's gene is causally involved in the production of the nucleotide-sequence of my gene in a way responsible for the similarity between the two nucleotide-sequences. Thus, the copying of genes will count as replication.

It is worth noting that under some conception of what genes are, what is being replicated in the gene replication case is taken to be (something like) a property, and not an entity. George Williams defines a gene as "any hereditary information for which there is a favorable or unfavorable selection bias equal to several or many times its rate of endogenous change" (Williams 1996, p. 25). Or, even more explicitly: "A gene is not a DNA molecule; it is the transcribable information coded by the molecule (Wimsatt 1992, p. 11). Under this conception of genes, what gets replicated is not an entity or replicator, such as the DNA molecule, but a piece of information, that is, a property of the DNA molecule. If we take sides with Williams' conception of genes, which I certainly do not want to do in this paper, and we want to maintain that genes replicate, then we need to endorse my account of replication.

How about the blueness of my eyes and the blueness of my father's eyes? This is the prime example of an inter-generational similarity that is *not* replication and, encouragingly, it does not count as replication according to my definition either. The blueness of my father's eyes is not causally responsible for the similarity between the blueness of my eyes and his.

The notion of replication I argued for in this section is, of course, very weak, as weak as Godfrey-Smith's (and significantly weaker than Hull's notion).[16] Not every replication process will be particularly interesting from an evolutionary point of view: photocopying or multiple orgasms, for example, may not. But this is what we should expect: the notion of replication is only the starting point for selectionist explanations. Many additional criteria need to be met in order for replication to lead to selection (or to cumulative selection) and many more criteria need to be met in order

[16] Further, in the definition above, I was talking about a property P of object a and property Q of object b. But if we allow a and b to be the same object, then even the sustenance of a solid object could be described as replication. Maybe there are interesting cases of sustenance as replication (for example, when a societal structure remains the same even though the members of the society change), but if we do not want these cases to count as replication, we can set a further constraint on replication, namely, that a and b must be different.

for selection to explain why things are the way they are (see, for example, Nanay 2005).

5 Is this still a version of the replication–interaction model?

With this new concept of replication as the copying of properties, we can now outline the modified version of the replication–interaction model. It is quite simple: selection consists of the repeated cycles of replication and interaction so structured that interaction makes replication in the next generation differential. Importantly, the cycle of replication is to be understood as the copying of properties, as I outlined in the last section.[17]

Two possible worries need to be addressed at this point. First, one may object that the account we ended up with is not interestingly different from the original versions of the replication–interaction model. Second, one may also object that the account we ended up with is so far removed from the original replication–interaction model that we should not consider it a version thereof. In fact, it may even be closer to the heritable variation of fitness model: the replication of properties could be thought to be quite similar to the notion of heritability, one of the three central concepts of the heritable variation of fitness model. I will take these worries in turn.

The first worry was that the switch from talking about entities to talking about properties cannot possibly be more than metaphysical slight of hand as every claim about entities can be rephrased as a claim about properties (and maybe even vice versa). I agree that merely switching from entity-talk to property-talk is unlikely to have any deep consequences, but my proposed new definition of replication does more than this. The definition of replication as the copying of properties is very different from the original definition of replication as the copying of entities. There are processes that count as replication according to the former, but not the latter definition, as we shall see in the next section (see especially the kinds of cases in Sect. 6(1), (2), (3)). Thus, the change in the way to think about replication is not a meaningless formal change, but a real substantive one.

Second, one may wonder at this point whether the revised notion of replication I argue for is different from the notion of heritability. The worry would be the following: by revising the notion of replication in such a way that it is properties and not entities that replicate, I may have ended up with the notion of heredity. Heredity, according to the heritable variation of fitness model, is necessary for selection; so it will not come as a surprise that replication, as I define it, will also be necessary for selection.

My response is threefold. First, following James Griesemer, we need to distinguish heredity (a relation), heritability (an ability) and inheritance (a process) (Griesemer

[17] In order to give a full account of selection along the lines I suggested, we may also need a new definition of the notion of interaction that would correspond to the new notion of replication. I am not convinced that the notion of interaction needs radical revision of the kind I aim to undertake in this paper with regards to the notion of replication (this move would also imply taking sides in various issues with regards to the metaphysics of causation, which I would like to avoid). So, for now, I will assume that the notion of interaction is defined in terms of interactors and not in terms of properties, just as it is in the original versions of the replication–interaction model. Nothing I say in this paper relies on the notion of interaction.

2005, pp. 77–78). Replication, as I defined it, is a process, so it is not clear how it could be considered to be equivalent to heritability or heredity, the concepts that play a key role in the heritable variation of fitness model. As inheritance is also a process, maybe it could be considered to be equivalent to replication, but it is much less clear that inheritance (as opposed to heredity or heritability) is an essential part of the heritable variation of fitness model. Importantly, although both replication and inheritance are processes, they are very different processes.

Second, my notion of replication is different from the notion of heritability: the blueness of my eyes is heritable (and inherited), but it does not replicate according to my account. Further, heredity is a relation between types (Griesemer 2005, esp. pp. 77–78), whereas the replicated properties in the process of replication are property-instances (and not property-types). Nevertheless, the two concepts could be thought to be related in intimate ways: for every heritable trait, there is a replication process that (casually) explains the heritability of this trait.[18]

Third, these two notions play very different roles in the two models of selection. Replication, unlike heritability, is one of two distinct steps that constitute selection. Replication is followed by interaction, which, in turn, is followed by another round of replication. The model I am proposing is a two-step model of selection and replication is one of these two steps. The heritable variation of fitness model, in contrast, is not a two-step model: heredity is not temporally alternating with any other process. It is a delicate question what exact role the concept of heritability (or heredity, or inheritance) plays in the heritable variation of fitness model (see Griesemer 2005; Godfrey-Smith 2007), but whatever it is, it must be different from the role replication plays in my account: as my account, like original versions of the replication–interaction model, is a two step process with replication as one of these, whereas the heritable variation of fitness model is not a two step process.

6 Defending the new model of replication

I will go through the seven objections against the original replication–interaction model that I enumerated in Sect. 3 and attempt to point out that all of them can be adequately addressed if we accept the definition of replication I proposed in the last two sections.

(1) *Selection can happen if there is sufficient phenotypic parent–offspring resemblance.*

If we accept my definition, then phenotypic traits can replicate. Take the population where rich individuals are more likely to survive and reproduce and also tend to give their money to their offspring, who will then be richer than other members of the population. As being rich is an entirely acquired trait with no genetic component, we have selection without replication, that is, without the copying of replicators.

According to my account, in this population, there is a property that replicates: the property of being rich. My instantiation of this property is similar to my father's (in as

[18] If one believes that the replication–interaction model and the heritable variation of fitness model of selection do not exclude one another, but are rather complementary ways of describing a selection process, as I suggested in Sect. 1, then this way of thinking about the relation between the two may be a fruitful one.

much as the degree of similarity between my wealth and my father's is higher than it is between the wealth of two randomly chosen individuals in the population) and his wealth is causally responsible for this similarity. Thus, we do have selection in this population, but we also have replication.

(2) *Selection can happen if there is comparative parent–offspring resemblance (phenotypic or genotypic).*

My account, as we have seen, does not imply that replication must have a high degree of fidelity. It is enough if the degree of fidelity is comparably high. If P of a is a replica of Q of b, then P and Q must be similar, but as we have seen in the last section, P and Q are considered to be similar if the degree of similarity between them is higher than it is between the corresponding properties of two randomly chosen individuals in the population.

Thus, Godfrey-Smith is right: Selection can happen if there is comparative parent–offspring resemblance (Godfrey-Smith 2007, p. 515). But if there is comparative parent–offspring resemblance, then, under my definition, there is also replication.

(3) *There are other ways of transmitting information (extragenetic inheritance, cultural transmission) that do not count as replication but that are (given other conditions) sufficient for selection.*

My response is that some versions of both extragenetic inheritance and cultural transmission can count as replication. Nothing in my definition of replication suggests that the replicated property needs to be a property of the DNA. Thus, extragenetic properties can replicate as much as the properties of the DNA can. If property P of the offspring is similar to property Q of the parent and the latter is causally responsible for this similarity, then we do have replication, regardless of whether these properties can be called genotypic or not.

Similarly, some instances of cultural transmission can also be considered to be replication. The example of richness I used in the exposition of and response to the first objection, for instance, may count as a case of cultural replication. An advantage of my proposal is that it allows us to talk about cultural replication without positing entities such as memes. I will return to this question in Sect. 7.

(4) *The replication–interaction model takes the notion of replicator and interactor for granted as fully evolved entities. Thus, we cannot explain the evolution of replicators and interactors.*

My definition does not talk about replicators and interactors. And the properties that are being replicated are certainly not considered to be fully evolved. The objection is difficult to even formulate in the case of my account, as it is difficult to make sense how a property could be considered to be fully or less fully evolved.

But maybe the worry could be rephrased with regards to the notions of replication and interaction (rather than replicators and interactors). Maybe it is the notion of replication and interaction that the replication–interaction model takes for granted as fully evolved processes. As I hope to point out in response to the next objection, my notion of replication is applicable to copying processes that are certainly not fully evolved.

(5) *We can apply the explanatory scheme of natural selection in cases where neither the replicators nor interactors were fully evolved.*

In the early stages of evolution, copying was not at all very accurate (Maynard and Szathmary 1995). But my notion of replication, as we have seen in response to the second objection, is not defined as a very accurate copying process. The accuracy of copying needs to be only *comparatively* high. Thus, we can indeed talk about replication in early stages of evolution.[19]

(6) *The notion of replication ignores development.*

Development could be incorporated into the replication–interaction model even within the original framework with very little modification: the life cycle consists of not two but three phases: the replication of replicators, the development of interactors from replicators and the interaction of interactors (Brandon 1990, cf. Blute 2007). The same move is also open to my account.

A related worry was that if we take seriously the lesson Developmental Systems Theory teaches us, then we should not draw a sharp distinction between 'genetic' and 'environmental' developmental causes. Neither do we need to, in order to salvage the notion of replication. As we have seen in response to objection (3), replication is not necessarily 'genetic', thus, the blurring of the distinction between 'genetic' and 'environmental' does not jeopardize this concept.

(7) *Griesemer's notion of 'reproducers' can do all the theoretical work the notion of replicators can.*

The notion of replication does not imply that there is a material overlap between the replicas. But Griesemer argues that there must be such material overlap between them. Why? Because, as he says, "offspring must be born with the capacity to acquire the capacity to reproduce" (Griesemer 2000, p. 74), and such complex capacity is unlikely to be transmitted "to unorganized bulk matter". Thus, he concludes, there need to be a material overlap between the parent and the offspring.

This argument is far from being conclusive even against the original replication–interaction model. It could be argued that if the transmitting mechanism is complex enough, then even extremely complex capacities can be transmitted without any material overlap. A replicator is not an entity that copies *itself*. The gene, for example, certainly does not make copies of itself (Lewontin 1991, pp. 48–49). Genes are copied by a complicated mechanism and replicators in general can also be copied by complicated mechanisms. But if this mechanism is complicated enough, then it is unclear why there would be any need for a material overlap.

[19] A similar worry is raised by the original notion of interactor, which I will address only in passing, as the aim of this paper is to reevaluate the concept of replication and not of interaction. The unit of interaction has been defined as an entity that interacts with the environment as a cohesive whole, but there were various stages of evolution where what interacted with the environment (what should be considered interactors) were not at all cohesive wholes (Michod 1999). It needs to be noted that although the notion of interactor was in fact defined by David Hull as the entity that interacts with the environment as a cohesive whole, this is by no means a necessary feature of the notion of interactor. Cohesive whole or not, interactor is the entity that interacts with the environment in such a way that this makes the next round of replication differential.

Further, if the Developmental Systems Theory is correct, then we cannot differentiate between genetic and environmental developmental causes. Thus, not all the capacity that is needed for the replicator/reproducer to acquire the capacity to develop into a replicable entity needs to be inherited. As the Developmental Systems Theory suggests, the inherited and the environmental component of such ability should not be differentiated. But in this case, we have no reason to believe that the inheritance of such capacity would require some hard conditions, such as material overlap, as it is unclear how much of this capacity is in fact inherited.

Even worse, as Peter Godfrey-Smith pointed out, there are cases of evolution by natural selection where there is no material overlap between parent and offspring (Godfrey-Smith 2009, pp. 83–84). The evolution of retroviruses is an actual example of this: the offspring of these viruses (both their RNA and their proteins) are built by the infected cell without any material overlap between the parent and the offspring retrovirus. It is worth noting that there is replication in the retrovirus case if we take replication to be the copying of properties (see Godfrey-Smith 2000; Nanay 2002 on whether and in what sense there is replication in the evolution of retroviruses).

In short, it seems that material overlap is not necessary for information transmission that could lead to selection. Thus, the notion of reproduction has no explanatory advantage over the notion of replication.[20]

7 An additional advantage: cultural replication without memes

A further advantage of my account of replication needs to be noted. As we have seen, the distinction between replicator and interactor was originally introduced "as a generalization of the traditional genotype–phenotype distinction" (Brandon 1996, p.125). This means that there can be, and supposedly there are, entities other than the gene that would count as replicators. The main candidates for such replicators have been memes.

Memes are defined as the 'units of the cultural transmission' (Dawkins 1976/1989, p. 192. See also Dawkins 1982a,b). According to the meme theory, cultural phenomena can be explained, at least partially, with the help of the following evolutionary model: Memes are pieces of information and they compete for survival in a quite similar way as genes do; the difference is that they compete for the capacity of our minds. A meme can be a tune, the idea of liberalism, or the habit of brushing one's teeth. Those tunes will survive that can get into and stay in many minds. The ones that fail to do so will die out.

Meme theory is still extremely popular (see Blackmore 1999; Dennett 2003, 2006; Aunger 2002; Distin 2005). But it has been severely criticized for various reasons, partly for worries about the ontological status of memes (Sperber 1996; Wimsatt

[20] As Griesemer's concept of reproduction and my new way of interpreting the concept of replication are in some sense competing for the same theoretical space (of an alternative to the original replicator concept), it is worth clarifying the major differences between the two. Griesemer's aim is to strengthen the concept of replication—arguably he ends up strengthening it too much. My strategy is the opposite: I weaken the original replication concept by conceiving of it as the copying of properties rather than entities.

1999; Fracchia and Lewontin 1999; Richerson and Boyd 2005; Sterelny 2006a,b). What are these cultural replicators supposed to be?

There have been various attempts to answer this question (Dennett 2006, pp. 80–81, 349–350, 2003; Aunger 2002, pp. 311–322; Distin 2005). An influential strategy is to say that both genes and memes are really just pieces of information and there is nothing ontologically worrying about the concept of information (this is Dennett's and Distin's response; but see Aunger 2000's more restrictive version). But note that this view violates the concept of replicator the original replication–interaction model was presupposing.[21] I would like to argue that an obvious way of bypassing these ontological worries would be to endorse my definition of replication. This move would, of course, replace the notion of memes with replicated cultural properties.

It has been argued that whether or not we buy into meme theory, there are processes in the cultural domain that can be described as replication (Richerson and Boyd 2005; Sterelny 2006a,b).[22] If we accept my definition of replication, then we can explain these processes without postulating ontologically suspicious entities, like memes.[23]

It is important to note that if we acknowledge that there are processes that could count as cultural replication, we do not need to be thereby committed to allow for cultural selection (as replication is not sufficient for selection), let alone cumulative cultural selection that could explain why certain cultural features are the way they are. We can be agnostic about the explanatory power of cultural replication while still allowing for the phenomenon itself. Or, to put it differently, if we accept my definition of replication, this will not salvage meme theory or even the very idea of memes. But it would make it possible to talk about cultural replication, without specifying what the replicated entities would be, or positing the existence of memes.

8 Conclusion: Why should we care?

Suppose that my argument so far has been valid. The modified notion of replication is indeed necessary for selection and we can salvage the replication–interaction model by modifying the notion of replication in the way I proposed. What is the relevance of this claim? After all, we have another well-respected account of selection that does not make any reference to replication: the heritable variation of fitness account. If that

[21] They are not reproducers either: a meme and its copy do not have any material overlap.

[22] Of course there are ways of giving an evolutionary account of cultural change without talking about cultural replication (see Cavalli-Sforza and Feldman 1981; Lumsden and Wilson 1981). My point is that if we want to talk about *cultural replication*, we can do so without positing memes.

[23] Note that this application of the account I proposed her would have interesting consequences with regards to an important debate within meme theory. A crucial question about meme replication is whether it needs to be specific to one kind of physical substrate. Robert Aunger argues that it does (Aunger 2002, pp. 154, 157). Kate Distin, in contrast, argues that memes can replicate in different substrates (Distin 2005, Chap. 11, see also Dennett 2003, 2006). If we accept my definition of replication, then replication does not have to be specific to one kind of physical substrate: it is properties that get copied, regardless of the substrate. And the same is true of cultural replication. Hence, my account is siding with Distin and Dennett (and not with Aunger) in this question. It is also important to note that the question of substrate-specificity is an important one as long as we allow for cultural replication, regardless of whether we talk about memes.

account works, why should we take the replication–interaction model seriously, even if it is tenable?

One reason why we should take the replication–interaction model seriously is that the heritable variation of fitness account, as the notion of fitness itself, is not as unproblematic as it looks. One of the most important debates of the last years in philosophy of biology has revolved around how exactly we are supposed to interpret this model of selection (Matthen and Ariew 2002; Walsh et al. 2002; Millstein 2006; Brandon 2006; Bouchard and Rosenberg 2004; Rosenberg and Bouchard 2005; Ariew and Lewontin 2004; Stephens 2004): is it a population-level explanation or is it an individual-level one? Is it a causal or a statistical model?

If we accept the replication–interaction model, these puzzling questions may become easier to address. According to the replication–interaction model, selection consists of repeated cycles of two different processes. Thus, instead of asking, for example, whether selection is a causal or a statistical process, we should really ask (a) whether the first process in this cycle (replication) is a causal process and (b) whether the second one (interaction) is a causal process. As in the case of the levels of selection problem I mentioned in Sect. 1, the answer we get may be very different in the case of replication and in the case of interaction.

The same point applies to the other highly contested question about selection conceived of as heritable variation of fitness: whether it is an individual-level or a population-level phenomenon. If selection is replication plus interaction, then this is the wrong question to ask. Instead, we should ask two questions: (a) whether replication is an individual-level or a population-level phenomenon and (b) whether interaction is an individual-level or a population-level phenomenon. These two questions may have very different answers. What I want to suggest is not that these questions about the nature of selection will go away as soon as we switch to the replication–interaction model, but that they may become easier to address if we pose them within this framework.

But there are stronger explanatory advantages of the replication–interaction model if we consider its biological applications. More precisely, there are instances of natural selection where it is not clear how the heritable variation of fitness model would work. An important aspect of the heritable variation of fitness model is that it talks about parents and offspring. Both in what Lewontin calls 'the principle of variation' and what he calls 'the principle of differential fitness' (Lewontin 1980, p. 76) are principles about the parent–offspring relation. But there are cases of natural selection where it is unclear what should be considered to be the parent and what should be considered the offspring. I will mention two such cases.

The first case is the evolution of clonal organisms. Clonal organisms like quaking aspen, violet or strawberry can produce what look like new plants asexually (of course, most clonal organisms *also* reproduce sexually). The new apparent plants are (or at least can be) physiologically separate but they are really just the growths of the old plant. To use the terminology of Harper (1977), we have one *genet*, but many different *ramets*. We face a thorny ontological question: how should we individuate clonal organisms? Is it a new organism that is being produced or is it just a new part of the existing organism? Should we identify clonal organisms with genets or with ramets? (see Jackson et al. 1985; Bouchard 2008 for summaries).

The consensus among biologists and philosophers of biology is that "genets are the fundamental units of populations upon which natural selection acts" (Jackson and Coates 1986, p. 8, see also Godfrey-Smith 2009, p. 72 for further representative quotes; Hull 1980, p. 328 for some worries; Jackson et al. 1985 for a summary). The reasoning is simple: gamets are produced in the same way as new branches of an oak tree. So we should not think of them differently. As Ariew and Lewontin say, "if a tree is an individual then so is the collection of all the ramets of a violet" (Ariew and Lewontin 2004, p. 360).

But then the heritable variation of fitness account has a problem. If ramet production is growth, then how could we even formulate what would count as natural selection in the case of the evolution of clonal organisms? As Godfrey-Smith says, "if a strawberry produces ramets that vary, that differ in their further ramet production, and that pass along their quirks to new ramets, then we do have the ingredients for Darwinian change" (Godfrey-Smith 2009, p. 85, see also Bouchard 2008, p. 562). But the ramet is not an offspring of the original strawberry plant: it is part of the very same individual. But if we cannot talk about parents and offspring in the case of the evolution of clonal organisms, then the 'the principle of variation' and 'the principle of differential fitness' are difficult to even formulate.[24]

The new version of the replication–interaction model, in contrast, can describe these cases without encountering any problem. Ramet production does count as replication according to my definition, regardless of whether we consider the new ramet to be a new individual or a new part of the same individual. Hence, we can describe selection in the case of clonal organisms as the repeated cycles of this replication process (interpreted as the copying of properties) and some interaction process.

The second example for a selection process that can be handled by the modified version of the replication–interaction model better than by the heritable variation of fitness model comes from microbial populations. A striking feature of microbial population is lateral gene transfer: the transfer of genetic material from one organism to another by conjugation, transduction or transformation (Bushman 2002; Thomas and Nielsen 2005, see O'Malley and Dupré 2007, esp. pp. 167–168 for a philosophical analysis of this phenomenon). Lateral gene transfer makes natural selection (and evolutionary change in general) in the microbial world more rapid and more frequent than it is among macrobes (see, e.g., Lewontin 2002). But how can we talk about heritable variation of fitness in the case of lateral gene transfer? Lateral gene transfer is not from parent to offspring. It is from offspring to offspring. This, again, makes it difficult to even formulate 'the principle of variation' and 'the principle of differential fitness' of the heritable variation of fitness model (see O'Malley and Dupré 2007 for a summary of how lateral gene transfer in the microbial world challenges our existing evolutionary models).

[24] My claim is not that this constitutes a knock-down objection to the heritable variation of fitness account, but that it is not at all clear how these cases could be dealt with within that framework and whether they require some important modifications of the account or of the concept of fitness. Ariew and Lewontin (2004) addresses these issues in Ariew and Lewontin (2004, pp. 360–361). See also Bouchard 2008's way of handling this problem. It is also important to note that as most clonal organisms also reproduce sexually, this makes even more difficult to calculate the fitness of a clonal organism.

But if we accept the new version of the replication–interaction model I proposed here, then we can easily explain natural selection by lateral gene transfer as lateral gene transfer will count as a replication process. Hence, we can describe selection in the microbial world as the repeated cycles of this replication process (interpreted as the copying of properties) and some interaction process. So we have found another example where the new replication–interaction model works better than the heritable variation of fitness model.[25] It is also important to note that these examples are not marginal cases of natural selection (on how widespread and important clonal reproduction is, see Godfrey-Smith 2009, pp. 71–72; Bouchard 2008, on the importance and relevance of the microbial world see O'Malley and Dupré 2007's manifesto).

My aim in this paper was to give a plausible version of the replication–interaction model of selection. The replication–interaction model was introduced as an improvement on the heritable variation of fitness model. It has been rejected, as I attempted to point out, for unjustified reasons. Hence, now we can begin to compare the explanatory power of the two models. The aim of the last section was to show that the replication–interaction model may have some very important explanatory advantages.

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[25] It is important to point out that while the example of microbial evolution only demonstrates that any version of the replication–interaction model can handle these cases better than the heritable variation of fitness model, the example of clonal organisms also shows that the new replication–interaction model, which takes replication to be the copying of properties, is superior to both the heritable variation of fitness model and to the old replication–interaction model, which takes replication to be the copying of entities.

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