



Rethinking hereditary relations: the reconstitutor as the evolutionary unit of heredity

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Abstract

This paper introduces the reconstitutor as a comprehensive unit of heredity within the context of evolutionary research. A reconstitutor is the structure resulting from a set of relationships between different elements or processes that are actively involved in the recreation of a specific phenotypic variant in each generation regardless of the biomolecular basis of the elements or whether they stand in a continuous line of ancestry. Firstly, we justify the necessity of introducing the reconstitutor by showing the limitations of other evolutionary conceptions of the unit of heredity, such as the replicator, the reproducer, and the Darwinian individual. We argue that these conceptions are based on the requirement of lineage formation (Stability of Lineages), which we argue to be unnecessary for the existence of evolutionary heredity. In the second part, we introduce the reconstitutor, which we base on the concept of Stability of Traits, and illustrate how it covers cases of hereditary phenomena (small RNAs, microbiota) not covered by the previous accounts. Secondly, we illustrate how the reconstitutor could serve as a platform to rethink ecological inheritance and other forms of inheritance that have been recently introduced under the song/singer model of evolution.

Keywords Biological individuality · Microbiome · Holobiont · Regulatory RNAs · Epigenetics · Niche construction · Interactor · Units of selection · Misplaced concreteness · Song/singer model

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1 Introduction

Understanding the phenomenon of biological inheritance, identifying the entities and processes responsible for hereditary relations, and isolating the unit of heredity have constituted a central task in the biological discipline for almost two centuries (Rheinberger & Müller-Wille, 2009).¹ A basic characterization of heredity, when the concept is understood in evolutionary terms, relates to the realization of the process of descent with modification. If evolution is the process in which the parents produce some offspring that are alike them in certain features (*descent*), but also differ from them in others (*with modification*), heredity must be conceived as the part realizing the production of alike-forms (descent). The evolutionary unit of heredity is then the unit that can be identified as the realizer of the process of descent in each generation. In this paper, we assume that there is a conceptual distinction between *heredity* and *inheritance*. We take *inheritance* to refer to the process(es) that ensures parent–offspring similarity; we take *heredity* to denote the resemblance relations between parent and offspring (i.e., the result of inheritance) (see Danchin & Pocheville, 2014; Lamm, 2018). Based on this we take the unit of heredity to refer to the unit where these resemblance relations get transgenerationally realized.

The contemporary understanding of the unit of heredity derives from its association with the concept of lineage formation and the empirical detection of lines of ancestry. This association crystallized during the second half of the twentieth century due to the combination of genetics and molecular biology (Fox Keller, 2000; Rheinberger & Müller-Wille, 2009) and triggered progress in biological research for several decades. Biological lineages are continuous lines of descent where identifiable relationships of ancestry connect the different nodes. Importantly, biological lineages are not necessarily molecular (DNA) lineages. There may be lineages of DNA, RNA, organelles, cells, functions, traits, etc. provided there are relationships of ancestry between these elements. These relationships allow the systematic establishment of a causal connection between parents and offspring across different generations (Neto, 2018), providing a solid material basis to establish relationships of transgenerational continuity by detecting what parents transmit to their offspring. In what follows, we will refer to lineages and lineage formation as “line of ancestry” or “continuous line of descent”. These expressions should be distinguished from “descent”. One may object that the concept of *descent* logically entails *ancestry* and *continuity*, but this is precisely what we challenge in this work. In our view, descent occurs when there is recreation of phenotypic variants, regardless of whether this happens via ancestry or whether there is any continuity, which we take to be two properties accidentally—although epistemically efficiently—connected to the notion of descent.

Recent biological research, however, has challenged the association between evolutionary heredity and lineage formation. Drawing on it, in this paper, we propose a new concept of the unit of heredity, the reconstitutor, that keeps the association between heredity and descent while simultaneously breaking up with the tradition that links heredity to lineage formation. We argue that the association between heredity and

¹ This paper is inspired by our previous Veigl et al. (2019), where we presented a very early and tentative version of the ideas that we elaborate here.

lineage formation is grounded on what philosophers call a fallacy of misplaced concreteness (Nicholson & Dupré, 2018; Whitehead, 1929). The fallacy results from the univocal identification of a complex process with a concrete type of structure, entity, or set of entities that is supposed to exclusively account for every possible instantiation of the process. The fallacy usually becomes salient when a process with the same characteristics seems to be occurring, yet the type of structures or entities that account for its realization are not the ones that were initially assumed to be necessarily involved in the process.

In the case of heredity, the fallacy arises after discovering processes of descent transcending the requirement of a single and continuous line of ancestry. In fact, advances in microbiology, embryology, developmental biology, and other fields have shown that some biological processes sustain heredity without being tied to the type of structures or entities usually matching the most accepted conceptions of the unit of heredity.

This situation generates a dilemma that is hard to solve. On the one hand, if one breaks up the association between heredity and lineage formation, one may risk making the concept of heredity unoperationalizable. As a matter of fact, lineage formation is an appropriate concept for elaborating the empirical tools allowing the detection of causal links whose history can be investigated. On the other hand, if one maintains the association, then it may be that several phenomena that entail a manifestation of descent would be excluded from hereditary research, mistakenly narrowing the field and even preventing scientific progress. It seems that both options are opposing sides of a dilemma that cannot easily be solved (e.g., Merlin, 2017; Pontarotti, 2015 for similar lines of argument and potential solutions).

In this paper, we offer an escape from the dilemma based on the introduction of two concepts that allow for the association of heredity with a broader array of biological structures while being at the same time restrictive enough to maintain a fruitful operationalization of the hereditary process. Our proposal is closely aligned to other dynamic conceptions of heredity already found in the literature, i.e., conceptions of heredity as a matter of systems dynamics (Oyama, 1985/2000; Jablonka, 2002), with the main difference that we offer our concept as a particularly useful tool in the context of the dispute about evolutionary conceptions of heredity. In addition to emphasizing the epistemic benefits of adapting our proposal, we consider our approach to constitute an ontological project for thinking about heredity. In that vein, we believe it could be regarded as a case study for those elaborating a process ontology of the life sciences (Dupré, 2012; Nicholson & Dupré, 2018). Overall, our paper shows the illuminating role of philosophical tools in analyzing contemporary scientific debates (Guay & Pradeu, 2015; Kaiser, 2019; Laplane et al., 2019; Suárez & Stencel, 2020; Veigl, 2017).

In Sect. 2, we distinguish between the organizational, developmental, and evolutionary conceptions of heredity and introduce the concept of *Stability of Traits* (SOT) as the appropriate way to characterize evolutionary hereditary phenomena, as opposed to developmental or organizational hereditary phenomena. We also show how the concepts of the evolutionary unit of heredity and the unit of selection differ under our approach. In Sect. 3, we show how other recent conceptions of the evolutionary unit of heredity (replicator, reproducer, Darwinian individual) are ultimately based on the concept of lineage formation, making them conceptually inadequate to capture SOT.

Drawing on this analysis, in Sect. 4, we introduce the concept of the *reconstitutor* as the unit of heredity. In Sect. 5, we exemplify the usefulness of the reconstitutor based on two case studies where the complexity of the hereditary process impedes the identification of lineages (Sects. 5.1, 5.2). In Sects. 6 and 7, we show that the application of the concept cannot be extended indefinitely, establishing limits that, we contend, make its operationalization feasible. These limits derive from the two requirements concerning the existence of “phenotypic variants” and “active maintenance”, included in the definition of the reconstitutor we offer. This allows us to contrast the reconstitutor and the views of the unit of heredity deriving from niche construction (Sect. 6), and the *It’s the song, not the singer* model of evolution (Sect. 7). Finally, we present our conclusions (Sect. 8).

2 Stability of traits and the different conceptions of heredity

To introduce the reconstitutor as the unit of evolutionary heredity, let us first examine its relation to other conceptions of heredity discussed so far in the literature, and the type of unit of heredity the latter are associated with. One conception is organizational. The organizational conception characterizes heredity non-evolutionarily as the set of elements that act as difference-makers for the transgenerational reconstruction of biological constraints (Moreno & Mossio, 2015; Mossio & Pontarotti, 2019; Pontarotti, 2015). While this approach may have evolutionary implications which require serious considerations (e.g., in case these constraints are a result of evolution or once they have appeared end up biasing the evolutionary history of a lineage or a clade), it is not in principle conceived to account for the role that the concept of heredity plays in evolution, insofar as it is not directly concerned with the idea of descent that we consider to be the pivotal element structuring any evolutionary account of heredity. Hence we contend that the type of unit of heredity isolated by those following an organizational account is neither co-extensional nor co-intensional with the type of unit of heredity we are concerned within this work.

A second conception derives from developmental biology and crystallized in the tradition known as developmental systems theory (DST) (Oyama, 1985/2000). Those working on DST have emphasized that the unit of heredity is the whole life cycle of an organism and not just specific sets of relations or parts within it. Under their interpretation, the concept of heredity expands to break the traditional dichotomy between organism and environment. This is because what had traditionally been conceived as environmental features are sometimes causally responsible for the higher reproductive success of a specific life cycle, and thus DST theorists deduce that they must be parts of the hereditary system as a whole. Griffiths & Gray, drawing on Oyama’s pioneering work, embrace such position succinctly:

[Under the DST interpretation of heredity, o]ne variant does better than another, not because of a correspondence between it and some preexisting environmental feature, *but because the life cycle that includes interaction with that feature has a greater capacity to replicate itself than the life cycle that lacks that interaction* (Griffiths & Gray, 1994, p. 300, emphasis added).

This leads to a view in which the whole matrix of developmental resources, including parental, collectively generated and persistent resources, is a component of their concept of the developmental unit of heredity. To express it in Oyama's terms, heredity concerns traits transmission (Oyama, 1988) via the "passing on of all developmental conditions [resources], *in whatever manner*" (Oyama, 1985/2000, p. 43, emphasis added).

The conception of heredity we elaborate on shares some elements and takes many lessons from these developmental conceptions. First, we agree with their emphasis on conceiving heredity as a dynamic process where restriction to a specific type of molecule, or its properties, is ungrounded. Second, we agree that a conception of heredity should not begin by assuming what is environmental as opposed to hereditary, but rather determine what is environmental by exploring the transgenerational relations between certain biological elements. Third, we agree that the process of heredity is connected to the existence of traits and how these reappear.

However, there is an important contrast between the developmental conception of heredity championed by DST and the conception we aim to put forward. Insofar as our conception concerns the process of descent in the context of the ontology of descent with modification, our conception of heredity must primarily encompass and make room for those cases where there is a capacity for modification or, to put it differently, the possibility of generating phenotypic variants. While someone working on DST deals with modification, she would do so only accidentally, as from the developmental perspective, the key question is how a life cycle re-appears (i.e., descent), regardless of the capacity for modification in the life cycle, or some parts of it. The relevance of this distinction will become salient in Sect. 6, but we think we have enough reasons to consider that this approach, like the organizational approach, is capturing a different concept of heredity.

Evolutionary biology provides a third conception for thinking about heredity, which more closely aligns with our interests in this work. While the conception initially appears with Darwin and Mendel, its current formulation can be traced back to Richard Dawkins and David Hull. They introduced and elaborated the concept of the *replicator* that would later develop into the concepts of the *reproducer* and the *Darwinian individual*—for concrete biological examples of each of the three categories see Sect. 3. The primary purpose of the authors elaborating these conceptions of heredity was to isolate the units that are relevant from an evolutionary perspective, insofar as they are useful to track how a population would change its composition (replicator) and, in some circumstances, the distribution of its phenotypic properties (reproducer, Darwinian individual), due to the action of natural selection and, potentially, other evolutionary factors.

Given that the conceptions of the unit of heredity that we examine were initially formulated in the context of the debates about units of selection, we first want to clarify the relationship between both concepts. Two types of projects have mainly addressed the question about the unit of selection: one, which could be referred to as the *Disambiguating Project*, seeks to distinguish different meanings of "unit of selection" under the assumption that it is a polysemic expression referring to different functional

properties. Some versions of this project distinguish two meanings, one referring to the unit that causes replication/reproduction to be differential (interactor), and another referring to the unit that forms lineages via replication/reproduction (replicator, reproducer) (Dawkins, 1976/2006; Hull, 1980), while others add more meanings in addition to these two (Lloyd, 2017). On the other hand, others believe that the expression is unambiguous (Godfrey-Smith, 2009), referring to the entity that simultaneously forms a single lineage and causes this lineage to modify its properties transgenerationally and adaptively via its differential replication/reproduction. Skillings, discussing whether holobionts are units of selection, expressed this latter idea succinctly:

If the set of lineages that make up the holobiont varies within and between host generations, then the holobiont cannot be a coherent unit of selection (Skillings, 2016, p. 8).

In both cases, the unit of heredity is always involved in the question about units of selection, either as a specific functional concept meant by the expression or as a necessary part of the unique concept of “unit of selection.”

While we agree that the question about the unit of heredity and the question about the units of selection are conceptually related, and we present our account by contrasting it to these other accounts of the unit of heredity deriving from the units of selection controversy, we think that they should not be conflated. Those who have thought of the units of heredity in the context of the debates about units of selection tend to associate the former concept to a specific copy-mechanism (replicator question) or to the realization of an adaptive process via lineage formation at the focal level (Darwinian individual). We think this is demanding too much from the concept of “unit of heredity,” as we will show in detail in the next section. In our account, the concept of the unit of heredity only refers to the unit that realizes the process of descent, regardless of the copy-mechanism involved, or whether this unit simultaneously causes the process to be realized differentially and/or adaptively at the same level of the unit of heredity. This will be crucial to understand what our conception of the unit of heredity contributes to current biological debates.

Thus, we assume that the expression “unit of heredity,” as we use it here, refers to the unit situated in the evolutionary tradition of thinking about processes of inheritance, but we contend that contemporary evolutionary approaches to heredity are severely limited because they fail to explain some cases of what we call Stability of Traits (SOT).

SOT: phenomenon manifested by the transgenerational persistence of variation in the phenotypic characteristics of some individuals in a population at a specific level of the biological hierarchy, not necessarily forming a continuous line of ancestry.

Under this approach, SOT is a functional concept to capture not only the phenotypic variants transmitted due to the existence of strict parent–offspring relationships, or

lineages, but also the cases of regular re-appearance of phenotypic variants despite the lack of these relationships. Epistemologically, SOT requires looking at the final phenotypic effects and their temporal continuity and inferring how phenotypic re-appearance must have occurred retrospectively instead of looking at what is transmitted before it may have any phenotypic consequences or once those have already evolved. Ontologically, SOT occurs because there is variation with respect to phenotypic types.

It is necessary at this point to distinguish between three concepts: *new traits*, *conservation of the same trait*, and *variation of the same trait*. Note that our characterization of SOT refers only to the latter. It refers both to the preservation of the phenotypic characteristics, or trait preservation, and the maintenance of their variants in the population. The requirement of trait preservation is due to the necessity of a criterion that allows distinguishing between the new features that appear in the transgenerational process of formation of new individuals and the appearance of the same features. We consider that this is the basis of the phenomenon that should underlie the ontology of heredity, and thus the reason we call it the *stability of traits*. But additionally, we demand that SOT manifests in the *variation of the same trait* if SOT is aimed to capture the ontology of evolutionary heredity. So one may object that it would be best to speak of *stability of variation*, or *stability of trait variation*, instead of stability of traits, simpliciter. We think this may induce confusion between the concepts of *new traits* and *variation of the same trait*, which we aim to avoid explicitly.

While some variations of SOT had already been introduced and defended in previous works (Doolittle & Booth, 2017; Lenton et al., 2018; Taxis et al., 2015; Lemanceau et al., 2018), it was then formulated to account for the accumulation of design at the functional level of genetic networks through multilineage convergence, even though it was clear that the concept could be applied to other types of hereditary systems (Bapteste & Papale, 2021; Suárez & Triviño, 2019; Suárez, 2020). How we understand SOT in this paper partially aligns with this latter application. Additionally, SOT aligns with other accounts that also conceive processes similar to it as a conceptual basis, and an empirical guide, for capturing the level in the biological hierarchy where the realization of hereditary processes is taking place (Charbonneau, 2014; Papale, 2021).

SOT is multiply realizable. Its realization in specific biological levels depends both on the phenotypic characteristics being investigated as well as the range of possible experimental or natural interventions that would significantly change the specific trait.

One way of maintaining a particular trait is through the Stability of the Lineage (SOL) carrying the trait. SOL primarily occurs through the transmission of an element or set of elements that are ultimately responsible for preserving the phenotypic characteristics. Such elements may be DNA molecules, RNAs, ribosomes, antibodies, specific microorganisms, or even traits or functions. Thus, a lineage of traits under the SOL conception does not necessarily rest upon the existence of molecular lineage, but simply upon the existence of clear relationships of ancestry between the elements of the lineage. DNA-based inheritance is a canonical example of this form of inheritance, realizing SOT because a specific element (i.e., DNA) causally related to the phenotypic characteristics is replicated in a semiconservative manner and transmitted from the parents to the offspring. In the offspring, the transmitted element is supposed to be causally related to the phenotype in a privileged way. A trait whose variants depend on

DNA transmission is eye color. Variants of eye color are maintained in a population due to DNA transmission.

But SOT, as we have characterized it, can also be transgenerationally realized beyond the transmission of elements. This would occur, for example, in cases where independent elements converge into a network and develop together to reconstruct the trait variant. This network of independent elements may consist of independent lineages that merge, but it may also consist of a set of relations between cellular, extracellular and environmental components with the capacity to reconstruct the property afresh, even without any form of direct transmission. Some traits involved in the acquisition of hematophagy in vampire bats result from the convergence of independent bacterial lineages, most of them coming from the environment. The traits are transgenerationally recreated and manifest as SOT via network recombination, and different trait variants are maintained in a vampire bat population via this specific recombination. On the other hand, small-RNA-based resistances to environmental stimuli in *Caenorhabditis elegans* persist transgenerationally through self-reinforcing interactions with heterogeneous sets of resources, leading to a manifestation of SOT, without lineage formation or lineage convergence between functionally equivalent small RNA states across generations.

Even though our characterization of SOT is grounded on the observation that SOL is not a good way of characterizing every phenomenon of SOT, the two concepts do not stand in opposition to each other. Technically, SOL is a way of realizing SOT, but *one out of a broader range of possibilities*. Yet, it is a specific way of maintaining hereditary relations that has inspired the conceptual association between *heredity* and *transmission*. Let us stress at this point that we prefer not to use the term “transmission,” as we aim to avoid what we take as a common conflation between transmission and heredity. We take transmission to refer to the process in which one element (or a discrete set of elements) is passed from one place to another while keeping structural and functional integrity.² The entity at the place of departure and the place of arrival is *the same*, not similar. Generally, in biological transmission, at least when the latter is conceived *stricto sensu*, the place of departure and the place of arrival of this element are points in a biological lineage (cf. Merlin & Riboli-Sasco, 2021 for a different conception of “transmission,” and our discussion in Sect. 3). The critical point of transmission is thus that the structural integrity and function of the transmitted element are maintained. But our point is that some recent examples of hereditary relations work even when the elements in a set of persisting elements do not maintain their structural and functional integrity to recreate the trait variant, nor are they passed on together. Rather, they maintain the tendency to interact together, and in doing so altering their structure and function, to recreate the trait variant—see Sect. 5 for examples. Therefore, SOT cannot be conceived simply as a form of transmission, even though it is sometimes realized via transmission.

With the concept of SOT at hand, in the next section, we will examine the family of projects that have elaborated specific conceptions of the unit of heredity. We will argue, however, that none of these is geared towards fully explaining all instances of

² By “functional” we mean that the element plays the same causal role, and not necessarily that it has a selected effect. Otherwise, *transmission* would be conflated with the existence of *selected mechanisms of transmission*.

SOT, given the tight connection they build between the concept of the unit of heredity and the concept of SOL.

3 Evolutionary conceptions of the unit of heredity: replicators, reproducers, and darwinian individuals

Introducing the differentiation between replicators and vehicles, Dawkins aimed to refocus debates regarding the units of selection and clarify what he perceived as an essential confusion (Dawkins, 1976/2006, 1982). Whereas previous accounts, such as Lewontin's (1970), required heredity and phenotypic variance to occur at the same focal level, Dawkins strictly limited the level of biological organization subjected to heredity to the level of the particles that replicate, or replicators, and envisioned a new concept, the vehicle, for the level at which phenotypic variance, and its systematic connection to fitness, occurs. In Dawkins' account, many levels of biological organization can be vehicles: cells, organisms, populations, and even genes. The replicator's role in biology is preserved for a particular entity, the DNA/RNA-gene transmitted in replication events, which is the sole source of heredity, and the ultimate source of evolution.³

Replication plays such a pivotal role in Dawkins' account because it is the mechanism that ensures biological stability from one generation to the next, and thus the replicator is the ultimate biological structure that should be studied when investigating the effects of evolution and natural selection (Dawkins, 1976/2006; Williams, 1966). Dawkins' attitude derives from his view that replicators are necessary causes of vehicles. Vehicles house replicators, represent the phenotypic effects of the replicators they contain, and are the proximate targets of selection; yet, in Dawkins' account, these ideas are also combined with his conviction that vehicles are merely machines programmed to preserve and propagate replicators (Dawkins, 1976/2006, 1982; cf. Nicholson, 2013, 2019). Hence, under such an account, replicators were considered the only channel of hereditary relationships, at least insofar as these were conceived evolutionarily.

The problem of such an assumption is that the whole debate about heredity was built upon the implicit assumption that it necessarily involves SOL in the form of transmitted elements, which, as we showed, is the most restrictive understanding of SOT. In fact, the replicator concept that was assumed in these debates was ultimately an abstraction of 1970s knowledge concerning DNA/RNA replication (Godfrey-Smith, 2009). In that vein, it implicitly contained all the presuppositions about heredity that DNA/RNA replication carries, including their semiconservative vertical transmission across generations which ultimately allows the creation of a continuous line of ancestry of replicators. It is precisely this conceptual association between the process of heredity and the idea of lineage formation that constitutes the fallacy of misplaced concreteness upon which the whole debate about the unit of heredity has been built, in this case, manifested via the isolation of the properties of a specific molecule.

³ Dawkins also introduces the concept of *meme* to account for the type of replicators that may work in other contexts, such as cultural evolution. Memes are however not as relevant for the type of phenomena we are considering, so we will ignore them here.

An important line of criticism to the concept of the replicator as the sole unit of heredity derives from the work on the Major Transitions in Evolution (MTE), starting with Maynard-Smith & Szathmary's (1995) and spanning since. Research on the MTE is grounded on the observation that different modes of reproduction have evolved and must have done so from molecules that lacked the specific properties attributed to the replicator (Maynard-Smith & Szathmary 1995; Okasha, 2006; Stencel & Suarez, 2021). The MTE framework puts some tension on the necessity of replicators as carriers of heredity and suggests that a new abstract concept to characterize the unit of heredity is needed (Bourrat, 2021).

Covering the gap opened by the MTE tradition, Griesemer introduced the concept of the *reproducer* as a more encompassing category than the replicator. According to him, replication is a particular form of reproduction, and reproduction is the real source of heredity. This idea derives from the observation that each MTE involves the evolution of a new mode of reproduction that includes more units and, sometimes, different copying properties than the mode of reproduction it evolved from (Maynard-Smith & Szathmary, 1995).

A key element of the reproducer account is that reproduction is a capacity that some organisms acquire via development. That is, Griesemer aims to integrate heredity and development into a single conceptual scheme. A reproducer develops and requires material overlap between parent and progeny. The overlap between parents and progeny is called *progeneration*, an increase in numerically distinct objects of a particular kind (Griesemer, 2000). The requirement for material continuity opposes merely the formal transmission of information by a specific privileged molecule, as was the case with the replicator (Griesemer, 2014). Reproduction comprises development and progeneration, and emphasizes the transfer of developmentally acquired material (be it DNA or any other). Under this framework, thus, DNA/RNA-genes should not be awarded a privileged explanatory or causal role as units of heredity, insofar as they do not develop the reproductive capacity in isolation, but merely as parts of a bigger, genuinely reproducing unit (see also Griffiths & Stotz, 2013; Waters, 2006).

It follows from this that, in Griesemer's framework, the capacity to inherit something is thus a system property that is developmentally acquired. In other words, a reproducer does not merely transfer the capacity to grow or survive, but *the capacity to develop the capacity to reproduce again* (Griesemer, 2000). Only those systems that have developmental capacities can be reproducers. Therefore, not every reproduction process is necessarily an inheritance process. Inheritance processes are those reproduction processes in which there are evolved mechanisms for producing hereditary relations in development again and again. In this picture, replication processes are inheritance processes that evolved coding mechanisms (Fig. 1).

While Griesemer's reproducer concept substantially advanced the debate about inheritance and helped avoid the identification of heredity with the coding properties of a transmitted molecule (hence avoiding that specific form of misplaced concreteness), it still constitutes an inadequate account of the unit of heredity. Note that Griesemer's definition of inheritance characterized it as a subset among reproduction processes; concretely, those reproduction processes in which there are evolved mechanisms for producing hereditary relations in development again and again. The problem with such an account lies in demanding the existence of evolved mechanisms before the

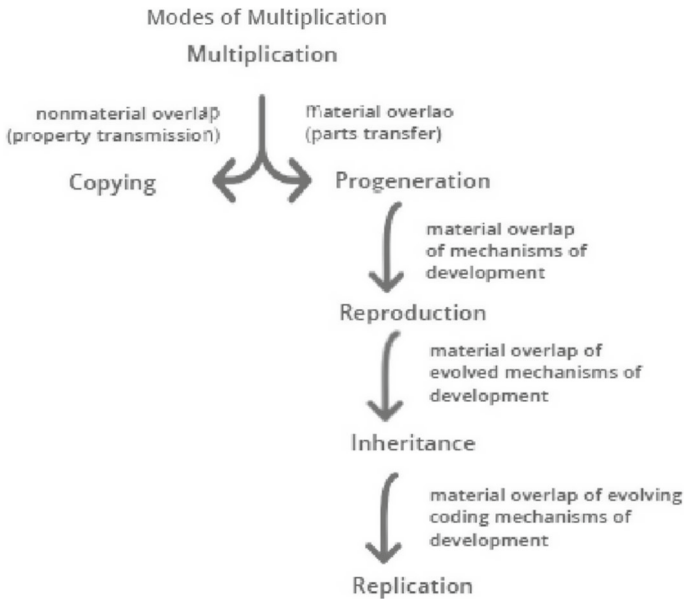


Fig. 1 Griesemer's conception of the reproducer. Adapted from Wimsatt and Griesemer (2007, p. 268, Fig. 7.9)

concept of heredity can be applied to a specific phenomenon of SOT. Bourrat (2014) proves why this demand mischaracterizes heredity by constructing six-individual models showing that the evolution of specific mechanisms of heredity is the result of the evolution of previously extant units which still lacked evolved hereditary properties (such as coping mechanisms or mechanisms for material overlap). We would reformulate Bourrat's criticism in our terms by arguing that the key paradox in Griesemer's account lies in his demand for specific mechanisms of transgenerational recurrence, i.e., mechanisms that guarantee the transgenerational continuation through lineage formation. In other terms, while the reproducer notably expands SOL beyond transmitted elements towards different transmitted mechanisms, it still fails to fully account for SOT insofar as it requires the existence of evolved, yet not necessarily coding, mechanisms of transmission. This, unfortunately, conflates a process with a product of the process and, in consequence, commits the author to a subtle version of the fallacy of misplaced concreteness. An adequate account of the unit of heredity must escape from the requirement of evolved properties, even though these are as diverse and heterogeneous as those included in Griesemer's account.

After Griesemer, Godfrey-Smith moved the debate about heredity forward by introducing his concept of a Darwinian individual. While Darwinian individuals are also entities that reproduce, and thus they are just reproducers with a different name, there are three differences between both accounts. First, Godfrey-Smith distinguishes different types of Darwinian individuals depending on the characteristics of the lineages and their mode of reproduction. Second, he conceives the Darwinian individual as *the unit of selection* and acknowledges that Darwinian individuality comes by degrees. Hence

it is a concept that could apparently solve two debates in a unified manner. Third, he does not think that material overlap is a necessary requirement for the existence of hereditary relationships. Let us explain these three characteristics in detail.

First, Godfrey-Smith introduces a three-fold anatomy of Darwinian individuals, depending on the mechanisms allowing the realization of reproductive relations. Darwinian individuals can be simple, collective, or scaffolded reproducers. A bacterial cell, whose parts cannot reproduce and which only requires some external resources to reproduce itself, constitutes an example of a single reproducer. A multicellular organism, which is made of cells that can reproduce but which reproduces itself by passing some privileged materials to its offspring through a bottleneck, constitutes a paradigmatic example of a collective reproducer. Finally, a retrovirus, which only reproduces by hijacking the reproductive machinery of simple reproducers, would be a canonical case of a scaffolded reproducer.

Second, in contrast with Griesemer, who introduces the reproducer as a substitute for the replicator without rejecting the validity of the other functional roles previously isolated in the debates about the units of selection (the interactor, most noticeably), Godfrey-Smith conceives the Darwinian individual as *the unit of selection*, rejecting the rationale and adequacy of the Disambiguating Project. In this sense, a Darwinian individual needs to express interpopulational variance in fitness (interactor) while simultaneously being able to transmit that variance in fitness at its own level through one of the evolved reproductive mechanisms isolated in his anatomy of Darwinian individuals (reproducer). Importantly, while the concept of the Darwinian individual encompasses the roles previously isolated in the debates about units, it gets realized as a graded property, i.e., elements and systems of elements can be more or less Darwinian individuals.

Third, by introducing his idea of formal reproduction, Godfrey-Smith made the concept of Darwinian individual independent from the material relationship between parent and offspring. Material overlap is an essential part of the process of reproduction for Griesemer, while Godfrey-Smith considers it does not need to be present in every example of reproduction.⁴ The concept of formal reproduction only involves an unspecified relation of causality between the form of the parent and the form of the offspring. In other words, formal reproduction occurs when parents are causally responsible for producing offspring without any privileged material basis causing the parent–offspring similarity relationship. Examples of formal reproduction constitute cases of minimal reproduction, e.g., scaffolded reproduction in which the virus only provides the coding sequence or protein transformation caused by prions. In the latter example, a protein is causally responsible for the change in the shape and properties of another but without materially contributing to its composition. Given that these cases can be considered examples of formal reproduction for Godfrey-Smith due to

⁴ It must be noted, although in passing, that Griesemer believes that Godfrey-Smith misinterpreted his conception, as his concept of the reproducer only demands “that at least some material parts of the offspring were formerly material parts of the parents” (Griesemer, 2014). Material overlap might be evolutionary necessary, because, contrary to an unstable environment, it robustly ensures the capacity to develop the capacity to reproduce. For the case of retroviruses specifically, Griesemer claims that there is material overlap of molecular generations.

the causal relationship between the two proteins, his account can accommodate cases of heredity without material overlap.

Godfrey-Smith's concept of the Darwinian individual constitutes a good framework to tackle the problem of the unit of heredity, and one that apparently could escape the fallacy of misplaced concreteness that we contend has characterized the study of the evolutionary unit of heredity. No entity or set of entities are privileged in his account and, given the inclusion of formal reproduction, no structure is privileged either. But we contend that this is not entirely accurate because, in his schema, a parent–offspring similarity counts as hereditary only if there is an evolved mechanism ensuring the exclusivity of parent–offspring causal connections and simultaneously ensuring the feasibility of their disconnection (in the sense of generation of a new individual). To quote:

The link between 'individuality' and reproduction is in some ways inevitable. Reproduction involves the creation of a new entity, and this will be a countable individual. But the right sense of 'individual' to use here is a relaxed one. Two Darwinian individuals might be genetic duplicates (physical duplicates, in fact). One individual might be genetically heterogeneous. *That is fine as long as we know who came from whom*, and roughly where one begins and another ends. (2009, p. 84, emphasis added).

And later, when studying whether some cases of symbiosis (the squid-*Vibrio* symbiosis) may be cases where there is one or several units of heredity (Darwinian individuals), he asserts:

Uptake of bacteria by the squid occurs not from its parents, but from bacteria in the sea. The parts of the ocean containing the squid have more of the bacteria than other parts of the ocean; there is a sense in which the squid are 'seeding' the ocean for other squid, when they expel excess bacteria each day. But if you are a squid, there is no mechanism ensuring that the bacteria in you are the offspring of bacteria in your parents, or any other specific individuals. The bacteria in you might come from many sources, and some might have not been inside squid for many generations. *Squid-Vibrio combinations 'make more of themselves' in one sense, but not in the sense that gives rise to parent–offspring lineages*. The parent–offspring lines connect only the parts – they connect bacteria with bacteria and squid with squid (Godfrey-Smith, 2013, p. 29, emphasis added).

This demand is even clearer in his more recent work, where he differentiates between reproduction and reconstruction, two phenomena commonly present in the biological world (Godfrey-Smith, 2015). Reproduction, according to him, occurs when there is a parent–offspring lineage, whereas reconstruction occurs when there is recurrence of biological objects, which is not due to lineage-formation, but to other processes. He exemplifies reconstruction with the case of the heart. When chordates reproduce, there will always be a heart both in the parents and in the offspring due to the reconstruction of the organ during development. The heart in the offspring is, however, not caused by the heart in the parent. It appears because there is a higher-level parent–offspring connection that includes hearts as one of its component parts. As a result, no heart-lineage is created, and thus hearts are not Darwinian individuals. The same would be

the case for the Squid-*Vibrio* system, as reflected in the previous quote. But in this case, in contrast with the heart example, the reconstruction happens because there is a lower level where parent–offspring relationships exist. Thus, according to Godfrey-Smith, the generation of “more of the same” Squid-*Vibrio* combinations is not causally triggered by previous Squid-*Vibrio* combinations but rather by the encounter between independent Darwinian individuals that can ephemerally live together.

All this shows that the existence of lineages sustaining the possibility of knowing “who came from who” is a condition sine qua non for reproduction, and hence for considering something a unit of heredity or Darwinian individual under Godfrey-Smith’s account. While the concerns about knowing “who came from whom” and detecting lineages are reasonable for certain purposes, including studying certain aspects of the units of selection (e.g., the accumulation of design at a focal level), we suspect this demand is ungrounded to characterize the evolutionary unit of heredity. Recall that in Sect. 2 we already said that the questions about the evolutionary unit of heredity and the unit of selection should be kept apart. Here, we complement that view with the observation deriving from MTE that indicates that evolved mechanisms of heredity (like those involved in the creation of lineages) evolve from units that lack these mechanisms. Demanding that parent–offspring lineages exist to detect a unit of heredity is conflating the existence of mechanisms of heredity with the existence of hereditary processes. As Bourrat (2014, p. 518; 2015) has convincingly shown, lineage formation at one level is an evolutionary complex adaptation conditional on the evolution of specific mechanisms, and the concept of Darwinian individuality already presupposes their existence. Thus, taking it for granted as the basis for the concept of an evolutionary unit of heredity may precisely mask the existence of some evolutionary units of heredity lacking these very mechanisms whose origin is investigated by studying the process of heredity.

Secondly, and more importantly for our purposes in this work, Godfrey-Smith’s account commits him to a strong position regarding SOL as the privileged way of characterizing SOT and hence to the fallacy of misplaced concreteness. This is especially clear and highly problematic in the case of the Squid-*Vibrio* symbiosis. Imagine that, for some reason, a jointly expressed trait, like bioluminescence, shows transgenerational phenotypic variation. In this case, there is a phenomenon of SOT at the system level. But since the system does not constitute the type of structure that Godfrey-Smith considers necessary to acknowledge the existence of a unit of heredity, he would say that this phenotypic variation needs to be carved up either at the level of the squid or at the level of the *Vibrio*, as they are the parts of the consortium forming lineages. This is, however, problematic, for this would precisely mask the actual level where hereditary relationships are manifesting. Of course, there may be good reasons to prefer Godfrey-Smith’s option. In the end, squid-lineages and *Vibrio*-lineages are easily traceable as both are collective and single Darwinian individuals, respectively. But this conflates ontology with epistemology, i.e., it conflates whether heredity is happening (and what heredity is), with the empirical tools we rely on to investigate whether it is happening. Furthermore, it conflates the evolutionary unit of heredity, i.e., the one where SOT is manifesting, with the evolved lineage-forming entities that compose the unit. This is not only conceptually problematic but also empirically so. It may mask genuine processes of heredity with real potential to evolve hereditary mechanisms at

that level, a point many authors before us have already emphasized (Lloyd & Wade, 2019; Molter, 2020; Stencel, 2016; Sterner, 2015; Suárez, 2020).

Overall, this section has shown that the problem that has masked the proper conceptualization of heredity lies in considering SOL as the only way of realizing SOT. We have shown how the concept of SOL has expanded from the early days when it was conceptually associated with the transmission of one or a few privileged elements (replicator concept) to the transmission of more complex structures via known evolved mechanisms (reproducer, Darwinian individual concepts). In fact, the expansion of the concept came together with an epistemological expansion of the methods to detect lineages. The paradox of such expansion, however, is that the conceptual association between heredity and SOL was maintained. We argued that this conceptual association ultimately blocks the development of an adequate concept of the unit of heredity, which depends on the detection of SOT at one specific level of the biological hierarchy. Relying on the notion of SOL to account for SOT constitutes a fallacy of misplaced concreteness, in which one structure is supposed to account for the existence of a complex phenomenon or process. In the next section, we introduce the concept of reconstitutor as the evolutionary unit of heredity, which, we contend, avoids the problem of reducing SOT to SOL that characterizes previous accounts of heredity.

4 The reconstitutor as the evolutionary unit of heredity

As an alternative to the concepts of the replicator and the reproducer, we propose the concept of the reconstitutor as the unit of heredity. There are two reasons for our preference to coin the new term “reconstitutor” to refer to the concept of unit of heredity that we introduce, rather than to present our approach as an intensional and extensional redefinition of the “reproducer.”

Firstly, “reconstitutor” is introduced for its semantic association with the idea of *reconstitution*, which the *Oxford Dictionary* defines as “the act of forming of an organization or a group again in a different way.” Semantically, it contrasts with reproduction because while both concepts emphasize the idea of doing or producing something “again,” only reconstitution emphasizes that this process can be attained “in a different way.” We interpret the latter meaning that reconstitution can occur via the interaction of different elements than the ones making up the first unit and through a plurality of ways of organizing these entities. Note that this strongly contrasts with the requirement concerning the establishment of continuous lines of ancestry. In this vein, the concept of the reconstitutor seems more apt to divorce the notion of heredity from the notion of lineage formation.

Secondly, the concept of the reproducer has been historically associated with the notion of an evolved unit that bears at least some evolved mechanisms allowing its components are transmitted together, or at least are transmitted in a way that preserves their structural and functional integrity. But, as we pointed out in Sect. 2, SOT does not require that the elements being passed preserve their structural and functional integrity, nor does it require that they are passed from the same source or via evolved mechanisms. Whether SOT (the empirical basis of heredity) is realized via these elements is a matter of evolutionary contingency, not of conceptual necessity. Therefore,

the concept of reproduction should be divorced from the concept of heredity (or conceived as a subset of hereditary phenomena) and substituted by a more adequate one to capture the real nature of hereditary phenomena. We believe the reconstitutor is the most adequate term to fulfil this role.

Ontologically, our discussion so far suggests that: (1) the concept of the evolutionary unit of heredity cannot privilege lineage-forming entities over those that do not form lineages; (2) the existence of evolved mechanisms for the re-creation of the form (i.e., reproductive mechanisms) is not necessary, even though it may be sufficient; (3) any characterization of the unit of heredity should be such that it accounts for the existence of SOT, and hence it should minimally incorporate the dynamic nature of hereditary phenomena. Based on this, the reconstitutor can be defined as:

Reconstitutor: *The structure resulting from a set of relationships between different elements or processes that are actively involved in the recreation of a specific phenotypic variant in each generation regardless of the biomolecular basis of the elements or whether they stand in a continuous line of ancestry (Fig. 2).*

Our definition allows the operationalization and empirical exploration of heredity via its appeal to two key requirements: the active involvement of the interacting elements and the requirement of distinct phenotypic variants.

By “being actively involved,” we mean that the elements that interact to give rise to a reconstitutor act as causal difference-makers in producing the trait. That is, the trait

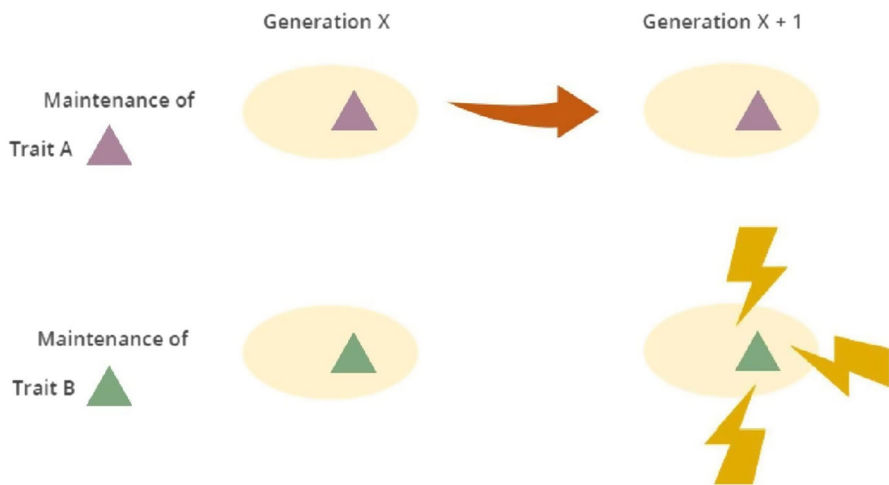


Fig. 2 Exemplification of what reconstitutors do, in two different ways: (a) through reassembly by direct transmission (thus through the reliance on the same material entity); (b) through reassembly without transmission (no reliance on the material continuity)

will not be produced, as such, if the elements are not present, but a different trait will be produced in their absence. Additionally, it requires that there is a certain selective affinity between the interacting elements, i.e., that the involved elements will tend to interact with each other to re-create the phenotype even when there are other elements available, and not simply as a side-effect of the interaction between other elements.⁵ The active involvement comes by degrees, ranging from cases where there is a history of design or natural selection causing the elements to come back together to recreate the trait variant (e.g., in asexual reproduction in unicellular organisms, in sexual reproduction in multicellular, in cases of vertical transmission of endosymbionts, etc.) to cases where the correlation between the elements is not a result of design, but it is nonetheless so persistent transgenerationally (e.g., due to the existence of constraints of various types) that it may, but does not need to, lead to the evolution of design in the long term.

By “distinct phenotypic variants,” we mean that the trait that is produced by an interacting set of elements may be qualitatively the same as the trait that a different set of elements would have produced, but it must simultaneously manifest certain differences concerning the properties of the trait that would have been produced if the set of interacting elements were different. Thus, a group of elements will be part of the reconstitutor and not part of the environment even in cases where these elements are shared among the members of the population only if those elements are actively involved in the re-creation of the phenotype, and their sharing does not result in breaking down the possibility of phenotypic variants among the members of the population.

Intensionally, our characterization of the reconstitutor shares Griesemer’s emphasis on the developmental processes as an integral component of the concept of the unit of heredity. But Griesemer’s condition of material overlap during progeneration and his requirement that development is conceived as “acquiring the capacity to reproduce” differentiate both concepts. Reconstitution describes cases in which the entities need not be transmitted for trait reappearance, as well as cases where the new entity does not acquire the capacity to reproduce on its own, insofar as it may be formed by independent lineage forming entities. Analogously to Godfrey-Smith’s Darwinian individual, reconstitution demands that certain conditions persist at the focal level so that SOT can be realized. But it does not require the “focal level” to be formed via the establishment of a continuous line of ancestry, be it at the material or formal level. The establishment of these lines is a product of evolution, and the evolutionary unit of heredity must be something that facilitates evolution rather than something that results from evolution. Extensionally, the relationship between the type of phenomena captured by the reconstitutor and other evolutionary units of heredity is summarized in Table 1.

Our concept of the reconstitutor and its exact relationship to the concepts of the reproducer and SOT can be specified as follows:

⁵ “Selective affinity” does not mean that the affinity has been naturally selected, or that the interacting elements have the evolved function to interact in that manner. Otherwise, our account would presuppose that evolution must have jointly affected the interacting elements, as it is presupposed e.g. in Jablonka’s (2002) account. We, however, reject that this is a requirement.

Table 1 Type of biological phenomena that would fall under the extension of the concept of different units of heredity. NC means "niche construction"; ITSNTS means "it's the song not the singer"; ITSATS means "it's the singer and the song"

	DNA	Sexual/ Mendelian reproduction	Retroviruses	Constraint related heredity (small RNAs)	Functional Microbiome	Taxonomic Microbiome	Ecological inheritance - phenotypic variant + active maintenance	Biogeochemical stability - phenotypic variant + active maintenance	Ecological inheritance - no phenotypic variant + active maintenance	Reference
Replicator	✓	✗	✓	✗	✗	✗	✗	✗	✗	Dawkins (1976), Hull (1980)
Reproducer	✓	✓	?	✗	✗	✗	✗	✗	✗	Griesmer (2000, 2014)
Darwinian Individual	✓	✓	✓	✗	✗	✗	✗	✗	✗	Godfrey-Smith (2009, 2013), Stalings (2016)
Reconstitutor	✓	✓	✓	✓	✓	✗	✓	✗	✗	This work
NC Theorists	✓	✓	✓	✓	✓	✓	✓	✗	✓	Laland et al. (2003)
ITSNTS	✓	✓	✓	✗	✓	✗	✓	✓	✗	Doelittle and Inkpen (2018)
ITSATS	✓	✓	✓	✓	✓	✗	✓	✓	✓	Baerleise and Papale (2021)

Boxes marked with "?" represent cases where to determine whether the phenomenon is a case of inheritance according to the concept is disputed or undetermined

- (1) *All reproducers are reconstitutors.* A reproducer is a reconstitutor in which trait-variant generation occurs from the interaction between a set of elements transmitted exclusively from one entity or a discrete set of entities (e.g., two in sexual species), resulting in the creation of a lineage. Units manifesting trait-variants via lineage formation are reconstitutors.
- (2) *Not all reconstitutors are reproducers.* Those reconstitutors that reassemble a local pool of elements (e.g., lineages) derived from many more than one previous entity, to the point that the set of entities is not identifiable or in a continuous line of ancestry despite the fact that the trait-variant is, are reconstitutors that are not reproducers. As a result, units that do not create lineages can be units of heredity.
- (3) *The material elements that comprise a reconstitutor can be empirically identified in the process of generation of the trait.* The entity or entities need to play an identifiable active role in generating a trait. This can be tested, e.g., by showing how they act as difference-makers for different variants of the trait, such that if one of these is removed, the trait variant is linearly or nonlinearly altered in a way that can be empirically recognized. Additionally, selective affinities between the elements comprising a reconstitutor can be empirically tested.
- (4) *The material elements that comprise a reconstitutor may derive their influence on the trait(s) from their joint effects (systemic properties).* These joint effects sometimes appear as a result of the convergence of several entities or as a result of the convergence of several lineages.
- (5) *The biological elements that comprise a reconstitutor may be stable if subjected to certain perturbations once the reconstitutor has been generated, but are not indefinitely stable.* Some elements comprising a reconstitutor may interact in a way such that the removal of one of the elements does not affect the expression of the phenotypic trait-variant.
- (6) *The existence of reconstitutors ontologically describes the minimal entities involved in SOT.* As the concept of the reconstitutor allows that the material basis grounding the reappearance of the phenotypic variant might change trans-generationally due to different elements (e.g., changes in the available elements in the population or their ways of assembling, environmental pressures, etc.), reconstitution can lead to a constant change in the material basis of heredity without necessarily altering the phenotypic variants being inherited. SOT occurs because there is a reconstitutor that allows that all these changes do not entail a change in the recreation of trait variants.
- (7) *The existence of a reconstitutor is not ontologically prior to the existence of the set of relationships that cause its appearance.* To put it differently, the reconstitutor is an effect of the set of relations and not its cause. If the situation were the inverse, then we would presuppose the existence of an evolved structure that re-orders the relationships, which would make the reconstitutor identical to the reproducer and hence prone to the same type of problems as the latter.
- (8) *The concept of the reconstitutor is not confined to specific levels or scales and applies up and down the biological hierarchy.* The concept of reconstitution is impartial regarding the units of heredity and other types of units of selection, being open to finding them across different levels of biological organization.

To sum up, the concept of the reconstitutor captures the mounting evidence that suggests that reproduction and lineage formation are not necessary for SOT, and thus not all hereditary phenomena rely on these types of processes (Charbonneau, 2014; Merlin & Riboli-Sasco, 2017, 2021; Papale, 2021; Rainey & De Monte, 2014; Suárez & Triviño, 2019). Previous works, however, have failed to introduce a specific unit that could be used as a single conceptual tool to account for all these phenomena in a way that unifies them as elements of the same family. The concept of the reconstitutor is thus an essential conceptual tool for contemporary biology, and a proper understanding of it has practical implications. In the following sections, we develop the concept by relying on examples.

5 The empirical grounds of the reconstitutor: two exemplars

Sections 5.1 and 5.2 present two cases from contemporary biology (small RNA states and holobionts) where SOT is observed, but the level where SOT is observed cannot be accounted for by the replicator, the reproducer, or the Darwinian individual. However, we will show that they have features attributed to reconstitutors. In this way, they are exemplars for our concept and a good way of illustrating the type of phenomena it aims to cover.

5.1 Small RNA states

Small RNAs are non-coding RNAs studied for their regulatory roles in almost all known species (Fire et al., 1998). They target and sometimes destroy complementary messenger RNAs, inhibiting the synthesis of a specific protein. Small RNAs are involved in several regulatory tasks, such as control of transposons (Malone & Hannon, 2009), metabolic regulation (Cai et al., 2009), and defense against viruses (Hamilton & Baulcombe, 1999). Small RNAs also have heritable effects. In several model organisms, they were shown to cause heritable responses to certain adverse environmental conditions (Heard & Martienssen, 2014). Thus, a trait variant produced by small RNAs, e.g., resistance to a particular virus, can sometimes persist transgenerationally (Rechavi et al., 2011).

Let us illustrate small RNA-based SOT with an example. This example is situated within one of the best-studied model organisms regarding small RNA-based inheritance: the nematode *C. elegans* (Rechavi & Lev, 2017). A particular environmental trigger causes a small RNA response. There are millions of small RNA molecules in each cell. They compete for effector molecules. These effector molecules are involved in small RNA-based silencing and partly in the transgenerational maintenance of small RNA-based effects.

Because small RNAs are responsive to environmental triggers, the proportions of types of small RNAs within each cell change if exposed to environmental triggers, i.e., more trigger-induced small RNAs compete with other species of small RNAs for effector molecules (Sarkies et al., 2013; Veigl, 2017). Trigger-induced small RNAs

are heterogeneous and partially specific to the particular trigger.⁶ Virus-induced RNAs are the most straightforward examples, as they are complementary to a particular virus (Rechavi et al., 2011). Starvation-induced RNAs include, e.g., small RNAs that regulate yolk-protein coding mRNAs (Rechavi et al., 2014). Environmental trigger-caused differences in proportions of different small RNA have been reported to persist throughout generations. Some environmental triggers cause the synthesis of small RNAs that were not present in the pool of small RNAs before the trigger (as is the case with the virus example), whereas in other cases, environmental triggers lead to an increased or decreased expression of certain species of already present small RNAs. As a result, the progeny of *C. elegans* challenged by a particular environmental stimulus display resistance to this stimulus.

A small-RNA-based trait induced by an environmental stimulus persists throughout generations without a corresponding DNA sequence causally relevant for that trait. As this trait is caused by small RNAs and maintained through changes in small RNA equilibria, many suspect that small RNAs are the heritable agents that ensure the persistence of the trait (Sarkies & Miska, 2014). One particular reason that suggests that small RNA inheritance phenomena should fit into the imperative of replication is that RNAs and DNAs have similar molecular properties and capacities. Most importantly, both can be replicated, and both are viewed as “coding” information (Smardon et al., 2000), and both were taken as paradigms in Dawkins’ abstract conception of the replicator. We believe, however, that transmission of small RNAs is at best an insufficient explanation for the observed SOT. In what follows, besides providing a proof of concept, we aim to offer a contribution to and critique of small RNA inheritance investigative practices which currently presuppose the necessity of the transmission of a particular entity (e.g., Gapp et al., 2014).

Several conceptual and empirical challenges dissuade a perspective that relies on faithful transmission of small RNAs. To explain, we will have to divert our attention to some facts about multicellular organisms’ physiology. Metazoans consist of many cells. *C. elegans*, a model organism highly esteemed for its relatively “small” number of cells, consists of about 1000 cells. An environmental stimulus is a stimulus to somatic tissues and not to the germline and causes changes in pools of small RNAs in somatic cells. This change does not happen in one somatic cell but a significant number of somatic cells.

A hypothetical transmission of causally relevant small RNAs would require that a particular set of small RNAs reaches the germline, be passed through it together with the reproductive cells, and then cause a phenotype in the next generation by reconstituting the RNA states of the parental generation.⁷ This would run along SOL, with material overlap, analogously to how the reproducer and the Darwinian individual would suggest. Empirically, the evidence for such processes taking place is scarce at

⁶ There are several reasons why trigger-induced RNAs are only partially trigger-specific. First, exposure to a trigger will also cause the upregulation of small RNAs that are involved with stress-responses in more general. In addition, given the quantitiveness of small RNA states, any change in small RNA concentrations (e.g. the synthesis of trigger-specific small RNAs) will also lead to changes in the amount of other small RNA species, given them competing for the same effector molecules.

⁷ Whether small RNAs in *C. elegans* can persist transgenerationally without reaching the germline—e.g. through secretion and intake—has, as of now, only been addressed theoretically (Sarkies & Miska, 2014).

best; more accurately put, non-existent. This is partly because faithful transmission of, e.g., trigger-specific small RNAs, poses an insurmountable “administrative” problem. No matter whether one, several, or a pool of small RNAs are causally relevant for a specific phenotype; it remains unclear how the specific causally relevant entities can be faithfully trafficked into sperm or unfertilized eggs. How would each germ cell acquire the same amount of an individual small RNA or the same concentration of pools of small RNAs? Besides, it is impossible in the next generation to divide small RNA populations amongst daughter cells following each cell division so that daughter cells would display the same or similar small RNA concentrations. Meiosis allots small RNAs in a way that might be somewhat 50/50, but as small RNAs have been shown to locate irregularly across the cytoplasm, their distribution could also be quite uneven (see the recent literature on germ granules, e.g., Dodson & Kennedy, 2019; Lev & Rechavi, 2020).

While we believe that the “administrative” problem is a crucial conceptual roadblock for SOL in the case of small RNA inheritance, we do not argue that there is no transmission of small RNAs: given that small RNAs locate in the germline, it is necessarily so that some small RNAs originating in the parental generation will materially persist and have effects in the offspring generation. However, the critical point is that the stability of some small RNA entities cannot fully explain the persistence of a small RNA state that induces a particular trait. Neither can lineages of cells instantiated through cell division. While at the organizational level of the cell, two segments in a lineage of cells resemble each other in displaying a particular trait (and a small RNA state that corresponds with the trait, e.g., resistance to a virus), the small RNA states are only functionally equivalent. At the organizational level of small RNA states in two segments of a cell lineage, these states might not be comparable (either because of different proportions of collinear small RNAs or the absence of collinearity), and cell division cannot ensure comparability. The case of small RNA inheritance is a case where the causally relevant elements do not form lineages. Small RNA pools of two units (say, the parental and the F1 generation) are not comparable; in fact, each and every RNA molecule might be different, but it can still cause the same phenotypic effect or a variant of such effect.

The process of reconstitution of small RNA pools that are functionally identical with the pool in the prior generation is feasible thanks to the interaction of a multiplicity of causally relevant elements. These elements include chromatin modifications in loci that affect the amplification of small RNAs (Lev et al., 2017), concentrations of small RNA effector molecules such as Dicers, Argonautes, and RNA-dependent RNA polymerases (Gu et al., 2009), and subcellular structures such as germ granules (Sundby et al., 2021). Also, second exposures to environmental triggers have been shown to stabilize small-RNA-caused phenotypes (Houri-Ze’evi & Rechavi, 2017). Second triggers extend the transgenerational persistence of a small RNA state for several generations. These elements, in turn, persist through a multiplicity of ways. Chromatin modifications can, in most cases, not be directly transmitted given their erasure after fertilization; yet, they are believed to be re-established by small RNAs complementary to the modification-bearing regions. Effector proteins are transmitted as well as newly synthesized. The expression of effector proteins has, however, been shown to be highly

influenced by small RNA-dependent chromatin modifications. Germ granules—sub-cellular condensates microscopically distinguishable through high densities of protein and RNA—are maternally deposited and re-nucleated later in embryogenesis. While these entities are transmitted in a way that conforms with SOL, their integrity in each generation and their further persistence are dependent on small RNAs. SOL of these elements can, thus, not cause the persistence of the phenotype if taken in isolation. A set of relations of various components that is itself dependent on interactions with small RNAs reassembles a small RNA state, which recreates the phenotype of the previous generation.

Let us stress that by a discontinuity of lineages, we mean that it is not necessary that any particular entity, determined by, e.g., a particular sequence of Uracils, Guanines, Adenines, and Cytosines, reoccurs. If one would sequence the small RNA pools in the parental and the offspring generation, both being causative of a particular trait that both parent and offspring bear, the sequences in the stimulus-specific RNA state in parent and offspring would not be collinear. This is because the small RNA state is not transmitted but reconstituted. Instead, the specific process reappears, caused by the reappearance of a functionally equivalent small RNA state. But the RNA molecules themselves cannot account for the trait, and neither do the cells carrying them. Even if a lineage of cells persists, it cannot guarantee SOT through SOL of small RNA states.

In conclusion, we need to rethink the questions we ask about small RNA inheritance. Instead of asking: “How can a specific molecule causally related to a phenotype be maintained?”, as is done in the small RNA inheritance field (Gapp et al., 2014), we need to ask: “Which conditions need to be met in order to guarantee the stability of traits?” The reconstitutor accounts for the stability of a particular trait caused by the stability of an RNA state, itself caused by processes that have nothing to do with lineage formation. While the material basis that caused a trait is not available because it cannot be vertically transmitted faithfully—there is no SOL—the reconstitutor reassembles the causally relevant structures, effectors, and sets of relations (e.g., exogenous small RNAs, endogenous small RNAs, modification on chromatin loci known to amplify small RNAs) in a way that recreates a functionally equivalent small RNA state (Fig. 3).

5.2 Holobionts

A growing amount of biological research has demonstrated that the microbiome has substantial effects on the host’s phenotype, necessitating the concept of the “holobiont” (host + microbiome) and its consideration as a unit of heredity. For instance, different symbionts are known to determine thermal tolerance, protection against parasitoids, and body-color in aphids (Dunbar et al., 2007; Oliver et al., 2009; Tsuchida et al., 2010); several representatives of *Spiroplasma* are known to protect flies of the species *Drosophila neotestacea* from parasitic nematodes that would cause infertility (Jaenike et al., 2010); acquisition of new endosymbionts might be implicated in causing speciation events in some insects (Brucker & Bordenstein, 2012, 2013; White, 2011); symbionts are supposed to be involved in the transition from sea to land environments in certain eukaryotes possible (Lipnicki, 2015); angiogenesis in mice is known to be driven by *Bacteroides thetaiotaomicron* (Stappenbeck et al., 2002);

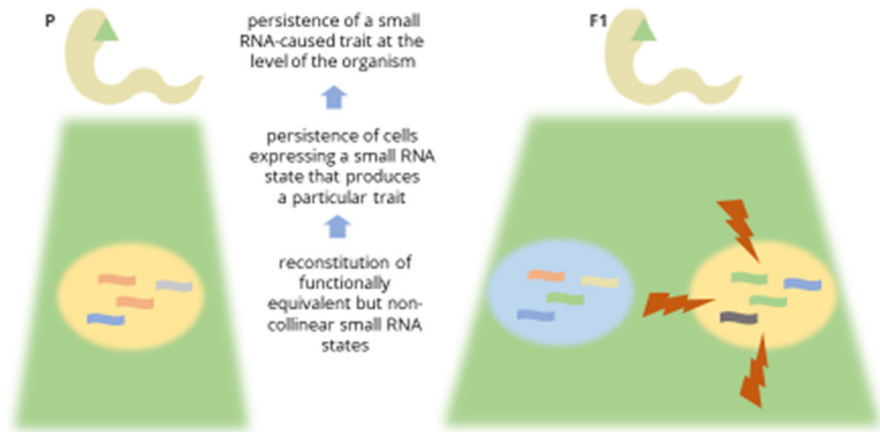


Fig. 3 Exemplification of reconstitutor-operated transgenerational maintenance of small RNA pools. In the parental generation, a particular small RNA state produces a particular trait (triangle). In the F1 generation, a functionally equivalent small RNA state (yellow) gets reconstituted (from the blue small RNA state) through the interaction of different elements. Thus, while at the level of the worm and the level of cells lineages are discernible, at the level of the small RNA there is no SOL, but SOT is guaranteed through reconstitution

butyrate production in humans is known to depend on a complex dynamic interaction between different bacterial species, including *Bifidobacterium* spp. and *Eubacterium hallii*, among others (Dethlefsen et al., 2007); hematophagy in vampire bats is known to be partially controlled by the microbiome (Zepeda Mendoza et al., 2018).

While in some of the cases mentioned above, the phenotypic modifications are caused by identifiable parent–offspring lineages of microbial species, this is not so for every case where microbiota have been proven to have a critical phenotypic effect on their host. Furthermore, empirical evidence suggests that the microbiome, as a whole, is not vertically transmitted when a host reproduces. Even while the evidence for/against vertical transmission is contradictory to a certain extent depending on the host species (Rosenberg & Zilber-Rosenberg, 2018), indeed, the microbiota that interact with a host suffer compositional changes due to environmental stresses, including factors such as dietary changes (Singh et al., 2017) infections (Li et al., 2019), seasonality (Stencel, 2021), etc. This observation reinforces the belief that vertical transmission is a rare event, and hence host-microbiota combinations do not form identifiable parent–offspring lineages. For these reasons, and grounded on the concept of the reproducer or Darwinian individual as the unit that captures what needs to be preserved to detect hereditary relations, several researchers have rejected the idea that there are hereditary relations among holobionts (Douglas & Werren, 2016; Moran & Sloan, 2015; Skillings, 2016; Stencel & Wloch-Salamon, 2018), or have restricted it to cases where the degree of vertical transmission is proportionally high (Lloyd & Wade, 2019). This leads to a view of the phenomenon of host-microbiome association that may diminish its connection to certain ways of evolving darwinianly, e.g., by developing higher-level adaptations for the host-microbiome system.

There is a general problem with this rejection, deriving from the narrow view of the process of inheritance encouraged by the assumptions of SOL and the reproducer/replicator frameworks. The problem concerns the requirement of identifying one specific microbial taxon instead of a possibly complex combination of taxa that can be deemed responsible for the phenotypic trait. The problem is the assumption that the process needs to be accountable in terms of the production of identifiable parent–offspring lineages at the host-microbiota level. Our primary concern is that this assumption is not correct in many cases because some recent research suggests that on occasions, the trait only appears due to the functional components and not the taxa composing the microbiome (Doolittle & Booth, 2017; Suárez, 2020; Taxis et al., 2015), and it is known that these functional components cannot form lineages independently of the taxa that bear them. Furthermore, the problem gets more complex because, on some occasions, the effect does not directly derive from the identification of one or several taxa or one or several functions, but rather from the interaction between several taxa in specific proportions, insofar as these proportions result in specific interaction patterns (Łukasik et al., 2018; Tanoue et al., 2019).

An elegant example of this complexity is provided in a recent study by Tanoue et al. (2019), where the authors show that a consortium of 11 bacterial strains of the gut microbiome enhances mice's resistance to *Listeria monocytogenes*. Different combinations of the same strain fail to elicit enhanced resistance. Another example comes from work on cicadas, where it is proven that three endosymbionts evolved from the same similar ancestor of the genus *Hodgkinia* bear complementary genes (Łukasik et al., 2018). This suggests a convergent evolution that has generated complementarity such that several phenotypic effects on cicadas may result from the joint action of these three endosymbionts. A theoretical example comes from Huitzil et al.'s (2018) modeling of holobiont evolution in terms of genetic networks, which could be used to interpret the evolution of sanguivory in bats (Suárez & Triviño, 2020). In the Huitzil et al. (2018) model, genetic regulation is not transmitted: instead, it is recreated in every new generation. Yet, the model clearly shows how this does not affect the possibility that these relationships are recreated, the traits are stabilized, and, in due time, this accumulation may occur and may have evolutionary effects.

These three examples illustrate that transgenerational preservation of host-microbiome traits may obey diverse causes: a consortium of species interacting in specific proportions and generating an emergent biological system, the complementarity of a few variants of the same species that had experienced genomic decay, or a whole arrangement of redundant functions encoded by several taxa within the microbiome, to name a few (Fig. 4). This shows that the establishment of re-current trait-forming interactions between a host and its microbiota may occur through many different routes, many of which do not require forming clear parent–offspring lineages at the level of the consortium.

We suspect, though, that part of the reason why the existence of heredity among holobionts has been neglected despite the evidence supporting the existence of SOT derives from two sources. On the one hand, microbiome scientists are frequently interested in some pragmatic problems concerning how phenotypes come about, and their research agendas are not primarily occupied with whether and, if so, how the processes triggering SOT can be conceptualized as hereditary phenomena (Huss, 2014). On the

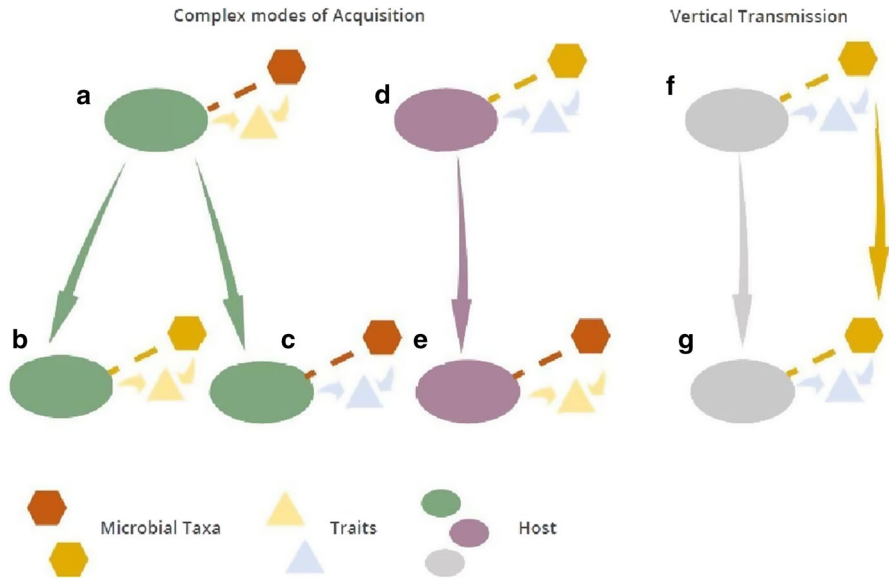


Fig. 4 Application of the ideas of reconstitution and SOTs to holobionts. Arrows stand for reproductive relations between hosts, while dashed lines stand for joint epistatic effects between some microbes and the host that lead to some traits at the community levels represented by colored triangles. Different colors represent different variants. According to the reconstitutor concept, *a*, *b*, and *e* on the one hand, and *d* and *b* on the other, would belong to the same hereditary lineage in virtue of their capacity to reconstruct the same traits. This is so even though *a*, *b*, and *c* on the one hand, and *d* and *e* on the other constitute a single host lineage. According to the reconstitution concept, they are not in genuine hereditary relationships concerning the trait, although they may be in hereditary relationships concerning other traits whose variation depends solely on the host. Finally, *f* and *g* constitute a case of reconstitutor based upon the vertical transmission of the elements and their phenotypic effects. It is a case in which the reconstitutor is also a reproducer

other hand, concepts such as the replicator, the reproducer, and/or the Darwinian individual, and the type of underlying assumptions concerning SOL they are based on, are frequently assumed in the study of hereditary relations among biological systems. The latter issue is especially important. Holobionts are composed of a host and a set of microorganisms of different species, each with independent reproductive capacity, and hence each suitable to be considered a replicator, reproducer, or Darwinian individual on its own, as each of these parts forms lineages and satisfies SOL. But holobionts, on the contrary, fail to form lineages and result from the collection and assemblage on independent lineages that combine horizontally to re-create the holobiont and its traits transgenerationally (Moran & Sloan, 2015; Godfrey-Smith, 2015; Douglas & Werren, 2016; Skillings, 2016; Stencel, 2016). Thus, under the narrow frameworks that conceive heredity as a product of lineage formation, the holobiont cannot be conceptualized as a hereditary system.

In contrast with this picture, we agree with Roughgarden (2021) that the lack of vertical transmission or any other of the properties required for bona fide heredity under the replicator, reproducer, and Darwinian individuality frameworks is simply an effect of the idea that host-multispecies systems explore heredity by other means, and hence

allow for the transgenerational effects that we have ascribed to SOT. The way in which hereditary relations among holobionts occur is still open to empirical investigation in different systems, and the answer to this question will be the ultimate answer to whether the traditional models of heredity fit the picture or not. Yet the reconstitutor provides a platform for thinking that, based on our contemporary knowledge about holobionts, there may be at least two different ways that sometimes will act in combination:

- (1) *selective affinity* between individual hosts and certain microorganisms, which leads to a transgenerational recurrence of the trait or traits expressed by the collective (Lloyd & Wade, 2019; Roughgarden, 2021). In this case, the epistatic effects between the host and the microbes would increase the frequency of transgenerational recurrence of host and microbial association, which would guarantee the transgenerational production of trait variants at the holobiont level. This is the case in squid-*Vibrio* symbiosis, but also in other symbioses in which the host/microbe serve as genetic environments for each other (Drown & Wade, 2014; Drown et al., 2013)
- (2) *specific joint effects* that will tend to drive the host-microbiome system towards specific points of equilibria where the phenotypic effects will be channelled and maintained despite perturbations (Suárez, 2020; Suárez & Triviño, 2020). An example would be the case of vampire bats, in which the host environment creates the conditions in which genetic amplification of specific variants, triggered by selection between microorganisms within the microbiome, and horizontal gene transfer generate a microbiome that is apt to cope with the challenges of sanguivory that the bat genome cannot cope with without its microbiome (Zepeda Mendoza et al., 2018). These genetic variants encoded by the microbiome are required to be amplified for sanguivory to be feasible and are amplified only after birth, being this the key moment when the holobiont transitions between a set of relationships to a new set of relationships (Chiu & Gilbert, 2015) as a result of microbiome-assemblage due to the joint host-microbiome effects. Establishing these relationships requires a specific pattern of genetic expression triggered by the host-microbes interaction, and this pattern is obtained without vertical transmission. To put it in our terms, the patterns need to be reconstituted through complex interactions between different lineages.

The combination of these two effects in the holobiont-reconstitutor recreates the holobiome trait-variants from the previous generation such that SOT is preserved at the holobiont level.

6 Phenotypic variation: the case of ecological inheritance

Some biologists have recently become attentive to the active role of organisms in adapting to their environments. The early population genetics models that inspired Dawkins' concept of the replicator assumed the environment as a constant, an invariant element in genetic modeling, with the organism (and consequently its replicators) being the part that changes or adapts to it. This new movement, in contrast, leads to a view in which organisms are actively changing their environments (Lewontin,

1982; Lewontin & Levins, 1985). As a result, they might alter the selective pressures affecting the whole population over time. This phenomenon has been called *niche construction* (Laland et al., 2000, 2014; Odling-Smee & Laland, 2011) to reflect the idea that organisms alter parts of their environments and hence adaptation is not one-sided, but a two-sided process of organism-environment accommodation (Krakauer et al., 2009; Tanaka et al., 2020).

Niche construction theorists emphasize that some of the environments actively built by some organisms are preserved so that their descendants can inherit them already tailored to their biological necessities. The totality of environmental changes inherited in this manner is considered ecological inheritance, by contrast to those forms of inheritance that do not directly concern the environment (Odling-Smee & Laland, 2011). For example, earthworms make tunnels in the soil to keep the right humidity conditions, and these tunnels will be kept for subsequent generations (Jones et al., 1994; Odling-Smee & Laland, 2011). Beavers build dams, and in doing so, they alter the selective pressures that affect populations around the lake where their dams are built. Future generations of beavers inherit these dams, which are often maintained or reconstructed by families of beavers for years with an important ecological impact (Naiman et al., 1988; Odling-Smee & Laland, 2011). Dam construction involves the alteration of the selective pressures affecting the beaver population, but once the dam is built, these alterations are kept intact for the next generations of beavers, which are not required to build the dam again, but only to maintain it.

According to niche construction theorists, these would be cases of ecological inheritance because a specific structure or resource (the earthworm tunnels, the dams, etc.) is preserved transgenerationally. Similarly, some examples of holobiome inheritance have recently been characterized as ecological inheritance, given that the tailored microbes can spread among the host population (Haag, 2018). Hence, we have to determine whether the reconstitutor is extensible to cases of ecological inheritance. And, if so, whether the criteria we have presented in our characterization would cover only parts or the totality of phenomena included under the general concept as this is presented by niche construction theorists (Odling-Smee & Laland, 2011).

We believe that an appropriate characterization of the evolutionary unit of heredity should only cover some instances of ecological inheritance while excluding many others. The reason is that doing otherwise would risk indiscriminately covering any factor that affects the recreation of phenotypes in a population. But this would turn the concept on the one hand unoperationalizable—insofar as the group of possible factors recreating phenotypes is enormous and potentially indefinite—and, on the other, conceptually invalid—as it would entail blurring the distinction between the unit of heredity and the background conditions provided by its environment. For instance, organisms cannot re-create their phenotypes without access to water, oxygen, or sunlight. Nor can they if two germline cells do not fuse or if the microbiome is not correctly assembled. Yet, there seems to be a qualitative difference between these two types of factors. Therefore, there must be a way of conceptually discriminating between them.

Concretely, to avoid these problems, the unit of heredity should cover the cases where there is SOT, but not those where a factor affects SOT recreation, but not differentially. To put it differently, any conception of the unit of heredity must provide

criteria to distinguish between the elements that generate *new traits*, that contribute to the *conservation of the same traits*, and that contribute to *trait variation* (Sect. 2). We contend that the reconstitutor provides those criteria. But we need to explain the reasons why we believe this is so.

One possible criterion to exclude cases of ecological inheritance from our conception of the reconstitutor appeals to the fact that the resources involved in these processes are frequently shared equally among every member of the population, transcending the parent–offspring bound. But note that this criterion would not be valid under the reconstitutor version of the unit of heredity we are defending, nor is it included in our definition. The concept of reconstitutor does not impose any limitation on how much the benefits of a trait variant can be shared or spread among the members of the population (cf. Godfrey-Smith, 2009), and in fact, we consider this to be one of its virtues to cover several cases of SOT excluded under other accounts. Hence, this criterion is not valid for our purposes.

As our definition of the reconstitutor appeals to the active involvement of the elements in the process of re-creating phenotypic variants, our criteria of inclusion/exclusion must be grounded on these concepts. Some cases of niche construction may, however, be conceived as cases of active maintenance, as we have defined above, so active maintenance is not the most profitable avenue to defend our position. But, we contend, many of the ecological resources considered heredity by niche construction theorists cannot be conceived as elements that cause the appearance of distinct phenotypic variants.

Let us illustrate the concept of “distinct phenotypic variants” by relying on two paradigmatic examples of a background condition and a genuine ecological hereditary element. Compare the roles of oxygen in the air and a symbiotic microbe in producing a phenotypic variant in a strictly aerobic organism. Oxygen in the air is necessary to develop most of the phenotypic traits, especially since strictly aerobic organisms would perish in the absence of it. But while being essential for producing *any phenotype* may suggest that oxygen could potentially play a role in the transgenerational re-creation of constraints and thus could be regarded as an inherited element from an organizational perspective, it is not discriminatory enough to play a role in the production of the *phenotypic variant*, scoring poorly as an element to consider part of the reconstitutor as we conceive it, playing an evolutionary role. To play such a role, it is necessary that one can distinguish between phenotypic variations in the absence of oxygen vis à vis phenotypic variations in the presence of oxygen, which is not even a possible scenario in strictly aerobic organisms. Rather, in our account, we would argue that some organisms have evolved to tolerate oxygen, process it, or consume it as an indispensable element for their survival, yet it lacks the necessary properties to qualify as a part of the inherited system.

Contrast this case with the example of microbes or the traits that they encode. As we argued in Sect. 5.2, microbes and their hosts interact in a way that differentially causes the appearance of specific phenotypic variants, to the extent that the type of SOT that are preserved when certain host-microbe combinations are generated differs from the type of SOT that results from other host-microbe combinations. Note that this can even happen for the same host lineage, allowing to distinguish phenotypic variations within one population, which we claimed to be key for the concepts of SOT

and, consequently, the reconstitutor. Hence the difference between the oxygen and the microbe in cases of strictly aerobic lineages stands from the fact that while the former does not contribute to the recreation of phenotypic variation, but rather to the recreation of *any phenotype* (i.e., it is not causally discriminating with respect to the variants being created), the latter does so.

To shed further light, consider now the case of the earthworms and the beavers that we used at the beginning of this section. All earthworms can use the tunnels excavated in the previous generation, and all beavers can use the same dams. The effect of this is that no distinct phenotypic variants arise from the interactions between the environmental factors that have been constructed and maintained and the organisms that reside in a niche where these factors have been altered. Thus, the alteration and maintenance of these environmental factors do not lead to phenotypic variation, or SOT, but rather to a new form of selective pressure affecting the population as a whole. We agree that this alteration may eventually lead to the evolution of a different type of reconstitutors at the organismic level, but we contend that it does not lead to such evolution at the organismic-niche level, suggesting that these environmental factors are background conditions for the establishment of SOT.

Finally, let us contrast two cases of symbiosis that we consider may shed further light on the type of relationships captured under the concept of the reconstitutor. Recall that, according to niche construction theorists, cases of transgenerationally recurring symbioses may be considered examples of ecological inheritance (Dall, 2007; Krakauer et al., 2009). But we believe it is not correct to consider every transgenerationally recurring symbiotic association a case of reconstitution, or at least it is not so without further limitations. Plants and bees, for example, have evolved some mechanisms allowing them to attract each other and interact transgenerationally (Leigh, 2010). Yet all bees in different colonies can interact with different flowers from different species, and flowers are not discriminatory with respect to the insects they interact with. This results in the evolution of specific mechanisms in bees (and other insects) that favor sucking the nectar of several flowers, as well as several mechanisms in plants to guarantee that bees (and other insects) carry their spores and deposit them in other plants. Distinct phenotypic variants evolve at the levels of bees and flowers because bees and plants have interacted over several generations, but no distinct phenotypic variants arise from the interactions at the bee-flower level. In other words, bees and flowers change the selective pressures acting on each other, which may have obvious evolutionary effects for each of the lineages, but that does not need to translate into the existence of a joint unit of heredity.

Contrast this case with fungi-growing termites Macrotermitinae, which have also been considered an example of ecological inheritance (Dall, 2007; Krakauer et al., 2009). Macrotermitinae create colonies that grow “fungus gardens” in symbiosis with the fungus *Termitomyces*. These “gardens” are manured by termite workers in a cork-like structure called the “fungus comb,” which is found within the underground chambers of the termite mound and is comprised of predigested plant material (Mueller et al., 2005, Schalk et al., 2021). The process of how “fungus gardens” are recreated constitutes a good example of transgenerational reconstitution of a distinct phenotypic variant, as opposed to mere background conditions for the recreation of such variations in the termites or the fungus. Termite colonies start with a reproductive couple:

a future queen and king. Once they find a proper place, they seal themselves in what later becomes a “royal chamber.” After some time, the queen starts laying eggs. When the workers of the first brood become mature, they forage and establish the first fungus garden. To do so, the first foraging workers must pick up spores of the right species of fungus along with the first forage brought in from outside. The spores picked up by the workers come from fruiting bodies (mushrooms) that arise from other mature termite colonies and are dispersed by the wind (Mueller et al., 2005). There is no lineage continuity, as each termite can pick up fungi from different lineages, and fungi from a specific lineage may be picked up by termites from a different colony, with no data on vertical transmission (Darlington, 1994; Nobre et al., 2010). After the fungi collection has finished and the “fungus garden” has been established, its maintenance would depend on the specific ways in which the termites and the fungi interact over time. The fungus garden is localized in the underground chambers of the termite mound, and thus each termite colony has its own garden, shaped according to its necessities. In Macrotermitinae, the fungus is essential in establishing a biochemical network that allows the termites to cope with the challenging task of plant biomass decomposition, and it may also play a role in prophylaxis (Otani et al., 2019; Schalk et al., 2021). The result of the interaction between termites and their fungi is the reconstruction of a distinct phenotypic variant, which appears through the process of fungi collection, and termite-fungi interaction during the construction of the “fungus garden.” Each “fungus garden” is unique to each termite mound (variation), yet the very phenotype itself can be identified as a unique phenotype. Therefore, fungi-growing termites and their “fungus gardens” manifest SOT and are a reconstitutor.

Our analysis of the cases of ecological inheritance illustrates the necessity of the generation of distinct phenotypic variants as a key characteristic of the unit of heredity. The confusion regarding ecological inheritance has derived, we contend, from the traditional connection between heredity and the transmission of certain elements or resources. Examples of ecological inheritance convincingly show that several environmental components are transmitted elements. Yet, it does not necessarily follow that SOT is realized at their level. SOT will be realized if and only if these transmitted elements also come with the capacity for phenotypic variations. We showed that most of the cases adduced by niche construction theorists fail to satisfy the latter requirement. For this reason, we exclude these cases from the unit of heredity. By doing so, we do not mean that none of these elements plays an important role in the evolutionary process: indeed, they play important and well-described roles in evolution. But the evolutionary process and the unit of heredity are not the same.

7 Active maintenance: the evolution of interconnected processes

A final case we would like to consider is whether the reconstitutor covers cases (empirical examples) of what Doolittle and collaborators have called “songs” in the so-called “it’s the song not the singer” (ITSNTS) model of evolution by natural selection (Doolittle & Booth, 2017; Doolittle & Inkpen, 2018). First, let us clarify that Doolittle and collaborators have not proposed a characterization of the unit of heredity per se. Yet his model could be used to characterize such a unit, and one may theoretically suspect

that it would be preferable to the SOT version of evolution we rely on. But this section will show that this is not correct, as Doolittle's model cannot account for certain hereditary relations that are at the core of SOT and thus are the basis for the reconstitutor. Second, his model counts as hereditary examples that we consider not to be genuine manifestations of the process of descent.

ITSNTS was initially envisioned to account for the historical persistence of certain processes that are susceptible of accumulating design of the type usually observed when natural selection acts on a biological lineage but whose instantiation does not occur at the lineage level. That is, ITSNTS was conceived to cover cases where several lineages come up together to re-produce the same processes.⁸ Doolittle refers to these processes as *songs*, which he characterizes as patterns of interactions that act as fitness bearers under certain conditions, and to the lineage-forming organisms that join and interact to reproduce these songs as *singers*. In this account, songs would result from the network interactions between lineages, which suggests how the relationship between singers and songs works: "Because there's a song, there are singers: because there are singers, there's a song" (Doolittle & Booth, 2017, p. 21).

Empirically, Doolittle envisions his ITSNTS account to explain the existence of apparent design in holobionts, where certain metabolic and other physiological properties seem to emerge transgenerationally from the interaction between the host and several bacterial species, despite the lack of joint reproduction. Yet Doolittle extends his account to other processes such as the nitrogen cycle or other geochemical cycles sustained on Earth for thousands of years because several organisms from different lineages re-produce them through their ecological interactions.

We are sympathetic to Doolittle's account, and we think his view of evolution has similar consequences to those that follow from our SOT proposal. First, we also suggest a functional conception of evolution, whereby similarity of variation in certain traits (Doolittle's songs) is ontologically prior to establishing structures that cause this perceived similarity (Doolittle's singers). Epistemologically, this entails that one must first investigate the existence of processes leading to a clear pattern of similarity and later investigate the source(s) of this similarity. Second, we agree with him that the unit of heredity should extend beyond lineage-forming entities (Doolittle's singers) and encompass cases that transcend the lineage (Doolittle's songs). Finally, we share Doolittle's intuition that it makes little sense to consider that certain patterns of interaction *reproduce*, thus ultimately rejecting the reproducer/Darwinian individuality model of the unit of heredity, while it still seems plausible to assert that they do something quite analogous. Doolittle and his collaborators choose the term *reproduce*, but we prefer to use the term *reconstitute* as we think it captures better the type of relationships that ultimately occur, and especially stressing the role of the set of relationships in bringing about the reconstitutor. To put it differently, Doolittle's

⁸ Note that in Doolittle's terminology, *re-produce* differs from *reproduce* because the latter occurs when there are clearly identifiable lineages carrying out the process (e.g., when there are Darwinian individuals), whereas the former results from the convergence of independent lineages, but it is not itself a lineage (i.e., a Darwinian individual). In a sense, Doolittle shares our intuition that some cases of heredity are not cases of reproduction, as the concept has traditionally been used. We, however, disagree with him about the type of cases, the definition of the process, and the most adequate name for the unit of heredity.

songs are analogous to the phenotypic traits under the reconstitutor account, but the reconstitutor itself focuses on the relationship making the latter arise.

But we also have some disagreements because we suspect that if we followed his account instead of the SOT model we have proposed, we would be prone to an inadequate conceptualization of the unit of heredity. Firstly, Doolittle requires that singers, that is, lineage-forming entities, re-produce the songs. Yet we have shown that the transgenerational recreation of small RNA states does not follow this pathway. Small RNA states get formed transgenerationally because certain components are actively assembled; but these components are not in a lineage relation. Thus, they would not be singers in Doolittle's account, which entails that the states they form through their interaction would not be songs. In other words, our account does not only reject the idea that heredity requires an identifiable parent–offspring lineage, but we even go beyond arguing that there is no need for lineages at all. Secondly, as Suárez (2020) has shown, Doolittle's approach cannot account for the role of the holobiont as a unit of heredity, even when the account was envisioned to do so. In Doolittle's account, songs are patterns giving rise to a (phenotypic) state through the interaction between the components. These patterns are metabolic, physiological, or any other. So each of these patterns would be a song in itself. The peculiarity of the holobiont, though, is the centrality of the host as the core element around which different lineages converge to recreate a multiplicity of patterns of interaction at different levels. ITSNTS model, however, lacks the adequate conceptual resources to deal with the host-centrality emphasized by Suárez (2020), later developed by Suárez and Stencel (2020), and which we take as a paradigmatic example of SOT and of how the concept of the unit of heredity should be expanded.

Recently, Baptiste & Papele (2021) have extended ITSNTS into the “it's the song, and the singer” (ITSATS) model, whereby what Doolittle initially characterized as singers (i.e., organisms in lineage relationships) are ultimately songs as well, and thus can also be modeled as networks of interactions between components. ITSATS conception entails a redefinition of singers *as any component of a song*, with the proviso that these components can simultaneously be modeled as songs from a different perspective, which generates a more complete model that can account for some examples that we argued Doolittle's model cannot. In effect, one could argue that RNA states are ITSATS-songs, whose components would be ITSATS-singers; and the same is true for holobionts, as Baptiste and Papale (2021) acknowledge by relating their account to Suárez's. Note that this possibility stands from their rejection that singers need to be lineage-forming entities. They only need to be components, irrespectively of whether they form a continuous line of descent.

But ITSATS account, as well as ITSNTS, entails the consideration of certain processes that we contend do not constitute cases of descent as cases of heredity. Concretely, global cycles such as the carbon and the nitrogen cycle or other biogeochemical cycles have been postulated as paradigmatic examples of ITSNTS/ITSATS. Our main objection is that they lack the type of active maintenance between the elements required to consider it an example of heredity. Recall that active maintenance requires difference-making and selective affinity between the elements, with the latter occurring in a principled way, i.e., not as a side-effect of the interaction between other elements. It is true that the nitrogen and the carbon cycles can be characterized as cases

where some elements are causal difference-makers for the processes to continuously re-appear and to modify over time. For instance, atmospheric nitrogen fixation would not be feasible without Mo-nitrogenase, a causal difference-maker in the process. But there seems to be a lack of selective affinity between the atmospheric nitrogen and the Mo-nitrogenase. The production of Mo-nitrogenase is common in several bacterial lineages, including free-living bacteria such as *Azotobacter* and symbiotic bacteria such as *Rhizobium*. In *Rhizobium*, Mo-nitrogenase is synthesized because it allows establishing a symbiotic relationship with legumes. The existence of a selective affinity between *Rhizobium* and legumes fosters and favors this process, and thus nitrogen fixation is not a trait being inherited on its own, but rather a side-effect of the interaction between *Rhizobium* and legumes and the generation of joint phenotypic variants that are themselves inherited (Sawada et al., 2003). In the case of *Azotobacter*, on the other hand, nitrogen fixation is a side-effect of bacterial metabolism. Bacteria have evolved to produce Mo-nitrogenase in the absence of nitrogen sources, but its production is known to stop if these sources are available (Bürgmann et al., 2003). Note that these two cases reveal the nature of the process: if Mo-nitrogenase and atmospheric nitrogen had a selective affinity with each other, then it would be expected that *Rhizobia* and *Azotobacter* produced it because atmospheric nitrogen is present. But experimental evidence suggests that they produce it when they need it, suggesting that there is no affinity between Mo-nitrogenase and atmospheric nitrogen in the process of nitrogen fixation.

Therefore, it follows from this discussion that some of the cases discussed by Doolittle and collaborators under the ITSNTS account, and Baptiste & Papale under the ITSATS account, would be excluded from our concept of the reconstitutor, as we elaborate it in this paper.

8 Conclusion

The study of evolutionary heredity in the twentieth century was based on the concept of lineage formation and the requirement that a continuous line of ancestry could be detected at a focal level to infer the existence of an evolutionary unit of heredity at that level. This view motivated the introduction of the concepts of replicator, reproducer, and Darwinian individual to conceptualize the evolutionary unit of heredity. However, several hereditary phenomena discovered in recent years have shown the insufficiency of these concepts and the necessity to introduce a new one to fully account for the forms of hereditary phenomena recently discovered. This paper fulfills that role by introducing the reconstitutor as the unit of heredity. We characterized the reconstitutor as the structure resulting from a set of relationships between different elements or processes actively involved in the recreation of a specific phenotypic variant in each generation regardless of the biomolecular basis of the elements or whether they stand in a continuous line of ancestry.

The reconstitutor is built upon the concept of Stability of Traits (SOT), the phenomenon manifested by the transgenerational persistence of variation in the phenotypic characteristics of some individuals in a population at a specific level of the

biological hierarchy, not necessarily forming a continuous line of ancestry. By identifying SOT as the concept behind heredity, we have also been able to diagnose why the replicator, the reproducer, and the Darwinian individual failed to capture certain hereditary phenomena that received increasing attention throughout the second half of the twentieth century. We explained that by appealing to the fallacy of misplaced concreteness (Dupré & Nicholson, 2018): all these concepts privileged the creation of parent–offspring lineages, either via coding or other evolved mechanisms, as the source of heredity. As a result, they limit heredity to those resources that are transmitted via these channels. We referred to this approach as the Stability of Lineages (SOL) view of heredity and argued for its conceptual and empirical inadequacy.

The concept of the reconstitutor, as we have introduced it here, constitutes a valuable tool for those elaborating a process view for thinking contemporary biology (Baptiste & Dupré, 2013; Baptiste & Huneman, 2018; Baptiste & Papale, 2021; Dupré, 2017; Dupré & O'Malley, 2009; Papale et al., 2020), insofar as it provides a platform for elaborating process thinking. This is because process ontology, without downgrading the importance of entities, emphasizes how the same dynamics can be instantiated in two different systems, even though the entities and activities causing their instantiation do not resemble each other, and the dynamics may have been thus reached by different means. The reconstitutor serves to operationalize the process view of the living world via its requirements of “distinct phenotypic variants” and “active maintenance.”

Introducing the reconstitutor as a conceptual tool has several practical consequences. For instance, small RNA inheritance research frequently concentrates on demonstrating the transgenerational reappearance of one specific RNA molecule that has been up or downregulated in response to a stimulus in past regenerations (e.g., Gapp et al., 2014) (Sect. 5.1); and hereditary research on holobionts concentrates on finding the channels of vertical transmission of the microbial components of a host's microbiome (e.g., Hester et al., 2016) (Sect. 5.2). In contrast to these practices, the reconstitutor concept encourages an entirely new type of research in both fields. Rather than looking for the reappearance of an entity or the discovery of a transmission mechanism, the focus should be on the active maintenance of the phenotypic effects. Are these transgenerationally kept? With what frequency? Which types of processes guarantee the persistence of phenotypic effects? Several explanatory avenues can be opened: the phenotype may reappear because a functionally equivalent small RNA state can be reconstituted in the next generation (Veigl, 2017). Or, in the holobiont case, it may reappear because the traits are hologenomic adaptations, and thus they are motile, engaged in abundant horizontal gene transfer within the microbiome, and functionally redundant (Suárez & Triviño, 2020; Suárez, 2020). Presupposing SOL instead of SOT limits the possible research avenues and phenomena amenable for scientific investigation. In contrast, the reconstitutor concept opens them to discovering new mechanisms and processes, new structures, or new forms of maintaining hereditary relations.

Overall, the reconstitutor stands as a unit of heredity that is neither as restrictive as the replicator or the reproducer nor as inclusive as some forms of ecological inheritance or inheritance beyond the organismic level. In this sense, it avoids the confusion of the concept of heredity, which is itself processual and inclusive, with the inheritance mechanisms that cause heredity, which are restricted to specific cases of hereditary

transmission. At the same time, it avoids conflating heredity with mere persistence, as it happens with some extended accounts of heredity. The reconstitutor should thus be conceived as a middle-ground concept to reconcile restricted accounts of inheritance with expanded accounts while keeping the darwinian spirit of looking for descent intact.

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Declarations

Conflict of interest The authors declare there is no conflict of interest.

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