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ORIGINAL PAPER

Evolution in biological and nonbiological systems under different mechanisms of generation and inheritance

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Abstract The majority of definitions of life and evolution include the notion that part of an organism has to be copied to its offspring and that this includes some form of coded information. This article presents the thesis that this conception is too restrictive and that evolution can occur in systems in which there is no copy of information between generations. For that purpose, this article introduces a new set of concepts and a theoretical framework that is designed to be equally applicable to the study of the evolution of biological and nonbiological systems. In contrast to some theoretical approaches in evolution, like neo-Darwinism, the approach presented here is not focused on the transmission and change of hereditary information that can be copied (like in the case of DNA). Instead, multiple mechanisms by which a system can generate offspring (with and without copying) and by which information in it affects the structure and evolution of its offspring are considered. The first part of this article describes in detail these new concepts. The second part of this article discusses how these concepts are directly applicable to the diversity of systems that can evolve. The third part introduces hypotheses concerning (1) how different mechanisms of generation and inheritance can arise from each other during evolution, and (2) how the existence of several inheritance mechanisms in an organism can affect its evolution.

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Introduction

In living organisms, a part of an individual, the genotype, is copied into its offspring. This genotype interacts with other cellular components inherited from the parent (or parents) and the environment to lead, over time, to the production of an adult that is able, in its turn, to produce other offspring. Evolution is normally regarded as the change in the structure of organisms over generations. This change is due, ultimately, to the accumulation of DNA changes in the genotypes that are copied between generations (although not exclusively; Oyama 2000; Jablonka and Lamb 2005). In that respect, the majority of definitions of life and evolution (Palyi et al. 2002; Cleland and Chyba 2002; Ruiz-Mirazo et al. 2004) incorporate the notion that part of an organism has to be copied to its offspring and that this includes coded information (Schrödinger 1944; Morowitz 1992; Santos et al. 2003). Here, I present the thesis that this conception is too restrictive and that evolution, under some circumstances, can also occur in several kinds of systems in which there is no copy of information between generations. This article also tries to identify the minimal requirements for a system to be able to evolve. For that purpose, this article introduces a new set of concepts and a theoretical framework that is designed to be equally applicable to the study of the evolution of biological and nonbiological systems. This framework thus has the dual motivation of helping in understanding the evolutionary process across systems and in some nonbiological systems by using an evolutionary perspective.

In contrast to some theoretical approaches in evolution, like neo-Darwinism, the approach presented here is not

focused on the transmission and change of hereditary information that can be copied (as in the case of DNA). Instead, multiple mechanisms by which a system can generate offspring (with and without copying) and by which information in it affects the structure of its offspring are considered. This article focuses on the effect that these different mechanisms can have on the forms and rates of evolution of different kinds of evolutionary systems.

This article only introduces a new nomenclature and perspective. It does not present any new results or tests, but helps in reinterpreting evidence in biological and nonbiological systems from a unified perspective. In doing so, it proposes new evolutionary hypotheses. These, however, are not tested. Instead, only some circumstantial evidence is presented to support them.

The first part of this article explains in detail some new concepts. It introduces specific definitions of some commonly used concepts such as evolution, evolutionary system, nonbiological and biological system. Some examples are discussed in this part but only to clarify concepts that may otherwise seem rather abstract. The second part of the article discusses how these concepts are directly applicable to the diversity of systems that are able to evolve. An extensive review of these systems is outside the scope of this article. However, examples will be presented only to clarify the utility of the new concepts. In the third part of the article, the concepts presented earlier are used to elaborate a set of hypotheses about how different mechanisms of generation and inheritance, including the ones found in living beings and others, differentially affect evolution. These hypotheses are concerning (1) how different mechanisms of generation and inheritance can arise from each other during evolution and (2) how the existence of several inheritance mechanisms in an organism affects its evolution.

While the second part tries to explain the applicability of the presented concepts to many different biological and nonbiological systems, the third part focuses only on those systems and conditions in which long-lasting and complex evolution is possible.

Definition of generative system

Generative systems are arbitrarily defined systems that fulfill the following requirements:

1. A *generative system* (the parent system) is able to generate (alone or with other generative systems), in a given environment, another generative system (the offspring system). For the purposes of this article, this means that some information existing in a parent generative system (called the *kernel information*) is, at least, causally responsible for the information in the offspring system that allows it to generate other generative systems (*kernel heredity information*). Parent and offspring are simply defined by the fact of generating (or being generated) by other generative systems.
2. A generative system is physically independent of the parent system(s). Physically independent means here that changes occurring in the parent system do not necessarily have an effect on the offspring system. This definition allows for a clear distinction between generation, development and evolution. Generation is the process by which a generative systems ends up producing an independent offspring system. This involves recruitment of material elements from the environment (or from the parent itself) and their organization in specific spatio-temporal patterns (the offspring system) through interactions with the organization of the parent(s) and the environment. The changes (if any) occurring in the offspring system from the moment it becomes physically independent to the moment in which it is able to produce other offspring systems are called, in here, the development of the offspring system. Generative systems can thus produce a sequence of successive offspring, offspring of offspring and so on. A *lineage* is the set of ancestors of a given generative system. An *offspring lineage* is the set of all the offspring and successive offspring of offspring of a generative system. Lineages do not need to be lineal sequences, since a generative system can have several parents and multiple offspring. The use of information and causality requires some clarification, which is presented in the following subsections.

Information

Information is used here as a specific pattern or configuration of arbitrarily defined relationships between arbitrarily defined elements, for example, spatial patterns (as distributions or configurations) of neighborhood between cell types in an animal, a specific pattern of friendship relationships between individuals in a club or a specific sequence of nucleotide bases in a RNA molecule. Thus, for example, two different spatial patterns of cell types are said to have different information. Given a common definition of states and their relationships, the amount of information in two or more generative systems can be quantitatively compared by using relative statistical measures as for example joint information or mutual information (Shannon 1948). In essence, thus, information is similar to organization. However, the concept of organization can be a bit misleading, since it may presuppose

(depending on the user) that some specific patterns are more organized than others on the bases of some criteria. This is not the case for information as defined in here nor is the purpose of this article to identify the real essence of organization or which systems are more organized. How information is measured and compared is not of vital importance for the ideas presented in this article but an example will be given for clarity.

The joint probability of two cell types [P(A,B)] can be calculated as the number of times a cell of a given type (for example, type A) is next to a cell of another specific type (for example, type B) divided by the total number of times a cell is next to another in a given spatial distribution of cells. The mutual information of a spatial distribution of cell types is then the summation of each joint probability multiplied by its logarithm. This mutual information is low, for example, in spatial cell distributions with two cell types in which each cell tends to be next to cells of the same type, intermediate when cell types are intermixed and high when each cell has exactly half of its neighbors of each cell type. By including neighbors at larger distances, higher scale features of a spatial distribution can be included in the calculations (Salazar-Ciudad et al. 2000).

Information here relates to the patterns of configuration of parts in a whole (for example, which atoms bind to which in a molecule) without consideration on the nature of these parts (although, at a lower level, the nature of these can come from the organization of its parts). This definition does not consider coding, as for example in DNA, because as it will be proposed, coding is an advanced outcome of evolution and not something required for it. In that sense, other more complex definitions of information (Jablonka 2002) are comparable to concepts like causality (see below) but not to information as used here.

It is important to stress the fact that the concept of information used here is unrelated to the common metaphor that DNA bears the information for the production of the phenotype or that is one among many of the resources required in development (Oyama 2000). It is not meant to replace the latter; it simply refers to a different thing.

DNA is routinely said to carry "information" that is read to produce the phenotype. In that sense, information has a meaning that depends on how this information is read. DNA conveys information about the phenotype in a manner that depends on the transcriptional machinery, translational machinery and, in general, in the cell's dynamic structure and developmental mechanisms. These are different in different organisms and their functioning is not always well understood. This makes this common conception of "information" very difficult to define and measure, because what information is depends on the mechanisms of generation and development. This lack of comparability is even more manifest when generative systems without DNA and copying are considered. The concept of information introduced here is not dependent on developmental mechanisms and cell structure nor does it imply any reading or meaning (and thus refers to something other than DNA-coded information). In other words, a different kind of genetic code (or different kinds of developmental mechanisms leading to a phenotype) would change what in the genotype is information in a meaning/reading definition of information but not in the concept of information used in here. As will be seen, it is equally applicable to any generative system and easily measurable and comparable (by joint information measures for example). This separation between information and the mechanisms of generation and development should facilitate the study of their relationship while avoiding possibly misleading analogies with living beings' genotypes and development. Thus, the processes of generation and development can be physically described as the transformation of information between a parent and offspring generative systems or within a generative system.

This article loosely refers to complex phenotypes as phenotypes with a large diversity of different elements and relationships between them or to some related measures (e.g., mutual information; Shannon 1948).

An organismal phenotype, for example as its cell types and their spatial relationships (which cell is next to which) or their molecules and their spatial relationships, can be described as information. There is nothing specially deep or mysterious about it. This is simply to state that a phenotype can be described as the spatial arrangement (or relational arrangement) of elements (for example, cells). It is important to note, however, that the existence of a phenotype of a generative system does not imply in general, the existence of a genotype, and in any case, the genotype of a living organism is part of its phenotype (its "DNA phenotype"). Thus, unless it is explicitly stated, statements about the phenotype of a generative system also apply to its genotype (in the case the generative system has one).

Heredity or hereditary information is all the information in the offspring system that is caused by information in the parent system(s) (this later information being called *parental hereditary information*). Note that the heredity information in an offspring system is not necessarily the same information as its parental hereditary information (that is, when that offspring system acts as a parent of another generative system). One clearly leads to the other, but other influences (for example, from other parents, from the environment or from informational transformations inside a generative system) can also affect the parental hereditary information and can even cause this information to be different at different times during the existence of a generative system.

An *inheritance mechanism* is defined here as any set of physical interactions (and their spatiotemporal organization) by which a parent can change or generate information in the offspring. The process of generation implies the existence of an inheritance mechanism, but some inheritance mechanisms may not be associated with the process of generation itself. For example, human parents can affect their offspring's information by teaching them some ideas (or modifying their bodies), but these ideas may not be required for the offspring to be born (although some of them may be required for ultimate offspring's survival in specific environments). *Reproduction* is understood in this article as the generation of offspring systems that resemble the parent. Then, as will be described, all reproduction is generation but not all generation is reproduction.

Causality

"Causally responsible" means in this article that the kernel information is strictly required for the offspring system to be produced and that variation in it would lead to variation in offspring's kernel heredity. Note that this definition of causality requires the existence or possibility of variation. This causality does not imply sufficiency or exclusivity; in other words, several parents may be required for the production of an offspring system (the kernel heredity information then being caused by several different kernel informations). In that respect, there are three main types of generative systems: *Informational parasites* are generative systems that require some external information in the environment to be able to generate the offspring system. This external information can come from another generative system or not. A clear example of this is viruses. To produce other viruses, a virus requires the transcriptional, translational and replicative machinery of a host cell. This machinery not only provides the energy necessary for the production of new viruses but the structural information required for such a process to be possible (this is the spatiotemporal arrangement of the molecules involved in the transcriptional, translational and replicative machinery). Note, however, that even if the host is strictly required for the production of offspring viruses, its variation does not normally produce variation in the offspring viruses and thus cannot be considered as a parent system. *Energetic parasites* are generative systems that require some energy input from some other generative systems to produce the offspring systems. *Autonomous generative systems* are generative systems that are not parasites. From an information point of view that would imply that no external molecules would be incorporated into a generative system or that their atomic configuration (its information) per se would not play any role in the generative system. This is probably not found in extant living beings. In addition, *nested generative systems* are generative systems that are part of another generative system and are informational parasites of it (some examples are presented below).

Since the process of generation involves information changes (even in the case of copying, elements from the environment have to be taken and organized to form the copy itself), it follows that generation involves changes in energy. If the informational changes involved in the generation process do not increase universe's entropy, then generation requires an input of energy from the environment (Prigogine and Nicolis 1977). In that case, generative systems are open dissipative systems that require energetic inputs. Material inputs can also occur. These material inputs can be information in itself that can be incorporated into the generative system. Some of these molecules may be required for development and later reproduction even if they may not be part of heredity. Humans, for example, as many other animals, are unable to synthesize many essential vitamins. These are often produced by bacteria and acquired through foods. These vitamins do not provide energy as such but some molecular organization that is required for body growth and, eventually, reproduction [for example, vitamin C is required for collagen hydroxylation, a process essential for the formation of tissues such as cartilage and blood vessels (Peterkofsky 1991)]. In that sense, most or all living beings can be considered informational parasites.

For the offspring system to be a generative system, it is required that the parents' kernel information required to generate it is also causative of the generation of the offspring's offspring. This implies that the kernel heredity in the offspring's offspring is indirectly caused by its grandparents' kernel information and successive ancestors. Thus, generative systems imply a transgenerational chain of causation. Note that only causality is required. It is not required that any information is passed or transmitted from generation to generation, but that, simply, information in one generation leads, causally, to information in later generations. Later sections will describe how this can be achieved without copying of parental information into the offspring system.

Evolutionary systems

The concept of generative system allows introducing what is meant in this article by evolution. *Evolution* is a change in the information (that is, the phenotype) of the generative systems in a lineage over generations. Individual generative systems can not be considered to evolve, but to develop. The definition of generative system implies the existence of a lineage and, in a sense, this definition of evolution is equivalent to change in the structure of individuals through a lineage.

It is often the case in living beings that parents and offspring are far from being identical. In obligate sexual animals, each gamete receives only a subset of the parent's genotype. This subset arises by the recombination of the genotypes inherited from the grandfather and grandmother. Each individual thus only shares around 50% of its genes with each of its parents. The pairing of chromosomes during meiosis and mitosis makes that normally each offspring receives a copy of every gene from each parent. However, this may not always be the case, because occasionally, nonhomologous recombination occurs in parts of the genotype. Genetic similarity of 50% does not imply, however, 50% phenotypic similarity, because maternal and paternal gene products often interact to repress each other or to produce an outcome that is not necessarily the average of the parents (dominance). Even, without dominance, the intricate genetic and epigenetic interactions among genes that are required for development ensure that 50% genetic similitude does not ensure 50% phenotypic similitude. Environment can also affect the phenotype in such a way that even in asexual organisms the phenotypes of offspring and parents are substantially different (reviewed in West-Eberhard 2003). In fact, experimental measurements in quantitative genetics show that, in general, the heritability of phenotypic traits can be very variable (Carlborg and Haley 2004). In other words, even if the offspring get their genomes by copying part of their parents' genomes, they are not always very similar to them. However, even when heritabilities are very low, evolution is still possible (although it is slower and less effective).

In living beings, where evolution is well studied and accepted as a phenomenon, DNA copying occurs between successive generations in a linage. In that case, evolution is still understood as change in the phenotype of individuals (which in the usage here also includes the genotype) over generations. Thus, although there is copying, what counts as evolution (or which aspects of the phenotype are regarded as evolving) is what changes between generations (so what is different from ancestors and what is not common). This is why evolution is here defined as change in lineages irrespective of the mechanisms by which lineages are generated. Thus, evolution does not require copying, but the existence of a lineage (thus causality between generations and the possibility of change in it over generations). Notice that this implies that in lineages in which parents and offspring do not resemble each other evolution may still be considered to occur. In that respect, this definition of evolution is general, since it pertains both to evolution in living beings and evolution occurring in other kinds of generative systems.

An evolutionary system is a generative system that is in an environment where its offspring evolves over time. Thus, the classification of a system as evolutionary is dependent on the environment. The same is true about generative system: a system's ability to generate further generative systems depends on the environment (for example, if the essential energetic and informational resources are available). Therefore, the classification of a system as generative or evolutionary should be regarded as a hypothesis about a system's offspring lineage behavior in the future (in a given environment). In that sense, a system is evolutionary in relationship to the expected behavior of the offspring lineages it produces. The term "biological system" as used in this article includes all the kinds of living beings known to science (monera, protista, animalia, plants, fungi) and also viruses and viroids.

Evolutionary systems and the environment

The environment of a generative system is defined here as anything that is not the generative system itself. What is, materially, part of a generative system and what is not is arbitrary except for the fact that a generative system should include the kernel heredity information (otherwise, it cannot be defined as a generative system). The evolution of a generative system can be affected by the environment in two major ways: *mutational environmental effects* (or mutations) are environmentally induced changes in the parental hereditary information or kernel information of a generative system, while *nonmutational environmental effects* are environmentally induced changes that do not affect the parental information of a generative system. In living beings, mutations can be changes in the genotype that have an effect on later development. In generative systems, without a genotype, as it will be described, the mutation itself is a phenotypic effect but one that leads to different phenotypic effects later in development.

The phenotypic effects of a mutation are defined as the phenotypic consequences of this change in the subsequent development of a generative system. Mutations clearly affect a lineage's evolution by changing the information that is causal between generations. Nonmutational effects can also affect evolution by affecting the probabilities by which a generative system will give rise to offspring systems. Nonmutational effects can be, for example, the relative abundance of some informational resource or energetic resource or any other factor (like predators or destructive accidents) that affects the probability of generation. Thus, nonmutational effects can lead to natural selection. In living beings, but not necessarily in other kinds of generative systems, what is normally considered as somatic mutations and environmental effects on development can also be considered as nonmutational environmental effects.

The range of mutational effects possible in an environment has a strong influence in the evolution of a lineage.

This range depends on the environment and on the structure of generative systems (in fact in the interaction between these). The *total variational properties* of a generative system are the set of different offspring systems that would arise from all possible mutations in a given environment. The *variational properties* of a generative system are a subset of the total variational properties that includes only the mutations that occur more often in a given environment. Depending on the generative system and the environment the total variational properties are infinite or not. The variational properties are a finite set, since they include only the most common mutations.

The evolution of a generative system and its total variational properties can be finite and predictable. This does not seem to be the case for living beings, but it is likely to be the case for some simple generative systems placed in simple environments. In that respect, this article is not only concerned with evolutionary histories that started in the past and are still going on (as in living beings) but also considers hypothetical or real started-and-ended evolutionary histories and evolutionary histories that start and end many times and very fast. Examples of this will be provided in the next section, but in general three different types of evolutionary histories for a lineage can be described: *monotonous evolutionary histories*, the repertory of changes occurring in a lineage of generative systems during evolution is finite and predictable; *recombinant evolutionary histories*, the repertory of changes in an evolving lineage is not finite but it can be understood as the combination of a finite number of basic changes; *open evolutionary histories*, when none of the previous applies. This categorization is similar to that of limited and unlimited heredity (Maynard Smith and Szathmáry 1995) except that it includes aspects of the environment and variational properties.

The concept of generative systems is vaguely reminiscent of that of "reproducers" (Griesemer 2000a). A reproducer is a unit of multiplication, hereditary variation and development in which a parent generates an offspring that is able to develop so as to generate its own offspring (as here). However, in contrast to the treatment here, what is meant by heredity is not defined by Griesemser and he does not consider informational transformation between generations. Reproducers are not defined on the bases of physical independence, kernel information and causality as in the case of generative systems. Material overlap is not required for generation in generative systems, but it is required in reproducers (although this may not be required for nonbiological reproducers; Griesemer 2000b). This allows applying the concept of generative systems to nonbiological systems as will be discussed below. In addition, it has been suggested that replicators (units of copying and variation as in DNA) are evolutionary ancestors of reproducers (Szathmáry 2006), while here it will be argued that generative systems are evolutionary ancestors of replicators (but some types of generative systems are descendants of replicators).

The next section discusses some examples of generative systems to clarify the concepts presented up to this point. Additional concepts will be introduced in the next section. These should be easier to understand after the discussion of concrete examples.

Examples

Five kinds of examples will be discussed in this article: living organisms, parts of living organisms, and psychological, computer-based and chemical systems. Many of these examples are not very well understood, many are not usually considered from an evolutionary framework, and others have not previously been described but are used for the purpose of clarifying some concepts.

Chemical generative systems and closed mutations

Some chemical systems could provide the simpler examples of generative systems. A system of reactions in a chemostat, where reactants are steadily supplied and some products removed, can in some circumstances be considered to comprise generative systems. This can occur in environments with readily available natural compartments (such as small cavities in rocks precluding the dilution of reactants) or by the chemical system producing its own compartment boundaries as a side reaction. An example could be a reaction in which a molecule A reacts with an externally supplied reactant R_a to produce a molecule of B and P_b (a product that is removed) and B reacts with R_b to produce a molecule of A. The molecules A and B can be considered as generative systems if there is at least one possible mutation (for example, due to some lateral reaction or some external radiation) that transforms A into another molecule A′ and this molecule A′ reacts (with R_a) to produce a molecule B′ (and P_b) that reacts (with R_b) to form a molecule A′. This is because the change in A (to A′) is responsible for the change in B (to B′) and this change also affects the information in B that is required to make its offspring generative system A (which then changes to A′). In other words, the change in every molecule (for example, from A to A′) is, from the definition above, a change in its information (typically the nature and spatial arrangement of atoms). Thus, the informational change in a generative system A (to A′) is responsible for the variation in information in system B (to B′). This change in B clearly affects the information in B that is required to produce its offspring system, because B′ produces A′ instead of A or

nothing. In that sense, there is a transgenerational causal chain.

I refer to this kind of mutation a *closed mutation*. An *open mutation* is a mutation in which, for example, A′ will lead to B′ but B′ leads to A (instead of A′). Open mutations, thus, have no effects on future generations, while closed mutations have. From the definition of generative system, it follows that any generative system should be able to sustain at least one closed mutation in its kernel information (otherwise, there is no causality between the information in a system and the information in its offspring).

Another category of mutation that can help to understand the nature of generative systems is a mutation in which A′ leads to B′ and B′ leads to A″ and A″ to B″ and successively A^n leads to B^n and B^n to A^{n+1}. This is also a closed mutation. In fact, there is nothing in the definition of generative system that forces a member of a generative system lineage to resemble any of its ancestors. In this example, the causal link between information in generations is not broken at any point. In fact, a lineage of systems in which A^1 leads to A^2 and A^2 leads to A^3, that leads to A^4 and successively A^n leads to A^{n+1} is a lineage of generative systems, insofar as there is, in a given environment, some possible closed mutation (leading, for example, to a lineage in which $A^{n'}$ leads to $A^{n'+1}$).

To my knowledge, no chemical system has been reported to be a generative system. Some artificial self-catalytic chemical systems have been studied in relationship to the origins of life (von Kiedrowski 1986; Terfort and von Kiedrowski 1992; Sievers and von Kiedrowski 1994; Bohler et al. 1995; Pitsch et al. 1995; Wintner and Rebek 1996; Lee et al. 1997). These exhibit generation, in the form of autocatalysis, but they do not seem to have the capacity to accumulate changes that lead to changes in the offspring (closed mutations). Thus, these systems cannot be considered capable of evolving (nor are they generative systems).

Although no artificial or natural chemical generative systems have been reported, some studies provide indirect evidence of their existence. In a recent study (Ashkenasy et al. 2004), it is shown that a polypeptidic molecule (T1) can be catalyzed from two other polypeptides (N and E1) by another T1 molecule. A T1 molecule is made of an N and an E1 molecule bound by a peptidic bond. In T1 catalysis, the N part of T1 interacts with an N molecule, while the E1 part interacts with an E1 molecule, positioning N and E1 in a favorable spatial arrangement for the formation of a peptidic bond (thus a new T1 molecule is catalyzed). This kind of autocatalytic reaction is also possible in nine other polypeptides (T2–T8) from *N* and nine precursors (E2–E9). All the E polypeptides are very similar: they differ only in one or few amino acids. Each T molecule can catalyze the synthesis (from N and a specific E) of several other kinds of T molecules (but not all of them). This pattern of cross-catalysis is rather complex (Ashkenasy et al. 2004), but it allows for the identification of generative systems. For example, in a chemostat containing only molecules of T2, N, E2 and E5, only the T2 molecules reproduce. However, if one mutation can transform T2 into T5, then T5 molecules would also be produced. In that sense, a T2 molecule is a generative system because it can generate other generative systems (other T2 molecules) and also sustain closed mutations (e.g., when T2 becomes T5, T5 can generate T5 offspring systems and not T2 systems) that change the kernel heredity of the offspring (T2 is changed to T5 and then can catalyze the production of T5), producing a different generative system. Both T2 and T5 can catalyze the formation of other T molecules if the appropriate E molecules are present. Thus, T molecules do not copy themselves (see definition of copying below) but simply remake themselves. In that sense, these generative systems are generative systems without copying. These kinds of systems may not be particularly interesting in a theoretical sense, because they are likely to lead to monotonous evolutionary histories.

In practice, it is likely that many generative systems without copying are possible. The critical question, then, may not be whether generative systems exist but which of them, and under which conditions, can produce many different kinds of closed mutations (and potentially lead to nonmonotonous evolutionary histories), and how they relate to living organisms.

Biological generative systems

Most living organisms fall into the category of generative systems introduced here. Exceptions include sterile organisms (like, for example, hybrids between different biological species). This distinction is similar to the one introduced by Gánti between life and evolution units (Gánti 2003) except that generative systems are not the same as evolution units.

Nested biological generative systems

Some parts of living organisms can be considered as nested generative systems. For example, cells extracted from animals and kept in cell culture (Rubin 1992) can be considered as generative systems in the limited environment of the culture itself. Each cell gives rise to daughter cells that are in turn able to generate other daughter cells. In addition, these cells are able to evolve in the sense that genetic mutations accumulate over generations (Rubin 1992). This does not imply that these changes are adaptive, although it is expected that in conditions of finite resources and space some kind of natural selection is likely to occur.

A more natural example at the cellular level is cancer. In cancer, a cell, or group of cells, starts to proliferate independently of extracellular regulatory signals. These deregulated cells can have an increased mutation rate (Chow and Rubin 2000). Mutants that increase proliferation rate and independence from the body's signals tend to increase in frequency. Mutants that can penetrate the body's mechanical and chemical barriers so as to spread (producing metastasis) have access to more resources and can increase their offspring and relative frequencies. If the body is taken as the environment, each cancer cell can be considered as an evolutionary system for which there are clear selective pressures. This evolution has a predictable end with the death of the host body, but this certainty does not affect the classification of cancer as an evolutionary process (simply each cancer has an evolutionary history with start- and end-points).

A similar example can be found in the evolution of genes, plasmids, transposons and DNA sequences in general. If the environment is taken to be the genome of a living organism and all its offspring, then any gene, transposon, plasmid or (in general) any DNA sequence that can be copied by cell's machinery can be considered to be a nested generative system. They are causative for their offspring sequences and the changes thereof. As in the case of cancer, these sequences can evolve under clear selective pressures (increase in its frequency in host's lineage) that are not always compatible with the selective pressures acting on the host. A good example of this is killer plasmids: plasmids that produce a long-lived toxin and a short-lived antidote for it. Daughter cells that do not receive a copy of the killer plasmid are killed by the long-lived toxin (inherited from the partitioning of the mother cell's cytoplasm) (Gunge 1986). This ensures the spread of the plasmid in populations, but is detrimental for the host's offspring lineages. The abundance of transposon sequences in many genomes (especially animal) also suggests the evolution of some gene sequences as evolutionary nested generative systems (Kidwell and Lisch 2001). In essence, these so called selfish sequences (Doolittle and Sapienza 1980; Hurst and Werren 2001) can be regarded as formally equivalent to viruses in which transmission is only possible to the host's offspring (behaving like vertically transmitted viruses in contrast to the normal "horizontal" ones).

Computer-based generative systems

There are several artificial life algorithms that implement generative systems. Many of them are artificial life programs, like for example tierra (Ray 1991) or avida (Adami and Brown 1994), that embody artificial organisms as a sequence of computer instructions (Bedau et al. 2000). These are copied, over generations, into the computer memory and mutated. These generative systems compete for memory space and CPU time (or some program-specific analog) and, by doing so, lead to a process of evolution by natural selection. To my knowledge, all artificial life studies use copying.

An imaginary example of a computer-based generative system without copying can be introduced to clarify the concept: an intelligent robot that is able to construct other robots that are also intelligent and able to construct other robots. These robots could learn from the environment and produce in each generation offspring that do not resemble their parents and that have different methods and motivations to construct offspring. Since robots' characteristics and generative capacities are due to the characteristics and generative capacities of ancestors (but are not a copy, rather a design based on life experience), these robots would be clear examples of generative systems without copying.

Computer viruses and worms are pieces of computer code that replicate as informational parasites in an environment comprising the world's computers. This does not imply that viruses and worms are generative systems. For that, it is required that they can sustain closed mutations. The high reliability of information copying in computers seems to preclude this possibility but, apparently, no systematic studies have been performed on this question.

Psychological generative systems

It has previously been proposed that ideas that tend to be transmitted between individuals can be understood as evolving selfish entities (Semon 1921; Dawkins 1976). This is assumed to happen, by some authors (Dawkins 1976), by imitation and then the copying of one idea in one host to the same idea in another host. How to define ideas and what are their mechanisms of generation are rather complex and controversial issues for which no important conclusions are going to be provided in this article. For the purposes of this article, an idea is any neurally encoded information that can be remembered. Here, as in some other works (Sperber 1996; Jablonka and Lamb 2005), it is considered that copying is not necessary for the evolution of ideas (as it is suggested for the case of memes; Dawkins 1976). Any idea that is communicated in some way can evolve insofar as its lineage can have closed mutations. Thus, some ideas could be generative systems with copying, some without copying and some are not generative systems.

In communication, a set of physical changes in the environment (which can be speech, chemicals, body gestures and writings, among others) produced by the emitter host are received and interpreted by the receiving host. In this process, a new idea arises in the receiving host. This

idea is caused by the idea in the emitter host, but may not be identical or similar to it. It is a common experience, in fact, that there is substantial variability in the way an emitted idea is understood by the receiver host. This is due not only to possible noise in the environment but also to the relative capacities and previous knowledge of the emitter and the receiver. The neurobiological bases of ideas are not currently understood. However, offspring ideas are not necessarily copied and then modified by other ideas in the host. The generation of an idea in the host is influenced (and likely also caused) by other resident ideas. In that respect, whether ideas use copying or some kind of recombination is an open question that may have multiple answers. This, in fact, does not preclude the evolution of ideas but, on the contrary, may enhance it (see below).

Mechanisms of generation

In this article, *mechanisms of generation* refer to the physical interactions (and their spatiotemporal organization) that are required in the parent generative systems to give rise to offspring generative systems in a given environment. Even in living organisms these processes are not fully understood. This article does not attempt to provide an exhaustive list of all possible mechanisms of generation, but a simple description of some of them. However, even at this coarse level, the differences between these mechanisms are large enough to allow tentative inferences about how they may affect the evolution of different generative systems.

Copying

Up to this point, I have made many references to *copying* without providing a precise definition (although DNA replication has been taken as the paradigmatic example). In copying, the parent generative system recruits elements from the environment (for example, nucleotide bases or their chemical precursors) and cause them to interact with part of its structure (for example, a template DNA strand and the replication machinery) to produce a copy of that part (for example, the DNA itself). A perfect copy of something is defined as something that is identical to it in all respects (a nonperfect copy being identical only in some aspects). Copying, as defined here, requires perfect, or nearly perfect, copies and physical interaction between templates and the forming copies. In living organisms, copying happens by the replication machinery acting on complementary DNA strands. In computer viruses, a template is also used. Information (the virus), as patterns of 0s and 1s, is stored as ordered spatial patterns of magnetization states (0 and 1) in a hard drive or some other storage media. Since different magnetization states allow or preclude the passage of current, a stored pattern of "on" or "off" current tracks (the 0s and 1s) can be recovered by passing a current over the magnetized media. In this way, information (as for example the virus program as 0s and 1s) is copied without the use of complementary interactions but by making environmental resources (current, free memory and CPU time) to interact with the structure of the parent (the template being the magnetization pattern that describes the parent system in the memory).

The concept of copying allows a precise definition of the genotype (for the purposes of this article) as the part of the phenotype that is copied. This definition does not make assumptions about the mechanisms by which this phenotype may affect the rest of an offspring phenotype.

The proportion of the phenotype that is copied by a copying mechanism can be different between types of generative systems. In computer viruses and in biological viroids (viruses consisting of a naked RNA molecule), for example, the whole phenotype is genotype. In most living organisms, the genotype is only a small proportion of the phenotype. Although the genotype may interact with the epigenetic structure of the zygote to regulate many aspects of the rest of the phenotype, in no case does the copying of the genotype imply that identical phenotypes arise (except for viroids and computer viruses).

The range of structures that can be copied by a copying mechanism can also be different. In computers, only 0s and 1s can be copied, while in living organisms four configurations can be copied (A, T, G and C). Ideally, however, the maximal rates of evolution by copying would be attainable if any simple aspect of the phenotype could be copied. In general, copying more complex phenotypes (for example, if protein three-dimensional structures, cells or cell spatial arrangements could be copied directly) can be expected to require more complex copying mechanisms. Note that even if tertiary RNA and DNA structure form spontaneously, these are not caused by the copying process itself. In other words, changes in the transcription or replication machinery have no effect on those structural aspects or their variation. For ideas, for example, the structure of a brain is required (and even then it is not necessarily the case that all ideas can be copied). This is not specific to copying as such, but simply comes from the general expectation that phenotypes that involve more parts (for example, cells, molecules or atoms) and more relationships between them (for example, types of cell neighborhoods or atom bindings in a molecule) may require more complicated processes in their production.

In addition, it can be expected that more complex copying mechanisms are also necessary for copying large numbers of structural changes in a structure (for example, to copy nucleic acids with more than four base types).

Remaking

Reproduction, defined here as the generation of offspring generative systems identical or similar to the parental generative system, can occur without copying. In *remaking* a part of an offspring, the generative system is identical to a part of a parental generative system, but this parental part is not mechanistically involved in the generation process. In remaking, the generation process intrinsically works to produce a generative system (lets say A) that is similar to that of the parent, but if the parent changes (to A′) it may still produce an offspring system looking like A. Thus, there is no model part of the parent (such as a genotype) that interacts to make a copy (so there is no copying as defined earlier). Instead, the offspring system arises only from the dynamics of the generation mechanism of part of the parental generative system.

The hypothetical chemical generative system described previously could be an example of remaking. As described, these generative systems can sustain closed mutations, but these do not arise because of copying but because of particularities of the generation process itself (note that as discussed a close mutation may lead to different phenotypes in different generations). Hypercycles, hypothetical self-catalytic cyclical reactions (Lee et al. 1997), are also an example of regeneration and so are simple cyclic reactions, such as the formose reaction (Fernando et al. 2005) (although they do not qualify as generative systems unless they can have closed mutations). Another clear, but so far only theoretical, examples are autocells (Deacon 2006).

Others

Other generation mechanisms are likely to be possible in kinds of generative systems that are currently poorly understood or merely imaginary (like the intelligent robot example). In fact, the classification of copying and remaking is not very detailed at the mechanistic level. The rest of the article, therefore, considers only the distinction between generative systems with copying and generative systems without copying.

A natural history of generative systems

From the previous discussion, it would appear that even if some generative systems without copying may exist, most generative systems known to science use copying. This article suggests that, in spite of that, generative systems without copying may be important both as possible precursors and descendants of generative systems with copying. This section discusses these possibilities from both the evolutionary dynamics of generative systems with copying and generative systems without copying.

The different types of generative systems presented and their mechanisms of generation could be compared in relation to the frequency and variety of adaptive mutations they can produce. This, however, depends on the environment and is too complex to be approached in this article. As a proxy to that, this section compares mechanisms of generation with respect to their capacity to produce closed mutations and also with respect to their capacity to produce complex phenotypes. It is not implied that evolution tends toward increasing complexity. The aim is simply to acquire some understanding about how that may happen when it does.

This section considers the idea that, in general, the likelihood by which a mechanism of generation (or development) will be found in the evolution of a lineage depends on two things: (1) the likelihood by which this mechanism can appear due to mutations in past generative systems and (2) the likelihood by which these mechanisms would produce closed adaptive phenotypic variation in a given environment.

Generative systems and the origins of life

Currently, one of the most widely considered hypotheses concerning the origins of life is the RNA-world hypothesis (Darnell and Doolittle 1986; Gilbert 1986). According to this idea, life arose through self-catalytic RNA molecules. This hypothesis was motivated by the identification of some unexpected catalytic activity in RNA molecules. However, extensive chemical research on RNA and related molecules (Monnard 2007; Chen et al. 2007) has identified only a limited number of catalytic capacities. Other biomolecules, like polypeptides, can catalyze a much richer range of reactions. Despite this, it seems that there are no straightforward mechanisms by which polypeptides could catalyze their own copying.

Even the simplest examples of RNA and DNA replication (found in some viruses) require complex enzymes. Life requires input of energy and molecular building blocks from the environment. Even if early life appeared in an environment with abundant energy and building blocks, replication would have required a minimal metabolism that may not be possible based on the limited catalytic capacities of RNA molecules. This metabolism would be required to cope with the energetic demands of generation, and possibly, maintenance. Early metabolism would also have needed to attain a molecular diversity from which complex processes such as RNA copying could arise (Dyson 1985; Morowitz 1992). In addition, as will be discussed, the replacement of a copying mechanism by another (of different chemical nature) is unlikely once

some degree of phenotypic complexity is attained. Thus, both the replication process and the minimal metabolism for life require a minimal threshold of molecular diversity and complexity. This threshold can be reached, hypothetically, by random molecular events in some special environment of the early earth special environment. Alternatively, it could arise by a process of evolution in simpler generative systems without copying. In other words, systems in which closed mutations can occur without copying should be considered as possible evolutionary precursors of life-like generative systems with copying. Evolution by closed mutations could increase the diversity of molecular species found in a lineage over time, and eventually the chances of reaching some life-like system with copying. However, this kind of generative system would need many closed mutations. This may also require some threshold of molecular diversity and complexity, but it is possible that this threshold is lower for noncopying generative systems. In fact, several researchers have proposed that early life would have appeared in chemical systems, called composomes, that can sustain heritable changes and generation but do not have copying. These systems thus fulfill the criteria to be generative systems without copying (Segre et al. 2001; Hunding et al. 2006).

The invention of the genotype and its advantages

Evolution by natural selection can be regarded as a process of learning in a population. In each generation, each individual produces a set of offspring variants (trials) and some of them may fit to existing selective pressures (tests). By the fact that the unfit produces fewer offspring, the population "learns" in which way to change. In many living organisms, this learning is mostly by trial and error at the genetic level (the trial being produced by random mutations at the genotypic level).

Copying can be seen as a mechanism of guessing that future selective pressures are going to be similar to current ones. In generative systems without copying offsprings' phenotypes can be, in principle, very different from those of the parents. In that situation, adaptation is only possible if the environment changes all the time and in a way compatible with the phenotypic variation encountered in each generation's offspring. For large populations of generative systems originating from the same ancestors, this is possible as long as many different offspring generative systems are produced in each generation (in proportion to the amount of possible adaptive phenotypes in a given environment). However, if generative systems without copying and generative systems with copying (assuming that they have similar variational properties) compete in an environment, then, the copying generative systems would be selectively favored if the environment typically remains constant between generations.

Another important advantage of copying is that it allows closed mutations. In DNA, mutations leading, after repair, to substitution of any of the four nucleotide bases, or deletions and additions, are closed mutations. Thus, DNA copying allows a large diversity of closed mutations (those appearing in any DNA sequence) by a single mechanism. Closed mutations should not be confused with modular heredity (Maynard Smith and Szathmáry 1995), in which, like for DNA, different parts can sustain mutations independently (modularity does not imply closedness nor its absence).

It is not clear if RNA was the molecule that first allowed copying in early life (in principle, other molecules could have been involved and later replaced by RNA), but once some part of a generative system is copied, it can be hypothesized that it can drive the subsequent evolution of its offspring lineage. This requires that the genotype is extensible (i.e., that it can increase in size due to mutation and still allow copying) and that it can affect processes other than its own copying (for example, metabolism). In that situation, even a small genotype with a short sequence can be expected to grow (*genotypic drive hypothesis*) to affect many aspects of the phenotype and be the main factor responsible for its evolution. This drive is not due to copying per se but to the closed mutations it allows, and hypothetically, it would also occur in generative systems without copying that can produce many closed mutations (see next section for possible examples).

This situation can be clarified with a hypothetical example consisting of a prebiotic system with a metabolism and a membrane-like amphilitic vesicle that splits in two after a threshold size is reached. If a copyable molecule (like some RNA) in it can bind to some reactants to affect their specificity or catalytic activity (even if just slightly), then some conservation of catalytic capacities would be ensured between generations (of course other mechanisms can also exist at that time). If duplications of this sequence and later mutational divergence of the duplicated sequences affect several metabolic molecules (of the same species or different species) differently, then this enhanced conservation can increase in frequency. More importantly, the effects produced by the RNA molecule on catalysis could sustain closed mutations. If these mutations lead to, at least, slightly different specificities or catalytic rates, then these RNA molecules may increase in number and type. This could increase the speed and reliability by which a lineage can respond to natural selection. The increasing chances of getting closed mutations would ensure that, over time, adaptive variation occurs most often in those aspects of the phenotype that are affected by the genotype. This process can go on as long as the copied

molecules (and their mutation) can specifically affect aspects of the metabolism and phenotype in general. It is interesting to note, in this respect, that there are a number of very widespread and conserved enzymes in living organisms that require and are affected by RNA molecules (Huttenhofer and Schattner 2006).

This hypothesis also suggests that early life may not have appeared from a set of self-copying molecules that later acquired metabolism but from a metabolism in which some closed mutations became possible and in which, ultimately, some molecules acquired self-copying ability (or collective self-copying). This allowed more closed mutations in metabolism. A side effect of this hypothesis is that the function of parts of a generative system that are not affected by the genotype may tend to be replaced by networks of other molecules that are affected by the genotype (and perform equivalent functions). This further suggests that in early evolution of life, a reduction in the diversity of types of molecules could have temporally co-occurred with the emergence of copying. Their functions would have been replaced by molecules that could have been affected by the genotype. For example, the number of types of amino acid in early life (or prelife) could have been larger than 20. Copying may have driven evolution to be based on a smaller number of amino acids combining into a large diversity of large polypeptides rather than into a large number of amino acid types combining into a large diversity of oligopeptides. Alternatively, molecular diversity may not have decreased in early life. In that case, the genotypic drive hypothesis still provides a possible explanation for why biomolecules are made of many different combinations of few types of monomers (like amino acids) and not of a few combinations of many different types of monomers.

The existence of copying causes a system to evolve mainly in those aspects of the phenotype that are affected by the genotype. The mechanisms of copying that are more likely to appear in a lineage are those that require fewer mutational changes from ancestral members in a lineage. These are likely to be simple mechanisms capable of copying only simple aspects of the phenotype and a small number of variations in it. This is because, in principle, it can be expected that simple mechanisms require less mutational change for their appearance. For example, in living organisms, only the DNA is copied (not more complex things such as protein conformations, cells or entire multicellular organisms) and only mutations that lead to substitutions with one of the four allowed bases are copied. DNA and RNA copying may have appeared because they were, on the early earth, the simplest, or among the simplest, molecular mechanisms to allow copying (and copying would have been a simple way to allow many closed mutations). Later, selection would favor developments in which the genotype (that is the copied phenotype) would come to affect most of the noncopied phenotypes.

In many living organisms, only mutations in a small proportion of the phenotype (those in the genotype) can be used as variation for evolution (mutations in other aspects of the phenotype of living organisms tend to be open). Complex multicellular organisms are generated from single cells (e.g., eggs) or small groups of cells (as in the blastemas that give rise to gemmation and other kinds of vegetative reproduction). In other words, the complex organization of multicellular organisms is not used to directly generate complex offspring systems; rather, single-cell gametes (or simple multicellular structures for vegetative reproduction) are used for this purpose. Thus, offspring are generated from the level at which more closed mutations are possible. This requires that the offspring must have a complex development in which genetically encoded proteins and RNAs interact with themselves and the epigenetic structure of a single egg or gemmule, to produce a complex multicellular organism with different molecules and cellular types in different parts. In other words, since mostly simple aspects of the phenotype (the DNA) tend to have closed mutations, complex aspects of the phenotype have to evolve and develop by multiple indirect interactions between what can be copied (and also between that and the epigenetic information of the egg cell or gemmule). This has been suggested to inevitably lead to a complex relationship between genotype and phenotype that, in many living organisms, determines, together with natural selection and genetic mutation, the dynamics of phenotypic evolution (Salazar Ciudad 2006). This arises both from entrenchment (Riedl 1978; Wimsatt and Schank 1986) (the recruitment of a gene in the production of multiple traits leading to the unreachability of an optimal sequence for it due to opposing selective pressures in those traits; Duboule and Wilkins 1998) and the fact that the developmental mechanisms that are most likely to appear, by random mutation, exhibit a complex relationship between genotype and phenotype (Salazar-Ciudad et al. 2001; Salazar-Ciudad and Jernvall 2004). This way of constructing the phenotype may not be exclusive to biological systems. Many complex ideas can arise through the interaction between several ideas. However, these ideas may be difficult to communicate directly (so then mutations in them are not necessarily closed) and need to be learned by the combination or interaction between other ideas (thus leading to a complex relationship between mutations and their phenotypic effects).

The invention of secondary mechanisms of inheritance

Evolution by selection of genetic mutations can be described, as mentioned, as a learning process. Learning by

trial-and-error is, however, not the most efficient way of learning one could imagine. Most known living organisms can change their development and physiology in response to changes in the environment (this plasticity can be something evolved or something inherent to many organisms). This way not only one, but several phenotypes can be exhibited by an individual. This is equivalent to organisms having a memory of some past adaptive phenotypes in a lineage and the capacity to decide, based on environmental clues, which one is more likely to be adaptive in their near future.

A different situation applies if individuals in a lineage or in a population can learn and communicate. In learning, the repertory of behavioral changes elicited by environmental changes is itself determined by previous influences from the environment in a generative system (in contrast to plasticity due to internally fixed responses to environmental changes). With communication, some information acquired by learning in a generative system can lead to information in its offspring generative systems. Thus, the repertory of responses (as phenotypes or behavioral phenotypes) exhibited in a lineage can change due to genetic mutations but also due to communication with other generative systems. In that way, learning plus communication is a mechanism of inheritance; some times called *cultural inheritance* (Jablonka and Lamb 2005). Learning plus communication in living organisms is a way to have more (closed and open) mutations. Many of them may not be possible or likely from changes in the genotype and in general different inheritance mechanisms may allow different kinds of changes. Learning itself allows some mutations (notice that this does not mean genetic mutations) to occur while communication allows some of these mutations (those that can be learned) to be closed (and at the same time allows the generation of offspring-idea generative systems).

A hypothesis (*secondary inheritance mechanisms* hypothesis) of this article is that secondary systems of inheritance are expected in generative systems with copying that have evolved to have complex phenotypes. This is because, as mentioned, during the evolution of complex generative systems closed mutations occur at the level of the copied elements (or at the level of the more closely mutable parts of the phenotype). Then complex phenotypes have to be generated by interactions among these elements. As described above, in living organisms with complex phenotypes, the relationship between genotype and phenotype tends to be complex. The emergence of secondary inheritance mechanisms allows more closed mutations (by copying or not) and possibly more complex (or simply other) closed mutations. The emergence of secondary inheritance mechanisms could thus be an event that would correlate with the evolution of complex phenotypes.

Learning plus communication often permits this secondary inheritance (although other systems of secondary inheritance exist; see Jablonka and Lamb 2005). There are many differences and similarities between this system of inheritance and the copying of the genome (Jablonka and Lamb 2005). The discussion of all of them is outside the scope of this article, but some of them are important for the concepts presented here.

Idea generative systems without copying are not necessarily less able to produce adaptive variation. Indeed, humans living in variable and complex environments may have benefited, in some situations, from the fact that received ideas tend to be affected by (or interpreted in the light of) previous ideas. Thus, for example, young generations may tend to interpret the ideas received from older generations in the light of the current context in which they live (and other existing ideas at the time). On the other hand, parents can try to affect the development of specific kinds of ideas in their offspring according to how they think the future is going to be. In that situation, the evolutionary guess is more variable than in the case of copying (the guess being, in copying, that selection will be similar in the future). From these perspectives, generative systems without copying may provide, in some environments, larger chances of producing adaptive variation than generative systems with copying.

It is not necessarily the case that all ideas can be communicated. It is possible, however, that human languages allow, potentially, for the expression of an infinite number of ideas. This is not necessarily informative about how many ideas a human can mutate. However, to make an analogy, it has been estimated that humans have on average 175 mutant nucleotides (Nachman and Crowell 2000) but less than 3% of the genome codes for protein-coding genes or regulatory regions. Thus, each human may have on average less than five mutations affecting proteins or their regulation (not considering synonymous changes). It is likely that an average human experiences more than five changes in his ideas over a lifetime (in fact, all or most ideas are learned) and thus it seems reasonable to assume that ideas can change, in a generation, faster, and in more complex ways, than genes.

Another important difference is that learning plus communication allows for play. By playing, an individual can explore the environment in a protected context (by direct protection from the parents, or indirectly by a nest or another modification of the local environment) in which the negative effect of maladaptive behaviors is minimized. In that situation, the offspring has more chance of learning to respond to specific environmental changes. This can substantially reduce the cost of selection. Depending on the environment and the number of offspring, this may save a lot of time and energy for the parents (compared with the

situation in which selective pressures are faced directly by the offspring in their early life).

In other aspects, genetic and cultural inheritance can be similar. Communication also exists at the level of DNA. Indeed, communication at this level can be quite frequent and not restricted to members in a lineage. Thus, many bacteria and protists can interchange genetic material. This process is called sex, although does not necessarily lead by itself to offspring. Many bacteria and some protists express specific structures enabling them to interchange DNA (Madigan and Martinko 2005). Many can simply acquire DNA molecules from the environment. Errors in the packing of virus DNA can also lead to the transfer of DNA between nonvirial hosts. These genetic transfers are possible, although rare, between organisms of unrelated species. This process is also possible, because all living organisms use DNA for copying and a similar genetic code [there is some small variation in the genetic code between prokaryotes and eukaryotes and within eukaryotes (Fox 1987)]. In the same way, communication in animals (especially in humans) is not necessarily restricted to individuals in the same lineage.

Conflicts between inheritance mechanisms

Some researchers have suggested that cultural inheritance allows, at least in humans, adapting to a wider range of environments than genetic inheritance (Jablonka and Lamb 2005). This would be consistent, as proposed here, with cultural inheritance allowing more and more complex kinds of mutations (as suggested). This greater adaptability of cultural inheritance may ensure that adaptation to selective pressures occurs more usually by changes in ideas than changes in the genotype. As a result, evolution at the genetic level could deaccelerate due to cultural evolution. This hypothesis is similar to a secondary inheritance mechanism drive hypothesis. This time it is the secondary inheritance mechanism that tends to control the evolution of other parts of the phenotype.

More generally, it can be expected (without taking into consideration specific aspects of a system's functioning) that when two or more inheritance mechanisms coexist in a given generative system the one that can produce more (or more diverse) closed mutations in a wider range of environments is likely to produce more adaptive variation and drive the evolution of the generative system (although, specifically, that would depend on which mutations each inheritance mechanism allows). This is because the driving inheritance mechanism would make many mutations in other inheritance mechanisms maladaptive (for example, genetic mutations increasing aggressiveness may be adaptive in social contexts in which communication is simple but not in contexts in which individuals may benefit from acquiring ideas from other individuals) or obsolete (for example, mutations increasing hairiness may be neutral if clothes have been invented).

Although cultural inheritance may currently drive human evolution, this still depends on the specific environments encountered. As stated by the "no-free-lunch" theorem, there is no single strategy that is the most adaptive in all environments (Wolpert and Macready 1997). Learning and communication are not necessarily better than random genetic mutation in finding adaptations to selective pressures. If the only possible adaptations in an environment are too complex to be learned by a type of generative system (meaning that they greatly exceed the understanding capacities of existing learning generative systems), other generative systems with copying may be more efficient in finding these adaptations. This can occur because generative systems that only use copying may require less energy and thus produce larger numbers of offspring (thus, by many unintelligent tries, the right adaptation may be found).

A similar "survival of the dumbest" situation can also occur if changes occur so fast that there is not sufficient time to develop (through learning and communication) a suitable adaptation. In that situation, it can be expected that the capacity of learning and/or communication would decrease in evolution, because it is costly in time and energy. In fact, there are cases in which intelligence has decreased during evolution (for example, in some salamanders; Roth and Wake 2001). It is interesting to note that, in spite of the amount of intelligent researchers devoted to it, pest control is a complicated subject and pests often (by simple mutation) manage to develop resistance to pesticides rather quickly.

In general, it can be expected (*multiple inheritance hypothesis*) that, in complex generative systems, multiple inheritance mechanisms coexist. As mentioned, once an inheritance mechanism exists, further evolution in complexity may arise by interactions between aspects of the phenotype that can exhibit closed mutations. Although different mechanisms of development can lead to qualitatively different relationships between the genotype and the phenotype (or between closed mutations and its phenotypic effects), it is very unlikely that all aspects of complex phenotypes can be inherited directly. Thus, further increases in phenotypic complexity would lead to very complex relationships between genotype and phenotype or, more generally, between mutations and their phenotypic effects (Salazar-Ciudad and Jernvall 2004, 2005). At that stage, the emergence of additional inheritance mechanisms would be quite adaptive, as discussed above, and as suggested, on different grounds, by other authors (Jablonka 1994). This is more likely than the transformation (or disappearance) of already existing

inheritance mechanisms into a mechanism able to make more complex aspects of the phenotype copyable or mutable in a closed fashion. This is because many developmental interactions are likely to be based on early inheritance mechanisms, and their variation is thus likely to disrupt most of the resulting phenotype. For example, a change in the genetic code (and the t-RNAs and associated amino acid metabolism) leading to codons with four bases (and then, potentially, to many more kinds of amino acids) is likely to be more disruptive in current living organisms (especially, in the more complex ones, in which many more developmental interactions are based on previous developmental time) than the addition of new inheritance mechanisms.

New inheritance mechanisms, like the cultural one, are less likely to interfere with the already existing development and are thus more likely to be adaptive. In this fashion, complex phenotypes would often arise by the progressive acquisition of inheritance mechanisms. For a while, once a new inheritance mechanism is attained, further complexity could be based on interactions between elements from the new inheritance mechanism that can have closed mutations, but eventually this system may also lead, as in the case of genetic inheritance, to entrenchment and to a complex relationship between mutations and their phenotypic effects. Thus, further evolutionary increase in complexity (when it occurs) may require additional inheritance mechanisms.

Other researchers have suggested that the acquisition of new inheritance mechanisms may constitute the most important transitions in evolution (Jablonka and Lamb 2005). It is also possible that the acquisition of new inheritance mechanisms will lead to the origin of new kinds of nested generative systems (as is the case with ideas).

Concluding remarks

This article has presented a set of arguments for the proposition that copying is not necessary for evolution to occur and that many nonbiological systems can be understood from an evolutionary perspective if new concepts that are not based on genetic analogies are introduced. This allows the introduction of some generic predictions about the evolution of complex phenotypes that are not dramatically dependent (within certain limits) on the physical nature of evolutionary systems but on the logic of their mechanisms of generation.

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