9 The Systems of Inheritance

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During the last two decades, most thinking about inheritance and evolution has been deeply influenced by what has been learned about the molecular nature of the gene. The structural organization of the gene, the conditions for its transmission, the way in which it is transmitted, and the way it varies, have shaped the modern view of heredity and have been very influential in molding ideas about evolution. This influence went beyond the strictly biological realm and affected ideas about the evolution of culture. However, for a gene-like concept to be used in explanations of nongenetic evolution, a more general concept was necessary. Such a concept, the "replicator," was suggested by Dawkins (1976). The replicator was defined as "anything in the universe of which copies are made" (Dawkins 1982: 83). This definition seems, at first sight, broad enough to accommodate different types of heredity and reproduction, since "copying" can be understood to include many types of processes. However, as Dawkins, Hull, and many others made clear, the replicator entails a very special kind of copying, which presupposes that only instructions or representations (which is what replicators embody) rather than the implementations of representations, can be meaningfully "copied" or inherited. Following the distinction between genotype and phenotype, which was suggested by Johannsen at the turn of the twentieth century and molded the theory of the emerging discipline of genetics (Johannsen 1911), Dawkins suggested a distinction between replicators and vehicles. He defined the vehicle as "any unit, discrete enough to seem worth naming, which houses a collection of replicators and which works as a unit for the preservation and propagation of those replicators" (Dawkins 1982: 114). The vehicle was called "interactor" by Hull, to emphasize its active functional role as a propagator of replicators (Hull 1980). Vehicles or interactors are, of course, not only carriers of replicators, but they are also their products. Development is something that happens to vehicles (and is controlled by replicators) to ensure the further propagation of replicators. While replicators are units of heritable variation, vehicles are targets of selection. The generation of new variant replicators is assumed to be independent of the selective environment (which acts on vehicles), and of the developmental process that vehicles undergo. The replicator is clearly very similar to the gene, the unit of Johannsen's genotype, and it carries much of the latter's baggage.

The view of inheritance embodied in the replicator concept affects the way in which evolution is understood, and leads to a view of evolution that reflects the modern neo-Darwinian version of Darwin's original selection theory. According to Darwin's theory, in a world in which there are interacting entities with the properties of multiplication, heredity, and heritable variation that affects the chances of multiplication, natural selection will necessarily occur, and in the long term, adaptive evolution will follow (Maynard Smith 1986). In this general form, Darwinian selection theory does not specify what the entities should be, how they multiply, how variations are inherited, or how they are generated. It also does not make a priori assumptions about the relationship between heredity and development. It is the generality of Darwin's selection theory that gives it its great explanatory power and its potential applicability to different domains of historical change.

For Darwinian selection theory to be fruitfully applied to a particular domain, its major concepts have to be specified for that domain. The replicator seems to fit particularly well the molecular, neo-Darwinian version of Darwinism (or genic neo-Darwinism). According to genic neo-Darwinism, nucleic acids are the sole units of heritable variation, the transmission of these units is independent of their expression, and the generation of genetic variations is not adaptively guided

by the selective environment or the developmental history of the organism.

This replicator-centered, gene-derived view of heredity is, however, not only severely limited, but also severely misleading. There are multiple inheritance systems, with several modes of transmission for each system, that have different properties and that interact with each other. They include the genetic inheritance system (GIS), cellular or epigenetic inheritance systems (EISs), the systems underlying the transmission of behavior patterns in animal societies through social learning (BISs), and the communication system employing symbolical languages (SIS) (Jablonka, Lamb, and Avital 1998). These systems all carry information, which I shall define here as the *transmissible organization of an actual or potential state of a system.*

In addition to the intrinsic properties of the different inheritance systems, the feedback loops formed between the organism's activities and its ecological and social environment often create conditions for the reconstruction of ancestral phenotypes in descendant generations. Developmental and ecological legacies may be said to be passed on between generations. Inheritance systems with replicator-like properties are very unusual, and certainly do not represent or sum up the many ways in which heritable variations are transmitted across generations. I use "transmission" in a general way, to denote all the processes leading to the regeneration of the same type of organization-states across generations. This includes the direct transfer of resources, as well as the activities that lead to the reconstruction of ancestral phenotypes. In what follows I shall discuss different inheritance systems and compare them with respect to those properties that seem to me most pertinent to the understanding of inheritance: the type of variation transmitted; whether or not information is encoded; the type of mechanism leading to the regeneration of variations in the next generation; the relationship between development and the generation of new heritable variations (table 9.1).

I shall then discuss the transmission of parental and group legacies through niche construction, and argue that it is the whole developmental system, with all its different and interacting inheritance systems, that has to be considered when we think about the transmission of variations from one generation to the next (Oyama 1985; Griffiths and Gray 1994). This means that the replicator/vehicle dichotomy has to be discarded, and we must go back to a single (though complex) minimal unit—a unit that is simultaneously a unit of development, multiplication, and heritable variation—the reproducer (Griesemer 2000).

I start with the most fundamental and best understood inheritance system in living organisms, the genetic inheritance system, which is based on DNA replication.

The Genetic Inheritance System (GIS)

The information in the genetic inheritance system is organized in the sequence of nucleotides in nucleic acids, which in most extant organisms is DNA. The gene is a template made up of nucleotides whose sequential organization can be transformed through a complex process of decoding into functional RNA and proteins. Genetic information is thus *encoded.* Encoding means that one system of transmissible elements (signs) represents not just itself, but also another system of elements that combine to form the actual, functional, messages. In the GIS, nucleotide triplets in a structural gene are elements of the DNA system, and they represent amino acids in a protein, which is the functional "message." In natural language, utterances represent actual objects and events in the world, as well as other words and meaning-relations.

Information is also carried in DNA regions that can control the decoding of other DNA sequences. The noncoding but regulatory regions in DNA cannot be said to encode information in the same sense as the coding regions. However, particular sequences (of varying length) are spread throughout the genome and perform

Table 9.1
Types of information and modes of transmission for different systems of inheritance

Inheritance System	Variation transmitted: Unit	Variation transmitted: Origin	Information: Alteration	Information: Encoding	Mode of transmission: Type	Mode of transmission: Direction	Type of Heredity
GIS (genetic)	DNA sequence (gene)	Blind & some patterned	Modular	Encoded	Modular	Mostly vertical	Unlimited
EIS (cellular)							Niche construction—variant interaction of organism and environment can be transmitted
Steady-state	Activity state of metabolic cycle	Blind & patterned	Holistic	Nonencoded	Holistic	Mostly vertical	Limited
Structural	3D complex	Blind & patterned	Holistic	Nonencoded	Holistic	Mostly vertical	Limited
Chromatin marking	Pattern of chromosome marks	Blind & patterned	Holistic	Nonencoded	Holistic	Mostly vertical	Limited
			Modular (methylation)	Can be encoded	Modular (methylation)	Mostly vertical	Limited?
Epigenetic (organismal) Inducing substance	Physiological state	Blind & patterned	Holistic	Nonencoded	Holistic	Mostly vertical	Limited
BIS (behavioral)							Unlimited at the cell level / Unlimited at the organism and lifestyle level
Inducing substance	Pattern of behavior	Often induced (patterned)	Holistic	Nonencoded	Holistic	Mostly vertical	Limited
Non-imitative social learning	Pattern of behavior	Often learned (patterned)	Holistic	Nonencoded	Holistic	Vertical & horizontal	Limited
Imitation	Pattern of behavior	Often learned (patterned)	Holistic	Nonencoded	Holistic	Vertical & horizontal	Limited
SIS (symbolical)	Symbolic form and content	Learned (patterned)	Modular & holistic	Encoded	Modular & holistic	Mostly horizontal, some vertical	Unlimited

sequence-typical regulatory functions, so general types of functions can be inferred from sequence organization. Such regulatory sequences thus form a kind of higher order "code."

The organization of information in DNA is modular (or digital), that is, it is decomposable into separate discrete units drawn from a standard set, (the units in DNA are the nucleotides A, C, T, G), and the information is alterable digit by digit. Following Szathmáry (1995), a replication process that proceeds digit by digit will be called *modular replication*. The genetic system is the prime example for a system that is modularly replicated. The enzymatic machinery that replicates the DNA, or that edits and repairs it, is largely indifferent to its sequence organization. This means that a sequence that has beneficial effects when decoded will be replicated and repaired with the same fidelity as one with deleterious effects, or a sequence that is completely nonfunctional. Furthermore, the transmissibility of the template remains unaltered following its replication. Usually transmission is vertical, from parents to offspring, but occasionally it can be horizontal, so genetic information can be transmitted between nonrelated individuals, including individuals belonging to different species.

The modular nature of the replication and alteration of information allows for the inheritance of many combinations of modules—a DNA molecule with ten linearly linked nucleotides has more than a million possible variant sequences. This means that the evolutionary potential of a modularly alterable and transmitted unit, such as a gene that consists of hundreds of nucleotides, is very large. The number of possible sequences greatly exceeds the number of individuals in any realistic system. Such a system can be said to have *unlimited heredity* (Szathmáry and Maynard Smith 1993; Maynard Smith and Szathmáry 1995).

Until recently, the generation of variations in DNA has been assumed to be random with respect to the selecting environment. Variations were assumed to be exclusively the consequence of the meiotic reshuffling of genes (in sexually reproducing organisms), and of several classes of errors in DNA maintenance. Errors can be due to physico-chemical damage to the DNA, they can occur during DNA replication and repair, and they can result from the activity of genomic parasites: genetic elements that multiply excessively and move from site to site in the genome. Errors that are not removed or repaired accurately by the DNA maintenance machinery were assumed to be the ultimate raw material for evolution by natural selection. Although there is no doubt that a lot of variation in DNA is indeed random in this sense, the view that *all* variation is random has been challenged.

The challenge has come from several directions. It has been shown that different nucleotide sequences differ in the likelihood that they will be damaged, invaded by genomic parasites or replicated inaccurately. The rate and type of new variation may thus depend on how the nucleotides in the sequence are organized, and this organization may be adaptive. For example, Moxon and his colleagues have shown that in the pathogen *Haemophila influenza* the genes that influence its antigenicity are highly mutable because the short tandem repeats in them make them prone to mutation by recombination and strand slippage. The high mutation rate in these genes is advantageous, because it enables this pathogenic organism to evade the immune system of the host (Moxon et al. 1994). The sites in which mutations preferentially occur are the result of adaptive evolution. Moreover, mutation rate can increase selectively not only at sites but also in conditions in which a higher mutation rate is selectively beneficial. Wright (1997) has shown that amino acid starvation in *E. coli* increases the transcription of genes that help the cells survive longer, and concurrently increases the mutation rate in these genes. This condition-dependent increase in mutation rate is adaptive since such targeted mutation in the relevant genes may "rescue" the cell without greatly increasing the load of mutation. It seems that through natural selection the mechanisms that allow selective control of gene expression have been coupled with mechanisms that determine the fidelity of copying, so that the inducible system that turns genes on and off also turns the production of mutations on and off.

Such "targeted" mutations cannot be said to be random in the classical sense, since adaptively advantageous mutations are preferentially (though not exclusively) induced under the appropriate conditions and in the relevant domains. Randomness has not been eliminated, but it has been restricted and channeled. However, the mutations are not goal-directed in any teleological sense, and their targeted production is the consequence of natural selection that had acted on random variations. Variation has been targeted by selection to be preferentially generated in a subset of sites, under particular conditions. It is difficult to know how to define such variations. The term *patterned variation*, which has been suggested by the economist Ekkehart Schlicht with respect to cultural evolution, is the one I choose to use in this paper (Schlicht 1997). It is better than previously suggested terms such as directed, adaptive, induced, and guided variation because it does not carry the teleological connotation of premeditated design, yet does carry the connotation of some degree of preexisting structuring (by past natural selection). Once a system for generating patterned variation has evolved, it channels and guides evolution.

From an evolutionary point of view the existence of a cellular system for the production of patterned variations makes good sense. It would be remarkable if a cellular system for targeting the generation of variations had not evolved during the four billion years since life appeared on earth. It is quite easy to see how the enzymatic genetic engineering kit that all cells use to rearrange, amplify, and delete pieces of their DNA could have been modified by selection to allow the genome to respond to different reoccurring types of environmental stress (Shapiro 1997).

The ability to generate patterned variations forges direct links between heredity, development, and evolution. The generation of patterned variation is part of the developmental process no less than changes in transcriptional activation of genes, although the effect of changes in DNA may often last longer than changes in transcriptional activity. The process of generating patterned variation is part of both development and evolution. Although there is a certain (short-term) degree of autonomy of heredity and development if mutations are random, if they are patterned, heredity loses this partial independence.

The Epigenetic Inheritance Systems (EISs)

Epigenetic inheritance systems are the systems underlying cellular heredity. It is well known that once cells become determined during development, they often maintain their functional and structural characteristics through many cell divisions, even though the stimuli that first induced their determined state early in development were transient, and are no longer present. Kidney cells and fibroblasts within the same organism have identical DNA base-sequences, yet each cell type "breeds true": Kidney cells transmit their functional state to daughter kidney cells, while skin fibroblast cells transmit their very different cellular phenotype to their descendants. The mechanisms that are responsible for this cellular inheritance have been termed epigenetic inheritance systems. The transmission of heritable epigenetic variations is possible not only within individuals, but also between generations of individuals, so EISs can have direct evolutionary importance.

Three types of epigenetic inheritance systems (EISs) have been described (Jablonka and Lamb 1995). The first type of EIS is the steady-state system, which is based on the activity of self-sustaining feedback loops. It was first described theoretically by Wright (1945), and has been found in many biological systems. In its simplest form, a gene produces a product as a result of

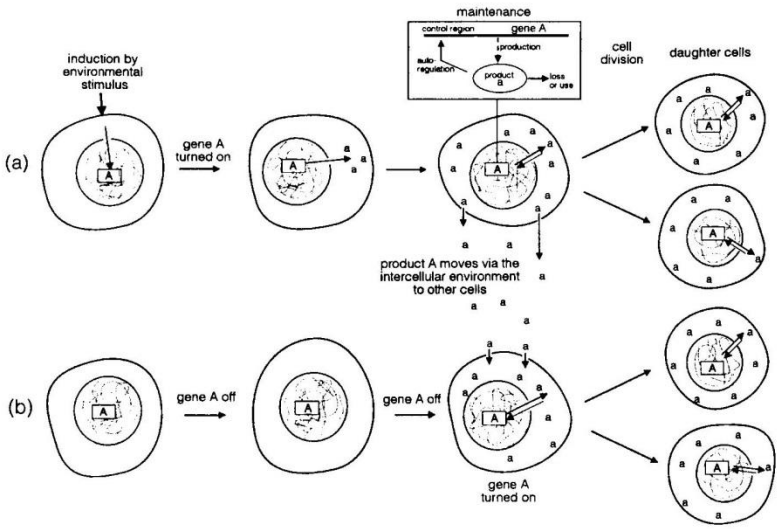

Figure 9.1
A steady-state system showing the perpetuation of an induced active state through cell division. (a) After induction, gene A is turned on and its product, *a*, positively regulates its own activity. The regulator *a* need not be a direct protein-product of gene A, but can be the metabolic product of the direct (protein) product, a small metabolite with regulatory function. The box shows the self-regulation of the genetic circuit. (b) The regulatory product *a* diffuses into the environment, enters into inactive cells, turns on gene A, and hence leads to the self-sustaining activity of the circuit in these cells.

induction by an external developmental or environmental stimulus, and this product then stimulates further activity of the gene (through positive self-regulation) even when the original external inducing stimulus has disappeared (figure 9.1). Once switched on, the cell lineage continues to produce the gene-product unless its concentration falls below some critical threshold value. Two genetically identical cells can therefore be in two alternative states ("on" and "off"), and both states can be self-perpetuating, even when the inducing environment changes. Thus two genetically identical cells in the very same environment may be heritably different because of the prior, different, developmental history of their ancestor cells. As long as the concentration of the products of the self-sustaining cycle does not fall below a critical threshold, the active, "on," state is maintained following cell division; once the concentration falls below the threshold, the cycle is in the "off" state, which is also maintained. The states of activity and inactivity are reproduced in daughter cells as an automatic consequence of cell division, and transmission is an integral part of growth and multiplication. The generation of the activity state is part of development, yet the developmental states can be faithfully transmitted within the cell lineage for many generations.

The information reproduced in this type of system is *nonmodular or holistic* (here I follow the distinctions, but not the terminology, of Szathmáry 1995). Although the cycle can be divided into discrete modules (modular gene A, modular product *a*, modular regulatory domain), the *functional* state cannot be transmitted module by module. It can only be transmitted when the processes of interactions among components are regenerated in the daughter cells. Changes in any one component usually prevent the transmission of the whole cycle. It is only the state of activity of the whole cycle that can vary. However, cellular states may also be transmitted horizontally, between lineages. If the positively regulating product is not only transferred to daughters cells as an inevitable part of cell division, but also diffuses to the cell's environment, it may "infect" neighboring cells from another lineage and induce its own activity state in them. Rather than inheriting the phenotype through descent, the nondescendant cells are interacting with the environment that the "infecting" cells have modified and become phenotypically identical to them through this interaction (figure 9.1b).

Often each individual self-sustaining cycle can have only two states ("on" or "off"), and the system can move only between two states, so nothing evolutionarily very interesting can occur at this level. The number of variant, functional, and heritable states that every single cycle can show is very small, much smaller than the number of individuals the population can include. The system therefore can be said to show *limited heredity*. However, within a cell there are often several independent cycles. More than a million variant cell states are possible if a cell has twenty different cycles! New developmental conditions can induce changes in the activity states of several cycles in cells, producing many variant states, which can then be subject to selection. Thus, at the level of the cell, the inheritance of functional states may be practically unlimited, and cumulative evolutionary change may occur. In this case, of course, many of the variations are clearly induced by the environment (although random environmental fluctuations may also generate some variants). The environment both induces a set of different adaptive variant states and fine-tunes the adaptation by selecting the most appropriate ones. In this inheritance system both the reproduction of the activity states in daughter cells and the generation of variations are part of the cell's development, and it is the phenotype (a dynamic activity state, a process) that is reproduced.

The second EIS is that of structural inheritance, where existing cell structures are used to guide, or template, the formation of new similar structures. Variant complexes or architectures made up of the same components can be stably inherited. Inheritance is through some kind of three-dimensional templating, with existing structural patterns facilitating the construction of similar "daughter" patterns. For example, in ciliates, genetically identical cells can have different patterns of cilia on their cell surfaces, and these different patterns are inherited. Prions seem to be another example of such structural inheritance (Grimes and Aufderheide 1991, Tuite and Lindquist 1996). In this structural inheritance system there are clear modules (the modular components of the complex), but transmission is holistic: The complex is not transmitted module by module, nor are the modules alterable unit by unit. The structural information may be transmitted by the fragmentation of the original complex, followed by growth, as in a crystal, or by other means where the interacting units within the complex form the conditions for self-organization of free floating units. There is no general, autonomous system of transmission independent of the structural properties of the particular complex. The reliability of transmission will be specific to each structural complex and depend on its unique properties. Variations in the

organization of the units into self-perpetuating complex-variants can be affected by environmental conditions, so variations are often patterned (figure 9.2). As with the steady-state EIS, structures are likely to be passed on vertically, by descent. However, horizontal transmission is also possible, as testify some prion diseases where the pathogenic prions are transmitted to nonrelatives and even to individuals of other species. The number of heritable states of each complex may be very limited, but in a cell with tens of complexes, there are practically unlimited heritable architectural states. "Copying" of complexes is part of development and multiplication; there is no specialized machinery that can copy different architectures. Variation, when patterned, is both developmental and evolutionary.

In the third EIS, the chromatin-marking EIS, states of chromatin that affect gene expression are clonally inherited. Genetically identical cells can have variant and heritable chromatin marks. Marks are protein or RNA complexes associated with DNA, or small chemical groups, such as methyl groups, that bind to certain nucleotides. The type, the density, and the pattern of marks on a chromosome region affect its potential transcriptional state, and changes in marks can be induced by the change in the environment. When the marks are protein complexes, their reproduction in daughter cells is probably similar to the reproduction of other three-dimensional complexes, although the DNA sequence to which protein marks bind may constrain variation and enhance the fidelity of reproduction. However, the best-understood chromatin marking EIS, the methylation-marking EIS, is somewhat unusual in its modular organization and mode of transmission. Nucleotides in many organisms can be in a methylated or nonmethylated state, and the alternative states can be clonally inherited. The most commonly methylated nucleotide is cytosine, and in most eukaryotes it is the cytosine in CpG doublets or CpNpG triplets that can be in an either methylated or nonmethylated state. The methylated state of the nucleotide has no effect on the coding properties of the triplet in which it participates, but can affect transcriptional activation in the chromosomal region in which it occurs. With this EIS there is a dedicated, function-independent, copying machinery (the enzyme methyl-transferase) that can copy patterns of methylation irrespective of their past or present function. Information is organized in a modular way (a nucleotide can be in two states—methylated or nonmethylated), so that methylation sites are alterable unit by unit, and transmission proceeds module by module (figure 9.3). However, the reproduction of methylation patterns is not always modular and does not always depend on the special enzymatic machinery. Methylation patterns can be transmitted sexually between organisms through sperms and eggs. As the germline becomes determined, there are widespread and sometimes radical changes in chromosome marks, including patterns of cytosine methylation on chromosomes. However, parental patterns of methylation can still be regenerated in the offspring because some traces of the past are retained, as partial (protein or methylation) marks, and these partial traces or "footprints" are reconstituted into full marks during the embryogenesis of the offspring. There seems to be a cycle of changes in chromosomal marks during germline formation and during early embryonic development that leads to the reconstitution of parental methylation marks (figure 9.4). Because changes in methylation marks, like changes in other types of heritable chromosome marks, can be induced by the environment and the variation can be inherited, some of the variation is patterned. Heredity in this system is unlimited when we consider the whole genome or several large chromosomal domains, but limited when a short DNA sequence is considered.

Unlike the GIS, with all EISs the generation of new variation is typically patterned (although it can also be completely accidental), and cannot be divorced from the physiological development of the cell as it interacts with the environment. In most cases, the transmission of information is

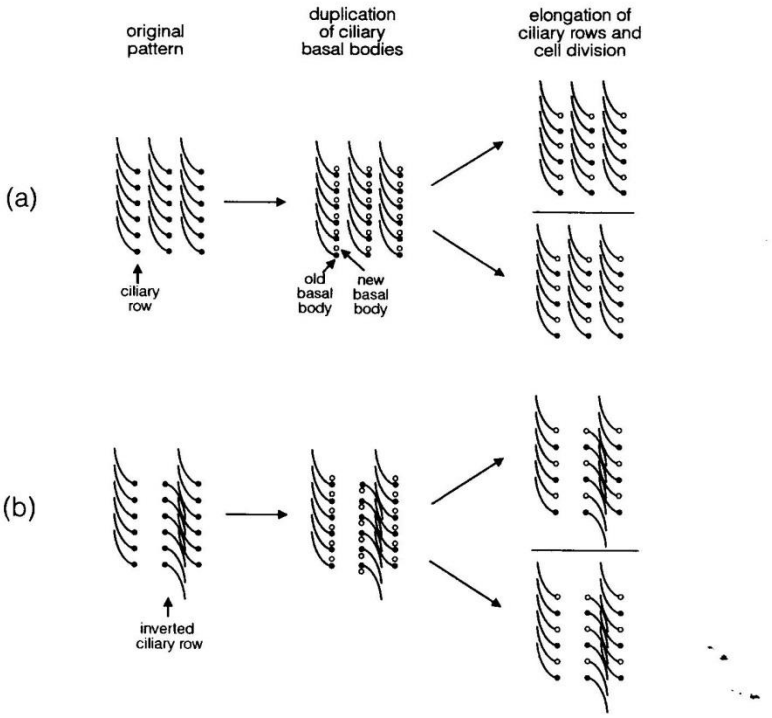

Figure 9.2
The perpetuation of two alternative organizations of ciliary structures in *Paramecium*. (a) The organization and perpetuation of normal ciliary rows through cell division (horizontal line). (b) The perpetuation of an experimentally inverted ciliary row.

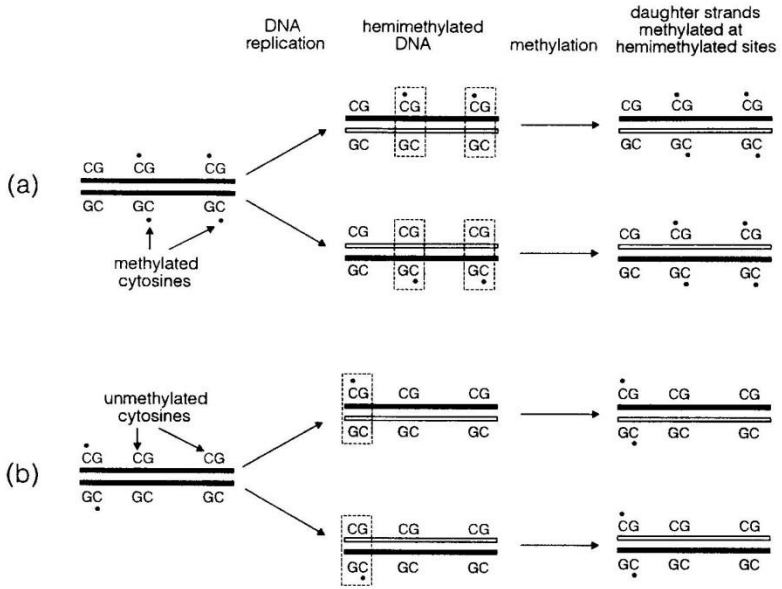

Figure 9.3
The inheritance of alternative patterns, (a) and (b), of DNA methylation. The black dots represent methyl groups. The dotted boxes show hemimethylated sites following the replication of DNA. These sites are preferential targets for a methylating enzyme, which methylates the opposite nonmethylated site in the DNA duplex. Different methylation patterns can therefore be perpetuated through cell division.

holistic. The processes that allow the faithful transmission of variant functional or structural states in the cell lineage do not utilize a dedicated, specialized, function-independent copying machinery (with the exception of the methylation EIS in somatic cells). Instead, these processes are by-products of general growth and multiplication processes. The fidelity of reproduction depends on the specifics of the cycle, or the three-dimensional structure of the complex. At the cellular level heredity is unlimited, although it may be very limited at the level of the functional, transmitted unit itself. Of course, when we are looking at the functioning of the cell, the different inheritance systems interact and cannot be treated as autonomous: For example, products of a steady state EIS can affect heritable chromatin marks and 3D structures, and vice versa.

If we move from the level of the cell to the level of the multicellular organism, there is ample evidence showing that the cells that begin new organisms, the egg and the sperm, can carry epigenetic information, and that variations in epigenetic information are often inherited (Jablonka and Lamb 1995, 1998). There is also another type of phenotypic information transfer between generations, which is more difficult to categorize because it does not occur at the cellular level, but at the level of the whole organism. The maternal environment in which the mammalian fetus develops sometimes has effects that can be carried over to later generations. For example, if female Mongolian gerbil embryos develop in a uterine environment in which most of the embryos are male, and they are therefore exposed to high level of testosterone, they mature late, are more territorial than other females and, in turn, produce litters with a greater proportion of males than the normal 1:1 sex ratio. The result is that their daughters, who usually also develop in a testosterone-rich uterine environment, also mature late, and produce mainly male offspring (Clark, Karpiuk, and Galef 1993; Clark and Galef 1995). The developmental legacy of the mother is transferred to her daughters, so there is a nongenetic transmission and repetition of this distinctive reproductive pattern. Without any genetic differences, two maternal lineages may differ, consistently, over many generations, in the sex ratio of the offspring they produce.

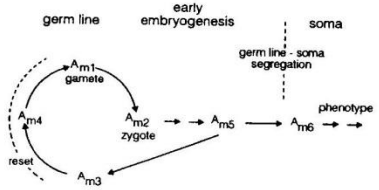

Figure 9.4
A normal cycle of changes in chromatin marks (e.g., methylation marks) during gametogenesis and early embryogenesis. As germ cells proceed through gametogenesis, the chromatin marks on DNA sequence A change (from m3 to m1). In the zygote, the mark on A is changed to m2, and during early embryogenesis to m5. When segregation of soma and germ line occurs, some cells with m5 marks become germline cells and again acquire mark m3. How an induced change in marks may alter the cycle is not shown here (for a figure and discussion of self-perpetuating cycles of changed marks, see Jablonka and Lamb 1995: 154–156).

Another example of phenotypic transmission at the organismal level is the transmission of microorganisms between generations through feces. Young of many species of mammals consistently eat their own and other individuals' feces, a habit known as coprophagy. Most of the mammals that practice coprophagy are herbivores, who consume cellulose-rich plant material and have a dense symbiotic bacterial and protozoan gut flora that helps them to break down and digest cellulose. The young of many herbivorous species eat their mother's feces, and in this way they directly inoculate their own guts with the maternal flora of useful microorganisms. Differences between the gut floras of different mothers, will be transferred to their offspring, and may be perpetuated for many generations. In many cases, these parental legacies affect behavior.

The Regeneration of Behavior: The Behavioral Inheritance Systems (BISs)

Behavior that can be transmitted has been categorized in many different ways. With social learning alone, more than thirty terms and distinctions have been suggested. For the purpose of this essay, which concentrates on the type and transmission of information, I will distinguish three general types of transfer of behavioral information.

The first is very similar to the whole-organism transgenerational reproduction of phenotypes discussed in the last section, but focuses on the reproduction of behavior. In this system the processes that lead to similarity between the behaviours depend on the transfer of behavior-affecting substances between interacting individuals. I therefore call this type of transfer the *inducing-substance* transfer. Unlike the other two BISs, transmission is not dependent on learning.

The transmission of food preferences via the transfer of substances through the placenta and the milk in mammals is a good example of this type of BIS. Mammal fetuses are able to smell semivolatile liquids transferred to them across the mother's placenta, and later show preference or aversion for food items carrying these smells (Smotheran 1982; Hepper 1988). Transmission of substances through milk has similar effects. The results of cross-fostering and other simple experiments with mice have shown that the food the mother prefers, and therefore frequently eats, biases the food preferences of the young so that those who feed on her milk tend to have the same preferences. Such results are typical for many mammals, including other rodents and ruminants (Galef and Sherry 1973; Provenza and Balf 1987).

There are other channels through which inducing-substances that bias behavioral preferences can be transferred (Avital and Jablonka 2000). Information transferred in inducing substances is not encoded, and its transmission is holistic. Usually (but certainly not always), transmission is vertical. The variation generated is commonly patterned (induced), and heredity is limited, although the number of variant preferences and the behaviors they influence may be quite large.

The second type of transfer of behavioral information occurs through nonimitative social learning. This has received a great deal of attention from experimental psychologists who differentiate between several different types of social learning that do not involve imitation and/or direct instruction (Zentall and Galef 1988; Heyes 1994). I call this type of social learning *nonimitative social learning*. In the cases covered by this category of social learning, the naive, observing individual (or "observer") learns about the environmental circumstances (including the objects, stimuli, and events) that elicit a particular behavior in the experienced individual. Two examples will help to illustrate the nature of such social mediation. When young monkeys become fearful of snakes after observing the panic-stricken reaction of adults to snakes, they too will avoid

Figure 9.5
A blue tit opening a milk bottle by tearing the foil cap (from Hinde 1982).

snakes. However, what they learn is not the motor flight behavior patterns of the experienced adults, but rather that snakes have to be avoided. The second example is the cultural spread of the blue tit's and the great tit's habit of opening milk bottle tops, a famous case of "cultural" transmission of behavior (Fisher and Hinde 1949). Tits learnt by observation the habit of removing the cap and getting at the cream at the top of the bottles (figure 9.5). This is probably another case of non-imitative social learning. The spread of the behavior from experienced to naive tits can be explained as the result of naive tits having their attention drawn to the milk bottle as a source of food, commonly through the behavior of an experienced individual (Sherry and Galef 1984). The method by which the top was removed was not imitated—each individual tit focused its attention on the milk bottle as a potential source of food and, after it own trial-and-error learning, finally learned how to remove the top in its own style. Such social mediation leads, in most cases, to similarity between the behaviors of the "observer" and the "model." The model guides

or enhances the attention of the observer to the environmental stimulus (such as a milk bottle, or a dangerous predator), which elicits a similar emotional and behavioural response to its own.

In this type of behavioral inheritance, information is not encoded. Variation is generated by the inventor of the new behavior through asocial learning. It is therefore patterned rather than accidental. It is holistically transmitted through social learning, and can be transferred both vertically and horizontally. Heredity is rather limited, since the number of variants the behavior pattern can assume may be restricted. However, at the level of the overall lifestyle, heredity may be practically unlimited, since different variant patterns of behavior may combine to form many types of lifestyle.

The third type of BIS is learning by imitation and/or instruction. I consider it to be another type of BIS because of its modular way of transmission. As Heyes (1993) has argued, there is no compelling evidence to suggest that imitation is inherently more cognitively demanding than several other types of social learning. However, the modular way of transmitting and altering behavior during imitation or instruction—the parsing of behavioral acts—sets it apart from other types of social learning. During imitation, the naive individual reproduces not only similar responses to the environment, but also the model's actual acts. Vocal imitation is very common among many species of bird, whereas motor imitation has been validated beyond reasonable doubt in only a few species. Humans, chimpanzees, dolphins, budgerigars, rats, and a few other birds and mammals have been shown to be able to imitate motor acts. However, because relatively few experiments have been designed to distinguish between imitative and nonimitative learning of motor acts, the extent of motor imitation may be underestimated. Intentional instruction seems to be very rare in the animal world, but again, this issue has not yet been systematically studied.

The information acquired during imitation and nonsymbolical instruction is, as with other types of social learning, patterned and nonencoded, and is transmitted both vertically and horizontally. Heredity is often limited although, in theory, if a behavioral act is made of many individually alterable and transmissible modules (for example, if the song of a songbird is made of many types of phrases), heredity may be unlimited. However, a huge number of combinations leads to a huge amount of nonsense-messages—to functionally useless or even positively harmful information. Only if there is some patterning or ordering of the combinatorial process can the search for functional sequences in the infinite space of possibilities yield functionally meaningful results (Schlicht 1998). It is only when there is a reasonable probability that variant behavioral modules combine to form different yet *functional* sequences of behavior that the modular transmission of sequences opens up truly wide evolutionary possibilities. Rule-bound organization and transfer of information is clearly necessary. We see this kind of rule-bound organization in systems of symbolic communication.

Symbolical Systems of Inheritance (SIS)

As T. W. Deacon stresses in his 1997 book on the evolution of language, symbols are not simple. The American philosopher Charles Peirce distinguished between three ways in which a sign (defined as information communicated between sender and receiver) can refer to something. First, a sign can refer to an object by resembling it. This type of sign is called an icon, and an example is a picture of a house, which refers to an actual house, or the pattern on a mimetic butterfly's wings, which resembles (and can be said to refer to) the pattern on a model's wings. Second, a sign can be an index and refer to an object by association, through being linked to the object in space or time. For example, the size and brightness of a male peacock's tail is an index of its health and vigor. Finally, a sign can refer to an object by convention, or according to a reference-rule that enables it to refer to other signs in the system.

Such signs are symbols. Symbols must represent objects, operations, and relations among signs (as in natural language and, in the purest way, in mathematical notations). The category to which a sign belongs depends on the interpretive system of which it is part, rather than on the isolated sign; a portrait, for example, though iconic, is also a part of a symbolical system, and should therefore be interpreted as a symbol. Natural human language is another example of a symbolical system. In the sentence I am writing just now, most words refer to other words rather than to objects in the world.

From the point of view adopted in this chapter, symbolical systems are transmitted by social learning, which often involves imitation and a greater or lesser degree of intentional instruction. Symbols are transmitted both modularly and holistically. For example, in the case of natural language, the narrative, the sentence, the word, the phoneme are all transmitted, but it is quite clear that a spoken narrative is (unless a story is learned by heart) more holistically transmitted than a single new word. Interpretation depends on the rules of the system (for example, grammatical rules), so symbolical systems are organized by those rules. Sometimes, as in natural language or mathematics, the organization is easily formalized (rules of language-specific grammar, mathematical axioms), but it can be more fuzzy (as in dancing, music, and the visual and motor arts). Information is (by definition) encoded and is almost invariably transmitted horizontally. Vertical transmission is common in some systems, however. For example, early language learning usually involves vertical parent/offspring interactions. In other cases, such as the transmission of painting skills, it is almost always nonvertical from master to student. Symbolical systems have unlimited heredity and huge evolutionary potential. The rules of symbolical systems organize the systems and order them, so variation is inherently constrained and patterned by these internal rules. New variations arise as a result of insight, trial-and-error-learning and accident.

Table 9.1 summarizes the different properties of the four types of inheritance systems and allows a comparison among them. What is clear is that a system based on encoded information, modular transmission, and modular alteration of the composing modules is a very special type of inheritance system. The two inheritance systems that have these properties and are closest to each other in this respect are the GIS and the SIS. However, the SIS is evolutionarily derived from the BISs, and it shares important characteristics with them. It is nevertheless significant that both the GIS and the SIS have unlimited heredity at the level of the transmitted units themselves, and not, as with other inheritance systems, only at a higher level of organization. Because of the ability to encode information, both the GIS and SIS transmit a lot of unexpressed information. Nonfunctional genes are transmitted, as also are nonimplemented ideas. This provides a huge reservoir of variation, which may become useful in new conditions. I believe that this ever-present potential gives these systems a particularly important role in long-term evolution. However, no inheritance system acts in isolation: inheritance systems interact both directly and indirectly. For example, the social animal, with its BISs, determines the selective regime in which genes are ultimately selected.

Another point suggested by the table is that by considering a higher level of organization, limited inheritance systems may become unlimited. Hence we see that EISs are limited inheritance systems at the level of the unit of transmitted information (cycle of activity, 3D complex, local pattern of marks), but may be unlimited at the level of the cell phenotype. A practically unlimited number of cell phenotypes can be generated. The same is true of BISs—at the level of a single behavior pattern there may be few variants, but the lifestyle as a whole can display many more variations. Although biological information at the lower level is holistically organized, at the higher level each state is treated as a module

that can combine with others and produce practically unlimited variation.

It seems that as a system becomes more functionally cohesive during evolution, evolving repair and compensatory mechanisms, its heredity becomes increasingly more limited. There is less selectable variation, and the result may be evolutionary stasis. There are two situations in which escape from such stasis is possible. One occurs when selection acts at higher level of biological organization (at the level of many combining units), that is, when a higher level of individuality emerges (Jablonka 1994; Jablonka and Lamb 1995). The second occurs when a system of encoding the information evolves. Both situations have occurred during evolutionary history.

The Transmission of Organism/Environment Variations: Niche Construction and Niche Regeneration

The right-hand side of table 9.1 shows that organisms often transfer variations in their epigenetic characteristics or their behavior patterns in an indirect way. By providing their descendants with the initial conditions that allow the repetition of their own developmental processes, similarity between generations is enhanced. Both Waddington (1959) and Lewontin (1983) stressed that living organisms are not passive entities, but ones that actively choose and construct their environment, and hence also the selective regime in which they live and in which they breed. The most obvious examples are the nests of birds and the dams of beavers. Such artifacts are often also passed on to the next generation.

Odling-Smee developed these ideas further, stressing the multigenerational transfer of many types of variations in niches. He argued that because through their activity and behavior organisms construct the ecological and social niche that they occupy, this "niche construction" may often ensure that the environmental conditions in which they have lived will be regenerated and reexperienced by their descendants (Odling-Smee 1988, 1995; Odling-Smee, Laland, and Feldman 1996). For example, males of some species of bowerbirds build small huts to attract females, bringing fruits, seeds, and fungi to decorate them. These decorations are often able to grow, so by their behavior bowerbirds also ensure the long term supply of the materials which they, and their descendants, will choose as decorations (Diamond 1986, 1987, 1988). Caching seeds is another example of a habit that may be reinforced through the effect it has on the local environment. By caching seeds, animals provide themselves with a source of food for harsh winters, but because some of the cached seeds germinate, caching also provides new plants that will form seeds and create future caching opportunities (Källander and Smith 1990; Smith and Reichman 1984).

Even more obvious examples of niche construction are the propagation of dialects in bird or whale groups, where the dialect of the previous generation is the condition for the acquisition of this dialect by the younger generation. Similarly, learning to speak by human children is guaranteed by the child's developing in a preexisting linguistic community. Such ecological or social niche construction ensures that the ecological and social milieu is transmitted. The conditions eliciting the ancestral behavior are reconstructed, and selection for the maintenance of the behavior pattern that fits the constructed niche occurs.

The regeneration of ancestral niches and selective regimes can occur at different levels of biological organization. At the cellular level, we saw that when the regulatory product of a steady-state cycle can diffuse into the environment it changes it, thereby creating the conditions that induce a cycle of self-perpetuating activity in neighboring cells. This is a simple form of niche construction. All types of niche construction depend on the formation of self-sustaining feedback loops between the developing organism and its niche.

A Different Kind of Darwinism

The diversity of inheritance systems that are able to transmit variation at different levels of biological and social organization should surely prevent developmental and evolutionary biologists from interpreting development and evolution in terms of genetic variation alone. Yet, not only are other sources of heritable variation neglected in gene-centered accounts, but also the whole dynamics of inheritance, which is an aspect of the developmental process, is ignored. This leads to a very faulty account and understanding of development and of evolution, and completely misses the complexity, possibilities, and limitations of developmental and evolutionary processes.

Moving from the gene to the more abstract replicator, and assuming that the replicator is the unit of variation and evolution, is also not satisfactory. The replicator/vehicle dichotomy, which is fundamental to the concept of a replicator, is meaningless in all cases in which the transmission of information or the generation of new heritable information depends on development. Yet, as table 9.1 illustrates, this is the usual case. The replicator-vehicle distinction cannot therefore be used to analyze heredity, development, or evolution. However profitable the distinction between replicator and vehicle may be for some evolutionary theorizing, this distinction simply does not apply to real organisms.

At the beginning of this chapter I suggested that in order to have a unifying concept of heredity that encompasses all the types of inheritance system, we need a theoretical framework that is broader than that used by genic neo-Darwinism. The developmental system approach suggested by Oyama (1985) and Griffiths and Gray (1994) provides such a framework, as it focuses on the developing and interacting individual, with the multiplicity of its inheritance systems and self-perpetuating feedback loops. The reproducer concept suggested by James Griesemer (2000) provides the unit of analysis for such an approach, for the reproducer is simultaneously a unit of development, of multiplication, and of heritable variation, as well as a target of selection.

The focus on units of reproduction introduces back into evolution the developing individual as an active evolutionary agent. This leads to the consideration of the different types of developmental processes that lead to the regeneration and reproduction of variant characters. It inevitably leads to concurrent attention to selection at different levels of organization—the gene level, the cell level, the organism level, and so on, and to different types of heritable variation—the genetic, the epigenetic, the behavioral, and the symbolical. It is this richer version of Darwinian theory that needs to be adopted.

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10 Niche Construction, Ecological Inheritance, and Cycles of Contingency in Evolution

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A recurrent theme of this book is the rejection of dichotomous thinking characterized by emphasis on processes that are regarded as either internal or external to living organisms. As Lewontin (1983) has pointed out, the tendency to think dichotomously is not confined to developmental biologists. Evolutionary biologists can also slip into a dichotomous mode of reasoning. One of the principal dichotomies in evolutionary theory, to which Lewontin draws attention, stems from the separation of the causes of ontogenetic variation, seen as coming from internal factors, especially Mendelian genetics, and the causes of phylogenetic variation, seen as something that is imposed by natural selection pressures arising from autonomous external environments (Lewontin 1983). In this chapter we shall focus primarily on the second half of this dichotomy. We want to reconsider the extent to which Darwinian natural selection pressures in external environments really are autonomous, that is, they really are independent of the organisms they select, and we shall suggest that often they are not. We shall also propose evolutionary processes that can cause naturally selected organisms to modify their own natural selection, as well as the selection of other organisms.

Classically, adaptation has been conceived of as a process by which natural selection, stemming from an external and independent environment, gradually molds organisms to fit an established environmental "template." The environment is seen as posing problems, and those organisms best equipped to deal with the problems leave the most offspring (Lewontin 1982, 1983). Although the environmental template may be dynamic, in the sense that processes independent of the organism may change the world to which the population adapts (Van Valen 1973), the changes that organisms themselves bring about are rarely considered in evolutionary analyses. Yet to varying degrees, organisms choose their own habitats, choose and consume resources, generate detritus, construct important components of their own environments (such as nests, holes, burrows, paths, webs, pupal cases, dams, and chemical environments), and destroy other components (Lewontin 1983; Odling-Smee 1988). In addition, many organisms choose, protect, and provision "nursery" environments for their offspring. On the basis of this kind of evidence, Lewontin (1982, 1983) has argued that the "metaphor of adaptation" should be replaced by a "metaphor of construction" (see also Gray 1988). We have sought to build on Lewontin's writings by exploring the consequences of these constructive processes, which we have collectively termed *niche construction* (Odling-Smee 1988; Odling-Smee, Laland, and Feldman 1996). We argue that through niche construction organisms not only shape the nature of their world, but also in part determine the selection pressures to which they and their descendants are exposed. Other authors in this volume have pursued similar themes (Gray 1988; Griffiths and Gray 1994).

Niche construction is not the exclusive prerogative of large populations, keystone species or clever animals; it is a fact of life. All living organisms take in materials for growth and maintenance, and excrete waste products. It follows that, merely by existing, organisms must change their local environments to some degree. In spite of its universality, niche construction is virtually never incorporated into evolutionary accounts. Niche construction is too obvious and ubiquitous for biologists to be unaware of it existence. If evolutionary biologists currently neglect niche construction, it is unlikely to be because they dispute that it occurs. We suspect that most evolutionary biologists feel that they can afford to neglect the effects of niche construction, because these effects are regarded as either trivial, inconsequential, not liable to change the nature of the evolutionary process, or unlikely to do so sufficiently often to warrant consideration. It is convenient, and also simpler, to regard niche construction as merely the product of natural selection, and not a process shaping selection pressures, because then the