

Excerpt from

## **Précis of *Evolution in Four Dimensions***

by Eva JABLONKA and Marion J. LAMB

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For this Special Issue of INTERS.org on **Theories of evolution: recent developments**, we propose an excerpt of the “Précis of *Evolution in Four Dimensions*”, published by Eva Jablonka and Marion J. Lamb on the authoritative journal *Behavioral and Brain Sciences* (BBS) two years later the release of the first edition of their book *Evolution in Four Dimensions: Genetic, Epigenetic, Behavioral and Symbolic Variation in the History of Life* (The MIT Press, Cambridge, MA and London, UK 2005).

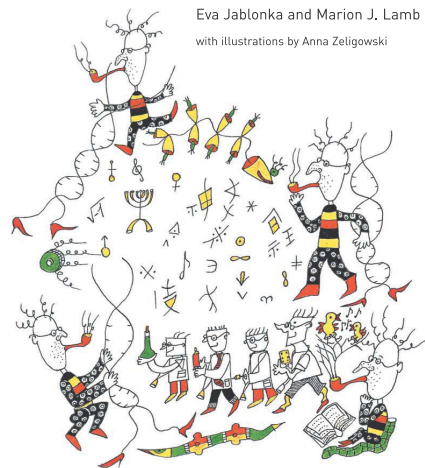
In its original form, as usual for BBS papers, the Précis proposed here was followed by 13 open peer commentaries by influent scholars in many fields (from biology and psychology, to computer science, anthropology, philosophy and medicine) and an authors’ response. We omitted the comments and the response in the present document.

The book *Evolution in Four Dimensions* represents a milestone of the conceptual developments in evolutionary biology from its Lamarckian roots and Darwinian “origin” to the current “Extended Evolutionary Synthesis”. More comments on the role played by this book for the furthering of evolutionary biology can be found in the opening document of this Special Issue.

## **Evolution in Four Dimensions**

Genetic, Epigenetic, Behavioral, and  
Symbolic Variation in the History of Life

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with illustrations by Anna Zeligowski



# Précis of *Evolution in Four Dimensions*

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**Abstract:** In his theory of evolution, Darwin recognized that the conditions of life play a role in the generation of hereditary variations, as well as in their selection. However, as evolutionary theory was developed further, heredity became identified with genetics, and variation was seen in terms of combinations of randomly generated gene mutations. We argue that this view is now changing, because it is clear that a notion of hereditary variation that is based solely on randomly varying genes that are unaffected by developmental conditions is an inadequate basis for evolutionary theories. Such a view not only fails to provide satisfying explanations of many evolutionary phenomena, it also makes assumptions that are not consistent with the data that are emerging from disciplines ranging from molecular biology to cultural studies. These data show that the genome is far more responsive to the environment than previously thought, and that not all transmissible variation is underlain by genetic differences. In *Evolution in Four Dimensions* (2005) we identify four types of inheritance (genetic, epigenetic, behavioral, and symbol-based), each of which can provide variations on which natural selection will act. Some of these variations arise in response to developmental conditions, so there are Lamarckian aspects to evolution. We argue that a better insight into evolutionary processes will result from recognizing that transmitted variations that are not based on DNA differences have played a role. This is particularly true for understanding the evolution of human behavior, where all four dimensions of heredity have been important.

**Keywords:** cultural evolution; Darwinism; directed mutations; epigenetic inheritance; evolutionary psychology; information transmission; Lamarckism; language evolution; memes; social learning

## 1. Introduction

Since its beginning in the early 19th century, the history of evolutionary theory has been a stormy one, marked by passionate and often acrimonious scientific arguments. It began with Lamarck, who 200 years ago presented the first systematic theory of evolution, but it was largely through the influence of Darwin's *On the Origin of Species* (Darwin 1859; henceforth *Origin* in this article) that evolution took center stage as the foremost integrating theory in biology. In the late 19th and early 20th centuries, the theory went through neo-Darwinian, neo-Lamarckian, and saltational upheavals, but eventually it achieved a 60-year period of relative stability through what is commonly known as the Modern Synthesis. The Modern Synthesis, which began to take shape in the late 1930s and has been updated ever since, was a theoretical framework in which Darwin's idea of natural selection was fused with Mendelian genetics. The stability it gave to Darwinian theory was the result of the elasticity biologists allowed it. By giving up some initial assumptions about strict gradualism, by tolerating selective neutrality, by accepting that selection can occur at several levels of biological organization, and by other adjustments, the Modern Synthesis was made to accommodate much of the avalanche of molecular and other data that appeared in the second half of the 20th century.

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One thing that most mid- and late-20th century evolutionists were unwilling to incorporate into their theory was the possibility that the generation of new variations might be influenced by environmental conditions, and, hence, that not all inherited variation is “random” in origin. During the first 50 years of the Modern Synthesis’s reign, Lamarckian processes, through which influences on development could lead to new heritable variation, were assumed to be non-existent. When induced variations eventually began to be recognized, they were downplayed. Developmental processes in general were not a part of the Modern Synthesis, and until recently developmental biology had little influence on evolutionary theory. This is now changing, and as knowledge of developmental mechanisms and the developmental aspects of heredity are incorporated, a profound, radical, and fascinating transformation of evolutionary theory is taking place.

In *Evolution in Four Dimensions* (Jablonka & Lamb 2005; henceforth mentioned as *E4D* in this précis), we followed the traditional 20th-century heredity-centered approach to evolutionary theory and looked at how new knowledge and ideas about heredity are influencing it. We described four different types of heritable variation (genetic, epigenetic, behavioral, and symbolic), some of which are influenced by the developmental history of the organism and therefore give a Lamarckian flavor to evolution. By systematically analyzing and discussing the processes involved, we examined the role and prevalence of induced variations, arguing that they are important and versatile and that the theory of evolution and studies based on it will remain deficient unless they are fully incorporated. Since the book was completed in 2004, a lot of new material has been published, and we refer to some of it in this précis.

We had several aims in writing *E4D*. One was to provide an antidote to the popular DNA-centered view of evolution. Many people have been convinced by eminent popularizers that the evolution of every trait – whether cellular, physiological, morphological, or behavioral – can be and should be explained in terms of natural selection acting on small variations in DNA sequences. In *E4D*, we tried to explore a different and, we believe, better type of explanation, which is based on behavioral ecology, experimental psychology, and cultural studies, as well as modern molecular biology. Because we wanted to catch the attention of lay people who are interested in evolution, we tried to reduce the amount of jargon used and made use of unconventional illustrations and thought experiments to explain our views. We also used the old philosophical device of a dialogue with a “devil’s advocate,” whom we called Ifcha Mistabra (“the opposite conjecture” in Aramaic), to explore the premises and difficulties of the approach we described. Obviously, in this précis for professional scientists we do not try to reproduce these stylistic features of the book.

## 2. The transformations of Darwinism

We started *E4D* with a historical introduction in which we described some of the shifts in ideas that we think are important for understanding how and why biologists arrived at the gene- and DNA-centered view of heredity and evolution that prevails today. We began with

Darwin, who gave his “laws” of biology in the closing paragraph of the *Origin*:

These laws, taken in the largest sense, being Growth with Reproduction; Inheritance which is almost implied by reproduction; Variability from the indirect and direct action of the external conditions of life, and from use and disuse; a Ratio of Increase so high as to lead to a Struggle for Life, and as a consequence to Natural Selection, entailing Divergence of Character and the Extinction of less-improved forms. (Darwin 1859, pp. 489–90)

Darwin’s laws were very general. How reproduction, growth, and inheritance are realized in different biological systems, how variability is generated, and what types of competitive interactions are important, all had to be qualified. Evolutionary biology since Darwin can be seen as the history of the qualification of these processes. As the quotation from the *Origin* makes clear, Darwin included “use and disuse” as a cause of variability: he accepted that there are Lamarckian processes in evolution.

August Weismann’s version of Darwinism, disapprovingly dubbed “neo-Darwinism” by Romanes, is an important part of the history of evolutionary thinking, and its influence can still be seen in contemporary views of heredity and evolution. Unlike Darwin, Weismann gave natural selection an exclusive role in evolution, ruling out change through the inherited effects of use and disuse or any other form of the inheritance of acquired somatic (bodily) characters. His reasons for doing so were partly the lack of evidence, but also the difficulty of envisaging any mechanism through which the inheritance of acquired characters could occur. Certainly, Weismann’s own elaborate theory of heredity and development did not allow it.

Weismann believed that there is a sharp distinction between cells of the soma, which are responsible for individual life, and germline cells, which are responsible for producing sperm and eggs. Only germline cells have all the hereditary determinants necessary for producing the next generation. As Weismann saw it, there was no way in which information from body cells could be transferred to germline cells: He assumed (incorrectly) that development and differentiation involve quantitative and qualitative changes in the cells’ nuclear contents, and that, as far as heredity is concerned, the soma is a dead end.

One of Weismann’s great achievements was to recognize the source of some of the heritable variation that Darwin’s theory of natural selection required. He saw how meiosis and the sexual processes could bring together different combinations of the parents’ hereditary determinants, thereby producing differences among their offspring. However, that still left the problem of the origin of new variants. It surprises many people to discover that Weismann, the great opponent of Lamarckism, thought that the source of all new variation was accidental or environmentally induced alterations in the germline determinants.

Weismann’s ideas and those of his supporters and rivals were debated vigorously during the late 19th century. His elaborate theory of heredity and development was never popular – and turned out to be largely wrong – but elements of it were influential during the foundation of genetics at the beginning of the 20th century and consequently became embedded in the Modern Synthesis. The distinction Weismann made between soma and

germline, his claim that somatic changes could not influence the germline, and his belief that heredity involved germline-to-germline continuity, helped to provide the rationale for studying heredity in isolation from development. One part of Weismann's thinking that was soon forgotten, however, was the idea that new germline variation originates through environmental induction.

In the early 20th century, most of the pioneers of the young science of genetics consciously ignored development and focused on the transmission and organization of genes. The Danish geneticist Johannsen provided the conceptual basis for modern genetics by distinguishing between the genotype and phenotype. The genotype is the organism's inherited potential (the ability to develop various characters), while the phenotype is the actualization of this potential in a particular environment. Hence, the phenotype is by definition the consequence of the interaction between the genotype and the environment. Johannsen's unit of heredity, the gene, was not a representative of the phenotype or a trait, but rather, a unit of information about a particular potential phenotype. Genes were generally assumed to be very stable, although through occasional accidents they changed (mutated) to new alleles. At the time, what a gene was materially was unknown, and how the phenotype was realized was a complete mystery. But for Johannsen and his fellow geneticists, the abstract concept of the gene meant that "Heredity may then be defined as *the presence of identical genes in ancestors and descendants*" (Johannsen 1911, p. 159, his emphasis).

This view of heredity became part of the "Modern Synthesis" of the late 1930s, in which ideas and information from paleontology, systematics, studies of natural and laboratory populations, and especially, from genetics, were integrated into the neo-Darwinian framework. Some of the assumptions on which the Synthesis was based were: (1) Heredity takes place through the transmission of germline genes, which are discrete and stable units located on nuclear chromosomes. They carry information about characters. (2) Variation is the consequence of the many random combinations of alleles generated by sexual processes; usually, each allele has only a small phenotypic effect. (3) New alleles arise only through accidental mutations; genes are unaffected by the developmental history of the organism, and changes in them are not specifically induced by the environment, although the overall rate of change might be affected. (4) Natural selection occurs between individuals (although selection between groups was not explicitly ruled out). Theoretical models of the behavior of genes in populations played a key role in the Synthesis, and Theodosius Dobzhansky, one of its leading figures, proclaimed that "evolution is a change in the genetic composition of populations" (Dobzhansky 1937, p. 11). This view was not shared by everyone, but the voices of embryologists and others who believed that heredity involves more than genes, were seldom heard and were generally ignored by evolutionists.

The advent of molecular biology in the 1950s meant that the Modern Synthesis version of Darwinism was soon being updated to incorporate the new discoveries. At first, these discoveries seemed only to reinforce the basic tenets of the Modern Synthesis. The gene, the unit of heredity, was seen as a sequence of nucleotides in DNA which coded for a protein product that determined some aspect

of the phenotype (or sometimes for an RNA molecule with a functional role in information processing). The seemingly simple mechanism of DNA replication explained the fidelity of inheritance. Information encoded in a gene's DNA sequence was first transcribed into RNA, and then translated into the amino acid sequence of a protein. According to Francis Crick's central dogma, information can never flow from a protein back to RNA or DNA sequences, so developmental alterations in proteins cannot be inherited. This, of course, was soon being seen as a validation of the neo-Darwinian view that "acquired characters" could have no role in evolution. Changes in DNA sequence – mutations – arise only from rare mistakes in replication or from chemical and physical insults to DNA. Although specific mutagens might increase the overall mutation rate, all changes were assumed to be blind to function. As molecular biology developed, DNA began to be seen as more than coded information for making proteins. Because its sequences carry regulatory and processing information that determines which protein is made where and when, DNA assumed a more directive role – it was seen as a plan for development, a program.

Some modifications of the original Modern Synthesis had to be made. It transpired that many variations in the amino acid sequences of proteins (and many more variations in DNA sequences) make no phenotypic difference: some genetic variations seem to be selectively neutral. Moreover, there are genes located in the cytoplasm, which do not obey Mendel's laws. It was also recognized that there are internal processes, such as the movements of "jumping genes" (transposons), that generate mutations. However, the Modern Synthesis version of neo-Darwinism was elastic enough to accommodate these findings.

Modern Synthesis neo-Darwinism took an interesting twist in the 1970s as a result of the attention biologists had been giving to the long-standing problem of the evolution and persistence of "altruistic" traits, which decrease the fitness of the individuals displaying them. For our purposes here, the solutions that were reached are less important than the broader effect the debate had, which was to lead to an even greater focus on the gene not only as a unit of heritable variation, but also as a unit of selection. Richard Dawkins developed and popularized this gene-centered view of evolution in *The Selfish Gene* (Dawkins 1976), and subsequently, it was adopted by most biologists. The gene was depicted as the unit of heredity, selection, and evolution. According to Dawkins, individual bodies live and die, but for evolutionary purposes they should be seen simply as vehicles, as carriers of genes. A gene is a replicator, an entity that is copied in a way that is independent of any changes in the vehicle that carries it, and adaptive evolution occurs only through the selection of germline replicators. Cultural evolution takes place through the spread of cultural replicators, which Dawkins called memes (see sect. 6).

The sketch we have just given shows that the historical route to the present gene-centered view of Darwinism has been evolutionary, in the sense that modifications that happened early on became the basis for what happened later. At an early stage, developmental aspects became vestigial and the significance of the germline grew disproportionately large; this form of the theory eventually became adapted to an environment dominated first by genetics and

then by molecular biology, so that first the gene, and then DNA, was seen as the source of all hereditary information. We believe that recent data and ideas may mean that the gene- and DNA-centered form of Darwinism is heading for extinction, and in *Evolution in Four Dimensions* we have suggested the sort of Darwinian theory that may replace it. It is a theory that sees DNA as a crucial heritable developmental resource, but recognizes that DNA is not the only resource that contributes to heredity. New discoveries in cell and developmental biology and in the behavioral and cognitive sciences mean that it no longer makes sense to think of inheritance in terms of almost invariant genes carrying information about traits encoded in DNA sequences. First, the genome has turned out to be far more flexible and responsive than was previously supposed, and the developmental processes that result in phenotypic traits are enormously complicated. Second, some transmissible cellular variations, including variations that are transmitted through the reproductive cells, are the result of spontaneous or induced epigenetic changes, rather than differences in DNA. Third, for animals, behaviorally transmitted information plays a significant role in evolution. Fourth, as is already well recognized, symbolic culture has powerful evolutionary effects in humans. All types of heritable variations and their interactions with each other and the environment have to be incorporated into evolutionary theorizing. This is particularly important for scientists trying to understand the evolutionary basis of human behavior, who throughout the history of evolutionary ideas have been active and passionate participants in the major debates.

### 3. From genes to development to evolution: A complex relationship

In the early days of genetics, the characters chosen for analysis were largely those that could be interpreted in terms of genes that behaved according to Mendel's laws of segregation. It soon became clear, however, that the relationship between genes and characters is complex: It is not a one-to-one relationship but, rather, a many-to-many relationship. An allelic difference in a single gene can lead to many character differences, and what is seen depends on the external environment, the internal cellular environment, the other alleles present in the genome, and the level at which the analysis is made. Furthermore, several different alleles, often located in different parts of the genome, may, as a combination, collectively affect a character. Often a variation in a single gene makes no difference to the phenotype.

Although these facts became obvious quite early on in the 20th century, the temptation to see a simple causal relation between genes and characters was not resisted. As we are well aware, the idea of simple genetic causality has been politically misused – most horribly by German eugenicists in the 1930s and 1940s, but in other places and at other times too. The attraction of simple linear causation is still present: It is not uncommon to read reports in the popular press about the discovery of a “gene for” obesity, criminality, religiosity, and so on. Many non-geneticists believe that knowledge of a person's complete DNA sequence will enable all their characteristics to be

known and their problems predicted. This widespread belief in “genetic astrology” leads to many unrealistic hopes and fears – fears about cloning and stem cells, for example, and hopes that genetically engineered cures for all individual ills and social evils are just around the corner.

As molecular biology developed, it did at first seem that the relationships between genes and biochemical characters might be simple. A small change in a gene's DNA sequence was seen to lead to a corresponding change in a protein's amino acid sequence, which eventually caused a change in one or more characteristics of the organism. In some of the so-called “monogenic” diseases, for example, a simple DNA change makes a qualitative difference in a protein, which leads to the malfunction of the system of which it is a part. However, it turned out that even in these cases the effects of the DNA change are often context dependent. Sickle cell disease is a paradigmatic example of a small DNA change (a single nucleotide substitution) that leads to an amino acid change in a protein (a subunit of hemoglobin), which results in a large phenotypic change (very severe anemia). Many of the details of how this substitution caused these changes were worked out in the early days of molecular biology. More recent studies have shown, however, that the severity of the disease depends markedly on other factors, including which alleles of other genes are present (Bunn 1997). Some Bedouin Arabs, for example, show only relatively mild symptoms, because they carry an unusual allele of a different gene that counteracts the effects of sickle alleles.

Even at the molecular level, the relationship between DNA, RNA, and proteins has turned out to be vastly more complex than originally imagined. First, most DNA does not code for proteins at all. Only about 2% of human DNA codes for proteins, and the current estimate of the number of protein-coding genes is around 25,000 (about the same as for the mouse – and not many more than for the nematode worm). Second, the RNA products of DNA transcription come in a variety of lengths and organizations. Because of various processes that occur during and after transcription, an RNA transcript often corresponds to several different proteins – sometimes hundreds. Third, much of the DNA that does not code for proteins is nevertheless transcribed into RNA. We know the functions of some of this RNA – it has many, including enzymatic and regulatory ones – but for much of it we are still very much in the dark about what, if anything, it does. Fourth, there are DNA sequences that are not transcribed at all (or so it is believed): Some act as binding sites for regulators, some act as structural elements, and others have no known function and may be genomic parasites. Fifth, DNA can be changed during development. It can be cut up, sewn together, and moved around. Sequences in some cells undergo amplification, or bits are deleted, or they are rearranged, as happens, for example, in the immune system. These are developmental changes, executed by the cell's own genetic-engineering kit. Sixth, not only does RNA have messenger, enzymatic, and regulatory functions, but it can also act as hereditary material which is replicated and passed on from mother cells to daughter cells, including germ cells.

Evelyn Fox Keller (2000) has described how the meaning of the term “gene” changed during the 20th

century, arguing that it had lost much of its clarity. What has happened in molecular biology in the first few years of the 21st century emphasizes this even more (Pearson 2006). It seems that “gene,” the “very applicable little word” coined by Johannsen, can no longer be used without qualification.

What the new knowledge about the relation between DNA and characters shows is that thinking about the development of traits and trait variations in terms of single genes and single-gene variations is inappropriate. It is cellular and intercellular networks (which include genetic networks) that have to be considered. If the effects of small changes in DNA base sequence (classical gene alleles) are highly context dependent and often, when considered in isolation, have, on average, no phenotypic consequences, then the unit underlying phenotypic variation cannot be the classical gene. A shift in outlook is needed. The concept of information in biology, which was inspired by and based on the notion of genes that carry information in their DNA sequences, needs to be changed and cast in more developmental and functional terms (Jablonka 2002). Because it is phenotypes, the products of development, that are selected, and heritably varying phenotypes are the units of evolution, the evolutionary implications of all the developmental resources that contribute to heritable phenotypes have to be considered. Moreover, since it is recognized that regulated DNA changes occur within a generation, the possibility that the mechanisms underlying such developmental modifications may also generate variations that are transmitted between generations cannot be ignored (Shapiro 1999).

Research on the origins of DNA variation challenges the idea that all variations in DNA (mutations) are blind or “random” (see Ch. 3 of *EAD*). The term “random mutation” is a problematic one that is used in several somewhat different ways. It is used to mean that mutations (1) are not highly targeted, that is, that identical (or very similar) changes in DNA do not occur in many different individuals within a population (although there are some “hot spots” in the genome where mutations are more likely than elsewhere); (2) are not developmentally or environmentally induced, that is, that identical changes in conditions do not result in identical mutations; and (3) are not adaptive, that is, that they do not increase the chances that the individuals carrying them will survive and reproduce. Each of these three senses in which mutation has been assumed to be random has been questioned. Mainly as a result of work in microorganisms, fungi, and plants, it is now recognized that some mutations may be targeted, induced, and adaptive.

The flavor of the data coming from this research can be appreciated from a few examples showing that DNA sequence variations can be both highly targeted and condition dependent. Under conditions of genomic stress, such as when two genomes from different sources meet (e.g., when plant hybridization occurs), there can be repeatable and wide-ranging, yet specific, genomic and chromosomal changes (for an eye-opening example, see Levy & Feldman 2004). Because hybridization is thought to be of major importance in plant evolution, the global modifications that hybridization induces are of great interest and importance. Nutritional or heat stress in plants can also lead to specific, repeatable changes in particular DNA

sequences. Certain microorganisms have what look like adaptive stress responses: Data from studies of the mutation rates in bacteria indicate that both the overall mutation rate and the mutation rate of specific genes may be increased in stressful conditions, and that these increases improve their chances of survival. The idea that these are evolved adaptive mechanisms is being actively explored (Caporale 2003). The mechanisms proposed do not make adaptive changes a certainty, but they do increase the chances that a DNA variation generated by the evolved systems that respond to stress will lead to a better-functioning phenotype.

How extensive and significant evolved mutational mechanisms are in animals is not yet clear, mainly because little relevant research has been done. Induced mutational processes are certainly part of the mammalian immune response, and there are hints that stress reactions similar to those found in plants may occur in mammalian germ cells (Belyaev & Borodin 1982), but little is known. Nevertheless, induced mutation is potentially enormously important for humans. If, as seems likely, bacterial pathogens exposed to pharmacological stresses have sophisticated mutation-generating mechanisms that enable them to adapt and survive, then a detailed understanding of these mechanisms is essential if we are to have a chance of combating the growing problem of drug resistance.

#### 4. Epigenetic inheritance

In the first part of *EAD* (Chs. 2 and 3), we showed that the genetic inheritance system, based on DNA, is not as simple as is commonly assumed. Not only is the relationship between variations in DNA sequences and variations in biochemical and higher-level traits more complex, but the idea that all DNA changes arise through random mistakes is wrong. Heredity involves more than DNA, however, and in the second part of *EAD* we looked at heritable variations that have little to do with DNA sequence differences. These variations are described as “epigenetic,” and the systems underlying them are known as epigenetic inheritance systems. Like almost everything else in the biological world, these systems depend on DNA, but, by definition, epigenetic variations do not depend on DNA variations.

The term “epigenetic inheritance” is used in two overlapping ways. First, epigenetic inheritance in the broad sense is the inheritance of phenotypic variations that do not stem from differences in DNA sequence. This includes cellular inheritance (see the second usage), and body-to-body information transfer that is based on interactions between groups of cells, between systems, and between individuals, rather than on germline transmission. Body-to-body transmission takes place through developmental interactions between mother and embryo, through social learning, and through symbolic communication.

Second, *cellular* epigenetic inheritance is the transmission from mother cell to daughter cell of variations that are not the result of DNA differences. It occurs during mitotic cell division in the soma, and sometimes also during the meiotic divisions in the germline that give rise to sperm or eggs. Therefore, offspring sometimes inherit epigenetic variations. In both soma and germline, transmission is through chromatin marks (non-DNA parts of

chromosomes and DNA modifications that do not affect the sequence or code), various RNAs, self-templating three-dimensional structures, and self-sustaining metabolic loops (Jablonka & Lamb 1995).

In *E4D* we treated cellular epigenetic inheritance separately (Ch. 4) from body-to-body information transmission, and divided the latter into transmission through social learning (Ch. 5) and transmission through symbolic systems (Ch. 6). As happens so often in biology, some phenomena did not fit neatly into any of these three categories. In particular, it was difficult to know where to put information inherited through routes such as the placenta or milk, and where to put the ecological legacies that offspring receive from their parents and neighbors. In this précis, we describe these important routes of information transfer in section 4.2.

#### 4.1. Cellular epigenetic inheritance

It is easiest to explain what epigenetic inheritance is about by using its most important and obvious manifestation – the maintenance of determined and differentiated states in the cell lineages of multicellular organisms. Most of the cells in an individual have identical DNA, yet liver cells, kidney cells, skin cells, and so on are very different from each other both structurally and functionally. Furthermore, many cell types breed true: mother skin cells give rise to daughter skin cells, kidney cells to kidney cells, and so on. Since they have exactly the same DNA, and since the developmental triggers that made them different in the first place are usually no longer present, there must be mechanisms that actively maintain their differing gene expression patterns, structural organization, and complex metabolic states and enable them to be transmitted to daughter cells. These mechanisms are known as epigenetic inheritance systems (EISs). Their study is a fast-moving area of research, because not only is epigenetic inheritance a central aspect of normal development, it is also increasingly being recognized as being of great importance in cancer and other human diseases. In addition, it is responsible for the transmission of some normal and pathological variations between generations.

Cellular epigenetic inheritance is ubiquitous. All living organisms have one or more mechanism of cellular epigenetic inheritance, although not all mechanisms are shared by all organisms. In non-dividing cells such as nerve cells, there is no epigenetic inheritance, but there is epigenetic cell memory: Certain functional states and structures persist dynamically for a very long time. This cell memory seems to involve the same epigenetic mechanisms as those that underlie epigenetic inheritance (Levenson & Sweatt 2005).

There are at least four types of EIS:

1. Self-sustaining feedback loops. When gene products act as regulators that directly or indirectly maintain their own transcriptional activity, the transmission of these products during cell division results in the same states of gene activity being reconstructed in daughter cells.

2. Structural inheritance. Pre-existing cellular structures act as templates for the production of similar structures, which become components of daughter cells.

3. Chromatin marking. Chromatin marks are the proteins and small chemical groups (such as methyls) attached

to DNA, which influence gene activity. They segregate with the DNA strands during replication and nucleate the reconstruction of similar marks in daughter cells.

4. RNA-mediated inheritance. For example, silent transcriptional states are actively maintained through repressive interactions between small, transmissible, replicating RNA molecules and the mRNAs to which they are partially complementary.

These four types of EIS are interrelated and interact in various ways. For example, RNA-mediated gene silencing seems to be closely associated with DNA methylation, a chromatin marking EIS, and some chromatin marks may be generated through structural templating processes. The categories are therefore crude, and there are probably other types of non-DNA cellular inheritance as well.

The epigenetic information that a cell receives depends on the conditions that ancestral cells have experienced – on which genes have been induced to be active, which proteins are present, and how they are organized. Passing on induced changes in epigenetic states is crucial for normal development. Unfortunately, transmitting cellular epigenetic changes can also have pathological effects, as it does with some cancers and during aging.

Heritable epigenetic modification sometimes affects whole chromosomes. This is the case in female mammals, where all (or almost all) of one of the two X chromosomes in each cell is inactivated during early embryogenesis, and this state is then stably inherited by all daughter cells in the lineage. Inactivation is brought about by chromatin remodeling and RNA-mediated epigenetic mechanisms. During mitotic cell division, the epigenetic state of the active and inactive X is very stable. However, during gametogenesis the inactive X is reactivated, so the different epigenetic states are not transmitted through meiosis to the next generation.

Sometimes epigenetic states that are mitotically inherited are reset, rather than abolished, during meiosis. A well-known example is genomic imprinting, in which the epigenetic state of a gene, chromosomal domain, or whole chromosome depends on the sex of the transmitting parent (and thus on whether the germ cells undergo oogenesis or spermatogenesis). The chromatin marks on genes inherited from the father are different from those on maternally derived genes, and consequently whether or not a particular gene is expressed may depend on the sex of the parent from which it was inherited. This has had interesting evolutionary consequences (sect. 7), the outcome of which is that when the imprinting system goes wrong in humans, the resulting disorders mainly affect growth and behavioral development (Constância et al. 2004).

With imprinting, the epigenetic state is reset when the chromosome goes through the opposite sex, but there is increasing evidence that some epigenetic variations are neither abolished nor reset during meiosis. They are transmitted and affect offspring, just like DNA variations. Indeed, often they were at first assumed to be conventional gene mutations. The number and variety of examples of these transgenerationally transmitted epigenetic variations is increasing rapidly. One case that we described in *E4D* was that of mice with an epigenetically inherited phenotype that includes yellow coat color, obesity, and a propensity for cancer. The degree of expression of this phenotype is inherited, and is correlated with the chromatin mark

(extent of methylation) associated with a particular DNA sequence. What is interesting about this case is that the phenotypes of offspring (and the underlying marks) can be changed by altering the mother's nutrition during gestation (Dolinoy et al. 2006). Other, comparable cases of induced effects are being investigated. A recent series of experiments with rats has shown how some industrial compounds that are endocrine disruptors can cause epigenetic changes in germline cells that are associated with testis disease states; the changes are inherited for at least four generations (Anway et al. 2005). In humans, Marcus Pembrey and his colleagues (2006) are studying the trans-generational effects of smoking and food supply in the male line, and have concluded from their analysis of body mass and mortality that some mechanism for transmitting epigenetic information must exist.

We could catalogue many more examples of trans-generational epigenetic inheritance in animals, but most of the best examples are found in plants. The scope and evolutionary importance of this type of inheritance in plants is well recognized and is receiving a lot of attention from botanists (e.g., Rapp & Wendel 2005). There may be good evolutionary reasons why plants show so much epigenetic inheritance. In contrast to most animals, where the germline is segregated off quite early in embryogenesis, the germline of plants is repeatedly derived from somatic cells (which is why we can propagate flowering plants by taking cuttings). Consequently, epigenetic states established during the development of the plant soma may sometimes persist and be transmitted to the next generation. This may be of adaptive significance. Animals can adjust to new circumstances behaviorally, whereas plants do not have this option and use non-behavioral strategies. We argued in *E4D* that induced epigenetic changes and their inheritance may do for plants what learnt behaviors and their transmission do for animals.

Although we think that EISs are particularly important in plants, we believe that epigenetic variation is significant in the evolution of all groups, including vertebrates. Unlike most genetic variations, commonly epigenetic variations are induced, are repeatable, are reversible, and often occur at a higher rate than gene mutations. These properties make their effects on evolution very different from those of genetic variations: Evolutionary change can be more rapid and have more directionality than gene-based models predict.

#### **4.2. Developmental endowments and ecological legacies**

It is not clear how much information in addition to that transmitted through DNA sequences is passed to offspring by the germline cell-to-cell route. It used to be assumed that the size of sperm means they can carry little information other than that in DNA, but it is now acknowledged that fathers transmit a lot through the cellular epigenetic routes we have just described. Mothers have additional routes of information transfer through materials in the egg and, in mammals, through the womb and milk. Both parents can also transfer information through faeces, saliva, and smells. The transmission of epigenetic information by body-to-body routes has been recognized in many different species of animals, and also in plants (Mousseau & Fox 1998). In all body-to-body inheritance

of this type, variations are not transmitted through the germline. Rather, offspring receive materials from their parents that lead them to reconstruct the conditions that caused the parents to produce and transfer the material to them, and thus they pass on the same phenotype to their own descendants.

The long-term effects of prenatal conditions and early parental care on human physiology are attracting increasing attention. A mother's nutrition during pregnancy, for example, is known to have profound effects on the health of her offspring when they are adults (Bateson et al. 2004; Gluckman & Hanson 2005). Sometimes the effects are surprising: for example, malnutrition during pregnancy increases the likelihood of obesity and related problems in adult offspring. There are interesting evolutionary theories about why this occurs (Gluckman & Hanson 2005). However, we are more interested in cases in which a phenotype that was induced during early development is later transmitted (or has the potential to be transmitted) to the individual's own offspring and subsequent generations, since it is then justifiable to speak about the "inheritance" of the induced trait. Examples of this type of heredity were recognized in animals many years ago (Campbell & Perkins 1988), and there is now some evidence that it occurs in humans (Gluckman & Hanson 2005). Most cases involve body-to-body transmission through the uterine environment. In *E4D*, we used the example of lines of Mongolian gerbils in which a male-biased sex ratio and aggressive female behavior is perpetuated, probably because the mother's phenotype reconstructs a testosterone-rich uterine environment that induces the same hormonal and behavioral state in her daughters.

Animals continue to receive information from their mother (and sometimes father) after birth. In *E4D* we used the results of experiments with European rabbits to illustrate the variety of routes through which youngsters acquire information about their mother's food preferences. These experiments showed that information is transmitted during gestation (presumably through the placenta or uterine environment), while suckling (either through milk or the mother's smell), and by eating the mother's faeces. The substances transferred enable the young to reconstruct their mother's food preferences. When they leave the burrow, knowing what is good and safe to eat is an obvious advantage.

Even when an animal becomes independent of the direct influences of its parents, it may inherit information from past generations because it occupies an ecological niche that they created. By affecting the development and behavior of animals as they grow up, the nature of the niche created in one generation may lead to the reconstruction of the same type of niche in the next. Odling-Smee et al. (2003) have described many examples of niche-construction activities in groups ranging from bacteria to mammals, and Turner (2000) has given some dramatic examples of sophisticated ecological engineering by animals. The paradigmatic example of niche-construction is the dam built by beavers, and the inheritance and maintenance of the dam and the environment it creates by subsequent generations. Ecological inheritance of this type is the result of developmental processes that are reconstructed in every generation. From the niche-constructing organism's point of view, the ancestrally constructed environment provides it with a developmental resource,



and through its activity, the organism, in turn, bequeaths a similar resource to its offspring.

### 5. Animal traditions: Transmission through socially mediated learning

It is very difficult to erect boundaries between epigenetic and behavioral inheritance. In *E4D*, we classified information transmission through the transfer of substances – a category of inheritance that Sterelny (2004) has called “sample-based inheritance” – with behavioral inheritance, because commonly, body-to-body substance transmission is the outcome of how parents behave. In this précis, we have grouped body-to-body information transfer with germline cell-to-cell epigenetic inheritance, because in both cases, information transfer is through material substances. Both ways of classifying inheritance seem to have legitimacy, although neither is entirely satisfactory.

In Chapter 5 of *E4D*, as well as considering transmission involving the transfer of materials, we looked at the transfer of visual or auditory information through socially mediated learning. No one doubts that socially mediated learning can have long-term, transgenerational effects that can sometimes lead to traditions, but for many years the amount and scope of this type of information transfer in nonhuman animals have been underplayed, and its evolutionary implications neglected. Only recently have animal traditions been given a more central role. There are now a number of new studies (e.g., Hunt & Gray 2003; Rendell & Whitehead 2001; Whiten et al. 2005) and several books about it (e.g. Avital & Jablonka 2000; Fragaszy & Perry 2003; Reader & Laland 2003).

In *E4D*, we distinguished between two types of socially mediated learning – non-imitative and imitation-based social learning – and used some well-known examples to illustrate them. For non-imitative social learning leading to an animal tradition, we used the ability of tits to open milk bottles. In parts of England and elsewhere, this behavior spread rapidly because naïve tits learnt, when in the presence of experienced individuals, that milk bottles are a source of food. A less familiar case is the tradition of opening pine cones and eating the inner kernels that developed in black rats living in Jerusalem-pine forests in Israel. In this case, maternal behavior provides conditions that enable the young to acquire this new and rather complex practice. Another time-honored example is that of the Koshima macaques, who learnt to wash sweet potatoes from an innovative young female. In all of these three cases, imitation was probably not involved – naïve animals learnt what to do from experienced individuals by being exposed to their behavior and its effects, but they did not learn how they did it. They seem to have learnt how to do these things through their own trials and errors, with the social environment providing selective cues and opportunities for learning.

With imitative learning, animals learn both what to do and how to do it by observing the way experienced individuals behave. Humans are great vocal and motor imitators, of course, but vocal imitation is also well developed in songbirds and cetaceans, and these vocal traditions have received a lot of attention. Motor imitation, on the other hand, seems to be much less common, although it is not

clear that there is not some degree of motor imitation in social mammals.

Information transmission by the body-to-body route, whether through substances or through behavior, has very different properties from transmission by the genetic and epigenetic cell-to-cell route. First, with the exception of information transmitted in the egg and, in mammals, in utero (which, with today’s technology, need not be an exception), body-to-body transmission is not always from parents to offspring. Information can be inherited from foster parents and, with imitative and non-imitative social learning, from related or unrelated members of the group or even from other species. Second, with behavioral transmission, in order for a habit, skill, preference, or other type of knowledge to be transmitted, it has to be displayed. There is no latent information that can skip generations as there is with the genetic system. Third, unlike most new information transmitted by the cellular route, new behaviorally transmitted information is not random or blind. What an innovating individual transmits depends on its ability to learn something by trial-and-error or by other methods and to reconstruct, adjust, and generalize it. The potential receiver of information is not a passive vessel, either: Whether or not information is transferred depends on the nature of the information and the experiences of the receiving animal.

In some cases, socially mediated learning may involve a combination of different transmission routes. These can cooperatively and synergistically combine to reinforce and stabilize the behavior pattern. Following Avital and Jablonka (2000), we argued in *E4D* that traditions – behavior patterns that are characteristic of an animal group and are transmitted from one generation to the next through socially mediated learning – are very common. They can affect many aspects of an animal’s life, from habitat choice, to food preferences and food handling, predation and defense, and all aspects of mating, parenting, and social interactions with other group members. Social learning, especially early learning, has very strong, long-term effects, and some traditions are very stable. They can evolve through cumulative additions and alterations, with one behavior being the foundation on which another is built. Different behaviors may reinforce each other, creating a stable complex of behaviors – in other words, a lifestyle. We suggested that such cultural evolution might be partly responsible for complex behaviors, such as bower-building by bowerbirds, which are usually regarded as exclusively a result of the stability of genetic resources.

Social learning that does not involve symbolic communication is as common in humans as in other mammals. Aspects of our food preferences, our choices of habitat and mate, our parenting style, and pair bonding are based on learning mechanisms that we share with other animals. However, in humans, every aspect of life is also associated with symbol-based thinking and communication, particularly through language. Because the symbolic system enables an expansion of information transmission that is so great and so different, we have treated it as a dimension of heredity in its own right.

### 6. Symbol-based information transmission

Similar to other inheritance systems, the symbolic system enables humans to transmit information to others, but in

this special case it also enables humans to communicate with themselves: the symbolic mode of communication is a mode of thought. It permeates everything that humans do, from the most mundane activities to the most sublime.

In *E4D* (Ch. 6), we stressed the special properties of symbolic communication, using the linguistic system as our main example. We defined a symbolic system as a rule-bound system in which signs refer to objects, processes, and relations in the world, but also evoke and refer to other symbols within the same system. Symbolic communication extends the quality, quantity, and range of the information transmitted, and, as symbols are units of meaning (words, sentences, images, vocal units, etc.), they are amenable to combinatorial organization, which can be recursive and theoretically unlimited in scope. However, combinatorial potential is not sufficient for a developed symbolic system: The rules that underlie and organize symbols into a system must ensure that most combinations will not be nonsensical, must allow rapid evaluation (at all levels – truth value, emotional value, action directive), and thus must have functional consequences. The symbolic system of communication enables reference not only to the here and now, but to past, future, and imaginary realities. It profoundly affects behavior by enabling reference to the not-here and not-now. This qualitatively extends the range of possibilities of symbolic communication. Because reference to past and future allows direct references to the relations between causes (past) and effects (present or future), as well as reference to abstract (i.e., logical) relations, symbolic systems enormously extend the potential for transmitting information. They also lead to a requirement for learning, because their own elements and structure undergo updating as the system becomes more sophisticated and is applied to new domains of life and thought.

Language is an excellent example of a symbolic system of communication, but so too are mathematics, music, and the visual arts. The various symbolic systems are, however, different – the type of modularity in each system, the “mobility” of the “units,” and the types of principles binding the system together are not the same and apply to different levels of individual and social organization. Symbolic information, like all information transmitted behaviorally, can be passed to unrelated individuals, but unlike the type of information discussed in the last section, it can also remain latent and unused for generations (most obviously with written words). In the latter respect, as well as in the wealth of variations that are possible, it is like the DNA-based system.

The work of anthropologists and social scientists has shown that cultural evolution rivals DNA-based evolution in its range and complexity. However, the two popular theories that dominate discourse on the evolution of culture – memetics and evolutionary psychology – provide what many see as unsatisfactory explanations of culture and the way it changes. We believe that this is because both are based on neo-Darwinian models of evolution that do not incorporate the developmental aspect of cultural innovation and transmission. Other approaches, such as that taken by Richerson and Boyd (2005), make development much more central and acknowledge the direct effects of developmental learning mechanisms on cultural evolution.

Memetics is a theory of culture which was developed in analogy with, and as an extension of, the selfish gene view

of Richard Dawkins. It is based on the idea that cultural units of information (memes) reside in the brain, are embodied as localized or distributed neural circuits, have phenotypic effects in the form of behaviors or cultural products, and move from brain to brain through imitation (Dawkins 1982). Memes are “replicators” and are comparable to genes. From our perspective, there is one basic problem with the meme concept, and this is that it ignores development as a cause of cultural variation. The assumption that the meme can be seen as a replicator, rather than as a trait that is the result of development, is false. How can a circuit in the brain, which is developmentally constructed during learning, be seen as anything other than a phenotypic trait? If we accept, as we must, that the brain circuit underlying a facet of culture is a developmentally reconstructed trait, then we have to accept that it is sensitive to environmental influences and that acquired (learned) modifications in it (and its many physiological correlates) are transmitted to others. The distinction between cultural “replicators” and cultural “phenotypes” is simply untenable.

Even focusing on “symbolic” memes, which can be communicated without concomitant actions (humans can pass on a command but not implement it), does not solve the problem, because development still cannot be ignored. Symbols and symbolic-system rules must be learnt, and learning is an aspect of development. Most imitation and the use of symbols is not machine-like – it is not blind to function, but is governed by understanding and by perceived goals. It is impossible to ignore the instructional aspects of the generation of new memes, which are central to the symbolic system. We therefore think that although memetics rightly stresses the autonomy of cultural evolution and the complexity of interrelations between memes, it is inadequate as an evolutionary theory of culture because of the false dichotomy that it has created between cultural memes and cultural phenotypes.

We are also critical of most versions of evolutionary psychology. Evolutionary psychologists stress the universal aspects of human-specific propensities and behavior, including cultural behavior. They focus on the genetically evolved basis of the human cultural ability. This, of course, is important. However, it leads to assumptions and inferences about the evolved structure of the mind and the evolved genetic basis of psychological strategies, which we think are very problematical. The main problem is the downplaying of the autonomy of cultural evolution and the conjecture that the diverse behavioral strategies are underlain by specifically selected genetic networks. In *E4D*, we illustrated the problem with a thought experiment that shows how purely cultural evolution could lead to a universal and stable cultural product (literacy) that has all the properties that would indicate to some that it has a specifically selected genetic basis, which it certainly does not.

We conclude that genetic and cultural selective processes are important in human evolution, but they cannot be considered independently from the social construction processes at the individual and group levels that have been recognized and emphasized by the social sciences. Development, learning, and historical construction are central to the generation of cultural entities, to their transmissibility, and to their selective retention or elimination.

## 7. Putting Humpty Dumpty together again: Interactions between genetic, epigenetic, behavioral, and symbolic variations

In the first two parts of *EAD* we described the genetic, epigenetic, behavioral, and symbolic systems of information transfer, stressing the relative autonomy of each. When looking at evolution, an analysis that focuses on a single system of transmission is appropriate for some traits, but not for all. Every living organism depends on both genetic and epigenetic inheritance, many animals transmit information behaviorally, and humans have an additional route of information transfer through symbol-mediated communication. These four ways of transmitting information, with their very different properties, mechanisms, and dynamics, are not independent, and their interactions have been important in evolution. The third part of *EAD* was an attempt to “put Humpty Dumpty together again” by looking at the interrelationships and evolutionary interactions between the different inheritance systems. At present, only a few have been worked out, and even those are only partially understood. However, the cases that have been studied show that there is a surprising richness in the multidisciplinary approach.

We started (Ch. 7) with a discussion of the direct and indirect interactions between the genetic and epigenetic systems. It is obvious that changes in DNA sequences must affect chromatin marks. A mutation changing a cytosine to thymine, for example, may abolish a potential cytosine methylation site. Similarly, changes in control sequences may affect the binding affinity of protein and RNA regulatory elements, and thus directly influence the epigenetic inheritance of states of gene activity. Even greater effects are seen when cells suffer a genomic shock, such as the DNA damage that follows irradiation: For several generations, the descendants of irradiated parents have elevated somatic and germline mutation rates, an effect that has been attributed to induced heritable changes in epigenetic marks on the genes involved in maintaining DNA integrity (Dubrova 2003). In plants, hybridization, another type of genomic shock, causes targeted epigenetic (and genetic) changes at particular chromosomal sites and in certain families of sequences. These sites and sequences are altered in a specific and predictable way, and the modifications are transmitted across generations (e.g., see Levy & Feldman 2004).

Not only do genetic changes affect epigenetic variations, but epigenetic variations affect DNA sequences. Changes in chromatin marks affect the mobility of transposable elements and the rate of recombination, so they affect the generation of genetic variation. Ecological factors such as nutritional stresses or temperature shocks can lead to targeted changes in both chromatin and DNA, and often the epigenetic changes are primary; they probably act as signals that recruit the DNA-modifying machinery (Jorgensen 2004). Direct interactions between the genetic and epigenetic systems seem to be of importance in plant adaptation and speciation (Rapp & Wendel 2005), but ecological and genomic stresses may also have direct effects on the evolution of animals (Badyaev 2005; Fontdevila 2005). The burst of interacting genetic and epigenetic variations that is induced by stress suggests that the rate of evolutionary change may be far greater than is assumed in most models of evolution.

As well as their direct influences on the generation of genetic variation, EISs have enormous indirect effects on evolution through genetic change. Without efficient epigenetic systems that enable lineages to maintain and pass on their characteristics, the evolution of complex development would have been impossible. However, efficient epigenetic inheritance is a potential problem for multicellular organisms, because each new generation usually starts from a single cell – the fertilized egg – and that cell has to have the capacity to generate all other cell types. We believe that past selection of genetic and epigenetic variations that improve the capacity of potential germline cells to adopt or retain a totipotent state may help to explain the evolution of features in development, such as (1) the relatively early segregation and quiescent state of the germline in many animal species; (2) the difficulty of reversing the differentiated state of their somatic cells; and (3) the mechanisms that erase chromatin marks during gametogenesis and early embryogenesis. The evolution of cellular memory necessitated the evolution of timely forgetting!

Not everything is forgotten, however. As we have already indicated, the new embryo does have epigenetic legacies from its parents, including those known as genomic imprints. We think that originally these may have been a by-product of the different ways that DNA is packaged in the sperm and egg, which resulted in the two parental chromosomes in the zygote having different chromatin structures. Some of these differences were transmitted during cell division and affected gene expression, so when and where a gene was expressed depended on whether it was transmitted through the mother or the father. When this was disadvantageous, selection would have favored genes in the parents and offspring that eliminated the differences, but occasionally the difference was exploited. Haig and his colleagues have suggested how the conflicting influences of parents in polygamous mammals may have led to the evolution of imprints and imprinting mechanisms that have effects on embryonic growth and development (Haig 2002). Epigenetic inheritance may also have had a key role in the evolution of mammalian sex chromosomes and some of their peculiarities, such as the relatively large number of X-linked genes associated with human brain development and the overrepresentation of spermatogenesis genes on the X (Jablonka 2004c).

There is a general sense in which the non-genetic inheritance systems can affect genetic evolution. In new environmental conditions, all organisms can make developmental adjustments through cellular epigenetic changes; animals can also make behavioral modifications, and humans can solve problems using their symbolic systems. If conditions persist, natural selection will favor the most well-adjusted phenotypes and the genes underlying them – the genes whose effects lead to a more reliable, faster, developmental adjustment, or the ones with fewer undesirable side-effects. Waddington (1975), whose work we discussed in some detail in *EAD*, coined the term genetic assimilation to describe the process through which natural selection of existing genetic variation leads to a transition from an environmentally induced character to one whose development becomes increasingly independent of the inducing conditions. A more inclusive concept, genetic accommodation, has been suggested by West-Eberhard (2003). Genetic

accommodation includes not only cases in which developmental responses become, through selection, more canalized (less affected by changes in the environment and the genome), but also cases in which they become dependent on different or additional features of the environment, which leads to altered or increased developmental plasticity. We think this concept is valuable, but at the time we wrote *EAD* we had not fully accommodated to it, so we used it more sparingly than we would do now, and framed most of our discussion in terms of genetic assimilation.

Genetic assimilation can occur only when the developmental response is called for repeatedly over many generations, which happens either (i) because the environmental change persists (e.g., a long-lasting climatic change), or (ii) because the organism's activities lead to increased ecological stability (e.g., through a constructed niche such as the beavers' dam), or (iii) through intergenerational epigenetic inheritance. In the last case, the transmitted cellular epigenetic state, behavior, or culture provides the transgenerational continuity necessary to effect significant genetic change.

In Chapter 7 we described several experiments showing how induced cellular epigenetic changes in organisms ranging from yeasts to mammals can reveal previously hidden genetic variation whose selection can lead to evolutionary change. The molecular bases of some of these examples of genetic assimilation have been worked out. In one particularly interesting case in the fruit fly *Drosophila*, the selectable variations that the inducing agent revealed were not previously cryptic genetic variations but new epigenetic variations.

Genetic assimilation can occur not only with environmentally induced changes in form, but also with persistent changes in behavior. In *EAD* (Ch. 8), we described Spalding's old (1873; reproduced in Haldane 1954) but entertaining scenario of a learned response (talking in parrots) that through selection for improvements in learning become an instinct. We went on to show how when previously learned behaviors are genetically assimilated and hence become more "automatic," this may enable the animal to learn an additional pattern of behavior because the former learning effort is no longer necessary. Avital and Jablonka (2000) called this process the assimilate–stretch principle and suggested that it could explain how lengthy and complex sequences of "innate" behaviors have evolved.

As with other learned behaviors, human culture has affected genetic evolution. A well-known example is the way in which the domestication of cattle led to changes in the frequency of the gene that enables adult humans to absorb the milk sugar lactose. As cattle were domesticated, milk became a potential source of energy, but adult humans, like most mammals, cannot break down lactose, so unprocessed milk causes indigestion and diarrhea. Nevertheless, drinking fresh milk has definite advantages in certain populations – most notably those in northern countries, where sunlight is in short supply and vitamin D is therefore scarce. Lactose, like vitamin D, enables calcium (which is plentiful in milk) to be absorbed from the intestine, and hence prevents rickets and osteomalacia. Consequently, in northern countries, people who carried the uncommon allele that enabled them to break down lactose when adult were healthier, and through natural selection this allele became the most

common one. The beneficial effects of milk drinking in northern populations are reflected in their myths, which presumably have an educational value and further encourage the dairying culture and milk-drinking habit.

A good example (which we did not use in *EAD*) of a cultural change that has guided genetic change is the effect of the cultural spread of sign language among congenitally deaf people (Nance & Kearsley 2004). Until the invention and use of sign language, deaf people were cognitively, socially, and economically handicapped, and rarely had children, but once sign language began to be used and they became cognitively adept, many of their social disadvantages disappeared. Naturally, they tended to marry other people with whom they could communicate. As a result of deaf-by-deaf marriage and the improved chances of their surviving and having children, in the United States the frequency of people with the most common type of deafness, connexin deafness, has doubled over the last 200 years. Nance and Kearsley suggest that the evolution of speech in the hominid lineage may have been promoted by a comparable process, in which those with effective oral communication chose others who were similarly endowed and in this way speeded up the fixation of genes affecting speech and speech-dependent characteristics.

Cultural practices probably affected not only the spread of genes underlying oral communication, but also the cumulative evolution of the language capacity itself. In *EAD* we argued that neither the Chomskians nor the functionalists provided a satisfactory explanation of this. The explanation we offered took as its starting point the suggestion that linguistic communication involves the grammatical marking of a constrained set of core categories that describe who did what to whom, when, and how. Following Dor and Jablonka (2000), we argued that the ability to rapidly learn to recognize and mark these categories evolved through partial genetic assimilation. There was a continual interplay between the cultural and genetic systems in which the invention and transmission of linguistic rules that were useful (e.g., the distinction between the categories of one/more-than-one) was at first cultural. Because individuals who had a genetic constitution that made learning the rule more reliable, rapid, and effective had an advantage, partial genetic assimilation occurred. Further linguistic innovation and spread led to more genetic assimilation. Thus, as they accumulated, the basic rules of language became very easy to learn. We believe that this type of process, in which cultural innovation and spread comes first and genetic change follows, has been important not only in the evolution of the language capacity, but also in the evolution of other aspects of human cognitive capacities.

## 8. The evolution of information-transmission systems

We argued in *EAD* that there are four types of heredity system that can produce variations that are important for evolution through natural selection. Some of the variations they transmit seem to be goal-directed: they arise in response to the conditions of life and are targeted to particular functions. In the penultimate chapter (Ch. 9), we looked at the evolutionary origins of these systems that enable "the educated guess" – systems that limit the

search space and increase the likelihood that some of the variations generated will be useful. There is no great mystery about their evolution: they arose through natural selection as a side-effect or modification of functions that evolved for other purposes. For example, stress-induced mutation probably evolved as a modification of mechanisms that were originally selected to repair DNA, and targeted mutation arose through the selection of DNA sequences that are prone to repair and replication errors.

The evolution of epigenetic inheritance systems, which are found in all organisms, must have begun in simple unicellular organisms. Some types of EIS, such as transmission of self-sustaining feedback loops and certain structural elements, would be automatic by-products of selection for the maintenance of cellular structures and functions. With others, such as chromatin marks and RNA-based inheritance, their evolution may have been tied up with the selection of mechanisms for the packaging and protection of DNA, and for defending the cell against foreign or rogue DNA sequences. Once adaptive epigenetic systems were in place in the cell, in certain conditions the ability to pass on their adapted state was an advantage. The environments that would favor transmitting existing epigenetic states to daughter cells are those that fluctuate, but not too often. When environmental conditions change very frequently, cells adapt physiologically; when the change occurs over a very long time-span (hundreds of generations), cells can adapt genetically; but when the changes occur on the intermediate time scale (every 2–100 generations), passing on the existing epigenetic state (having cell memory) is beneficial. Daughter cells get “free” information from their parents and do not have to spend time and energy finding appropriate responses themselves.

Behavioral transmission also results in progeny getting selected useful information from their parents, and is also of advantage in environments that fluctuate. The body-to-body transmission of various substances through the egg, uterus, milk, feces, and so on is probably inevitable, but when it is advantageous to the young, selection would favor genetic changes that made the transmission and the response to it more reliable. Similarly, socially mediated learning is inevitable when youngsters learn in social conditions, but it became a major route of information transfer through selection for paying attention to and learning from those from whom the young can acquire information about what is good to eat, how to find it, how to avoid predators, and so on. In the hominid lineage, the social system resulted in communication traditions that led to selection for genetically better communicators and better ways of communicating. Ultimately, partial genetic assimilation of the ability to learn useful vocal and gestural signs and rules produced the relatively easy-to-learn symbolic systems of human societies.

The origins of all the non-genetic inheritance systems, which sometimes transmit induced and targeted information to daughter cells or organisms, are unexceptional. However, the effects they had were dramatic. We argued in *E4D* that some of the greatest evolutionary transitions were built on new ways of transmitting information, which opened up new ways of adapting to the conditions of life. The transition from unicells to multicellular organisms with several types of cell would be impossible without

quite sophisticated EISs; behavioral information transmission was crucial for the formation of complex social groups; and in the primate lineage, the emergence of symbolic communication led to the explosive cultural changes we see in human societies.

## 9. Conclusions

At the beginning of this précis we suggested that evolutionary theory is undergoing a profound change. Instead of the DNA-centered version of Modern Synthesis Darwinism that dominated the latter part of the 20th century, a new version of evolutionary theory is emerging, in which:

(i) heredity is seen as the outcome of developmental reconstruction processes that link ancestors and descendants and lead to similarity between them. It includes both function-blind replication processes (such as DNA replication) and reconstruction processes that depend on and are determined by function. As Oyama (1985) and Griffiths and Gray (1994) have argued, DNA is a crucial, but not exclusive, heritable developmental resource.

(ii) units of heritable variation are genes (alleles), cellular epigenetic variations (including epialleles), developmental legacies transmitted by the mother during embryogenesis, behavioral legacies transmitted through social learning, symbolic information, and ecological legacies constructed by ancestral generations. All can be thought of as “units” of heredity, although commonly they are not very discrete.

(iii) new heritable variation can be purely fortuitous in origin and blind to function (like most classical mutations), but some is directed, produced as a developmentally constructed response to the environment.

(iv) units of selection or targets of selection are what James Griesemer (2002) terms reproducers. These are entities that display differential reproduction – mainly individuals, but also groups and species, and, in the pre-cellular world, replicating molecules and molecular complexes.

(v) units of evolution are heritably varying types (mainly types of traits), the frequency of which changes over evolutionary time.

(vi) evolution occurs through the set of processes that lead to changes in the nature and frequency of heritable types in a population.

One of the main things we wanted to establish in *E4D* is that there is a wealth of data showing the richness and variety of heredity processes. Epigenetic inheritance is present in all organisms: It is not an unusual and bizarre exception to the rules of heredity, but an important, mainstream, hereditary process. Behavioral inheritance is an uncontroversial mode of information transmission in social animals, and symbols are central to human life and hominid evolution. All these modes of transmission lead to transgenerational phenomena and processes that are of huge practical importance for medicine, for agriculture, for ecology, and for conservation issues. It is clearly not possible to reduce heredity and evolution to genes, not just because the interrelationships are very complicated (which they are), but because of the partial autonomy of different systems of inheritance. Although the view we suggest is in some ways more complex than the

gene-based view, it leads to more realistic and often simpler alternative interpretations of developmental and evolutionary events and processes.

As biologists recognize that the concepts of heredity and evolution have to go beyond DNA and “selfish genes,” and acknowledge that behaviorally and culturally transmitted variations have been significant in the evolution of animals and man, some of their antagonism towards the social sciences may disappear. Incorporating a broader concept of heredity into evolutionary thinking may also help to remove some of the social scientists’ prejudices about biological interpretations of human behaviors and societies. In future, a biologist will need to be more of a social scientist, and a social scientist will need to be more of a biologist.

We predict that in twenty years time, the late 1990s and the first decade of the 21st century will be seen as revolutionary years for evolutionary theory. The effects of the synthesis that is now emerging, and which incorporates development, will be comparable, we believe, to the revolutionary change that followed the introduction of Mendelian genetics into evolutionary thinking during the Modern Synthesis of the late 1930s. Like the former synthesis, the emerging “post-Modern” synthesis is the result of a collective effort. It brings together the mass of information coming from the many branches of molecular biology, developmental biology, medicine, ecology, hybridization studies, experimental studies of behavior, developmental and social psychology, the cognitive sciences, anthropology, and sociology. The new version of evolutionary theory can no longer be called neo-Darwinian, because it includes, in addition to the neo-Darwinian process of selection of randomly generated small variations, significant Lamarckian and saltational processes. Whatever it is called, a new transformed Darwinian theory is upon us.

## References

[The letters “a” and “r” before author’s initials stand for target article and response references, respectively.]

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