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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 nongeneticists 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 *E4D*). 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 *E4D* (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 *E4D* 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 transgenerational 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 transgenerational 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 *E4D* 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 *E4D* 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 *E4D*, 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 *E4D* 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 *E4D* (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 *E4D*) of a cultural change that has guided genetic change is the effect of the cultural spread of sign language among congenitally deaf people (Nance & Kearsey 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 Kearsey 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 *E4D* 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 *E4D* 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

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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 precellular 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.

Open Peer Commentary

Those dreaded memes: The advantage of memetics over "symbolic inheritance"

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Abstract: Jablonka & Lamb (J&L) reject "the dreaded memes," but memetics can explain human uniqueness and culture (as a product of the ability to imitate) without depending on their slippery notion of symbolism. Modern memes show the beginnings of a division into replicators and vehicles, and the replacement of reconstructive processes with systems of blind copying, variation, and selection.

This wonderful book (Jablonka & Lamb 2005) has opened up my thinking on evolution and thrown unexamined assumptions into disarray, for which painful intellectual turmoil I am grateful. The sections on memes are, however, confused and inadequate, and it is these I address here.

Unlike the first three dimensions, the fourth is largely restricted to one species – humans – and so its existence raises questions about the origin of human uniqueness. Jablonka & Lamb (J&L) argue that humans are unique because of the complexity and power of culture, and attribute this uniqueness, as have so many before them, to the acquisition of symbols. For the authors, the turning point in human evolution was something like Deacon's (1997) "symbolic threshold" – once this threshold has been crossed, the argument goes, a species can have symbolic culture that can evolve. This is why they call their fourth dimension the "symbolic inheritance system."

Memetics, by contrast, does not depend on the notion of symbolism. For memetics, the turning point in hominid evolution was the appearance of imitation. Imitation is a kind of copying, and the information that is copied (memes) varies and is selected, which necessarily creates a new evolutionary process. Once this process got underway, the evolving culture could interact with genetic and epigenetic systems to transform the species in which it arose, resulting in modern humans along with their complex and powerful culture (Blackmore 2001). Memes are defined as "that which is imitated" (or more generally, whatever is copied; Dawkins 1976). So although many memes, including words, sentences, or diagrams, are symbolic, others, such as tunes, cars, or hairstyles, are not. Symbolic thought is a result of cultural evolution – not its starting point.

I am suggesting that one advantage of memetics is its ability to explain human uniqueness and culture (as arising from imitation) without getting bogged down in the slippery notion of symbolism, and J&L do get bogged down. Let us consider some of their examples of symbolic culture. They frequently refer to songs and dances, but although words are symbolic, one can copy a dance or tune without any symbols being involved. They also claim that "ideas, artifacts, and institutions are almost entirely based on symbols" (p. 205). But is this so? A lifelike statue is symbolic, but what about an abstract sculpture or painting? An institution such as a bank or hospital has symbolic facets such as paper money or patient records, but buildings are not symbolic, nor are vaults or hospital beds. For J&L, this must surely cause confusion; for memetics, there is no problem. The important questions concern which memes are copied and why – for example, why most hospital beds use a particular design, or why some institutional structures thrive and are copied, even though they are not optimal (Runciman 1998).

Perhaps memetics suffers from far more serious drawbacks. J&L certainly think so, referring to "the dreaded memes" (p. 224). Memetics is, they say, "seductively simple" but flawed: "The flaw stems from the distinction that is made between replicators (memes) and their vehicles (human brains, human artifacts, and humans themselves are all given this role)" (p. 208). The authors refer to Dawkins's treatment of memes in *The Extended Phenotype* (Dawkins 1982, sometimes called Dawkins B) but in his initial formulation in *The Selfish Gene* (Dawkins 1976, or Dawkins A), Dawkins does not claim such a strong distinction, and nor do others who followed him (e.g., Blackmore 1999; Dennett 1995). The confusion is compounded by J&L using the example of tits opening milk bottles, which is generally agreed to have spread by stimulus enhancement rather than imitation (Sherry & Galef 1984), and by their incorrectly describing a "memeplex" as "the set of memes in a brain," rather than as a group of memes that are copied together.

My own view is that the division into replicators and vehicles was a useful development in biological evolution because copying the instructions for making a product is less prone to accumulated error than copying the product directly. So the very efficient system by which genes (instructions) are copied accurately down the germ line while phenotypes (their product) are not directly copied, itself evolved.

Turning to the evolution of memes, we can see the same development happening. Early memes such as dances, songs, spoken language, or ways of making tools were directly (and inefficiently) copied, but now we have production lines, computer programs, printing presses, and Web servers that make multiple copies of

products and accurately copy the instructions for making more. The fact that this development is happening all around us means that memetics can provide insight into how evolutionary processes themselves evolve.

An important question J&L do discuss is whether memes are copied or reconstructed, and the extent to which development, learning, and meaning are involved in the process. I agree that many mathematical models assume a simple copying process and therefore do not accurately reflect the complexity of memetic transmission, but this complexity is no reason to reject memetics. If blind copying is better than the messy processes of development and meaning-sensitive reconstruction, then we should expect to see memetic systems evolving in this direction – and that, I would argue, is precisely what we do see. Compare the spread of gossip through word of mouth with its spread on the Internet. In the former, copying and selection occur together through the low-fidelity processes of listening, learning, recall, and reconstruction. In the latter, the copying is done by high- (almost 100%) fidelity digital processes, and only selection remains meaning dependent. We might expect to see, and may already be seeing, digital selection processes that bypass low-fidelity human processes. Or take J&L's own example of the spread of clothes fashions. Long ago, someone who admired another's dress would have had to find the cloth, draw a pattern, and cut and sew a copy. Now they simply go to the high street and choose from among all the myriad copies accurately produced in factories. All this is much better explained by memetics than by a "symbolic inheritance system," for what does a new dress symbolize?

I find it odd that J&L, who have so effectively revealed the complex, fluid, and messy nature of other evolutionary processes, do not welcome memetics as another way of showing that not all evolution is clean, tidy, and gene-like. This is why Dawkins invented the concept in the first place, and J&L would seem ideally placed to have taken it on.

Genetics and the control of evolution

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Abstract: This book presents a survey of the molecular basis for the genetic control of living organisms and their evolution. The authors consider four dimensions of control over what shapes life forms: genetic, epigenetic, behavioral, and symbolic/cultural. They pay particular attention to the epigenetic realm, and they defend a view recognizing the genetic incorporation of acquired characteristics – a neo-Lamarckian tack.

This book (Jablonka & Lamb 2005) is a well-written effort to deal with a topic that is a bit beyond the realm of familiarity for the average well-educated reader. That realm deals with the molecular underpinnings of evolutionary biology. Early on, evolutionary thinking is introduced in straightforward fashion, with Lamarck, Darwin, and August Weismann all given the recognition they deserve. The authors then present their case, noting how history has tended to leave out some of the aspects of the thinking of each scientist. Gregor Mendel, Hugo de Vries, William Bateson, T. H. Morgan, and the establishment of the field of genetics are all treated in this book, and Theodosius Dobzhansky and Ernst Mayr and the establishment of "the Modern Synthesis" are also dealt with, although here difficulties arise. To illustrate the interpretive twists in the issues presented, each chapter ends with a "dialogue" between a mythical inquisitor, I.M., and the authors, identified as M.E., which stands for the initials of their first names. I.M., in turn, stands for Ifcha Mistabra, which means "the opposite conjecture" in Aramaic, the language of the Biblical Christ.

Eva Jablonka is a professor at the Cohn Institute for the History and Philosophy of Science and Ideas at Tel Aviv University in Israel and is obviously familiar with Semitic languages. When the authors are discussing "Genes and Behavior, genes and language," which is Chapter 8, the example depicted is in English, Hebrew, and Polish to illustrate some of the thinking of Noam Chomsky. The dialogues at the ends of each chapter are thoughtful efforts at articulating some of the various interpretations of the material being presented. The tenth and final chapter is a prolonged I.M. and M.E. dialogue summarizing the issues covered in the whole book. This is followed by 30 pages of chapter notes, which in turn are followed by 30 pages of bibliography, making the whole an admirably documented production. Throughout, line drawings by Anna Żeligowski illustrate the matters being presented. Ifcha Mistabra appears smoking a pipe in the cartoon on the cover, in the Prologue, and at the end of the last page of the text.

Any work that can offer an informed critique of the Universal Grammar outlook of the MIT linguist Noam Chomsky on the one hand, and then, on the other, note that methylated cytosines are very prone to change spontaneously into thymines – and that changing a C to a T in a DNA sequence could have serious consequences – is automatically going to earn the respect of the reader. Jablonka & Lamb (J&L) clearly are in command of a vast literature in linguistics and, if credible, of even more in biology and genetics. They present a view of "evolution in four dimensions": genetic, epigenetic, behavioral, and symbolic/cultural, with the last being particularly important for the human part of the picture. Each of these dimensions gets a chapter, but perhaps the epigenetic chapter is the most crucial.

Epigenetic inheritance systems (EISs) are baffling to us because it is not clear how they work. The cells in each tissue, whether kidney, liver, heart, or whatever, divide to form more cells of the organ in which they are found, although their DNA is the same in each of the different kinds of cell. Presumably this is just the result of the cytoplasm in each cell simply replicating in the daughter cells, whereas the nucleus duplicates itself also but does not control the process.

Four types of epigenetic systems are discussed: self-sustaining or feedback loops; structural inheritance or templated assembly; chromatin-marking systems and their role in DNA methylation; and RNA interference (RNAi) which leads to the silencing of certain genes.

It is in the separation of genetics and epigenetics that there are going to be questions raised about the usefulness of the authors' efforts. Most evolutionary biologists emphasize learning about the genome and its control over the assembly of the organism, and the subsequent effects of selection, and are going to feel uneasy about the tack taken by J&L. The authors critically cite the approach taken by the esteemed John Maynard Smith and his coauthor Eőrs Szathmáry in their 1995 volume, *The Major Transitions in Evolution*, in which the picture of how evolution works is the one accepted by most evolutionary biologists. Genetic variation occurs and is then acted upon by selection to produce evolutionary change. As the authors under review declare, "there is no room in Maynard Smith and Szathmáry's evolutionary ideas for instructive processes, other than in human societies. This, we believe, is a mistake" (Jablonka & Lamb 2005, p. 344). Most of us in this business would disagree. It is true that much is not known about the details of genetic control, but, at the present time, their level of faith seems more like wishful thinking.

J&L also take issue with the views expressed by Ernst Mayr and John Maynard Smith in opposition to the inheritance

of acquired characteristics, and they try to defend a kind of neo-Lamarckian stance. Again, this is something that most working in the field would reject.

Another point the authors do not face is the possibility that a single nucleotide or a codon triplet can often function as a single gene. They mention the recent estimate that the number of genes in the human genome may be as low as 25,000, although they suggest that it also may be "very much higher." It has long been known that there are some 40 human hemoglobin variants inherited as single gene traits, and that each is the result of a single amino acid substitution (Baglioni 1967). Amino acids, in turn, are specified by nucleotide codons, and often by one or two nucleotides. The human genome has 2.9 billion nucleotide base pairs (Wade 2002), although, with the quantity of junk DNA in the genome, most of them are not coding for anything (Britten 1986). Still, that is a huge number of base pairs, and the number of amino acids in a given human being is mind-bogglingly large. If each is produced by a single gene, then the number of genes present has got to be in the many millions at least.

One-generation Lamarckism: The role of environment in genetic development

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Abstract: Environment can provide information used in development – information that can appear to be genetically given and that was previously assumed to be so. Examples include growth of the eye until it achieves good focus, and structuring of receptive fields in the visual cortex by environmental information. The process can be called one-generation Lamarckism because information acquired from the environment is used to structure the organism and because the capacity to acquire this information is inherited.

Jablonka & Lamb (J&L) address the puzzling question of how the same genotype can yield different phenotypes in different environments (Jablonka & Lamb 2005). An extension of this problem is how a genotype can create a well-adapted organism in a variety of environments, and how this can be accomplished in the human with only about 30,000 genes. One of the secrets is that the process of development uses more information from the environment than anyone had envisioned. It is a kind of one-generation Lamarckism: the environment tuning the phenotype in adaptive directions, using genetic systems that assemble information from the environment rather than providing the information themselves. Rather than an inheritance of acquired characteristics, it is acquiring characteristics from the environment, guided by inheritance. Parts of organisms must be competent to use environmental information, though, even if the information is not in the genes.

A relatively simple example of environmental influence on phenotypic development is in the vertebrate eye. The genes could have guided the development and growth of a precise geometry for the eye, forming a focused image just at the retinal surface. This would require a lot of genes working in just the right way, and the last bit of precision would require most of the genetic instruction. Instead, the eye is formed in a general way by the genes, and is made too small. Another genetic mechanism gives the command, "grow until focus is good, then stop." Thus, the eye achieves precise focus without precise instructions (Wallman et al. 1978).

Another use of environmental information became clear when I had the privilege to participate in the discovery of visual cortex receptive fields (RFs) that are far outside the normal range, due to rearing in an abnormal visual environment. Helmut Hirsch of the Stanford University psychology department had raised kittens through the critical period for RF development wearing masks that presented a pattern of large vertical stripes to one eye and large horizontal stripes to the other, focused at optical infinity. Before Hirsch performed behavioral testing for his Ph.D. dissertation, Nico Spinelli of the Stanford Medical School psychiatry department suggested that our team in Professor Karl Pribram's laboratory record RFs from the primary visual cortex of the kittens, using extracellular microelectrodes. Spinelli, Robert Phelps, and I had been recording from single neurons of cat and monkey cortex with an automated method for more than a year (Spinelli et al. 1970).

For our first recordings, Hirsch brought a kitten to our laboratory in a light-tight box (the kittens had been dark-reared except for a few hours of mask exposure per day). Our technique scanned a 25 × 25 degree field with a small moving spot in a raster scan. The first RFs were diffuse and ill-defined. That evening, a RF appeared on our screen unlike any we had ever seen – it was more than 20 degrees long, and monocular. We checked the stimulation apparatus and the recording, and found everything normal. The RF orientation happened to be in the direction of the scan, so a spontaneous burst of firing might have caused the result. We then rotated the scan direction 90 degrees and mapped the field again, but it appeared just as before. We looked at each other, dumbfounded: this kitten's cortex had a completely different organization!

Later that night, we recorded several more such RFs, all monocular, all huge, and all following the orientation of the kitten's mask. Other kittens yielded similar results (Hirsch & Spinelli 1970). The implication is that the cortex had reorganized itself to produce RFs reflecting the structure of the visual world the cortex encountered. It was the first indication that the environment could not only bias the statistics of existing RFs, but could also create completely new ones never seen in nature.

J&L provide a context for examining the implications of this and subsequent results. If a bizarre environment can induce bizarre RFs, what induces normal RFs? The only answer consistent with the fields found in the mask cats is that the environment is doing the same thing in the normal case as in the mask case. The statistics of the environment become reflected in the structure of the visual system's RFs, which therefore become adapted to best code events during the animal's lifetime. According to this idea, the RFs in the cat cortex, and by extension in monkey and human as well, are the way they are because they are tuned by the environment during early development.

The properties of the normal visual world, then, are reflected in the structure of RFs in normal cortex. The world contains contours at all orientations, but more in the horizontal and vertical directions, similar to the RF distribution. In the spatial frequency domain, 1/f power describes both the world and the RF distribution. Motion is ubiquitous in the environment and in RF sensitivities. Like the optics of the eye, the structure of cortical connections is very general, requiring very little genetic information. The high-resolution information comes from the world, not the genes. The system is adapted to adapt.

The arrangement can be described as one-generation Lamarckism because acquired characteristics, from the structural regularities of the environment, come to define what had previously looked like fixated, genotype-directed development. Unlike the original Lamarck proposal, the environmental information does not make it into the next generation, but the genetic strategies supporting the competence to acquire these characteristics do.

We do not know how far one-generation Lamarckism extends. What would be the color sensitivities, for example, of cortical RFs

in animals raised in monochromatic or bichromatic light? We know that we are stuck with genetically specified receptor pigments, but the cortex may be a different story. There's lots of work to do, using what Hirsch calls "environmental surgery" to investigate the extent and importance of environmental influences on phenotype development.

Designed calibration: Naturally selected flexibility, not non-genetic inheritance

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Abstract: Jablonka & Lamb (J&L) have presented a number of different possible mechanisms for finessing design. The extra-genetic nature of these mechanisms has led them to challenge orthodox neo-Darwinian views. However, these mechanisms are for calibration and have been designed by natural selection. As such, they add detail to our knowledge, but neo-Darwinism is sufficiently resourced to account for them.

Evolutionary theory is about design. Organic life demonstrates design at different levels, from the functional fit between the parts of an organism, to the adaptive fit between the whole organism and its environment. There is no getting away from the requirement to explain this. The most successful evolutionary theory to date is neo-Darwinism, which has, at its core, gene-level selection. Jablonka & Lamb (J&L) seek to challenge the explanatory hegemony of gene-level selection with a case for three other sources of intrinsic design. Using diverse evidence and adopting an accessible style, J&L's book (Jablonka & Lamb 2005) is a useful addition to debate. However, we take issue with their interpretation both of the evidence and of evolutionary theory.

At the centre of J&L's thesis is a concern with information. They define information in terms of source and receiver; the input from the source becomes informative if the receiver interprets it. Thus, information denotes a functional relationship between an input and a system – a system will only respond systematically to those inputs for which it is prepared, or designed.

DNA molecules are organised systems that respond to particular inputs, leading to the assembly of polypeptide chains. The nucleotides on a strand of DNA have a one-to-one relationship with RNA nucleotides such that the latter's sequence may be predicted from the former. This specificity is maintained in messenger RNA (mRNA) with a one-to-one correspondence between mRNA sequence and the sequence of amino acids in a protein. From there, the rest unfolds (or folds) with greater complexity. Protein folding and mechanisms underlying gene expression are not fully understood. Views of how DNA works might well be altered in light of new science, but whatever new mechanisms are uncovered, they will doubtless conform to information theory. It might be said that this notion of information is axiomatic.

J&L take issue with neo-Darwinian inheritance. While we might conservatively say that parental nucleic acids are inherited and initiate development, J&L would add that various other factors (epigenetic and behavioural) can alter design later in development in ways not directly captured by the regularities of genetics, and that these factors are inherited differently. The authors proceed by analogy.

DNA replication is compared with photocopying: a process indifferent to content. Photocopying differs from teaching and learning, because different content can be more or less difficult to learn; and, by content J&L can only mean inputs. This content-sensitivity has an affect on what can be transmitted, whereas DNA just replicates and any accidental variation can only be tested through selection, which is wasteful (it is not clear from what perspective this is wasteful). This analogy nudges us to think that some downstream redesigning in light of ecological facts is likely in order to reduce waste; some systems might be content sensitive and calibrate themselves accordingly. And this recalibration, of course, is the role for the other three sources of design that J&L lay claim to.

Downstream recalibration cannot be caused solely by extra-genetic, inherited content. Any content sensitivity that one encounters can only be the consequence of a system that is specifically designed to take and react to inputs. So, one can imagine a complex decision-rule architecture that captures a large number of possible environmental variations, and thus a large number of differing inputs. Organisms in the same species have this architecture set differently according to local environmental idiosyncrasies, which leads to different content-sensitive effects; but we must note that the degrees of freedom for such calibration are finite. This, incidentally, is how learning works (Gallistel 1999).

Calibrating processes are fascinating, and J&L have given us rich detail on a number of candidate mechanisms. One interesting fact about these mechanisms is their codependence of parts and of the whole system to its inputs, which equates to the evidence for design. As such, J&L require a theory of design to account for them. Again, the most successful theory is neo-Darwinism, to which J&L offer no alternative. Minimally, we must say that the finite possible calibrations that can be made by developmental processes reflect facts about the ecology and evolutionary history of a species.

We come now to J&L's description of genetic, mutational change. Irrespective of the nature of the genetic system, a key neo-Darwinian supposition is that genetic change is random with respect to the function selected. In Chapter 3, J&L offer a helpful typology of the kinds of non-random change which have been observed. They describe three tribes whose social structure is by analogy similar to germline mutation strategies: (1) a conservative tribe, members of which stick with what works and devote resources to the maintenance of tradition; (2) a tribe of explorers who foster individual discovery by all members and have no respect for tradition; and (3) a tribe of interpreters who engage in creative enterprise constrained by tradition. Given a certain level and scope of variation, we are led into the intuition that the most successful strategy is the interpreters', which corresponds with a strategy of induced mutations in which random nucleotide change is triggered by specific events and/or in specific genomic regions.

This is an interesting example and the plausibility is further enhanced when J&L remind us that the adaptive value of sex and recombination is studied within orthodox evolutionary circles. But there are several missing elements. First, such evolutionary research is often concerned with questions about levels of selection. A key aim of many theorists has been to explain the evolution of sex in terms of individual-level selection, which has been achieved in the context of the Red Queen hypothesis. So J&L must consider their criterion of plausibility in this light. Might it not be best for individual genomes to tend towards conservative stances? Pleiotropic gene effects, which raise the costs of mutations, would not seem to tilt the scales one way or the other (contra J&L) and might easily be overcome by mutations in specific regulatory domains or in genes with tissue-specific expression. Indeed, the optimal stance adopted might vary between lineages and between parts of the genome. The answer to this question does not seem obvious to us and it may be best to stay close to the evidence (which presently supports

neither fully directed mutations nor an adaptive strategy underlying adaptive induced mutations; see Brisson 2003).

In summary, we welcome J&L's efforts to bring together research in genetics and development in the context of evolution. But, while they discuss many interesting calibrating processes, J&L do not offer any alternative theory of design to explain these. Hence, their book does not amount to a fundamental challenge to the explanatory resources of classical neo-Darwinism.

Evolutionary string theory

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Abstract: *Evolution in Four Dimensions* claims that epigenetic, behavioral, and symbolic inheritance systems should be considered equal partners to genetics in evolutionary biology. The evidence for, and applicable scope of, these additional inheritance systems is limited, particularly with regard to areas involving learning. It is unclear how including these extra dimensions in mainstream evolutionary thinking translates into testable hypotheses for a productive research program.

Evolution in Four Dimensions (*E4D*; Jablonka & Lamb 2005) argues that evolutionary biologists experience only one dimension of evolution – genetics – and proposes that epigenetics, culture, and language are additional evolutionary dimensions. Similarly, we experience four physical dimensions, but string theory in theoretical physics suggests that there are really ten dimensions (Woit 2002). In both cases, the extra dimensions are attractive to theorists, but their experimental value is as yet unproven.

Evolutionary theory's power lies in its breadth. Its scope is such that a microbial ecologist (ADB) and a neuroethologist (ZF) have a meaningful common language. Any major extension to evolutionary theory, which *E4D* aspires to provide, should be broadly applicable to most organisms. This may be why combining evolution with genetics (the Modern Synthesis) and developmental biology (evo-devo) has been successful.

E4D is often irritating in its zeal to demonstrate its thesis that genetics has been overemphasized. It often uses disparaging terms ("genetic astrology," p. 62), and makes argumentative statements (the alleged need to "abandon" the central dogma, p. 153).

E4D makes its strongest case for epigenetic inheritance systems, because they have the greatest potential for generality. Epigenetic inheritance is a cellular phenomenon, and all living things have cells that could be influenced through epigenetic mechanisms. Genetics and epigenetics could be grouped together as "cellular inheritance systems." That there are several heterogeneous epigenetic mechanisms diminishes the prospects for wide applicability, however (indeed, "epigenetics" originated as a catch-all term meaning "not genetics"; cf. Morange 2002).

The evidence for epigenetic inheritance having major impacts on evolution is limited, however. *E4D* frequently uses thought experiments to make its arguments. The concrete examples, such as an epigenetic morph of the flower *Linaria vulgaris* that has been stable for many generations, are more convincing than the thought experiments; and some of the interpretations of the real examples are questionable. Chapter 7 describes experiments in which silver foxes were selected for tameness. This selection for behavior generated morphological changes. It was hypothesized that stress caused the activation of "dormant genes" (Belyaev et al. 1981a; 1981b). As far as we can determine, this hypothesis has not found strong empirical support during subsequent decades of further research (Gulevich et al. 2004; Lindberg et al. 2005; Trut 1999). An alternative hypothesis is that the morphological changes result from selective pressures on genes that have a common influence on both behavior and morphology, which can be tested as part of ongoing research on silver fox genetics (Kukekova et al. 2004; 2007).

Such matters raise practical concerns of how one might predict whether a particular organismal feature is likely to be inherited by epigenetic mechanisms, and if so, by which one. Epigenetic mechanisms also lack clear rules for determining how features will be inherited across generations, in contrast to the clear understanding of genetic inheritance.

Cultural (Ch. 5) and symbolic (Ch. 6) can be grouped together as brain-based inheritance systems. Thus, the book's argument that these should have equal status to genetics in evolutionary theory is immediately weakened, because not only do the organisms involved need brains, they need complex brains with particular properties. The cultural dimension applies only to a very limited number of animals (Whiten & van Schaik 2007), and the symbolic dimension applies only to humans.

To make incorporating brain-based inheritance into evolutionary biology worthwhile, Jablonka & Lamb (J&L) need to show that the similarities of cellular and brain-based inheritance are greater than the differences. But the differences are profound, and trying to put both systems in a common framework obscures more than it reveals. By analogy, a jet plane and a tricycle are both forms of transport. Some aspects of their behavior can be described in common terms: Knowing the velocity of the plane or the tricycle allows one to calculate its distance traveled over a given time, for example. But understanding how a jet plane works requires extensive understanding of aerodynamics; understanding a tricycle does not. Similarly, understanding a brain-based inheritance system will require a deep understanding of neurobiology and ethology; a cellular inheritance system will not.

There is no substantive discussion of nervous systems in *E4D*, which seems to consider animals as generic information processors. Animals are often constrained (Breland & Breland 1961; Wells & Wells 1957) or specialized (Healy et al. 2005) in their cognitive abilities, including what they can learn, because of the particular neural circuitry of each species, which results from selection for capabilities that are relevant to each species' ecology (Healy et al. 2005). As with epigenetics, there are not yet general rules to help us to make strong predictions about which behaviors or symbols are liable to be transmitted across generations, or in which species such behaviors are liable to be important. For example, many animals vocalize, but vocal learning (surely relevant to cultural inheritance) is limited to three orders of birds (songbirds, parrots, and hummingbirds; Baptista & Trail 1992), cetaceans (Deeckea et al. 2000), bats (Boughman 1998), elephants (Poole et al. 2005), and humans. Why is vocal learning present in these taxa, but not others?

The Baldwin effect is mentioned as a way that behavior might influence genetics, but *E4D* admits there have been no experimental tests of these phenomena (p. 311), nor are there suggestions for testing the Baldwin effect (apart from suggesting *Drosophila* as an experimental organism). *E4D* also tries to distance symbolic inheritance from the meme concept, although we struggled, and ultimately failed, to understand why.

E4D is provocative in both the worst and best senses of the word. Despite our many reservations, disagreements, and outright annoyances with this book (provocative in the worst sense), it led us to find interesting research that was new to us, and forced us to consider our theoretical points of view carefully (provocative in the best sense). Ultimately, the book's biggest missing piece is that it does not suggest a research program for empirical evolutionary biologists. In contrast, the "gene's eye

view" (Dawkins 1976; Wilson 1975), which *E4D* criticizes so strongly and at such length, galvanized biology because it generated myriads of testable hypotheses.

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Only three dimensions and the mother of invention

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Abstract: Although the first three dimensions of evolution outlined by Jablonka & Lamb (J&L) are persuasively presented as aspects of evolutionary science, the fourth dimension, symbolic evolution, is problematic: Though it may in some metaphorical sense be happening, there cannot be a science of symbolic evolution. Symbolic evolution essentially involves meaning, which, besides being nonphysical, resolutely resists scientific categorization.

Evolution in Four Dimensions (Jablonka & Lamb 2005) is a beautiful book, but I have a cavil. I am completely sold on the first three dimensions of evolution professed by Jablonka & Lamb (J&L): genetics, epigenetics, and behavior. I even grant that the proposed fourth dimension, the symbolic, has the three essential attributes of evolution: symbols are reproduced, their reproductions inherit some qualities from their ancestral line, and selection determines which reproductions will reproduce again. Nevertheless, honesty demands I explain why, as I see it, there cannot be a science of symbolic evolution.

We take it for granted that there are a finite number of biological species at any given time, and scarcely reflect how crucial this is for the theory of evolution. Imagine instead a continuous series of animals between humans and the chimps, between all hominids, between all mammalian species, and indeed between the different orders of extant organisms. In this continuum-world, Linnaeus would not have seen natural kinds like oaks, maples, pines, firs, and so on, but a confusing profusion of treelike organisms that graded off into other organisms along various dimensions; for example, into bushes and grasses along one dimension and into animals along another dimension, by way of carnivorous plants with digestive tracts and sensorimotor processes for the capture of animals. Even if evolution were occurring, it would be virtually impossible to tell.

The domain of the symbolic is like the continuum-world rather than the actual biological world. Symbols do not come in natural kinds but, instead, in shades of meaning that grade off along various dimensions. Note that when J&L speak of the symbolic dimension of evolution, the term "symbol" must be understood semantically rather than syntactically. What they are interested in, as they say many times, is the cultural transmission of information, because cultural information transfer causally interacts with biological evolution. Information is often transmitted symbolically, as in the sentence, "Snakes are dangerous," which makes us cautious around snakes and so improves our fitness. However, this implies that the syntactically disparate sentence in French, "Les serpents sont dangereux," is the same symbol as the English sentence, for it carries the same information. This example shows that symbols, in the sense J&L intend – and, in fact, require – for the fourth dimension of evolution, are individuated in terms of their semantics (not their syntax): in terms, that is, of their meaning. Sadly, there are no individual species of meaning, but only continuous shades of meaning. There exists no underlying code, no analogue of genes or DNA, and therefore no specific units of meaning that could undergo a process of evolution. In other words, there little hope for a genetics of semantics.

Need I point out that meanings are not physical, and that this poses a problem for physical sciences such as biology? Meanings are transmitted via physical events, certainly, such as the changing of a traffic light from green to red or the saying of words, but they cannot be discovered, even in the most minute scientific investigation of these events. Meaning is bestowed by cultural conventions, which are invisible (even though our physical movements are visible), implicit (upon what we do and say), and indeterminate (just as the cultural boundary between crime and misdemeanor is indeterminate). And even if the cultural conventions were scientifically determinate, what a symbol means is not always determined thereby. You may, for instance, know the rules of bridge, but the meaning of your partner's playing the king of clubs may still be a mystery. Is she flushing out lesser trumps? Or has she violated good strategy to inform you she is bored and would like to go home? We know that the spectrum of starlight may mean to an astrophysicist that the star is a red giant, even though the light left the star before human beings even existed, and before the cultural creation of astrophysics was even dreamt of. Therefore, the information contained is a function of the information the receiver is able to extract via his or her cultural information, whether we consider the playing of a king of clubs (a cultural event), or the light of a star (a merely physical event). Thus, there is no scientific measure of the information contained in a cultural act – the very concept may not be meaningful.

Worse yet, there is no sharp boundary between the literal and the metaphorical. The word "stick" comes from the Old English "sticca," meaning twig. As it happens, twigs will get entangled in sheep's wool and in people's hair, and this primitive form of adhesion eventually led us to speak of tape "sticking" to a surface. Nowadays, we say tape literally sticks to things, but this meaning must have first been a metaphor: Some forgotten poetic genius first used the botanical noun to communicate the adhesive verb. Likewise, sticks of celery and candy were first metaphors inspired by their resemblance to twigs, whereas getting stuck by a pin was a metaphor inspired by pointed twigs' potential for puncture. This seems to be the universal pattern: literal usage is extended – but not literally – into a new domain by metaphor. The literal is just frozen metaphor, but is not literally frozen. Or is it? The "point" I am trying to make is that there is no sharp boundary between the literal and the metaphorical, as I said in the first sentence of this paragraph. You knew what that sentence meant, but did you stop to think that both "boundary" and "sharp" are metaphors? No. Nor is there any sense in doing so. Better that we realize that meaning is the bastard offspring of the literal and the metaphorical, so to speak, and reject the notion that cultural artifacts, words included, have literal meanings waiting to be discovered like phenotypes or genotypes. But this implies that there is no science of meaning, because science is the domain of the literal.

Finally, the human brain, which receives and replicates information, is utterly unlike the relatively monotonous biological mechanisms that copy DNA. The brain is a chaotic, unpredictable, disequilibrium device (Foss 1992; 2000) – the most complex thing in the known universe. Its workings have transformed the world itself in a quantum leap that created pizzas, movies, atomic bombs, lasers, and genetic engineering, all by means of meaning. Meaning is the mother of this explosion of invention, and J&L's metaphorical fourth dimension is this mother of invention. However, she is richer and wilder by orders of magnitude than anything else which science has encountered. She is, for now, one of the boundaries of, rather than one of the dimensions of, biological evolution. Her evolution, by contrast, remains a mere metaphor.

Epigenetic and cultural evolution are non-Darwinian

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Abstract: The argument that heritable epigenetic change plays a distinct role in evolution would be strengthened through recognition that it is what bootstrapped the origin and early evolution of life, and that, like behavioral and symbolic change, it is non-Darwinian. The mathematics of natural selection, a *population-level* process, is limited to replication with negligible *individual-level* change that uses a self-assembly code.

Jablonka & Lamb (J&L) have produced an admirable synthesis in *Evolution in Four Dimensions* (Jablonka & Lamb 2005), showing how processes with vastly different underlying mechanisms constitute important, interrelated facets of evolution. Ironically, although their intent is to highlight Lamarckian aspects of evolution, their framework discourages it. If genetic and cultural evolution were viewed not as components of one big, four-dimensional evolutionary process but as two intertwined evolutionary processes, one primarily Darwinian and the other primarily Lamarckian, there would be no need to rely heavily on genetic assimilation as the means by which behavioral and symbolic systems exert a lasting evolutionary effect. (Behavioral and symbolic systems affect cultural evolution regardless of whether they affect genes.) The focus on genetic assimilation leads to a gradualist scenario for the transition to symbolic thought that is unsupported, as is the contention that symbolic thought followed naturally from possessing a larger brain (p. 304). Leakey (1984) writes of human populations in the Middle East that had brains that were modern in shape and size, but virtually nothing in the way of symbolic culture, and concludes, "The link between anatomy and behavior therefore seems to break" (Leakey 1984, p. 95). This suggests that encephalization was followed by an enhanced capacity to *make use of* a larger brain. To my mind, the most reasonable explanation for the transition to symbolic thought is that genetic mutation facilitated the capacity to subconsciously shift between focused and defocused attention, thereby shifting between analytic thought, which is conducive to logic and symbol manipulation, and associative thought, which is conducive to analogy and "breaking out of a rut" (Gabora 2003). Onset of this capacity would confer upon the mind both hierarchical structure and associative richness conducive to language and other complex tasks. Another hypothesis is that once culturally generated artifacts created sufficient change in the environment, cultural evolution simply snowballed, without any underlying genetic change at all (e.g., Donald 1991; 1993). Explanations such as these that do not rely on genetic assimilation cannot be ruled out.

The authors' reason for treating behavioral and symbolic transmission as distinct dimensions is that behavior must be displayed, whereas symbols can transmit latent information that skips generations (Jablonka & Lamb 2005, p. 202). This distinction breaks down when one considers real transmission among creative individuals operating in different contexts with different abilities. Consider the following simple scenario. Ann pats the cat. Bob, who is sitting in a chair holding a baby, sees this and nuzzles the cat with his foot. Cindy, who sees Bob but not Ann, pats the cat. Thus, the patting skipped a generation. The other rationale given for treating symbols as distinct – that symbols must be taught, whereas behavior need not be – is also not strictly true. In my view, both behavior and symbol use reflect the primarily non-Darwinian cultural evolution of a world-view – the individual's means of internally construing the world and his or her place in it. At any rate, a stronger argument should be made for treating symbols and behavior separately.

Throughout the book, J&L assume that epigenetic, behavioral, and symbolic change proceed through natural selection (a move Darwin himself never made). They speak of "selection of epigenetic variants" (p. 359) and "a change in the parents' behavior that generates a new behavioral variant" (p. 166), and refer to their theory as a "version of Darwinism" (p. 356). However, for a process to evolve through natural selection, inheritance of acquired characteristics must be negligible compared to change resulting from differential replication of individuals with heritable variation competing for scarce resources. What necessitated the theory of natural selection, a theory of population-level change, is that acquired traits are not inherited from parent to offspring at the individual level. In a world in which if a cat bites off a rat's tail, the rat's offspring are not born tail-less, how does one explain how change accumulates? That was the paradox Darwin faced – the paradox for which natural selection provided a solution. There is no such paradox for early life or culture, because they do not replicate using a template, a self-assembly code that is both actively transcribed to produce a new individual and passively copied to ensure that the new individual can itself reproduce. The individual may change, but the passively copied code does not. The mathematical framework of natural selection is not transferable to evolutionary processes that are not code-driven (Gabora 2006). Such processes are correctly described in terms of "actualizing potential" rather than "selecting amongst variants."

I suspect many will find the arguments concerning the key role played by epigenetic processes ultimately unconvincing, because of the paucity of heritable epigenetic change. (How much of what we or Jaynusians [inhabitants of the imaginary planet Janus that J&L refer to at length in their book to illustrate key points] learn or acquire in a lifetime is transmissible through the germ line?) The authors' position could be strengthened by considering recent work which indicates that epigenetic inheritance not only began in simple unicellular organisms (as they rightly point out), but was the means by which early life evolved (Gabora 2006; Vetsigian et al. 2006). Given the book's breadth, it is understandable that the origin of life is considered "outside the scope of this book" (p. 320). However, to me this felt like going on a treasure hunt, peeking down the alley that holds the treasure, and passing it by. When one realizes that there existed a time in which self-organized structure replicated (albeit sloppily) through autocatalysis prior to the onset of template-mediated replication, one appreciates that epigenetic processes are what provided the means by which this primitive structure evolved the genetic code itself.

J&L's contention that epigenetic processes constitute a distinct and important dimension of evolution is indeed strengthened by the realization that they cannot be described by natural selection, which is intimately tied to the genetic code. This also gives us a clear rationale for treating cultural evolution, a non-Darwinian process with behavioral and symbolic components, as distinct from genetic evolution (and the epigenetic processes it grew out of). Indeed, it has been suggested that cultural evolution operates through a mechanism very similar to the one by which early life evolved (Gabora 2004). The evolving entity, the individual's conceptual network, or (from a subjective perspective) internal model of the world, or worldview, is – like a primitive life form – integrated, self-organizing, and self-mending.

What is so informative about information?

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Abstract: Understanding evolution beyond a gene-centered vision is a fertile ground for new questions and approaches. However, in this systemic perspective, we take issue with the necessity of the concept of information. Through the example of brain and language evolution, we propose the autonomous systems theory as a more biologically relevant framework for the evolutionary perspective offered by Jablonka & Lamb (J&L).

In *Evolution in Four Dimensions* (*E4D*; 2005), Jablonka & Lamb (J&L) present an interesting synthesis of evolution that takes a stance against purely gene-centered approaches. By providing evidence of the importance of not only gene-based mechanisms, but also epigenetic, behavioral, and symbolic mechanisms in the establishment of inheritable traits, the authors propose to reconsider evolution as being dependent and effective along these four dimensions. This way, the authors seek to reconcile genetic, developmental, behavioral, and socio-cultural studies under a systemic, comprehensive framework for evolutionary theory. Through interpretative mutations as another mechanism of variation, both Darwinian and Lamarkian aspects find their place in this proposal; we believe it represents a much needed, challenging, and serious attempt at moving forward our understanding of evolution. However, while the authors dispute the gene-centered notion and consider evolution as a systemic multilayered phenomenon, we believe they fall short in one critical aspect: J&L rely heavily on the notion of "information transmission" in a rather loose manner. Their approach is liable to the argument that in order to have any such thing, one needs a transmitter, a message, and a receiver – something that is not easily found when dealing with biological phenomena.

What distinguishes living systems from the rest is a difficult question that can have non-trivial consequences for our understanding of evolution. One influential hypothesis states that living systems are those that maintain organizational closure: they are constituted by networks of self-sustaining processes, regardless of the materials used to instantiate such loops; that is, they are autonomous systems (Maturana & Varela 1973; Varela 1979). When one understands organisms this way, the notion of information transmission becomes less appealing: a closed system cannot "have" information in itself. As *E4D* describes, information transmission depends on the existence of a source, a receiver, and a functional relationship between them. However, although J&L recognize the modulation of source organization and dynamics by the receiver's processes through feedback loops (first-order cybernetics), they fail to incorporate the role of the observer (second-order cybernetics). This step is fundamental if one is to distinguish both ends of the information transmission process. Moreover, the authors state that the "receiver's functional state is changed in a way that is related to the form and organization of the source. There is nothing intentional about the receiver's reaction and interpretation" (Jablonka & Lamb 2005, p. 54). This description is incompatible with systems biology, in which organisms are fundamentally intentional and interpretative, thereby reacting to perturbations precisely according to their internal state. There is nothing informative or valuable in a sucrose gradient until a hungry cell interprets it.

There are, however, several statements in the book that lead us to think that the authors are somehow aware of these aspects. Take the following assertions: "the receiving animal is not just a vessel into which information is poured" (p. 172), "Animals must therefore possess some kind of internal filter – some set of principles or rules" (p. 175), and "what is information for them (animals) may not seem like information to us, and vice versa" (p. 334). All of these quotations point to autonomy in a very direct manner. In fact, the last claim begs the question for the utility of using the lens of information when understanding the properties of the interaction even in communicative actions between animals.

We understand that the use of the concept of information attempts to support the existence of the three non-gene-based systems of inheritance, as a consequence of the discussion about units and levels of selection. However, taking the above considerations into account, and in the context of the evolutionary problem, we believe that inheritance can be understood as the subsistence of an adapted (and adaptive) organization across generations. Considering that DNA, self-sustained cytoplasmic loops, behavior, and so on, transmit information among organisms, has no explanatory value when trying to formulate an evolutionary theory. This process is more easily conceptualized as the reconstruction of a "parent" organization by an "offspring," using multilevel templates (genetic, epigenetic, behavioral, and symbolic), in the context of unpredictable environmental influences. No information metaphor is thus needed. Indeed, when J&L define inheritance as a bias in the reconstruction of an activity or state in next generations, they are emphasizing the active and autonomous role of the offspring in inheriting, without using an information metaphor.

In a quite similar line, Aboitiz (1992) discussed the relevance of development and morphogenesis for a better understanding of adaptive evolution, considering in his proposal Darwinian and Lamarckian mechanisms, as well as behavioral and social levels of organization. We consider that the proposals of *E4D* and Aboitiz are consistent in this respect. Nevertheless, Aboitiz did not use the information transmission analogy, remaining in a purely biological framework. We want to briefly elaborate on this view by looking at the evolution of the nervous system and language.

Perspectives such as generativist grammar, sociobiology, and evolutionary psychology share a common tendency to explain human behavior and brain operations relying heavily on genetic mechanisms. *E4D* presents an alternative view where genetic factors are considered to have relevant, but not exclusive, roles in development. Considering this scenario, we agree with J&L that when talking about a whole-organism operation such as language, it cannot be understood as a genetically determined function, but as a consequence of genetics, development, and culture. Faster language learners evolved thanks to many social, ecological, and morphological conditions, some of which are precisely described by J&L. Aboitiz (1988) also considers genetic factors, but calls attention to the epigenesis of the human brain through the regulation of nerve cell proliferation and the selective stabilization of synapses in a specific context of interactions.

Genetic mutations may contribute to sharpening neural networks involved in specific cognitive processes such as phonological perception and certain aspects of syntactical processing (Aboitiz 1995; Aboitiz et al. 2006), but a large amount of the connectional repertoire may also be shaped by epigenetic processes such as the selective stabilization of neural networks and biomechanical structures within the context of social interaction. In this context, imitative capacities, specified by highly elaborate neural networks already present in nonhuman primates, begin to work as a mode of cultural propagation of adaptive behavioral and symbolic patterns. More specifically, in early proto-humans, the development of a short-term phonological memory system within the framework of an elaborate imitative capacity permitted the acquisition and subsequent elaboration of highly sophisticated proto-linguistic utterances. This short-term memory network eventually expanded, contributing to the establishment of increasingly complex networks generating higher levels of linguistic interaction. Through tight social coupling and sophisticated imitative mechanisms such as the mirror-neuron system, language can evolve without any information being "passed on".

We can see how genetic, epigenetic, behavioral, and symbolic patterns of organization evolved into language through an interplay of conservation and transformation of their properties along generations. Using this brief example, and in the context of our discussion of biological autonomy, we pose the question: Is the analogy of information transmission truly necessary in a new evolutionary framework?

Evolution in the symbolic dimension: The devil is in the details

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Abstract: *Evolution in Four Dimensions* argues convincingly that non-genetic inheritance systems have the potential to be agents of evolution and that, in some circumstances, acquired information can be heritable. However, we found the authors' four-dimensional approach to evolution problematic, and doubt that symbolic evolution can be adequately modeled as a distinct dimension of organismal evolution.

There is growing recognition of the importance of epigenetic and behavioral inheritance systems as potential agents of evolution. However, the evolutionary importance of each system and the relationships among different inheritance systems cannot be rigorously examined or modeled in the absence of a coherent conceptual framework. Jablonka & Lamb's (J&L's) ambitious efforts to construct this framework result in a fascinating and thought-provoking compilation of information and ideas, which together comprise a compelling argument that there may be more to heredity than genes.

The principal success of *Evolution in Four Dimensions* (Jablonka & Lamb 2005) lies in its presentation of a convincing, well-supported argument that traits acquired in an organism's lifetime can under some circumstances be heritable. Although this is no longer a new or particularly controversial idea among many behavioral scientists and ecologists, its presentation in this volume is unusually clear-sighted. The presentation and careful explanation of selected research on epigenetic and behavioral inheritance and the use of thought-provoking examples make it clear that the near-universal anti-Lamarckian bias in the biological sciences is based more on the uncritical acceptance of dogma than a logical interpretation of available evidence.

Unfortunately, J&L are less successful at clarifying the relationships among inheritance systems and their larger implications for evolution and natural selection. While the delineation of four distinct modes of inheritance is helpful as a heuristic device, we find the notion that they function as distinct "dimensions" of evolution, forming a coherent four-dimensional whole, problematic. For most living things, which neither behave nor employ symbols, information can only be transmitted from one generation to the next through genetic and possibly epigenetic inheritance. Therefore, the authors' argument that a four-dimensional approach to evolution is of general applicability is a stretch. More important, the authors' failure to provide broadly applicable definitions for key terms such as *evolution*, *inheritance*, and *information*, and the unexplored assumption that evolution can be reduced to the inter-generational transmission of information, make it difficult to evaluate their four-dimensional model.

We are particularly unconvinced by the argument that behavioral inheritance and symbolic inheritance comprise distinct dimensions of evolution. We agree with the authors that the evolution of language and the ability to explain and interpret our experiences is a key to understanding human uniqueness. However, although the authors outline differences in the ways in which behavioral and symbolic information are coded, a compelling argument that the means of transmission for behavioral and symbolic information are mutually independent is not presented. Is it possible to produce or transmit complex symbolic information without behaving? If not, then can behavior and symbols really be said to represent two distinct dimensions of evolution? Even if we accept the authors' multi-dimensional approach to evolution, describing symbolic inheritance as a special case of behavioral inheritance may be more appropriate.

Symbolic evolution must have included at least two phases: (1) the evolution of the ability to create, acquire, and use symbols (the evolution of "explaining man"), and (2) the ongoing development of symbolic systems. These two steps may have occurred through different processes, and may have different implications for human evolution. The evolution of linguistic beings from nonlinguistic ancestors likely involved an interaction among genetic, developmental, and behavioral processes, which were related to the fitness of the organisms themselves. Although it is not yet clear when or where symbolic communication first appeared, the growing body of fossil, archaeological, and genetic evidence for the recent replacement of other *Homo* species by a new species, *Homo sapiens*, adept in the use of symbolic communication (e.g., Caramelli et al. 2003; Serre et al. 2004; Sokol et al. 1997), demonstrates the potential evolutionary importance of the ability to use language. Indeed, we were disappointed by the authors' failure to include relevant information from fields such as paleontology, archaeology, and neuroscience in this otherwise well-researched volume.

Following the evolution of the human capacity to create, produce, and transmit language, symbolic systems have continued to change and diversify through processes that might more reasonably be described as being independent of genomic or behavioral evolution. In their discussions of the symbolic inheritance system, J&L do not examine the nature of the symbolic entity that is reproduced or the mechanism of competition among variant symbols. The idea that symbolic inheritance represents a fourth dimension of evolution that is complimentary to the other three, cannot be rigorously evaluated in the absence of a clear explanation of what exactly is meant by symbolic evolution and how it relates to the organisms producing the symbols. The latter omission highlights a key difficulty with the authors' argument. If symbolic evolution is completely decoupled from the symbol-producing organisms' fitness and behavior, and transmission is independent of reproduction, then the analogy to biological evolution is an interesting thought exercise, but it would be difficult to support the argument that symbolic variation represents a fourth distinct dimension of biological evolution.

The other main argument presented in the volume, that the production of variation may occur via non-random as well as random processes, has important implications and should be carefully examined. Although the authors provide interesting examples of mechanisms by which appropriate variants may be generated (as opposed to being merely selected) by the interactions of organism with environment, we are not convinced that directed genomic evolution is common. Indeed, the failure of organisms to evolve traits that would be useful seems to be more the rule than the exception, and the diversity of different solutions that evolution has created to the same ecological problems cautions against the interpretation that the environmentally directed generation of useful variants in limited regions of the genome is a more important mechanism of evolution than random variation coupled with selection.

The book uses an interesting twist on standard scientific writing by framing the discussion as an actual dialogue between the authors and a fictional devil's advocate, Ifcha Mistabra. While we found this approach entertaining, this particular devil seemed to function mainly to set up straw men for the authors to knock down, rather than to address serious

challenges to their ideas. We wish that our own critics would be so kind.

Extended evolutionary theory makes human culture more amenable to evolutionary analysis

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Abstract: Jablonka & Lamb's (J&L's) extended evolutionary theory is more amenable to being applied to human cultural change than standard neo-Darwinian evolutionary theory. However, the authors are too quick to dismiss past evolutionary approaches to human culture. They also overlook a potential parallel between evolved genetic mechanisms that enhance evolvability and learned cognitive mechanisms that enhance learnability.

In *Evolution in Four Dimensions* (Jablonka & Lamb 2005, henceforth J&L), the authors do an admirable job of outlining an extended, "four-dimensional" evolutionary theory, one in which inheritance can be non-genetic as well as genetic, organisms are active rather than passive, and variation may be directed as well as blind. Their effort can be placed alongside others, such as niche construction (Odling Smee et al. 2003), developmental systems theory (Oyama et al. 2001), and evo-devo (West-Eberhard 2003), that seek similar expansions, and point towards a new synthesis for not only the biological sciences, but also the behavioural and social sciences.

As J&L note, this extended evolutionary theory is much more amenable to being applied to human cultural change (their "fourth dimension") than the standard neo-Darwinian view. Social scientists who are critical of cultural evolution argue that human culture does not evolve because cultural change is guided or directed (Bryant 2004; Hallpike 1986), because cultural protagonists actively shape their environments (Ingold 2000; 2007), and because cultural inheritance is horizontal/blending (Moore 1994). Many of these objections derive from a lack of knowledge of such processes as epigenetic inheritance (Jablonka & Lamb 1995), niche construction (Odling Smee et al. 2003), horizontal genetic transmission (Rivera & Lake 2004), and adaptive mutation (Rosenberg 2001). Biological and cultural evolution are not as fundamentally different as these critics surmise. As J&L note, "Darwin's Darwinism" – the replicator-neutral, Lamarckian-inheritance version of evolution that Darwin outlined in *The Origin of Species* (Darwin 1859; henceforth *The Origin*) – is closer to their extended evolutionary theory than strict neo-Darwinism. Indeed, if we take "Darwin's Darwinism" as a benchmark, we find that broadly comparable evidence exists for cultural evolution as that which Darwin presented for biological evolution in *The Origin* (Mesoudi et al. 2004). Given these broad similarities between biological (genetic) and cultural evolution, we can profitably borrow tools, methods, theories, and concepts from evolutionary biology to analyse cultural change (Mesoudi et al. 2006), such as phylogenetic analyses (Lipo et al. 2005), population genetic models (Boyd & Richerson 2005), and experimental simulations (Mesoudi 2007).

This, however, leads me to a criticism of J&L's book – that they are too quick to dismiss past evolutionary approaches to human culture, and apply unfair double standards when judging the merits of cultural evolutionary analyses as compared to similar analyses in biology. They dismiss mathematical models of gene-culture coevolution and cultural evolution (Feldman & Laland 1996; Laland et al. 1995) as too heavily based on neo-Darwinian population genetics models and as ignoring developmental/reconstructive aspects of culture (J&L, pp. 205–206), consequently arguing that such models "can provide only limited information about the spread of cultural variants" (J&L, p. 206). In fact, gene-culture coevolution models have significantly improved the understanding of some of the very issues that J&L discuss, such as the coevolution of lactose absorption and dairy farming (Feldman & Cavalli-Sforza 1989; see J&L, p. 293), the conditions under which social learning should be favoured over individual learning (Aoki et al. 2005; Boyd & Richerson 1995; see J&L, p. 158), the consequences of vertical versus horizontal cultural transmission (Cavalli-Sforza & Feldman 1981; see J&L, p. 188) and Lamarckian inheritance (Boyd & Richerson 1985; see J&L, pp. 228–29). That is not to say that such models might not be improved by taking into account factors such as development, as emphasised by J&L, but dismissing them in a single sentence is unjustified. After all, J&L would surely not also dismiss the vast body of population genetic models in biology, which use the same mathematics and simplifying assumptions as gene-culture coevolution models, and which, despite also omitting factors such as development, have nevertheless proved enormously useful (Crow 2001).

J&L also dismiss the concept of the meme, arguing that "it is impossible to think about the transmission of memes in isolation from their development and function" (J&L, p. 209) and that "[in cultural evolution] there are no discrete unchanging units with unchanging boundaries that can be followed from one generation to the next" (J&L, pp. 211–12). Yet a large section of their book is devoted to making identical arguments for genes – that genes are not discrete units with unchanging boundaries, that genes cannot be thought of in isolation from their development and function, and that there is no simple one-to-one mapping between genes and characters. Yet, while the meme concept is dismissed as invalid, the gene concept, which was subject to the same criticisms as the meme, is not dismissed with the same conviction. Perhaps J&L would argue that the terms *meme* and *memetics* carry too much undesirable historical baggage (e.g., an association with "selfish genes"), yet they advocate keeping the term "Lamarckism" (J&L, pp. 360–62) despite similarly negative historical connotations. In my view, far more can be achieved by seeking to improve existing research traditions and concepts than by dismissing them entirely.

Finally, it might be instructive to draw further parallels between the different inheritance systems discussed by J&L. For example, there is a potential parallel between the genetic mechanisms that enhance evolvability (such as increased mutation during times of stress or in regions of the genome that deal with rapid environmental change) and cognitive heuristics that enhance creativity. The latter are learned strategies of learning that increase one's chances of making a useful discovery. These heuristics have been studied experimentally, such as Kaplan and Simon's (1990) "notice invariants" heuristic, in which focusing on aspects of a problem that change the least can increase the probability of a successful solution. Other heuristics have been identified using historical records. For example, Carlson (2000) identified, from Thomas Edison's notebooks, a small number of strategies that Edison repeatedly employed that increased his chances of inventing something successful, such as "simultaneously pursue multiple lines of investigation" or "repeat components in multiple inventions." These cognitive heuristics are the result of prior learning (individual and/or social) that guide future learning in directions that favour successful innovation, in the same way that the genetic mechanisms are the result of prior genetic evolution that guide future genetic evolution in directions that favour adaptive mutation. In addition to following fixed heuristics, however, humans can also actively and flexibly simulate the future, and this "mental time travel" does not appear to have any parallel in genetic evolution. That, however, is a story for another *BBS* article (Suddendorf & Corballis 2007).

Computational cognitive epigenetics

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http://www.biosciences.bham.ac.uk/staff/staff.htm?ID=90

Abstract: Jablonka & Lamb (J&L) refer only implicitly to aspects of cognitive competence that preceded both evolution of human language and language learning in children. These aspects are important for evolution and development but need to be understood using the design-stance, which the book adopts only for molecular and genetic processes, not for behavioural and symbolic processes. Design-based analyses reveal more routes from genome to behaviour than J&L seem to have considered. This both points to gaps in our understanding of evolution and epigenetic processes and may lead to possible ways of filling the gaps.

Jablonka & Lamb's (J&L's) book *Evolution in Four Dimensions* (Jablonka & Lamb 2005) exposes many tangled connections between genome, behaviour, and environment, but it skims over gaps in our knowledge about the information-processing capabilities underlying observed behaviours – ignoring important mechanisms with epigenetic features. Much is said about the physical and chemical mechanisms involved in development, but behavioural competences are described mostly from the outside. Explaining the internal information processing requires the design stance (Dennett 1978).

External behaviours of many animals indicate that they have mechanisms concerned with internal symbolic competences, required for perceiving or acting in structured situations, including planning, predicting, identifying information gaps to be filled, formulating goals, executing plans, learning generalisations, and creatively combining different competences. We need to explain what these competences are, what mechanisms make them possible, how they develop in individuals, and how they evolved. Such competences (in humans and other animals) seem to presuppose something like internal symbolic languages with very specific properties.

When the variety of structurally different combinations of situations and goals rules out preconfigured responses, animals need the ability to represent and make inferences about existing and future configurations and changes; for example, configurations of a partially constructed nest made of interlocking twigs and the affordances (Gibson 1979) for inserting the next twig. This requires internal formalisms for representing structures and possible processes and for constructing, comparing, and planning, including selecting actions from branching collections of possible future sequences. Later, the animal has to produce the actions under the control of the representation. So action sequences linked to complex internal symbolic structures occurred before external linguistic behaviour evolved. Animal behaviours demonstrating such competences include tool-related behaviours (Kacelnik et al. 2006) and the remarkable symbolic competences of the grey parrot Alex (Pepperberg 2001).

Our epigenetic hypothesis about how information-processing develops under the influence of the environment avoids two extreme theories: (1) that all animal competences are somehow encoded separately in the genome, possibly in a large collection of innate modules, and (2) that a small collection of general learning mechanisms (e.g., reinforcement learning) is genetically determined, and everything else is a result of applying those general learning processes. Our "middle way" also synthesizes two apparently opposed views, one expressed by Karmiloff-Smith (1994, p. 693): "Decades of developmental research were wasted, in my view, because the focus was entirely on lowering the age at which children could perform a task successfully, without concern for how they processed the information"; the other by Neisser (1976, p. 8): "We may have been lavishing too much effort on hypothetical models of the mind and not enough on analysing the environment that the mind has been shaped to meet."

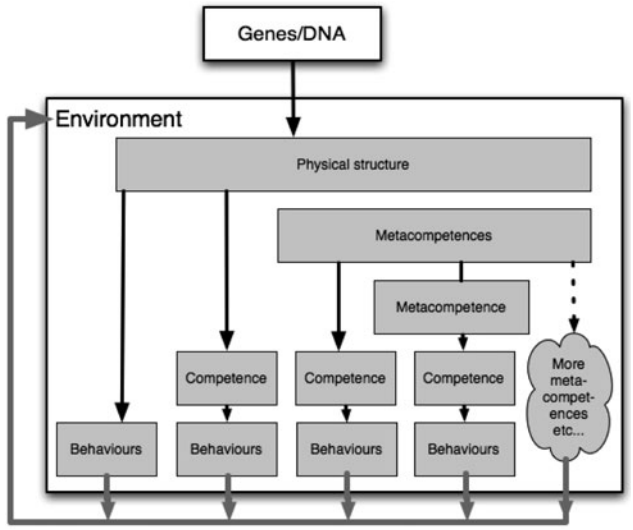

Figure 1 (Sloman & Chappell). The environment (including the body and new brain states) can affect all the processes. There are multiple routes from genome to behaviours, some used only after others have produced new competences and metacompetences. (Based on Chappell & Sloman 2007)

What an individual can learn often changes dramatically during its life, indicating a cascaded development of competences partly under the influence of the environment, including competences to acquire new competences (metacompetences), some of which are themselves the result of interaction of earlier metacompetences with the environment. We summarise this relationship in Figure 1, showing multiple routes from the genome to behaviours of various sorts, with competences at different levels of abstraction and different sorts of specificity developed in different ways at different stages. This implies that learning in some parts of the brain is delayed until others have acquired a layer of competences to build on. So if prefrontal lobes are associated with processes further to the right of the diagram, occurring only after many cycles of simpler development, we would expect prefrontal lobes to develop after low-level visual and motor control mechanisms. Evidence consistent with this conjecture has recently been reported in human infants by Gilmore et al. (2007).

J&L discuss the evolution of language and, like many others (e.g., Arbib 2005), focus mainly on external language used for communication. This assumes that first there were simple forms of language (e.g., gestures and sounds), and complex forms evolved later. In contrast, we suggest that language first evolved for "internal" use. Because some people restrict the label "language" to symbol systems used for external communication, we use the term *g-language* (generalised language) to refer to a wider class that includes internal languages. A g-language allows rich structural variability of various kinds as well as compositional semantics for dealing with novel configurations of objects or processes.

Most people assume that language started simple and external and then grew more complex externally before being internalised. We, like Bridgeman (2005), suggest that complex g-languages evolved in many non-human species and also develop in young children, who cannot yet talk. Internal g-languages are needed to provide forms of representation of current and possible future situations and processes that allow

wide structural variation in what is represented, with compositional semantics to cope with novelty (Sloman 1979). So, rich internal g-languages are precursors to external human languages both in evolution and in child development. After g-languages had evolved for other purposes, including constructing plans that were used to control behaviour, some animals may have started mapping their internal structures onto external behaviours for communication purposes.

Insofar as animals and children can look at different parts of a scene and combine information from most recent saccades with information about parts of the scene that are no longer in view, when planning what to do, they must use representations of spatial organisation of information as well as temporal organisation. In some ways, this requires more complex forms of representation than human spoken languages, combining aspects of verbal language and pictorial languages (analogous to maps, diagrams, and drawings; see also Trehub 1991).

G-languages probably evolved for internal information processing and control of behaviour (through the generation of goals, plans, or instructions), along with generation of questions to specify missing information, and perhaps to formulate hypotheses, explanations, and suppositions. External human language (spoken and gestural) and other symbolically based aspects of human culture (e.g., music, mathematics) also might have built upon these preexisting internal symbolic foundations.

Eventually, instead of a specific g-language, evolution produced competences to acquire a variety of g-languages expressing different kinds of information. This implies that some nonhuman animals' behaviour will be directed and shaped by their internal g-languages, which in turn are shaped by the structure of the external environment, directing evolution down particular paths, and perhaps causing "convergent" evolution of closely related cognitive abilities in birds and mammals with overlapping perceptual and manipulative competences.

If abstract and complex g-language constructs have to be learnt at a late stage of development, but are particularly useful to a species, then some of them could become genetically assimilated or accommodated; in which case they will themselves become heritable and can direct development in particular ways. Environmental cues encountered by these animals will be filtered through their cognitive architecture, thereby tightening the knots between the genome, behaviour, and the environment. Chappell and Sloman (2007) suggest that this employed a separation between parts of the mechanism producing a general class of behaviors and parts that provide parameters that select from that class. The generic competence and the particular parameters might undergo separate trajectories in evolution and development.

If J&L's "assimilate-stretch" principle were extended to cope with the evolution and development of internal g-languages and associated mechanisms, this might be a significant, previously unnoticed, factor in the evolution of cognition. Their examples suggest that assimilate-stretch extends behaviour additively. But qualitatively new capabilities might emerge. For example, if a learned capability becomes genetically assimilated or accommodated, it could form a building block for qualitatively diverse competences. Information that some objects can be deformed by manipulation, can be broken into smaller pieces, can be inserted into spaces, and can, if appropriately assembled, produce fairly rigid structures, might form fundamental parts of a very complex collection of learnable competences, including constructing nests, making or using tools, or extracting objects from containers.

The ideas in this book may turn out to have far-reaching significance for many disciplines. We have tried to show, briefly, how some of that could affect studies of cognition, and internal g-languages, with implications for the evolution of language and many forms of learning. As our cited paper indicates, these forms of development may be required also for intelligent robots that are learning to cope in a wide variety of environments.

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Is symbolic inheritance similar to genetic inheritance?

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Abstract: Jablonka & Lamb's (J&L's) book is refreshing in that it debunks the exclusively gene-centered approach used these days to explain almost anything about life and human behavior. The book is very accessible and most convincing when the authors discuss biological theories of genetic and epigenetic inheritance, but it does not shy away from the more slippery terrain of behavioral and symbolic inheritance, and specifically the origins of language. But is the analogy appropriate?

The gene-centered view on language evolution simply equates genetic evolution with linguistic evolution. It argues that humans genetically inherit a language organ that determines to a large extent the structure of their language and conceptual system, and that this instinctive organ has emerged and been shaped through genetic evolution by natural selection, just like the liver. This strong nativist position is now widely disputed, both on linguistic grounds (the innate universal grammar has remained elusive even after almost 50 years of research) and on biological grounds (it is unlikely that the genome determines at such a fine-grained level the information processing of the brain).

In *Evolution in Four Dimensions*, Jablonka & Lamb (J&L, 2005) survey these counterarguments and then put forward an alternative, more indirect interaction between genetic evolution and language evolution. They argue that the language system is built from neural mechanisms that are generically applicable to a wide range of cognitive functions and that the conventions of a specific language are acquired by general-purpose, socially mediated observational learning mechanisms, possibly with a slight bias established through genetic assimilation. In this scenario, the linguistic system gets copied with variation from adult individuals to the next generation, similar to the way the genome gets copied, but now by cultural instead of genetic means, thus starting a process of cultural evolution (Boyd & Richerson 1985). What was coded as genes according to the language instinct hypothesis becomes a collection of memes transmitted through imitation (Dawkins 1975).

Although I am generally in favor of the cultural hypothesis, I believe there is a major problem with this cultural transmission scenario because of the notion of information that it implies. Information is never simply there, independent of the processes that use that information. We always need to consider the physical structure, which is potentially informative, and the interpreter, who brings out the information and uses it in a specific context. For example, the information in a computer program (which is, after all, just a sequence of switch settings in a computer's memory) only becomes information when it is interpreted by a specific compiler and an operating system, using the primitive machine instructions that the computer can perform. The exact behavior of a running program depends in addition on the specific inputs and prior information states

present at the time of program execution. If none of this is right, the program is totally useless and does not run at all.

Similarly, the DNA macromolecule only becomes information when there is the interpretive machinery that can act upon it. Otherwise, it is a totally dead piece of matter. Just like a computer program, the interpreting process integrates the environment. So in that sense, the genetic system is not just the replicating DNA; it necessarily requires and includes the epigenetic system. Likewise, aspects of a situation in the world or of the behavior of another individual only become informative after an enormously complex process of perception and context-sensitive interpretation has been able to make sense of what is going on. A collection of speech sounds only becomes a carrier of information if there is somebody that can parse and interpret it in a specific context.

The cultural transmission scenario assumes that enough information is present in the perception of behaviors or in language sentences so that the system needed to interpret and reuse them can be copied by imitation from one individual to another. Here lies the difficulty. The imitator/learner must have a sense of what aspects of reality are relevant and what the underlying intentions are before he or she can imitate. The real world is infinitely complex. Without an interpretive capacity in place, the imitator cannot know what exactly needs to be retained in his or her own behavior and when it is appropriate. For example, the tones of a vowel are very relevant in Chinese but are irrelevant in English, so an English speaker trying to imitate Chinese will not properly pay attention to the tonal distinctions, let alone be capable to replicate them.

This is the fundamental paradox for all models of behavioral or symbolic inheritance that rely on imitation or observational learning – to imitate, you must know what counts as information and what is the intention of the producer. So the behavioral or cultural transfer of the interpretive capacity must already have taken place before the imitative act. This is in contrast to the copying mechanism underlying genetic inheritance, which does not need to know anything about what it is copying. This paradox explains why attempts to operationalize imitation in artificial systems have failed, despite a lot of effort (Dautenhahn & Nehaniv 2002). Models of cultural evolution based on imitation appear to assume what they try to explain.

So what is the alternative? Perhaps it is not such a good idea to make the analogy between different forms of inheritance so strong. Szathmáry (2006) draws our attention to a distinction between replicators and reproducers that may be helpful here: Replicators multiply with heredity plus variation, so that selection can act on the population of variants. However, replicators cannot replicate on their own. To copy them, a reproducer is needed – an entity that can do the replication. Genes are replicators but not reproducers; the cell is the reproducer. But because the cell can perform replication of genes, which can then reproduce another cell given the right additional (epigenetic) context, they can also be regarded as replicators. Viruses, in contrast, are replicators but not reproducers themselves, because they need another living cell to reproduce.

This indicates the following analogies and differences between genetic inheritance and symbolic or behavioral inheritance (Steels 2004): Utterances or features of utterances can be viewed as replicators (as in Croft 2000). Every time the same sort of utterance (or feature of an utterance) is produced, it is a replication, unavoidably, with some variation. The reproducer is the speaker's total language system, which might have had to be expanded to achieve the speaker's communicative goals for that utterance. When the utterance is interpreted by the hearer, he or she will have to exercise his or her own total language system, possibly expanding or adjusting it as well. Within this scenario, language systems are not transferred by imitation or observational learning but are actively constructed by speakers and hearers and are aligned to maximize success in communication. With enough interactions, the language system of a speaker will seem to have been transmitted to the hearer; but in fact, the transmission does not at all take place by copying, the way it does for DNA.

The missing chapter: The interaction between behavioral and symbolic inheritance

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Abstract: A strength of Jablonka & Lamb's (J&L's) book lies in its accessible as well as thorough treatment of genetic and epigenetic inheritance. The authors also provide a stimulating framework integrating evolutionary research across disciplines. A weakness is its unsystematic treatment of the interaction between behavioral and symbolic inheritance, particularly in their discussion of language.

In *Evolution in Four Dimensions*, Jablonka & Lamb (J&L, 2005) provide a coherent, unifying evolutionary framework that does not compromise complexity but, instead, embraces it. From our perspective, the meatiest chapters of this book are those on genetic and epigenetic inheritance systems and their interaction. The authors' rich discussion of epigenetic inheritance is fascinating, and their criticism of pure selfish-gene theory compelling. Despite these positive impressions, we found J&L's discussion of behavioral and symbolic inheritance to be less thorough than their comprehensive coverage of genetics and epigenetics, from which we learned so much. Our primary concern is the lack of focus on the interaction between behavioral and symbolic inheritance. This limitation is surprising, given that gene–behavior and gene–language interactions are provided special attention. The relationship between genes and language may be more controversial than that between behavioral and symbolic inheritance. Nevertheless, we argue that behavioral–symbolic interactions are crucial for understanding symbolic language. A discussion of this interaction would have made J&L's four-dimensional framework more complete. In short, there is a chapter missing from this book. In what space we have, we propose some of the material such a chapter could contain.

First, inheritance through behavior-influencing substances may have an analogue in the prenatal auditory experience infants receive in the womb. Research by Shi and colleagues (Shi et al. 1999) suggests that very early auditory perceptual abilities may contribute to grammatical development. Shi et al. studied 1- to 3-days-old infants' responses to two separate classes of words: function words (short, unstressed words such as *will* and *for*) and content words, such as nouns and verbs. Infants detected a change when different words were presented auditorily, but only when the change happened across these classes. Other experiments have demonstrated perceptual sensitivity to the difference between the mother's voice and a stranger's voice in utero (Kisilevsky et al. 2003). J&L argue that maternal diet has chemical consequences that bias a child's early culinary sensitivities. Similarly, one may argue that maternal language has perceptual consequences that bias early linguistic sensitivities. Some may debate the functional significance of this phenomenon, but it is a reasonable possibility that it provides an advantage for children whose prenatal auditory experiences permit early preferences to the mother's language (DeCasper & Fifer 1980; DeCasper & Prescott 1984).

J&L's second behavioral inheritance system, non-imitative social learning, seems also to have a role in early language learning. Some developmental psychologists have argued that socially mediated environmental contingencies promote vocal and social learning that is crucial for later language skill. Watson's (1966; 1985) early work on infants' contingency perception offered a possible basis for emerging social skill. More recently, Goldstein and colleagues (Goldstein et al. 2003) showed that, during early stages of vocal development, social contingencies can affect characteristics of vocalization, including the quality and quantity

of vowel sounds. These contingencies likely emerge in the language-learning environment while the caregiver naturally attends to vocalization by the child (see also Goldstein & West 1999).

Imitation, J&L's third type of behavioral inheritance, likely has some role in language development as well. In fact, numerous authors have argued for an intimate link between imitation and language (e.g., Meltzoff 1988; Tomasello 2003). A great number of studies have suggested a variety of imitative behaviors by young children. Although debate continues about certain studies and their relevance, researchers have reported imitation of basic facial expressions and gestures early in infancy (Meltzoff & Moore 1977), sound productions that reflect the ambient language environment (de Boysson-Bardies & Vihman 1991), and reproduction of novel words modeled by adults (Tomasello & Barton 1994). These imitative actions may be foundational for aspects of language acquisition, including gesture, phonology, and word learning.

As children progress from early word learning to more advanced stages of language use, they seem to rely increasingly on symbolic inheritance to further their language skills. In formal education and informal learning alike, children and adults can learn the meanings of new words through multi-word definitions coming from direct instruction or even reference books. Thus, language knowledge is a product of both behavioral and symbolic inheritance (with the symbolic feeding back onto itself).

As J&L argue, there is a reasonable basis for distinguishing symbolic and behavioral inheritance. We urge, however, that this distinction can be subtle and deserves more attention, particularly when considering language and its acquisition. For example, is language learning transmitted mostly through symbolic inheritance, or through behavioral inheritance? At the early stages of learning, when the most fundamental linguistic conventions are developing, behavioral inheritance seems more relevant than symbolic inheritance. In fact, many have found it challenging to draw a cutoff where a child's language becomes symbolic. Language itself may be symbolic to varying degrees, depending on factors such as the extent to which a lexical item is generalized across environmental contexts, and the extent to which a phrase is conventionalized (Bates et al. 1979; Bybee 2006; Tomasello 2003).

We should note that, scattered throughout J&L's book, one can find reflections similar to those presented here. For instance, the authors discuss Chomskyan and functionalist theories about the role of input in language acquisition; they also suggest genetic adaptations in cognitive faculties that promote language learning. However, they rarely relate these reflections to their behavioral inheritance dimension. A formal discussion of behavioral–symbolic interaction would be useful for multiple reasons. First, behavioral inheritance underlies our ability to transmit symbolic information. Second, symbolic information presumably affects what is transmitted through behavioral inheritance. Finally, behavioral inheritance may be one route through which the genetic and symbolic dimensions interact.

Authors' Response

Bridging the gap: The developmental aspects of evolution

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Abstract: The commentaries on *Evolution in Four Dimensions* reflect views ranging from total adherence to gene-centered neo-Darwinism, to the acceptance of non-genetic and Lamarckian processes in evolution. We maintain that genetic, epigenetic, behavioral, and cultural variations have all been significant, and that the developmental aspects of heredity and evolution are an important bridge that can unite seemingly conflicting research programs and different disciplines.

When writing *Evolution in Four Dimensions* (Jablonka & Lamb 2005; henceforth *E4D*), we often thought about our future readers and tried to engage in a dialogue with them, a dialogue that is reflected in some parts of our discussions with the fictional Ifcha Mistabra, our devil's advocate, which are found at the end of each chapter. We hoped that the book would be read not only by professional biologists, but also by non-biologists, such as psychologists, social scientists, and scholars of culture, who either directly or indirectly draw on evolutionary theory. The commentaries in *BBS* now give us a welcome opportunity to participate in a real cross-disciplinary discussion. As anticipated, most commentators have focused on the relations among heredity, evolution, and development at the behavioral and cultural level. Some doubt that our challenge to neo-Darwinism is necessary, and question the productivity of the Lamarckian perspective and the importance of epigenetic inheritance in evolution; others feel that we did not go far enough. The commentators also refer to our scant discussion of the evolution of cognitive plasticity, question the nature of cultural and behavioral inheritance and their interrelations, highlight the ambiguous and evasive nature of the notion of symbol-based evolution, and present different views about semantic information and the evolution of language. They made us think about areas that we did not explore or did not explore fully, and we are grateful to them.

The two topics that gave us most difficulty when writing *E4D* were finding a satisfactory way of clarifying the notion of semantic biological information, which is a unifying concept in the book, and elucidating the nature of symbols and of symbol-based evolution. Both issues were picked up by several commentators, and we are glad to have the opportunity of saying more about them. But before we do so, we briefly address the more general issues that were raised in the commentaries, which are (1) the relation between developmental plasticity and heredity, (2) our position vis-à-vis terms like neo-Darwinism and Lamarckism, and (3) the evolutionary importance and scope of non-genetic inheritance systems.

R1. The developmental aspects of heredity and evolution

In *E4D* we emphasized developmental aspects of heredity, but our focus and starting point was heredity, not development. We discussed developmental plasticity and environmentally induced variations as they contribute to heredity, concentrating on what can be thought of as temporally extended, intergenerational, developmental plasticity – on the inheritance of environmentally influenced changes. The evolution of plastic responses that are limited to a single generation was not a major topic in the book, although we did discuss (p. 312) the conditions under which plasticity in the classical sense would evolve. It was

the evolutionary effects of the inheritance systems that allow developmentally plastic phenotypes to be transmitted between generations, and the evolution of the mechanisms and processes underlying such intergenerational transmission, that concerned us. Because we focused on heritable developmental variation, the evolution of developmental plasticity itself received scant treatment, and many important issues were left open. We are therefore not surprised that our commentators have addressed the gaps we left.

R1.1. Developmental plasticity and the constraints on adaptation

Bridgeman gives a beautiful example of developmental plasticity that was revealed during studies made in his laboratory. He and his colleagues found that unusual visual experiences in kittens can construct new and previously unseen receptive fields in the visual cortex. This example is comparable to that of the two-legged goat described by Slijper in 1942 and discussed in detail in West-Eberhard's important book, *Developmental Plasticity and Evolution* (West-Eberhard 2003). This now famous goat was born without forelegs, adopted a semi-upright posture, and moved bipedally on its hind legs. After its death in an accident, extensive postmortem examination revealed many coordinated changes in the goat's morphology, such as changed hind leg and pelvic structure, curved spine, unusually thick and large neck, and various functionally correlated changes in its skeleton and musculature. Like the examples of plasticity discussed by Bridgeman, this case shows that pre-existing genetic and developmental capacities allow physiological adaptations that would not have been foreseen and could never have been directly selected for in the past. We can safely assume that walking on their hind legs did not regularly re-occur in the evolutionary history of goats, and that being visually exposed to vertical and horizontal stripes from birth was not part of the evolutionary experiences of cats. The reactions to these conditions of life, which are beyond the normal ontogenetic and evolutionary range of experiences, highlight the way organisms can make phenotypic adjustments to a new set of inputs by reorganizing their anatomy and physiology. Another case, which is of relevance to the evolution of language and which we described in *E4D* (Ch. 9), is the remarkable ability of the bonobo Kanzi to comprehend basic spoken English. The unusual rearing environment in which Kanzi developed led to new communicative behaviors and, we assume, to a novel reorganization of brain activity. West-Eberhard (2003) gives numerous other examples of adaptive plasticity at the morphological, physiological, and behavioral levels. She believes that new plastic responses to changed conditions can be the basis of evolutionary of novelties.

The limits of plasticity, therefore, are not always as narrow as is sometimes assumed. In their commentary, **Dickins & Dickins** claim that the degree of freedom for plastic calibration of the phenotype is very constrained. Constraints certainly do exist, but the type of evidence just outlined suggests that plasticity is surprisingly extensive. Moreover, many plastic responses at different levels can be inherited. Although it is true, as **Faulkes & Baines** note, that not all the "rules" of epigenetics have been unraveled, this does not mean that we can ignore the influence that induced changes may have on evolution. Not only are plastic responses and epigenetic inheritance the basis of cellular differentiation, but plastic responses and the transmission of induced changes also have a role in lineages of both non-behaving organisms, such as plants, and behaving organisms, such as animals and humans.

We consciously decided not to discuss the evolution of the nervous system and neural plasticity in *E4D*, because we wanted to focus on the trans-generational transmission of behavior. However, we recognize, as **Foss** stresses in his commentary, that when discussing animal and human evolution, there is a need to consider the evolution of the nervous systems and neural structures that enable meaning to emerge. In *E4D* we merely looked at the mechanisms and implications of between-generation transmission of the phenotypic products of developmental (non-neural as well as neural) plasticity mechanisms. We return to the problems of the evolution of neural structures underlying meaning in section R3.1.

R1.2. Descriptive terms: Lamarckism, neo-Darwinism, genes, and memes

Bridgeman describes the type of plasticity that he has studied as "acquiring characteristics from the environment, guided by inheritance," a phrase that aptly describes the necessary interplay of both inherited and environmental information in development. However, we cannot support his suggestion that this should be thought of as "one-generation Lamarckism," although we understand the temptation to do so. "Lamarckism" has always been a broad term: Nearly a century ago Delage and Goldsmith wrote, "Lamarckism . . . is not so much a system as a point of view, an attitude towards the main biological questions" (Delage & Goldsmith 1912, p. 244). Nevertheless, Lamarckism does describe a view of evolution that usually incorporates the inheritance of acquired (induced or learnt) characters, not just the inheritance of the mechanisms enabling characters to be acquired. For this reason we would not like to expand it in the way that Bridgeman suggests. In *E4D* we used it in its conventional sense to highlight the evolutionary significance of the inheritance of developmentally induced and regulated variations.

Our use of the term Lamarckism, and our claim that neo-Darwinism, as developed in the Modern Synthesis, is no longer an adequate framework for evolutionary biology, drew understandable fire from **Dickins & Dickins**, **Faulkes & Baines**, and **Brace**. Dickins & Dickins claim that because non-genetic inheritance systems, which they see as mechanisms for calibrating genetic responses, have been designed by natural selection, neo-Darwinism, with its focus on genetic adaptation, can retain its privileged position in evolutionary theory. We certainly agree that there is no alternative to natural selection as an explanation of complex adaptations. However, what we tried to do in *E4D* was not to challenge the explanation of adaptation by natural selection, but rather, to extend the scope of natural selection, and offer a more inclusive framework for thinking about the origin of selectable heritable variation. We maintain, first, that there are more foci for evolution by natural selection than gene-centered neo-Darwinism assumes, because

evolution by natural selection can occur through the selection of heritable variations that are independent of variation in DNA base sequence; and, second, that adaptation and diversity, which are both central to the study of evolution, will be better understood if the processes that generate heritable variation are studied. It is true that incorporating the developmental processes that underlie induction and learning into the evolutionary framework may seem to undermine natural selection, because these factors may, in some conditions, be more powerful than natural selection and lead to the spread of neutral or even deleterious variations. However, it is much more likely that developmentally induced heritable variations will be beneficial, because they are modulations brought about by an already adaptive regulatory system. Furthermore, just as incorporating drift into evolutionary theory does not lessen the importance of natural selection, so also the biases introduced by development and learning do not generally diminish the importance of selection. In fact, as we stressed repeatedly in *E4D*, they tend to extend its applicability.

Brace seems to disagree with our criticism of Maynard Smith and Szathmáry (1995) for generally ignoring the role of developmental variations as direct agents of evolution, and also with our criticism of Mayr's (1982) sweeping claim that there is no place for "soft inheritance" (Lamarckism) in evolutionary thinking. We know that the stance Brace takes is still the majority position, but we do not understand why he adopts it, because he gives no arguments against our point of view other than the argument from authority. Much of our book is a detailed attempt to show that, contrary to the majority position, soft inheritance is of importance in both heredity and evolution. In Chapter 9 of *E4D* we gave several examples which explain why we think that incorporating epigenetic inheritance would have enriched Maynard Smith and Szathmáry's explanations of evolutionary events. We have developed these ideas further in an article commemorating the enormous contribution that Maynard Smith made to evolutionary thinking (Jablonka & Lamb 2006b).

Although he is much more sympathetic towards our way of seeing evolution, **Mesoudi** queries why we stick to problematical terms like "gene" and "Lamarckism" yet reject the useful term "meme." The answer to the gene/meme part of his question is that there is a profound difference in conceptual clarity between memes and genes. Even though the term "gene" is very difficult to define and, like every term that has been around for a long time, carries a lot of historical baggage, we do know a few things about genes (Keller 2000). We know how DNA, the structural basis of genes, is replicated; we know that genes are usually located on chromosomes and that they mutate, are repaired, recombine, and so on. It is useful to have a term that relates to an entity with these properties, however problematic the precise functional definition of that entity may be. What memes are meant to be is far from clear, as even Dawkins has recently acknowledged, although he seems to think this is unimportant (Dawkins 2006, p. 192); and it is not obvious to us what, except a lot of metaphorical baggage, they carry. As for "Lamarckism," we stick to the term for the same reason that we stick to the term "Darwinism": both Darwinism and Lamarckism are historically based umbrella concepts, and their historical baggage is part of their nature and their value (see Jablonka & Lamb 2007).

Lappan & Choe criticize us for our failure to provide definitions of key terms such as evolution, inheritance, and information. In fact, we did define information (*E4D*, p. 54), but it is true that we did not formally define evolution and heredity. We recognize this oversight, and included formal definitions of these and other terms in the Précis.

R1.3. The generality and scope of heritable non-genetic changes

Some commentators, including **Faulkes & Baines** and **Lappan & Choe**, question the generality of the non-genetic inheritance systems, assuming or claiming that we give them all equal weight. In reply we can only repeat what we have said many times: of course the importance of the four inheritance systems is not the same in all organisms. What is true for a human being is not necessarily true for an elephant, and what is true for the elephant is not necessarily true for the bacterium. Bacteria cannot read *BBS*, but many human beings can and do, so surely the evolution of culture, and of the mechanisms of information transmission that underlie it, have to be part of any discussion of why this is so. We certainly do claim that all four inheritance systems need to be incorporated into evolutionary thinking, but obviously only when all four are applicable! At the very end of the "Interim Summary" chapter of *E4D*, in which we compare the different systems of inheritance, we wrote, "Clearly, the different dimensions of heredity and evolution have different significances in different groups, and equally clearly, they all interact" (p. 238). In Chapter 9, where we discuss the evolutionary origin of the different systems of inheritance, we recognize that they have appeared at different times in evolutionary history and so cannot have the same significance in all groups. We tried to capture this idea in Figure 9.6, the legend to which reads, "The evolution mountain: moving up the mountain, there are new types of inheritance systems underlying new types of organism" (p. 341). Obviously behavioral and cultural inheritance cannot apply to non-neural organisms, but we believe that behavioral transmission is important in the evolution of social animals, especially birds and mammals. Fully blown symbol-based evolution seems specific to humans, but surely its uniqueness does not diminish its importance in the past and future evolution of human populations? We also argued in *E4D* (pp. 106–107) that developmentally induced changes in germline DNA ("directed" mutations) have probably not been very significant in multicellular organisms, because the constraints imposed by multicellularity would make even highly targeted mutations have functionally "random effects," but that does not mean that developmentally induced changes in DNA are not significant in the evolution of microorganisms.

Some commentators questioned not only the generality, but also the scope and the importance of the different types of heritable variation we describe. How common and significant is epigenetic inheritance? What is the importance of behavioral inheritance in birds and mammals? How important are developmentally regulated changes in DNA?

Scope and importance are, of course, not synonyms. Some processes may be rare but nevertheless very

important (e.g., single classical mutations with large effects). However, as far as we can gather, epigenetic inheritance is ubiquitous; its extent and range are becoming increasingly evident. Because both in their origin and in their transmission the properties of epigenetic variants are very different from those of genetic variants (see tables on pp. 234 and 236 of *E4D*), we believe that it is inappropriate to lump them together as "cellular inheritance systems," which is how **Faulkes & Baines** suggest they should be treated. It is true, as Faulkes & Baines note, that there are several different epigenetic inheritance systems, although their claim that "epigenetics" and "epigenetic inheritance" originated as catch-all terms (a view that they mistakenly attribute to Morange 2002) is not true. The historical origins and changing uses of these terms have been well documented and discussed in, for example, Van Speybroeck et al. (2002) and Jablonka and Lamb (2007). However, we agree with Faulkes & Baines that it is unlikely that the various types of epigenetic inheritance will all have had the same significance in evolution: Their scope and importance may be very different in different groups. What the growing knowledge about epigenetic inheritance tells us is that we have to take more care in studying heredity, and include (or rule out) the different epigenetic inheritance mechanisms on a case by case basis. This does not diminish the fundamental significance of variants transmitted through epigenetic inheritance; it just makes their study, at the present time, rather difficult and labor consuming.

The evidence that was summarized at book length by Avital and Jablonka (2000) suggests that what in *E4D* we called "behavioral inheritance" is very common among social animals. We therefore dispute **Faulkes & Baines**'s claim that the scope of behavioral inheritance is very limited, although it is true that certain types of cultural inheritance, for instance, those based on sophisticated cognitive abilities, are rare. They are as rare as these cognitive abilities themselves are (Whiten & van Schaik 2007). However, we do not see this as a good reason for grouping behavioral transmission with symbolic transmission and calling them both "brain-based inheritance," as Faulkes & Baines suggest. As our summary tables (pp. 234 and 236 of *E4D*) show, they have different properties. It is true, as **Lappan & Choe** insist, that transmitting symbolic information must involve behavior. However, different symbol-based information can be transmitted through the same behavior, just as different heritable epialleles can be carried by identical DNA sequences. This is why we can distinguish between behavioral and symbolic variation, and between genetic and epigenetic variation.

In *E4D* we did not attempt to review the already vast and rapidly growing literature on non-genetic inheritance, and we cannot attempt to do so here. We recognize the importance of evidence, of course, and give some additional examples and references in the Précis of our book. One of us (Jablonka) is currently preparing a review in which she intends to summarize the evidence about epigenetic inheritance, and from the information already collated it is clear that the number of well-studied examples has reached triple figures. All we can do here is point to some more articles that give an idea of the range of non-genetic heredity phenomena to which we have referred. Information about the transmission of cellular epigenetic variations, particularly through the chromatin marking and RNA-mediated systems, is abundant: extensive reviews of cellular epigenetic inheritance in plants and its significance for understanding ecological and evolutionary processes are presented, for example, in Grant-Downton and Dickinson (2005; 2006), Rapp and Wendel (2005), Takeda and Paszkowski (2006), and Zilberman and Henikoff (2005). Recent examples of epigenetic inheritance in animals are to be found in Chan et al. (2006), Chong and Whitelaw (2004), Rassoulzadegan et al. (2006), Richards (2006), and Vastenhouw et al. (2006). There is now evidence that in microorganisms such as yeast and *Podospora*, prion variants are transmitted through meiosis (Tuite & Cox 2006; Wickner et al. 2004), and examples of transmitted self-sustaining loops have been discovered and studied in microorganisms (Casadesús & Low 2006; Rando & Verstrepan 2007; Zordan et al. 2006).

Avital and Jablonka (2000) have given many examples showing that information is transmitted through maternal substances that effect embryonic development, as well as through the effects of milk, feces, saliva, and various forms of maternal and paternal behavior on the newborn. A fascinating case of behaviorally mediated inheritance in the rat has now been described in molecular detail (Meaney 2001; Weaver et al. 2004). The importance of the effects of transmitted substances and early behavior on human embryonic and postnatal development is increasingly well recognized (Gluckman & Hanson 2005; Gluckman et al. 2007).

Taken together, the data suggest that one would be hard pressed to think of a biological feature that cannot be influenced by some type of developmentally responsive inheritance mechanism, be it epigenetic, behavioral, or symbolic. As **Faulkes & Baines** rightly claim, these modes of inheritance have not yet been incorporated into mainstream evolutionary thinking, but this is not surprising. Ever since the beginning of the 20th century, when genetics began to be unraveled, evolutionary thinking seems to have lagged 15 to 20 years behind the findings from experimental studies of heredity. The same seems to be happening today, although as we describe in section R4, evolutionary biologists are beginning to incorporate information about non-genetic inheritance into their models and experimental studies.

Another issue which worries some commentators is the status of developmentally induced and regulated DNA changes that are transmitted from one generation to the next. **Lappan & Choe** and **Dickins & Dickins** raise doubts about the importance of such mutations in evolution. This type of mutation does seem to be of particular importance in unicellular organisms, and, as we have already noted here, in *E4D* (pp. 106–107) we argued that they are likely to be less significant in multicellular organisms. However, we may well have been too negative about this, because recent studies of stressed plants – plants subject to internal stresses resulting from hybridization or polyploidization, or to external stresses involving nutrition or infection – have shown that stress can sometimes induce targeted and reproducible changes in the genome. (See articles in *Biological Journal of the Linnaean Society* 82(4), 2004.) It begins to look as if there may be complex evolved mechanisms that, in severely adverse conditions, alter the gross

structure of the genome in a targeted manner. As yet, these genome-altering mechanisms are poorly understood, but we believe that they are probably adaptive. Note that we think the mechanisms are adaptive – we are not saying that the mutations they generate are necessarily of adaptive value: The mechanisms increase the probability that a mutation or genomic change that is of adaptive value will occur. The situation may be comparable to the evolved mechanisms of targeted mutation in the immune-system genes; these mechanisms that produce mutations are adaptive, although most of the specific mutations they generate are non-adaptive. We follow Darlington (1939) in thinking that the way the genetic systems have evolved has been an important factor in evolutionary history, and add our voice to those of McClintock (1984) and others who have suggested that organisms may have evolved the capacity to make genomic changes in response to conditions of extreme stress. We return to this point in section R4.2, where we consider the predictive value of the four-dimensional approach that we advocate.

R2. The concept of information in evolution

When writing *E4D*, it became clear to us that neither the notion of information used in everyday language, nor that usually used in mathematical information theory, were appropriate for our purposes. We needed to define and elucidate "information" in functional terms because information would be a unifying concept in the book. The need to give a definition was reinforced by the publication of Maynard Smith's (2000) paper entitled "The concept of information in biology," which focused on genetic information and the way that it can be said to specify the form and function of living organisms. According to Maynard Smith, in biology the notion of information is linked with the idea that both the signal carrying the information (e.g., DNA) and the response to it are products of natural selection. Although Maynard Smith's paper rekindled interest in the notion of semantic information, we were worried that his narrow, gene-oriented approach would come to dominate thinking in this field. We needed a broader definition, one which would accommodate information stemming from environmental cues as well as from evolved signals, and which would enable a comparison between the modes of information transmission in the different types of inheritance systems that we were describing. Our definition had to be appropriate for genetic, epigenetic, behavioral, and cultural-symbolic transmission.

After many weeks of reading and mental struggle, we eventually came to a receiver- and development-oriented definition of information: A source becomes an informational input when an interpreting receiver can react to the form of the source (and variations in this form) in a functional manner (*E4D*, pp. 52–56). Because our focus in *E4D* was on heredity and evolution, and hence on the transmission of information between generations (through reproduction or through communication), we used the term mainly in the context of hereditary transmission. According to our notion of information, in order for information to be hereditary (1) a receiver has to developmentally reconstruct (we use "interpret" and "interpretation" in this sense) an informational input from a sender who was previously a receiver; (2) such a reconstruction must lead to the same (or a slightly modified) organizational state as that of the sender. Jablonka (2002) has given a more extensive discussion of these issues. In *E4D*, the nature of information (whether holistic or modular, limited or unlimited), the mechanisms generating informational variations, the direction of transmission, and so on, are explicitly discussed in each of the chapters dealing with the different inheritance systems (Chs. 2–6), as well as in the Interim Summary (pp. 233–38).

Oyama (1985) has challenged the usefulness of information-talk in biology, and has shown the flaws and dangers of using gene-based information metaphors. We agree with much of what she has said, but believe that part of the problem is that "information" has been defined too narrowly. We hoped that our explicit, development-oriented approach would overcome this and allow fruitful comparison between the different systems of heredity. The approach we have taken is, in fact, very similar to that which **Steels** takes, because we focused, just as he does, on the role of developmental reconstruction rather than on the blind copying of information. This is of central significance when behavioral and symbol-based transmission are discussed (*E4D*, pp. 211, 219–23). Although we did not reiterate this approach when we considered language evolution (Ch. 8), we think that our general discussion of symbolic communication makes it clear that our notion of language evolution is very similar to that of Steels: We do not think that language transmission is through blind imitation, and we see community language as an attractor in the sense described and used by Sperber (1996, Ch. 5). However, as we discuss in section R3.3, we find the concept of a replicator problematic, even when used in Steels's careful way.

Our view of the properties of living organisms is very close to that of **Hamame, Cosmelli, & Aboitiz** [**Hamame et al.**], who see them as interpretative and intentional systems. (On a minor point, when we said in *E4D* that there is nothing intentional in the receiver's reactions, we used "intentional" in the sense of "having intentions," not in the philosophical sense of "being about something.") However, if Hamame et al. see interpretation as a central distinguishing feature of living systems, what, if not information, do they think is interpreted? How can one get rid of the concept of information if one wants to discuss interpretation? We agree, of course, that information-transmission is a reconstruction process, and we stressed this many times in *E4D*, but if one wants, as we did, to compare different systems of heredity, some common denominator has to be found. We believe that a function-sensitive notion of information, which must therefore be related to fitness, is this common denominator. Moreover, using the term "information" helps to highlight the different natures of the things that contribute to the reproduction of organisms. As Avital and Jablonka (2000) and Lachmann et al. (2000) have argued, there is a profound difference between giving (or sharing) information and giving (or sharing) material and energy. The crucial difference is that whereas there are laws of conservation of mass and energy, there is no law of the conservation of information. Information can be given without losing it (often at a very small energetic cost to the

animal). This has profound effects in evolution. We believe that the notion of "information" (like the related notion of "function") is a specifically biological notion: Only living, or designed-by-living, organisms can be said to have information. Put differently, there is no information without interpretation. And since interpretation, as Hamame et al. argue, is a distinguishing feature of living systems, so is information.

R3. The "evelopment" of culture

The major interest of almost all of the commentators is the evolution of culture, and in particular, the evolution of human symbolic culture and human language. For us, the evolution of symbolic culture was the most difficult problem we had to tackle. We had to establish that symbolic "cultural evolution" is a coherent concept; and to try to see how one can study it; we had to define symbols, and distinguish between symbol-based cultural evolution and nonhuman, behavior-based cultural evolution. And we had to examine the relations between genetic and cultural evolution and to distinguish between evolution through culture, the evolution of cultural entities (at different levels), and the evolution of the capacity to develop culture. Because we saw cultural evolution as a process involving both the development and the selective retention of variants, we invented an ungainly hybrid term for this kind of process: "evelopment" (*E4D*, p. 148).

R3.1. Symbols and the problem of meaning

We defined symbols as a special type of signs, and defined signs as pieces of information transmitted from sender to receiver. The signs-that-are-symbols have a double nature: They refer to entities, processes, or relations in the world, and they also refer to and evoke other signs in the symbolic system, in a way that is bound by more or less binding rules, specific to the symbolic system in question (*E4D*, pp. 194–201). This definition of symbols focuses on communication between individuals and on the intersubjective aspect of meaning. People agree on a set of meanings, or definitions of a situation, and that is what makes communication among them possible. The ability to agree (implicitly or explicitly) is based on people sharing common cognitive properties and common-sense, so that fairly complex meanings (such as the meanings being conveyed in this exchange in *BBS*) can be reconstructed and understood by people who have never met each other. Clearly, a lot is left out of such intersubjective exchange: the idiosyncratically subjective, for example, as well the private aspects of emotion and feelings, which are very difficult to communicate even through facial and body gestures. Intersubjective communications through symbols come only through abstracting from private personal experience. The symbol, and especially the linguistic symbol, is far more discrete and digital than the analog, rich, and fuzzy reality and the private experiences to which it refers (Dor & Jablonka 2000). Nevertheless, the very limitations of the symbol are also its strength: Communication through symbols allows effective communication of (partial) meanings, and sometimes, albeit rarely, as through great poetry, it even enables the communicators to transcend the inevitable loneliness of private experiencing. Of course, once symbols exist, they too assume a private aspect: we engage in internal monologues (communication with oneself); symbols become objects of deep emotions; and symbolic communication influences our most basic behaviors.

Our notion of symbols and symbolic language is very different from that of **Sloman & Chappell**. For us there are no "internal symbolic structures," although naturally there are neurological processes and structures that underlie the ability to communicate through symbols. We regard planning, imagining, predicting, identifying informational gaps, and so on, all of which are characteristics of intelligent mammals, as complex cognitive abilities that are necessary but not sufficient conditions for symbolic communication and representation. We believe that conventionalization, which requires a degree of abstraction from the private, is a necessary condition too. When thinking about natural human language, we incorporate into its conception a set of constraints and affordances at both the neural and the cultural levels. We strongly agree with Sloman & Chappell's view that it is necessary to focus on development, and on the likely involvement of a complex hierarchy of developmental stages. However their "g-language" is, for us, a precondition for language, which is, as they argue, apparent in nonhuman species and young children. The complex cognitive processes and behaviors that animals and young children display are not, therefore, "symbolic" according to our definition of the term. In addition, although we agree with Sloman & Chappell that these cognitive capacities had to evolve before human language emerged, we think that symbols and symbolic human language evolved in the context of communication, because the external aspect of the linguistic symbol, the intersubjective communication that requires private experiences to be pared down, is a precondition for symbolic language. It was this experiential poverty that made the symbol into such a phenomenally powerful tool for communication (of certain things), and at the same time, such an inadequate tool for the expression of the rich and holistic states of many private experiences.

This leads us to **Foss**'s comments about the evasive nature of meaning, and his claim that "there cannot be a science of symbolic evolution" because symbols have meanings, and meanings are analog rather than digital. There are no discrete meanings but, rather, shades of meaning, Foss claims; meanings cannot be measured, and there cannot be units of meaning that can undergo evolution. Foss is highlighting the deeply subjective aspect of communication, and he criticizes us for not addressing this in *E4D*. This is indeed something that we avoided in *E4D*, and the reason we did so was that our focus was on heredity and evolution. However, Foss is right in his assertion that meaning is crucial for understanding cultural, symbol-based human evolution, because symbols, with their double, private–public faces, are the strong attractors of culture. They construct shared public values which are imbued with strong private meanings, and are crucial drivers of cultural evolution.

Can meaning be defined and measured? We think that meanings can be defined and are amenable to evolutionary

study. We agree with **Foss** that there is no precise measure for meaning. However, there is also currently no measure for function: The function of a DNA sequence is difficult to measure because it is both context and content dependent. (For an important first step in defining the fitness value of information, see Bergstrom & Lachmann [2004].) The difficulty in measuring function does not, however, preclude the scientific discussion of the function and function-dependent evolution of genes, proteins, and other biological entities. From this point of view, the cultural act is no different. However, because the cultural act has not only function but also meaning, and meanings can drive cultural evolution, a discussion of meaning is certainly desirable.

We believe that an evolutionary account of meaning is possible, but that it requires a discussion of the ability of animals to experience the world rather than merely to process sensory information and respond to stimuli as plants do. In other words, it requires an evolutionary explanation of the nature of feelings and the reward systems underlying them. Ginsburg and Jablonka (in press) propose that something can be said to have "meaning" when the animal experiences (feels/thinks) it and has a system that assigns a value to it. Value systems are organismal neural structures and processes that "reward" (positively reinforce) any state that enhances a fitness-promoting action, and "punish" (negatively reinforce) any state that leads to actions that decrease fitness. Sensory-cognitive states, such as balance (versus imbalance) in primitive metazoans and pleasure (versus pain) in more evolved animals, are the attractor-states towards which the animal strives, and are usually associated with behaviors that promote fitness. In humans with symbolic communication, ideas of the good (versus the bad), truth (versus untruth), beauty (versus ugliness), justice (versus injustice), and so on, are the culturally constructed attractors towards which we strive. Because these symbolically encoded attractors are based on publicly shared and constructed culture, they transcend the individual and can be (and often are) decoupled from inclusive fitness considerations. In other words, the symbolic system can "subvert" biological fitness more readily than the goal-directed ontogenetic reward systems of animals, because symbolic values may have little to do with the here and now. This means that cultural, symbol-based evolution has to be studied in ways that incorporate this potential decoupling. However, we do not see why this complication precludes an evolutionary approach to culture.

R3.2. How should we approach cultural evolution?

The similarities and differences between genetic and cultural transmission, and the role of genetic assimilation in evolutionary processes, are topics addressed by several commentators. Sometimes we felt that their comparisons were based on a very sophisticated notion of culture and a rather unsophisticated view of genetics. When, as we now know, a gene can code for several different protein products, some genes code only for RNA, two genes can use overlapping DNA sequences, selectable phenotypes are the products of complex developmental networks, and we have no idea what role, if any, most DNA plays in the cell, we think it is no longer appropriate to reduce the genetic dimension of heredity and evolution to the replication and selection of DNA sequences called "genes".

Gabora calls attention to the differences between gene replication and the type of transmission involved in cultural evolution. She has pointed out (Gabora 2004) that behavioral and cultural evolution involve uncoded, interpreted information, rather than the coded, uninterpreted information of the genetic system of inheritance. In her commentary, Gabora claims that, "The mathematical framework of natural selection is not transferable to evolutionary processes that are not code-driven," and therefore that behavioral and cultural evolution, just like the origin and early evolution of life, is primarily non-Darwinian. They should be seen as Lamarckian processes. According to Gabora, for a process to be Darwinian, "inheritance of acquired characteristics must be negligible compared to change resulting from differential replication of individuals with heritable variation competing for scarce resources."

There are elements of **Gabora**'s thesis with which we are in total agreement. Like her, we believe that the nature of the information – in our terms, whether it is modular or holistic – influences how evolutionary change occurs, and that both Lamarckian and Darwinian processes occur in evolution. We also agree that the fidelity of reconstruction (a word that we prefer to replication) is important. Where we disagree is with Gabora's fundamental assumptions that natural selection "is intimately tied to the genetic code," and that when induced (or "acquired") variations are transmitted, evolutionary change has to be regarded as non-Darwinian. We see no justification for these assumptions, and know of no reason why natural selection – a Darwinian process – and induced heritable variation – a Lamarckian process – should not both be involved in an evolutionary change. We think that by using the gene-based replicator as a conceptual framework, Gabora has fallen into the trap of assuming that if an evolutionary change cannot be explained in terms similar to classical gene-based explanations of adaptation, Darwinian selection is not involved. One of the reasons why we find Griesemer's (2000) reproducer concept so valuable is that it avoids this pitfall. As we suggested in *E4D*, the significance of induced variation and Lamarckian processes probably varies widely; they are obviously important in cultural evolution, but that does not mean that Darwinian selection is not occurring.

While **Foss** maintains that culture cannot be said to evolve, and **Gabora** claims that cultural evolution is purely Lamarckian, **Mesoudi** sees merit not only in the concept of cultural evolution, but also in present-day models of it. He takes us to task for dismissing an impressive body of theoretical work on cultural evolution, and he is right to do so. We should have discussed the models of cultural evolution in greater detail, although in self-defense we have to point out that we did direct interested readers to the relevant sources (see notes to p. 205 on p. 402 of the book). Nevertheless, we still think that there is a need for rather different models of cultural evolution – models in which induction/learning and selection are coupled, and in which there are strong cultural "attractors." However, this is a challenge for the future. Models in population genetics have generally ignored development, because developmental induction is less central to the genetic inheritance system than it is to cultural

transmission. We think that classical population genetics, which was very important for the development of evolutionary biology because it demonstrated theoretically that small selection coefficients and chance mutations could bring about "descent with modification," is of limited predictive value now that genes are seen as part of developmental networks. The very simplistic assumptions of classical population genetics are no longer thought to be appropriate, and different mathematical models are being constructed to accommodate the growing recognition that the effects of gene interactions complicate selection theory. Similarly, we think that a new family of models is necessary for dealing with cultural evolution. They will need to incorporate multilevel exploration, learning, and selective stabilization processes based on genetically and culturally evolved mechanisms and attractors. These models will recognize that although the selective retention of some cultural entities may be a result of a benefit accrued to an individual or a group, it may also result from the strength of symbolic, publicly shared conventions, which serve as attractors and can override individual or group benefits.

Unlike **Faulkes & Baines**, **Mesoudi** finds the analogy between cultural and genetic evolution useful, and suggests that cultural creativity and genetic mechanisms of evolvability may be analogous processes. We think that this is a very interesting suggestion. Evolvability has recently become a hot topic in evolutionary biology, and the heuristics of cultural creativity may inspire interesting new directions in the study of the poorly understood genetic "heuristics." We completely agree with Mesoudi that "mental travel time" is a crucial and unique feature of human culture with no parallel in gene-based evolution, and we believe it to be a result of the symbolic nature of human language (see, e.g., *E4D*, p. 203).

Whereas most commentators have focused on comparisons of gene-based and cultural evolution, or the interactions of the genetic and cultural systems, **Warlaumont & Dale** stress the importance of the relationships between behavioral and symbolic inheritance, and they criticize our neglect of this topic. All that we can say about Warlaumont & Dale's comments is that they were right to criticize us, and we appreciate the excellent examples they have provided to show how the development of symbolic linguistic communication and representation is deeply interrelated with behavioral transmission. Although we were aware that behavioral transmission underlies symbol-based information transmission, and that symbolic communication and thought affect behavior, in our effort to show the autonomy of symbol-based transmission we dealt with the interactions between the behavioral and the symbolic in a non-systematic manner. We think that this is a regrettable omission for exactly the reasons stated by Warlaumont & Dale. Moreover, had we discussed the interrelations of the two brain-based inheritance systems more systematically, some of the apparent weaknesses in our position, to which the commentaries of **Faulkes & Baines** and **Lappan & Choe** point, would not have arisen.

Lappan & Choe argue that, "If symbolic evolution is completely decoupled from the symbol-producing organisms' fitness and behavior, and transmission is independent of reproduction, then the analogy to biological evolution is an interesting thought exercise, but it would be difficult to support the argument that symbolic variation represents a fourth distinct dimension of biological evolution." A similar point is made by **Faulkes & Baines**, who regard brain-based and cell-based inheritance as being so disparate that a comparison between them has no value. Our view is that it is the dynamics of historical, intergenerational change that unites the different systems of transmission. Obviously the systems are very different, and we stressed these differences in Parts I and II of *E4D*; in the Interim Discussion we also pointed to what they had in common. We wrote the book believing that a focus on information – on its storage, its transmission, and its development – could unify and illuminate aspects of evolution and lead to a useful research program. In the last part of the book, we showed that the systems are intimately related, and that evolution on one (non-genetic) axis can profoundly affect the genetic axis. The case of language evolution discussed in Chapter 8 illustrates how the genetic, behavioral, and symbolic systems are intertwined: language evolves on a symbolic-cultural axis; the dynamically changing, yet consistent, linguistic niche affects the selection of behavioral and hence genetic variants; and the selected behavioral and genetic changes then affect the evolution of the capacity for language. We suggested how genetic assimilation, and its more sophisticated manifestations such as "the assimilate-stretch principle," can link the constructed and inherited cultural (linguistic) niche with the genetic underpinnings of the language capacity and lead to a sophistication of linguistic behavior over generations. Symbolic evolution in this case (and in many others) is related to fitness, although on certain time-scales the dynamics of symbol-based evolution have to be considered autonomously. This very autonomy, however, contributes to the "stretch" phase in the process, which leads to genetic assimilation.

The role of genetic assimilation, which seems to **Gabora** to be over-emphasized in our account of language evolution, and which **Steels** implies is under-emphasized, is an issue about which we were very careful. We do not know how much genetic assimilation was involved in the evolution of language, although for the reasons we gave in *E4D* (pp. 308–10, 316) we have little doubt that it was involved.

R3.3. Memes and replicators: Why object?

Blackmore advocates a meme-oriented approach to the evolution of culture, and takes issue with us over our less-than-enthusiastic treatment of memes. For her, a meme is "that which is imitated," and she suggests that it is imitation, rather than symbolic representation and communication, that is the basis of human uniqueness. She finds our notion of symbols incoherent, because it is not clear to her what an abstract picture or a new dress symbolizes, and she is also unhappy with the way we use the term "memeplex." Blackmore sees an increase in the fidelity of copying as an important part of both genetic and cultural evolution.

As we pointed out in section R3.1, our notion of "symbol" is not dependent on the iconicity of the symbol, but rather, depends on the duality of its reference – to things in the world and to other symbols – and on it being a component of a rule-governed symbolic system. We agree with

Blackmore that songs and dances are not inherently symbolic. Indeed, in *E4D* (p. 202), we described how the same type of behavioral entity (e.g., a song) can be transmitted in different ways: A song's development can depend mainly on genetic resources (as with the courtship song of male *Drosophila*), or on socially learnt resources (as in some song birds), or, as in humans, on symbolic resources. An abstract painting is a symbolic object par excellence, because it can be properly interpreted only within the context of modern (symbolic) art. A new dress fashion has a symbolic aspect because it implies and refers to status, or to social class, or to a public icon, and because it points to and evokes a host of related and interacting behaviors and artifacts. Tunes, cars, hairstyles, and buildings are also part of a symbolic system, although they are not "pure" symbols like words or numbers.

We agree with **Blackmore** that symbolic thought is a product of cultural (and genetic) evolution rather that its starting point, and in Chapter 5 of *E4D* we described non-symbolic cultural evolution in animals. Cultural evolution does not depend on imitation, although when imitation exists the range of cultural variants may be greatly expanded. We also agree with Blackmore that the development of high-fidelity copying is very important both in genomic and in cultural-symbolic evolution. However, we do not see how her focus on imitation avoids the fundamental problems to which we pointed in *E4D*. Most human (non-machine) imitation involves active exploration and learning, governed by understanding and by perceived goals (*E4D*, pp. 210–12; Jablonka 2004b). **Steels** rightly points out that models of cultural evolution based on imitation assume what they try to explain, because a person has to know what counts as information and what the intention of the producer is before they imitate. Perhaps his explanation will help **Faulkes & Baines** to understand why we distance ourselves from memes. Both imitative and non-imitative socially learnt behaviors are developmentally reconstructed, not copied in a machine-like or gene-like way. One of the examples of memes that Dawkins gave in the *Extended Phenotype* (Dawkins 1982, p. 109) was the socially learnt skill of opening milk bottles by tits, so we used it in *E4D* to show how, in this case, the meme (a habit) is transmitted through a process of developmental reconstruction, not imitation. But, even with cases of copied behavior that do involve imitation (e.g., engagement in a religious ritual), it can still be readily shown that developmental reconstruction and active, environment-sensitive learning are involved (*E4D*, pp. 210–12; see also Avital & Jablonka 2000, pp. 25–29).

As we argued in *E4D*, we think it is the notion of the meme as a replicator that is fundamentally flawed. In his latest book, *The God Delusion*, Dawkins describes memes as replicators, and defines a replicator as "a piece of coded information that makes exact copies of itself, along with occasional inexact copies or 'mutations'" (Dawkins 2006, p. 191). **Blackmore** claims that the distinction between replicator and vehicle is not crucial for her notion of the meme, yet she still finds the meme concept that emerged from this distinction productive. The difficulty we have is that we cannot see how what is referred to as a "meme" can be anything other than a heritably varying phenotypic trait, which is developmentally constructed and reconstructed through social learning (be it by imitation, by emulation, or by any other type of socially mediated process). If "meme" had been used in this way – as a term for socially transmissible behavioral phenotypes, preferences, or products of behavior – we would have seen it as a useful piece of shorthand. However, the wish to see the meme as a replicator akin to a gene, rather than as a character, leads to conceptual complications which render the concept incoherent. The term "memeplex" also seems to us to be full of problems. It is defined by Dawkins (2006, p. 198) as "a set of memes which, while not necessarily being good survivors on their own, are good survivors in the presence of other members of the memeplex." His memeplex therefore refers to the functioning of a set of memes, not to their transmission; it is an analog of an adapted gene complex. It is not a unit that is copied, which is the meaning that Blackmore gives to the term in her commentary. However, it is the conceptual problems with the meme, rather than those with the related terms used in memetics, that in our view prevent memes and replicators from being fruitful tools for understanding cultural evolution.

The problems associated with the replicator concept are also reflected in **Gabora**'s and **Steels**'s commentaries. Gabora identifies Darwinism with the replicator-centered interpretation of it that Dawkins and others have constructed, and is therefore driven to a position that denies that natural selection is of relevance to models of cultural evolution, a position that we think is untenable. Steels's point of view is different. He recognizes the value of the reproducer concept (Griesemer 2000) and suggests that, when considering language evolution, a useful distinction can be made between replicators and reproducers, with utterances being replicators and the language system as a whole being seen as a reproducer – an entity that develops to reproduce and is a target of selection. This is an interesting suggestion, but we think that it is better to give up the replicator concept altogether, and think in terms of reproducers that have heritably varying traits, which are more or less sensitive to developmental regulation through induction and learning. The specification of the spectrum of developmental sensitivities, the genetic and/or cultural evolution of such a spectrum, and selection for increased or decreased modularity of heritably varying traits would then become well-defined research questions.

R4. Implications and predictions

Because **Faulkes & Baines** suggest that *E4D* presents a kind of biological "string theory" – something of theoretical interest but of no empirical value – we end here by illustrating how a research program that incorporates a multidimensional view of heredity and evolution can be productive. We have already pointed to some of the implications of our perspective in various places in *E4D*, particularly in the concluding chapter, but it may be helpful to bring these together, focusing on its predictive importance for studies of heredity and evolution.

R4.1. Practical applications

Although much remains to be done, the roles of the additional inheritance systems we discussed in *E4D* are already being investigated and are affecting research programs. The most dramatic impact is that of the

epigenetic dimension in medicine. It is becoming clear, for example, that in addition to the genetic and environmental factors that influence the incidence of disease, the probability that an individual will develop certain diseases is affected by the environments to which their ancestors were exposed, which have effects that carry over to their descendants (Jablonka 2004a; Jirtle & Skinner 2007). In cancer research and developmental medicine, epigenetic inheritance is being intensely studied, and it is considered by leading authorities in the field to be of central significance for diagnosis and potential treatment. (For recent work and reviews, see Baylin & Ohm 2006; Burdge et al. 2007; Chan et al. 2006; Gluckman et al. 2007; Pogribny et al. 2007; Rodenhiser & Mann 2006; Yoo & Jones 2006.)

Understanding epigenetic inheritance is crucial for further progress in cloning (Jaenisch et al. 2004). Epigenetic effects in bacteria are also of medical importance, because cells that in normal growth conditions are killed by antibiotics can become "persistent." Persistence, an epigenetic phenomenon, occurs when some of the bacterial cells in a population switch into an antibiotic-tolerant state, usually by decreasing their growth rate (or by abolishing growth altogether). Tolerant cells are genetically identical to sensitive cells, and cells can switch from sensitive to tolerant states and vice versa. These switches occur stochastically, but the rate of switching may be affected by environmental conditions (Kussell & Leibler 2005; Lewis 2007). Because this system has been partially unraveled, drugs that can counter the persistence of the pathological microorganisms are being developed. There is also increasing theoretical work being done on how such epigenetic systems may operate, and how they have evolved. This is reflected in the modeling that has been carried out (Jablonka et al. 1995; Kussell et al. 2005; Lachmann & Jablonka 1996; Pál 1998; Walczak et al. 2005).

It is not only in medicine that the recognition of epigenetic inheritance is having an effect. In agriculture epigenetic inheritance is known to be fundamental to constructing strains of genetically modified plants and animals that do not silence newly introduced, potentially beneficial genes, and is also important for carrying out risk assessments of such new strains. Ecologists, especially plant ecologists, are now beginning to study the frequency of epigenetic variants (e.g., DNA methylation marks) within plant populations (Ashikawa 2001; Cervera et al. 2002; Knox & Ellis 2001; Liu & Wendel 2003; Riddle & Richards 2002; Wang et al. 2004). Although simple methods for detecting epiallelic differences other than DNA methylation have yet to be developed, many ecologists are clearly aware of the importance of epigenetic variations, as well as genetic variations, for understanding biological diversity, conservation, and adaptation.

Epigenetic inheritance is a very young field of study, so there are still many basic questions that have not yet been addressed, although they are amenable to experimental investigation. In *E4D* and elsewhere, we have pointed to several of them. For example, does the number of generations organisms spend in a particular inducing environment have cumulative effect on the stability of marks (Jablonka & Lamb 1995)? What is the epigenetic basis of Lansing (cumulative parental age) effects (Jablonka & Lamb 1990, 1995; Lamb 1994)? Are there cumulative effects on epigenetic marks when chromosomes are transmitted through one sex for several generations (Jablonka 2004c)?

There is also much basic work that needs to be done on the transmission of information by behavioral means in nonhuman animals. In their book *Animal Traditions*, which discusses the formation and evolution of cultural traditions in nonhuman animals, Avital and Jablonka (2000) described many simple and interesting experiments that would shed light on the evolution of culture. Their suggestions include: First, studying how coprophagy (feces-eating) affects food preferences in mammals by cross-feeding young with feces from adults kept on different diets. (Such studies might also reveal whether feces are a route for the transmission of disease-causing prions. It seems to us possible that the prion diseases in wild deer populations in the United States could be transmitted by feces-eating.) Second, studying the transmission through social learning of self-medication in animals by experimentally altering medicinal practices or their objects. Third, investigating the possibility that not only material and energy, but also information (e.g., about food and other ecological preferences) is transmitted during courtship feeding. This could be done by studying the behavior of females with courtship-feeding males that have different ecological preferences. Fourth, in bird species with learnt duets, investigate by modeling and through observation the possible role of nontransferable information in the long-term stabilization of the pair bond. Fifth, in species with helpers, where the helper's feeding or caring habits might be transmitted to the cared-for young, find out whether, and to what extent, future adult behavior is affected by looking at individuals that are cared for by helpers with different (natural or experimentally manipulated) habits.

R4.2. Evolutionary implications

The view that non-genetic inheritance is important has many implications for evolutionary theory, which we have discussed in *E4D* and later publications (Jablonka & Lamb 2006a; 2007). They include:

1. Adaptation can occur through the selection of heritable epialleles. This may be particularly important if populations are small and lack variability. Because epigenetic variants may be induced when environmental conditions change, and many individuals in the population may acquire similar modifications at the same time, adaptation through the inheritance of newly induced epigenetic variants may be very rapid (Kussell & Leibler 2005; Lachmann & Jablonka 1996). Although epialleles may not be as stable as genetic alleles, adaptations based on such variation may enable a population to survive long enough for a process of genetic accommodation to occur (*E4D*, Ch. 7; Pál 1998; Sangster et al. 2004; Siegal & Bergman 2006).

2. Chromosomal evolution may be affected by epigenetic variations. Jablonka (2004c) suggested how the selection of inherited epialleles can influence the evolution of sex chromosomes in mammals, and Rodin et al. (2005) discussed how heritable epigenetic silencing can affect the fate of duplications.

3. Epigenetic variations can affect the generation of genetic changes by biasing the rates of point mutation, transposition, recombination, and other genomic reorganization processes (*E4D*, Ch. 7; Jablonka & Lamb 1995).

4. The epigenetic and genetic responses to stress may lead to very rapid evolution by reorganizing the genome.

Genomic stresses such as hybridization and polyploidization are known to induce massive genetic and epigenetic reorganization in plants (Rapp & Wendel 2005), and possibly also in animals, and ecological stresses involving nutrition have been shown to induce significant changes in repeated sequences in plants (Cullis 2005), probably via epigenetic mechanisms.

5. Heritable epigenetic and behavioral variations may initiate the population divergence that leads to speciation. Reproductive isolation may begin when non-genetic behavioral differences between individuals from different populations prevent mating taking place (*E4D*, Ch. 5; Avital & Jablonka 2000), or because their chromosomes carry incompatible chromatin marks and, as a consequence, the viability or fertility of hybrid offspring is reduced (*E4D*, Ch. 7; Jablonka & Lamb 1995).

6. When learning from and through others is important, as it is for many social birds and mammals, group selection may play a greater role in evolution than is generally assumed (Avital & Jablonka 2000).

7. In birds and mammals, the necessity of exchanging information suggests alternative or complementary interpretations of the evolution of relationships between mates, and between parents and offspring. Avital and Jablonka (2000) believe cooperation, rather than conflict, may have driven or helped to drive the evolution of some of the observed behaviors. They also suggest that the preferential expression of maternal (imprinted) genes in the cortex of mammals during postnatal development may reflect the outcome of selection for compatibility between mother and offspring, rather than being the result of mother–offspring conflict, because the young have to be maximally attuned to the mother during the critical period of early learning. Hager and Wolf (2006) believe that the need for compatibility between mother and offspring can explain many patterns of genomic imprinting. They suggest that during mammalian development in utero, selection for coadapted maternal and offspring traits may drive the evolution of genomic imprinting, and they have supported this suggestion with a simple model.

8. Because cellular epigenetic inheritance acts as a powerful constraint on the evolution of development, discussions of problems such as the origins of multicellularity, irreversible cellular differentiation, the segregation of germlines, and so on need to include the epigenetic dimension (*E4D*, Ch. 7; Jablonka & Lamb 1995).

9. Zuckerkandl and Cavalli (2007) have suggested that so-called "junk DNA," which consists largely of repeated sequences, might be a carrier of epigenetic marks that can be communicated to and established in other regions in the genome. An altered mark could therefore result in coordinated hereditary changes in the expression of several different genes simultaneously, and thus accelerate adaptive evolution.

10. Non-genetic mechanisms of inheritance have played a central role in all the major evolutionary transitions identified by Maynard Smith and Szathmáry (1995). We outlined how they did so in *E4D* (Ch. 9) and later discussed this subject in greater detail (Jablonka & Lamb 2006b). In the latter article, we also discuss one of the transitions that Maynard Smith and Szathmáry did not identify, although it certainly must rank as "major," namely, the transition from non-neural animals to animals with a nervous system.

11. Because non-genetic mechanisms of information transfer play such key roles in evolution, the evolution of the non-genetic inheritance systems themselves is of fundamental interest.

R4.3. The case of the silver foxes

We end this section by discussing the study by Belyaev and his colleagues of the domestication of silver foxes, which **Faulkes & Baines** think we have misinterpreted. Belyaev (1979) called the domestication of animals over the past thousands of years one of the greatest experiments in biology, stressing the amount of very rapid evolutionary change that had taken place. He used his studies of the domestication of foxes as a framework for discussing the nature of these changes, and came to some interesting conclusions.

Selection for tame behavior in silver foxes began at the end of the 1950s and is still going on (Trut et al. 2004). It resulted in a very interesting complex of heritable changes: In addition to becoming doglike in their behavior, the foxes showed various other modifications such as changes in the skeleton, decreased sexual dimorphism, hormonal changes, spotting, altered tail and ear posture, and an increase in the incidence of small, supernumerary chromosomes. Belyaev and his group studied the pattern of inheritance of spotting, an easy character to assess, and found that it was strange: Spotting behaved like a dominant or semi-dominant trait, but it had a high rate of reversion. The rate of appearance and disappearance of the character was far too high for mutation to be likely, and it could not be explained as an effect of inbreeding, because the coefficient of inbreeding was only 0.03 (Trut et al. 2004).

Overall, the high rate of morphological and hormonal change that took place as the foxes were selected for tameness, and the parallels with the changes that occurred during the evolution of the dog, suggest that the genes selected during domestication probably had big effects on rates of development, particularly in the nervous system and associated developmental pathways. But what exactly was selected? Was it alleles, epialleles, or both?

Belyaev's suggestion was that the conditions of domestication led to neuro-hormonal changes that reactivated dormant genes in the soma and germline, thereby revealing previously hidden genetic variation. Not all genes can be reactivated in a way that is transmitted transgenerationally, but the selection for tameness picked out those allelic variants that could. This interpretation in terms of certain alleles being able to switch from dormant to active states explains the high rate of appearance and disappearance of the phenotypes. In the terms used today, we would say that Belyaev was probably studying epiallelic variants. Support for this interpretation comes from the fact that at least two of the genes (*Agouti* and *C-kit*) that Trut et al. (2004) think may be involved in the pathways leading to the change, are known to have heritable epigenetic variations in mice. Thus, if Belyaev is correct, selection for tameness involved changes in the frequency of both alleles and epialleles: Stably reactivated epialleles of reactivatable alleles were picked out during selection. We think that this interpretation is consistent with the observed patterns of inheritance. Now that the dog genome project is yielding more and more information, and the genes underlying morphological development as

well as aggressiveness in foxes are being studied (Popova 2006; Trut et al. 2004), looking for the epiallelic variants of these genes is becoming a possibility. Belyaev's bold conjecture can now be tested directly.

R5. Conclusions

The approaches to evolution, especially human evolution, have been many and varied, but since the mid-20th century, a gene-based neo-Darwinian theory has remained central. The only exception has been with human behavior and culture, where it is often acknowledged that there is a Lamarckian element, because the conditions of life influence the generation of variation as well as its selection. Modifications brought about by learning and development can be passed to future generations and bring about evolutionary change. The view of evolution that we developed in *E4D* is Darwinian, because it recognizes the power of natural selection, but it also incorporates Lamarckian elements. We believe that the information that is the basis of selectable phenotypic variation can be transmitted from generation to generation by several different routes, and that some new variations are developmental responses to internal or external environmental conditions.

The commentators on *E4D* show various degrees of enthusiasm for our version of evolutionary theory, which in many ways is closer to Darwin's thinking than to that of the neo-Darwinians. Some think we are basically right, and they have suggested additional arguments supporting a move away from the gene- and replicator-centered perspectives that dominate evolutionary thinking today. Others think that the existing neo-Darwinian framework is perfectly adequate, and in some instances criticize us for being over-zealous in our advocacy of a broader approach to heredity and evolution. We end here by saying something in response to the latter point.

We do not apologize for our zeal, because we believe that the debate about the nature of heredity and evolution is important, and not just for biologists. We think that what we call genetic astrology, the "it's-all-in-the-genes" view of human differences that is aired repeatedly in the media, especially when describing the potential power of new technologies, is not only wrong but also dangerous. It was, after all, the basis of the eugenics movement that had such disastrous consequences in many countries.

How biologists describe evolution also matters. Part of the reason why many young people reject or lose interest in evolutionary ideas is that when evolution is reduced to the selection of randomly occurring changes in genes, it seems to have little to do with their own understanding and experiences of the world around them, especially their understanding of human nature. Even social scientists often regard evolutionary biology as of no relevance to their discipline. Yet evolutionary ideas should help us to understand ourselves, our societies, and the rest of the living world. We hope that the broader approach to evolution that we advocate, which is an approach that recognizes the importance of development and learning, will help to bridge the gap between evolutionary biologists and others who seek to understand the living world, human behavior and culture, and the complicated interactions between them.

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

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