The major evolutionary transitions

Eörs Szathmáry & John Maynard Smith

There is no theoretical reason to expect evolutionary lineages to increase in complexity with time, and no empirical evidence that they do so. Nevertheless, eukaryotic cells are more complex than prokaryotic ones, animals and plants are more complex than protists, and so on. This increase in complexity may have been achieved as a result of a series of major evolutionary transitions. These involved changes in the way information is stored and transmitted.

THE major evolutionary transitions[1] are listed in Table 1. There are common features that recur in many of the transitions: (1) Entities that were capable of independent replication before the transition can only replicate as parts of a larger unit after it. For example, free-living bacteria evolved into organelles[2]. (2) The division of labour: as Smith[3] pointed out, increased efficiency can result from task specialization (for a comprehensive review of this subject in the classical literature, see ref. 4). For example, in ribo-organisms nucleic acids played two roles, as genetic material and enzymes, whereas today most enzymes are proteins. (3) There have been changes in language, information storage and transmission. Examples include the origin of the genetic code, of sexual reproduction, of epigenetic inheritance and of human language.

Complexity

There is no generally accepted measure of biological complexity. Two possible candidates are the number of protein-coding genes, and the richness and variety of morphology and behaviour. Table 2 shows the sizes of the coding regions of various organisms[5]. The trend is fairly robust: eukaryotes have a larger coding genome than prokaryotes, higher plants and invertebrates have a larger genome than protists, and vertebrates a larger genome than invertebrates. The last observation is puzzling: perhaps the nervous system of vertebrates requires the extra genetic information. Unfortunately, the data do not tell us much about structural or functional complexity, because we do not know the mapping between genotype and phenotype.

Bonner[6] measures complexity in terms of the variety of behaviour. For example, the emergence of humans depended on a greater behavioural variety. The point need not be confined to ethology: complexity increases with the diversity of actions an organism can carry out. For example, phagocytosis is a complex behaviour that depends on the eukaryotic cytoskeleton: prokaryotes cannot do it. The number of cell types in an organism can be taken as a measure of its complexity. Unfortunately, it is hard to quantify this aspect of complexity, or to get beyond the common-sense, but rather boring, conclusion that complexity has indeed increased in some lineages.

It is more interesting to list the mechanisms whereby the quantity of genetic information can increase. The three main possibilities—duplication and divergence, symbiosis and epigenesis—are shown in Fig. 1.

Transition from independent replicators

In many of the transitions listed in Table 1 we find the common phenomenon that entities capable of independent replication before the transition can only replicate as parts of a larger whole afterwards. Examples include the origin of chromosomes; the origin of eukaryotes with symbiotically derived organelles; the origin of sex; the origin of multicellular organisms (the cells of animals, plants and fungi are descended from unicellular protists, each of which could survive on its own: today, they exist only as parts of larger organisms); and the origin of social groups. Note that the last two examples differ from the previous ones: the cells of multicellular animals did not form the organism through a symbiosis of independent entities, but they consist of entities (the cells), the analogues of which do exist as independent forms. Thus, units of evolution at the higher level may either be analogous (multicellular organisms) or homologous (eukaryotes) to an 'ecosystem' of lower-level units.

Given this common feature of the major transitions, there is a common question we can ask of them. Why did natural selection, acting on entities at the lower level (replicating molecules, free-living prokaryotes, asexual protists, single cells, individual organisms), not disrupt integration at the higher level (chromosomes, eukaryotic cells, sexual species, multicellular organisms, societies)? The problem is not an imaginary one: there is a real danger that selection at the lower level will disrupt integration at the higher. Some examples are[1]: (1) If Mendel's laws are rigorously obeyed, a gene can only increase its representation in future generations by ensuring the success of the cell in which it finds itself, and of the other genes in the cell. Hence Mendel's laws ensure the evolution of cooperative, or 'coadapted', genes. But the laws are broken, in meiotic drive[7], and by transposable elements[8]. These are examples of the more general phenomenon of intragenomic conflict[9]. (2) A sexual population has an advantage, in rate of evolution, and in the elimination of harmful mutations, over an asexual one. But a parthenogenetic female has, in the short run, a twofold advantage over a sexual one, and parthenogens are not uncommon[10]. (3) A gene in a somatic cell of a plant might best ensure the transmission of replicas of itself by giving rise to a flower bud, even if this reduced the success of the whole plant. (4) A bee colony produces more reproductives if the workers raise the queen's offspring. But workers do lay eggs (which are unfertilized, and hence male)[11].

We cannot explain these transitions in terms of the ultimate benefits they conferred. For example, it may be that, in the long run, the most important difference between prokaryotes and eukaryotes is that the latter evolved a mechanism for chromosome segregation at cell division that permits DNA replication to start simultaneously at many origins, whereas prokaryotes have only a single origin of replication[12]. At the very least, this was a necessary precondition for the subsequent increase in DNA content, without which complexity could not increase. But this is not the reason why the change occurred in the first place: the new segregation mechanism was forced on the early eukaryotes by the loss of a rigid cell wall, which plays a crucial role in the segregation of eubacterial chromosomes. Or, to take a second example, meiotic sex was an important preadaptation for the subsequent evolutionary radiation of the eukaryotes, but it could not have originated for that reason.

The transitions must be explained in terms of immediate selective advantage to individual replicators. We are committed to the gene-centred approach outlined by Williams[13] and made still more explicit by Dawkins[14]. There is, in fact, one feature of the transitions listed in Table 1 that leads to this conclusion. At some point in the life cycle, there is only one copy, or very few copies, of the genetic material: consequently, there is a high degree of genetic relatedness between the units that combine in

TABLE 1 The major transitions[1]

Replicating molecules to populations of molecules in compartments
Unlinked replicators to chromosomes
RNA as gene and enzyme to DNA and protein (genetic code)
Prokaryotes to eukaryotes
Asexual clones to sexual populations
Protists to animals, plants and fungi (cell differentiation)
Solitary individuals to colonies (non-reproductive castes)
Primate societies to human societies (language)

TABLE 2 Genome size and DNA content[5]

	Genome size (base pairs $\times 10^9$)	Coding DNA (%)
Bacterium (*E. coli*)	0.004	100
Yeast (*Saccharomyces*)	0.009	70
Nematode (*Caenorhabditis*)	0.09	25
Fruitfly (*Drosophila*)	0.18	33
Newt (*Triturus*)	19.0	1.5–4.5
Human	3.5	9–27
Lungfish (*Protopterus*)	140.0	0.4–1.2
Flowering plant (*Arabidopsis*)	0.2	31
Flowering plant (*Fritillaria*)	130.0	0.02

the higher organism. The importance of this general principle was first emphasized by Hamilton[15] in his explanation of the evolution of social behaviour, but we believe it to be quite general. To give two other examples: multicellular organisms develop from a single fertilized egg, so that their cells are genetically identical, except for somatic mutation; most eukaryotes inherit their organelles from one parent only, so that the organelles in a single individual are almost always genetically identical[16,17]. We think that a similar principle operated in the origin of the earliest cells[18–20]: this example is discussed further in Box 1.

In several of the listed transitions, one is effectively dealing with a group of replicators: when does such a group qualify as an organism, or—viewed from the level of the component replicators—a 'superorganism'? Wilson and Sober[21] define a superorganism as a "collection of single creatures that together possess the functional organization implicit in the formal definition of organism". They suggest that groups are superorganisms if they satisfy the following criteria[21]: the population consists of several groups; there is a difference between the groups in their contribution of progeny to the next generation (differential group fitness); variation of group fitness is due to heritable genetic variation; individuals within the group have the same fitness.

Obviously, the last criterion cannot hold at the time of origin itself; it is precisely this absence of between-individual, within-group selection that has to be explained. The real question is whether a mechanism, suppressing internal competition, will invade when rare. The answer is known to be yes for certain cases (Box 1). For such a mechanism to spread, selection between groups must be effective. It is most efficient if the following criteria are met[22]: the number of groups must be much larger than that of the units within each group; there is no migration (horizontal transfer) between groups; each group has no more than one parental group.

The effect of these conditions is that there will be genetic differences between groups, but individuals in a single group will be similar. If so, the groups will be units of evolution[23] and will evolve by natural selection.

The principle of the small number of founders is important at the time of the transition. Two other processes—contingent irreversibility and central control—help to explain the maintenance of higher-level entities once they have arisen, although they are less relevant to the origin of such entities[1].

■ **Contingent irreversibility.** If an entity has replicated as part of a larger whole for a long time, it may have lost the capacity for independent replication that it once had, for accidental reasons that have little to do with the selective forces that led to the evolution of the higher-level entity in the first place. For example, mitochondria cannot resume independent existence, if only because most of their genes have been transferred to the nucleus; cancer cells may escape growth control, but have no independent future as protists; worker bees may lay male eggs, but cannot establish a new colony on their own.

The contingent nature of irreversibility is perhaps best illustrated by the reversion from sex to parthenogenesis. Mammals are never parthenogens, probably because, at some loci in some tissues, only the allele inherited from the father is active: hence, in an embryo with no father, some essential gene activities are missing. Gymnosperms are also never parthenogens, perhaps for a different reason: chloroplasts are transmitted in the pollen. Anamniote vertebrates, although they may be parthenogens, always require sperm from the males of another species to initiate development, perhaps because the sperm provides a centriole. The relevance of these sexual hangups—and there are many others—is to show how various and accidental are the reasons why reversal is difficult or impossible.

■ **Central control.** If a 'selfish' mutation occurs in a chromosomal gene, a suppressor mutation at any other locus in the genome would be favoured by selection. Hence the rest of the genome may win the contest, not because of any analogue of majority voting, but because of the large number of loci, and hence of possible suppressor mutations, that are available for each selfish mutation. It may be relevant that attempts to use driving chromosomes in biological control have so far failed because of the rapid evolution of suppression. It is in this sense that Leigh's idea[24] of a "parliament of genes" should be understood.

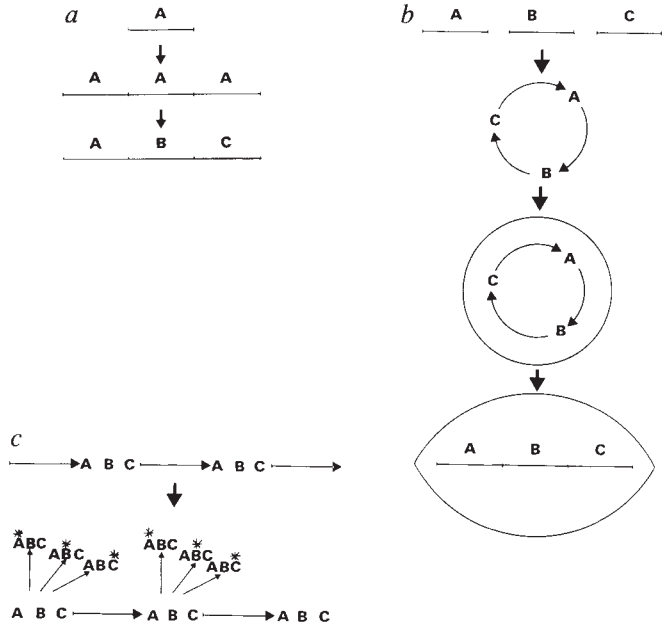


FIG. 1 Processes whereby the quantity of information can increase during evolution. *a*, Duplication followed by divergence: this is the main process whereby information increases between the major transitions. *b*, Symbiosis: the figure illustrates first a set of independent replicators, then a hypercycle[60], in which the replicators interact to form a stable ecological cycle, then the enclosure of the hypercycle in a compartment, and finally the physical linkage of replicators, so that when one replicates, all do. *c*, Epigenesis: A, B and C are different genes; asterisks indicate states of gene activity transmitted through cell division.

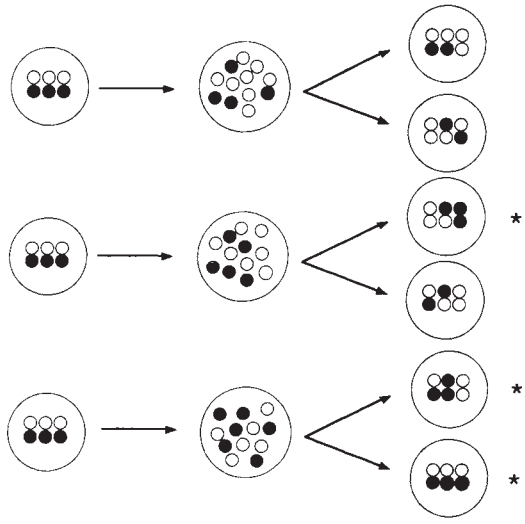
BOX 1 From naked genes to compartments to chromosomes

THE idea that the primordial genome must have consisted of unlinked genes comes from a paradox of Eigen. He demonstrated that the tolerable mutational load of a population sets an upper limit to the length of the genome: it is proportional to the reciprocal of the mutation rate per base per generation (the error threshold)[60]. Early genomes could not have been much longer than a contemporary transfer RNA owing to the low fidelity of replication. A population in mutation–selection balance of such molecules (a so-called quasispecies[61]) cannot harbour a set of sufficiently dissimilar genes (to encode different functions) in appreciable concentration. Therefore, a collection of unrelated and unlinked genes is needed, but they will compete with each other until only one gene (with the highest replication rate) survives, and in turn the genome is doomed to extinction; hence the paradox that long chromosomes are unstable because of excessive mutational load, and a set of small genes is unstable because of internal competition. As Eigen recognized, some functional coupling among the genes is necessary[60].

A possible resolution of Eigen's paradox is the 'stochastic corrector' model[18–20] (figure). The assumptions are as follows: (1) Unlinked genes replicate in compartments. There are two types of gene, which replicate at different expected rates, so that there is between-gene selection within compartments. (2) Compartments reproduce when the number of genes they contain has doubled. The rate of compartment growth depends on the kinds of genes it contains, and is fastest when different kinds are present. Thus different genes contribute non-additively to compartment fitness, as emphasized in our discussion on the division of labour. (3) Replication is a stochastic process and assortment of genes into offspring compartments is random.

Given these conditions, there is efficient group selection at the compartment level. Despite internal competition, natural selection maintains a stable compartment distribution, and neither type of gene is lost. This is due to the stochastic processes generating variation between compartments, on which selection—between the protocells—can act.

The spread of chromosomes has also been analysed in the context of the stochastic corrector model. Chromosomes (linking complementing genes), when introduced in small numbers into some protocells of a simulated population, are established in the population, despite a twofold within-cell replicative disadvantage relative to individual genes[62]. The reasons are first, that linkage is a safeguard against internal competition of genes, as one cannot replicate without the other, and second, that it pays for a gene to sit on a chromosome, because it does not then run the risk of finding itself, after cell division, in a cell with low fitness due to the absence of its complementing partner. The chemical feasibility of this transition has been worked out[63], resting in part on the 'genomic tag' model[64], which argues for an ancestral role of tRNA-like structures as signals for RNA replication.



The stochastic corrector model[18–20]. Empty and filled circles represent two kinds of gene; the former have an average within-cell replicative advantage, but all genes replicate faster in cells with equal numbers of the two kinds. Stochastic processes (in replication and cell division) ensure the reappearance of cells, marked with asterisks, with the optimal gene composition, despite contrary within-cell selection.

The division of labour

The most familiar examples of the advantages arising from a division of labour concern caste differentiation in the social insects. Bell[25] applied Adam Smith's ideas to a less familiar example, cell differentiation in the Volvocales[26]. The specific name *Volvox weismannia* is a reminder that these algae are an excellent example of the segregation of germ line and soma. Most members of the order possess only a single cell type, which fulfils all vegetative and reproductive functions. In *Pleodorina*, there is partial division of labour: some cells start with vegetative functions, but later differentiate into gonidia (asexual propagules). The genus *Volvox* has a *bona fide* segregation of germ line and soma: germ cells are immotile in the centre of the spheroid, and somatic cells bear cilia but cannot divide. The benefit of differentiation has been demonstrated: colonies produce a larger bulk of smaller offspring than do single cells of a similar size.

One precondition for the division of labour in the Volvocales is that motile cells cannot divide, and mitosing cells cannot move, because the same organelles are used either as basal bodies or as centrioles[27]. A similar argument was used by Buss[28] for the flagellated blastulae of the lower metazoa.

Some other cases in which a division of labour is evident are[1,29]: (1) The evolution of many specific enzymes from a set of multifunctional low-efficiency enzymes. If the first protocells were equipped with a few multifunctional enzymes, more efficient enzymes could evolve only by duplication and divergence[30]. (2) In the RNA world, RNA served both as genetic material and catalyst: today, DNA is the genetic material, and most enzymes are proteins[31]. (3) In prokaryotes there is a single cell compartment, whereas in eukaryotes the genetic nucleus and metabolic cytoplasm are separated, and additional organelles have evolved, some recruited symbiotically[2]. (4) In sexual populations, isogamy has repeatedly evolved to anisogamy, with differentiated sperm and ova[10]. (5) Hermaphrodites are replaced by separate sexes: the most convincing explanation for why some organisms are hermaphrodite and some dioecious is in terms of the advantages of a division of labour[32].

If cooperation is to evolve, non-additive, or synergistic, fitness interactions are needed. If two or more cooperating individuals can achieve something that a similar number of isolated individuals cannot, the preconditions exist: the image to bear in mind is that two men, each with one oar, can propel a boat, but one man with one oar will row in circles[1]. But the dangers of intragenomic conflict remain: both relatedness and synergistic fitness interactions are likely to be needed.

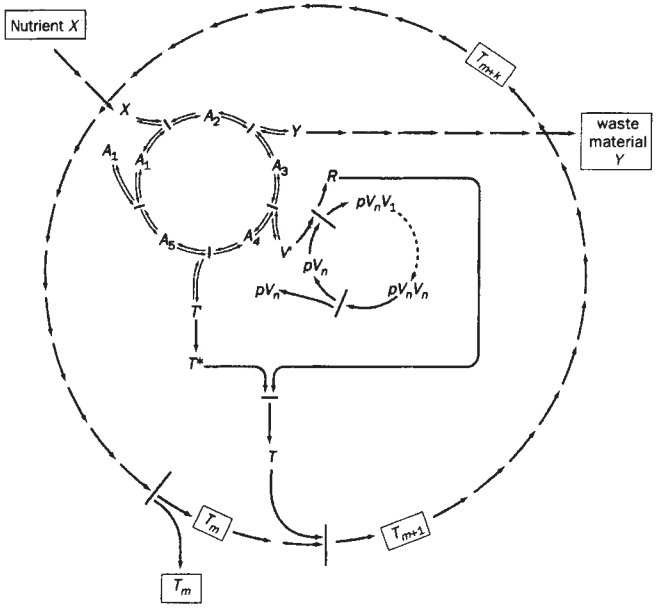
The evolution of heredity

Heredity means that like begets like: it requires some means whereby information can be transmitted. A crucial distinction is between systems of 'limited heredity', in which only a few distinct states can be transmitted, and systems of 'unlimited heredity', capable of transmitting an indefinitely large number of messages. We suggest the following stages[1].

(1) The origin of simple autocatalytic systems with limited heredity[33–35]. Autocatalysis, whereby a single molecule gives rise to two molecules of the same kind, is essential for growth, but does not by itself imply heredity, which requires that, if the nature of the initial molecule is changed, two molecules of the new kind are produced. Several authors have suggested that

BOX 2 The chemoton as a sensible protocell model and its importance in explaining the first major transitions

THE basic model of the chemoton[34,54,65] (figure) consists of three subsystems: the metabolic 'engine', which is an autocatalytic cycle; a self-replicating template macromolecule; and a bilayer membrane. The autocatalytic cycle produces the building blocks of the two other subsystems as well, at the expense of the energy and material difference between X and Y. The condensation byproduct R serves as a stringent stoichiometric coupling between template polycondensation and membrane growth. It is easy to see that the whole system grows in synchrony. Division is a more tricky problem. There are calculations (which need to be verified experimentally) showing that a chemical system with the described couplings would indeed undergo spontaneous fission into two offspring compartments, owing to the interplay between growth, osmotic relations and surface tension of the membrane[66,67]. The two questions we will discuss in turn concern the realistic feasibility of the subsystems and how our central themes manifest themselves in the origin and evolution of such a system.



The chemoton[54]. The metabolic subsystem, with intermediates A_i, is an autocatalytic chemical cycle, consuming X as nutrient and producing Y as waste material; pV_n is a polymer of n molecules of V, which undergoes template replication; R is a condensation byproduct of this replication, needed to turn T' into T, the membranogenic molecule; the symbol T_m represents a bilayer membrane composed of m units made of T molecules. It can be shown that such a system can grow and divide spontaneously.

A remarkable example of an autocatalytic network is Butlerow's formose 'reaction', synthesizing sugars out of formaldehyde with the catalytic aid of pre-existing sugars (see for example, ref. 68). Other, still non-enzymatic, cycles were suggested as variations on the theme of the reductive citric acid cycle[69], as yet without experimental evidence. The origin of RNA-like self-replicating molecules is still a problem, as discussed in the main text (compare ref. 31). It is worth emphasizing the special kinetic effects within the chemoton, however: replication occurs only upon reaching a certain threshold concentration of V within the compartment, and this happens only once during the protocell cycle[34]. The autocatalytic formation of membranes without enzymes is now proven[70]. Experiments should now concentrate on the division mechanism.

As to our common themes, the following considerations are worth noting.

Complexity. Nobody thinks that a simple cycle in its drawn form could be realistic. Inevitably, one should have a network. The increase is complexity of such a network is an open problem. Chemical symbiosis[71], or the grafting of novel extensions onto the pre-existing network[35] could have resulted in heritable, 'macroevolutionary' changes in the system. Apart from this, it is a family of templates, arising by mutation, duplication and divergence that can lead to a complex set of templates, which make use of digital information, first in the form of ribozymes catalysing steps of the metabolic network, and later as protein-coding genes.

Division of labour. It is common that symbiotic partners provide complementary metabolic 'toolkits' for the new unit[2]. The chemoton's subsystems serve exactly such complementary roles, and their unit can be regarded as a very special case of chemical symbiosis. It is obvious that the membrane itself would be an inferior metabolic subsystem, and that templates could provide only leaky boundaries, so indeed there is an advantage in the union of specialized subsystems: molecular 'jacks-of-all-trades' are replaced by 'masters'.

Competition of replicators. The organization of the basic chemoton model is such that the complementary subsystems cannot get "out of phase"[34,54]. This is not so within subsystems, most notably for the digital information carriers[19,20]. Through microevolution, selfish mutants can arise. It is the stochastic corrector principle (Box 1) that can prevent the system as a whole from deteriorating.

Heredity. Two of the subsystems (the membrane and the metabolic cycle) carry only analog information[72] and are at the most limited hereditary replicators. It is the realistic versions of the pV_n that can provide the system with unlimited heredity, because of their digital information[72]. First this is used for ribozymic activity, then in translation.

autocatalytic networks could have this property, but at best they could display limited heredity, with only a few molecular types able to reproduce themselves.

(2) The origin of polynucleotide-like molecules, providing unlimited heredity[31]. This transition has proven surprisingly difficult to explain. Even if one assumes the presence of all the necessary chemical constituents, there are severe obstacles to continued replication, such as enantiomeric cross-inhibition (mirror-image building blocks pair but do not form a covalent link with the growing chain[36]) and non-separation of template and replica due to the many hydrogen bonds formed between them. Oligonucleotide replication is a possible intermediate stage leading to that of polynucleotides. Most such experiments use chemical analogues of oligonucleotides (such as the first successful attempt by von Kiedrowski[37]). The short length allows for the spontaneous dissociation ('melting') of template and copy, so ongoing replication is possible, although an increased concentration is unfavourable for the latter because two complete strands find each other more readily. This results in a parabolic (subexponential) growth of such replicators[37], which in a competitive situation leads to a stable "survival of everybody"[38,39] (see also ref. 40 for review). "Survival of the fittest" needs an exponential growth tendency[39] and therefore more efficient strand separation. The latter could have been accomplished by RNAs with replicase function.

(3) The origin of the genetic code in the context of the RNA world, before translation. The essence of the code is that specific amino acids should be attached to specific oligonucleotides: today, it depends on the attachment of amino acids to transfer RNA molecules. Several workers in the field have realized that translation and coding are difficult to evolve simultaneously. One way to avoid such an evolutionary trap is preadaptation: rudiments of a complex adaptation may have evolved by selection for some other function. Concrete versions of this idea suggest that aminoacylation helped the replication of RNA, or that peptide-specific ribosomes with an internal message antedate general protein synthesis using external templates (messenger RNA). The idea that we favour is that amino acids were used as coenzymes of ribozymes, and were equipped with unambiguous trinucleotide handles, enabling them to bind to a ribozyme by base pairing[41]. Such handles would enable the same amino acid to be used by several ribozymes. Each new amino acid that acquired a specific handle would increase the enzymatic versatility of the organism, so that the difficulty of a complete adaptation being acquired in a single step largely disappears.

(4) The origin of translation and encoded protein synthesis. The details of this transition are discussed in ref. 1.

(5) The replacement of RNA by DNA as the genetic material could well have happened before the origin of genetic code, because it is chemically a much less complicated transition[42]. The primary selective force for this may have been the increased chemical stability of thymine (as opposed to uracil) and deoxyribose (as opposed to ribose)[43]. The usual argument that there is no mismatch repair or repair of damage in RNA misses the point that, chemically, all these processes would be feasible in double-stranded RNA.

(6) The emergence of hereditary regulative states in prokaryotes and simple eukaryotes. Already in prokaryotes, patterns of methylation are transmitted through cell division and can be responsible for states of phenotypic differentiation. Thus there is a dual inheritance system, in which heredity depends either on differences in DNA sequence or on transmissible states of gene activation[44,45]. Such a system is crucial for the development of animals and plants, but what selective forces were responsible for its evolution in single-celled organisms? Jablonka suggests that it was the need for protists to adapt to regular changes in the environment, the timescale of which was too large in comparison with generation times, and too small relative to the time required for typical evolutionary changes[44]. In this case, a heritable mark M1 on some gene could have been beneficial in environment E1, and an alternative mark M2 (maybe simply the lack of M1) could have been beneficial in environment E2. An alternative suggestion is that morphological and physiological adaptations of sexual protists could have been preadaptations for simple forms of multicellularity, as alternative phenotypes, specific cell adhesion, cell-to-cell signalling and cell-division arrest play a crucial role in both[46].

(7) The evolution of epigenetic inheritance with unlimited heredity: the emergence of animals, plants and fungi. The transition to multicellular organisms with many kinds of differentiated cells occurred on three occasions, suggesting that it may not have been particularly difficult. This would be explained if the main cellular novelty required was an epigenetic inheritance system, as this existed already in protists. If so, the emergence and radiation of the metazoa had to wait only for suitable environmental conditions[47].

(8) The emergence of proto-language in *Homo erectus*—a cultural inheritance system with limited potential in which, because of the absence of grammar, only certain types of statement can be made[48].

(9) The emergence of human language with a universal grammar[49] and unlimited semantic representation[50]. Grammar enables a speaker with a finite vocabulary to convey an indefinitely large number of meanings, just as the genetic code enables DNA to specify an indefinitely large number of proteins. We accept Chomsky's argument that grammatical competence is unique, both in the sense of being peculiar to humans, and of being special to language, and not merely an aspect of general learning ability. But we are puzzled by the reluctance of many linguists, including Chomsky himself, to think about the evolution of this competence. The objection takes the form of asserting, not only that human language is different in kind from animal communication, but that no intermediate is possible between the two.

It is argued that any rudimentary form of grammar would not allow one to generate some types of sentence. This is true but irrelevant: by analogy, it is better to have some light-sensitive cells than none at all; a perfect eye is not the only useful solution to the problem[51].

It is in fact rather easy to think of intermediates between protolanguage and true language. There remains the question of the evolutionary origin of grammatical novelties. It is reasonable to assume that this happened by genetic assimilation[51], new rules being made up by individuals as non-genetic innovations, then learnt by members of the community, then hard-wired into the 'language organ' subsequently. It has been demonstrated that learning and selection can lead to such an assimilation in extreme cases when the latter alone could not get anywhere[52]: learning can transform an initially flat fitness landscape with a needle-like peak into a well-behaved Fujiyama-like one.

Perhaps the most convincing evidence both for the belief that grammatical competence is to some degree independent of general learning ability, and for the possibility of functional intermediates between no grammar and perfect grammar, comes from studies of hereditary variation in linguistic competence. One remarkable case involves a family in which a so-called feature-blind dysphasia seems to be inherited in a mendelian fashion, a single dominant gene being responsible[53]. Members cannot automatically generate plurals and past tense. Although they understand the meaning of plural and past perfectly well, they have to learn each new case anew: *paint* and *painted*, *book* and *books* must be learned separately (in the case of exceptions such as *go* and *went*, we must do the same). To be sure, this is not a genetical violation of one of Chomsky's rules, but it demonstrates that there can be something useful between perfect grammar and protolanguage: it also holds out the hope that we will in future be able to dissect language genetically, as we are today dissecting development.

Constructive evolution

Although some of the key intermediate stages of evolution seem to have vanished, their experimental recreation could teach us a lot. Examples include:

- The *de novo* synthesis of a living chemical system, such as the chemoton[54] (see Box 2 for a summary of this idea, and how several of our discussed points integrate into a unified picture at a certain level of organization).
- *In vitro* construction of a truly self-replicating RNA.
- *In vitro* generation of ribozymes, using amplification and selection by affinity chromatography[19,55]. The first such example has been given[56]. In a similar vein, the generation of RNA molecules of importance for primordial coding and translation, essentially by the same protocol, should provide us with useful information about feasible scenarios of the code's origin[19,55]. Recently Famulok reported the *in vitro* selection of an RNA binding ornithine and citrulline[57]. Similar tests using proteinogenic amino acids would be welcome.
- The establishment of artificial symbioses should help to clarify several aspects of some of the transitions. A first example is Jeon's bacteria, originally parasitizing an amoeba, which became obligatorily dependent on these bacteria later[58].
- Finally, recreation of extant species or forms may be in certain cases possible. The recreation of a fossil fern species from genomes of extant polyploids is a remarkable example[59].

Conclusions

A central idea in contemporary biology is that of information. Developmental biology can be seen as the study of how information in the genome is translated into adult structure, and evolutionary biology of how the information came to be there in the first place. Our excuse for writing an article concerning topics as diverse as the origins of genes, of cells and of language is that all are concerned with the storage and transmission of information. The article is more an agenda for future research than a summary of what is known. But there is sufficient formal similarity between the various transitions to hold out the hope that progress in understanding any one of them will help to illuminate others. □

Eörs Szathmáry is in the Collegium Budapest (Institute for Advanced Study), Szentháromság u. 2., H-1014 Budapest and Ecological Modelling Research Group, Department of Plant Taxonomy and Ecology, Eötvös University, Ludovika tér 2, H-1083 Budapest, Hungary; John Maynard Smith is at the School of Biological Sciences, The University of Sussex, Brighton BN1 9QG, UK.

1. Maynard Smith, J. & Szathmáry, E. *The Major Transitions in Evolution* (Freeman, Oxford, 1995).
2. Margulis, L. *Symbiosis in Cell Evolution* (Freeman, San Francisco, 1981).
3. Smith, A. *The Wealth of Nations* (1777).
4. Rensch, B. *Evolution above the Species Level* (Wiley, New York, 1966).
5. Cavalier-Smith, T. (ed.) *The Evolution of Genome Size* (Wiley, Chichester, 1985).
6. Bonner, J. T. *The Evolution of Complexity by Means of Natural Selection* (Princeton University Press, Princeton, NJ, 1988).
7. Crow, J. F. *BioEssays* **13,** 305–312 (1991).
8. Charlesworth, B., Sniegowski, P. & Stephan, W. *Nature* **371,** 215–220 (1994).
9. Hurst, L. *Proc. R. Soc. Lond.* B **248,** 135–140 (1992).
10. Maynard Smith, J. *The Evolution of Sex* (Cambridge University Press, Cambridge, 1978).
11. Wilson, E. O. *Sociobiology: The New Synthesis* (Belknap, Harvard University Press, Cambridge, Massachusetts, 1975).
12. Cavalier-Smith, T. *Ann. N. Y. Acad. Sci.* **503,** 17–54 (1987).
13. Williams, G. C. *Adaptation and Natural Selection* (Princeton University Press, Princeton, NJ, 1966).
14. Dawkins, R. *The Selfish Gene* (Oxford University Press, Oxford, 1976).
15. Hamilton, W. D. *J. theor. Biol.* **7,** 1–52 (1964).
16. Hurst, L. D. & Hamilton, W. D. *Proc. R. Soc. Lond.* B **247,** 189–194 (1992).
17. Hutson, V. & Law, R. *Proc. R. Soc. Lond.* B **263,** 43–51 (1993).
18. Szathmáry, E. & Demeter, L. *J. theor. Biol.* **128,** 463–486 (1987).
19. Szathmáry, E. *Oxf. Surv. Evol. Biol.* **6,** 169–205 (1989).
20. Szathmáry, E. *Trends Ecol. Evol.* **4,** 200–204 (1989).
21. Wilson, D. S. & Sober, E. *J. theor. Biol.* **136,** 337–356 (1989).
22. Leigh, E. G. *Trends Ecol. Evol.* **6,** 257–262 (1991).
23. Maynard Smith, J. *Proc. R. Soc. Lond.* B **219,** 315–325 (1983).
24. Leigh, E. G. *Adaptation and Diversity* (Freeman, Cooper and Co., San Francisco, 1971).
25. Bell, G. in *The Origin and Early Evolution of Sex* (eds Halvorson, H. O. & Mornoy, A.) 221–256 (Liss, New York, 1985).
26. Kirk, D. L. *Trends Genet.* **4,** 32–36 (1988).
27. Koufopanou, V. *Am. Nat.* **143,** 907–931 (1994).
28. Buss, L. *The Evolution of Individuality* (Princeton University Press, Princeton, NJ, 1987).
29. Molnár, I. *Abstr. botanica (Budapest)* **17,** 207–224 (1993).
30. Kacser, H. & Beeby, R. *J. molec. Evol.* **20,** 38–51 (1984).
31. Joyce, G. *Nature* **338,** 217–224 (1989).
32. Charnov, E., Maynard Smith, J. & Bull, J. J. *Nature* **263,** 125–126 (1976).
33. Ycas, M. *Proc. natn. Acad. Sci. U.S.A.* **41,** 714–716 (1955).
34. Gánti, T. *A Theory of Biochemical Supersystems* (Akadémiai Kiadó, Budapest and University Park Press, Baltimore, 1979).
35. Wächtershäuser, G. *Microbiol. Rev.* **52,** 452–484 (1988).
36. Joyce, G. F., Schwartz, A. W., Orgel, L. E. & Miller, L. S. *Proc. natn. Acad. Sci. U.S.A.* **84,** 4398–4402 (1987).
37. von Kiedrowski, G. *Angew. Chem. Inter. Ed.* **25,** 923–935 (1986).
38. Szathmáry, E. & Gladkih, I. *J. theor. Biol.* **138,** 55–58 (1989).
39. Szathmáry, E. *Trends Ecol. Evol.* **6,** 366–370 (1991).
40. von Kiedrowski, G. *Bioorg. Chem. Frontiers* **3,** 113–146 (1993).
41. Szathmáry, E. *Proc. natn. Acad. Sci. U.S.A.* **90,** 9916–9920 (1993).
42. Benner, S. A. *et al. Cold Spring Harbor Symp. quant. Biol.* **52,** 56–63 (1987).
43. Lazcano, A., Guerrero, R., Margulis, L. & Oró, J. *J. molec. Evol.* **27,** 283–290 (1988).
44. Jablonka, E. *J. theor. Biol.* **170,** 301–309 (1994).
45. Jablonka, E. & Lamb, M. J. *Epigenetic Inheritance and Evolution: The Lamarckian Dimension* (Oxford University Press, Oxford, 1995).
46. Szathmáry, E. *J. theor. Biol.* **169,** 125–132 (1994).
47. Wolpert, L. *Biol. J. Linn. Soc.* **39,** 109–124 (1990).
48. Bickerton, D. *Language and Species* (University of Chicago Press, Chicago, 1990).
49. Chomsky, N. *Aspects of the Theory of Syntax* (MIT Press, Cambridge, MA, 1965).
50. Pinker, S. *The Language Instinct—The New Science of Language and Mind* (Penguin, London, 1994).
51. Pinker, S. & Bloom, P. *Behav. Brain. Sci.* **13,** 707–784 (1990).
52. Hinton, G. E. & Nowlan, S. J. *Complex Syst.* **1,** 495–502 (1987).
53. Gopnik, M. *Nature* **344,** 715 (1990).
54. Gánti, T. *The Principle of Life* (OMIKK, Budapest, 1987).
55. Szathmáry, E. *Nature* **344,** 115 (1990).
56. Prudent, J. R., Uno, T. & Schultz, P. G. *Science* **264,** 1924–1927 (1994).
57. Famulok, M. *J. Am. chem. Soc.* (in the press).
58. Jeon, K. W. in *Symbiosis as a Source of Evolutionary Innovation* (eds Margulis, L. & Fester, R.) 118–131 (MIT Press, Cambridge, MA, 1991).
59. Rasbach, H., Rasbach, K., Reichstein, J. J. & Vida, G. *Ber. Bayer, Bot. Ges.* **50,** 23–27 (1979).
60. Eigen, M. *Naturwissenschaften* **58,** 465–523 (1971).
61. Eigen, M. & Schuster, P. *Naturwissenschaften* **64,** 541–565 (1977).
62. Maynard Smith, J. & Szathmáry, E. *J. theor. Biol.* **164,** 437–446 (1993).
63. Szathmáry, E. & Maynard Smith, J. *J. theor. Biol.* **164,** 447–454 (1993).
64. Weiner, M. & Maizels, N. *Proc. natn. Acad. Sci. U.S.A.* **91,** 6729–6734 (1994).
65. Gánti, T. *BioSystems* **7,** 189–195 (1975).
66. Koch, A. L. *J. molec. Evol.* **21,** 270–277 (1985).
67. Tarumi, K. & Schwegler, H. *Bull. Math. Biol.* **47,** 307–320 (1987).
68. Cairns-Smith, A. G. & Walker, G. L. *BioSystems* **5,** 173–186 (1974).
69. Wächtershäuser, G. *Progr. Biophys. molec. Biol.* **58,** 85–201 (1992).
70. Bachmann, P. A., Luigi, P. L. & Lang, J. *Nature* **357,** 57–59 (1992).
71. King, G. A. M. *BioSystems* **13,** 23–45 (1980).
72. Wächtershäuser, G. *Proc. natn. Acad. Sci. U.S.A.* **91,** 4283–4287 (1994).

ACKNOWLEDGEMENTS. This work has partly been supported by the Hungarian National Scientific Research Fund (OTKA).

ARTICLES

A mechanism for decoupling within the oceanic lithosphere revealed in the Troodos ophiolite

Susan M. Agar* **& Kim D. Klitgord†**

* Northwestern University, Evanston, Illinois 60208, USA
† US Geological Survey, Menlo Park, California 94025, USA

Contrasting kinematic histories recorded in the sheeted dykes and underlying plutonic rocks of the Troodos ophiolite provide a new perspective on the mechanical evolution of oceanic spreading centres. The kinematic framework of the decoupling zone that partitions deformation between the sheeted dykes and plutonics contrasts with low-angle detachment models for slow-spreading ridges based on continental-rift analogues. A model for the generation of multiple, horizontal decoupling horizons, linked by planar normal faults, demonstrates new possibilities for the kinematic and rheological significance of seismic reflectors in oceanic lithosphere.

IMPROVED acoustic images of the oceanic lithosphere have spawned new hypotheses on its architecture and evolution[1–3]. Sub-horizontal reflectors have been interpreted as lithological transitions, metamorphic fronts, the roofs of active or fossil magma chambers and zones of magmatic underplating[4–10]. Dipping reflectors have been equated with magma chamber margins[11] and faults[1,5,10]. Some of these 'faults' appear to shallow at depth into sub-horizontal reflectors and have been interpreted as low-angle detachment surfaces either within seismic layer 3 (the lower crust) or along the Moho[1,5,10]. Mechanical models of sea-floor spreading centres have attempted to integrate these subsurface features with fault geometries and kinematics interpreted from sea-floor morphology[1,3,12]. Recognized limitations to such an integration are the sparse subsurface features that can be traced to the sea floor, the ambiguous origins of reflectors and the lack of constraints on fault displacement vectors and timing. Consequently, spreading-centre models have incorporated geometries and kinematics established for continental extensional fault systems[1,13–20]. Low-angle detachments, which arise at brittle–ductile transitions, have been proposed as a mechanism for the exhumation of deep crust and mantle sections on rift flanks during amagmatic cycles[15–20]. The test for such models depends on critical evaluations of the distribution, timing and magnitude of deformation in the lithosphere.

Ophiolites provide the three-dimensional exposures and age relations needed to complement seismic images and drill holes