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pp.21-37 **Return to CONTENTS**

Selection, Adaption and Evolution

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Nothing in biology makes any sense except in the light of evolution. (Dobzhansky, 1973)

In a way, evolution proceeds like a tinkerer who, during millions of years, has slowly modified his products, retouching, cutting, lengthening, using all opportunities to transform and create. Jacob (1983)

ON THE SHOULDERS OF A GIANT

As Dobzhansky (1973) so rightly stressed, the realization of evolution forms the very core of modern biology. We are used to understanding how good "design" of organisms comes about through evolution, but sometimes forget that design flaws are also understandable as arising the same way. As Jacob stresses, the processes of evolution work with what has gone before rather than producing new organisms afresh when faced with new niches or other evolutionary opportunities. Dawkins (1982) notes two examples of poor design whose origins are readily understandable in evolutionary terms: the contorted faces of flatfish, and the long detour taken by the recurrent laryngeal nerve in a giraffe's neck. It is no wonder, even leaving aside the evidence from natural and experimental populations, that scientists abandoned special creation of any kind as a general explanation for the living world.

As Stent (1972) notes, progress in science, as in art, has a certain inevitable aspect to it: an advance not made by person A today will be made by person B tomorrow. But occasional individuals make their advances with such thoroughness that we can be certain that, without them, we would have to have waited much longer to reach where we are today. Such a person was Charles Darwin, who not only generated an explanatory theory for evolution that is quite modern, but amassed such a treasure trove of data that the survival of the new paradigm could not be long in doubt. In Simpson's (1964) phrase, modern biology is truly the "world into which Darwin led us".

Darwin's vision and breadth of knowledge were awesome, as anyone reading through his works can testify. Not only did he establish the main outlines of evolution, but he clearly foreshadowed such modern topics as variation in evolutionary rates (as discussed by Penny, 1983), and even the whole field of sociobiology (not only and obviously in "The descent of man" but also in his discussions of social insects in "The origin of species").

Darwin's vision was far-reaching, but today, standing on his shoulders (to paraphrase Newton), we see much farther than he could possibly do (a point sometimes missed by philosophers of science). To acknowledge that our vision is different, even for concepts such as natural selection,

clearly does nothing to reduce our appreciation of Darwin: stasis in evolutionary understanding would be a failure, and Darwin did not fail.

GENETICS, CHANCE, AND NECESSITY

The greatest single cause of advance in understanding evolutionary mechanisms since Darwin has been the rise of genetics. Evolution is clearly understood now as genetic change in a lineage, and evolutionists generally engage in studying the causes and consequences of such change. Both cause and consequence can, of course, be complex, as I will try to make clear below.

Is there a role for chance in this view of life's history? Yes, and not just one role. One role is that of constant origin through mutation of new alleles differing so trivially in effect from others that their fixation is due to chance. Such a process is envisaged by the Neutralist school of molecular evolutionists as the main one giving rise to molecular change (Kimura, 1983), whereas other evolutionists suggest that the neutralist domain is relatively minor.

The other role for chance concerns the ultimate effects of early choices: whether a population takes one fork in the evolutionary road instead of another may be a matter of chance, yet the two roads may lead to quite different results. Such early choices have a considerable effect, not only on the adaptations that are later available to the population, but also on the way in which different organisms adapt. Monocotyledons and dicotyledons have both evolved tree growth forms on occasion, but in different ways: palms are of necessity constructed very differently from true trees because they are monocotyledons, and monocotyledons lack the ability for secondary strengthening expressed by dicotyledons (Maynard Smith et al., 1985).

Monocotyledons have to some extent overcome the basic constraints of their architecture by evolving tree forms in their own way, but Gould (e.g., 1980) has frequently stressed that such constraints will often be insuperable, limiting the possible array of forms. The concept of constraint is a useful one, in that it must sometimes be correct (Bull and Charnov, 1985) and in that it can lead to tests and be falsified on occasion. For example, LaBarbera (1983) showed that, contrary to general belief, the scarcity of wheels among higher organisms is not because they can't evolve them, but because they are less efficient than the observed forms of transport (moreover, where they are more efficient, they have in fact evolved). Apart from neutralist explanations for morphology and life-pattern, there are therefore three basic kinds of explanation of why certain features are seen and others are not (Crozier and Page, 1985): *structuralist* (absent features are either impossible or the way to them is blocked by impossible intermediates), *historicist* (there has not been sufficient time for the missing features to have been evolved, or only some of the adaptive peaks are occupied so far), and, of course, *adaptationist* (missing features confer less fitness than the ones that are present). Structuralist and adaptationist examples overlap, because an impossible phenotype is certainly also lethal, and nearby phenotypes, even if not impossible, are almost certainly of low fitness. For example, a clam shell which cannot open would be a lethal combination, and ones that can only open a tiny fraction would be of low fitness.

Of necessity, I will not spend further time here discussing chance, but rather concentrate on the process of adaptation, the most interesting aspect of evolution to most people.

ADAPTATION AND RAPID EVOLUTION

Adaptation is the evolution of characteristics better suiting an organism to its environment. Or perhaps, in view of considerations to be dealt with further below, adaptation is best describable

more vaguely as evolution better suiting whatever is evolving to its environment. [*Of course*, we can also speak of "an adaptation", as a characteristic that strongly favors the survival of an organism in its environment, and even as "adaptation" as being the state reached by adaptive evolution, but I will try to stick to just the one meaning in this essay.]

Clearly, adaptation is close to just being evolution driven by natural selection, which in the longer term is the replacement of one favorable mutation by another even more favorable (in Monod's (1972) terms, the action of necessity on the products of chance). The distinction is worth retaining because evolution can lead to lower fitness for a lineage or population (Blick, 1977; Crozier 1979; Paquin and Adams, 1983); I won't discuss these cases here either, save to point out that Darwin, lacking knowledge of modern genetics, did not realise that selection could lead to lower fitness. Here especially we see further than Darwin, standing on his shoulders.

Darwin had, of necessity, to infer that adaptation occurs by documenting the end results and by considering the Malthusian pressures certain to bring it about. For humans to observe adaptation in progress, it has to be rapid; since Darwin's time both strong selection and rapid adaptive evolution have been observed many times. It is worthwhile looking at some of these instances.

Most of the cases of rapid evolution that we know of have been driven by human activities. This happenstance is understandable from standard evolutionary theory, because a changing environment is the simplest cause of strong selection, and we are the major perturbing force on the planet today. Some of these cases involve introductions, such as of the house sparrow, *Passer domesticus*, into North America.

Winter stresses, particularly due to storms, form a major component of selection acting on North American sparrow populations (Bumpus, 1899; Fleischer and Johnston, 1982; Lande and Arnold, 1983). These winter stresses lead to both stabilizing and directional selection on the birds, and fall differently on males and females (Fleischer and Johnston, 1982).

From their reanalysis of Bumpus's (1899) data, Lande and Arnold (1983) estimated that, in females, departure by one standard deviation from the mean in either direction for the first principal component led to a decrease in relative fitness by 45%, and directional selection showed similar but lesser strength for several morphological measures (Lande and Arnold, 1983; Fleischer and Johnston, 1982). We would therefore not be surprised to find that significant changes had emerged in house sparrow populations since their introduction, and indeed such changes have occurred. Not only have the various North American populations differentiated markedly since introduction (Johnston and Selander, 1971), but also similar (but not yet identical) trends in variation with climate have emerged to those seen in the ancestral European populations (Murphy, 1985; Figure 1).

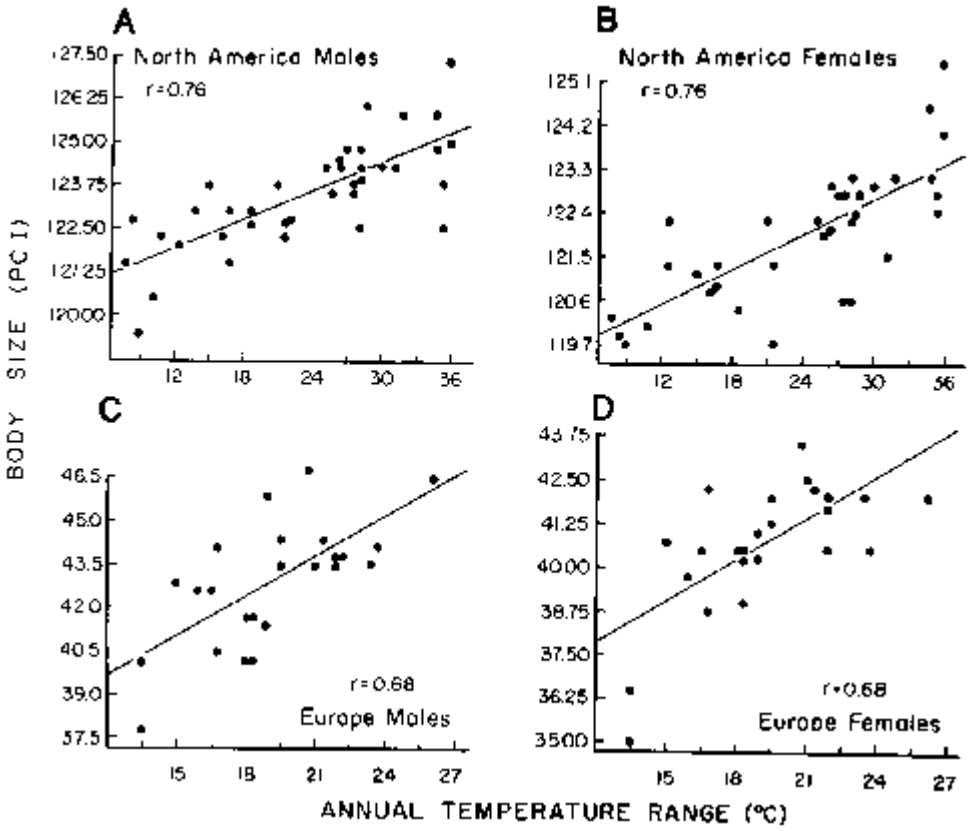

Fig. 1. Body size of house sparrows (divided according to origin and sex) versus annual temperature range. Size was measured by the first principal component derived from 14 skeletal measurements. Each point plotted represents the mean of a locality at which at least ten individuals were measured. Taken from Murphy (1985), reprinted with permission.

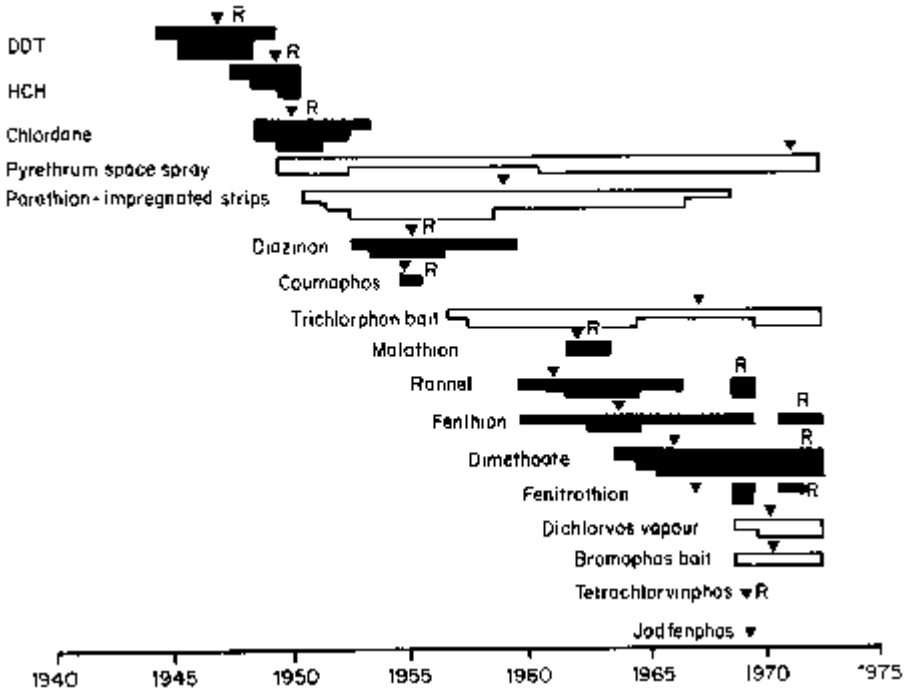

Fig. 2. Evolution of resistance to insecticides by houseflies on Danish farms. The width of each bar reflects the extent of use, the inverted triangle symbol the date of the first confirmed case of resistance of a given insecticide, and the letter R the date when most populations were resistant. Open bars refer to residue sprays and solid ones to other methods of application. From Wood and Bishop (1981), reprinted by permission.

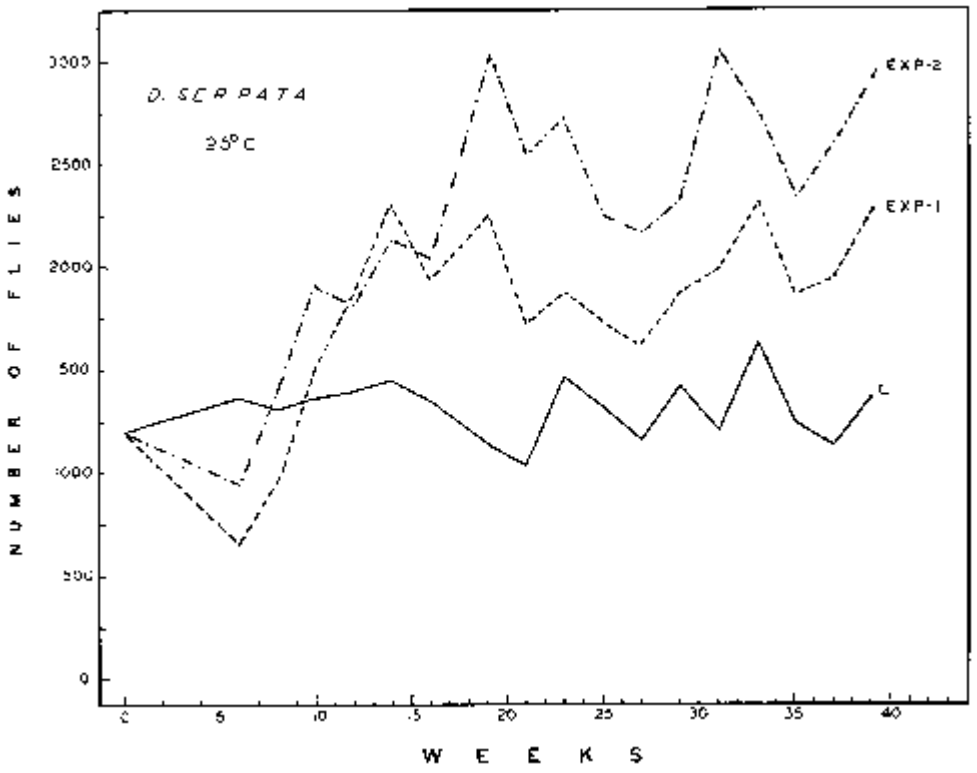

Fig. 3. Effects of radiation on inbred *Drosophila serrata* populations. An inbred population was split into three parts, two of which were irradiated. The resulting changes in population size are readily interpreted as indicating the induction of favorable mutations in the irradiated populations. Other experiments gave similar results. From Ayala (1966), reprinted by permission.

Although this does not appear to be so, the house sparrow case should be as famous as the text-book case of rapid evolution, that of the large-scale replacement of the normal white-peppered phenotype of the moth *Biston betularia* by a dominant allele for essentially black moths. The *Biston* case provides a further example of humaninduced changes, although this time through the modification of the environment: the killing by air pollution of encrusting lichens yielded black rather than light-colored tree-trunks, resulting in a shift in bird predation pressure in favor of the previously-unknown allele.

Many other cases are known of both strong selection and the response to it. Of course, the peppered moth and house sparrow cases are almost exclusively scientific in their appeal, but natural selection can be of practical importance as in the many cases of the evolution of resistance by insects to insecticides (Figure 2) or by bacteria to drugs. Resistance to biocides usually arises with dismaying speed, as perusal of the figure will show. The speed is both understandable (we are applying extremely strong selection pressures) and serious: we are in an evolutionary race between our ability to produce new compounds and that of the pest species to produce counteracting mutations in its degradative enzymes I'll allow a somber thought to

intrude here: as we become an evermore important part of the planet's total biomass, we thereby increase the selection payoff for pests to attack us, as well as our crops.

PRIMING THE PUMP: FAVORABLE MUTATIONS

Now, as I mentioned above, evolution can only continue for so long by the replacement of one existing allele by another. If the response to selection involves only the shuffling of preexisting genetic variation, without new alleles arising by mutation and being selected for, then evolution will not continue long. But this is not the case: the occurrence of favorable mutations has been repeatedly demonstrated. This demonstration is a classroom exercise for prokaryotes, because one can start with a single cell, known to lack a favorable trait (such as resistance to a given drug), and then demonstrate the occurrence of the trait in the progeny. Because prokaryotes have only one copy of each gene, the experimenter can be certain that the genes for resistance were not hidden in the initial bacterial cell. Long-running experiments of this kind can further produce significant improvements in the metabolic prowess of bacteria (Mortlake, 1983); these changes mimic the impressive powers of natural populations to evolve the abilities to use new carbon sources, of which a prime example is a wholly new enzyme attacking nylon (Olino, 1984)!

The detection of favorable mutations in eukaryotes is a little harder, because the habitual diploidy of many eukaryotes means that it is much harder to be certain that genes selected for were not present originally as concealed recessives, rather than newly-arisen by mutation. Yet, here too, the demonstration of favorable mutations has become both commonplace and economically valuable, through the induction of agriculturally-destrable mutations with radiation. Such induction is most easily done nowadays using large cultures of single cells from which whole plants can be grown, but has a long history. For animal populations, the study by Ayala (1966) is worth noting. Ayala inbred laboratory populations of the flies *Drosophila errata* and *D. birchii*, thus reducing both the level of variation and the average fitness of the population. Radiation led to the irradiated populations achieving larger sizes than non-irradiated controls (Figure 3), thus demonstrating that there are favorable mutations in amongst the harmful ones.

SINGLE LOCI DO MATTER

But is it worthwhile looking at the effects of single loci? It is a truism in genetics that the overall makeup of an organism results from a complex interaction between its genes and between them and the environment in which it develops. In fact, what evolves is not a collection of hair, skin, eyes (or cilia and flagella, or leaves and roots), but rather a developmental system that produces such characteristics under appropriate conditions. Given such complexity, will changes of one allele for another at one or just a few loci make any difference?

Yes. We have evidence from two sources that single gene changes are important in evolution.

Firstly, there are not as many functional genes as was once thought. The view of only two decades ago that organisms are determined by millions of genes appears quaint now, but it is easily understood. After all, there are about 2.7×10^9 (yes, only a little under three *billion*) nucleotide pairs in mammals such as the house mouse (Sang (1984) gives a table of genome sizes in various animals). An average protein is about 400 amino acids long, so that it could be encoded by 1,200 nucleotides, meaning that there is enough DNA in mammals for 2,250,000 genes.

But we now know that most eukaryote genes are made up to a large extent of *introns*, sequences within them that are cut out of the primary RNA transcripts before they leave the nucleus and

which therefore do not code for amino acids. For example, the protein ovalbumin from chickens *could* be coded for by just 1,879 nucleotides (the length of the final messenger RNA), but the gene itself is stuffed with introns and is about 7,600 nucleotides long (Sang, 1984)! Furthermore, much of the genome is made up of spacer sequences between genes and various other noncoding sequences, so that the dramatic differences in DNA content sometimes found between relatively closely related organisms (e.g., the pea *Pisurn sativum* has more than nine times as much DNA as the mung bean *Vigna radiata*: Sang, 1984) reflect differences primarily in the non-coding and not in the coding sequences.

How many genes are there then coding for the complexity of organisms? And how can we find out? For the tightlyorganised genomes of prokaryotes, lacking the introns and general profligacy of non-coding DNA of higher organisms, dividing the amount of DNA by the average size of a gene is enough (Watson, 1977). To estimate the relatively rare coding sections in the comparative vastness of a eukaryote genome requires indirect methods, such as estimating the number of loci at which lethal mutations can occur (Raff and Kaufrnan, 1983) or by estimating the number of different messenger RNA molecules produced (Raff and Kaufman, 1983), and using this figure as an estimate of the number of active genes. By such means we can arrive at the following highly approximate numbers of genes in three organisms whose disparities are only matched by the affection that geneticists feet for them:

ORGANISM	NUMBER OF GENES
Escherichia coli (bacterium)	3,000
Drosphila melanogaster (fly)	8,000
Homo sapiens	30,000

It would be a mistake to think that these figures mean that humans are only ten times as complex as bacterial! The complexity of a developmental system depends on the *interactions* between genes, and is therefore dependent on some power of the number of loci, and is not a simple arithmetic function of the number. As an analogy, consider the complexity of a telephone exchange: the number of possible dyadic connections increases with the number of telephones, *n*, according to:

$$\Sigma^{n} (i - 1)$$

where $i = 1$ through n

so that, for three telephones there are three connections, but for six there are fifteen. The complexity of the system grows much more rapidly than the number of subscribers.

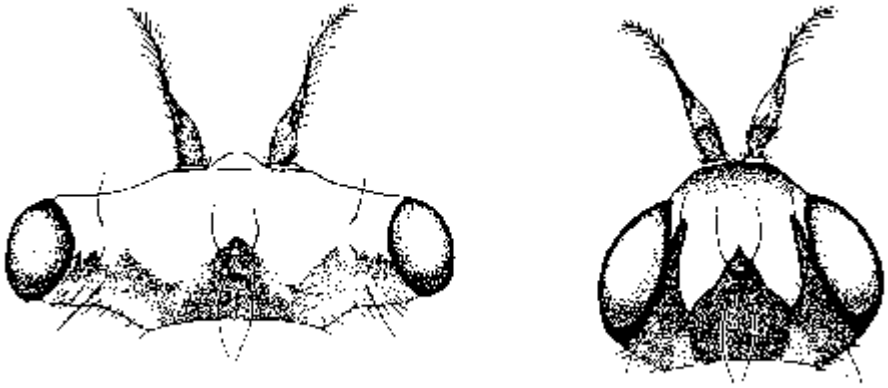

Fig. 4. Heads of males of (left) *Drosophila heteroneura* and (right) *D. silvestris*. Fom Kaneshiro and Val (1970), reprinted with permission

More direct evidence that single genes do matter comes from studies on interspecific differences. One rather spectacular example concerns the closely related flies *Drosophila heteroneura* and *D. silvestris*, whose males have dramatically different head shapes (Figure 4). These two species are extremely similar genetically, and can be induced to mate under laboratory conditions, when they yield viable and fertile hybrids (Vat, 1977; Templeton, 1977). Analysis of the head shape in the hybrids and backcrosses shows that the differences between the two species in male head shape are determined by only about six segregating units (Lande, 1981). Because these segregating units may contain two or more tightly linked genes, the number of gene loci is probably around about 12.

The case of these Hawaiian Drosophila, and others such as that of the peppered moth, show that major evolutionary changes may involve substitutions of one allele for another at as few as half a dozen loci, with further evolution involving "fine tuning" at other loci to ameliorate side effects of the major changes. There is, for example, some evidence that the heterozygotes for the allele for black coloration in *Biston betularia* have come more nearly to resemble the homozygotes for this gene today than was the case last century (Wallace, 1981).

WHEELS WITHIN WHEELS: SELECTION AT MANY LEVELS

Selection is the only evolutionary force resulting in adaptation, because only selection discriminates between the mutations that constantly rush like a gale through the genome. But selection can take place at many levels, and adaptation at one level can take place at the expense of adaptation at another.

While the levels at which selection can occur are many (Table 1), we can certainly fix our ideas by noting that the levels of the individual, above the individual, and within the individual satisfactorily include them all!

TABLE 1
LEVEL OF SELECTION

Levels of selection during genetic evolution, determined on the basis of which entities are interactors (see text). With the possible exception of ecosystems, entities higher than the species are not interactors. Genera, for example, may evolve through selection within or between their constituent species, but are not themselves interactors. Other levels of selection may occur without affecting genetic evolution: for example, competition between cell types within a metazoan or metaphyte is unlikely to lead to an increase in the frequency of the winners' genes next generation.

1. Direct selection on DNA sequences
    a. Selection for the number of copies
        i. Sequences capable of infective transmission
        ii. Sequences incapable of infective transmission (highly repreated sequences e.g. *Alu*)
    b. Selection between aleles (meiotic drive)
2. Selection on genotypes
    a. Selection on haploid phases (bacteria, gametes of higher eukaryotes)
    b. Selection on diploid phases
        i. Cells within organisms (likely to be only rarely converted to evolutionary change)
        ii. "Normal" complete higher organisms
3. Supra-individual selection within species
    a. Intra-demic group selection
        i. Groups of non-relatives
        ii. Groups of relatives (=kin-selection)
    b. Inter-demic group selection (this is the classical meaning of grouip selection)
4. Selection at the species level
    a. Non-random speciation (bias in characteristics of new species)
    b. Non-random distribution of speciation rates (species with more optimal values of some characteristic speciate at a higher rate than others)

c. Non-random distribution of extinction probabilities (species with more optimal values are less likely to become extinct)

5. Selection at the ecosystem level (Wilson, 1976). Assemblages of species differ in their persistence, thus favoring "good mixers" among their constituent species

It is worthwhile pursuing the levels-of-selection approach a little further. Selection may take place at different levels in two ways: by acting at different levels of complexity in the physical hierarchy of life (as displayed in Table 1), or by acting at both the genetic and behavioral levels (of which more later). With regard to the hierarchical approach, we can turn to Hull (1980), who built on ideas enunciated by Dawkins (1976), and distinguish between replicators and interactors. To paraphrase Hull:

> A *replicator* is an entity that passes on its structure directly in replication.
>
> An *interactor* is an entity that interacts directly with its environment.

An interactor is made up of one or more replicators, usually very many. Selection acts by affecting the relative success of different interactors in passing on the replicators of which they are composed. The properties of interactors are of course formed by more than their replicator makeup, with environment and history playing their parts too. Selection is thus effective in causing evolution only to the extent that fitness differences between interactors mirror differences in their replicator makeup.

Levels of selection are thus determined by the level of interactor. But how can this level be recognised? It is not enough to look for average fitness differences between interactors because, for example, two populations may differ in average fitness only because one is made up of fitter individuals than the other.

I suggest that the best criterion for determining the level at which selection is acting in any one case is to discover which is the lowest level allowing a complete formal description of the situation. Thus, if population 1 is made up only of AA individuals, which are of higher fitness than the BB individuals of which population 2 is exclusively composed, then fitness differences between genotypes (level 2(b) in Table 1) is the appropriate level, and higher levels such as 3(a) or 3(b) are not usefully invoked.

Interactors above the gene level are usually not replicators as well, because their reproduction involves their dissolution into their constituent parts: when you reproduce, you do not pass on your genetic endowment intact but rattier shuffled subsets of your genes packaged as gametes.

Higher taxonomic categories such as genera and families cannot therefore be units of selection, because they are not interactors. It is also problematic whether they "evolve" as some authors consider they do (e.g., Arnold and Fristrup, 1982), because they lack objective reality, being simply assemblages of species set up to facilitate cataloging. But 11 would be justifiable to consider a lineage evolving, because that can be objectively defined, in principle, and genera and families that are defined strictly on lineage lines could be said to evolve. Such lineage-defined taxa would then be roughly analogous to populations, which evolve even where selection is strictly at the level of the individuals within them.

There are exceptions to the rule that only genes are replicators. Individuals reproducing asexually and without meiosis do replicate their genetic makeup exactly, and lichens, which are simple

communities made up of particular combinations of fungal and algal species, reproduce by dispersing fragments that form new colonies elsewhere.

SELECTION AND SOCIAL BEHAVIOR: THE EMERGENCE OF NON-GENETIC EVOLUTION

The realisation that selection can take place at many levels helps a great deal in understanding the evolution of cooperation. While much cooperation involves reciprocity, what has been called "social compensation" ("you scratch my back and I'll scratch yours"), there remains the large and important category of *altruistic* behavior. Altruistic behavior is defined biologically as behavior in which one individual reduces its reproductive capacity in favor of that of another (this differs from the everyday definition of altruism, in which *intention* and not effect is all that matters (Stent, 1978), but then we cannot really be sure of the intentions of other humans, let alone of non-humans). Social insects provide an extreme example of altruism: in many species, the workers are sterile and labor solely to aid their mother in producing some reproductive individuals to perpetuate the colony (E.O. Wilson, 1971).

The problem of altruism, as was seen by Darwin, is that it is hard to understand at first glance how reproductive restraint and, especially, sterility can be selected for! Darwin also glimpsed the process which was later explored quite fully by Bill Hamilton (e.g. 1963, 1964, 1972) and named *kin-selection* by John Maynard Smith (1964). Quite simply, kin-selection is group selection in which the groups are made up of relatives (Wade, 1980a). Reproductive self-sacrifice can be selected for if the result is to increase the overall frequency of the genes responsible for the behavior through their increased replication via relatives of the altruist. This approach provides a framework not only for understanding the evolution of groups such as the social insects (e.g. Crozier, 1982), but also for framing laboratory tests (Wade, 1980b).

No treatment of the evolution of social behavior would be complete without at least a brief mention of Maynard Smith's Evolutionary Stable Strategy approach (Maynard Smith, 1982). Briefly, an ESS is a phenotype that, if present alone in a population, successfully excludes any other. The inclusion of the word "strategy" belies the generality of this approach, which of course is applicable to more than just behavioral differences. Furthermore, the ESS approach is a largely successful attempt to model evolution quantitatively without needing the precision in specifying parameters that bedevils efforts to apply population genetics to long-term evolutionary change.

As an example of the ESS approach we can take the explanation afforded by it to one of the puzzles of animal contests: owners of resources often fight fiercely for the resource whereas challengers are timid. This asymmetry can be shown in many cases to be uncorrelated with the fighting ability of the contestants. Maynard Smith pointed out that a strategy following the rule "If owner fight hard, if challenger retreat quickly" would replace either of the unconditional approaches of fighting hard every time ("hawk") or of always giving up easily ("dove"). This replacement is expected because the followers of this *bourgeois* strategy would never fight each other hard, whereas the "hawks" would fight each other hard each time they met, and the "doves" would easily lose whatever resources they had acquired.

Behavior leads us to consider the emergence of non-genetic evolution. Culture, defined biologically, is the transmission of learned behavior patterns. Evolution now becomes rather complex, because in organisms which are social, and these include most higher animals (Wilson, 1975; Wittenberger 1981; Trivers, 1985), there is the passing on of *genes* (of course), and, potentially, of *environment* (a result of parental activities) and *learned behaviors* as well (Cavalli-Sforza

and Feldman, 1981). Only genes show obligatory and exclusive vertical transmission; behavior can be passed horizontally (such as between non-relatives) and its transmission thus resembles the typical infective transmission of microorganisms (Cavalli-Sforza and Feldman, 1981; Lumsden and Wilson, 1981; Boyd and Richerson, 1985).

We expect our own species to be the champion at culture, but cultural transmission certainly occurs in other species too. Bonner (1980) considers examples, such as the occurrences of birds learning from others to pierce the caps of milk bottles, of chimpanzees passing on termite-hunting skills, and of the transfer between individual Japanese macaque monkeys of skills in processing new foods (such as paper-wrapped toffees supplied by scientists!).

Clearly, genetic and cultural evolution are both "biological", and we would expect them to interact, and the theory of this interaction is now a healthy field of study. Some authors, such as Lumsden and Wilson (1981) have stressed cases, such as tendencies to avoid incest, in which cultural biases reinforce genetic ones, but the two need not agree. Dawkins (1976) pointed out that cultural patterns may reduce the genetic fitness of carriers, and Boyd and Richerson (1985) provide a strong beginning to understanding this phenomenon quantitatively (celibacy is a possible example -assuming that by adopting their calling priests and nuns are not somehow preferentially helping their non-celibate relatives!)

Wyles et al. (1983) point out that cultural evolution should tend to increase the rate of genetic evolution, because the acquisition of new behaviors will change the environment and hence the selective milieu of the creatures involved. They use the example of the distribution of the ability to digest lactose as adults: human groups which keep dairy cattle have this ability, and those which don't keep cattle lack it, suggesting that drinking milk as an adult (a cultural trait) selects for adult lactase secretion. In agreement with this suggestion is the quite close concordance observed between relative brain size and morphological evolutionary rate seen in Table 2, on the assumption that larger brains confer a greater ability to support culture. Of course, as stressed by Lumsden and Wilson (1981), it is possible that cultural evolution in our own species could become so rapid as to lead to a rate of environmental change that genetic change cannot keep up with!

TABLE 2
BRAINSIZE AND EVOLUTIONARY RATE

Relative brain size (as a proportion of body weight) compared with anatomical evolutionary rate, as calculated by Wyles et al. (1983)

Taxonomic group	Relative Brain Size	Evolutionary rate
Homo (humans)	114	>10
All hominoids	26	2.5
Songbirds	23	1.6
Other mammals	12	0.7
Other birds	4.3	0.7
Lizards	1.2	0.25
Frogs	0.9	0.23

Salamanders	0.8	0.26

Findings such as those of the occurrence of cultural transmission in species other than our own give a new twist to the Darwinian revolution. Darwin showed that humans have a heritage derived from that of other animals, and indeed rooted in the whole living world. This insight was greeted with antagonism by many fearing that it demonstrated that humans have an "animal nature". The recent studies on social behavior in other species complete Darwin's revolution by turning this popular understanding on its head. Rather than evolutionary biology saying that humans share unpleasant characteristics with other creatures, we can now see that other creatures are really quite like us in many of the things that we pride ourselves on!

ECOSYSTEM SELECTION: THE PINNACLE OF GROUP SELECTION?

Although cultural evolution is seen by some social scientists as necessarily involving the highest level of selection (e.g., Plotkin and Odling-Smee, 1981), this standpoint is arguable because more than one species displays cultural evolution and hence any level inclusive of more than one species would therefore seem (to me, anyway) to be "higher". This highest level is that of the community, as argued by D.S. Wilson (1976).

D.S. Wilson's (1976) argument is couched in terms of interaction coefficient matrices, but he also provides a simple thought-experiment that makes the point. Consider two plant species that are identical in how they profit from the rest of the members of their community, but differ in how they affect them. In particular, consider the effect of species A being harmful to earthworms whereas species B is beneficial to them. Species B could therefore be described as a "good mixer" and species A a poor one. Within communities, there are no systematic effects altering the relative abundances of the two species, but one emerges when the persistence times of communities with differing proportions of species A and B are considered: the more species A individuals there are, the worse off are the earthworms, and the shorter is the persistence time of the community. Overall, given enough time, species B should replace species A because communities with high proportions of species B should persist longer than communities with high proportions of species A (and hence be more important sources of species for new communities).

By the criterion I introduced above, of searching for the lowest possible level at which models adequately describe the biology of the situation, Wilson's model is truly one of selection at the ecosystem level, and not one of selection at the level of the species. If there were only one huge ecosystem, then the relative abundances of species A and B would depend only on stochastic effects. The occurrence of many communities with finite persistence times is needed before species B will necessarily replace species A; hence, the community is the level of selection. [Now we need ecologists to provide material, rather than thought, experiments on this process!]

Given the division of the living world into communities, Wilson's (1976) model indicates that species will evolve not only competitive properties but also cooperative ones. There is a place for "good mixers" in the wider world of life, after all.

FROM ECOSYSTEM TO CELL: THE CHAOS WITHIN

I find it somehow pleasingly paradoxical that consideration of the highest level of selection leads naturally to consideration of the lowest: that within the genome.

The idea of selection, and for that matter genetic drift as well (Van Valen, 1983), occurring within the genome is at first odd, schooled as we all have been in the impartial precision of

Mendelian meiotic mechanics. But there are sections of the genome that do not obey Mendel's rules. These sections form a large part of the DNA, in some species a larger part than that that of the law-abiding genes we were taught 21bout that obey the principles worked out by the seer from Brno.

George Mikios, in his companion article, will say more about our current picture of the chaos within the cell that is the genome, so that only the briefest outline is needed here to make my points. The non-Mendelian sequences in the genome fall into two types:

1. Transposable genetic elements ("jumping genes"), are of two types:
    a. Insertion sequences (ISs, which possess only the necessary flanking repeats to allow insertion, and the gene for the enzyme, transposase, necessary for copying themselves).
    b. Transposons. (Tns, which are composed of insertion sequences plus additional genes, such as for antibiotic resistance, that may affect the biology of the organism as a whole).
2. Highly-repeated DNA. Various "families" of relatively short sequences of apparently non-coding DNA whi thousand.

Initially, the traditional view of selection as focussed at the level of the individual channelled thinking so that biologists racked their brains for some function for all that non-coding DNA. The first break came from Dawkins (1976), who wrote that "The simplest way to explain the surplus DNA is to suppose that it is a parasite, or at best a harmless but useless passenger..." carried along by the rest of the genome. Although attempts to explain the large amounts of non-coding DNA in terms of such functions as regulating coding DNA (Davidson and Britten, 1979) still appeared after Dawkins's book, it was not long before biologists concerned with genome structure and function seriously considered the view expressed by Dawkins, with particularly influential papers being those of Doolittle and Sapienza (1980), Sapienza and Doolittle (1980), and Orgel and Crick (1980). From these papers, and from those that followed them, emerged the term "selfish DNA", and an acceleration in efforts by population biologists to understand the dynamics of selection at the level of the genome.

The dynamics of intra-genomic selection is a field of study of intense interest, both for experiment and theory. Furthermore, as stressed by Mikios (1982), and by Doolittle (1982), the difference between effect and function is especially important when considering both transposons and highly-repetitious DNA. Transposons, in particular, have numerous effects on the biology of the organisms in which they occur, but it is uncertairhas to whether or not these effects are the crucial agrents in determining the levels of representation of these elements.

A detailed examination of the large "selfish DNK literature is not feasible here, but a few remarks are worthwhile in the general context of levels of selection.

The first important fact is that even just an excess of DNA will be deleterious at the level of the individual through effects on such factors as cell cycle times and organ growth rates, although the strength of this factor will differ between organisms and between environments (Grime and Mowforth, 1982). Secondly, the evolutionary dynamics of transposable elements are markedly different from those of highly-repetitious DNA, and should be considered separately. Thirdly, because the dynamics of these two classes of non-Mendelian DNA interact, and highlyrepetitious DNA occurs only in eukaryotes, there are likely to be significant differences in the dynamics of transposable elements between prokaryotes and eukaryotes. Fourthly, the diploidy of most eukaryotes will also give rise to different dynamics relative to those of

prokaryotes and of those eukaryotes which are usually haploid, because of the impact of diploidy in allowing storage of genetic variation.

Transposable elements generally insert only at specific sites (Finnegan et al., 1982), but these are usually very short. Because they are so short, the number of possible insertion sites is effectively infinite (e.g., for those cases in which the insertion site sequence is three bases long, there would be about 45 x 10^6 such sites in the human genome). Transposable elements are also excised from the genome with reasonable frequency; it is likely (but not certain) that this removal is caused by the genome's recombination machinery (and is not under the direct control of the element itself).

Transposable elements cause mutations, because they disrupt the functioning of the sequence around their insertion site. Usually the mutation involves the inactivation of one or more genes, but activation of genes is also possible, as is major rearrangement of the genome (Finnegan et aL, 1982; Syvanen, 1985).

The similarity of the genome to a community of species can now be made more explicit. Traditional Mendelizing genes are relatively law-abiding members of the community of genes (could we call this the "endoblome"?). Transposable elements form a class of genes which have moved outside the tidy legal framework established by Mendel. Whereas insertion sequences do little but replicate themselves and cause mutations, transposons code additionally for functions potentially beneficial to the whole community of genes. Transposons are thus the equivalent of "good mixer" species at the ecosystem level of organization, despite their usually deleterious mutational effects.

Debate continues as to whether insertion sequences are maintained primarily by selection at the level of the individual (the complete community of genes) or at their own level (as "selfish DNK'), a debate exacerbated by the apparent inability of some participants to see that both kinds of selection will occur. Two further findings are relevant. In at least some cases, the rate of transposition of a transposon can be shown to be inversely proportional to the number of copies already present (Doolittle et aL, 1984). In general, a single transposon copy promotes its own transposition while inhibiting that of other copies (Syvanen, 1985). Syvanen (1985), a strong proponent of the importance of selection at the level of the individual or higher, argues that selection at the species level has promoted mechanisms to hold transposition in cheek until a new environment favors a new genetic makeup, which releases the transposons to cause the mutations necessary for adaptation. Doolittle et al. (1984) place the level of selection at that of the community of genes: "self-restraint" by a transposon type will prevent the genome silting up with copies and hence promote its greater longevity (i.e., transposons with selfrestraint are good mixers). But while both of these levels may be important, there is a third hypothesis, namely that this "self-restraint" is, instead, a competitive mechanism between transposons. This last hypothesis accords with the suspicion (Syvanen, 1985) that, in nature, there is considerable sequence divergence between copies of nominally the same transposable element.

There remains the distinction between effect and function. Chao et al. (1983) found that strains of the bacterium *Escherichia coli* "infected" with either of the transposons TnS and Tn10 tend in laboratory culture to outcompete strains lacking these elements. In the Tn10 case, the greater success of this strain occurred because of alteration to a specific site in the gpnome as a whole, and those cases in which the Tnl 0 strain lost in competition were also those in which this particular transposition event did not occur. Do these results necessarily indicate that transposable elements primarily are selected for at the level of the organism because of their production of mutations potentially increasing fitness at this level? No. While the competition experiments demonstrate that inter-bacterium competition is one component of the system, we

still need to know the relative importance in nature of this selective pressure and those imposed by cross-infection and transposition.

In higher eukaryotes such as ourselves and *Drosophila*, diploidy leads to large stores of genetic variation impossible in prokaryotes, and hence to a much lesser reliance on mutation to provide an immediate response to selection in eukaryotes. Furthermore, many, and in some species most, transpositions will not result in mutations because they will occur within non-coding DNA. Furthermore, the much greater occurrence of genetic recombination through sexual processes in higher eukaryotes will probably, in combination with the two factors listed above, lead to a much greater significance of infection as against interstrain competition as a form of selection acting on transposable elements. This is not to deny that transposons can cause mutations in eukaryotes (Spradling and Rubin, 1981; Engels, 1983; Syvanen, 1985), or that such mutations do give rise to an increase in response to selection (Mackay, 1985), only that such effects may be less important than selection within the genome for transposition rate. The theoretical work already done (e.g., Charlesworth and Charlesworth, 1983; Kaplan et al., 1985; Ohta, 1985) needs to be extended, the phylogenetic distributions of transposable elements further elucidated (e.g., Brookfield et al., 1984; Hunt et al., 1984), and, especially, *much* more work done on the occurrence and effects of these elements in natural populations (e.g., Montgomery and Langley, 1983; Mackay, 1985)

Highly-repeated DNA sequences lack the sophistication of transposable elements, being simply the same (or very similar) sequences repeated thousands or hundreds of thousands of times. Insertion sequences may be derived from the breakup of transposons, and transposons from retroviruses (Flavell, 1984), but highly-repeated DNAs must have a different origin. It seems that, in some cases at least, they result from DNA fragments being made from RNA transcripts (a reversal of the normal cell functioning), with these fragments then being more or less randomly inserted into the chromosome again (Ullu and Tschud, 1984; Sharp, 1983; Brown, 1984; Rogers, 1986; — the process of the reintegration of DNA copies made from RNA, while well-documented, remains even more mysterious than most molecular evolutionary events).

While there is sufficient difference between the various copies of highly-repeated DNA "families" elements to indicate the workings of quite old-fashioned mutation (Mikios, 1982, 1985), various people have been sufficiently impressed with the overall similarity of such DNA sequences within species as against between them to coin the term "concerted evolution" to describe this apparent correction of sequences. Dover in particular (e.g., Dover, 1982; Dover et al., 1982) has suggested that this process may be important in speciation (through reducing compatibility of chromosomes in the meiosis of hybrids), and, although many cases of speciation are known where this factor is absent (such as in several Hawaiian drosophilids), it remains an exciting possibility. More important for our theme is the suggestion (Hickey, 1982; Dover, 1984) that such sequences could have significant fitness effects at the level of the individual.

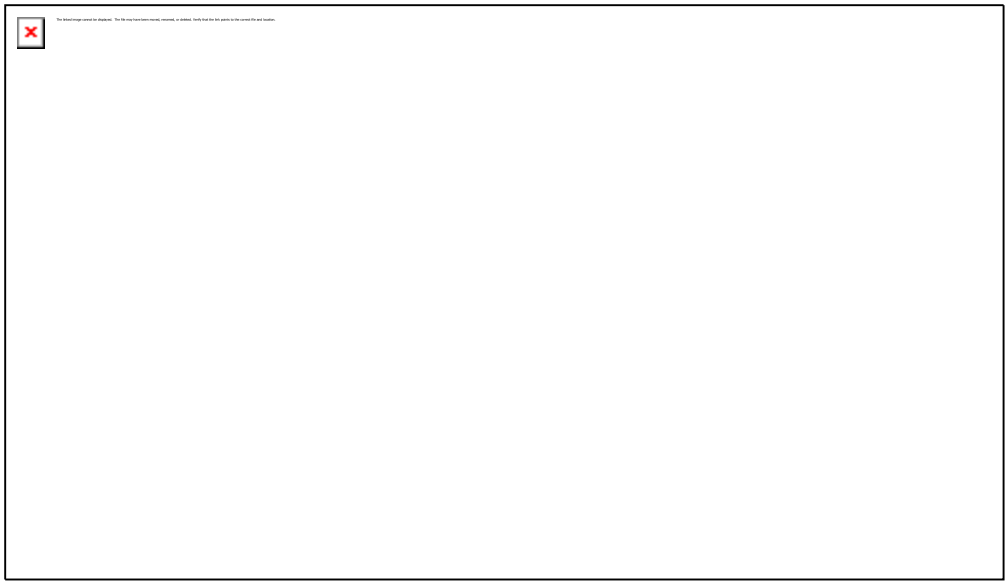

Fig. 5. Unequal crossing-over as a mechanism promoting change in copy number or "correction" of repeated sequences. R and R' are different members of the same highly-repeated DNA sequence family, and repeated rounds of unequal crossing-over, accompanied by genetic drift, lead to the loss of one or the other. Generation of chromosomes with increased numbers of copies may (a) or may not (b) result.

Much interest has centered on the mechanism of "concerted evolution". One factor is unequal crossing-over, resulting from an uneven alignment before recombination of DNA sequences which have several copies of a sequence. Unequal crossing-over leads to changes in the number of copies and through drift to strings of identical copies (Figure 5). While unequal crossing-over will thus have the effect sought, attention has shifted to gene conversion as more likely to be a strong enough force to explain the observed levels of similarity (Dover, 1982; Nagylaki and Petes, 1982). Gene conversion is a consequence of normal meiotic processes in which repair mechanisms occasionally lead to a departure from 1:1 ratios in the gametes produced by a heterozygote. For example, a meiosis in a heterozygote A/A' may yield three A and one A' gamete. Selection in this context would occur by the A allele being copied at a higher rate than the A' allele, through, for example, having a stronger affinity for the DNA synthesizing enzyme.

Gene conversion could be a significant evolutionary force only if one allele is systematically favored over the other. A survey by Lamb (1984) shows that such biases in conversion occur often, which then validates the burgeoning literature on the evolutionary importance of the phenomenon (e.g., Ohta and Dover, 1984; Lamb, 1985; Walsh, 1985). Of particular interest is the case where fitness at the level of the individual is affected by the number of copies of A' it has, rather than of A. Ohta and Dover (1984) conclude that, under appropriate relative strengths of selection and conversion bias, selection could be ineffective in preventing the spread of A, even if it is deleterious to individuals, because all members of the population at any one time are very similar with respect to number of A' copies. By contrast, Walsh (1985) concludes that selection at the individual level would usually prevail, if present.

Clearly, the jury is still out in the matter of the evolutionary dynamics of highly-repeated DNA sequences. There are two further considerations. Firstly, efforts to find effects of highly-repeated DNA at the organism level are relatively scanty, but efforts to find these effects by varying the amounts of such DNA have failed to do so (Mikios, 1982, 1985). Secondly, gene conversion can only systematically change the total copy number of a family to the extent that non-homologous

pairing can occur, and this seems likely to be small. However, because unequal crossing~over lacks any apparent capacity for bias, it would seem a poor candidate for a mechanism to produce very large numbers of copies of particular sequences.

The mechanism for producing many copies of a sequence therefore seems most likely to be reverse copying from RNA. There are two models for how this may occur. In one model, a great many of the repeats observed have the ability not only to be transcribed and then reverse-transcribed but also to be then reintegrated into the genome. There is evidence that many repeated sequences contain recognition sites for the appropriate enzymes, leading Rogers (1985) to stress this model. The other possibility is that there is, for each family, a parent sequence that has a normal organismic function, which is then copied via processes not yet understood into many sites on the genome. Some genes are "amplified" by the production of extra-chromosomal DNA copies at times during development of high demand for their products (Sang, 1984), and these may be particularly susceptible to this process. Brown (1984) stresses this model for Alu sequences because of the discovery that they are stripped-down versions of a functional gene and Rogers (1986) believes that the LINE sequences (1ong interspersed elements" several kilobases long) may be copies of an (unknown) parent sequence. It does not seem to have been generally noticed that most of the original basis for proposing "concerted evolution" is removed under the parentsequence model, because the concert can then be orchestrated by changes in one or at least very few coding sequences (Brown, 1984). Given that DNA is also often excised by mechanisms at present poorly understood, the picture then is one of constant insertion of new sequences from RNA copies of certain sequences whose messages lend themselves to it, and their removal by the enzyme products of what might be called "sheriff" genes (Rothstein and Barash, 1983). As the coding genes evolve, so also changes the spate of reverse-transcribed copies, giving rise to apparent "concerted evolution". In the known cases of this phenomenon, the reverse-transcribed copies are sometimes themselves transcribed but, it appears, never translated. It seems reasonable to suggest that a copy of a coding gene which produced function-coding copies in abundance would thereby unbalance the cell's metabolism, screening out such cases, and perhaps even selecting for alleles of the coding locus which can only give rise to nontranslatable reversed transcripts!

It seems worthwhile to note that phylogenetic analysis can be used to estimate the relative importance of the two factors proposed for producing highly-repeated sequences. Sequencing information can be used to produce phylogenetic trees of the sequences in any one genome. Under the parent-copy model, the resulting tree would consist of a single long stem with unbranched shoots, whereas under the autonomous-replication model the tree would be "shrubby" due to the repeated branching of the shoots. There are general statistical methods exist for testing the shape of trees (e.g., Penny and Hendy, 1985; Crozier et al, 1986), and these could then be used to test the relative difference of observed dendrograms from purely "shrub-like" or "tree-like" forms. A simple test of this nature indicates that Alu elements probably can transpose: Economou-Pachnis and Tsichlis (1985) found that an apparently new Alu copy has higher similarity to other Alu copies than to the functional gene believed to be the ultimate ancestor for this gene family.

LINKS: AND TOWARDS A MOLECULAR POPULATION GENETICS

Clearly, not only do we need links between the various levels at which evolution is studied, but this need is being filled through the emergence of a molecular population genetics. Or, more strictly, a molecular population genetics concerned with multi-level selection, because, as chronicled by Kimura (1983), a molecular population genetics dealing with the relative strengths of drift and selection has been around since the mid-1960s, concerned with evolution by

substitution of one base for another in coding DNA. A prime task of the new molecular population genetics should be finding out the actual molecular changes which occur in response to selection, both in the short and in the long term.

Evolutionary biologists generally fall into one of two types. There are those who uncover what forms of selection are acting on organisms to bring adaptive evolution about. Then there are those who study the genetic machinery in the search for long-term effects of selection. While these two groups have not always taken much notice of each other, the two approaches are not only complementary, but should be combined. The realisation that DNA can be, and almost certainly often is, selfish should heighten the appreciation of the links between the two approaches.

Finally, I ask you to look again at the heads of those two remarkable animals, *Drosophila heteroneura* and *D. silvestris*. What has happened at the molecular level to bring about such a massive change? I hope that molecular population geneticists will see it as a major task to map the genetic changes involved in major alterations in development of this kind and then sequence them to see what has happened. When we know what happened to so diversify these two bizarrely-different yet closely related fly species, we will know a lot more about the process of adaptation itself.

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Return to Top