NATURAL SELECTION REEXAMINED

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The process of natural selection is defined from the standpoint of population genetics. Examples are presented to show how natural selection might act gradually to eliminate harmful mutant genes from the population or to maximize the reproductive potential of gene combinations which are successful in a given environment.

A harmful mutant gene which confers some definite advantage in the heterozygous condition may persist in a state of balanced polymorphism. It is shown that balanced polymorphism is of limited significance, producing variation within the kind but as far as is known, no innovative evolutionary development of new kinds.

Supposedly helpful mutations might conceivably accumulate in a population by action of natural selection, but such a process would be so exceedingly slow that it would not account for the major adaptations of plant and animal species. Although genetic drift might cause a more rapid shift in gene frequencies, it produces non-adaptive changes and is not a valid basis for evolutionary progress. It is concluded that natural selection may lead to variations within the created kinds but it gives no promise as a mechanism of evolutionay descent.‡

Definition and Discussion of Evolution

The term "evolution" must be carefully defined.[1] Confusion has arisen because the word can express at least two different meanings. The word "evolution" is sometimes employed to indicate *variation* or genetic change observable within a living kind (i.e., special evolution).

In a more comprehensive sense, the term is used to designate the *great changes* which are supposed to have occurred throughout vast epochs of time—changes by which all living forms are thought to have descended from common ancestry (i.e., general evolution).

Undoubtedly some variation occurs and a small amount of natural selection has been observed among the variants. But the likelihood of variation and natural selection causing *evolutionary change of one kind to another kind* (general evolution) must be evaluated as a separate question. "Evolution" throughout this paper will refer to *supposed major changes among kinds* (general evolution). Observable minor changes will be called "variation" or simply "change within the kind?*

Some of the concepts necessary to understand the argument of evolutionists are expressed succinctly in biology textbooks. Since some readers may be non-biologists and may not have taken a course in biology recently, most concepts employed will be defined and developed in some detail. Equations will be derived and illustrated so that the non-mathematician may appreciate the quantitative implications of natural selection.

The Evolutionary Method Expounded

Many evolution theorists hold that gene combinations (not single genes or individual mutations) undergo evolutionary change to yield new combinations of genes. These accumulate to such an extent, in their view,. that changes of species or higher categories are produced by a large number of genetic changes. One evolutionist has summarized this view in private correspondence as follows: "Life *proposes* (by mutation), the environment *disposes* (through natural selection), and the population *evolves* (as new gene combinations are continually being tested against a changing environment)." The evolutionist believes that minor variations will ultimately yield what we would think of as new kinds through this process of evolution.

Evolutionists hold that all living forms of today have descended from one or at best only a few common ancestors. Many of them believe that natural selection of gene mutations and gene combinations in a population is the means by which the proposed major changes occurred.

As a result, some evolutionists hold that sexual recombination is more the crux of the evolutionary mechanism than is mutation. By means of sexual reproduction, they visualize that genes are constantly being reshuffled and new combinations are being tested in the changing environment. The sexual process is possible only by means of gamete formation (involving meiosis) and union of gametes (syngamy).

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*"Kind" here has reference to the created types of animals and plants in the sense developed in the book of Genesis. In most translations God is spoken of as creating "Kinds" of organisms (Heb. *MIN).* Marsh has coined the term *BARAMIN* to designate these "kinds" which is synonymous with the present usage here.

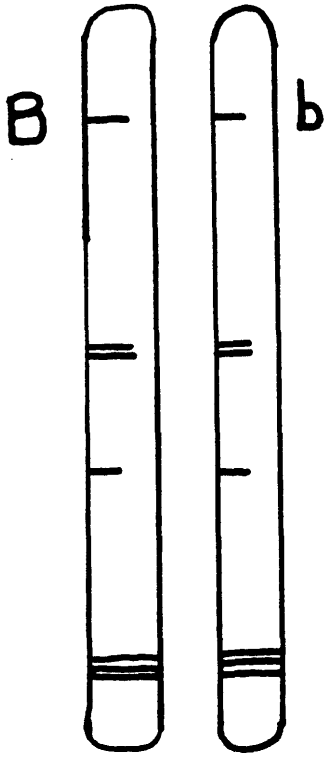

Figure 1. Gene Loci and Alleles. Here is a diagramatic view of two similar chromosomes. Assume that there is a spot *(locus)* near the top of each where a gene for coat color exists. Assume that gene *B* is for black coat color and that gene *b* yields brown color. Genes *B* and *b* are thus *alleles* since they occur at the same locus on the same kind of chromosome. Any genes for color which could occur at this locus in corresponding chromosomes of other guinea pigs would also be alleles in this same series. Note also that each chromosome has many other loci, some of them represented here as lines along the length of each chromosome.

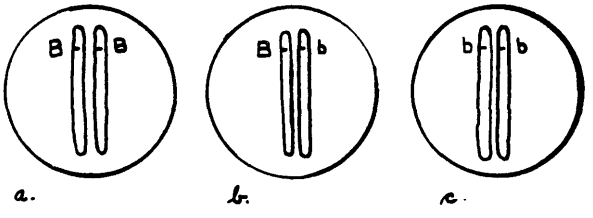

Figure 2. Chromosomes in a Body Cell. A body cell in an animal such as a guinea pig is said to be *diploid* because it contains two chromosomes of each type. Some animals may be "pure-line" or homozygous black (B/B) and they will have gene *B* in each of the two chromosomes of this type as shown in (a) at left. Some guinea pigs might be heterozygous for coat color with gene *B* in one chromosome and gene *b* in the other member of the pair—see (b) center sketch. At right in (c) is a chromosome set from the body cell of an animal homozygous for gene *b*.

William Stroud[2] has shown recently, however, that evolutionists are unable to account satisfactorily for the *origin* of sex. The formation of new kinds by evolution would require the action of preexisting sexual systems, the origin of which cannot be understood by the evolutionary process. This is a definite weakness in the evolutionary view.

But sexual reproduction actually serves only as a reshuffling agent in the breeding population. Sex can do little more than recombine the same genes already present in the population. Despite all statements to the contrary, the real dynamic in the evolutionary model must be the *gene mutation* which is then acted upon by natural selection. Although some evolution theorists assert that evolution results from changes in *combinations* of genes, this action, too, must consist ultimately of selection for or against the *individual mutant* genes which make such favorable combinations possible.

Natural Selection Among Alleles in the Gene Pool

The current neo-Darwinian concept of selection is held to operate by "differential reproduction" rather than by the physical "competition" of Darwin's original theory. Any gene combination which would improve survival among offspring would accordingly become the prominent combination in future generations. In this theory, the favorable genes upon which the fortunate combinations depend, will become more and more prevalent in the populations as time elapses.

In a large breeding population of sexually reproducing animals (or plants) the idea of the "gene pool" represents the totality of all genes carried by all gametes in the breeding members of the population. Thus the gene pool quite obviously gives rise to the next generation. Evolutionary natural selection is therefore said to operate primarily within the gene pool.

An "allele" is one of the genes at a given locus on a specific chromosome, Since chromosomes in cells of most larger plants and animals are present in pairs (a maternal and a paternal chromosome of each kind), a cell has two chromosomes of every kind normally, and thus a cell has two representative alleles of each gene locus.

For this reason, body or somatic cells carry two of each gene type, but reproductive cells (which have only one representative of a chromosome kind) carry only one of each gene. It is possible for every gamete in the population to bear the same allele at a given gene locus, but it is more likely that some sex cells will contain one allele and others will carry a different or contrasting allele (see Figures 1 and 2).

Every cell in a guinea pig, for example, carries two particular chromosomes, each with a gene for coat color. The population of guinea

pigs would produce gametes–sperms and eggs–and every gamete would possess one chromosome carrying the gene for coat color. Some gametes might carry the allele for brown coat color, *b*, at the color locus while other gametes could possess the dominant allele, *B*, for black coat color. The color of the next generation of animals would depend directly upon combinations of gametes from the gene pool that carried gene *B* and those that carried *b* (see Figures 3 and 4).

If any change is to occur in the gene combinations, it must arise as a shift in the frequencies of various alleles in the gene pool. For example, if gene *B* were to become increasingly more prevalent in the gene pools of future generations (and gene *b* were to diminish), this would be a change in gene frequencies in the gene pool—a change that would be reflected in coat color of future populations. There would be appreciably more black animals and fewer brown ones in time.

For the sake of clarity in explanation, the foregoing illustration has dealt with only two alleles of a particular gene locus. Actual allele combinations may be somewhat more complex as in the coat color of rabbits where genes for agouti (c^+), chinchilla (c^{ch}), Himalayan (c^h), and albino (c) are all alleles-situated potentially at the same locus. Although the study of multiple alleles in a population will be more complex, the principles involved are similar to those seen in the present example, and the conclusions are essentially the same.

The Theoretical Hardy-Weinberg Equilibrium

If five basic assumptions are granted regarding a breeding population, Hardy and Weinberg noted that there would be no change expected in the frequencies of genes in the gene pool, nor would there be any variation in the successive generations. There would be no change via natural selection in a population of organisms:

1) if the population is large,
2) if there is no mutation,
3) if there is random mating among all genotypes,
4) if there is no selective advantage of any genotype above the others, and
5) if there is no selective immigration or emigration.

If these five assumptions would hold simultaneously, then the same gene frequencies would prevail in the gene pool and the percentages of various genotypes in the population would remain unchanged.

Such a stable, theoretical population is said to be *panmictic* and to exist in a condition of *Hardy Weinberg equilibrium. The* term "panmictic" indicates that the population experiences no change in gene frequencies as time passes. It

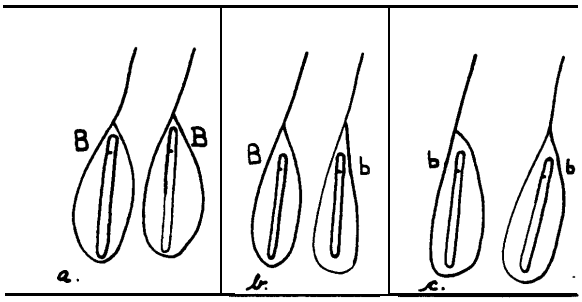

Figure 3. Gametes. When gametes form, the chromosome pairs separate. If cell (a) from Figure 2 produced gametes, the gametes (sperms or eggs) would all bear gene *B (see* left). If cell (b) of Figure 2 produced gametes, about half of them would carry a chromosome with gene *B* and the other half with gene *b (center).* Finally, if the animal with cells homozygous for gene *b,* (cell c) of Figure 2, formed gametes, they would all have the allele *b (right).*

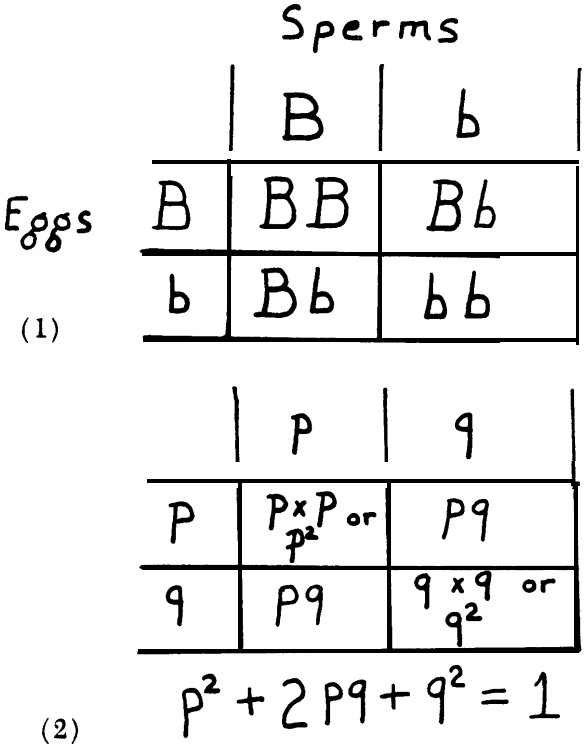

Figure 4. Punnett Square. The letters *B* and *b* are used to represent the two kinds of sperms possible, and also the two possible egg types (see Figure 3). With sperms on the horizontal top axis, and eggs on the side vertical axis in square (1) above, the various kinds of offspring become apparent: BB, *2Bb,* and *bb.* If the gene frequencies for *B* and *b (p* and q respectively) are substituted in place of the given letters, a new checkerboard results as in (2) above. This Punnett square shows the basis for Equation 2 of the text. Each individual box in the square represents one sperm-egg combination and all the boxes taken together represent the whole population of young formed from those matings.

also means that the population has no reproductive barriers, and that no natural selection is taking place.

Hardy and Weinberg pointed out early in this century that the workings of two alleles in such a theoretical population could be characterized by a very simple mathematical expression.[3] Let *p* stand for the frequency (fraction of gametes) in the gene pool that carries one allele (gene *B*), and let *q* represent the frequency of the other allele (gene *b*) in the gene pool. If the number 1 is used to represent the entire gene pool (totality of all gametes in all breeding members of the population), then:

$$p + q = 1 \qquad \text{(Equation 1)}$$

Another way to state equation 1 is:

$$\%p + \%q = 100\%.$$

With these equalities in the gene pool established, the relative abundances of genotypes in the next generation may be calculated if they all have equal survival value. The chance of a sperm containing the *B* gene uniting with an egg carrying that same kind of allele is a simple case of multiple probability in which the probability of a complex event (fertilization or syngamy) is the product of the probabilities of each of the two independent events (independent involvement of a *B* sperm and a *B* egg.)

The chance of a B-bearing sperm uniting with a B-bearing egg is the product of *p* times *p* or p^2. By the same token, the chance of individuals arising that have two genes *(bb)* is q^2.

Heterozygous individuals may arise in the population two ways: 1) the egg could carry the *B* gene and the sperm the *b,* or 2) the egg might carry the *b* while the sperm provides the *B* allele. The result in either case is the same—*Bb.* Hence the Hardy-Weinberg expression for the heterozygous term is *2pq.*

With gene frequencies of *p* and *q* for the *B* and *b* alleles, respectively, the new generation will have the following distribution of genotypes:

p^2 of *BB; 2pq* of *Bb:* and q^2 of *bb*

(see Figure 4).

This is the Hardy-Weinberg equation and it indicates that the potential for the whole new generation can be summarized as:

$$p^2 + 2pq + q^2 = 1 \qquad \text{(Equation 2)}$$

Given a large population of guinea pigs (returning to the example under study) if there is no mutation, if there is random mating without selective advantage, and if there is neither selective immigration nor emigration, then subsequent gene pools would be as former ones were, and genotype frequencies in the population would remain unchanged throughout successive generations.

All of these stringent conditions are never fully met in any one natural population. For example, if there is a slight selective advantage, one would expect this to cause a slight but measurable change in the gene frequencies.

Gene Pool Equations

Despite the fact that selection of a mate is far from random in human populations, consider the following theoretical example which is nontheless based on fact. Two red blood cell antigens have been discovered and are called *M* and *N,* respectively. Researchers have noted that certain people have the *M* antigen only, some carry both the *M* and *N* antigen, some people carry only the *N* antigen.

Geneticists suggest that two allelic genes are operating. A person with the *M* determining gene at these loci is of the *MM* genotype and only the *M* antigen is formed in his red cells. Another person who is heterozygous, having a gene for *M* and one for *N,* will be of the *MN* genotype and produce both the *M* and *N* antigens. Then anyone who has both alleles for *N (NN)* will produce only the *N* antigen.

If one knows the number of people of each genotype *(MM, MN,* and *NN)* in a population, he may calculate the frequencies of the two genes in the gene pool.

Assume that a geneticist found among 100 people the following distribution of blood types: 64 *MM,* 32 *MN,* and 4 *NN.* The *NN* class here is 4/100 or 0.04 which is the q^2 term in Equation 2. If $q^2 = 0.04$, $q = \sqrt{q^2} = \sqrt{0.04}$. Hence $q = 0.2$ and $1 - q = p = 0.8$.

Applying such considerations to an actual count arising in the literature, W. C. Boyd tested 151 Aguaruna Indians of Peru and found that 40.4% were *MM,* 45.7% were *MN,* and 13.9% *NN* as reported by Gardner.[4] These percentages will be rounded to 40, 46, and 14 for ease of computation. From Equation 2, allowing *p* to represent *M* and *q* to stand for *N,* p^2 *(MM)* is 0.40, *2pq (MN)* is 0.46, and q^2 *(NN)* is 0.14.

It is obvious, once again, that selection of a mate is not random in human populations. Nevertheless, few Indians (or people of any race, for that matter) would allow information about *MN* blood type to determine their choice of mate! Hence, reproduction can be considered nearly random as regards this trait. If it is then assumed for purpose of illustration that this population was in Hardy-Weinberg equilibrium, it would be possible to calculate the fraction of gametes in the gene pool that would bear each gene:

$$p = \sqrt{p^2} = \sqrt{0.4} = 0.632$$
$$q = 1\text{-}p = 0.368$$

But a common sense relationship exists which will aid in deriving other equations. If each individual were to produce one and only one gamete, one could tally the gametes and thereby compute *p* and *q.* Using percentage figures from Gardner,

40% of 151 or 60 people were *MM*, 46% or about 69 out of 151 were *MN*, and 14% or about 22 were *NN*.

Thus, 60 of the *MM* people would produce gametes that carried gene *M*. By the law of averages, half of the *MN* people would produce *M* gametes and half would yield gametes carrying gene *N*. Finally, all of the *NN* people would form *N* -bearing gametes. These results may be tabulated as follows:

People of Each Genotype

MM	*MN*	*NN*
60	69	22
Gametes with *M*		Gametes with *N*
60.0		22.0
34.5		34.5
94.5 *M*		56.5 *N*

Gametes with *M*	Gametes with *N*
60.0	22.0
34.5	34.5
94.5 *M*	56.5 *N*

(Gamete Totals)

$$\frac{94.5}{151} = M \cong 0.63 \sim p$$

$$\frac{56.5}{151} = N \cong 0.37 \sim q$$

These obvious relationships can be summarized in terms of new and helpful equations. Let *x* stand for the number of individuals in the population who are of the *MM* genotype. Let *y* designate those of *MN* genotypes and *z* those of *NN* genotype.

$$p = \frac{\frac{1}{2}y + x}{x + y + z} \quad \textbf{(Equation 3)}$$

$$q = \frac{\frac{1}{2}y + z}{x + y + z} \quad \text{(Equation 4)}$$

Substituting:

$$p = \frac{34.5 + 60}{151} = \frac{94.5}{151} \cong 0.63$$

$$q = \frac{34.5 + 22}{151} = \frac{56.5}{151} \cong 0.37$$

Once the gene frequencies have been calculated (by use of either set of equations) one can calculate the fraction of the next generation which would be of each genotype under Hardy-Weinberg equilibrium conditions; *MM* (p^2), *MN* (2*pq*), *and NN* (q^2):

$$p^2 = (0.632)^2 = 0.4$$
$$2pq = 2(0.632)(0.368) = 0.46$$
$$q^2 = (0.368)^2 = 0.14$$

Assuming for simplicity that there would again be 151 people in the new generation, calculation of *x*, *y*, and *z* will show that this new generation would be essentially unchanged from the previous generations:

$$x=p^2 \quad (151) = (0.4) \quad (151) = 60$$
$$y=2pg \quad (151) = (0.46) \quad (151) = 69$$
$$z=q^2 \quad (151) = (0.14) \quad (151) = 22$$

This could be repeated time and again. Given the second generation, by means of Equations 3 and 4 one could calculate the gene frequencies and then produce another generation. Repeated calculation will show that a Hardy-Weinberg equilibrium would permit no change in gene frequencies in one generation, 10 generations, or 1000 generations. As long as the population is large, as long as there are no mutations, and as long as panmixis generally prevails in regards to this trait, there would be no natural selection.

Although these data given from Boyd were actual counts, one must remember that this population may not have been panmictic regarding the *M* and *N* alleles, and remember that it was simply being used as an example for understanding concepts which are to follow.

How Does the Hardy-Weinberg Principle Relate to Natural Selection?

The evolutionist uses this stable base from which to launch an argument for change within gene pools. He asserts that the assumptions listed are important precisely *because* they are impossible to attain in actual populations. For this reason he views the Hardy-Weinberg equation as describing a hypothetical null set or "non-evolving" population to which real populations may be compared.

It is obvious, for example, random mating is generally unattainable in animal and human populations for a variety of reasons. Not all populations are large—some isolated population units may be quite small. It is also likely that mutations will be occurring (although infrequently). It is reasonable to suggest that a mutant gene will either help or hinder to some extent in the process of reproduction.

Assume that a mutation occurs which in combination with other genes is harmful when compared to other alleles of the same gene. The mutant could be either dominant or recessive, but assume it to be recessive as many deleterious changes are. Imagine that it is so harmful that when present in the pure-line (homozygous) condition, it often leads to death of the individual creature. This is simply to consider what geneticists know as a recessive "lethal" mutation.

In his genetics textbook, Gardner describes several recessive lethal mutations:

> Recessive lethals are carried in heterozygous condition and may come to expression

when matings between carriers occur. Genes in *Drosophila* that control such conspicuous phenotypes as curly wings (Cy), plum eyes (Pm), and stubble bristles (Sb), adversely influence the viability of the flies when they are heterozygous. When homozygous, these genes are lethal. They are dominant with respect to the phenotypes for which they were symbolized, but recessive with respect to their lethal action. The reasons for lower viability and lethal action have been determined experimentally in some cases. Apparently, when alone, each of the genes can interfere with vital processes and thus influence viability, but a double dose of the same gene makes it impossible for the organism to live beyond a certain stage in development.[5]

It is clear in Dr. Gardner's discussion that recessive lethal genes are not only lethal, they reduce the viability of the heterozygote. For example, a homozygous plum-eyed fruit fly (Pm/Pm) would die. A heterozygous fly (Pm/+)* would survive, but would be less viable than the wild-type fly (+/+).

One can contemplate a large fruit-fly population which contains a certain percentage of genes for plum-eye in its gene pool. The population would certainly not be in a condition of Hardy-Weinberg equilibrium. The choice of mates would also be affected because the (Pm/Pm) individuals do not survive to reproduce. Since individuals of that genotype die, there is a selection coefficient (S) against the (Pm/Pm) genotype. Here the numerical value of S, the selection coefficient against genotype (Pm/Pm) is 1. The 1 indicates that *all* the flies homozygous for plum-eye will die before reproducing.

Since most mutations are harmful,[6] a value of 1 is high but not astoundingly so. Any recessive lethal mutation which confers no selective advantage on the heterozygotes will establish a selection coefficient of 1 against the genotype it forms. A mutation which is harmful, but not altogether lethal in the homozygous condition, will have an S value less than 1 but greater than zero.

How can the change of gene frequency within the gene pool be calculated in such a case? The (+/+) individuals survive and so the *x* term remains unchanged (see Equations 3 and 4). Likewise (Pm/+) flies live so that the second term persists as *y*. But all the (Pm/Pm) individuals die, and some measure of change must be introduced in forming the value for *p* and *q* from the population. A revised version of Equation 4 will suffice for *q:*

$$q = \frac{\frac{1}{2}y + z(1-S)}{(x+y+z) - Sz} \qquad \text{(Equation 5)}$$

The new equation accounts in the numerator for the fact that the fraction of all (Pm/Pm) individuals will not reproduce. With an S value of 1, the 1 minus S term (Equation 5) becomes zero, indicating that the (Pm/Pm) individuals will not pass on any gametes to the gene pool. Likewise, the denominator must then be adjusted for the fact that the fraction of all the z group (Pm/Pm) will not be included in the total gamete pool so that Sz should be subtracted from the denominator.

By use of this equation, it is possible to show what would happen in one or more generations to the frequency of a recessive lethal gene. If the gene originally had a distribution in a fruit-fly population of 10,000,000 as follows: (+/+) 6,400,000; (Pm/+) 3,200,000; and (Pm/Pm) 400,000, using Equation 4, the frequency of genes Pm in the gamete pool by the Hardy-Weinberg formulation would have been:

$$q = \frac{1{,}600{,}000 + 400{,}000}{10{,}000{,}000} = \frac{2}{10} = 0.2$$

(See Equation 4).

But since there is natural selection against the (Pm/Pm) genotype, Equation 5 must be used and calculations continue as follows:

$$q = \frac{\frac{1}{2}y + z(1-S)}{(x+y+z) - (Sz)} = \frac{1{,}600{,}000 + 0}{10{,}000{,}000 - 400{,}000} = \frac{1{,}600{,}000}{9{,}600{,}000} = 0.167.$$

In one generation the frequency of gene Pm would be expected to have dropped from a value of 0.2 to 0.167. In future generations it would continue to fall gradually. Since nearly all mutations are of a negative character, this indicates that natural selection will serve largely to reduce the gene frequencies of harmful mutations in the gene pool.

Such "weeding out" would occur very slowly as demonstrated by Dobzhansky[7] in a table entitled, "The Progress of Selection against a Recessive Gene." A gene starting from a frequency of 0.55 with an S value of 1 against it (as in the previous illustration) after 10 generations would have fallen to a frequency of 0.085, and after 1000 generations would still persist at a frequency of 0.001.

This slow reduction would not be expected to completely eliminate the gene as it would still be expected to remain within a large population, at low frequency in the gene pool. These considerations apply to eugenic plans. The elimination of all harmful genotypes in the human population would be at least as slow as natural selection.

*Note that "+" in the geneticist's symbols refers to the wild-type allele for any gene.

Sickle-Cell Anemia

Many of the lethal or extremely harmful gene mutations will be reduced gradually to infinitesimally low levels in the gene pool by action of natural selection. Gardner reports that between 10% to 40% of certain human populations of tropical background are heterozygous for the gene which produces sickle-cell anemia.[8]

Sickle-cell genes arise in human beings as a mutation or variational change of a normal allele which causes the formation of normal hemoglobin pigment in the blood. A normal person will thus have two alleles for production of normal hemoglobin (hemoglobin-a) and normal red cells (Hb^a/Hb^a). A person heterozygous for sickle-cell anemia (Hb^s/Hb^a) would manifest the condition slightly but still survive.

The heterozygous gene combination is important in particular points because it has saved lives of African peoples faced with the spread of malarial parasites. People heterozygous for this mutant gene (Hb^s/Hb^a) have a greater resistance to malaria than those homozygous for normal hemoglobin (Hb^a/Hb^a).

The person having the sickle-cell gene at both loci (Hb^s/Hb^s) generally dies without reproducing. There are differing degrees of severity in this condition and sometimes the effect can be alleviated by diet—even in the homozygous condition.

Natural Selection and the Sickle-Cell Mutation

A human population with Hb^a (gene for normal hemoglobin) and Hb^s (gene or sickle-cell anemia and abnormal hemoglobin-s) present is certainly not in a condition of Hardy-Weinberg equilibrium. The choice of a mate is affected because the (Hb^s/Hb^s) individuals do not usually survive to reproductive age. Next, mutation has taken place and has led to the presence of this sickle-cell gene Hb^s.

Furthermore the equilibrium is violated since there certainly is a selection against genotype (Hb^s/Hb^s). Individuals that are (Hb^s/Hb^s) will die, thus there exists a definite selection coefficient (S) against this genotype. In this case S approaches 1 because (Hb^s/Hb^s) individuals generally die, as already indicated.

If there were no selective advantage conferred on the heterozygous individuals (Hb^s/Hb^a) it might be expected that natural selection would lead to the slow but inexorable demise of gene Hb^s in the entire human gene pool—as in the case of the plum eye gene (Pm) in fruit flies.

Sickle-Cell Genes and Balanced Polymorphism

Why do human populations of tropical ancestry (specifically Negroes) have this deleterious gene persisting in their gene pools—a gene that is virtually non-existant in gene pools of other populations? Merrell[9] states:

> This gene has a surprisingly high frequency in some parts of the world. In these areas malaria is endemic, and it has been found that the heterozygotes (Hb^s/Hb^a) for the sickle cell gene are significantly more resistant to subtertian malaria than are the homozygotes (Hb^a/Hb^a) for normal adult hemoglobin. Thus where malaria is prevalent, the heterozygotes are better adapted than the homozygotes, which are apt to die either from anemia on the one hand (Hb^s/Hb^s) or malaria on the other (Hb^a/Hb^a).

Although the gene for sickle-cell anemia in human beings resembles the gene for plum-eye in fruit-fly in that both are highly lethal in the homozygous condition, the sickle-cell gene differs in that it confers a *slight* but definite *advantage in the heterozygous condition*.

"Balanced polymorphism" is that condition in which a creature heterozygous for a lethal or otherwise harmful gene is adaptively superior to either homozygote. In tropical areas where malaria is a severe health problem, individuals who are heterozygous for sickle-cell anemia (Hb^s/Hb^a) are adaptively superior to the people homozygous for normal red cells (Hb^a/Hb^a), when malaria strikes. Under such conditions, both genotypes are preserved by natural selection—persons homozygous for normal red cells and those heterozygous for sickle-cell anemia.

A mutation for sickle-cell anemia may be considered somewhat advantageous in certain limited areas of the earth if conditions select against its normal allele. The sickle-cell gene may actually increase the survival potential of people in the tropics. Presumably the sickle-cell trait would have a greater geographic distribution in human populations if malaria organisms were of worldwide distribution. When such balanced polymorphism exists, a harmful gene will remain at a surprisingly high level in the gene pool. Populations of Afro-Americans living in Chicago, however, will probably tend to lose this gene if modern public health measures are maintained there.

Balanced Polymorphism and General Evolution

It is obvious, on the other hand, that sickle cell genes hardly qualify as examples of completely helpful mutations. It must be remembered that a fatal anemia usually develops when it is in the homozygous condition, i.e., Hb^s/Hb^s. Although the gene does provide some resistance to malaria in the heterozygous condition, *this effect will be beneficial only in the tropics where malaria is a continuing problem*. It is further obvious that the sickle-cell gene is not absolutely essential to human survival even in the tropics, because only about 18% of the people (48% at the greatest) carry this allele.

Even though the sickle-cell gene persists by balanced polymorphism in the gene pool of human populations of tropical history, there is no indication that the gene ever loses its harmful side effects. There is no basis on which to assert that it could change further to become a gene that would direct some new and totally helpful phase of metabolism. Its perpetuation in tropical populations is a local variation with no indication that any evolutionary event (change of "kind") has taken place.

Natural Selection and Creation

Perhaps natural selection was intended to offset the injurious effects of mutations coming after the Fall. Natural selection may have been initiated to minimize the burden of genetic mutation which would accumulate in future generations of human beings, plants, and animals. Certainly the "weeding out" effect of natural selection would fit with a creationist's concept of minor changes *within the* created kinds.

Perhaps the Creator used a certain amount of variation to provide adaptive potential for species in the changed environments after the flood. If a pre-flood vapor canopy collapsed during the deluge, the post-flood mutation rate would likely have increased because of a greater flux of cosmic radiation and scatter radiation.

While these suggestions are only speculative, they indicate that the small amount of natural selection known to occur in nature is not at odds with the Biblical account of creation. It is also possible that natural selection may very rarely act to preserve mutations which occur at the same time the environment changes so as to favor them. It is just as unlikely that a mutation could normally be an improvement in an organism as it is that a random change in the mechanism of a watch or a computer could improve it.

Slightly Harmful Genes

In the discussion to this point, one kind of evidence has been particularly scarce—evidence that natural selection will suffice for the formation of new adaptive types (new kinds). Despite the evidence that most known mutations are harmful, evolution theorists propose to fill this void by asserting that there are probably numerous mutations which have a very slight beneficial effect on the organism. For example, Snyder and David propose that:

> 15. Mutations with slight effects are much more common than those with marked effects. . . .
> 16. Mutations with no visible effects are the most common of all mutations. . . .[10]

To evaluate these statements, one must consider the origin of mutations.

The Origin of Mutations

The origin of mutations is not well understood. Mutation rates can be accelerated by a variety of agents including certain chemicals and ionizing radiation. Even in the absence of these agents, however, living things mutate constantly and at a rate too great to be accounted for by any known mechanism.

This much is known, however: mutations are changes in the genetic information carried in the cell nucleus—changes in the DNA (deoxyribosenucleic acid) of which the chromosomes are composed. Some mutations are chromosomal in their origin (polyploidy, chromosome inversions, etc.). Here we consider those mutations which strictly involve the gene as such.

Chromosomes are duplicated in the *interphase* or so-called "resting" stage of cell division in which the cell engages only in its normal metabolic activities. This is necessary if the cell is to divide, because each daughter cell must possess a copy of all the genetic information the mother cell had.

Replication occurs in the interphase because only then is the DNA "unwound." During mitosis the DNA is bound up into the packages known as chromosomes. In interphase every bit of it is expanded and in contact with its molecular environment. In the average cell the total length of the thread of DNA in interphase is estimated at 38 inches!

DNA can be considered a polymer comprised of many *nucleotides* linked in a long chain. Each nucleotide consists of (1) a phosphate group, (2) a pentose or five-carbon sugar (deoxyribose), and (3) an organic base. These are linked thus:

...phosphate-sugar-phosphate-sugar-phosphate...

The organic bases are arranged at right angles to the phosphate-sugar chain. There are our kinds of bases:

Adenine, Guanine } purines Cytosine, Thymine } pyrimidines

They are linked to *sugar* moities, but any base can be linked to a sugar, and they can occur in any order, for example:

Phosphate > Sugar–Adenine
Phosphate > Sugar–Cytosine
Phosphate > Sugar–Thymine
Phosphate < Sugar–Guanine

Nucleotides can also be linked to each other by means of their bases which are linked to one another through a hydrogen bond, for example: Adenine-H-Thymine. Adenine will pair with thymine; cytosine will pair with guanine. These are the only possible combinations. For instance, adenine will *not* pair with guanine or cytosine.

Generally, two strands of DNA are linked in a *double helix* by means of their bases:

Ph < S–Ad–H–Th–S > Ph
Ph < S–Cyt–H–Gua–S > Ph
Ph < S–Th–H–Ad–S > Ph
Ph < S–Cyt–H–Gua–S > Ph
Ph < S–Ad–H–Th–S > Ph

One can predict the composition of one strand if he knows the composition of the other. This is something like the relationship that exists between a photographic negative and its print.

In interphase the helix separates into its component strands. The hydrogen bonds break and the strands separate (like a zipper). Each strand

then forms a new strand from free molecules (under the control of enzyme systems). In each case a new double strand then comes into existence, and *two double* strands of identical composition are formed.

Like DNA, RNA (Ribosenucleic acid) has a skeleton of phosphate and a sugar (ribose), but the base *uracil* is present in RNA in place of thymine. In general structure, however, RNA and DNA are similar.

Just as a strand of single DNA can build up a complement of itself (under the influence of appropriate enzymes and in the presence of essential starting molecules) a negative image or counterpart of DNA can be synthesized as RNA. The pairing of bases takes place similarly but is not quite the same because of the substitution of uracil. Thus:

Cytosine in DNA leads to an opposing Guanine in RNA
Guanine in DNA leads to an opposing Cytosine in RNA
Thymine in DNA leads to an opposing Adenine in RNA
and Adenine in DNA leads to an opposing Uracil in RNA.

It has recently been shown that the RNA of certain bacterial viruses is also capable of replicating itself in much the same way that DNA does in larger creatures. RNA also contains certain exotic bases in addition to uracil. These additional bases appear to be of only minor importance in larger creatures.

In cells of larger organisms, DNA occurs primarily (but not exclusively) in the nucleus. RNA is found in large quantities both in the nucleus and the cytoplasm. RNA occurs in three major forms:

1. Ribosomal RNA. Ribosomes are complex little organelles made of protein and RNA. Ribosomes are associated with the "rough" endoplasmic reticulum. They seem to be manufactured in the nucleus and stored in the nucleolus. Evidently they are released into the cytoplasm by the dissolution of the nuclear membrane in mitosis.

2. Messenger RNA. This is an unstable substance which is manufactured in contact with DNA and reflects the information content of the DNA, which is "coded" in the sequence of bases which occur on the DNA strand. Messenger RNA will bear the complement of the genetic code specified on the DNA which has acted as its template.

3. Transfer RNA. This is never in contact with DNA. It has three free bases on one end which compose a triplet. These three bases pick up one appropriate and specific amino acid in the cytoplasm. Sixty-four combinations are theoretically possible. This particular amino acid is delivered to strands of messenger RNA which provide the scaffolding for the arrangement of the amino acids into proteins.

When the protein is complete, it is released and the RNA can make another like it. Since all life activities of the cell are controlled by enzymes, and enzymes are proteins, it is easy to see that the assembly of proteins controls the life of the cell and thus the life of the entire organism.

The general sequence of information transfer in the cell is:

DNA → mRNA → Ribosomal Assembly of tRNA → protein strand

If there is any biological fact that should stir one's heart to awe of the Creator, it is this: the libraries of information necessary to produce and operate the fantastically complex body of a human are contained (for the most part) in a nuclear package far too small to be seen by the unaided eye! There is no other example of miniaturization to be remotely compared with this.

These relationships are summarized in Figure 5. As is easily seen, a change or deletion in a single DNA base, or a misreplication of a single base will ultimately be reflected as a change in the amino acid sequence of the finished protein. Not so easily seen, perhaps, is the fact that it will usually affect not just one of the amino acids but the whole strand.

Sickle-cell anemia results from a change of just *one* amino acid out of the hundreds found in normal hemoglobin. A substantial change in any of the enzymes of the cell will make them non-functional. Thus a mutant is generally an

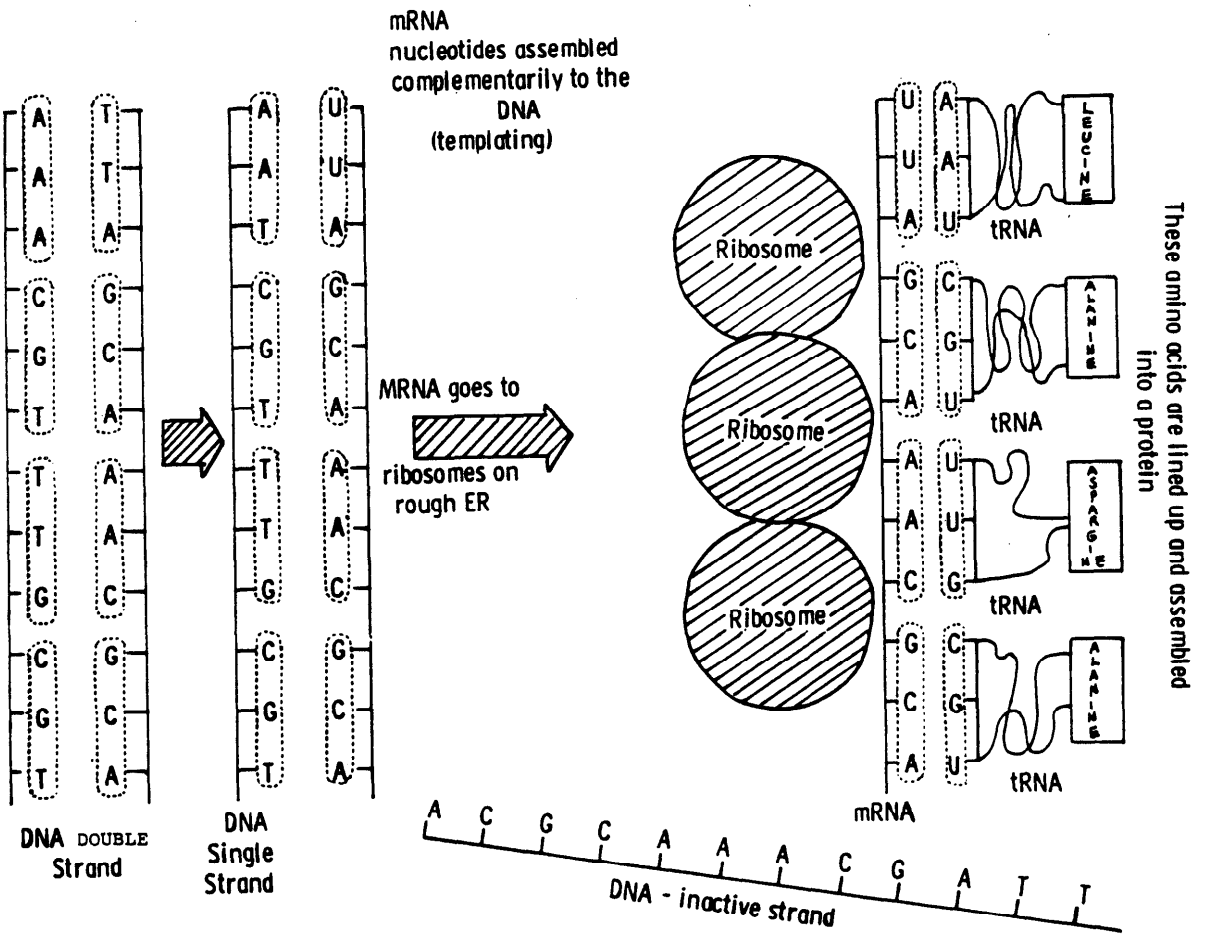

Figure 5. Information Transfer, DNA to Protein. The nucleotide sequence of one strand of DNA is complementary to that of its mate. During protein synthesis, the strands separate and the active strand assembles a unit of mRNA whose sequence is complementary to it. The mRNA then becomes associated in the cytoplasm with ribosomes which it binds together into *polyribosomes.* tRNA fragments are then assembled on the mRNA strand in a sequence complementary to it. Each tRNA bears an amino acid protein fragment. In this way the amino acid sequence of the finished protein is determined. Thus the nucleotide sequence of DNA ultimately determines the amino acid sequence of the finished peptide or protein.

individuals in whom some of the enzymes have been rendered useless or ineffective. The ultimate result is almost always a defective organism. It is no wonder that supposed evolutionary change through mutation and natural selection would proceed slowly. One is entitled to wonder if it proceeds to any important extent *at all.*

Slightly Harmful Mutations in Populations

Within the gene combinations of creatures in a breeding population, what would happen to such a mutation if it were only slightly harmful? If it were recessive, it would experience natural selection as the plum-eye gene did but would register a lower numerical value for the selection coefficient (S) against it.

Assume that there is a mutation in which the selection coefficient against a genotype (a/a) is only 0.01. This indicates that only one out of every 100 *(a/a)* individuals would die before reproducing.

If the population had been: (A/A) 6,400,000: (A/a) 3,200,000; and *(a/a)* 400,000 (expected gene frequencies of $p = 0.8$ and $q \approx 0.2$, respectively, for genes A and a), the population with selection coefficient of 0.01 against the *(a/a)* genotype would have a reduced frequency of gene a according to this calculation:

$$q = \frac{\frac{1}{2}y + z(1-S)}{(x+y+z)-(Sz)} =$$

$$\frac{1{,}600{,}000 + 400{,}000\ (0.99)}{10{,}000{,}000 - 400{,}000\ (0.01)} = \frac{1{,}996{,}000}{9{,}996{,}000} = 0.1997$$

The future of a slightly harmful mutation in such equations is obvious. It too will decrease

as successive generations follow. The only difference between a slightly harmful mutation (S = 0.01 against it) and the more drastically harmful mutation (S = 1) is that the slightly harmful mutation will be weeded out *more slowly* than the seriously harmful gene. But eventually and inevitably it too will vanish. Thus, previous calculations for plum-eye (Pm) indicate a reduction in the gene pool to 0.167 from 0.2 in one generation, while corresponding calculations for the slightly harmful gene have suggested a reduction from 0.2 to only 0.1997 in one generation. The effect is the same in either case—weeding out.

Selection Coefficients and Slightly Helpful Genes

Evolutionists often speak of minor mutations which have a slightly beneficial effect in combination with other genes. Data on these "slightly beneficial" mutations are hard to find, and most of the discussions are theoretical—being based on population equations and a priori demands of the evolution concept.

For the sake of following the arithmetric argument, however, assume that there are some of these slightly helpful mutations, and that they establish a selective coefficient of 0.001 favoring the recessive mutant genotype. What would a favorable S value of 0.001 indicate in the population? It would lead one to suggest that 1000 individuals of the mutant genotype *(a/a)* in the population will survive and pass on offspring for every 999 individuals of other genotypes *(A/a)* or *(A/A)* who survive.

Natural selection ought to cause such a slightly helpful recessive mutant gradually to infiltrate the gene pool after many generations. A new equation or *q:*

$$q = \frac{\frac{1}{2}y + z(1+S)}{(x+u+z)+(Sz)} \quad \text{(Equation 6)},$$

denotes that each new generation would be expected to show a slightly *higher* fraction of gametes in the gene pool that carry the "helpful" mutant *(a).* Continued indefinitely, natural selection ought to yield a population in which nearly all gametes contain gene *a.*

Rates of Natural Selection Estimated

Evolutionists maintain that there would be thousands and tens of thousands of gene combinations moving through the process of natural selection concurrently. By this means they "envision" the gradual changes in populations which they feel would explain ultimately the major transitions of general evolution—"amoeba to man" or "alga to magnolia," as the case may be.

One feature hindering the use of natural selection as the mechanism for general evolution is the rate. It would require extremely long periods of time for each mutant gene to become established in a gene pool by this mechanism.

From calculations by Patau (based on a generous, favorable selection coefficient of 0.01), John Klotz[11] shows that it would take nearly 1,000,000 generations for a recessive gene to pervade 99.99% of the gene pool. Klotz discusses the dynamics of such change and concludes that a recessive gene must have a rather high initial frequency before it could play any important part in natural selection.

Yet a high initial frequency could arise only from a high mutation rate, or genetic drift (a topic which will be covered shortly), and each of these is an unlikely mechanism. Evolutionists assert that natural selection leads to variation in gene combinations and that this variation would ultimately yield new kinds, but calculations indicate the process would be fantastically slow.

Calculations of Dodson[12] indicate a similar dilemma in that it would require 321,444 generations for such a slightly helpful gene to go from the level of 0.000,001 to 0.000,002 in the gene pool. Continued calculations based on Equation 6 above would indicate that natural selection favoring a slightly helpful mutant gene would be amazingly slow. An organism such as a bear that gives birth to its young only once each year would require about 1,000,000 years to establish such a new gene throughout all or nearly all of its gene pool.

Concerning the rate of horse evolution through the supposed geologic ages, G. G. Simpson[13] estimates that it took 50,000,000 years for the small, many-toed *Hyracotherium* (eohippus) form to yield the modern horse *(Equus).* Since he assumes that the direct lineage would involve some eight genera, he calculates the evolution of each intermediate genus as requiring about 6,250,000 years on the average.

Since 1,000,000 years might have been required to establish one partially adaptive mutation, Simpson's estimate of 6,250,000 years for the production of a genus is not particularly convincing. Realizing that various genera in the supposed horse series appear in the strata at different rates, Simpson flatly rationalizes, "It is easy to see that the rate of evolution may vary greatly at different times within a single line of descent."[14]

In another book,[15] Simpson estimates that there would have been enough mutations (1,500,000) in enough individuals (1,500,000,000,000) to have changed the various organs of *Hyracotherium* into those of a modern horse. Instead of providing solid support for natural selection as an evolutionary mechanism, Dr. Simpson's calculations merely show that evolution theory is sufficiently vague to escape absolute refutation. Thus it becomes what philosophers call an "unfalsiflable hypothesis," meaning no compliment thereby.

Rather than asking if a tooth of *Hyracotherium* might have yielded a different tooth configuration in *Equus* 50 million years later, Dr. Simpson ought to ask if natural selection can explain the origin of the *major types.* A typical uniformitarian such as Dodson[16] allows only 19 million years for the entire Eocene epoch of the Tertiary period.

Yet Dewar[17] reports the presence of fossils classified in the following new placental mammal orders: carnivores, odd-toed ungulates, even-toed ungulates, Hyracoidea (hyrax types), edentates (sloths), rodents: Proboscidea (elephant, etc.), Ceteacea (whales), sea-cow, bats, primates, pangolins, ardvaarks, and at least six other orders now extinct.

These manifold mammal orders are all evident in *lower* Eocene strata but they are absent from the Paleocene (the next deeper layer). Using the uniformitarians' own time scale, this would indicate that most major mammal orders, and some orders now extinct, must have arisen within one or two million years (at the longest) between the Paleocene and Eocene.

If it requires 6.25 million years (as Simpson believes) for genera to evolve in horses, and if natural selection takes about 1,000,000 generations to establish just one new allele in a population, how could most major mammal orders have arisen in the comparatively short period between Paleocene and Eocene?

This same acute problem is repeated in the origin of most major groups and their subgroups —fish, reptiles, amphibia, and birds. As a mechanism for general evolution, natural selection appears to be inadequate even in the perspective of a vast time scale.

Effect of Natural Selection Examined

Coupled with this sluggish rate, comes the realization that the end result of natural selection would not be a spectacular evolutionary event or change in "kind" as "evolution" was previously defined. Gigantic spans of time would be required for any *one mutant gene* to become entrenched in the individuals of a breeding population.

If the slow workings of natural selection would involve about 1,000,000 generations to establish one new recessive mutation, how long would it take before the coordinated assemblage of genes necessary to form an eye would combine successfully in some ancient ancestor? It is possible that several genes would be evolving together so that it would not necessarily take longer for a dozen non-allelic changes to occur than for one. Yet even so, the time spans projected are staggering.

If one considers variations in plants, where S is 0.001 or even 0.01, how long would it take, and how many mutations would be required to yield mation of pollen dusting structures in a flower? It is obvious that natural selection of a *dominant* helpful mutation would be slightly more rapid and it is also obvious as stated, that more than one gene could be experiencing selection at a the combination of genes which govern the forgiven time in the same population. Nevertheless, the time requirements for natural selection are known to be extremely large when compared to the "explosions" of new *kinds* apparently seen in the fossil series.

Genetic Drift Considered

Preceding calculations were all based on a large population with a certain amount of mutation and natural selection occurring. Evolution theorists reason that populations frequently become small and that variations may occur rapidly within small populations.

One must agree that greater deviations from expected ratios may arise (by chance alone) in small samples than in large ones. Someone who tosses a coin 10 times would expect (from theoretical probability considerations) to experience five "heads" and five "tails." Yet it is obviously possible in 10 flips of a coin to have eight heads and only two tails.

Such deviation may occur regularly in a small sample but becomes exceedingly less likely as sample size increases. In 1000 tosses of the coin, the chance of having 800 heads and only 200 tails is infinitesimally small. The 1000 flips would approximate the theoretical 50:50 ratio quite closely.

The principle of random variation in small populations is known as "genetic drift" or the "Sewall Wright effect." Genetic drift is a random shift in gene frequencies within a small population. It is believed to occur (as did the deviation in coin tossing) through a random sampling error in a small group.

Through genetic drift, one can conceive of a certain gene becoming quite prominent in the gene pool of an isolated population of squirrels. In an hypothetical example, assume that the recessive gene for albinism *(a)* is present in squirrel populations generally, but only 1 in 10,000 animals is homozygous albino *(a/a).* In addition, suppose that there exists a small population of about 20 squirrels isolated in a remote canyon and that one of the 20 happens to be an albino *(a/a).*

Imagine that a rock slide, flood, hunting overkill, or some other local catastrophe eliminates all but six squirrels. If one of the six surviving animals had happened to be the albino, the frequency of the albino gene *a* in such a gene pool would have increased markedly. Such change would be an example of genetic drift.

Because the population was small, a random "error" caused a change in gene frequencies that would have taken hundreds or thousands of generations to occur by natural selection in a large, panmictic population. The effect of such minor fluctuations within a larger group would have been minimized, or "swamped out," by the total numbers of surviving animals. Yet in a small population it would be important and would lead to a significant shift in gene frequencies. Similarly, any factor affecting gene frequencies will be more important in a small population—not just the chance factors.

If any evolutionary change is to occur (or even any *permanent* modification of gene frequency), an environmental change would be necessary at the same time as this population reduction to confer some selective advantage to the white mutant form. Otherwise natural selection would erase the effect of genetic drift, despite the temporary major shift in gene frequencies after the rock slide.

Perhaps the rock slide which drastically reduced the population would also expose a new greyish white surface of rock over a vast area, thereby making white fur an advantage through camouflage! A coincidence of this sort is quite unlikely and major permanent changes wrought by genetic drift are also unlikely.

Albinism—A Harmful Mutation

Although this has been an hypothetical example, albinism in squirrels does exist and has been described in Jack Kemmerer's fascinating account of the white squirrel populations in Olney, Illinois.[18] According to the best historical records available, it all started when a certain man, Ezra Stroub, discovered an albino male and female squirrel while hunting. These young albino squirrels were raised and bred. As time passed other townspeople pampered and protected the growing population, finally turning them loose.

A testimony to the deleterious character of this mutation is the fact that there are very few colonies of white squirrels in the world. Albino squirrels are more conspicuous to their enemies than the normal colored squirrels and they have ". . . weak eyes due to lack of protective pigment."[19]

If the albino mutation is generally harmful, one may ask why the population at Olney has increased. The answer is very simple—they have been preserved and protected by man. A city ordinance of Olney (1925) protects the nearsighted squirrels by giving them the right-of-way on any street or crossing in town. Since 1943 there has been an Illinois state ordinance making it illegal to molest, trap, or otherwise kill a white squirrel.

Thus a white squirrel population in Illinois is to zoologists what navel oranges or delicious apples are to horticulturalists! If man acts to preserve a variant (artificial selection), rapid change can be wrought *within the kind,* as Walter E. Lammerts has so clearly indicated in previous papers. For a white squirrel population to be established by genetic drift, however, would be quite unlikely.

Genetic Drift—Another Illustration

Another illustration of genetic drift comes from envisioning a group of people stranded on a desert island. A preacher is along and over the course of years almost everyone gets married and has children. However, one of those present has the rare blood type known as "Duffy," and does not realize it. He happens to be one of the few who does not marry, because he is unpleasant. But his disposition can have no bearing on the selective advantage or disadvantage of his blood type! That gene is thereby eliminated from the gene pool at his death, *by chance.* Such an event is much more likely to happen in a small population than in a large one. In a city, for example, the unfortunate man might have been able to find a girl who would put up with him!

Significance of Natural Selection and Genetic Drift

Evolutionists maintain that evolution of the kinds of plants and animals proceeded despite the slothful pace of natural selection. It is further presumed by evolutionists that genetic drift took place at times in small populations to yield more drastic and rapid changes.

It is possible that changes comparable to those described as genetic drift may occur upon occasion in nature. But such variations would, by their very random nature, usually be temporary and non-adaptive. A gross change in color might follow, but with no apparent value or survival advantage for the animal species. Variations might be expected to arise by such means, but with no particular priority of meeting the environmental demands.

Since genetic drift and natural selection are by no means guided, they could not yield the directed change which would obviously be required by a plant or animal in producing a new organ, new organ system, or basic adaptation. Genetic drift provides no guarantee that changes will introduce "favorable" genes at an opportune time.

Overtones in Molecular Biology

Although genetic drift is a random process, some evolutionists suggest that natural selection enables genetic drift to play a creative or adaptive role in evolution—such as directing the species towards greater fertility. King and Jukes

go so far as to maintain that evolutionary changes in DNA and protein are primarily due to neutral mutations and random genetic drift.

> There appears to be considerable latitude at the molecular level for random genetic changes that have no effect upon the fitness of the organism. Selectively neutral mutations, if they occur, become passively fixed as evolutionary changes through the action of random genetic drift.[20]

Bryan Clarke, however, believes that the role of neutral mutations is not clearly established in protein "evolution." He suggests that natural selection is still the key mechanism rather than genetic drift. In de-emphasizing the role of genetic drift, Clarke states:

> King and Jukes argue that random genetic drift has been primarily responsible for the majority of amino acid substitutions, but the weight of evidence does not support them. Protein sequences, like other characters, seem to have evolved under the dominating influences of natural selection.[21]

Miracles

If some (such as King and Jukes) would argue that genetic drift might have occurred at just the correct intervals of time, and in the proper localities to foster adaptive conversions of type or kind, then the timing of such events would be almost an appeal to the miraculous. A more satisfactory mechanism admittedly involving miracles is the Biblical account of God originally creating the plants and animals "after their kinds."

The creationist stance doubtless involves a step of faith, but it requires yet greater faith to hold that natural selection and genetic drift formed the basic kinds of living creatures. Apparently neither genetic drift nor natural selection lead to anything more than variation within the existing taxa.

Conclusions

Evolutionists foreward natural selection in breeding populations as a key mechanism in producing a general evolution of major kinds of life. Under close scrutiny, however, natural selection is seen predominantly as a "weeding out" operation in which harmful mutations are slowly reduced in future populations. Any favorable selection which might occur would be uncoordinated and quite slow.

Associated phenomena of balanced polymorphism and genetic drift likewise supply little or no basis upon which to support the sweeping changes which general evolution would require.

A Biblical format of origins "after their kinds" through rapid creation provides a suitable framework for viewing the magnificent adaptations of plants and animals. Furthermore, whatever change does actually take place in breeding populations fits well with a creationist's understanding of modification occurring "within the kind."

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