economic dilemma is the most obvious instance) are hardly encouraging.

It is true that some of his work has dated. Archaeological discoveries since his death (for instance, the implications of radio-carbon dating and the findings at Olduvai) suggest that some of his arguments derived from what is known of prehistoric man must be qualified.

Some of the farming methods he opposed have had a greater short-term success than he anticipated, though whether long-term effects may offset this remains to be seen. The triumphs of one generation tend to become the curses of the next (a fact that made Massingham suspicious of all quick solutions), and it may be a long time before a balanced assessment of his criticisms and recommendations will be possible.

But, whatever qualifications of detail may have to be made, his overall "philosophy" (if that is not too abstract a term) remains impressive. His faith in the ultimate unity of "Mother Earth and the Fatherhood of God" (*TL*, 15) is a continuing inspiration. His attitude is constant without being inflexible.

Above all, his vision is one of life. The local community is seen as "the cell of the national body corporate" (*R*, 120); membership of the Church is seen in terms of "the idea of the cell within the organic body" (*R*, 125). "Organic," "living," "growth" are keywords. He never despaired. Even when most pessimistic he was always prepared to hail "the germination of a new sacramentalism towards nature which is implicitly religious" (*TL*, 189). Appropriately, his confession of faith at the close of the twelfth chapter of *Remembrance* ends with the words, *"Spero et credo."*[6]

References

[1]*Remembrance: An Autobiography* 1942. London: Batsford, p. 125. Hereafter cited in text as *R*.

[2]*The Tree of Life* 1943. London: Chapman & Hall, p. 11. Hereafter cited in text as *TL*.

[3]*The English Countryman: A Study of the English Tradition* 1942. London: Batsford, p. 11.

[4]Massingham, it should be noted, did not idealize the Middle Ages in sentimental fashion; his treatment is informed, qualified and judicious.

[5]*The Natural Order: Essays in the Return to Husbandry* 1946. London: Dent, p. 7. Hereafter cited in text as *NO*.

[6]For further critical discussion of Massingham and his work, see the chapter devoted to him in my full-length study *The Rural Tradition* (Toronto: University of Toronto Press; Hassocks, Sussex: Harvester Press, 1974), and Nicholas Gould's article, "A Eulogist of Traditional Husbandry," *The Ecologist*, 6, 128-131. May, 1976.

GENETICS AND CREATION STUDIES†

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Modern creationists, with few exceptions, have not given much attention to modern genetics. Yet the study could be most useful. On the other hand, it reveals many difficulties for evolutionists. Indeed, the subject might better be called, not evolutionary genetics, as is sometimes done, but rather population genetics.

In this article much recent work is mentioned. Difficulties are pointed out for both theories commonly proposed from an evolutionary viewpoint: the classical theory and the balance theory. But some points emerge which creationists have come to believe on other grounds, for instance, that many creatures have far more potential for variation than has been suspected until recentley. This can be seen to be a provision by the Creator, to allow creatures to cope with changing conditions which might arise.

The "evidence" for alleged macro-evolution is generally collected from many different disciplines. Still, decisive proof, if any, might be supplied by two scientific areas only; other disciplines might furnish only "circumstantial evidence".

It is clear which these two areas are: *geology* and *paleontology* should supply the *historical* evidence (fossils, essentially) which would prove that a general evolution had actually taken place. And *genetics* should display the *biological* mechanisms which prove that a general evolution is actually possible and likely.

Modern creationists have dealt a lot with historical geology and paleontology, but relatively little with modern "evolutionary" genetics. Their arguments usually amount to stating that natural selection only eliminates harmful mutations, and that mutations are very rare and nearly always deleterious. Such a simplification involves two dangers: first, that of seeming to ridicule a very difficult and rich science practised by some very bright scientists; and second, of missing the important recent discoveries which, properly understood, strongly support the creationist point of view.

Modern "evolutionary" genetics is, of course, based on a strong presupposition which is directly expressed in the name. This presupposition is that general evolution has in fact taken place. The name "evolutionary genetics" implies this; but it promises far too much.

†This article consists basically of material which was presented, under the title, *The Present State of "Evolutionary" Genetics*, at the Third National Creation Science Conference at Minneapolis, MN, 15-18 August, 1976. The abstract, illustrations, and some of the references, have been added for the present paper.

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The evolutionist and the creationist, in fact, agree perfectly on one point: that "evolutionary genetics" has nothing to say whatsoever on the problem of the supposed general evolution. That is, it has nothing to contribute to the problem how genera, families, and all the higher taxa may have originated during the supposed general evolution. It should therefore be simply called "population genetics", or, if preferred, "speciation genetics"; because it may only hope to say something about the origin of new species—which are no problem for the creationist. Yet, even in this respect this science is still very weak. The geneticist professor Richard C. Lewontin wrote[1] in 1974:

> While population genetics has a great deal to say about changes or stability of the frequencies of genes in populations and about the rate of divergence of gene frequencies in populations partly or wholly isolated from each other, *it has contributed little to our understanding of speciation and nothing to our understanding of extinction.* Yet speciation and extinction are as much aspects of evolution as is the phyletic evolution that is the subject of evolutionary genetics, strictly speaking" (Emphasis added).

Lewontin, zoology professor at Harvard, is a brilliant geneticist and one of the present leading evolutionists of the world. The quotation from his book describes the material that I wish to summarize, from the creationist point of view, in this article.

Genetic Variation

If Darwin made any valuable contribution to science it was the emphasis he laid upon variation. Instead of considering variations as annoying aberrations from stable and ideal standard types, his genius distinguished their enormous importance as a possible source of adaptation and speciation. The set of presuppositions on which the modern creation model is based fully allows for these phenomena.

However, Darwin made the fundamental error of extrapolating from variation to macro-evolution. But still, variation is the cornerstone of genetics; it is the triumph of modern genetics that it explains in one synthesis both the constancy and variation of inheritance. Modern genetics fails, however, to show the relevance of these concepts to the concept of general evolution; in fact, genetics even fails quantitatively to explain what happens on the species level.

The variation studies in genetics is twofold: *genetic* variation (consisting of discrete genotype classes of the classical Mendelian type) and *phenotypic* variation (usually quasi-continuous in character and the supposed target of natural selection). Now it has been recognized for a long time (though not by many laymen) that "evolutionary" genetics is not very much helped by a study of *single* mutants with *drastic* effects but by a study of *large* gene combinations with *slight* phenotypic effects.

However, here one meets with an essential weakness of "evolutionary" genetics. The really interesting hereditary traits (such as size, intelligence, fecundity, viability) are so subtly influenced by certain gene combinations that this genetic variation is usually completely overwhelmed by the whole genetic background and particularly by environmental influences. The variation one can measure is therefore actually uninteresting for the "evolutionary" geneticist; and what he is interested in is actually unmeasurable.

Two Views Proposed

Therefore, the central problem of "evolutionary" genetics at present is to assess the amount of *hidden* genetic variation, or, in other words: At what proportion of its gene loci is an average diploid individual heterozygous? Two important polar predictions have been made on this point, called by Dobzhansky the "classical" and the "balanced" theory of population structure.[2] The *classical theory (CT)*, defended, e.g., by H. J. Muller, M. Kimura, J. F. Crow, T. Ohta, J. L. King, and T. H. Jukes, assumed (up till about 10 years ago) that at nearly every locus every individual is homozygous for a "wild-type" gene; in addition, it is heterozygous for rare deleterious alleles, at a few percents of the loci.

The *balance theory (BT)*, defended, e.g., by Th. Dobzhansky, B. Spassky, F. J. Ayala, J. A. Sved, and W. W. Anderson, assumes, on the very contrary, that at nearly every locus every individual is heterozygous, that there are no such things as "wild-type" genes, and that the number of alternative alleles must be large at each locus (to guarantee permanent heterozygosity). See Figure 1. Unfortunately, creationists usually seem to have heard of the former theory only.

The implications of these two theories and their differences are large. (1) If the *CT* were correct, genetic differences between populations would be much more important than under the *BT* (and this would, for one thing, supply a stronger basis for racism, by the way).

(2) The *CT* assumes that the chief action of natural selection is to eliminate deleterious mutations, that the fittest genotypes are the homozygotes for the wild-type alleles at all loci, and that favorable mutations maintained in the population are extremely rare. The *BT*

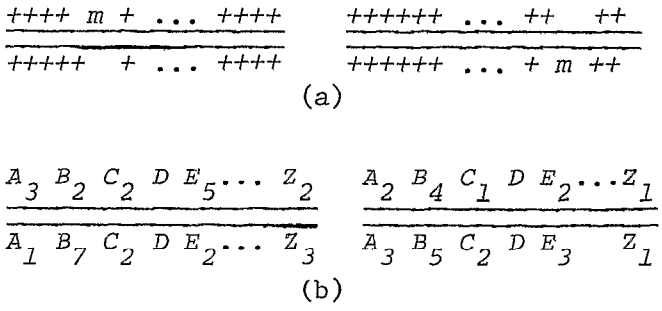

Figure 1. (a) The genotypes of two randomly sampled individuals from a population, according to the classical theory; + signs indicate wild-type alleles and *m* a deleterious mutant. Each individual is heterozygous for an occasional locus, the particular gene being different in each case. (b) The same, according to the balance theory; not only are most loci in the heterozygous condition, but different individuals are homozygous for different alleles at different loci when they are indeed homozygous. An occasional locus with only one wild-type form like *D* is not excluded in the *BT*; and an occasional allele, e.g. B_7, may be very rare and extremely deleterious so that, like the *CT*, the *BT* predicts deleterious consequences of close interbreeding.

assumes that natural selection chiefly occurs in the form of balancing selection, probably selective superiority of heterozygotes, actively maintaining the alternative alleles in the population.

(3) The *CT* assumes that speciation entirely depends on the occurrence of new favorable mutations and their maintenance in the population, which would make speciation (needing many new genes) a much rarer event than seems to be the case. However, the *BT* assumes that the genetic variation for speciation is already there, so that each new biotope should quickly lead to new speciation, indeed much more quickly than seems to be the case.

(4) The *CT* is deeply pessimistic; it states that genetic change can only be for the worse, and therefore supplies propaganda for a genetic elite and eugenic methods. The *BT*, strongly influenced by Herbert Spencer's view that evolution is essentially progressive, is profoundly optimistic; it states that natural selection usually "leads to increased harmony between living systems and the conditions of their existence" (Dobzhansky). However, Lewontin rightly remarks that neither view admits the possibility that genetic variation is irrelevant to the present and future structure of human institutions (values, morals, truth), yea, that the unique feature of man is that he is *not* constrained by his genes. The Marxist (if I am rightly informed) Lewontin agrees herein with the Christian, although their motives are totally different.

Attempts to Decide Which View is Right

Now, which theory is right? In other words, how much genetic variation is there in natural populations? Until about 1965 no definite answer could be given. Screening for "visible" mutations (with clear morphological effects) and lethal alleles (through complicated crossing techniques) has shown that such genes are extremely rare in natural populations, which seems to support the *CT*. However, the *BT* objects that by *heterosis* (relatively high fitness of heterozygotes) still a number of "balanced polymorphisms"[3] for visible and lethal alleles may occur. Other attempts, such as studying the variation of so-called "fitness modifiers" (a third group of genes) and studying the fitness of heterozygotes, have also failed to distinguish between the *CT* and the *BT*.

The best evidence of widespread genetic variation for genes that are relevant to characters of adaptive significance has been obtained from *artificial selection* experiments. If artificial selection succeeds in changing, in a heritable way, the phenotypic distribution in a population, it follows that there must have been non-trivial amounts of genetic variation for that character in the population to begin with.

Now, the remarkable thing in the history of artificial selection is the high frequency of success. It has been responsible for immense changes in domesticated plants and animals (although the enormous contribution of the improved technology of husbandry and agriculture should not be forgotten); while in *Drosophila* population genetics it is a commonplace that "anything can be selected for" in a non-inbred population. The variety of possible selection responses is so extraordinarily large that genetic variation relevant to all aspects of the organism's development and physiology must exist in natural populations.

Although these results do not prove that *large* numbers of genes are segregating relevant to any particular character—even one locus could provide a slow and steady response to selection if heritability is low or if alternative alleles at the locus are near fixation—they do show that, if nearly any character can be selected for rather easily, many genes must be segregating in natural populations. This certainly contradicts the most extreme form of the *CT* which allows only a handful of rare mutations to be heterozygous in each individual.

Still, even if there were definite proof that a very large genetic variation exists in natural populations (which there is *not*) one still would be no closer to an accurate, satisfactory genetic description of populations, i.e., of the frequencies of alternative alleles at various loci in different populations and at different times. But this is exactly what would be needed for an "evolutionary" genetics!

The methodological problems are enormous here. I pointed already to the dilemma that, on the one hand, phenotypic effects of various alleles at one locus must be distinguishable between individuals and from those of another locus so that ordinary Mendelian analysis is possible; whereas, on the other hand, what is really of interest is the variation that is the genetic basis of the *subtle* changes in development and physiology that make up the bulk of micro-evolutionary change.

These two demands, which conflict with each other, both conflict with a third demand for a program to enumerate genotypes in populations: if one wants to calculate what proportion of the genes is segregating in a population the assessable loci should be a random sample of all the genes, which requires that they be sampled irrespective of their variation whereas in fact they are studied because of their variation.

Molecular Genetics Applied to the Problem

The solution to these methodological dilemmas has been found in molecular genetics.[4] The amino acid sequence of proteins is a phenotype that satisfies all the requirements mentioned because:

(1) a single allelic substitution is detectable unambiguously since it results in a discrete phenotypical change: the substitution, deletion, or addition of an amino acid.

(2) The conflict between the *discrete* phenotypic effects demanded by Mendelism and the *subtle* phenotypic differences relevant to micro-evolution is resolved by looking directly at the gene products and not at their physiological and morphogenetic effects.

(3) The apparent paradox of trying to detect invariant genes is resolved because invariant proteins can very well be detected in a population, and molecular genetics usually equates one protein to one gene (sometimes a protein consists of two polypeptides each coded for by a gene).

How can one use these considerations in a program for measuring variation? At the moment, one cannot use the primary amino acid sequence of proteins directly as a phenotype because it is just not possible totally

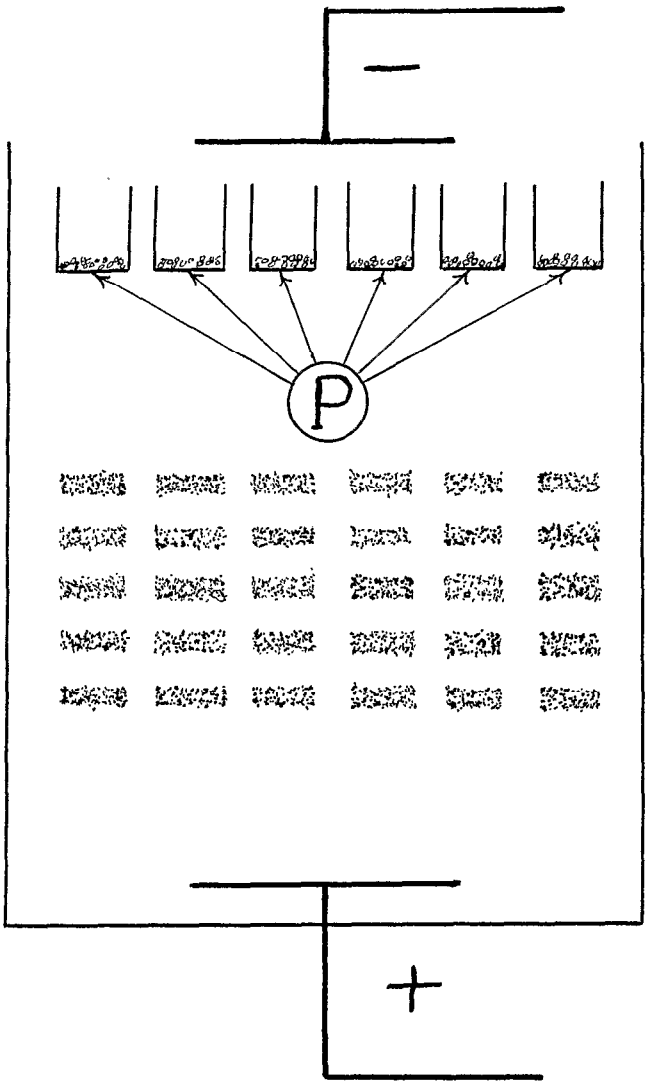

Figure 2. A diagram of a vertical slab gel-electrophoresis apparatus. The positive and negative signs indicate the electrical poles. P indicates the sample pockets, in which the homogenate can be seen in its initial position. Under the influence of the electric field the negatively charged proteins move down, different kinds at different speeds; and the bands of protein, into which the kinds have been separated after the electrophoresis, can be seen.

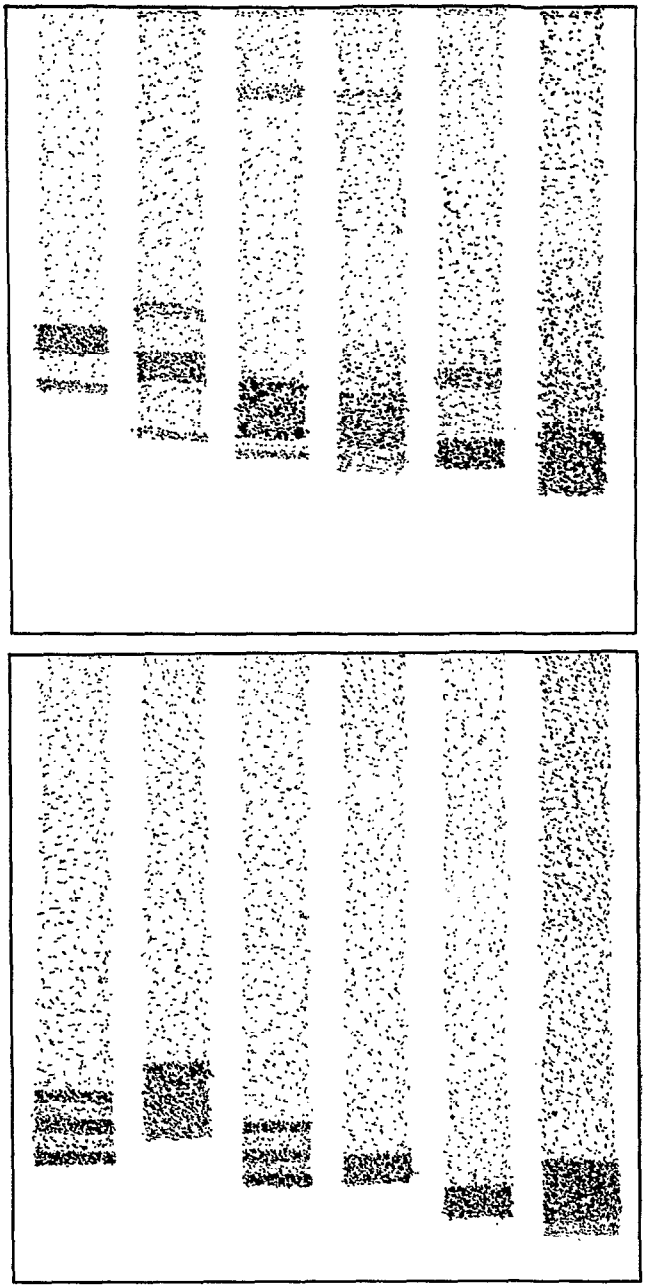

Figure 3. This shows allozyme phenotypes, as separated and revealed by electrophoresis. Above, homozygotes for six different alleles at the *esterase-5* locus in *Drosophila pseudoobscura*. Below, several different heterozygotes between alleles. Drawn after Hubby and Lewontin.

to analyse the structure of scores of proteins in hundreds of individuals. What is needed is some characterization of proteins that is sensitive to single amino acid substitutions but allows reasonably rapid examination of many individuals and many proteins. For that purpose, geneticists have turned to the use of the physicochemical properties of proteins.

Most amino acids are electrostatically neutral; but two of them have a positive charge and two have a negative charge. A polypeptide made up of a mixture of these three types of amino acids will therefore have a net negative or positive charge, varying with the pH. If an allelic change at a locus results in the replacement of an amino acid by one with a different charge, the net charge of the protein will be altered. Such changes in net charges can be used to separate proteins and thus to identify the products of different alleles of the same locus, by means of a technique called *gel electrophoresis*.[5] This technique is illustrated in Figure 2.

About 10 years ago the first revolutionary results with this method regarding the central problem of "evolutionary" genetics were published. The group of H. Harris in London had studied 10 human enzymes,[6] and the group of R. C. Lewontin in Chicago had studied eight enzymes and 10 larval hemolymph proteins from *Drosophila pseudoobscura*.[7]

Both studies showed a great protein variation; while in the *Drosophila* study it was shown that, for every varying protein and enzyme, the variation was the

result of the segregation of alleles at single loci. See Figure 3. This allows the calculation of the amount of polymorphy and of heterozygosity in a population, and, moreover, the approximate equation of each of the invariant proteins to an invariant locus.

It turned out, in both studies, that a third (!) of all loci were polymorphic, and that the average individual is a heterozygote at one out of eight or 10 (!) loci. These estimates become even more impressive when one realizes that a majority of amino acid substitutions do *not* involve charge changes and thus escape attention. It is possible, therefore, that the average heterozygosity per locus is about 35%, and essentially every gene is polymorphic! At first view, the *CT* seems to be firmly refuted in favor of the *BT*; but, as will be seen, the situation might still be quite different.

Results Confirmed in Further Studies

Much more extensive and accurate studies, also in other species, followed the pioneer work of 1966, although the danger increased that studies were biased because of known variability. However, all the more recent adequate and reliable studies completely confirm the results of Harris and Lewontin.

In a dozen species examined the median proportion of polymorphic loci turned out to be 30% and the median heterozygosity per individual 10.6%. Remember that these are minimum estimates since they are based on only those gene substitutions that are detectable electrophoretically. Similar, though less accurate, results are known in several other species, including plants.

Although these results are impressive one really must ask how representative the genes examined are. They thus far have been restricted to genes coding for soluble enzyme proteins and a few nonenzymatic molecules; nothing is known of structural protein or of controlling genes.

There is evidence, it is true, that the soluble enzymes examined are representative of enzymes in general, and that the enzymes give a fair estimate for all the coded proteins. Still, one must remain very careful when it is considered that the few scores of genes examined in, e.g. man, are few indeed compared with the three million genes that might be coded for by the 3×10^9 nucleotides in the DNA of each of his sperm.

Classical versus Balance Theory Reconsidered

It is striking that, apparently, natural selection can both preserve and destroy intrapopulation variation. Some cases of polymorphism have been clearly shown to be due to *balancing selection*, arising from a fitness superiority of heterozygotes over homozygotes or other causes. On the other hand, natural selection can decrease genetic variation in a population by selection against deleterious genes or against heterozygotes.

This paradox would have surprised Darwin, who recognized that intrapopulation variation is the source of the eventual interspecies variation, but had to assume that variation was constantly reduced by "the survival of the fittest" without knowing the source of new variation. This gap was filled by Mendelism.

The *CT*, however, is the direct inheritor of pre-Mendelian Darwinism because it still considers natural selection as antithetical to variation. It holds that the genetic basis for further (micro-) evolution is either lacking or extremely rare most of the time in the history of a population; because natural selection is efficiently sweeping out any variation that might otherwise accumulate.

On the contrary, the *BT* asserts the Mendelian possibility that natural selection preserves and even increases the genetic intrapopulation variation. It therefore regards adaptive evolution as immanent in the population variation at all times.

One might think that the clear evidence of vast quantities of polymorphism and heterozygosity would have utterly refuted the *CT* and firmly established the *BT*. This is not at all the case, however.

The *CT* has replied by stating that the substitution of a single amino acid, although detectable in an electrophoresis apparatus, is in most cases not detectable by the organism, and therefore may be completely indifferent to the action of natural selection. They are "genetic junk", or, *neutral mutations* from the standpoint of natural selection.

This new version of the *CT* has been called the *neoclassical theory (NCT)* or *neutral mutation theory*, and its proponents *neoclassicists* or *neutralists*. It states that:

(a) *many* mutations, it is true, are subject to natural selection, but these are almost exclusively deleterious and are removed from the population;

(b) a second *common* class is that of redundant or neutral mutations, and it is these that will be found segregating when refined physicochemical techniques are employed;

(c) a third group consists of *rare* favorable mutations which will be fixed by natural selection (since "after all adaptive evolution does occur"!) and of *occasional* heterotic mutants.

It is to be noted that the *NCT* cannot be disposed of by pointing to instances in which single amino acid substitutions do have large consequences, or to occasional observations of balanced polymorphisms (like the wearisome single example of sickle-cell anemia), because the *NCT* does not deny that such cases exist, but only that they are common and explain a significant proportion of natural variation.

The argument is made up of two parts: (a) an attempt to refute the *BT*, and (b) an attempt to show that the *NCT* is compatible with the data. It is applied to two different sets of facts: (1) the amount of heterozygosity in populations, and (2) the rate of substitutions of alleles in micro-evolution. The *NCT* holds that both (1) and (2) are too large to be accounted for by selection but can be satisfactorily explained by assuming that the genetic variation is largely neutral and that structural differences in most proteins are the result of random fixation of the alleles concerned during micro-evolution. Consider briefly *NCT* statements about points (1) and (2):

(1) The large amount of heterozygosity. The most telling evidence against the *BT* as the explanation for the observed standing variation in populations is that the predicted "inbreeding depression" (i.e., decrease of fitness) under the *BT* would be vastly greater than what is observed unless heterozygote fitness is extremely

small. On the other hand, the application of the *NCT* to every case of heterozygosity can just as well be shown to lead to absurd results. Also, some results from studies on allelic frequency distributions between reproductively isolated groups are strongly against a hypothesis of random drift of allelic frequencies, i.e., the *NCT*.

(2) The high rate of allelic substitution. The rate of amino acid substitution in "micro-evolution" has been shown to be suspiciously fast under an adaptive theory (the *BT*), but is perfectly consonant with random, non-adaptive distribution as supposed by the *NCT*. However, the proponents of the *BT* have objected that, if indeed the vast majority of amino acid substitutions in micro-evolution have been the result of the random fixation of neutral alleles, the *NCT* would have to assume, even if it allowed as much as 10% of substitution to be adaptive, that neutral mutations are 4,000 times more frequent than mutations with a very slight advantage (0.1%), and in a more usual version of the *NCT* this is 40,000 times!

What Can the Creationist Make of This?

First conclusion for the creationist: It is quite embarrassing for the evolutionist that there are very strong reasons for rejecting both the *BT* and the *NCT*, which are the only elaborate models for explaining the genetic variation that is supposed to be the basis for evolutionary change! How can such a rich theoretical structure as population genetics fail so completely to cope with the body of facts? The problem must be in the structure of this science.

First, there are too many parameters in the theory that are not measurable to the degree of accuracy required so that often no discrimination between alternative hypotheses is possible. Lewontin says that where that is the case

> the theory becomes a vacuous exercise in formal logic that has no points of contact with the contingent world. The theory explains nothing because it explains everything. It is my contention that a good deal of the structure of evolutionary genetics comes perilously close to being of this sort.[8]

Secondly, population genetics can only refer to equilibrium states and steady-state distributions, whereas in fact it should be applied to historical processes.

Thirdly, the usual treatment of the genome as a collection of single loci ignores both physiological interaction and linkage between genes. If different loci are correlated in their allelic distributions, then the dimensionality of the dynamic system is much greater than the number of loci.

It is the merit of Lewontin that he has urged that a theory needs to be developed which takes into account the "evolution" of the genome as a whole rather than the independent "evolution" of each gene.

It is likely that the very weak state of present "evolutionary" genetics will improve in the future. But at present every creationist may be assured that any assertion of evolutionists that genetics has proved that evolution is possible and likely is totally false. *Not only has "evolutionary" genetics nothing to say to the supposed phyletic evolution, not only has it no quantitative model even for species formation, but it has not even reached agreement on the possible meaning of the genetic variation observed in natural populations.*

Genetics of the Formation of Species

As was just stated, it is the irony of "evolutionary" genetics that it has made no direct contribution to Darwin's fundamental problem: the origin of species. It is not that there are no interesting theories about it; but geneticists are a long way from describing speciation in general genetic terms to constructing a quantitative theory of speciation in terms of genotypic frequencies.

This is a long way off, largely because virtually nothing is known about the genetic changes that occur in species formation. To have even the beginnings of a quantitative theory of speciation it is necessary to characterize the genotypic differences between populations at various stages of phenotypic divergence; but even such a characterization has hardly begun.

The general theory of geographic speciation postulates, on the basis of some evidence, that the speciation process begins with a geographical isolation between populations. After that, some distinguish three subsequent stages which are briefly considered here, with a summary of the evidence collected for each of these stages:

(1) Reproductive isolation, i.e., the appearance of genetic differences sufficient to restrict severely the amount of gene exchange that can take place between the populations if they should again come into contact. This might be largely caused by a divergence in ecological niche; but this point is still very vague. Now the genetic question is: How much and what kind of genetic differentiation is required for primary mechanisms of reproductive isolation to arise? Which fraction of the genome is involved in it?

Information to this first stage is nearly completely lacking. Only one case has been studied in which populations had newly acquired reproductive barriers in isolation from each other. S. Prakash discovered between 1967 and 1972 that the Bogotá population of *Drosophila pseudoobscura* (which is far removed from all the other *pseudoobscura* populations) is in the first stage of becoming a new species.[9]

One of the first steps of this process is apparently that Bogotá females crossed with males from any other locality produce completely sterile sons, while the reciprocal cross produces normal sons. The Bogotá population probably colonized the area not much before 1960, apparently from a small number of flies; because a study of its genetic variation showed that it is only half as heterozygous as the rest of the species populations.

The genetic basis for its apparent reproductive isolation must be very restricted; because there has been no genetic differentiation at the 24 loci examined for it. The distribution of some of these species is shown in Figure 4.

(2) *The reinforcement period*, i.e., the renewed contacts between the isolated populations and the subsequent reinforcement of the reproductive barriers by natural selection, in that the newly arisen physiological differences cause hybrid offspring to be less viable or fertile so that they are selected against. Here the genetic

question is: How much more genetic divergence must occur to produce ecologically differentiated, stable members of the species community?

Evidence for this second stage is also very scarce. The chief difficulty is how to recognize sympatric elements as being in their second speciation stage. On the one hand, some morphological or cytological differentiation between the entities must be observable; on the other hand, evidence of selectionally less favorable hybridization between them must be available.

A few cases are known: the pair of subspecies of the house mouse in Denmark, and the complex of "semispecies" of *Drosophila paulistorum* in South America. In the last case, there is not yet a marked genetic differentiation among the semispecies, whereas there is a considerable differentiation within the group of four sibling species to which *D. paulistorum* belongs, the so-called *willistoni* group.

(3) *The completion of speciation*, i.e., continued, mutually independent micro-evolution, each of the newly formed species becoming simply a part of separate communities of species undergoing further splitting or extinction. The genetic question here is: How much genetic similarity is there between more closely or less closely related species? What is the rate of independent genetic divergence in absolute and "taxonomic" time?

This is the stage about which a little bit more is known although: (a) studies are difficult because artificial hybridization between species, completely isolated reproductively in nature, is rarely possible; and (b) when species are farther removed from each other the only reason for saying that they have common ancestors may be the evolutionistic prejudice (i.e., the question may be begged).

Investigations have concentrated upon morphological differences or chromosomal bases of hybrid sterility but have yielded very few quantitative data about how much genetic difference there is between species. Here again the geneticists were led to the study of a random sample of specific enzyme and protein molecules by means of gel electrophoresis. By use of enzymes of which the genetics have been established by intraspecific study, species can be sampled and compared even when they cannot be crossed; although crosses should be made whenever possible to establish gene homologies.

The pioneering work in this field was done in two studies by Hubby and Throckmorton in *Drosophila virilis* and its relatives.[10] First, they compared soluble proteins from ten species (divided into two phylads) of the *virilis* group. Although several difficulties made their analysis imprecise (largely because the genetics of the group was then unknown), the results suggest that on the average about 14% of the proteins of each of the species may be unique to it, and that the ten extant species most probably trace back to four immediate ancestral forms.

It was concluded that at a minimum of 8.5% of the proteins in the extant species have arisen since their speciation, during whatever stage (1, 2, or 3); and that at a minimum 23.5% of the proteins have changed from the ancestral form of the two phylads.

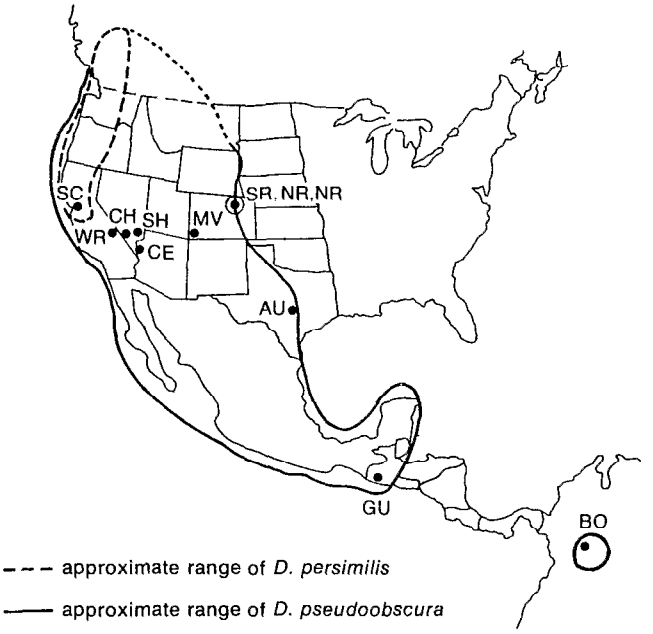

Figure 4. This shows the distribution range of *Drosophila pseudoobscura* and *D. persimilis*, and the locations of the populations sampled, as referred to in the text. BO indicates the Bogotá population, which is also mentioned in the text.

The second study was more precise because it was restricted to enzymes and larval hemolymph proteins, each of which is the product of a separate gene. Nine triads of species were chosen, two members of each triad being (morphological) sibling species, the third being a nonsibling member of the same species group. The results showed a much greater degree of genetic divergence than the results of *D. virilis* had suggested, but also corroborated the view that total morphological similarity between species is a reliable indication of genetic similarity.

A strong improvement of such studies would be not to compare single strains of all the species examined but allelic frequency patterns in related species. Prakash did this in the sibling species *D. pseudoobscura* and *D. persimilis*.[11] The remarkable result was that there turned out to be very little differentiation in gene frequencies, and not a single case of even near-fixation for alternative alleles in the two species. This suggests that species-differentiating genes must be relatively small in number, probably less than 10% of the genome. This is the more remarkable in view of the high degree of polymorphism *within* species.

Later investigations by Ayala and Powell[12] revealed that, for these two species, there are four "diagnostic" loci out of 39 studied, i.e., loci for which the frequencies of the diploid genotypes are sufficiently characteristic for a species to assign an unknown individual to that species with a probability of error of 0.01 or less. These authors carried out the most extensive and interesting comparison of gene frequencies among species, viz. in the four sibling species of the *willistoni* group. They found several instances where species are nearly fixed for alternative alleles, but also some impressive similarities between pairs of species at highly polymor-

phic loci. The fraction of diagnostic genes varied between 14 and 35%.

Second conclusion for the creationist: All these observations together imply that where species are highly differentiated in their alleles there is at least a low-level polymorphism in one species for the genes that characterize the other. There is then a potential genetic transition between species *that does not require the chance occurrence of new variation by mutation*, i.e., the overwhelming preponderance of genetic differences between closely related species is latent in the polymorphisms existing within species.

Even when species diverge farther and farther in the course of micro-evolution within a genus this greater differentiation requires only the occasional input of mutational novelties, while the earlier stages (1, 2, and 3) *make use of an already existing repertoire of genetic variation.* These very interesting observations are the chief consequence, for the process of speciation, of the immense array of genetic variation that exists in populations of sexually reproducing organisms.

For the creationist, this is a very important conclusion because it accurately confirms one of the predictions of the creation model, "involving creative forethought on the part of the Creator, who equipped each kind of organism with a wide variety of potential structures to enable it to adapt rapidly to a wide variety of potential environments in order to conserve and preserve its basic kind".[13]

The creation model predicts that *micro-evolution is not based on the occurrence of random, deleterious mutations but is adaptation based on the innate genetic variation in populations, a prediction fully confirmed by the molecular-genetical results of the last ten years.* "Evolutionary" genetics assumes that the appearance of many novel genes plays a role only in much later stages, possibly macro-evolutionary stages beyond the limits of the basic "kinds". But since macro-evolution does not happen, such things would be irrelevant.

The Assessment of Natural Selection

Neo-Darwinism is based on two notions: random mutations and natural selection. Yet it is totally unknown what proportion of the supposed evolution, or even of micro-evolution, could be possibly ascribed to natural selection. Indeed, evolutionists strongly disagree on this point: for the *BT*, natural selection is the causative agent in the divergence between isolated populations; for the *NCT*, natural selection is always primarily a cleansing agent, sifting out unfavorable gene combinations.

Moreover, studies of genetic variation have not enabled the geneticists to discriminate between these two theories. Now, one could entirely reverse his approach to the problem of whether natural selection mainly conserves genetic variation, or, conversely, decreases it (i.e., whether the *BT* or the *NCT* is right) by trying to measure natural selection directly in nature.

The problem is that it is impossible to determine the over-all importance of balancing selection by demonstrating (by means of examples) that it exists. Of course it exits. But the question is: What *proportion* of observed genic variation is maintained by selection?

This can only be solved by taking some *arbitrary* set of genetic polymorphisms and attempting to establish, for each case, the selective forces involved, through an exhaustive study of natural history and demography. This is the strategy of the school of "ecological genetics", largely inspired by E. B. Ford and the cradle of the *BT*.

The difficulty of such an "un-Popperian" strategy of confirmation rather than of exclusion is that if, say, 100 polymorphisms are objectively examined for balancing selection with a success of 98 positive cases, it could not be reasonably doubted (Popper or no Popper) that balancing selection is the chief cause of polymorphism; if, however, only two cases were proved it might imply either that balancing selection is unimportant or that it is extremely hard to demonstrate.

In fact, only a very few cases of evident balancing selection of polymorphisms have been proved, one of which, the case of the snail *Cepaea nemoralis*, is regarded as a paradigm by selectionists (*BT*). But even this rare instance is not without objections. What is necessary is, eventually, to measure the reproduction of the various genotypes at a locus and to calculate fitness values.

However, although there is no difficulty in theory in estimating fitnesses, in practice the difficulties are virtually insuperable. To the present moment *no one has succeeded in measuring with any accuracy the net fitnesses of genotypes for any locus in any species in any environment in nature.* Even attempts to estimate some individual components of fitness, involving the danger of giving a distorted picture of total fitness, have met with many difficulties.

Less pretentious attempts have restricted themselves to show at least that selection must be operating, even though it cannot be measured, by correlating the frequencies of alternative alleles with temporal and spatial differences in environment. A pretty large number of cases, published in, say, the last 10 years, has shown that undoubtedly polymorphism for electrophoretic variants is indeed under the influence of selection in some cases; nevertheless, they do not reveal how much selection goes on in nature.

If selection cannot be measured or even demonstrated as a general principle in nature, the selectionist can take yet another step back and make a still weaker demonstration. If it could be demonstrated that in laboratory conditions there was selection for some allele at a polymorphic locus, then it would be established that the substitution of such an allele does, in fact, make a significant physiological difference to the organism.

However, the record of detected selection of polymorphic enzyme loci in laboratory conditions is not a very large or convincing one. The most carefully designed and controlled work, that of Yamazaki[14], revealed no selection.

Some geneticists have suggested that the fitness of a given genotype is not fixed but is *frequency dependent.* This would imply that a stable equilibrium of gene frequencies is possible without heterosis, indeed even with an inferior heterozygote. Such a specialized model of fitness can be justified from the simplest ecological consideration. If resources are in short supply and if

each genotype exploits them in a slightly different way, then each individual is in more intense competition with others of its own genotype than with those of other genotypes.

Evidence of frequency-dependent selection, especially viability of larvae competing for resources, is abundant. On the other hand, all that this hypothesis has done is to transfer the problem of information from the sorting of genotypes ("genetic load") to the sorting of environmental niches.

The selectionist (*BT*) can still take one more step in his retreat from the direct measurement of fitness in nature. The *NCT* predicts that the vast majority of amino acid substitutions observed to be segregating in populations have no effect on the physicochemical properties of the enzymes, i.e., are selectively neutral.

A valid attack on the *NCT* would then be a demonstration that the kinetics of different allozyme variants are indeed different. A number of studies of the activity of enzyme alleles did in fact show significant differences, sometimes correlated with clines in nature; this certainly puts the *NCT* in a shaky position.

Third conclusion for the creationist: Not only on the basis of changes in genetic variation is the geneticist unable to discriminate between the *BT* and the *NCT*, as was shown before, but it now appears that neither is he able to do so on the basis of observations of natural selection. On the contrary, it is *still not even known whether natural selection plays at all a considerable role in micro-evolution, let alone that one could quantitatively determine this contribution.*

Upon comparing this with the second conclusion, it is seen that, of the two elements of Neo-Darwinism: random mutations and natural selection, the first hardly plays a role in micro-evolution; and the second perhaps no more.

This conclusion is not an invention of fanatical creationists, but one that is explicitly drawn and corroborated in the important book of a convinced and leading evolutionist, Richard C. Lewontin.[15] Neither need creationists repeat this conclusion with a sort of unholy glee, for creationists need have nothing against population genetics—on the very contrary!

No, there are other reasons to describe this present state of "evolutionary" genetics: *if population genetics so far has not even supplied a quantitative description of micro-evolution the assertion that genetics has supplied a basis for a belief in macro-evolution is utterly unfounded.* This simply takes away one of the pillars of the evolutionary doctrine—and creationists are well aware of the fact that the other pillar, paleontology, is not a bit more reliable.

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[5]Very briefly, an electric current is passed along a strip of gel. Electrically charged proteins (or other electrically charged particles) which have been suitably applied at one end of the strip have a force acting on them, due to the electric field; and they are slowly swept along the strip. Different proteins, because of differences in their structures, are moved with greater or less ease; hence the different kinds of protein are separated into bands spaced along the strip, as shown. Then they can be identified. See Wieme, R. J. 1959. Studies in agar gel electrophoresis. Arscia Uitgaven N. V., Brussels.

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