DOES THE SCIENCE OF GENETIC AND MOLECULAR BIOLOGY REALLY GIVE EVIDENCE FOR EVOLUTION?

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By reference to beans, roses and corn, variation is shown to be limited and not unlimited as Darwin thought. Mutations are generally harmful. Even assuming a 1% advantage, which no mutation reported has actually shown, rate of accumulation of mutations in a species is so slow that it would take about 1,000,000 years for a species population to become uniform for one mutation. This makes them ineffective even in microevolution as means of accumulating the constant features distinguishing species. The giraffe is used to illustrate this fact.

Also biological species show remarkable variation in chromosome number and form. Translocation and inversions occur rarely and spontaneously in species populations. Most translocations in the fruit fly, Drosophila, are either invariable when homozygous or cause a reduction in fertility in one or both of the sexes. In plants, homozygous translocation are usually normal in fertility and vigor. However, none of them are more vigorous than the normal or standard type. Accordingly there is simply no way for them to become established as homozygotes in all the individuals of the population. Experimentally produced polyploids are variable in chromosome number due to quadrivalent formation and so their offspring have a variable chromosome number. Also they are reduced in fertility, so could not become established in nature, since natural selection would operate against them.

Some idea of the complexity of the DNA-RNA system is given. This remarkable interlocking system could not be the result of chance variation. Also reference to work with bacteriophage and tobacco mosaic virus shows that these organisms will not stand the slightest change in the nucleotide bases or their order in the very long and complex DNA molecule. Only the genius of a remarkably intelligent Being we worship as God could have designed such an efficient yet intricate system.

I

Variation and Mutation in Relation to the Evolution Theory

According to Charles Darwin, natural selection of advantageous variations leads to the formation of new species, genera, families, and indeed all the marvelous variation we observe in the realm of biology. He considered variation as essentially unlimited with those individuals most fitted to the environment being naturally selected. In the following generation, the same range of variability would occur again. Thus in the classical case of the evolution of the giraffe, quoting Darwin[1]:

> So under nature with the nascent giraffe, the individuals which were the highest browsers, and were able during dearths to reach even an inch or two above the others, will often have been preserved; for they will have roamed over the whole country in search of food. . . . These slight proportional differences, due to laws of growth and variation, are not of the slightest use or importance in most species. But it will have been otherwise with the nascent giraffe, considering its probable habits of life; for those individuals which had some one part of several parts of their bodies more elongated than usual, would generally have survived. These will have intercrossed and left offspring, either inheriting the same bodily peculiarities, or *with a tendency to vary again in the same manner;* . . . By this process *long-continued,* . . . combined no doubt in a most important manner with the *inherited effects* of increased use of parts (the neck), it seems to me almost certain that an ordinary hoofed quadruped might be converted into a giraffe (Emphases added).

It should be noted that Darwin assumes (1) continuous variation, each generation showing the same *range* in variation of neck length, and (2) effect of continuous use or disuse. He even devised a theory of pangenesis, now disproven, to explain this presumed inheritance of the effects of use and disuse.

The inheritance of acquired characters is no longer believed by any competent biologist. There still seems to be some confusion as regards variation. Most major genetic factors such as tall vs. dwarf in the garden pea have modifying factors. Accordingly, slightly taller plants may be obtained by selection.

Since the pea and also the bean used by W. L. Johannsen are self-pollinated, they breed true, or as we say technically, are *homozygous* for most or all of their genes. Thus Johannsen grew one crop from the largest beans and another from the smallest, in separate plots of ground.

Then in each of the two fields he selected the longest and the smallest beans and planted them in four separate plots. Measurement of the bean in these plots showed **no greater variation** than was found in the first generation. Continued selection for four more generations showed that after the first generation, selection had no effect in either increasing or decreasing bean size.

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Roses, Corn Have Greater Differences

Roses and corn are cross pollinated, hence there is a much greater store of genetic differences. All individuals are *heterozygous* for most character distinctions, or do not breed true when self-pollinated. In fact because of the accumulation of harmful recessive genes, experimentally-forced self-pollination results in a population of plants, most of which are much weaker than the parental stock. Selection therefore to establish true breeding lines takes much longer.

Thus, in roses, there is a great variation in bud length from the short rather stubby bud of Crimson Glory to the lovely long bud of Charlotte Armstrong. Intercrossing of rose varieties with the longest buds results in a great increase in bud length in the next generation. Less effect is noted in the second generation and after five or six generations no further increase is found. Today roses are partly self-pollinated and partly cross-pollinated, so they are more variable than peas or beans.

Corn, however, is completely cross-pollinated. Hence a maximum amount of variation is found. Even so corn breeders made phenomenal progress in increasing yield during the first twenty years. By then the inbred lines used to produce the famous high-yielding hybrid corn seed had accumulated most of the major factors for high yield, so relatively little increase in yield is now possible.

Selection for high and low oil content in corn still continues after 50 generations. Starting with an average yield of 4.7% oil, the high oil content line is now about 15.6% and the low is 1%. Though the selections for low oil content have shown little change since the 35th generation, selection for high oil content has increased from about 11% in the 35th generation to 15% in the 50th. Evidently a very great number of rather slight factors for higher oil content exist in corn. However, the limit is obviously being reached.

All of our breeding experience with peas, beans, roses and corn indicates that contrary to what Darwin believed the variability potential of each species is definitely limited.

Mutations Are Favored Mechanism

On what, then, do present-day Neo-Darwinian evolutionists rely for the mechanism of evolution? The answer is mutations, which occur with varying frequency in plants and animals. Actually, as will be shown later, these result from a "mistake" in the process of gene reproduction or duplication of the DNA molecules which either are or "house" the genes determining the characteristics of plants and animals.

Now it is generally conceded that most mutations are harmful. Estimates of the number of mutations that show no harmful effects vary from 1 in 1000 (according to one source[2]) to 1 in 5000, depending on the person making the estimate.

The odd feature of these estimates is that actual data on just how much *better* a mutation is under usual conditions are very difficult to locate. Timofeef-Ressovsky[3] indeed did show that the viability of the mutation "eversae" in *Drosophila melanogaster* was 104% of the normal at 75-77°F. At lower temperatures it was only 98.3% of the viability of the normal fly.

A theistic evolutionist correspondent of mine once criticized my use of this quotation from Fraenkel-Conrat[4]: "One can assume that the protein coat of the common strain of virus as it evolved by natural selection is highly efficient, and that any mutation is likely to reduce the virus' ability," as evidence for the impossibility of explaining evolution by mutation. My correspondent felt that I had not paid attention to the words "is likely to."

Yet, neither in Fraenkel-Conrat's report nor Seymour Benzer's study[5] of the rH mutants in the T_4 bacteriophage were any beneficial mutants reported! In fact Benzer located 250 of an estimated 350 sites, and all reported are defective.

But let us grant that mutations with a 1% advantage under usual environmental conditions are found. What are the chances of establishing this mutation in *all* the individuals of a typical large species population? Patau[6], a keen student of population genetics, estimated that such a mutation would increase in frequency from 0.01 to 0.1 percent of the population only after 900,230 generations. Another 100,500 generations are needed to increase the frequency to 100 percent!

Serial Beneficial Mutations Incredible

Even geologically speaking this is a mighty long time when one is considering plants or animals with an annual breeding cycle. And when one begins to think of the incorporation of such changes in the presumed ancestors of the human race, this means about 18-20 millions years for the incorporation of one presumably beneficial mutation.

It is true that one can *postulate* the simultaneous accumulation of such mutations. But it is stretching credulity to postulate the fortunate chance that a *series* of beneficial mutations, leading to such progressive changes as the lengthening of a giraffe's neck, would all occur *at about the same time.*

Admittedly, mutations affecting any given characteristic occur strictly according to chance. Therefore, accumulation of beneficial changes would have to be sequential. Also, even with a feature as *relatively* simple as the height of a

giraffe, elongation of bone structure in both neck and forelegs have to be correlated with lengthening of muscles, and also strengthening of them. Truly, evolution of a giraffe would be a most time consuming process. Their breeding cycle is about 5 years, or 5,000,000 years per mutation and geologically speaking not much time was left after the presumed ancestor of the giraffe first appeared in the Eocene era.

Bacterial Studies Reported

With the discovery that strains of bacteria resistant to penicillin, aureomycin, or chloromycetin always showed up when these drugs were used to effect cures for various diseases, evolution-minded biologists believed that here at last real proof for beneficial mutations existed.

Soon it was discovered that these mutations did not arise as a result of exposure to the drugs. Rather they occur at a constant rate. Associated with the resistance, there is a decrease in viability under *normal* conditions. Hence they are soon "swamped out" and carried along in a very small number of individual bacteria.

When a strain of bacteria is exposed to antibiotics, either the mutation rate for these otherwise defective resistant mutations is so high that eventually one occurs, or an already established one is given the starting advantage of having no competitors. Soon the entire population in the sick human being is the resistant type, and new medication is necessary to effectively treat the disease.

C. P. Georghiou[7] has discussed resistance of house flies to DDT and other chemicals. The results are parallel to those found in bacteria. The author writes:

> It is now well established that the development of increased ability in insects to survive exposure is not *induced directly* by the insecticides themselves. These chemicals do not cause the genetic changes in insects; they serve only as selective agents, eliminating the more susceptible insects and enabling the more tolerant survivors to increase and fill the void created by the destruction of susceptible individuals.

In addition he observed that flies at a cattle feed lot and at a nearby poultry ranch showed little resistance to any organophosphates or carbonates, since they had not been sprayed very often with them. Yet *agricultural* crops in that area had been treated regularly. Evidently the resistant strain of flies, though able to maintain itself once established is *incapable of spreading* through the whole range of the species even in a given area such as Blythe, California, where this work was done. Surely the flies in the nearby agricultural area became resistant as a result of the frequent spraying of the crops, yet feed lots and poultry farms had a low level of resistance. Also in no instance were 100% of the flies, *even in the areas most exposed to spray,* resistant to the chemicals used.

We may therefore conclude that resistant strains of various organisms simply do not become established in species populations, hence can hardly lead to permanent evolutionary change.

II
Can Translocation, Inversions and Polyploidy Explain Chromosome Variation in Species?

For many years it has been known that the genes determining various characteristics of plants and animals are located in rod-shaped bodies called chromosomes. As implied by the name, these structures absorb stain more readily than the rest of the cell or nucleus when various colored dyes are used on them.

Each organism has a characteristic number of chromosomes. Also the appearance of them varies greatly from species to species. Usually their minute structure is studied in plants by examining them in very early stages of cell division when the pollen grains are being formed. Then it can be clearly seen, when the pollen mother cells are examined under an oil immersion lens at 1500 diameters, that the hereditary factors are located in small bead like bodies called **chromomeres** located serially along the entire length of the chromosome. A great deal of mapping of these chromosomes has been done in corn and tobacco, correlating hereditary characteristics of the plants with either the presence or absence of special chromomere sequences.

Most studies of species differences as regards the precise arrangement of the genes have been done in the fruit fly, *Drosophila.* Details of the chromomere arrangement are very difficult to study during reduction division, or formation stage of eggs and sperm in the fruit fly. Fortunately the chromosomes increase greatly in length in the salivary glands, where they are clearly defined cylindrical bodies.

The size, shape and staining reaction of the discs or striations found in the chromomeres vary sequentially along the length of the chromosomes. However, they show a remarkable constancy for each species, and reflect in their order of arrangement the known order of gene arrangement as determined by genetic analysis.

Chromosome Breakage Diagramed

Very rarely chromosomes break spontaneously. X-ray or neutron radiation greatly increases breakage. If two breaks occur in a given chromosome, the broken piece may become inverted end for end before reuniting with the rest of the chromosome. Thus, if the original chromosome

is a b c d e f g, breakage in two places may be indicated as follows: ab cde fg. The newly reconstituted chromosome if the broken segment c d e is inverted 180° will be a b e d c f g and is called an **inversion.**

Similarly breakages may occur in two different chromosomes and the segments reciprocally transferred from one to the other. This phenomenon may be shown schematically as follows:

Chromosome 1	Chromosome 2
Translocated Chromosome 1	Translocated Chromosome 2

When gametes are formed by an individual heterozygous for a reciprocal translocation, about 50% of the gametes will be inviable since they will lack either part of chromosome 1 or chromosome 2. Only gametes having both chromosomes of the normal type, or both translocated 1 and translocated 2 will be viable.

Fruit Fly Species Compared

Now let us compare two species of *Drosophila,* namely *D. miranda* and *D. pseudo-obscura.* Externally these two species are remarkably similar. D. *miranda* is somewhat larger in size, darker in coloration, with less distinct grayish stripes on the thorax and slightly wider cheeks. But internally the chromosomes are profoundly different as shown in Figure 1.

Genes which are next to each other in one species are often far apart in the other, or may even be located in other chromosomes. This is considered as indication that inversions and translocation have occurred. Many chromosome sections are so dissimilar that no resemblances in chromomere structure could be found at all. Dobzhansky[8] suggests, "that some chromosome sections have been so thoroughly rebuilt by repeated inversions and translocation that their disc patterns in the salivary chromosomes no longer resemble each other and no pairing of the homologous genes takes place."

As regards the rest of the chromosomes, Dobzhansky and Tan[9] estimate that a minimum of 49 breakages and rebuilding by inversion and translocation are needed to account for the origin of these species from a common ancestor. This magnitude of differences in chromosome pattern is typical of that found in many "closely related" species. In tobacco Elvers[10] has shown that practically no homologous sections are found in the chromosomes of the hybrid *Nicotiana glutinosa X N. tomentosa.*

Chromosome Arrangement Explained

Now what is really involved in attempting to account for differences in chromosome arrangement in externally similar species having the same chromosome number? Most of the specialists in *Drosophila* research believe that the gradual sequential accumulations of inversions and translocations would account for the differences.

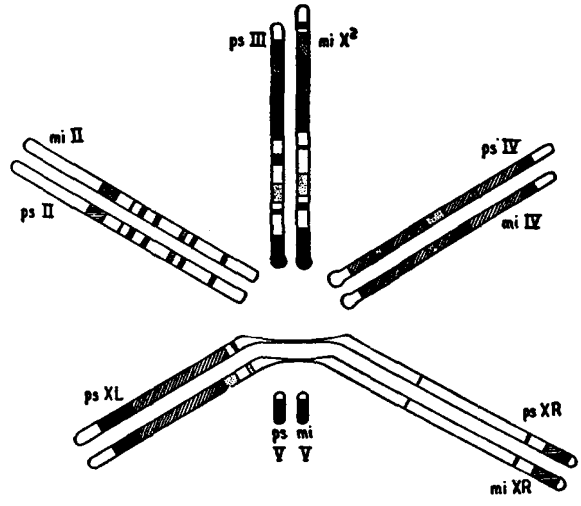


Figure 1. Comparison of chromosomes of *Drosophila pseudoobscura* and *D. Miranda.* Sections having the same arrangement of genes are white. Inverted sections are cross-hatched; translocation stippled; sections completely dissimilar are in black. ps = *D. pseudoobscura* and mi = *D. miranda.* The chromosomes in the salivary glands are actually much more elongated relative to their diameter.

However, many chromosome breakages are accompanied by slight deficiencies or losses of essential genetic factors. When the sex cells or gametes carrying such deficiencies unite, the resulting individual fly lacks an essential bit of hereditary material in both the maternal and paternal chromosome. In practically all cases it either dies very early in life or is relatively weak. Though some translocation have been established in homozygous condition the fertility of either males or females is lower than the wild type or normal fly.

The situation is quite different in plants where homozygous translocation established in corn by Burnham[11] showed no external differences from the normal plants and were fully fertile. Similar results have been reported for sorghum.[12] A wheat rye hybrid translocation and its derivative Transec "enjoy a fairly normal phenotype which is a reflection of the fact that both involve the loss of genetically unimportant wheat chromatin."[13]

Four reciprocal translocation homozygotes have been obtained in the watermelon, in hopes of getting seedless melons by crossing them with the normal strain.[14] Except for a slight amount of reduction in pollen fertility they were quite

normal. A number of translocation homozygotes such as Sonora and S2303 have also been reported in wheat.[15] Though not discussed presumably these varieties are normal in vigor and fertility.

The interesting feature of the inversions and translocation referred to above is that none of them is reported to be *superior* to the standard type in either vigor or fertility when in homozygous condition. As discussed above, mutations with even a 1% advantage would become fully established in 100% of the population only after about 1,000,000 generations. How then could translocation and inversions having **no selective advantage** ever become established and thus form new species as distinctive as *D. pseudo-obscura* and *D. miranda?*

The spontaneous occurrence of both inversions and translocation is very low, that is between 1 in 100,000 and 1 in 1,000,000 fruit flies. The heterozygotes are reduced in fertility; thence, the chances of establishing translocation and inversions in homozygous condition are very low. However, it has been experimentally determined that inversion heterozygotes (and possibly translocation ones also) usually show hybrid vigor, and thus are superior to the homozygotes in vigor.

Dobzhansky has shown that the heterozygous inversion obtained by crossing the standard with the CH strain has an adaptive value of 1 or 100%. as compared to 90% for the standard (ST) and 40% for the CH strain.[16] Furthermore the frequencies of the various inversions varied with the altitude in the Sierra Nevada Mountains near Mather. At the lowest elevations the ST chromosomes are the commonest, but at 10,000 feet the Arrowhead strain (AR) is most frequent with CH greater in frequency than ST.

Uniform Population vs. Natural Selection

The main feature of these observations is that natural selection does not make the population uniform for any one of the inversions in any of the observed locations. Yet, uniform populations in which each fly is homozygous for a particular inversion are precisely what are needed as evidence that inversions can be appealed to as the cause of chromosome differences in gene arrangement in species. For species are remarkably uniform as regards the order of arrangement of the genes as shown by the discs in the salivary gland chromosomes.

Bruce Wallace indeed makes the above claim in his book *Chromosomes, Giant Molecules, and Evolution.*[17] He gives the distinct impression that the inversion races of *D. pseudo-obscura* are uniformly characteristic of the various geographical races and occur therefore as true breeding homozygotes. If this were true, it would indeed be a strong argument for the origin of the much more complex species differences, even though the exact mechanism for the natural establishment of homozygous inversions has not been demonstrated.

However his thesis breaks down completely when we realize from Dobzhansky's experiments that the heterozygotes are usually more vigorous; hence, natural selection would never establish a new species by the sequential accumulation of homozygous inversions. In fact the fifth chapter, "Reconstructing Inversion Sequences," in Wallace's book so carefully avoids the problem of establishing inversions and translocation in a population, that one wonders if omission of the problem might not be deliberate!

Looked at from the creationist viewpoint the remarkable complexity of the chromomere or disc arrangements which distinguish one species from another are clear evidence of the Creator's amazing ability to give individuality to each of His many creations, which we as scientists are only beginning to appreciate.

Chromosome Doubling Considered

Interspecific hybridization followed by doubling of the chromosomes of the hybrid has often been appealed to as the means by which species having multiples of a basic number have evolved. Such, for example, are the wheat series having 7, 14, and 21 pairs of chromosomes.

Much has been made of Karpechenko's famous hybrid of the radish and the cabbage, each of which have 9 pairs of chromosomes. Chromosome doubling in the F_1 hybrid is claimed to have resulted in a true breeding cabbage-radish hybrid having 18 pairs of chromosomes. However as I discussed in detail in the *1964 Creation Research Society Annual* (p. 50), later work by Richaria[18] and Howard[19] showed that even in the fourth generation the chromosome number varied from 33 to 37 chromosomes and the plants varied in fertility from 5 to 42%!

Also discussed in the current 1969 *Annual* by Klotz,[20] *Primula kewensis,* usually reported in evolution oriented books as having regular 18 pairs, actually forms several groups of four chromosomes or quadrivalents. Accordingly plants vary in chromosome number from 34 to 37.

My own *Nicotiana rustica X paniculata* amphidiploid showed the same chromosome variability. In fact all experimentally produced polyploids show this variable behavior. They can therefore hardly be compared to such naturally occurring polyploids as wheat which regularly has 21 pairs of chromosomes, or tobacco *(N. tabacum)* which has 24 pairs and are fully fertile.

III
Complexity of DNA-RNA System, a Clear Indication of Design in Nature

Only in recent years have biochemists found out precisely what the factors or genes governing the heredity makeup and physical development of an organism really are. Thanks primarily to the work of Watson and Crick we now know that the genetic code is based upon the order of arrangement of two purines, adenine and guanine, and two pyrimidines, cytosine and thymine. Any one of these when united with phosphoric acid and deoxyribose sugar makes up a nucleotide.

These nucleotides are arranged in two strands twisted about one another in the form of a double helix or coil. In this coil adenine is always paired with thymine and guanine with cytosine. On one side of this double coil the nucleotides are arranged in any order. However once that order is established the other strand is determined, that is, an adenine nucleotide must *always* pair with a thymine, and the guanine with a cytosine. The interconnections are simply endless repetitions of sugar-phosphate-sugar-phosphate.

As explained by Duane T. Gish[21] the simplest code by which the 20 amino acids used in making proteins could be specified involves at least three nucleotide pairs, such as ATT, GCA, TCG, ACC. (A = adenine, C = cytosine, T = thymine, and G = guanine.) Thus, if we postulate that only one nucleotide governs the use of one amino acid, only four amino acids could be coded.

Likewise any two nucleotides could code only 16 amino acids since there can only be 16 combinations of two nucleotides (AA, AT, AC, AG, TA, TT, TC, TG, CA, CT, CC, CG, GA, GT, GC, GG). Combinations of any three nucleotides can actually specify 64 amino acids, far more than the 20 actually used.

Actually experiments indicate that several different triplets of the DNA may be used to code the same amino acid. Also some of the triplets may be used in such important factors as timing of the union of various amino acid sequences.

Now the "message" evidently begins at a fixed point at one end of the gene and as shown above is read three bases at a time. Thus if for some reason the reading starts at the wrong point the message would fall into the wrong set of three and so would be incorrect. Thus for each correct reading of the code there are two incorrect ones.

Code Translation Explained

How is the code actually translated into action? Briefly, this is effected by the complementary RNA which differs from DNA only by having ribose instead of deoxyribose sugar. RNA is found in the cytoplasm of the cell. The actual location of the protein synthesis is at the ribosomes which are small granules in the cytoplasm. Thus, the RNA is a reverse copy of its DNA template and acts as a messenger, passing into the cytoplasm and effecting a particular pattern on the ribosomal surface.

Other sorts of RNA, already free in the cytoplasm, pick up individual amino acids and carry them to the ribosomes. This type of RNA is called transfer-RNA. There is a distinctive form of transfer-RNA for each of the 20 different amino acids. The amino acids are then arranged *according to the order* of the surface pattern previously set up by the messenger-RNA. They are then linked together into a polypeptides chain, part of a protein molecule, and these polypeptides when united form the protein. Very briefly stated, this is the way DNA specifies, and RNA supervises, the formation of whichever proteins are needed by the organism.

When the complex interrelationship of DNA and RNA are carefully considered it becomes rather obvious that *any tampering* with the code is likely to have serious consequences. In this connection a brief review of Seymour Benzer's work with T_4 bacteriophage referred to above is in order. The advantage of using bacteriophage is that in a 20 minute experiment by the use of a single test tube, a quantity of data can be obtained which would require the entire human population of the earth!

Phages are virus organisms having a hexagonal head, and a complex tail used for attaching themselves to a bacillus wall. Within the head is a long-chain molecule of DNA having a weight about 100 million times that of hydrogen. After attachment, the DNA alone moves into the bacillus cell and takes over the reorganization of the cell so as to manufacture 100 or more copies of a complete virus. The bacterial cell then bursts open and liberates these virus organisms, each of which attacks another bacillus.

It is estimated that the bacteriophage DNA contains about 200,000 base pairs. Each base pair is one letter of a minimum three letter word which specifies which of the 20 amino acids is to be linked up into the polypeptides chain. Sometimes an entire "paragraph" of such code words is needed to specify the sequences of amino acids needed for *just one* polypeptides chain and several such chains make up a complex protein.

"Typographical" Errors May Occur

"Typographical" errors may occur in the replica of the DNA molecule. Transpositions, deletions, additions, or inversions may occur. As Seymour Benzer says, "In a daily newspaper the result is often humorous. In the DNA of living organisms, typographical errors are never funny and are often fatal." (Emphasis added)

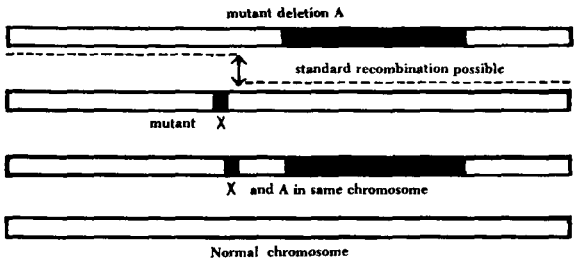


Figure 2. Showing how deletions are used to test location of mutants. Point mutation "x" will recombine with deletion A since it is several nucleotides to the left of it. If located within the length of the deletion it will not show recombination with it.

However these "typographical" errors or mutations can be used to analyze a small portion of the information carried by a T_4 bacteriophage, and thus reveal the amazing complexity of not only the DNA code but the processes of cellular activity as well.

One group of mutants called rH mutants can be identified quite easily by the appearance of clear regions they form on the surface of a culture in a glass dish where phage particles have multiplied and destroyed the bacterial cells. The shape and size of these clear regions are hereditary characteristics of the phage that can be easily identified and scored. A clear region or plaque produced in several hours will contain about 10 million phage particles, the progeny of a single phage particle.

The T_4 phage can produce plaques on either host strain B or K. This standard form gives rise to mutants easily recognizable by a distinctive plague on B cultures. But the mutants cannot form plaques on bacteria strain K. This is the "key" to the whole mapping technique used by Benzer; for an rH mutant can grow on strain K only if the cell is simultaneously infected with a particle of the standard type. Now the function of the standard type phage has been traced to a small portion of the T_4 phage genetic map known as the rH region.

The various different-appearing plaques are due to mutants which arise spontaneously in this area. They may be crossed with each other by adding them to a liquid culture of B cells. This gives the progeny an opportunity to recombine portions of genetic information. If the two mutants resulted from typographical errors in different parts of the DNA molecule, some individuals of the standard type will be regenerated. (see Figure 2) A sort of *crossing over* occurs. These reconstructed standards will produce plaques on the K strain, whereas the original two mutants could not. In this way single recombination among billions of offspring can be detected. This allows the resolution of rH mutants only one base apart in the DNA molecule chain.

Recombination Mechanism a Problem

The exact mechanism of recombination is not known. It seems that two defective DNA molecules may break apart and reunite to form one non-defective molecule which then is replicated. Or it has been suggested there is "copy choice" so that only good portions of the two mutants are copied. This seems to be granting quite a remarkable power of selectivity to some "curative" agency in the T_4 phage cell.

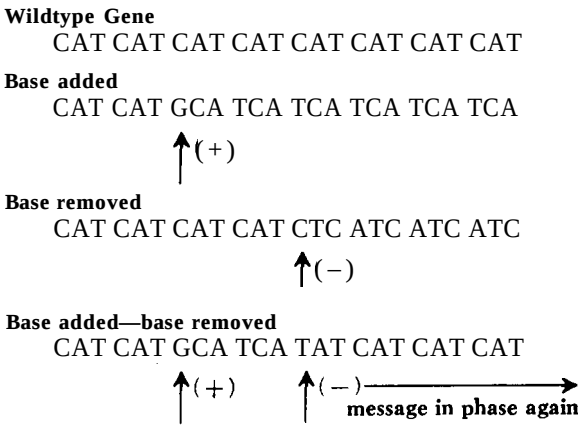
The results of long and elaborate study of hundreds of non-reverting rH mutants shows that *all* can be represented as containing deletions of various sizes in a single linear structure. By contrast, the rH mutants mentioned above behave as if their alterations were localized at single points. By testing against the non-reverting segments or deletions at this particular area of the T_4 phage DNA molecule, all mutants located within a given segment will not recombine when tested against it.

By use of about 80 such non-reverting segment mutations, the rH point mutations may be assigned to the proper one. Finally those localized in one small segmented deletion length or segment are tested against each other. Those showing recombination are obviously at different sites, and then each site is named after the mutant indicating its location. Finally the *order* of the sites within a given segment can be established by measuring the recombination frequencies. Of an estimated 350 sites about 250 have been located and all are defective.

Why No "Good Mutations?

Why then are "good" mutations needed by evolution not found? The reason is clearly shown in a paper by F.H.C. Crick.[22] He shows that the sites discovered by Benzer correspond to changes in the DNA base nucleotides. Mostly the defects are the result of adding or deleting one base, or sometimes a small group of bases. Such addition can be produced at random by compounds called acridines. As described above the code reading starts at the beginning of one gene and is read three bases at a time. Hence the addition or deletion of a base makes the gene completely non-functional.

Experimentally this means that, if a mutant due to the addition of a base is combined with another due to the addition of a base, the combination is still non-functional. Likewise this is true of a minus combined with a minus. But if a plus is combined with a minus the function is restored. This is because starting at one end of the rH region of the B cistron (or gene), the message would be read correctly until the extra mutation-causing base is reached. Then the message would not make "sense" until the minus mutation or missing base is reached, after which



The imaginary message is CAT, CAT . . . Adding of a base shifts the reading to TCA, TCA. Removing a base makes it ATC, ATC. Addition and removal puts the message in phase again. The reading is from left to right in triplets of 3 nucleotide bases.

Figure 3. Showing effect of mutations

the message would come back into phase again or make "sense." This is possible because parts of the rH part of the B cistron are not critically important, hence the message can be garbled for a short distance and still be functional. If the distance is too long, the combination will not function. This is shown in Figure 3 adapted from Crick's paper.

Fraenkel-Conrat reports substantially the same thing in reporting on about 200 chemically induced mutants of the tobacco mosaic virus. Some mutants render the RNA incapable of even forming the protein coat of the virus, others make it more capable of being harmed by the digestion of enzymes.

Genetic Code Is Complex

The picture emerging is that the genetic code is marvelously complex and will stand very little in the way of alteration either by addition, subtraction, or change of any of the nucleotide bases. Only because the rH region was *relatively* unimportant in function was it possible to accumulate the large number of mutations, making possible the detailed analysis of this rather minute portion of the T_4 phage DNA molecule. Yet even here most portions of the code message are of such vital importance that even a *short* portion out of phase causes a completely non-functional message, hence the mutations do not survive.

In discussing the functioning of the DNA system relative to the organism resulting from it Marcel Schutzenberger makes a very pertinent criticism of Neo-Darwinism.[23] He says,

> According to molecular biology, we have a space of objects (genotypes) endowed with nothing more than typographic topology. These objects correspond (by individual development) with members of a second space having another topology (that of concrete physico-chemical systems in the real world). Neo-Darwinism asserts that it is conceivable that without anything further, selection based on the structure of the second space brings statistically adapted drift when random changes are performed in the first space (the DNA system—W. E. L.) in accordance with its own structure. We believe this is not conceivable.

He suggests that algorithms in which the very concept of syntactic correctness has been incorporated, are needed to fill the gap.

As creationists, we believe that the marvelously intricate DNA system, and its RNA counterpart, are God's mechanism for providential care. "For in Him we live and move and have our being." (Acts 17:28). The marvel of it all is that this complexity is based on essentially only five elements: carbon, hydrogen, nitrogen, oxygen, and phosphorus. No one could predict from the physical characteristics of these elements that they could ever be combined to form the intricate series of reactions we know as life.

As molecular biologists continue to learn even more as to exactly how organisms develop by the expression of the DNA code, should they not have a feeling of reverent awe for a Being who could plan all of this, and who continues daily to make it work? Here indeed in a fine field for some of our younger creation-minded students to enter. For here they are truly seeing God at work as He daily maintains and preserves all creatures.

References

[1]Darwin, Sir Charles. 1859. The origin of species. Everyman's Library, Dutton, N. Y. p. 202.

[2]Biological sciences: an inquiry into life. 1963. Harcourt, Brace and World, Inc., New York. p. 613.

[3]Timofeef-Ressovsky, N. W. 1934. Uber die Vitalitat einiger Genmutationin und ihrer Kombinationen bei *Drosophila funebris* und ihre Abhangigkeit vom "genotypischen" und von ausserem Mileau, *Zeitschrift fur Ind. Abstam. und Vererbungslehre,* 66:319-344.

[4]Fraenkel-Conrat, Heinz. 1964. The genetic code of a virus, *Scientific American,* October.

[5]Benzer, Seymour, 1962. The fine structure of the gene, *Scientific American,* January.

[6]Patau, K. Die Mathematische Analyse de Evolutions vorgange. *Zeitschrift fur Ind. Abstam. und Vererbungslehre,* 76:220-228.

[7]Georghiou, C. P., *et al.* Housefly resistance to insecticides, *California Agriculture,* 19:8-10.

[8]Dobzhansky, T. Genetics and the origin of species. Columbia University Press, N. Y. p. 146.

[9]Dobzhansky, T. and C. C. Tan. 1936. Studies on hybrid sterility III: A comparison of gene arrangement in two species, *Drosophila pseudo-obscura* and *Drosophila*

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firmament showeth His handiwork (Psalm 19:1), and furthermore, this "declaration" of God in His created cosmos can never contradict the subsequent and more specific declarations of His written Word.

Some people have questioned the concept of "creation research," since creation is no longer occurring (as stressed both by Genesis 2:1-3 and by the Law of Energy Conservation), and therefore is not subject to experimental investigation. The term of course is intended rather to describe research demonstrating the *fact* and purposes of special creation and the falsity of alternate theories of origins, and thus is a very appropriate name for our activities.

Several articles in this *Annual*, in fact, report results of important original research supporting and illuminating the truth of Biblical creationism. One of these–the thrust-fault study of Burdick and Slusher–was made possible by the first grant from our C.R.S. Research Fund. We hope this is only the beginning of a greatly-extended ministry of this sort. New research proposals, with supporting cost estimates and justification, are invited from our members; and new contributions to the Research Fund for such purposes are also solicited.

In addition to doing and publishing such research, we need to explore better ways of making the results known, especially to the scientific world and to leaders in the Christian world. Such things as gift subscriptions of the *Quarterly* to scientist friends, libraries, church leaders, and others, might well be promoted on a larger scale than has been possible to date. I would be happy to receive any suggestions along this line, and to pass them on to the Board of Directors for consideration.

There are undoubtedly many present and prospective members who have good research ideas, as well as the needed talents and training, for fruitful research in some aspect of creationism but who for various reasons have not yet implemented them. I would encourage you to "get going," in confidence that the Lord will surely guide and use such efforts. The time may be short and the need is great.

HENRY M. MORRIS
President

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miranda, Zeitshrift fur lnd. Abstam. und Vererbungslehre, 72:88-114.

[10]Elvers, I. 1934. Interspecific hybridization in *Nicotiana* XIV. The cytology of *N. glutinosa X N. tomentosa, University of California Publications in Botany,* 17: 341-354.

[11]Burnham, C. R. 1934. Cytogenetic studies of an interchange between chromosomes 8 and 9 in maize, *Genetics,* 19:430-447.

[12]Huang, C. C., J. G. Ross and H. D. Haensel. 1963. Reciprocal translocation in *Sorghum vulgare, Canadian Journal of Genetics and Cytology,* 2:227-232.

[13]Driscoll, C. J. and L. M. Bielig. 1968. Mapping of the transec wheat-rye translocation, *Canadian Journal of Genetics and Cytology,* 2:421-425. (Quotation from p. 424.)

[14]Oka, Hiroyuki, Watanabe, Tadahiro, and Nishiyama Ichizo. 1967. Reciprocal translocation as a new approach to breeding seedless watermelon, I. Introduction of reciprocal translocation strains by X-ray irradiation, *Canadian Journal of Genetics and Cytology,* 9: 482-489.

[15]Baker, E. P. and R. A. McIntosh, 1966. Chromosome translocation identified in varieties of common wheat, *Canadian Journal of Genetics and Cytology,* 8:592-599.

[16]Dobzhansky T. 1961. Evolution, genetics, and man, John Wiley and Sons, Inc., New York, London. p. 147.

[17]Wallace, Bruce. 1966. Chromosomes, giant molecules and evolution. W. W. Norton and Co., Inc., New York. pp. 28-34.

[18]Richaria, R. H. *1937.* Cytological investigation of hybrids, *Journal of Genetics,* 39:19-44,

[19]Howard, H. W. 1937. Fertility of amphidiploids, *Journal of Genetics,* 16:239-273.

[20]Klotz, John. 1969. Chromosomal changes—mechanisms for evolution?, *Creation Research Society Annual,* 6: 45-48, 54.

[21]Gish, Duane T. 1967. DNA: its history and potential, *Creation Research Society Annual,* 4:13-17.

[22]Crick, F. H. C. 1962. The Genetic code, *Scientific American,* October.

[23]Schutzenberger, Marcel P. 1967. Algorithms and the neo-Darwinian theory of evolution in Mathematical challenges to the neo-Darwinian interpretation of evolution. Edited by Paul S. Moorhead and Martin M. Kaplan. The Wistar Institute Press, Philadelphia, Pa. p. 74.