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MUTATIONS REVEAL THE GLORY OF GOD'S HANDIWORK

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Mutations have been studied through three levels of investigation: (1) their original discovery and proof of inheritance according to Mendelian principles; (2) the artificial production of them by radiation and mutagenic chemicals and parallel with this work, the study of their actual behavior in natural populations; and (3) the molecular genetic approach.

In spite of great enthusiasm and many claims, no investigator has shown as yet that ***any*** *mutation is so advantageous as to spread through an entire species population of plants or animals. Molecular geneticists, such as Seymour Benzer, conclude, "in the DNA of living organisms, typographical errors are never funny and are often fatal."*

The technique used by Benzer in analyzing T_4 bacteriophage virus mutations is described, and it is shown that all mutations in this phage are either deletions of varying length, nucleotide base changes, or addition or loss of a base. When either an addition or loss of a base occurs the remainder of the code becomes a nonsense code and the combination is non-functional.

Molecular genetics shows the DNA code to be a marvelously complex one. Surely in studying it we are coming close to understanding how God is daily at work maintaining and preserving all creatures.

For many years mutations, or suddenly appearing changes in either the appearance or behavior of individual organisms, have been considered the material basis of evolution. However, as more is learned about the exact nature of mutations, the less likely do they seem as building blocks for the origin of even varieties, let alone species of plants. Indeed, cyto-genetic research, and especially molecular genetics, has revealed an ever-increasing complexity of the physical basis of inheritance called the "gene."

First Level of Investigation

The study of mutations has involved probably three levels of investigation. First, after their discovery by such pioneers as Hugo DeVries, there was the painstaking work of T. Hunt Morgan and his associates, Calvin Bridges and A. H. Sturtevant. These men patiently accumulated information on the naturally occurring changes, or mutations, in eye color, wing form, eye structure, bristle arrangement and numerous other features of the fruit fly, *Drosophila melanogaster.*

Careful intercrosses and back-crosses showed that these mutations could be grouped into four linkage groups corresponding to the four chromosomes of the species. Within each chromosome the mutant genes were located serially, like beads on a string. The order of their sequence was determined by crossing-over studies; those far apart showing much recombination, while those close together, very little. As a result "chromosome mapping" could be done with fair precision, though odd "clumping" of genes in certain areas remained puzzling. Similar detailed chromosome maps were made in corn, tomatoes, flour beetles, and various grains, such as wheat.

Meanwhile the *process* of mutation was greatly speeded up by X-ray irradiation of the fruit fly. Muller first made this discovery in 1928. Here was a way by which biologists could, in a few years, obtain more mutations than Morgan and his associates found in a lifetime of patient observation. Thus quantitative studies as to the percentage of harmful vs. neutral, or possibly advantageous mutations, could easily be made.

Here was the first disappointment for evolution-minded biologists, for most mutations found

were harmful. In fact only about one in a thousand seemed to be even neutral or showed **slight** advantage under laboratoy methods of nutrient agar culture. Unfortunately X-rays did not prove very effective as regards inducing variations in plants though some success was obtained by pollen irradiation in such plants as corn.

Here again most mutations were semi-sterile types and many proved to be the result of translocation so that portions of chromosomes formerly separate were now attached, and reciprocally, portions previously in one chromosome were transfered to another. These recriprocal translocations were in fact quite common and may schematically be represented as follows:

Chromosome 1	**Chromosome 2**
Translocated Chromosome 1	**Translocated Chromosome 2**

There was considerable enthusiasm for a while that translocated chromosomes might explain the origin of new chromosomal arrangements, but soon it was found that all were lethal when homozygous.

The era from 1920 to 1945 might well be termed the period of great discovery and freedom to speculate that biologists were finding the "real" physical basis of evolution. Mutations were considered by many biologists as really *new* entities, useful as building blocks so to speak by the process of natural selection.

Then came mathematical treatises, by such masters at the art, as J. B. S. Haldane, R. A. Fischer, and Sewell Wright. They argued most convincingly that even though only one in a thousand mutations were advantageous to the extent of even a 1% advantage, these would slowly accumulate under the pressure of natural selection in a population and lead to evolutionary change.

Thus Patau showed that a mutation with a 1% advantage would increase according to the pattern shown in Figure 1. Increase from .01% to .1% of the population would occur only after 900,230 generations in a large population. Though millions of years would be needed to effect the transformation of the small five-toed, dog-sized Eohippus to the modern large one-toed horse, still geologists claimed abundant time was available, so all seemed well with the general theory.

Second Level of Investigation

Then came what might be called the second level of investigation. Population geneticists decided to study the actual way in which mutations did or did not accumulate under actual

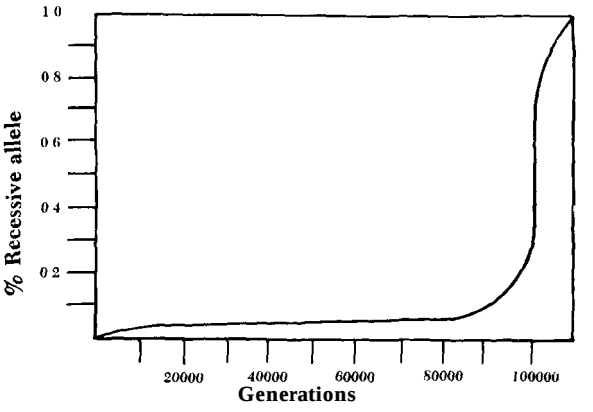


Figure 1. Showing very slow increase of a recessive mutation with advantage of 1 in 1000.

natural, field conditions. And parallel to these intensive studies of other means of inducing mutations were conducted which involved use of gamma radiation, neutron radiation, and various mutagenic chemicals.

Thus, as I reported in the 1965 *Creation Research Society Annual*,[1] neutron radiation of axillary leaf buds, or "budding eyes" of roses, was a highly effective way of obtaining mutations. In fact more mutations were obtained by the radiation of 50 rose "budding eyes" than one could find in a field of a million rose plants in a whole lifetime of patient searching for "sports."

Here was a splendid tool also for measuring the vigor and viability of mutations. For, as a measuring stick so to speak for calibration, we had the original unvarying variety easily propagated by budding, Accordingly, by rebudding the mutant forms *at the same time* as the original variety, and growing under comparable hothouse conditions, accurate comparison as to vigor, pollen fertility, and other characteristics could be made.

An interesting feature of this work is as follows: although some mutations showed useful horticultural variation, such as an increase in petal number, or loss of the unpopular magenta coloration, **ALL** without exception were weaker than the variety originally radiated. This was true even with the remarkably vigorous variety, Queen Elizabeth. Some fairly vigorous and interesting coral and white mutations were obtained, but these failed commercially since they were not vigorous enough under varying garden conditions.

Similar results were obtained by other workers using gamma radiation, and it is now quite clear that mutations in plants are usually significantly weaker, or have a reduced fertility, in terms of either the percentage of good pollen, or number of seeds produced per plant. Arti-

ficial induction of mutations is useful horticulturally because sometimes a mutation may show some much-needed commercially desirable characteristic. Thus, even though somewhat more difficult to grow or giving less seed, such a variety is worth using. This is particularly true of ornamental plants. Some of the neutron-induced rose variations may prove useful if the original variety is super-vigorous but lacks petal number. A mutation from it with enough petals to be commercially desirable would be worth growing even though less vigorous than certain overly vigorous types such as Queen Elizabeth.

In recent years, enthusiasm for demonstrating evolution by a study of induced mutations has about died out, since clear-cut cases of obviously advantageous mutations simply do not occur.

Meanwhile such population geneticists as Band[2] were showing what natural selection can and cannot do. Her work was with fluctuations in naturally occurring out-door populations of the fruit fly, *D. melanogaster,* so carefully studied under laboratory conditions from 1947 until 1962. One of her most remarkable conclusions was that natural selection does not increase the most viable or best true-breeding lines or homozygotes in natural populations!

Most pertinent were observations made following the unusually severe winter of 1960-61 at Amherst, Massachusetts. September temperatures were the highest on record and the samples were collected then. In 1962 collections were made during the driest season on record. The results of genetic analysis of variability and viability in 1961 and 1962 were compared to the more normal season of 1960 and the earlier ones of 1947-49. Her conclusions follow:

1) Natural selection is highly efficient in maintaining population fitness during stress as in the summer of 1962. The effects are shown only in the heterozygotes.

2) Stabilizing selection has led to the retention of most components of genetic diversity.

3) There is no evidence of improvement in viability of the homozygotes (those showing the mutation and breeding true for it.)

4) No decrease in genetic load was shown. This is because most load components (recessive mutations) remain concealed in the random heterozygotes.

5) Hence joint effects of directional selection and stabilization are directed to the interaction of genes and gene complexes in the heterozygous condition.

6) A slight reduction in total genetic diversity resulted from stress conditions.

Band does not stress the most interesting conclusion: namely, that there is no evidence that selection has been primarily directed to the elimination of harmful variations or mutants. Neither do such variants appear to reduce the viability of the heterozygote. Her Figure 1 is fascinating in that it shows no improvement in average viability of the homozygotes mutations, or any reduction in the magnitude of the genetic load.

From the viewpoint of evolving new characteristics these conclusions are indeed pertinent. The only source of new and distinctive features leading possibly to species formation are mutations. These must gradually be accumulated in true breeding or homozygous conditions, since of course species and even varieties differ from each other in various traits which are *constant.*

Yet Band's research shows that even the most viable homozygotes do not increase in number. Furthermore *no improvement* in their viability occurs. Since even drastic mutations show no harmful effect, if recessive in the heterozygcus condition, there simply is no mechanism for eliminating them. Now the ratio of "harmful" to "useful" mutations is at least 1000 to 1. Quite obviously, if a species really did evolve by natural selection, the genetic load of drastic or harmful mutations would become so high in a few hundred generations as to result in all offspring having some defect, because of chance mating of identical genotypes and resulting homozygosity. The fortunate fact that this is not yet true, in the human race or in most plant and animal species, argues strongly for the special creation of the species unit, and especially for its existence for a *relatively short time* instead of hundreds of thousands or millions of years.

Catastrophic Selection

With the discovery, that strains of bacteria resistant to penicillin, aureomycin or chloromycetin always showed up, when these drugs were used to effect cures of various diseases, great enthusiasm was aroused for a while among evolution-minded biologists. Here at last was "proof" that beneficial mutations really did occur.

But enthusiasm was short lived, for it soon became clear that these mutations did not arise as a result of exposure to penicillin. Rather they seem to occur at a *constant rate.* Associated with the resistance, there always is a decrease in viability under *normal* conditions. Accordingly, under normal conditions, they are soon "swamped out;" and, either are completely eliminated, or are carried along as heterozygotes in a very small number of individual bacteria. Now most bacterial cells appear to be haploid, but there is increasing evidence that sexuality does

occur; hence some cells are, for a time at least, diploid; hence heterozygosity does occur even in bacteria.

When a strain is exposed to antibiotics, either the mutation rate for these otherwise defective resistant mutations is so high that sooner or later one occurs, or an already established one is given the starting advantage of having no normal competitors. Soon the entire population is of the resistant type, and new medication is necessary. However, as soon as treatment is relaxed, the normal type bacteria take over, and the resistant strain is either eliminated or reduced to a minute fraction of a percentage of the population.

The story has been remarkably well presented as regards the housefly in a recent issue of *California Agriculture* in an article entitled "Housefly Resistance To Insecticides."[3] The conclusions on the housefly parallel those based upon studies of bacteria. Thus, the article 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 only serve as selective agents, eliminating the more susceptible insects and enabling the more tolerant survivors to increase and fill the void created by destruction of susceptible individuals.

There are several fascinating observations:

1) Resistance to DDT and dieldrin continued at a high level *in an area where these sprays were used,* in spite of the flies not having been sprayed with either chemical for about ten years. In other words, once established, resistant strains maintained themselves without selection pressure.

2) 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 the 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 species even in a given area such as Blythe, where this observation was made. Surely flies in the nearby agricultural area became resistant from 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 most exposed areas,* resistant to the chemicals used.

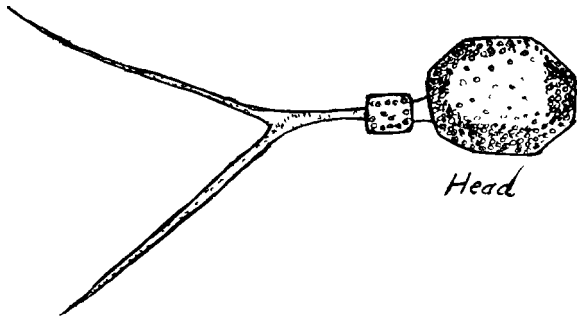


Figure 2. Diagrammatic representation of T-2 Bacteriophage magnified 500,000 diameters. Weighs 100 million times the hydrogen atom. Head has 1 chromosome with 200,000 base pairs (After electron micrograph, Benzer, p. 71.)

Third Level of Investigation

An explanation of just why most mutations were harmful, or once established, tended to maintain themselves at various percentages of the total natural population without further selection pressure. was so far wanting. With the advent of molecular genetics the third level of understanding has now been reached.

Thus Seymour Benzer[4] has found that the T_4 bacteriophage, which infects the colon bacillus. is a most useful organism for mapping in detail the molecular limits of gene structure. In a 20 minute experiment by use of a single test tube, a quantity of genetic data can be obtained, which would require the entire human population of the earth, if such a complex organism were used for study!

Phages are virus organisms characterized by a hexagonal-looking head, and a complex tail by which they attach themselves to the bacillus wall. (Figure 2) Within the head is a long-chain molecule of DNA having a weight of about 100 millions times that of hydrogen. After attachment, the DNA alone moves into the bacillus cell and takes over reorganization of the cell machinery to manufacture 100 or so copies of a complete virus and the bacterial cell then bursts open liberating these virus organisms.

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

Now "typographical" errors may occur in the replication of the DNA molecule. Transpositions, deletions, additions, or inversions may occur. As Seymour Benzer says, "In a daily

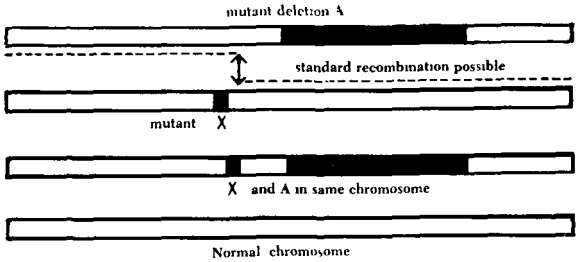


Figure 3. Showing how deletions are used to test location of mutants.

newspaper the result is often humorous. In the DNA of living organisms, typographical errors are *never* funny and are often fatal." (Emphasis added)

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 very processes of cellular activity as well.

One group of mutants called rH mutants can be identified quite easily by the appearance of the plaques or 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 plaques are hereditary characteristics of the phage that can be easily identified and scored. A plaque produced in several hours will contain about 10 million phage particles, the progeny of a single phage particle.

Now the T_4 phage can produce plaques on either host strain B or K. This standard form gives rise to rH mutants easily recognizable by a distinctive plaque on B cultures. But these mutants cannot form plaques on bacterial strain K. This is the "key" to the whole mapping technique used by Benzer; for an rH mutant can grow on bacterial strain K, if the cell is simultaneously infected with a particle of the standard type. 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.

As mentioned before, various different-appearing plaques or mutants arise spontaneously in this area. These may be crossed with each other by adding each of them to a liquid culture of B cells. This gives an opportunity for the progeny to recombine portions of genetic information from either parent.

If the two mutants resulted from typographical errors in different parts of the DNA molecule, some individuals of the standard type will be regenerated. A sort of "crossing-over" occurs. (See Figure 3) These reconstructed standards will produce plaques on the K strain, whereas the original mutants cannot. In this way one can detect a single recombination among billions of offspring. This allows the resolution of two rH mutants that are only *one base apart* in the DNA molecular chain.

The exact mechanism of recombination is not known. However, it seems that two defective DNA molecules may actually break apart to form one non-defective molecule which then is replicated; or, in the course of replication, there may be "copy choice," such that only good portions of the two mutant molecules are "copied." This appears to me as granting quite a remarkable power of selectivity to some "curative" agency in the T_4 phage cell.

At any rate, the results of a long and elaborate study of hundreds of non-reverting rH mutants shows that **all** can be represented as containing deletions of one size or another in a single linear structure. By contrast, the rH mutants discussed above behave as if their alterations were localized at single points. By testing against the non-reverting segments 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. (Figure 4)

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 segmental 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 in this small area, about 250 have been located, and only a hundred or so remain to be found. All are defective. Furthermore, certain chemicals, such as 5-bromouracil, increase the mutation rate at certain sites by a factor of 10,000 or more, yet affect no change at other sites. All of these also are defective changes.

Where then are the "good" mutations, needed for evolutionary progression?

The reason for this state of affairs is clearly shown in a paper by F. H. C. Crick[5] in the *Scientific American* for October of 1962. 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 at most a small group of bases, and are not merely the result of altering one of them. Such addition can be produced at random by compounds called acridines. Just how this chemical works is not fully understood. However, since the resulting changes can be combined or

broken up, there seems little doubt as to the fact that they are additions or deletions.

As has been explained by Duane T. Gish,[6] the simplest code by which 20 amino acids could be specified involves at least three nucleotide pairs, or a triplet of "letters" such as ATT, GCA, TCG, ACC. (A-adenine, C-cytosine, T-thymine, and G-guanine) The "message" evidently begins at a fixed point at one end of the gene, and is read three bases at a time. Then, if for some reason the reading starts at the wrong point, the message would fall into the wrong sets of three, and so would be incorrect. For each *correct* reading of the code there are two incorrect ones.

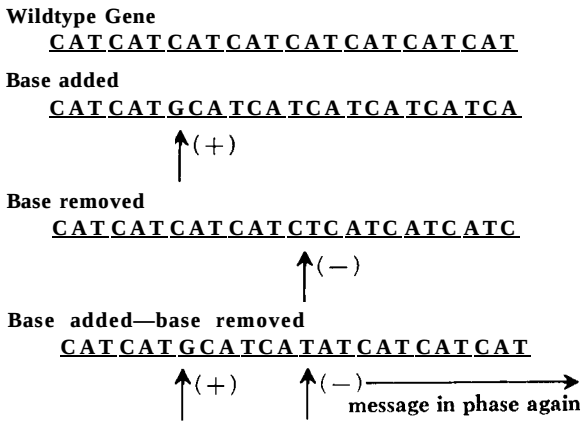
That is why the addition or deletion of a base in most parts of the gene makes it completely non-functional. The reading from that point onward would be totally wrong. Experimentally, this meant that if an additional base of plus mutation is combined with a plus, the combination is non-functional.

Likewise a minus with a minus is non-functional. But, if a plus is combined with a minus close to it, 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 was reached. Then, the message would not make "sense" until the location of the minus mutation or missing base is reached, after which the message would come back into phase or make "sense" again.

In other words, the function of the rH part of the B gene does not seem to be important. Accordingly, the message can be *"wrong"* for a short distance, and still be functional. But, if the distance is very long between the plus and minus mutations, the combination will not function. This is shown in Figure 4, adapted from Crick's paper.

Now, if only a sequence of three bases is needed to specify an amino acid, 64 could be specified instead of the actual 20 available. Off hand, it would seem that most "messages'" are nonsense messages or triplets. However, the experimental results show that most of the 64 possible triplets, or codons, are not nonsense, but actually stand for amino-acids. Hence probably more than one codon can "call" for the same amino acid.

The picture emerging from the work of molecular geneticists is a marvelously complex code which will stand mighty little in the way of alteration, either addition, or subtraction, or change of *any* of the nucleotide bases. Only because the rH region is *relatively* unimportant in function was it possible to accumulate the large number of mutations, making possible the detailed



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 4. Showing effect of mutations.

analysis of this rather minute portion of the T_4 phage DNA molecule. Now evidently, most portions of this molecule code message is so important, that even a *short* portion out of phase causes a completely non-functional "message," hence mutations do not survive.

The virus organism has only one chromosome; yet, "higher" animals, including man and, of course, all of the plants except algae, have many chromosomes—each one made up of organized protein and DNA molecules. How did this organization come into being?

The only solution so far offered by evolution-minded molecular geneticists is a sort of molecular level "polyploidy." They picture an organism such as a bacterium, which has a single circular chromosome, as giving rise to one with two chromosomes. Then, presumably, mutations could accumulate in the "extra" chromosome, and be shielded by the normal genes of the original one. But, sooner or later, sexual union or conjugation of two bacteria would occur. Then, some of the resulting descendants would have only a pair of the "new" chromosomes, and no original normal genes to shield them from the possibility of having a lack of balance in the plus or minus mutations, which occurred during the time before conjugation.

Surely, it is stretching credulity a bit to picture these "new" chromosome pairs as having such a finely balanced set of plus and minus mutants as to have "correct'" messages for all needed functions. In fact, it is difficult to see how any really new functions, such as the change from single celled organisms like bacteria to even the simplest multicellular green algae, such as a *Pleurococcus,* could ever come about by accum-

ulation of such defects as are so far reported by molecular geneticists.

It is true that the very nature of such experiments, as those of Benzer, where the K strain is used to reveal recombination, would tend to concentrate attention on defective changes. Still, since these are picked up as changes in appearance of plaques on the B strain, *some* should be of a positive nature and grow on the K strain better than the standard type. Such seem never to have been found or at least remain unreported.

From the creation viewpoint, we could of course expect the DNA system to be a marvelously intricate one. Since designed to accomplish very complex tasks even in the "simplest" organism such at a T_4 phage virus, it obviously could stand little in the way of tinkering. In fact, in light of the picture of just how DNA, RNA, the ribosomes, and the cytoplasm interact to form the needed proteins, we cannot but marvel at the complexity of all these reactions taking place at one time in a single cell.

Surely, the ingenuity of man is taxed to find ways of experimentally solving the exact way in which even a "simple" type of phage operates. Should we not then be filled with a feeling of reverent awe at the glory of God's handiwork as shown by this revelation of the complex way in which His created organisms carry on, their tasks? Truly the calling of a molecular biologist is a great one. Let us hope, that some of our young creation minded students approach this field, realizing that here they are coming close to seeing God at work as He daily maintains and preserves all creatures.

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IS DNA ONLY A MATERIAL CAUSE?

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By means of philosophical considerations and, secondly, through specific examination of experimental facts, the author inestigates the notion that DNA is "the secret of life."

An objection is raised that use of the word "code" in references to DNA involves nothing more than a metaphor. This and other objections are studied regarding DNA as a material, efficient, and formal cause. Objection is raised against the idea that memory is the encoding of experiences in DNA.

Examination of experimental data brings out denial of the normal expectation that complicated organisms would have larger amounts of DNA than less complex forms. Facts indicate that DNA is influenced by environment as well as heredity.

Comparisons are presented between results of ***in vitro*** *and* ***in vivo*** *experiments involving DNA.*

The author concludes from his theoretical arguments and from experimental evidence that DNA is not the whole cause of life and heredity. DNA is a material cause, but the author asserts there still must be a formal cause.

The one thing that most distinguishes living beings is their ability to reproduce themselves. In so doing, they are, of course carrying out God's command to "Be fruitful, and multiply. . .," (Genesis 1:22).

It is true, perhaps, as has sometimes been remarked, that things which are not living, for instance crystals under suitable circumstances, may "grow." Be that as it may, certainly the things which are not living do not show the same striving to reproduce themselves; if the crystals ever received a commandment to multiply they have not yet done much about it.

A second difference is that the living things are alike "after their kind" (Genesis 1:24); much more so than those that are not living. A snowflake, for instance, is a common crystal, or collection of crystals. Whether or not it be true, as is so often said, that no two snowflakes are alike, certainly there is much variety among