# Mutations and Evolution

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**ABSTRACT**

*Genes, which are the carriers of heredity, sometimes undergo non-directed changes that are expressed in the hereditary make-up of the offspring, and are also passed on to future generations. These unrepaired changes known as **mutations**, from the Latin for **changes**, are also relatively rare, occurring only once in thousands of DNA divisions. Orthodox evolution teaches that a continuing series of multi-trillions of small inheritable mutations gradually developed the estimated more than two million species of organisms alive today from simple organic molecules. For natural selection to select, differences must exist to select from — and these differences are ultimately provided only by mutations. The evidence reviewed indicates that mutations cannot provide the raw material for natural selection, and thus evolutionary naturalism has no viable mechanism.*

## INTRODUCTION

Mutations, as used in this paper, are defined as random changes in the DNA base pairs that are passed on to the plant or animal's offspring. It includes the addition or loss of DNA bases, or a substitution of correct bases with other ones. Although the vast majority of mutations are acknowledged to be negative or harmless, evolutionary naturalists conclude that enough rare positive mutations must have occurred in the past to have evolved all forms of life. They reason that this must be true because life exists, and mutations are the most viable naturalistic explanation for it.[1]

Because only natural explanations are acceptable in orthodox science, the best explanation in this framework, even if less than satisfactory, must be accepted until a superior naturalistic explanation replaces it. As Gamlin and Vines note,

> *'the original source of all variation for evolution to select from is mutation '* and '*mutations are essentially random in nature and most are either neutral or harmful in their effects'.*[2]

In short, mutations are life's creator, the raw material with which natural selection has fashioned the entire living world. The mutation concept as the ultimate source of evolution was first proposed in detail by Hugo DeVries[3] and has been extensively researched ever since.

## THE OCCURRENCE OF MUTATIONS

In the nucleus of each eukaryote cell exists the genetic material packaged in chromosomes (46 in humans). In organisms with a sexual cycle, both the sperm and the egg cells have only half this number — 23 in humans — called the haploid number. When a sperm cell unites with an egg cell, the resultant fertilized egg called a **zygote** becomes a new 'individual' which again has the diploid chromosome number. The net effect of this process is that approximately half of the new organisms' genes come from the mother, and half from the father. Characteristics are in this way inherited from both parents.

In each chromosome exists the units of heredity, the genes — estimated up to as many as 200,000 in humans. Over 3 billion base pairs form the entire gene code. On average, mammals have 5,000-8,000 base pairs per gene. Much of the work in genetics is done on bacteria, organisms which are often called simple, but which contain about 5,000 genes or 4.7 million base pairs. They are relatively simple compared to humans, but still enormously complex.

The number of harmful mutations in a human is estimated

by Mader to be about one out of 100,000 genes.[4] Herbert *et al.* estimate the rate is four to eight per human.[5] Some genes, though, are more prone to mutate than others, and many produce well-known deleterious or lethal effects. Given this data, and an estimated 200,000 genes per person,[6] the average person would carry only two harmful genetic mutations.[7] This, of course, does not include the somatic mutations, those that occur in individual body cells not involved in sexual reproduction. Occasionally, when doing DNA fingerprints (an evaluation of sections of DNA known to vary greatly), one uncovers a spontaneous mutation that occurred during embryogenesis that creates a band in the child's fingerprint that has no match in parental DNA.[8] This change in the child's DNA could be due to gene shuffling mechanisms discussed below, or due to a mutation in one of the parent's gametes.

One reason the number is so low is because several repair mechanisms lower enormously the mutation rate as expressed in the genes. After repair, an estimated rate of $10^{-8}$ to $10^{-5}$ per gene per generation is common in sexually reproducing organisms.[9] For populations, the total number can be high — in the United States with 250 million people the total equals 5 million to 5 billion new mutations in this population. Most of these, though, cause no problems or are for known diseases, and many occur over and over. Some are so commonly seen that scientists have names for them, often whimsical names such as *stuck, radish* and *shot-full-of-holes.* Few if any of these mutations can provide new material for evolution to select from. One of the most critical arguments against the mutation theory is the conclusion reached by molecular biologist Ray White that at most *'on average, only one in 500 base pairs will differ from person to person.* ' For exons (protein-coding DNA) humans are over 99.99 per cent identical.[10] This means that during the 1 million years speculated to have elapsed since our common ancestor evolved, virtually no changes in the gene pool have occurred.

Mutations are now divided into two types for convenience — **induced**, those due to a known cause, and **spontaneous**, those due to a so-far unknown cause or random event. Most mutations are caused by physical or chemical assaults on the DNA molecule. A chemical or physical factor that can cause mutations is called a **mutagen**. These include primarily chemicals, viruses, and ionizing radiation such as X-rays and cosmic rays. Mutations of somatic cells can cause cancer, and those that occur in the germ cells can cause genetic disease in the offspring. Many agents are both mutagenic and carcinogenic, including arsenic, asbestos, some chromium compounds, benzidine, vinyl chloride, thorium dioxide, mustard gas and melphalan. Another reason for mutations is:

> '. . . *because of the speed of replication (50 to 500 nucleotides per second) and partly because of*

**GLOSSARY OF TERMS**

**Bacteriophage** — an organism that infects bacteria. Although the relationship is not fully understood, the existence of bacterial phages has been critical in DNA technology, and they also serve a major function in distributing genes between different bacteria, which confers resistance, allowing bacteria to survive.

**DNA** — stands for deoxyribonucleic acid which consists of four chemical bases, a sugar backbone (deoxyribose) and phosphorous bonds. The arrangement of the bases contains the information necessary to produce proteins.

**Mutation** — in this paper we are referring to changes in the DNA base sequence through random events which result in base substitution, base addition, or base deletion. In this paper we are not referring to processes producing genetic variety through orderly and systematic means such as sexual reproduction with crossing over.

**DNA fingerprinting** — a technique used to distinguish DNA differences in the population analogous to identifying persons by fingerprints. A special enzyme is used to cut the DNA into short pieces. The cuts occur at specific base sequences such that different DNA strands will be cut into different lengths of short pieces, or fragments. Then a technique called gel electrophoresis separates the DNA fragments according to size. The resultant differences form a unique set of bands. The patterns of these bands can be used to distinguish persons, identify disease, etc.

**RNA** — an abbreviation for ribonucleolic acid which is a compound very similar to DNA except its sugar backbone is slightly different and RNA uses uracil instead of thymine. Also RNA is typically single stranded. The basic types of RNA are messenger RNA, transfer RNA, and ribosomal RNA.

**mRNA** — the cell makes a copy of a required DNA sequence, called messenger RNA or mRNA which then travels outside of the nucleus into the cytoplasm, where it hooks up with ribosomes which then manufacture the protein coded by the mRNA.

**tRNA** — in order to manufacture protein, the proper amino acids must be lined up on the mRNA strand. The tRNA, meaning transfer RNA, is designed to achieve this function. Each amino acid has its own type of tRNA which lines up the amino acid next to the specific 3-base code for that amino acid on the mRNA.

*spontaneous chemical flip-flops in the bases, DNA polymerase occasionally incorporates incorrectly matched bases, perhaps one mistake for every 10,000 base pairs. In mammalian cells, however, the completed DNA strands contain only about one mistake for every billion base pairs.*'[11]

In this way, some mutations, such as that which causes hemophilia disease, are assumed to be caused not by mutagens, but by a spontaneous mutation that occurs due to 'chance', actually unknown factors — and are usually, but not necessarily, obtained through heredity. A famous case of hemophilia involved Queen Victoria, a carrier who passed it on to many of the royal families of Europe including Russia. Since no known record of hemophilia exists in her ancestors, it is assumed that her genes, or those of her immediate ancestors, were the source of the mutation. Once this mutation becomes part of the gene pool, it is usually passed along by female carriers, and only males can develop the disease. If a mother is a carrier, half of her daughters will also be carriers and half of her sons will develop hemophilia, on average.

Many mutations produce barely perceptible changes — such as causing eye development in humans that is a millimetre or so farther apart than normal. Some cause a defective enzyme that is relatively unimportant, or that can be dealt with by diet or lifestyle changes. Leu and Dill claim that many biomolecules seem to be relatively insensitive to single substitutions, especially those in the amino acid chain ends or terminal portion.[12] This is one reason why most mutations are neutral and do not result in phenotypic changes. Protection from these changes could also be due to over design or because of a built-in protection mechanism. In addition, a mutation in a body cell of an adult is often not a problem because most cells around it are normal. Although this situation may result in cancer or another problem, it is not a means of providing variety for evolution because these changes are not inherited. Nor are the many lethal ones which often cause spontaneous abortions, estimated to be as many as one third of all human conceptions.

A major difficulty is determining whether or not a biological innovation is actually due to a mutation, or is a simple variation caused by normal gene shuffling, crossing over or other complex mechanisms designed to produce variety. For example, hairiness in tomatoes is often regarded as a mutation, but it may be a normal trait variation or be due to gene shuffling. It cannot easily be determined if an innovation is from a mutation or due to a mechanism designed to produce variety. This is emphasized in the discovery of genes that produce two or more different proteins by alternative splicing of an mRNA transcript. This allows one gene to produce two or more forms of a protein for different stages of development or for use in different cell types.[13]

An example of what may be a control mechanism that is often labelled a mutation is called an **amber mutant**. Bacteriophage (bacterial viruses) with one will grow only in what is called a 'permissive host', one which contains a 'suppressor' that can bypass the 'mutation'. The 'mutant' is a single-base 'change', that alters the amino acid coding triplet to become UAG, a stop codon. It is read as such under normal 'non-permissive' conditions, terminating the protein coding before it is completed. A so-called permissive host bacterium said to 'suppress' the mutation is actually compatible to it because it has a species of tRNA which translates UAG as an amino acid. Thus it does not function as a stop codon. The specific amino acid coded depends upon the class of permissive host in which the bacteriophage resides. Thus, a structurally unique system in the host allows the bacteriophage to produce the needed protein and serves as a means of controlling the phage.[14]

## WHY MANY MUTATIONS ARE NEUTRAL

Mutations often have no effect on the phenotype for many reasons. Some genes exist in multiple copies, and for these cases if a mutation occurs on one of these genes, no discernible change may occur in the organism. The redundant genetic codon system allows the code to change, and yet the proper amino acid can still be produced. This is because several codes exist for many amino acids. For example, the amino acid leucine is coded by UUA, UUG, CUU, CUC, CUA and CUG, and the same amino acid will be coded if the code is CUU or CUC, a change which results if the last U is replaced by a C. Consequently, a large number of mutations will have no effect on the protein coded (see Figure 1). This system is said to be a **degenerate code**, meaning it can 'degenerate' from the original and still code the correct amino acid sequence. Of course, in spite of a degenerate code, mutations will often produce defective proteins — and most mutations usually result in defective polypeptides. A mutation in a repressor gene could allow the excess production of a protein to the point of lethality. A defective protein could be poisonous to cellular systems. Since humans have two sets of chromosomes, a mutation will usually affect one gene, consequently the 'insurance gene', the second one, will still produce the correct functioning protein. The person may have less of the normal protein, but can often function. They are thus said to be 'recessive'. If dominant, loss of one gene will always cause major problems.

For this reason, typically only a double mutant gene will actually affect the health of the organism — as, for example, with sickle cell disease. The major exceptions are the many sex-linked genes that are on the X chromosome, which have no corresponding allele on the Y. Consequently, recessive genes such as colour blindness and hemophilia are often expressed in males, but rarely in females. Other exceptions include alleles that are **co-dominant**, meaning that the heterozygote form results in two separate phenotype traits, such as blood groups. A homozygous $I^A$ will cause the blood cells to have only glycoprotein sequence A, which produces type A blood, and an $I^B$ homozygous will cause the person to have type B blood. The A and B refer to two types of glycoproteins attached to the red blood cells. One who is

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|---|---|---|
| **Amino acids:** (translation) | | -glycine-serine-leucine-proline-valine (protein) |
| | tRNAs { | □□□ □□□ □□□ □□□ □□□ |
| | | CCA AGA AAU GGA CAA (STOP) |
| **mRNA:** | | — GGU — UCU — UUA—CCU — GUU — UAA |
| (transcription) | | ⇧ ⇧ ⇧ ⇧ ⇧ ⇧ |
| **DNA:** | | — CCA — AGA — AAT —GGA — CAA — ATT— |

*The scheme for protein production from the information in the DNA. DNA has four nucleic acid bases, cytosine, guanine, adenine and thymine (C,G,A,T) arranged in sequence. The four bases pair up: cytosine with guanine and adenine with thymine to form the complementary strand of DNA to give the familiar double helix. Groups of three bases (triplets) code for the 20 amino acids which make up proteins, as well as a 'stop' signal. There are 64 possible combinations of three bases, so more than one triplet can code for the same amino acid. Enzymes unwind the DNA helix to allow copying of the required information from one strand to produce a strand of mRNA. In RNA the nucleic acid base uracil (U) replaces the thymine in DNA, so uracil pairs with the adenine in DNA during copying. Transfer RNA (tRNA) molecules attached to each of the amino acids each have complementary triplet codes to those on the mRNA. The mRNA is then 'read' by ribosomes which line up the tRNA molecules along the mRNA to produce the sequence of amino acids prescribed by the DNA sequence (see Figure 1). Other enzymes join the amino acids together. Only five of the twenty amino acids are shown.*

heterozygous ($I^A$ and $I^B$) would produce type AB blood, and if one has neither allele (called an I° gene) no sugar sequence will be made on the erythrocytes, and recessive type O blood results. No problems normally result from inheriting one blood type as opposed to another, but this is not always the case for other traits.

A primary concern in understanding mutations is the fact that other causes exist for defective genes aside from genetic code changes. Ultraviolet light can cause two thymine bases located adjacent to each other to bond, forming a **thymine dimer** which is not always properly corrected by repair enzymes. Numerous enzymes hover in and around the chromosome, like worker bees, which unwind, wind, repair, transcribe, replicate and replace defective parts of DNA. Many kinds of genetic mistakes can occur as a result of faulty enzymes or proteins due to diet or environmental factors. A phenomenon called **hybrid dysgenesis** results when an inherited combination of genes produces an interaction that causes or allows the development of harmful traits. Hybrid dysgenic fruit-flies produce abnormal eggs and sperm, and have other developmental and health problems.

In one study of hybrid dysgenic effects in *Drosophila melanogaster*, the researchers used two strains called M and P types. Crosses between P males and M females produced dysgenic offspring because the P type flies have a transposon in their chromosomes which functions to block the expression of certain genes in the M type fly. A transposon is a block of genes that can move to another location and, as a result, represses or activates genes near its new location. The control genes are usually kept in place by specific proteins — but in a P sperm and M egg zygote, a transposon in the fertilized egg is unaccompanied by the necessary suppressing proteins. These 'jumping genes' sometimes insert themselves at wrong sites in the chromosomes, consequently turning genes off or on and thereby causing developmental abnormalities.

One example of an incorrect use of the word mutations is to refer to changes caused by jumping genes, such as that which produces the variegated colours of maize. In the case considered here, researchers found that the P type flies contained transposons at **numerous** incorrect sites in the chromosomes in contrast to the M type which, with rare exceptions, had none. Other investigations have found that many of the 'frequent mutations' in the reproductive cells of dysgenic hybrids were due to the incorrect movement of the transposons to other parts of the DNA molecule, not a change in the base pairs.

Many so-called mutations are actually developmental abnormalities that are not caused directly by changes in the base codons in the DNA macromolecule. Errors in transcription not due to DNA

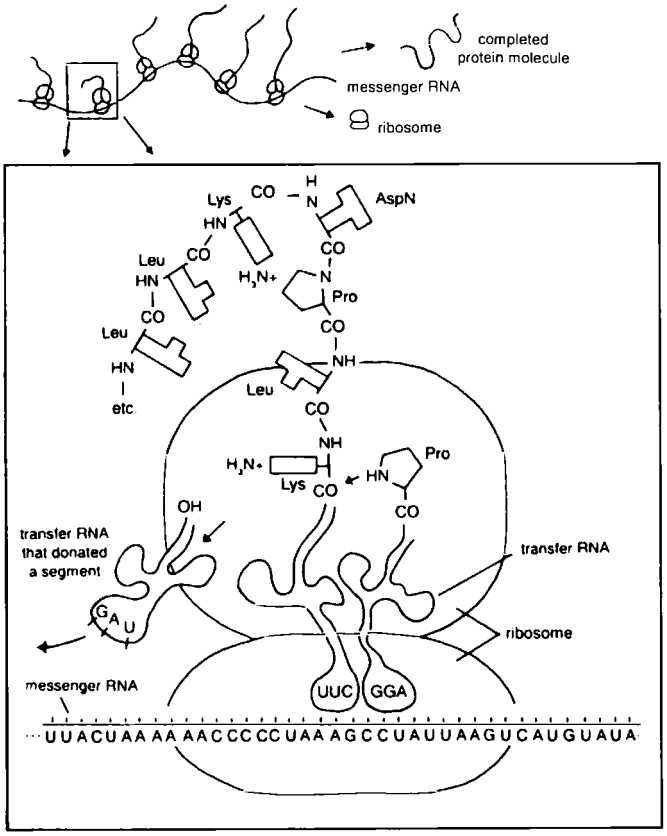


*Figure 1. The synthesis of protein by ribosomes using mRNA.*

mistakes because of faulty enzymes are examples. Poor diet, toxins and temperature changes during embryogenesis that produce abnormalities and other environmental influences are all important causes.

Other changes are due to chromosomal abnormalities. An example is tβhe incorrect splicing of the β-globin gene that occurs when the mRNA is assembled.[15] Yet another example is gross changes in the chromosome, as *Wolf-Hirschhorn syndrome* which is caused by a chromosome defect, namely a missing tip of the short arm of chromosome 4, which results in mental retardation and a misshapen skull.

## THE STUTTERING GENE, DYNAMIC MUTATIONS

**Dynamic mutations** are another example of a non-base pair change. An example of this mutation type is *Huntington's Chorea*, a rare hereditary neurological brain disease characterized by severe mental deterioration, speech disturbances, and involuntary movements due to the degeneration of the cerebral cortex and basal ganglia.[16] It develops in later life and affects as many as 1 in 10,000 persons in the United States. The cause is not a change in the gene spelling, but simply repeats of subsets of a gene in addition to the existing gene base pairs that exist in healthy persons. Located on chromosome 4, it is a dominant gene. In victims of the disease, 42 to 100 repeats of a block of three units of the normal DNA sequence occurs, and the more repeats the earlier Huntington's usually strikes and the worse the disease. In each new generation of males, the 'stuttering' (repeats) becomes longer so that the disease may strike victims as young as age 20. The genetic copies may be related to the fact that the brains of Huntington victims produce abnormal amounts of the excitotoxin quinolinic acid which overstimulates certain nerve cells, evidently in time causing their death. The same gene passed in the female produces both later and less severe stuttering. This lends evidence to the conclusion that the gene repetition problem is greater in cells that become sperm than those that become eggs.

Several dynamic mutations have been identified aside from Huntington's Chorea. These include Kennedy spinal and bulbar muscular atrophy, spinocerebellar ataxia, Fragilette's syndrome, and dentatorubral-pillidoluysian atrophy.[17] Dynamic mutations are of special interest because they indicate that even normal gene sequences, if repeated beyond a certain point, could code for a level of a needed protein that kills the cell. This may also be true of subunits of normal DNA sequences which evidently do not code for deleterious excess of protein, or are not the result of a classical mutation. The classic example is *Downs syndrome*.

Because this research is preliminary — the extra DNA sequence on the fragile X syndrome site was isolated only in 1991, and in other examples the dynamic mutations were only discovered in 1992 — few conclusions can be drawn about this type of mutation. They speculate that the control over the fidelity of the DNA replication in these cases may be lost, as occurs in the familial form of colon cancer, hereditary nonpolyposis colon carcinoma or HNPCC). The speculation about the cause of the repeats centres around the idea that '*the gene-replicating machinery somehow slips when it comes to a repeated region*' and copies are repeated, adding '*between one and five new copies of the repeat*'.[18] This indicates that, just as a few extra resistors in an electrical circuit may cause problems, additions which individually are not a problem can be deleterious when added to the DNA circuit. Further, they hint that all DNA, or most DNA, has a function, and that the addition of useless, non-coding DNA may have adverse consequences for the organism.

## THE RESEARCH ON *DROSOPHILA* AND MUTATIONS

Extensive experiments have been conducted on the effects of mutations, especially with the fruit-fly known as *Drosophila*. This tiny insect, whose favourite habitat is around rotten bananas and other fruits, is an ideal subject for this research. It is a short-lived prolific reproducer, and has large chromosomes which can easily be examined. In addition, it requires very little space, food or care to breed a few thousand fruit-flies for research. Most other potential research animals are far more expensive to feed, maintain and keep. A female *Drosophila* produces as many as 600 offspring in as little as 24 days, and consequently 90 *Drosophila* generations will be born and die in a mere three years. This equals more human generations than have lived since the birth of Christ!

Thomas Hunt Morgan first began to study *Drosophila* at Columbia University in 1909. In 1910 he bred two red-eyed flies from a long line of red-eyed ancestors and came up with white-eyed offspring — reported to be the first laboratory example of a non-lethal detrimental mutation — although some have concluded that the white-eyed characteristic he found was always part of the fly's gene pool. When researchers continually mate animals, they often eventually come up with a variety of 'new' characteristics because of the operation of the many systems designed to produce variety, such as genetic crossing over, that are built in every animal.

In 1926, Muller discovered that by exposing fruit-flies to high levels of radiation (such as X-rays or gamma rays from radioactive materials), the mutation rate in their offspring can be increased by as much as 150 times.[19] A conservative estimate is that Muller increased the level of mutations about twenty-fold over the normal rate, a discovery for which he was awarded the 1946 Nobel Prize in medicine and physiology. His work in this area '*convinced him that the vast majority* [of mutations] *were deleterious*' and consequently that exposure to radiation should be strictly controlled.[20] Morgan had, in total, bred about 900 consecutive generations of fruit-flies, the equivalent of 25,000 years of human reproduction. Even with directed artificial selection, he ended up with nothing more than deformed fruit-flies.

Wallace and Simmons explain that of the hundreds of specially constructed 'mutant' stocks that now exist as a result of this work, all are only variations, most or all would be undesirable in the wild, and all are still fruit-flies:

> *'At the University of Edinburgh, some flies (Drosophila melanogaster, the vinegar fly) from Kaduna, Africa, were introduced into such a cage in the early 1940s. During the late 1960s, some twenty-five years after the fly population was started, one could still obtain "Kaduna" flies from the geneticist at Edinburgh. To the best of our knowledge, the population still exists — now nearly fifty years old. The flies in a population cage live between twenty and thirty days. In such a cage, kept at 25°C, a fruit-fly generation is estimated to span two weeks, perhaps a bit longer. Thus, over 1200 generations of flies have lived in the Kaduna population cage (that many generations of humans, for whom a generation equals twenty-five years, would span about 30,000 years).'*[21]

## HUMANS AND MUTATIONS

Only amino acids are normally used as the primary building blocks of life. A group of three DNA bases called a codon can code for one amino acid. Since DNA uses four bases, there exist 64 ($4^3$) possible codons, more than enough to code for the common 20 amino acids. For a small peptide, given 20 different amino acids, $20^{20}$ ($>10^{26}$) kinds of polypeptides would be possible. This enormous level of variety is necessary to code the estimated 60,000 or more types of proteins needed for human life. The correct sequence is ordinarily of utmost importance for most of the proteins — but sometimes proteins with a few incorrectly placed amino acids can still function, although often not as well. An example is sickle cell anemia in which a mere single incorrect amino acid in 300 — the replacement of glutamine with valine — produces 'sickle cells' which do not function well under certain circumstances, causing anemia, severe pain and even strokes.[22] Often mutations prevent a functional enzyme from being produced, and consequently a necessary component or capacity is missing from the cell.

The development of research on radionucleotides has afforded scientists excellent opportunities to observe mutations. The mutations produced, though, have almost without exception proved disappointing to Darwinists. Those observed virtually always have no discernible positive effect or injure the animal — some estimate that over 99.99 per cent of all mutations are in this category — and most are recessive. Actually, as Gamlin and Vines note,

> *'the chance of a random change improving a highly specialized molecule like an enzyme is very small indeed.'*[23]

As Rust summarizes:

> *'Each of the newly emerged minimal functions* [from mutations] *must be capable of improvement by random mutations — up to the near-perfection usually found in present organisms. This* ***seems*** *to be more easily accomplished than the emergence of a new functionality, but it is not self-evident that it is possible.* ***Not even a single "positive" or adaptive mutation, in the sense of an improved function previously unavailable, has been documented in any organism.*** *Takeover of functions from other organisms, by means of* ***episomes, transduction genetic recombination, allele assortment and the like, cannot be counted as an emergence of a new or improved function*** *in the biosphere,* ***nor can regaining a function lost previously, or the display,*** *under stress, of a temporarily unused function.'*[24] (Emphasis mine.)

In spite of statements by evolutionists that

> *'it is of benefit for mutations to occur occasionally because variation is the raw material for the evolutionary process,'*[25]

no reputable scientist has claimed that radiation pollution or other means of increasing mutations can accelerate evolution. Almost 50 years ago a **Life** magazine article reported that the '*radiation from the explosion of an atomic bomb is a geneticist's nightmare*'.[26] Some geneticists, **Life** reported, believed that the mutations which occurred in the Japanese exposed to radiation from the Hiroshima and Nagasaki bombs '*may plague the human race for thousands of years*'.[27] Actually, the bombs have had far less mutational effects than first projected.[28] Current research on their long term effects has found that the damage was actually much less than expected partly because the highly effective human genetic repair process has negated much of the harm done. An example of the effectiveness of this repair process in normal cells is as follows:

> *'Three enzymatic processes carried out by a DNA polymerase complex are responsible for the high fidelity of DNA replication. First, the complex chooses a nucleotide triphosphate from the cellular pool that is complementary to a template nucleotide. Then the nucleotide triphosphate is converted to a nucleotide monophosphate that is aligned with the template nucleotide. Only if the fit is stable is the nucleotide incorporated into the daughter strand. If the fit is not stable, the nucleotide is restored to its triphosphate form and released. A mismatched nucleotide slips through this selection process only once per 100,000 base pairs. Such errors can still be caught, however, because the DNA polymerase complex also carries out a proofreading function. The mismatched nucleotide causes a pause in replication, and during this time, the mismatched nucleotide is excised from the daughter strand. After proofreading has occurred, the error rate is only one mistake per 10 million base pairs.'*[29]

Although mutations are claimed to be the 'prime power of evolution' which produced humans from a one-celled ancestor which appeared eons ago, mutations are universally feared rather than exploited as a potential means of improving at least some plants and animals. It would seem that humans

could improve at least **some** domestic animals by using artificial selection on animals that have been exposed to massive doses of radiation to cause mutations. Yet efforts in this direction have clearly failed, and new techniques such as intelligent use of recombinant DNA are now being used with much success.[30]

The results of radiation and its deadly genetic effects were first extensively studied at the University of Washington in the 1940s. Fish were used in these experiments because of cost concerns, and for the reason that they react to radiation in similar ways as do higher vertebrates, including humans. Mutations caused horribly deformed offspring of irradiated parent trout, producing the following summary:

> *'Five years of tests have shown that radiation produces no abnormalities that do not occasionally show up in nature. But irradiated parents produce a much higher percentage of malformed offspring —as high as 59% following an X-ray dosage of 1000 roentgens. No useful mutations have appeared, and none is anticipated. Biologist L. R. Donaldson, director of the study, explains, "So far as we know we're not getting any good characteristics. You can't add when you are subtracting. " '*[31]

This level of radiation and experiment duration has increased the number of mutations to the extent that his experiments have produced the 'equivalent' of at least half a million years of human evolution! In an equal period of time, many human ancestors (as well as modern humans) were theorized to have evolved. These experiments are not definitive, though, because the intensity of mutagenesis in these experiments is so high that an individual with a rare positive mutation would be invariably eliminated because they also carry a large load of harmful mutations. When the mutagenic intensity is low (as in nature) and the population extremely large, the positive mutations can in theory occur in isolation in an individual, and therefore may have an impact. Although these experiments cannot exactly mimic the natural environment, extensive research has since then supported these early conclusions. Thus, the concern expressed by Shapiro:

> *'For the past century or more, the human race has been exposed to an ever-increasing number of synthetic chemicals, the great majority of which have been prepared for the first time in the history of this planet. Since the 1940s, it has also been clear that more than a few chemicals can cause mutations, changes in the DNA text, by reacting with DNA.'*[32]

The evidence for the case in favour of mutations as the creator of the living world was eloquently summarized by Giertych in the following words:

> *'Mutations figure prominently in the evolution story. When in the early sixties I was starting breeding work on forest trees everyone was very excited about the potential of artificial mutations. In many places around the world special "cobalt bomb" centres were established to stimulate [higher] rates of mutations. What wonderful things we were expecting from increased variability by induced mutations. All of this work has long since been abandoned .... All we got were deformed freaks, absolutely useless in forestry. Maybe occasionally some oddity could be of ornamental value, but never able to live on its own in natural conditions . . . literature on mutations outside forestry quickly convinced me that the pattern is similar everywhere. Mutations are either neutral or detrimental. Positive ones if they do occur are too rare to be noticeable. Stability in nature is the rule. We have no proofs for evolution from mutation research.'*[33]

Many animals, notably larger mammals, produce relatively few offspring in their lifetime. Consequently, very few exist to produce many mutations as a source of variety, yet the number of extant species is now estimated in the multi-millions. Given this fact, if evolution could occur, it would be extremely slow — and millions of years would be needed for even minor species changes. Large-scale evolutionary changes, not small changes, are needed to produce a new species which would require major changes in the gene pool. Yet, the *Drosophila* research has given little cause for hope that mutations could have provided the necessary variations. Thousands of generations of high level mutation reproduction have caused the wings, legs, colour and body shape all to vary beyond their normal limits, but the insects are all still clearly fruit-flies.

Some conclude that only a flood of new mutations in a comparatively brief period of time is able to produce a 'new' family type. In other words, apes gave birth to humans in a relatively short time and neither has changed much since. This view, in its earliest form was called the **hopeful monster theory**, but today's related and refined version forms one possible mechanism within what is now called **punctuated equilibrium**, which is now widely accepted by evolutionists. The term 'monster' is used not in a pejorative sense: any offspring that possesses major developmental anomalies that causes it to be drastically different from its parents is called a monster. The term, which originally meant a divine portent or warning, is from the French word *mohere*, meaning 'to warn'. This view also suffers from the same problems as the gradualist's mutation theory — if small mutations cannot be shown to be beneficial, ones causing greater change are even less likely to confer positive functional changes. It also suffers from other problems, such as the source of suitable partners with whom the hopeful monster can breed.

The fact that humans and many lower organisms share many genetic code sequences, and some are virtually identical and yet they are alleged to be multi-millions of evolutionary years apart, is also problematic for evolution. Many of the changes are in codes that produce small but necessary differences between organisms, or those that make no difference. Such relatively small changes in the genes, assuming that evolution is valid, also would reveal the slow rate of change that mutations cause. And the finding that two genes are identical or very similar is put in perspective

when it is realized that many human genes have thousands of base pairs. For example, one of the larger genes, the human growth hormone gene, is 150,000 base pairs in length.[34] The gene defect that causes neurofibromatosis is a mistake in a series of small exons spread over at least a 200,000 base pair section on chromosome number 17.[35]

## THE EFFECTS OF MUTATIONS

To study the effects of mutations, researchers have now located in the relevant genes '*at least 60 different mutations*' causing cystic fibrosis — and thousands of human diseases exist which are believed to have a genetic basis, demonstrating the enormous number of possible mutations. Yet, not one of these known mistakes has been shown to be beneficial, except in extremely limited and unusual circumstances, such as sickle-cell anemia, a disease that affects about one out of 625 Blacks (0.0016%). Even these situations are rare and in most cases cause a problem only if homozygous.[36] Cystic fibrosis is a disease characterized by a faulty sweat and mucous gland mechanism which causes excess chloride loss in sweat and thick sticky mucus that tends to trap and hold bacteria in the bronchial tubes causing respiratory infections. In about 70 per cent of cystic fibrosis patients, the disease is caused by the loss of three base pairs resulting in a single amino acid —phenylalanine — missing from the protein that the gene produces.[37] A total of over 20 rare mutation types which can cause cystic fibrosis have now been located, all of which render the critical proteins non-functional.

The accumulation of mutations has caused the defective gene load in humans to gradually increase, until now an estimated 4,000 diseases exist which are caused by 'mutations'. Certain mutations are often found only in a specific population and can sometimes be traced back to their source. Sickle-cell anaemia has been traced to an African who lived in Sudan, East Africa, and from there spread '*along with slash-and-burn agriculture*'.[38] The farther back we go in history, therefore, we would expect to find fewer mutations in the human gene pool that cause diseases. This is clear evidence for de-evolution, because the farther we go back in history, the more perfect our genome. As the mutation load increases, more and more mutation-caused diseases enter the human gene pool. The evidence produced from tracing mutations back in time finds that, although some have repeatedly occurred, such as Duchenne's muscular dystrophy, many gene mutations evidently have occurred only once or a few times in history, such as Tay-Sachs disease (*amaurotic familial idiocy*) or sickle-cell anaemia.[39]

This conclusion also supports the observation that, the genetic repair system ensures that many mutations are impossible or extremely unlikely; primarily those genes that are not protected can express mutations, and it is these mutations that cause de-evolution. The major way that this problem can be dealt with is by use of genetic engineering to develop gene therapy methods to circumvent a mutation. For hemophilia, for example, we can inject patients with factor Vlll protein, the clotting mechanism that their blood lacks, or endeavour to carry working copies of the gene into the patient's genes by the use of retroviruses as vectors.[40]

## SUMMARY: ARE HUMANS ONLY GENES?

A major concern of many is that science has, by unlocking the gene code, reduced humans

> '*to little more than a string of four different chemical bases in various combinations and paramutations, and that this ultimate exercise in reductionism will undermine their moral and spiritual standing and their dignity.*'[41]

In response to this, a Case-Western University biomedical ethicist has noted that Beethoven's ninth symphony consists of only 12 notes, and this knowledge in no way reduces the grandeur of his works. The fact that something so beautiful can be made from something so simple should increase our wonder. He adds that likewise, as we have grown to understand the four base pair system that codes for life, it

> '*may increase our appreciation for the Creator of all life. After all, Beethoven had 12 notes to work with, but the Creator had only four*'.[42]

This huge number of possible gene sets — billions exist in humans, and multi-trillions of gene combinations are possible — has coded for more than 2 million species, and has also achieved an enormous level of trait variation within each species. By careful selection of parent stock, geneticists have bred an amazing variety of dogs, cattle, chickens, horses, and hundreds of other animals. But no historical evidence exists that mutations have produced a new useful gene, or a new order, or family type, or a major new biological innovation such as a wing. Recombination and other natural means of producing variety have evidently created all the 'new' characteristics observed, most all of which are variations of the existing genotype. This is not Darwinian evolution, but only the creation of new traits from novel combinations of genes that already exist in the family gene pool. Only qualities which already exist can be 'developed' or altered by selective breeding. The laws of heredity can produce a wide variety of traits, but dogs remain dogs, and humans remain humans.

Wide variations within the family can often be accomplished only by conscious intelligent selection of certain traits. Left to themselves, the specialized varieties that humans have bred from deliberate interbreeding of animals for a select trait will usually 'breed out' in the wild state. As a result, their progeny soon revert back to the original wild form. The controversial field of eugenics is actually an attempt to use selection to improve the human family by directing or speeding-up evolution (although it actually only changes the frequency of existing traits). The bridge between different major groups of animals has not been narrowed by breeding.

Much empirical support exists for the conclusion that if

mutations have any effect, they weaken and kill, and few, if any, are clearly beneficial. Although many varieties of life exist that can interbreed and produce virile offspring, gene mechanisms often de–stroy the varieties that stray too far, ensuring that each family continues to bring forth only '*after its kind*'. Mutations are not the answer to the question of what gave 'nature' the materials to select from in order to cause macro-evolution. The answer lies in what we vividly see everywhere in our physical world: purpose, design, economy, variety and wonder.

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## QUOTABLE QUOTE: Artificial Life

*'A-life may already have achieved this goal, according to the evolutionary biologist John Maynard Smith of the University of Sussex. Smith, who pioneered the use of mathematics in biology, took an early interest in work at the Santa Fe Institute and has twice spent a week visiting there. But he has concluded that artificial life is "basically a fact-free science". During his last visit, he recalls, "the only time a fact was mentioned* was *when I mentioned it, and that was considered to be in rather bad taste." '*

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