Why Mutations are Lethal to Darwinism

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Abstract

The primary means by which evolution is believed to occur are mutations, independent assortment of genes, gene shuffling and natural selection. Mutations are non-directed DNA changes that can be expressed in the offspring's phenotype and passed on to the organism's progeny. Unrepaired mutational genetic changes are relatively rare, however, occurring only about once per billion DNA bases. For natural selection to operate, there would need to be biological variants from which to select, and these differences must ultimately be provided only by mutations in the macroevolutionary scenario. A review of many types of mutations, however, indicates that they cannot provide the raw material necessary for natural selection for various reasons. Consequently, evolutionary naturalism still lacks a mechanism to produce new information and biological novelty.

Introduction

Modern Darwinists teach that a continuing series of small heritable genetic changes called mutations gradually produced by means of natural selection the estimated ten million species alive today from comparatively "simple" protocells. Mutations normally are defined as non-directed random changes in the DNA base pairs that are passed on to a plant or animal's offspring. *Genotype* (the information on the DNA) changes may or may not be expressed in the *phenotype* (the organism's physical characteristics). Mutations are extremely important to evolutionary naturalism because according to this world view the ultimate diversity of all of life forms depends on mutations favored by natural selection (Bergman, 1992). 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" (1991, p. 25). In short, modern Darwinism teaches that mutations are life's diversifying agents, the raw material from which natural selection has led to specialization throughout the entire biosphere. It is well to note, however, that although mutations can lead to heterogenicity at existing loci, the creation of gene loci themselves cannot be explained by mutations and is, to all intents and purposes, unknown.

The view that mutations are the ultimate source of evolution was first proposed in detail by Hugo DeVries and has been extensively studied ever since (DeVries, 1910). The topic of mutations is of major concern in medicine because mutations are a major cause of disease and therefore "a better understanding of the characteristics of deleterious mutations is ... imperative" (Peck and Eyre-Walker, 1997, p. 136).

For many years, creation scientists have been proclaiming the fact that mutations cannot serve as a mechanism for neo-Darwinian evolution because almost all expressed mutations are deleterious, and those that can be beneficial in unique situations virtually always render the animal less fit in the wild (Lammerts, 1971; Tinkle, 1967). Lammerts, in a study of neutron radiation induced mutations, concluded that "all of the mutations were defective variations" (1971, p. 278). The vast majority of all non-neutral mutations are harmful in most environments, partly because they usually result in loss of information.

The two types of heritable changes that can occur in the DNA nucleotide sequence include a normal shuffling of genes (such as by sexual reproduction or genetic crossing over) and copying mistakes. Copying mistakes are alleged to account for most evolutionary changes because normal gene shuffling can only rearrange existing information (Spetner, 1997). Only those expressed mutations, or those that affect the organism's health or viability, can be selected for. Aside from loss mutations and the few situations discussed below, no *documented* case of a mistake or error type of mutation that has produced a phenotypic change that is beneficial in the wild exists, although some mutations are beneficial for humans such as seedless fruit, cattle lacking horns, or beans with strings (Tinkle, 1967, p. 68). Mutations are hypothesized ultimately to account for all life's diversity, but we have no *evidence* of any clearly beneficial mutations except possibly

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in a few unique situations such as a heterozygous sickle cell anemia mutation may protect persons against malaria.

The "rate and magnitude of deleterious mutations have substantial implications for conservation genetics and therefore, for preserving the diversity of life on earth" (Peck and Eyre-Walker 1997. In the medical field, non-neutral mutations are assumed to be harmful without exception. Some add the caveat: given enough mutations, one is bound to be helpful, at least in some situations (Price, 1996). Although the vast majority of mutations is known to be negative or harmless, evolutionary naturalists assume that enough rare positive mutations *must* have occurred in the past to have evolved all life forms.

The reason mutations are accepted as the source of variation is because only naturalism is acceptable as an explanation in contemporary orthodox science, and the best naturalistic explanation must be accepted until replaced by a superior naturalistic explanation (Bergman, 1992). Darwinists reason that this helpful character of some mutations must be true because life exists, and aside from normal variations such as those produced in sexual reproduction, the mutation theory is considered the only viable naturalistic explanation for diversification at existing gene loci. Mutations are the "driving force of evolution" and if the "DNA archive was absolutely constant and unchangeable, evolution would be impossible" (Sedivy and Joyner, 1992, p. 13). Evolutionists assume that if the mutation rate is slow enough, macroevolution can occur and life will not become extinct. But if too many mutations are produced, they believe extinction will occur (Nussenzweig, 1998). Furthermore, many mutations would have to occur almost simultaneously before a major change such as bipedal locomotion could evolve from quadipedal locomotion.

The Cause of Mutations

The primary building blocks of proteins are the 20 amino acids that are assembled into proteins according to the DNA genetic blueprint. A group of 3 DNA bases called a *codon* codes for each amino acid. Since the DNA code uses 4 bases (adenine thymine, cytosine, guanine), 64 (4^3) possible codes exist, more than enough to code for the 20 common amino acids used in most forms of life. Even for a very small peptide consisting of only 20 amino acids, fully 20^{20} (200 pentillion) kinds of polypeptides are possible. This enormous level of variety is necessary to code the 150,000 types of proteins now estimated to be required for human life, and also for the hundreds of thousands of proteins necessary for other life forms.

The correct amino acid sequence is ordinarily critical for most of the protein chain, but proteins with a few incorrectly placed amino acids can sometimes still function, although often not as well (Friedberg, Walker and Siede, 1995). Conversely, many single changes can be critical or lethal. An example is sickle cell anemia in which there is one single incorrect amino acid out of 300. The replacement of glutamine with valine produces red blood cells that tend to deform (called sickled cells) in certain environments. In the homozygous sickle cell condition the blood functions poorly under certain circumstances, causing anemia, severe pain, and even strokes in children (Feigle, Hill and Boschmann, 1991). Many other mutations prevent the production of functional enzymes. The consequent lack of a necessary component in the cell causes dysfunction or disease.

It is not always possible to determine the exact cause of each specific mutation (Clark and Wall, 1996 p. 147). Mutations are classified as either *random*, meaning they stem spontaneously from an unknown cause; or as *caused*, indicating that they were induced by a known agent called a mutagen. The potential causative agents of spontaneous mutations include cosmic rays (which are part of background radiation) and gene processing interference. The potential cause of induced mutations include carcinogens and mutagens such as those in cigarette smoke.

To distinguish between *spontaneous* and *induced* mutations requires a great deal of research. First, the researcher must isolate mutants from the culture which contains both mutated and normal cells. Knowing if the mutation is spontaneous or induced is important in certain areas, such as controlling bacterial infections. The use of penicillin provides an example: penicillin kills nonresistant bacteria, thus selecting for the resistant "mutant" strain. Why this occurs needs to be understood in our fight against disease. In many cases, changes labeled as mutations are not mutations proper because they do not involve actual DNA base changes in a particular "gene" but are the result of gene transfer or recombination.

Another example is the changes that are often improperly called "mutations" is the primary mechanism that the immune system of many organisms use to develop antibody diversity. A wide variety of antibodies is needed to respond to the billions of possible antigens, and there are not enough genes in the entire cell to produce the required diversity. The antibody diversity system was designed to achieve this requirement. The antibody gene system is actually quite complex and assembles gene products in a variety of ways so that a wide variety of antibodies is produced. This whole mechanism of antibody production by way of gene product recombination or shuffling is more accurately classed as a complex system which produces antibody diversity.

Normal Variations

It is difficult to prove that a biological trait or variation is due to a mutation. A primary concern in understanding mutations is the fact that changes in DNA base order can be produced by many factors other than genetic code changes. Changes caused by the independent assortment of alleles during sexual reproduction, chromosomal crossover, and other normal means of producing organized genomic variety are not mutations as normally defined.

For example, hairiness in tomatoes is often assumed to result from a mutation, but may be caused by a normal gene shuffling. Some genes can produce two or more different proteins by alternative splicing of the mRNA transcript. This allows one gene to produce two or more forms of a protein for different developmental stages or for use in different cell types (Watson, et al. 1992, pp. 135, 140). Much normal variety exists in life: humans alone possess an estimated 10 million normal variations called *polymorphisms*, meaning humans vary at about 1 per 500 base pairs (Jorde, Carey and White, 1997). Over 100 normal blood groups alone are now known to exist.

So called *amber mutants* provide an excellent example of what is often labeled a mutation but are more accurately described as genetic control mechanisms. An amber mutant bacteriophage will grow *only* in a bacterium termed a "permissive host" which contain a suppressor gene that can bypass the so-called mutation. The mutation involves a single-base substitution that alters the amino acid coding triplet to become a stop codon (UAG). It is read as stop under "non-permissive" conditions, terminating the protein coding before the entire gene is read. The so-called permissive phage host bacteria can "suppress" the mutation by producing a tRNA species which translates the UAG stop codon as an amino acid, so that it no longer functions as a stop codon. The specific amino acid coded depends upon the permissive host class in which the bacteriophage resides. This structurally complex and unique system possessed by the host allows the bacteriophage to produce the protein needed for its survival (Adams, et al., 1992, p. 79).

Mutation Hot Spots

A major problem for evolution is that, although many different mutation sites are possible for most genes, mutations are much more common in certain gene areas called mutation hot-spots. Although for some genes such as the beta-globin gene over 100 types of mutations have been reported, mutations for most genes tend to occur in the same base pairs (Jorde et al., 1997).

As a result, the same mutations occur over and over in the same locations, meaning the potential for improvement is close to zero. For some illnesses, many or even most new cases result from new mutations: for achondroplasia fully 80% of all cases diagnosed are caused by new mutations, for neurofibromatosis type I about 50%, and for Marfan syndrome 30%. These mutations tend to be in the same area, or even on the same base, of the gene affected. The situation is similar to a typist repeating the same type errors over and over and over because each base pair does not have an equal likelihood of a mutation. Some mutations are so common that scientists have given them whimsical names as *stuck*, *radish*, and *shot-full-of-holes*. Few if any of these mutations provide new material needed for evolution by natural selection.

Back Mutations

Because mutations also tend to occur in hot-spots, a great likelihood exists that a back mutation repair will occur, i.e. another mutation will repair the damage by changing the base back to the original form. Even the most common mutations can and commonly will back mutate, and genes that are "highly susceptible to mutation" must also be "highly susceptible" to back mutations (Beisner, 1987, p. iv). Wilson and Balding (1998) even conclude that back mutations are evidently so common that it is difficult to use mutational data to estimate populational histories and evolutionary processes. Thus, back mutations would tend to dilute any positive effect that certain mutations would be hoped to have in evolution.

The One-Directional Nature of Mutations

Most mutations usually cause changes *in one direction* only, resulting in *not* increased diversity as evolution requires, but a *reduction* of diversity. For an analogy, assume that typewriter mistakes tended to produce one letter most often. Then the possibility of a mistake improving the text is considerably *less* than chance. Thus typing letters by chance would be more likely to produce an improvement in the paper. An example is deamination of cytosine to thymine, which causes the compliment base to be converted from G to A. This change results in fewer and fewer G bases and more and more A base pairs in the genome, reducing information instead of increasing it. If G to A mutations were more likely to occur in a set such as AGATCGCGAT, the code would eventually become AAATCACAAT.

The best known example of a mutation that tends to go in one direction is the two-base dinucleotide sequence CG. Methylated genes are particularly prone to mutations, specifically deamination, and this causes changes in *one* direction only. In mammals, close to 80% of CG dinucleotides are methylated, meaning a methyl group is

bonded to the cytosine base. The compound this mutation produces, 5-methylcytosine, causes cytosine to lose an amino group, converting it to thymine. This mutation would tend to convert the cytosine to thymine, causing thymines to become more common and always resulting in a *loss* of information.

This change results in no small difference. The CG dinucleotide mutation rate is fully 12 or more times higher then other dinucleotide sequences, and CG dinucleotide mutations have been identified in a large number of important human diseases (Jorde et al., 1997 pp. 39–40). This tendency would constantly result in a greater number of thymines and would in the long run always be degenerative.

The Rate of Mutations

Each chromosome contains the units of heredity, the genes, which are now estimated at 150,000 in humans and an average of 5,000 to 8,000 genes on each chromosome for mammals. Over 3 billion base pairs are required to form the entire human gene code, and bacteria contain about 5,000 genes or 4.7 million base pairs. The background mutation rate ranges from 1 per thousand to 1 per billion cell divisions—and it is usually about one mistake per billion bases (Cain et al. 1999).

Much genetic research has been completed on bacteria, relatively simple organisms compared to humans, but still enormously complex. Microorganisms are also extremely useful in the study of mutations because of their short generation time and the low cost of maintaining large populations for research. Comparisons of normal and "mutant" organisms can easily be done and this is an important method to study metabolic pathways.

The number of mutations in humans is estimated to be about two per gamete (Mader, 1998, p. 307). Many of these mutations are lethal and result in a miscarriage or a spontaneous abortion, and consequently they are eliminated from the gene pool. Some genes, though, are far more prone to mutate than others, and many of these mutations produce well known deleterious or lethal effects. Given these data and an estimated 150,000 human genes, the average person would carry only about two to eight harmful genetic mutations, most of which are not expressed because their other allele is normal (Gonick and Wheelis, 1991, p. 80). This number does not include somatic mutations, which are those that occur in individual body cells and are not involved in either sexual reproduction or evolution.

One reason that the number of mutations is comparatively minute is that at least eight known repair mechanisms lower the rate of expressed mutations by an enormous amount. Numerous enzymes hover in and around the chromosome like worker bees and help to unwind, wind, repair, transcribe, replicate, and replace defective parts of DNA. These repair systems correct about 99.9% of all initial errors (Jorde et al., 1997). After repair, a rate estimated at 10^{-7} mutations per gene is common in sexually reproducing organisms (Mader, 1998). Therefore, even if .0001% of all mutations were beneficial (a high estimate), the total number of beneficial mutations in a population would be extremely small, especially in relatively small animal populations such as exist for most mammals and almost all primates.

For a large population, the total number of non-lethal mutations is so small that the potential of mutations to produce both new information and beneficial effects is close to nonexistent. A critical argument against the mutation theory is the conclusion that at most "on average, only one in 500 base pairs will differ from person to person." Thus, at the genetic level the exons (protein coding DNA) are over 99.99% identical in humans (Jaroff, 1991, p. 23). This means that during the one-million years speculated to have elapsed since our common ancestor evolved, virtually no signficant changes in the gene pool could have occurred.

The Effects of Mutations

In studying human diseases, researchers have located thousands of the mutations responsible for a particular disease. Over 800 different mutations have been located in the cystic fibrosis genes alone—yet, not *one* of these known mistakes has been shown to be beneficial (Mak et al., 1999). None of the millions of different mutations that has been identified have been proven to be beneficial except a few that are useful only in extremely limited and unusual circumstances. The most well known example is sickle cell anemia, a disease that affects about one out of 625 Blacks (.0016%). Even mutations that are beneficial in very limited situations are rare, and can sometimes cause problems even if the mutation is heterozygous (Jaroff, 1991, p. 8). The homozygous sickle cell anemia gene condition causes a serious illness in all cases, and even the heterozygous form causes health problems and in some circumstances sometimes death.

Cystic fibrosis is caused by a mutation in the gene that processes the chloride ion. A faulty sweat and mucous gland mechanism results that causes excess chloride loss in sweat. More serious is the production of an abnormally thick sticky mucus that tends to trap and hold bacteria in the bronchial tubes, causing major respiratory problems and infections. About 70% of cystic fibrosis cases are caused by the loss of just three base pairs that results in the loss of a single amino acid—phenylalanine—from the protein that the gene produces (Jaroff, 1991, p. 36). In addi-

tion to this mutation hot spot, many other rare mutation types that can cause cystic fibrosis have now been located. All of those render the critical proteins either less functional or even non-functional.

The Neutrality of Most Mutations

Changes in the third and sometimes the second codon, for example, often do not alter the protein produced. If several codes are used for an amino acid, a number of changes in the code, especially in the last base, will not change the amino acid that is coded. For example, leucine is coded by CUU, CUC, CUA, CUG, UUA, and UUG. Thus, the same amino acid will be coded for the CU code regardless of what base is last in the codon. Consequently, a large number of single point mutations will have no effect on the protein coded. This system is said to be *degenerate*, meaning it can "degenerate" from the original code and still code for the correct amino acid sequence (Jorde et al., 1997). Only if an amino acid with a very different side chain is inserted, such as valine replacing glutamate, is the protein rendered nonfunctional. Mutations can also be neutral if the substitute is an amino acid with a similar side chain, such as the replacement of leucine by isoleucine or aspartate by glutamate. In these cases the proteins function is often not affected or is affected only slightly.

Many mutations produce barely perceptible, often inconsequential, changes. One such mutation affects human eye development so that the eyes are a millimeter or so farther apart than normal. Some mutations cause a defective enzyme that is relatively unimportant or can be dealt with by diet or lifestyle changes. Leu and Dill (1990) claim that one reason most mutations are neutral and do not result in phenotypic charges is that many biomolecules seem to be relatively insensitive to many single substitutions, especially those in the amino acid chain ends or terminal portions. These terminal base pairs serve as spacers, for protection, or are cut out during processing. Mutations often cannot damage these areas but cannot improve them either. Some organisms are protected from mutation-caused damage due to over design or built-in protection mechanisms such as the genetic repair mechanisms. None of the mutations discussed above are known to result in adding information to the genome. If most mutations are neutral regarding selection, the neo-Darwinian theory is in jeopardy because mistakes accumulate which are not selected against but that in time, according to some evidence, adversely effect the organism's health.

A mutation in a somatic cell of an adult is often of little consequence because most of the cells around it are normal. Some somatic mutations may result in cancer or other problems, but they cannot provide the variety needed for evolution to work because they are not inherited. The many lethal mutations which often cause spontaneous abortions, estimated to be as many as one-third of all human conceptions, are also not passed on.

The Recessive Character of Non-Neutral Mutations

Mutations often produce defective proteins, but a mutation usually affects only one gene and humans have two sets of chromosomes. In the case of mutant recessive genes, the "insurance gene" can still produce the correct functioning protein. For this reason, only when both alleles are recessive mutated genes is the organism usually adversely affected, such as the sickle cell anemia mutation. The organism with one mutation may have less of the normal protein, but still can often function satisfactorily. A major exception to this occurs with the sex linked genes that are located on the X chromosome which have no corresponding locus on the Y chromosome. Consequently, recessive genes located on the X chromosome, such as color blindness and hemophilia, are often expressed in males, but rarely in females.

A major reason why improvements from mutations are highly unlikely and why mutations present little hope in producing evolutionary innovations is that most mutations are recessive and therefore must exist in pairs to be expressed. The presence of a recessive mutant allele in the homozygous condition is an extremely unlikely possibility except in consanguinous marriages:

> When an allele of a gene is mutated to the new allele, it tends to be recessive and its effects are normally masked by its partner allele. Only in the homozygous condition can such mutant genes be expressed. Thus a population carries a reservoir of mutant recessive genes, some of which are lethal when homozygous but which are rarely present in the homozygous condition. Inbreeding encourages the formation of homozygotes and increases the probability of recessive mutants being expressed in the phenotype (Hickman, Roberts and Larson, 1997 p. 79).

Both beneficial and deleterious recessive mutations are more likely to be expressed in consanguinous marriages. This is one reason why marriage to close relatives is illegal or discouraged in many countries, and may be one of the reasons it was forbidden in the Bible. Serious health problems result in animals produced by the inbreeding practiced to produce thoroughbreds: a mutt or mongrel is often the healthiest animal. While discussing recessive alleles, it is important to note that in the case of dominant mutant genes, a defect in only one gene will often produce major problems. The result is production of a protein that does not fold properly, and for this reason is cut up by the cells' proteolytic system. Lack of a functional protein can cause

an illness, or a defective protein can be poisonous to one or more cellular systems. Furthermore, mutations that affect only one gene may cause mispairing during meiosis and may well be lethal.

The Demise of Even Helpful Mutations

Most mutations, even if they have positive selective values, will eventually be eradicated by random effects that have close to an equal probability of killing individuals carrying the beneficial mutation or any other member of the population (Spetner, 1997). Positive single mutations have only a small chance of survival: even if a single mutant appeared, it would likely vanish through random effects such as floods, disease, or predators much like the descendants of the animal without the mutant. Its positive selective value would do little to help it survive these random effects that work to destroy it.

Consequently, a tendency exists for one new offspring per member of the population to survive long enough to reproduce. Organisms generally give birth to many more than one offspring. Frogs can lay hundreds of eggs several times during their lifetime, a herring will lay about 50,000 eggs per year, an oyster can produce 100 million eggs in its lifetime, and a lower plant can produce a trillion spores (Spetner, 1997). Some cannot reproduce and others that can reproduce might not do so because of being eaten by predators or dying in a catastrophe such as a drought, flood or fire. Often, they die not through any fault in their own but through random events (Raup, 1991).

An optimistic calculation suggests that an animal in a stable population produces an average of five offspring in its lifetime, an average of only one out of five will survive to reproduce. Therefore, the chances are only one out of five, or 20 percent, that a particular animal will survive long enough to reproduce. Furthermore, whether or not a family member survives is largely a matter of luck (Raup, 1991). A mutant with a selective value 0.1 percent higher than its siblings means that its chances of surviving is 20.02 percent instead of 20 percent (Spetner, 1997). A single mutant with a survival value of 0.1 percent better then its siblings in the first generation has a likelihood of survival of 0.2002 compared to 0.2000, and of surviving 2 generations 0.1347, and the chance that it would survive each succeeding generation is even less. For this reason, the chances of survival the first few generations are affected very little by the mutant's selective value. Furthermore, four significant figures are required to see the difference between the mutant and the rest of the population.

As a result, virtually all positive mutations are unlikely to play a role in evolution and a positive mutation normally can play a role in evolution *only* if many of that type occur simultaneously, which has close to a zero probability of occurring. Simpson (1969) and others have concluded that a single mutation has little chance of staying in a population. If the chance of one mutant surviving is 0.002, a gene mutation with a selective value of 0.1% will have only one chance in about 500 of surviving the random effects that tend to wipe it out (Spetner, 1997). If 500 mutations with a selective value of 0.1% existed, the chance that at least one would survive would still be only about .6. If 1000, their chances would be about .87, and only when there were about 2500 would their chance of surviving be more than 99%. For this reason, positive mutations will take over the population only if many of them occur simultaneously, otherwise they will likely disappear (Spetner, 1997).

The key to Darwin's theory is that "slight modifications, which in any way favoured the individuals of any species, by better adapting them to their altered conditions, would tend to be preserved" (1872, pp. 82–84). The facts contradict Darwin's belief that natural selection will preserve even the slightest variation and increase in number until it takes over the population. Most larger mammals and many other animals produce relatively few offspring in their lifetime. Consequently, for most animals, relatively few exist to produce mutations, yet the variety of extant species types is now estimated to number in the multi-millions. Given this fact, if macroevolution were possible, it would be extremely slow—and multi-millions of years would be required for even minor species changes (Howe and Davis, 1971).

The fact that many genetic code sequences are shared by humans with many lower organisms, some of which are virtually identical, yet alleged to be many millions of evolutionary years apart, is also problematic for Darwinism. This is a problem because why stasis existed in these genes while other loci presumably underwent extensive changes often cannot be explained. For example, many human genes are so similar to yeast genes that "human DNA can be substituted for the equivalent yeast gene—and it works just as well" as far as we can determine (Pines, 2001, p. 10). Yet evolutionists claim the two are separated by "over at least a billion years of evolution." And the finding that many human and yeast genes are identical, or very similar, is put in proper perspective when it is noted that many human genes consist of thousands of base pairs. It will not suffice to ignore the problem by claiming, as certain Darwin apologists do, that these similar sequences were simply "conserved" by selection across many eons of evolutionary time while other genes evolved. The "conserved" label does not explain the problem, but only identifies it.

For example, one of the larger genes, the gene for human growth hormone, is 150,000 base pairs in length (Jaroff, 1991, p. 52). A defect in the gene that causes neurofibromatosis occurs in a series of small exons spread over at least a 200,000 base pair section of chromosome number 17 (Jarroff, 1991, p. 60). Many of the code differ-

ences result in small but necessary differences between organisms, although some make no difference. Even assuming that evolution is valid, these relatively small changes in the genes also would reveal the extremely slow rate of change that mutations cause.

The Reasons Why Non-Neutral Mutations Are Usually Fatal

Negative mutations produce quite a different effect from neutral/beneficial mutations. A major problem for the mutation theory is the fact that gene functions are highly interrelated and damage to one gene usually has effects on *many* systems. Most genes are *pleiotropic*, meaning that they have multiple effects, and if both are damaged, adverse effects can result in many different organs and functions (Jorde et al., 1997 p. 72). One reason why genes are pleiotropic is that they produce enzymes that are part of shared biochemical pathways. As a result, when pleiotropic genes mutate, many problems result. This is why genetic disorders typically cause "complex, multiorgan, and systemic conditions" (Jorde et al., 1997 p. 232). Even if a mutation is clearly beneficial in some way, many mutations will have major adverse affects on the organism due to the pleiotropic effect.

Given a sequence of several hundred amino acids, a single mutation can result in a misshaped protein that may render the entire protein non-functional. A defective protein can also often cause the entire structure that the protein is part of to be defective, and as a result produce disease. A typographical error in *any* one word in a sentence will likely render the entire sentence incorrect—an analogy that is limited because a sentence may function with certain typographical errors if readers can discern its meaning by the use of intelligence. With proteins, one incorrect amino acid can result in an error in a protein that can produce a useless protein. If the protein is an enzyme, it can adversely affect all of the structures that the enzyme works on. As a result a single or only a few changes in the DNA code often can have drastic effects.

Mutations Cause Devolution

"Devolution" is the corruption of the genome. The accumulation of mutations has caused the defective gene load gradually to increase until now an estimated 5,000 mutation-caused diseases exist in humans (Cotran, 1999). Some mutations are much more frequent than others, with certain mutations having evidently occurred only once or a few times in history. It is for this reason that certain mutations are often found only in specific populations and can sometimes be traced back to their source. Sickle-cell anemia has been putatively traced to an African who lived in Sudan, East Africa, and from there spread "along with slash-and-burn agriculture" (Weiss and Mann, 1990, p. 491).

We would also expect that the farther back we go in history, the *fewer* the number of disease causing mutations would have been in the human gene pool. This is clear evidence for devolution because the farther back in history, the more perfect our genome would have been. An estimated 80 to 85 percent of mutations are familial, and the rest are acquired de novo by the affected individuals (Cotran, 1999). As the mutation load increases, more and more mutation-caused diseases enter the human gene pool. Spetner noted that:

> all mutations studied destroy information. None of them can serve as an example of a mutation that can lead to the large changes of macroevolution. The neo-Darwinians would like us to believe that large evolutionary changes can result from a series of small events if there are enough of them. But if these events all lose information they can't be the steps in the kind of evolution the NDT [Neo-Darwinian Theory] is supposed to explain, no matter how many mutations there are. Whoever thinks macroevolution can be made by mutations that lose information is like the merchant who lost a little money on every sale but thought he could make it up on volume (Spetner, 1997, p. 160).

Spetner's conclusion is also supported by the fact that genetic repair systems insure that most all mutations will be corrected. Primarily those genes that have mutation hot spots will produce unrepaired mutations, and it is these hotspot mutations that cause devolution. The major way that this problem can be dealt with is by the use of genetic engineering to develop gene therapy methods to circumvent mutations. For hemophilia, for example, temporary relief can be obtained by injecting patients with factor VIII protein, the clotting mechanism that their blood lacks. But any permanent correction will require the deliver of working copies of the genome into the patient's genes by the use of retroviruses as vectors (Adams, Knowler, and Leader, 1992).

The evidence produced from tracing mutations back in time reveals that, although some have occurred repeatedly, such as Duchenne muscular dystrophy, certain other gene mutations evidently are relatively uncommon, such as Tay-Sachs disease (*amaurotic familial idiocy*) or sickle cell anemia (Livingstone, 1976). Mutations are common in the case of Duchenne Muscular dystrophy because the DMD gene is the largest known human gene. It is 2.4 million DNA base pairs long and produces 14 Kb of mRNA which manufactures a protein chain that consists of 3685 amino acid residues! The larger the gene, the greater the likelihood of a mutation because larger targets are more likely to be damaged by cosmic rays, and the more the bases, the greater the likelihood that any one will be dam-

aged by any type of mutation. The gene codes for *dystrophin*, which is involved in maintaining the integrity of the cells' cytoskeleton (Jorde et al., 1997).

Conclusions

There exists a huge number of possible gene sets in the over a million known species, and there also exists a high level of trait variation *within* each species. No historical evidence exists, however, to support the contention that mutations have produced a new order, a new family, a single major biological innovation (such as a wing), or even a single useful gene (Rust, 1992; Spetner, 1997).

Recombination and other natural means of producing variety have evidently produced most if not all of the "new" characteristics observed, such as the minor variations common in existing genotypes (Cain et al., 1999). This is not Darwinian evolution, but merely the formation of new traits from novel combinations of genes that already exist in the family gene pool. Only qualities which already exist in the gene pool can be "developed" or altered by selective breeding.

Major variations within the animal and plant world can be accomplished only by intelligent selection. But even then, when left to themselves, the selected traits that humans produce from deliberate animal interbreeding are usually lost in the wild state. Their progeny soon revert back to the original wild type as in the case of wild dogs. The gap between different major groups of animals has not been narrowed by breeding as Darwin wrongly predicted it would be.

One may conclude from the empirical evidence that the primary effect of mutations is to weaken and kill because few if any mutations are clearly beneficial. Although there are many varieties of life that can interbreed with similar organisms to produce virile offspring, various gene mechanisms and natural selection often destroy any varieties that stray too far from the norm, insuring that each group continues to bring forth only "after its kind," as the Bible states. Natural selection operates as a conserving mechanism which *reduces* the mutation load, and research has found that mutations are established in a breeding population at a very slow rate (Howe and Davis, 1971, pp. 37, 40–44). Nor can mutations account for the origin of the gene loci themselves. The answer lies in what is vividly seen everywhere in our physical world: the Creator's design and intelligence.

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Scientists Speak About the Origin of Insects

Today two-thirds of all species on the earth are flying insects, and their ways of life and ecological niches are almost incomprehensibly diverse.

James H. Marden, *The Sciences* Nov/Dec 1995, p. 26

The fossil record does not give any information on the origin of insects.

The New Encyclopedia Britannica, Vol. 19, 15th edition, 1986, p. 804

Certain modern species are reasonably similar, in their anatomy, to fossils of winged insects dating back 325 million years. The problem is, wings appear in the fossil record already fully formed.

James H. Marden, ibid. p. 27

What could be more familiar than the hover and dart of a fruit fly, going about its business? And what could be more mysterious? Thirty-odd muscle pairs, in coordinated motion, flap the wings up and down 200 times a second—rapidly enough to sing a baritone G below middle C. Biologists have tried for decades to sort out the complexities of insect flight. The wing hinge, where the wing joins the thorax of the insect, is, in the words of Michael H. Dickinson, a neuroethologist at the University of Chicago, "the most morphologically complex joint in the animal kingdom."

Mary B. Aberlin, *The Sciences*, Nov/Dec 1995, p. 13

So miraculous a thing is insect flight that nearly all insect biologists believe it could have evolved only once.

James H. Marden, ibid. p. 28