Mutation And Natural Selection: The Central Dogma of Neo-Darwinian Evolution

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Abstract

The central dogma of neo-Darwinism is: Mutations provide variety that is selected by natural selection. Mutations and natural selection are ultimately the only driving force of evolution, but they are effective only if one or more mutations prove beneficial to an organism in a given environment. In evolution, natural selection can do nothing without mutations. Since macromutations are virtually all near-neutral or harmful, micromutations are the only means by which new species could possibly evolve. The probability that micromutations will lead to new species is so low as to be close to zero. One reason is the net deterioration of the genome that occurs as a result of mutations, especially near-neutral mutations, which selection and medical advances cannot counteract. Furthermore, nonrandom "mutations" are controlled by in-built cellular processes and, therefore, cannot be a cause of evolution. A large number of studies clearly show that the central dogma has failed, and this devastates the credibility of neo-Darwinism. Furthermore, the increasing knowledge of the immense complexity of life at the molecular level has buttressed both the Creation and Intelligent Design movements.

Introduction

What drives the alleged evolutionary process? Does science know the underlying mechanism that can convert one genera into a new genera, one basic type into a new basic type, or explain the origins of the differences between the higher level taxa? Darwin thought of evolution primarily in terms of the natural selection of normal biological variations existing in all life, resulting in survival of the fittest in the struggle for existence. The literature today refers to natural selection as a process, a force, or a mechanism, and even as an active, almost intelligent, agent that "picks" what will survive. Darwin thought that normal biological variation could be extended to the point that entirely new species are formed over long periods of time. His major proposed mechanism of pangenesis (reviewed in Bergman, 2003) has been scientifically discredited.

Genetic information is contained in the aperiodic arrangement of nucleotides in DNA and is unique not only for any species in organisms but also for variations and individual mem-

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bers within a species. New genetic information would be the *additional* information contained in species B if it were unique from and required for it to evolve from species A or if new species B had been created independently from species A. Unless it is experimentally proven that as a result of random mutations the aperiodic arrangement of nucleotides in species B positionally fits exactly (or almost exactly) the aperiodic arrangement of nucleotides in species A that is already known, there is no basis for concluding that species A evolved into species B. Such proof does not exist.

Other attempts to explain the source of new genetic information are summarized by Bergman (2003). They include Lamarckianism, orthogenesis, Bergson's creative evolution theory, theistic evolution, DeVries's macromutations, Goldschmidt's "hopeful monster" theory, panspermia, punctuated equilibrium, symbiogenesis, and chaos theory. None of these theories has survived scientific scrutiny, and none has been able to successfully challenge the present-day neo-Darwinian theory of mutation as the source of new genetic information, a mechanism that is also increasingly recognized as inadequate. As will be shown, mutations have not been able to explain the source of new genetic information.

Neo-Darwinism

According to the modern or synthetic theory of evolution, now called neo-Darwinism, evolution's driving force is random mutations that cause phenotypic variations, which are then acted on by natural selection. The discovery of the DNA double helix molecular structure, the function of genes, and the laws of inheritance all have provided neo-Darwinism with a mechanism to allow organisms to evolve by altering the genetic program encoded in the genes. Darwin knew nothing of DNA, the material of inheritance that transmits an organism's characteristics to its descendants, or of mutations, the laws of inheritance, or the extreme specified complexity of living cells.

Naturalistic evolution theorizes that nonliving simple molecules evolved into the first living cell (through self-organization), and from there all life, including humans, developed through the selection of beneficial mutations by natural selection. The process occurred without guidance, direction, or purpose, except for the purpose of survival itself. It began about three billion years ago with the appearance of the first unicells, followed by the Cambrian explosion of complex, multicelled organisms 530 million years ago, and since then the evolution of all complex life on earth has occurred. This theory gradually overthrew the once dominant view called creationism, and conquered academia, education, and the media.

The mechanism of evolution at the molecular level consists of the rearrangement of DNA base pairs of a particular species by copy errors in such a way that they produce new genetically encoded traits that are selected, eventually producing a new species. Since mutations can only produce a few base pair changes at a time (because a large number of changes tend to be lethal), a new species is slowly evolved. If it could be demonstrated that evolution through random mutation of DNA bases is highly unlikely, or even impossible, then no known mechanism to generate new information for evolution of new species would exist. As will be shown, natural selection, and thus macroevolution, cannot work without mutations.

The nonexistence of information-adding mutations has equally serious ramifications for both naturalistic and theistic evolution, because, while some theistic evolutionists have difficulty accepting randomness, they assume the same history and mechanisms for evolution as do naturalistic evolutionists.

Random Mutations

Can Macro- and Micromutations Produce New Species?

The major driving force of the naturalistic theory of neo-Darwinism is random mutations. No macroevolutionary process can exist without a constant supply of mutations (Bergman, 2005a). The concept of macromutations appears to have different meanings, or at least different formulations: (1) Apparently it was first used of saltational changes in Goldschmidt's "hopeful monster" theory. (2) The concept depicts macromutations as affecting regulatory genes that activate other genes, which cause the synthesis of proteins. (3) The concept depicts macromutations as the cause of major alterations in the phenotype, the visible properties of an organism, which virtually always, if not always, are destructive to the organism. This is the way macromutation is used in this paper.

Mutations can have both external causes (radiation from the sun, space, or earth) and internal causes (toxins, radiation from food, and free radical damage) that damage the genes. Several thousand mutated genes that lead to serious illnesses and death have now been identified. Since these destructive mutations cannot be the driving force of evolution, I will ignore harmful and fatal mutations in this analysis of evolutionary mechanisms.

Most known macromutations are harmful and, therefore, cannot be a significant source of genetic variation for evolutionary development. For this reason, micromutations (small changes in the structure or arrangement of the genes, usually in a single nucleotide change) will be considered. These micromutations are assumed by both evolutionists and some creationists to be the ultimate source of genetic variation that produces phenotypic variation within the species, but in general only evolutionists ascribe micromutations to

be the cause of the step-by-step continual formation of new species over millions of years. So far no documented examples of mutations exist that meet the necessary conditions for the cumulative selection required to evolve new life forms.

Most micromutations cause small changes, resulting in no clear destructive genetic effects, and for this reason the cell often can tolerate them. The smallest mutation type, a point mutation, changes one base into another and is usually the result of a copying error that occurs when chromosomes duplicate themselves during cell division. Most copying errors are random, a requirement of naturalistic evolution. Although a small number of errors remain, the cell's ability to "proofread the text" and repair mistakes reduces the errors a hundredfold or more.

The large number of highly effective genetic repair systems in the cell fall into three major categories: error avoidance, error correction during DNA replication, and advanced repair of errors missed by the first two systems (Bergman, 2005b). These small errors demand millions of years of evolution to be able to produce a substantial change in a gene. If certain errors cannot be repaired, tumor suppressor genes send the cell into a complex programmed destruction cycle called apoptosis (Bergman, 2008a). Creationists believe that the many repair systems are part of the Creator's design to maintain the information in the basic created genome.

Proteins are constructed according to instructions emanating from the information in DNA. The proteins consist of long amino acid chains that are "folded into specific, intricate, three-dimensional shapes that contain all the twists, turns, folds, pockets, and loops essential for performing the numerous functions required by the cell" (Bergman, 2006). Proper folding requires "chaperones" and other molecular machinery called "folding factors." The folding is double-checked several times by numerous other quality control systems. Mutational changes sufficient to produce new proteins would also likely require a new set of custom chaperones and enzymes to fold the new protein properly. These new chaperones and enzymes would also require new genetic information. After folding, a protein must successfully pass through multiple layers of monitoring before it can be sent to the needed location in the cell. Both the astounding genetic repair systems involved in the transcription step and the elaborate quality control factory for proteins are major problems for neo-Darwinian evolution and support the concept that the origin of life was by direct, miraculous creation.

Another serious problem for evolutionary theory is that, due to these error correction/quality control systems, copying errors are relatively rare. Neo-Darwinism assumes only slight genotypic and phenotypic changes that occur over long periods of time; otherwise the organism usually will not survive. The frequency of copying errors escaping the repair mechanisms for most organisms is about one per ten billion transcriptions per nucleotide (Spetner, 1997). Spetner calculated the possibility that a new species can be formed by random micromutations by determining the following parameters:

(1) the chance of a mutation occurring, i.e., the mutation rate
(2) the fraction of the mutations that have a selective advantage
(3) how many replications occur in each step of the chain of cumulative selection
(4) how many steps are required to achieve a new species.

Values for these parameters are all either known or can be estimated. For organisms other than bacteria, the first of these parameters, the mutation rate, is about 10^{-10} per nucleotide per live birth (Spetner, 1997, notes p. 123). However, not just any copying error can serve as a step that aids cumulative selection; it must add information to the genome that produces a phenotype having a positive selective value. Fisher, who did much of the original work in population genetics, found that even "beneficial" mutations are likely to disappear from the population for various reasons and that a single mutation, even if favorable, will have only a very small chance of establishing itself in the species population (Fisher, 1958). Although Fisher, an English statistician, evolutionary biologist, and geneticist, published this 50 years ago, his conclusions are still valid today (Spetner, 1997).

If evolution is to function, large numbers of adaptive mutations must appear. Only then could mutants survive the vagaries of selection. But adaptive mutations are very rare. Simpson, generally acknowledged as the dean of modern evolution, determined a "frequent selective" value at about 0.1% (Simpson, 1953). Although published 45 years ago, Spetner and others use Simpson's 0.1% as a typical selective value (Spetner, 1997).

From the inferred changes in the fossil record of the so-called horse evolution during the past 65 million years, Spetner concluded that one typical small step of evolution would require about 50 million births (Spetner, 1997). The probability of at least one such mutation during this time is 50 million times 10^{-10}, or one in two hundred per nucleotide (for how Spetner arrived at 50 million births and a mutation rate of 10^{-10} per nucleotide, see Spetner 1997, pp. 121–124). Assuming that there is an equal chance that the base will change to any one of the other three bases in the nucleotide triplets (codons) that specify a single amino acid, the chance of obtaining a specific change in a specific nucleotide is a third of that, or 1 in 600. (Spetner, 1997, p. 100, slightly altered). This probability is the second of the above parameters.

Fisher's calculations (see reference above) show that for only one mutation

with a 0.1% selective value, the chance is 1/500 that it will survive, and this is the third parameter used. The chance that a specific copying error will appear is 1/600, and for it to both appear and survive to take over the population is 1/600 x 1/500 = 1/300,000—less than the chance of flipping 18 coins and coming up with only heads.

G. Ledyard Stebbins (1966), considered the founder of evolutionary botany, 40 years ago estimated that to evolve a species requires about 500 steps, an estimate still accepted as the lower level today. Spetner uses this as the fourth of the above parameters (Spetner, 1997). Other researchers estimate a much larger number of steps or transitions are required to evolve a new species, which would considerably lessen the probability for this to occur. The adaptive mutation must survive each of these steps. The chance for this is estimated at $(1/300{,}000)^{500}$ or about 2.7×10^{-2739}, or more than 2500 orders of magnitude below 10^{-50}, which statisticians consider the level of probability approaching impossibility!

Therefore, there appears to be an impenetrable wall separating the basic organism types. On this basis Spetner rejected neo-Darwinian theory. His calculation spans several pages, some of which is included in Larssen (2001), with a more detailed presentation in a book by Larssen (2004). Using different paths, others, such as Ho and Saunders (1979), have come to the same conclusion as Spetner. Using a different mathematical proof in a book of surprisingly few pages, Hoyle and Wickramasinghe (1982) arrived at close to the same result.

Gnawing doubts about beneficial results of random mutations have led some to propose the idea of quantum mutations. One proponent of this idea is Kenneth R. Miller, a theistic evolutionist, professor of biology, and outspoken anti-creationist (Miller, 2000). Quanta at the subatomic level allegedly can evolve the first living cell by chance fluctuations of vibrational energy changing a molecule's configuration and producing a spontaneous mutation. That quantum effects can be actualized and magnified in this way is not accepted by most evolutionists. Furthermore this constant vibrational energy would cause havoc in the genome.

Mutational Deterioration of the Genome

The deterioration of the genome as a result of mutations is well known and widely acknowledged by geneticists. Examples include a book focusing on the aging part of genomic deterioration (Vijg, 2007), the article "Darwinism and the Deterioration of the Genome" (Bergman, 2005a), and the work of John Sanford (Sanford, 2005), which will be discussed in the next section. Bergman concludes that the production of new information by mutations is at the core of the validity of neo-Darwinism. He attributes the degradation of the genome to many causes, such as (1) the tendency for mutations to produce a highly disproportionate number of certain nucleotide bases, and (2) many mutations occur in only a relatively few places within the genes called hot spots. Bergman finds that there is little or no evidence for beneficial mutations that can produce macroevolution and that the genome is deteriorating. He shows that nearly neutral mutations create major problems for evolutionary theory because they cannot be removed by selection, eventually causing mutational meltdown (Bergman, 2008b).

Origin and Maintenance of the Complex Genome

The genome is not only extremely large but also exceedingly complex and full of bends and branches, with genes that control other genes that control still other genes. The same row of genetic letters can code for completely different instructions. Tens of thousands of different types of sophisticated nanomachines carry out incredible chemical feats in the living cell. The information used to produce and regulate these components is encoded by the genome. When asked how all this could have come into existence, the standard answer is what Sanford calls "The Primary Axiom": "Mutations combined with selection have created all biological information" (Sanford, 2005).

Sanford concludes that the overwhelming deleterious effect of mutations can clearly be seen in the demonstrated lack of information-creating mutations. He is not convinced that a single, clear example exists of a mutation that unequivocally has created new information, even in the case of antibiotic resistance in bacteria. Anderson (2005) provided a detailed analysis of antibiotic resistance, and showed that they are the result of deleterious genetic events, the opposite of new information. Bergman made a literature search, finding that of over 450,000 "mutation" hits, only 186 even mentioned the word "beneficial." or similar terms. Those labeled beneficial were beneficial only in a very narrow sense and consistently involved loss of information (Bergman, 2005a).

Population geneticists know by experience that virtually all mutations are largely neutral or deleterious. Mutations that have positive effects on fitness are so rare that they are excluded from distribution diagrams that show mutational effects on fitness as a function of mutation frequency. Kimura (1979) argues that most mutations are nearly neutral and are, therefore, not subject to selection. He shows this in distribution curves that include a relatively narrow box on each side of the zero point, representing a "no-selection zone." No beneficial mutations are shown to the right of the zero point (see Figure 1). Sanford (2005) shows a corrected distribution (see Figure 2), compared to the one used by Kimura. An interesting feature incorporated in Sanford's diagram (Figure 2) that is not found in Kimura's diagram is the tiny

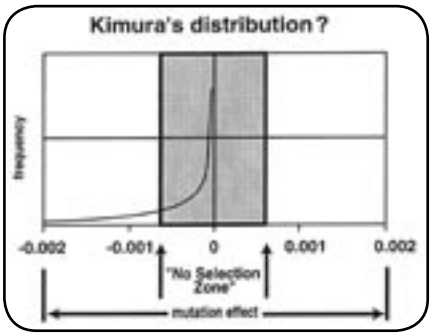

Figure 1. Distribution diagram of mutation effect adapted from Kimura (1979). Most mutations are nearly neutral approaching the zero point. Deleterious mutations are farther to the left. The "no-selection zone" is shown by the box.

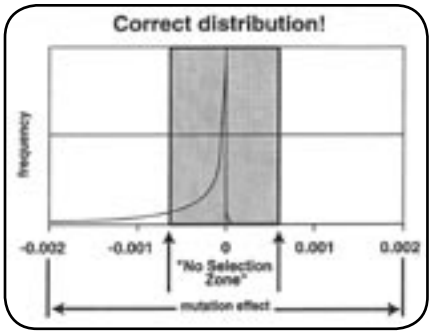

Figure 2. A corrected Kimura diagram (from Sanford, 2005) showing where beneficial mutations would occur. Their distribution is reduced in range and scale by a factor of ten thousand to one million. This part of the mutation distribution could not be drawn small enough, and a relatively large triangle is shown just to the right of the zero point. Even with beneficial mutations greatly exaggerated, essentially all beneficial mutations will still fall within Kimura's "no-selection zone."

triangle slightly to the right of the zero point, entering the beneficial mutation area. Because of the diagram's scale, Sanford could not draw this section of the distribution diagram in proportional

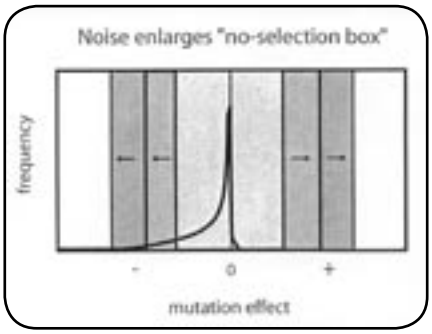

Figure 3. Sanford's much larger no-selection zone due to several noise factors (reproduced from Sanford, 2005, Figure 9, p. 104).

scale to the deleterious section. The chance of a beneficial mutation is so rare that it would otherwise not show up in the distribution diagram.

Sanford (2005) believes that there *are* beneficial mutations in some sense, but they are still usually part of an overall erosion of information and are much too rare for genome building and thus are well inside the "no-selection zone." The box size should further be enlarged by all nongenetic factors that affect reproductive probability, and "anything that decreases the signal-to-noise ratio will make proportionately more of a genome's nucleotides utterly un-selectable" (Sanford, 2005, p. 22). Sanford shows a distribution with this and other noise factors (see Figure 3) that has a much wider no-selection zone than the one used by Kimura (see Figure 1). This implies that the chance of the appearance and survival of a beneficial mutation through 500 steps of supposed evolution is infinitesimal and, in fact, impossible (Spetner, 1997). Further, there exists no selection scheme that can reverse the damage caused by continuous mutations (Sanford, 2005). This agreement between the conclusions of a molecular biologist and a geneticist is notable and compelling.

Newer discoveries show that the mutation rate for reproductive cells in humans is at least 100 nucleotide substitutions per person per generation (Sanford, 2005, pp. 34, 70, 120, 125). Even if a good portion of DNA is actually "junk," every person is a mutant carrier. Most "junk" DNA also deteriorates. Although these data are valid only for point mutations, there are many other common mutations, such as frame-shifts, redistributions, duplications, inversions, etc. These nonrandom "mutations," which are discussed further below, also do not support neo-Darwininism.

Sanford asserts that neither natural nor artificial selection (such as human countermeasures from advances in medicine) can eliminate this deterioration. For this reason, reducing the continued accumulation of damaging mutations by denying large segments of the population worldwide the right to bear children is both impracticable and socially unacceptable.

Mutations and natural selection, Spetner's calculations on species formation, and Sanford's mutational deterioration of the genome are discussed in Larssen (2007).

Nonrandom "Mutations"

Genetic Changes

Neither macro- nor micromutations can be the driving force of evolution, and natural selection is impotent without them. What, then, is the cause of variation within the species? Part of the answer is nonrandom genetic changes. These changes are still considered by some to be "mutations," although evolutionary theory is based on randomness. Nonrandom "genetic rearrangements appear to be just as normal in the cell as cell division, although they do not occur often. They are affected by specific enzymes, which the cell synthesizes precisely for that purpose" (Spetner, 1997).

Genetic recombinations show evidence of having been initiated by the en-

vironment (climate, temperature, food) to achieve adaptation by mechanisms such as epigenetics. The possibility of adaptation to the environment already exists in the genome. An example of these variations is the long and short beaks of the Galapagos Islands finches, which Spetner discusses in some detail (Spetner, 1997, pp. 202–205). Spetner argues that the diversity of finches (1) could have come from nonrandom variation, (2) could have come from the direct influence of the environment, and (3) alternatively, could have happened through a built-in genetic switch triggered by the environment.

Another example is the ratio of light to dark peppered moths on the dark tree trunks caused by pollution during the industrial revolution in England, which was reversed when the tree trunks later became lighter due to pollution control. Light and dark moths are always present at some level, indicating that the difference exists in the moths' wild type gene pool. Evolutionary literature often erroneously describes peppered moths as normally sitting on tree trunks, when in fact they typically rest high up in the canopies protected by the foliage (Wells, 2002, p. 149). Both finches and peppered moths possess variations within their species or basic type, and they both demonstrate that genetic changes are controlled by processes in the cell.

While random macromutations alter genes, recombinations (including duplications, inversions, deletions, and translocations) only move existing genes around. Recombination is not a simple process, and we do not fully understand how it proceeds as precisely as it usually does. Specific genes exist that affect recombination effectiveness. These genetic rearrangements occur in response to the surroundings and can result in phenotype changes, but not in new species. These nonrandom genetic rearrangements that appear as a response to the surroundings may explain the enormous variation existing within most every species, such as finch beak size differences and in the appearance of dogs. These recombinations cannot contribute to Darwinian evolution because as far as we know they do not increase information and do not occur as a result of randomness.

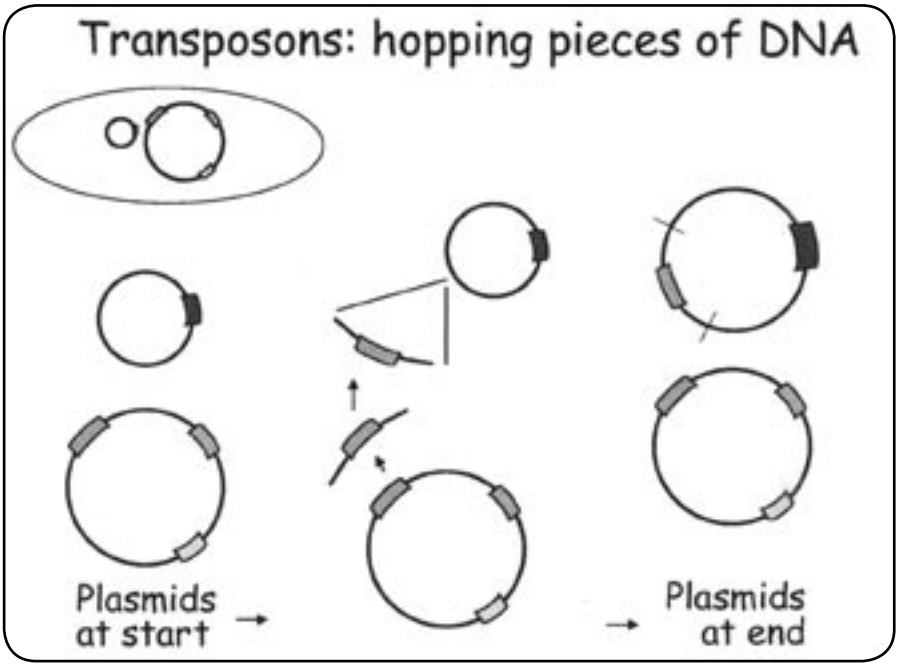

Figure 4. Transposons, containing DNA segments (larger units not shown), can jump from one location to another in a chromosome. Plasmids are small ring forms of DNA containing genes. The genes are "curved parallograms." Plasmid to the left ends up identical in another place on the chromosome to the right. The center illustrates how the jumping occurs (from a lecture to students by Luca Comai in 2001, University of California-Davis).

The inbuilt ability to respond to the environment also may be due to epigenetics, which refers to heritable traits that do not involve changes in the underlying DNA sequence but rather methylation that turns certain genes on or off. Further, some variations may be caused by micromutations, which, as shown above, are too rare or too minor to have any significant evolutionary effect. These also are not neo-Darwinian changes. When the very first of any basic species type was created, it already had the mechanisms and a genetic pool broad enough to produce the variations necessary to adapt to many environmental changes.

Transposons

One means of nonrandom genetic variation is found in the transposition system. DNA segments termed transposons, which consist of as many as several thousand nucleotides, can move to different positions of the genome of a single cell (see Figure 4). Transposons have DNA components that code for the two required enzymes that facilitate their movement. Integration of a transposon into a gene results in its disruption. These "mutations" are under strict cellular control and therefore are not the result of a random process. The more notorious transposons in bacteria have genes that confer resistance to several

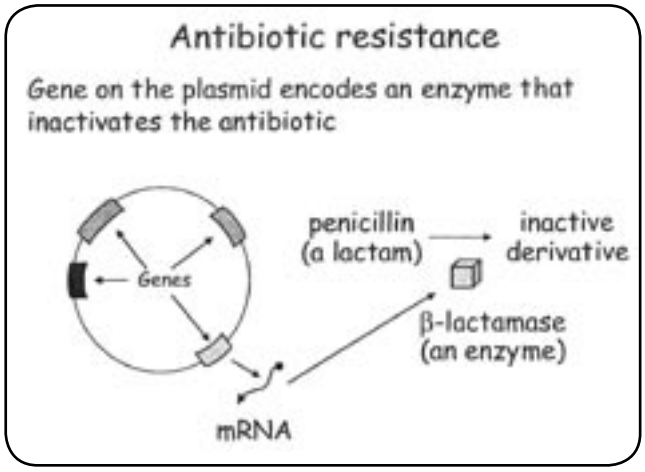

Figure 5. Antibiotioc resistance. One gene in a plasmid, through messenger RNA, is coding for an enzyme, beta-lactamase, shown as a small cube. The enzyme produces penicillin immunity by making it inactive in the bacterium (from a lecture to students in 2001 by Luca Comai, University of California-Davis).

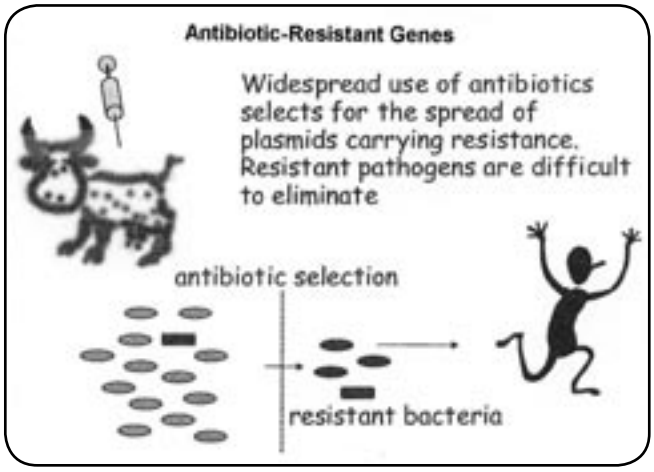

Figure 6. Antibiotic-resistant genes. Transposons contain such genes as seen in the plasmids in Figures 4 and 5 above. After injection of the antibiotic in the animal, the resistance-carrying plasmids, which are naturally found in bacteria and are inherited, are spreading among bacteria. The bacteria to the left become the resistant bacteria shown on the right. The bacteria causing illness are difficult to eliminate, illustrated by the reluctant black figure (from a lecture to students in 2001 by Luca Comai, University of California-Davis).

types of antibiotics. These transposons can be quite large—the transposon that carries resistance against ampicillin, streptomycin, and sulfanilamide contains around 20,000 nucleotides.

Plasmids, small ring-shaped extrachromosomal DNA naturally existing in all bacteria (see Figure 5), copy themselves without external control. They can carry transposons and are inherited by the host cell's progeny. Plasmids are passed between bacterial cells during conjugation and transposon-carried genes can move from a plasmid of one bacterium into the genomic DNA of another bacterium (Spetner, 1997, p. 187). Aside from spreading antibiotic resistance, the normal functions of transposons are not fully understood, but we do know that the resistance they provide does not occur as a result of evolution (see Figure 6). On the experiments with bacteria, Spetner writes that if

> adaptive mutations are stimulated by the environment, they contradict the basic dogma of neo-Darwinism. …that mutations are random, and the kind of mutations that occur are independent of the environment. If mutations are…non-random (and/or) the environment can stimulate adaptive mutations, the paradigm of Darwinian evolution, which has dominated the biological sciences for close to 150 years, must be replaced (Spetner 1997, p. 190).

A comprehensive review of genetic transposition has been published by Bergman (2001). He describes the discovery of transposons, their complicated mechanisms and functions, their types, movements, molecular biology, and significance for creation. In agreement with Spetner, Bergman refers to experiments showing that the movement and placement of transposons are regulated by enzymes and elaborate control systems in the cell, negating the randomness demanded by neo-Darwinism. He concludes that the "extremely complex transposition mechanism could not exist without simultaneous presence of all its main parts, supporting the concept of irreducible complexity" (Bergman, 2001, p. 145).

Selection

Natural Selection Is Not a Selective Force

Organisms with mutations regarded as beneficial are presumed to be "chosen" by the process of natural selection. Natural selection is a descriptive term meaning that some members of an organism's offspring are more likely to survive in the struggle for existence. Unaware of mutations and the laws of genetics, natural selection was the only "mechanism" Darwin could use to explain how evolution occurred. His overemphasis on selection has been maintained by most in the evolutionary community ever since.

French zoologist Pierre-Paul Grassé, a former president of the French Academy of Sciences, rejects both of the assumed mechanisms of evolution, random mutations and natural selection. He asserts that the role of natural selection in evolution is not based on a single fact: "Guided by the overwhelming selection, randomness becomes some type of providence which, under cover of atheism, is not mentioned but is worshiped in secrecy" (Grassé 1973, translated 1977, p. 107). In a review of the original French edition, the Russian geneticist Dobzhansky says of Grassé's book:

> [It] is a frontal attack on all kinds of "Darwinism." Its purpose is "to destroy the myth of evolution, as a simple, understood, and explained phenomenon," and to show that evolution is a mystery about which little is, and perhaps can be, known. Now one can disagree with Grassé but not ignore him. He is the most distinguished of French zoologists, the editor of the 28 volumes of Traité de Zoologie, author of numerous original investigations, and ex-president of the Academie des Sciences. His knowledge of the living world is encyclopedic (Dobzhansky, 1975).

For the purpose of this analysis, let us assume that no new mutations exist in the zygote and only the genetic laws of inheritance operate. If the descendants share only the parents' characteristics and nothing new exists on which selection can work, evolution is effectively dead. Natural selection depends on the existence of mutations: no mutations, no natural selection, and no evolution.

Macromutations are destructive, and beneficial micromutations (such as copying errors) are too rare to be able to furnish anything on which selection can work. The assumption in the above statement is therefore immaterial and unnecessary. Accordingly, there is no evolution!

Some leading evolutionists openly admit that, in practice, the theory of natural selection is a tautology, a way to say the same thing twice. The theory predicts that the most viable organisms will produce the most offspring, and the most viable organisms are defined as those that will produce the most offspring. Harvard University geneticist Richard Lewontin opines:

> Evolution cannot be described as an adaptive process, because all organisms are already adapted. Natural selection is functioning essentially to make it possible for organisms to maintain their status of adaptation instead of improving it. Natural selection does not seem to improve the chance of the species to survive in the long run, but simply makes it possible for them to trace, or keep up with the surroundings, that constantly change (Lewontin, 1978, pp. 213).

The famous founder of the strictly methodical research on heredity, Danish professor and physiologist W. L. Johannsen, concludes from his crucial early experiments on hens:

> What happens in natural or artificial selection is merely that a fixed life form steps forward from its hiding place in the mixture. Selection is therefore not able to produce anything new. It can only select something that already exists. ... It is quite evident that genetics has removed the foundation from the Darwinian selection theory (Johannsen, 1915, p. 169 as quoted in a book by professor and theologian I.P. Seierstad, 1946, pp. 64–65).

Johannsen uses "mixture" instead of "gene pool" because he was not familiar then with later developments about what "mixture" really meant. Nonetheless, his conclusion is still valid today.

Can Selection Stop Genomic Deterioration?

Sanford determined that the human genome deteriorates due to mutations. Mutations are either near neutral or deleterious, leading to the loss of genetic information that in time will result in reduced adaptation. This decline in adaptation is assumed to occur at the rate of 1–2 percent per generation. A steadily increasing adaptation loss will lead to dramatic degeneration of the human race within around 300 generations. Although medical and technical advances are still increasing our life span, "human geneticists would probably all agree that eventually selection must be increased if we are to stop genetic degeneration" (Sanford, 2005 p. 45). Conversely, Sanford concludes that no form of selection can ultimately stop genetic degeneration, only slow it down (Sanford, 2005, p. 117).

The question is, how much selection is required to stop the degeneration, and can it be stopped altogether? A small, closely-knit and highly specialized group of population geneticists tied to the primary axiom ideology have analyzed what selection can and cannot do. These population geneticists have effectively demonstrated the limitations of natural selection, with some even talking about a "mutation meltdown" (Sanford, 2005, p. 115; Lynch et al., 1995a).

Lynch defines mutational meltdown as when "the population size declines, random genetic drift becomes a more significant evolutionary force and the rate of accumulation of deleterious mutations increases, causing further decline in population size. We refer to this extinction phenomenon, which can be quite rapid, as a mutational meltdown" (Lynch, 1996, p. 488; see also Lynch et al., 1995a, p. 1069).

The Lynch et al. (1995a) article deals primarily with asexual populations but one illustration (Figure 6a, p. 1075) deals with a sexual population. When the mean time to extinction is plotted against the reproductive rate, the mean extinction time is short only for very low populations, from 8 to over 32,000 generations for populations of only 4

to 16 individuals. Conversely, Sanford (2005) deals with higher organisms and with the largest human population that can be considered, over 6 billion people (see below).

Lynch et al. (1995a) present results based on computer simulations supported by analytical approximations. For higher organisms they use a genomic mutation rate of one per gamete per generation (Lynch et al., 1995a, p.1067) and one per individual per generation (p. 1068). They arrive by this process at very long mean extinction times as a function of reproductive rate for sexual populations at various population sizes (p. 1975, Figure 6a), even for populations of 16 individuals and smaller. However, they state that credible arguments have been made that the mutation rate may actually be greater than one, in which case the mean extinction time that they have reported to be very long "may be vastly overestimated," and "it appears that sexual populations as large as 1000 individuals.... are potential victims of the mutational meltdown" (Lynch et al., 1995a, p. 1078).

Neel et al. (1986) show that the deleterious mutation rate must be much higher than one per person per generation, in which case mean extinction time is substantially decreased. Sanford (2005) quotes Lynch as saying, "Our results provide no evidence for the existence of a threshold population size beyond which a population is completely invulnerable to a mutational meltdown" (Sanford, 2005, p. 173, quoting Lynch et al. 1995b, p. 510).

Mutation rates have been investigated by a number of other geneticists. Drake et al. (1998) report mutation rates in higher eukaryotes are roughly 0.1–100 per genome per sexual generation, and for humans 1.6 per effective genome per sexual generation. Nachman and Crowell (2000) report that the genomic deleterious mutation rate for humans is at least 3 per genome per generation, concluding that synergistic epistasis among harmful mutations may be common. They also indicate that male mutation rate is 4 times the female mutation rate. Kumar and Subramanian (2002) conducted computational analysis of several thousand genes from major groups of placental mammals to determine mutation rate differences among genes in a genome and among various mammalian lineages. They find that the average mammalian genome mutation rate of 2.2×10^{-9} per base pair per year is constant and largely similar among genes and also similar among lineages with vastly different generation lengths and physiological attributes (Kumar and Subramanian, 2002., p. 803). This is at variance with current thought that mutation rates widely vary among genes within a genome and among lineages in mammals.

Can this genomic degeneration problem be solved? Several possibilities exist, but they all fail. For example, if we could select against all "mutations," no life-form could reproduce, causing rapid extinction. Every life-form inherits a large number of deleterious mutations and so collectively carries billions of near-neutral or harmful mutations. From the estimated total of 600 million new mutations that enter the entire human gene pool in our own generation, it is estimated that in a 6-billion world population as many as one third, or 2 billion people, would be precluded from having children (Sanford, 2005, p. 71). This would still leave 400 billion new mutations to burden the next generation. Even if we assume that two thirds of the remaining mutations are completely neutral, about 133 billion harmful mutations would be added to the population in the next generation. If we doubled the intensity of selection, we still would have 67 billion damaging mutations for the next generation. The cost of selection sets distinct limits to how many mutations can be eliminated per generation and for this reason "*mutations will continue to accumulate, and the species must degenerate!*" (Sanford, 2005, p. 72).

Crow agrees that because mutations are accumulating, population fitness is declining, and due to chance some individuals would experience more mutations than others (Crow, 1997). If more mutations in the population could be eliminated at less "cost" (by focusing selection on the individuals with the most mutations), the number of mutations per person might be stabilized, and the fitness decline of the population would taper off. However, of primary importance is not who has the most mutations, but who has the most damaging mutations. Crow's model answers only the former and ignores the latter. The fitness impact of different nucleotides can vary by many orders of magnitude, and one minor mutation can overshadow the effects of a million near-neutral mutations. Crow's model has limited significance in the real world because it is based on several unreasonable assumptions (Sanford, 2005, pp. 106–108 and Figure 10a, p.112). Crow's model is unrealistic because

> [it] is designed to make the problem of mutation accumulation "go away". It assumes all mutations have equal value, are all individually very subtle, yet none are so subtle as to be "nearly neutral", that all selection is based upon "mutation count."... None of these assumptions are even remotely reasonable. Even though all these assumptions are artificial and unreasonable, the numerical simulation still shows severe mutation accumulation (Sanford, 2005, p. 112).

Walter ReMine (2005) has developed software for doing numerical simulations of Crow's model (represented by Sanford's [2005] Figure 10a, p. 112). This shows a fitness curve *versus* generations (Sanford, 2005, Figure 10b, p. 113), where fitness clearly declines to zero after roughly 300 generations. If the

Lynch et al. (1995a) reservation about "vast overestimates" is not considered, this finding deviates sharply from the long extinction times result (Lynch et al., 1995a). If it is considered, there is less disagreement between Lynch et al. (1995a) and Sanford (2005).

Without intervention, the extinction of the human genome appears as certain as the extinction of stars and the death of organisms. We as individuals will not only die as a result of the deterioration of our own genome, but without outside intervention the human race also will eventually die for the same reason. This agrees with current speculation by some evolutionists that over 99 percent of all species that have existed in the past have gone extinct.

In the previous section it was concluded that there is no evolutionary process. In this section Sanford's (2005) findings lead to the conclusion that living organisms exhibit a process of devolution that he calls genetic entropy.

Plotting the human life spans of the descendants of Noah against the number of centuries after Noah when a given individual was born shows a dramatic reduction in life spans, which has a strong likeness with a biologic decay curve (see Figure 7). The data reveal an exponential curve that fits well with a correlation coefficient of 0.90. The Biblical data are in very good agreement with modern theories of genomic degeneration caused by mutation accumulation (Sanford, 2005, pp. 148–149, Figure 14, p. 152). The curve is very similar to theoretical curves shown by Sanford that reflects genomic degeneration (Sanford, 2005, Figure 4, p. 65 and Figure 10b, p. 113).

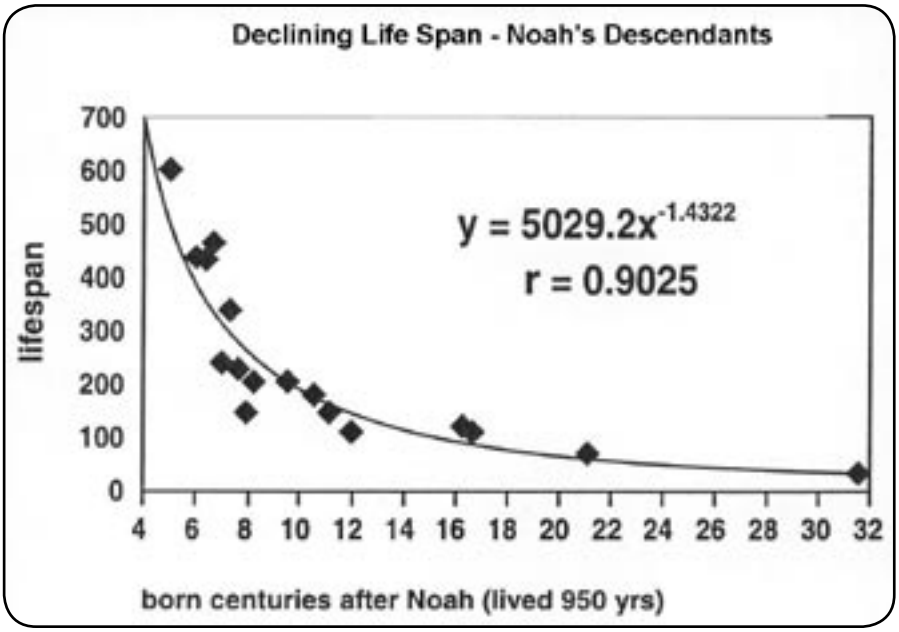

Figure 7. Human life spans in early history (from Sanford, 2005). When Biblical life spans are plotted against time for generations after Noah, a dramatic decline in life expentancy is shown. The curve shows a strong evidence of biological decay. It reveals an exponential curve following the formula $y = 5029.2x^{-1.43}$. The curve is consistent with the concept of genomic degeneration caused by mutation accumulation.

Summary

Random mutations combined with natural selection is the central dogma of how the neo-Darwinian theory works. This review shows why macromutations cannot be the driving force of evolution, and calculations show that micromutation copying errors are far too rare and cannot be the basis for evolving new species. Natural selection's role in evolution depends on the existence of mutations, and natural selection is inadequate to halt genomic degeneration.

How the complexity and the incredible chemical feats of the living cell came into existence through random mutations and natural selection, referred to as the primary axiom or the central dogma, defies explanation and is considered, as shown, an impossibility. The nearly neutral mutations within the "no-selection zone" of the mutation distribution curve creates major problems for evolutionary theory due to their increasing accumulation. The adaptation loss at 1–2% per generation will lead to dramatic degeneration of the human race within around 300 generations, and this genomic deterioration cannot be rescued by selection.

The combined impact of the inability of mutations to produce new species and selection's inability to halt genomic deterioration demonstrates the failure of evolution's central dogma to explain the complexity that we see in the realms of molecular, cellular, and systems biology, and strikes a devastating blow to neo-Darwinian evolution theory.

These facts are a sufficient blow to motivate a paradigm shift. The well-known Swedish evolutionary biologist, the late Søren Løvtrup of Umeå University, in his book *Darwinism: The Refutation of a Myth*, evidently perceived such an outcome when he wrote, "I believe that one day the Darwinian myth will be ranked the greatest deceit in the history of science" (Løvtrup, 1987, p. 422).

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