Statistical and Philosophical Notions of Randomness in Creation Biology

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Abstract

Mutations that occur in the absence of selection have often been cited as evidence that mutations are philosophically random—that is, that they do not follow a predetermined pattern but occur haphazardly. Mutation in absence of selection, however, may be a part of an adaptation process, specifically engineered for maintaining variability in organisms as a hedge against future environmental changes. A statistically random distribution of a constrained set of semi-specific (i.e. nonphilosophically random) mutations in populations would enable them to preserve a fairly constant percentage of mutants, which would foster survival in adverse conditions.

Introduction

Much is often made, both in creationary and evolutionary circles, of the notion that genetic mutations are, at least in general, "random." There are multiple definitions, however, of the word "random," and knowing which definition is being used can help analyze both the veracity of the claim and how the claim impacts our understanding of biology if the claim is true. Specifically, the Lederberg and Luria-Delbrück experiments are often considered evidence that mutations are random in the sense that they are haphazard. While these experiments do demonstrate the randomness of many mutations for certain definitions of randomness, another possible model is that some of the mutations are deliberate in order to maximize the population's genetic diversity (Anderson, 2003).

Other types of mutations exist that may fall outside of the "random" designation (Purdom and Anderson, 2008). These mutations, while extremely interesting, are not within the scope of this paper. Rather, in this paper I will consider only those mutations that occur in the absence of any selective pressure and use these mutations as a springboard for thinking about concepts of randomness as applied to Creation theory.

Definitions of Randomness

In evolutionary biology there are three definitions of chance or randomness that are offered—noncorrelation, statistical randomness, and philosophical randomness. Much confusion in both the scientific and popular literature arises from treating all varieties of randomness the same. At their core, most notions of randomness involve some sense of unpredictability from at least one vantage point (Eagle, 2005). This is somewhat of a minor point, however, compared to the variety of notions for which randomness is used. Clarifying the concept of randomness will aid not only in communicating the properties of systems clearly, but also in discovering new avenues of design in biology.

Noncorrelation is the simplest notion of randomness, as all that it implies is that two variables have no relationship to each other. This does not

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Accepted for publication June 11, 2008

imply anything about the operation of the variables themselves – they may be perfectly nonrandom on their own. As long as they vary independently of each other, they can be considered noncorrelative. Also, noncorrelation can rarely be generalized – if a variable is noncorrelated with another variable, that does not prevent it from being correlated with another similar but different variable. In current mutation theory, mutations are said to not be correlated with the fitness needs of the organism that they occur in (e.g. Templeton, 2006, pg. 3).

Another notion of randomness is what I call "philosophical randomness." This is the notion that a given event is happenstance. That is, a given event occurred outside the plan or guidance of any overarching control system. These events may be deterministic, but are fundamentally uncontrolled. When using this concept as applied to biology, Eble called this the "evolutionary notion of chance" and applied it to large-scale events in natural history as well as to the mutations on which natural selection acts (Eble, 1999, p. 75).

The last notion of randomness is what I call "statistical randomness." This is the notion that a given set of events, whether deterministic or indeterministic, follows a specific set of statistical properties. Statistical randomness was originally specified by Von Mises, who stated that a process is random if all infinite subsets of the process's outputs that can be specified using recursive algorithms are random (see Eagle, 2005, p. 756). Events that have a low likelihood of occurring and are statistically random are fairly easy to detect by counting those events in groups. If the mean and the variance of the counted groups are roughly equal, it provides good evidence that the process is statistically random (Luria and Delbrück, 1943).

Noncorrelation and statistical randomness, however, are quite distinct from philosophical randomness. Noncorrelation does not imply anything about whether or not a given output was planned, and statistical randomness is actually harnessed in many applications to produce a planned result. In philosophical randomness, the output event is outside of a control. But in many cases programmers and engineers put statistical randomness under specific controls to produce specific outputs. Programmers use statistical randomness to minimize worst-case runtime performance in a variety of settings. As an example, some algorithms perform poorly on specific input orders, and therefore by randomizing the input order as the first step, the average computation time for the algorithm will be maintained even if the input ordering is always the worst case. Slot machine designers use statistical randomness to guarantee that (a) the result of any individual pull of the slot machine will not be determinable externally, and (b) the slot machine owner will give out less money than he received. Statistical randomness utilized within controlled systems is fundamentally different from philosophical randomness, in which the outcomes are not under any system's control.

There are also other notions of randomness. For example, algorithmic randomness is determined by the compressibility of a string of results (e.g. Eagle, 2005, p. 769). It is safe to say that there are many varieties of randomness available, but here we will focus on noncorrelation, statistical randomness, and philosophical randomness. Specifically, we will look at the Luria-Delbrück and Lederberg experiments and see what sorts of randomness they imply.

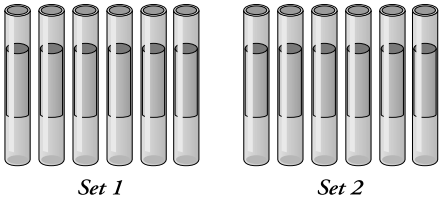
The Luria-Delbrück Experiment

The Luria and Delbrück experiment (Luria and Delbrück, 1943) is often hailed as definitive proof that mutations are philosophically random—that they are haphazard events, not derived from the "need" of the organism. The test is known as the "fluctuation test." The test (illustrated in Figure 1) works as follows:

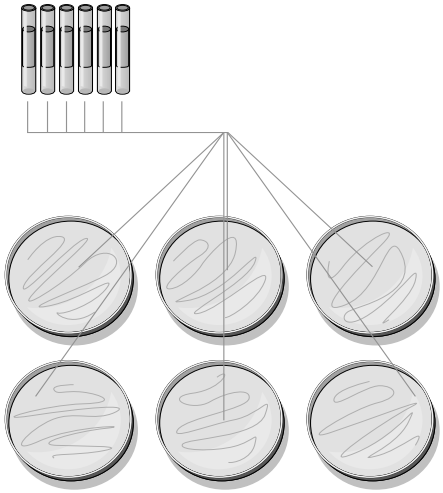
1. Start with a single colony of bacteria.
2. Cultivate the colony in several separate tubes and separate these into two sets.
3. After cultivation, the tubes are separated into two groups.
   a. In the first group, the bacterial populations from each tube are randomly mixed and then spread onto several Petri dishes containing a selection agent, such as a bacteriophage.
   b. In the second group, the population from each tube is spread onto separate Petri dishes (i.e. the populations are not mixed) containing the same selection agent.
4. The number of surviving/resistant colonies on each dish are counted, and the mean and variance for each group is totaled. If the mean and variance are roughly equal, the distribution is considered a Poisson distribution, indicating statistical randomness. The possible results are as follows:
   a. If the first group is not a Poisson distribution, then the experiment was either not conducted correctly or the mutational pattern is not assessable by this experiment.
   b. If the first group is a Poisson distribution and the second group is also a Poisson distribution, then the selection event is the cause of the mutation.
   c. If the first group is a Poisson distribution and the second group is not a Poisson distribution, then the selection event is not the cause of the mutation.

The test of the first (mixed) group tells whether or not the mutation itself is statistically random and if the experiment was set up correctly. If the

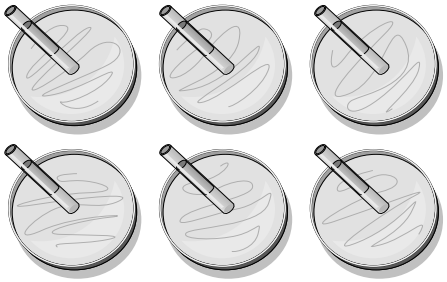
Step 1: Start with a single colony of bacteria



Step 2: Culture it into two sets of tubes and allow them to grow

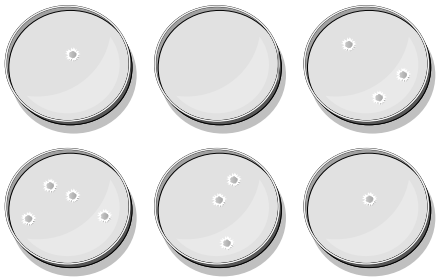


Step 3: ***Set 1:*** For Set 1, mix up all of the cultures and spread it on plates containing a selection agent

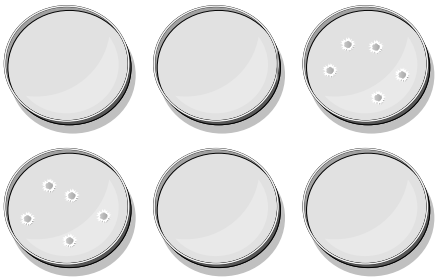


Step 3: ***Set 2:*** For Set 2, spread each culture onto its own plate containing a selection agent

Wait for colonies to form...
Count the colonies that formed on each plate and measure the average number of colonies per plate for each set, and the variance for each set.



Possible Result 1: Poisson: Mean = Variance



Possible Result 2: Non-Poisson: Mean ≠ Variance

Figure 1. In the Luria-Delbrück experiment, a single colony is cultivated in two sets of test tubes (Step 1 and Step 2). In step 3 they are spread across Petri dishes containing a selection agent such as a virus that normally kills the bacteria. The test tubes in Set 1 are all mixed before spreading onto plates, while in Set 2 each plate is spread from exactly one test tube. After waiting for colonies to form, the colonies on each plate are counted. The time to allow for colony formation is dependent on both the selection agent and the bacteria—in the original experiment colonies were counted after 24 and 48 hours. If the mean colony count for a set is roughly equal to its variance, then it is likely that set follows an underlying Poisson distribution. The experiment will give a Poisson distribution for Set 1 and a non-Poisson distribution for Set 2 if the mutation is statistically random and not correlated with the selection agent.

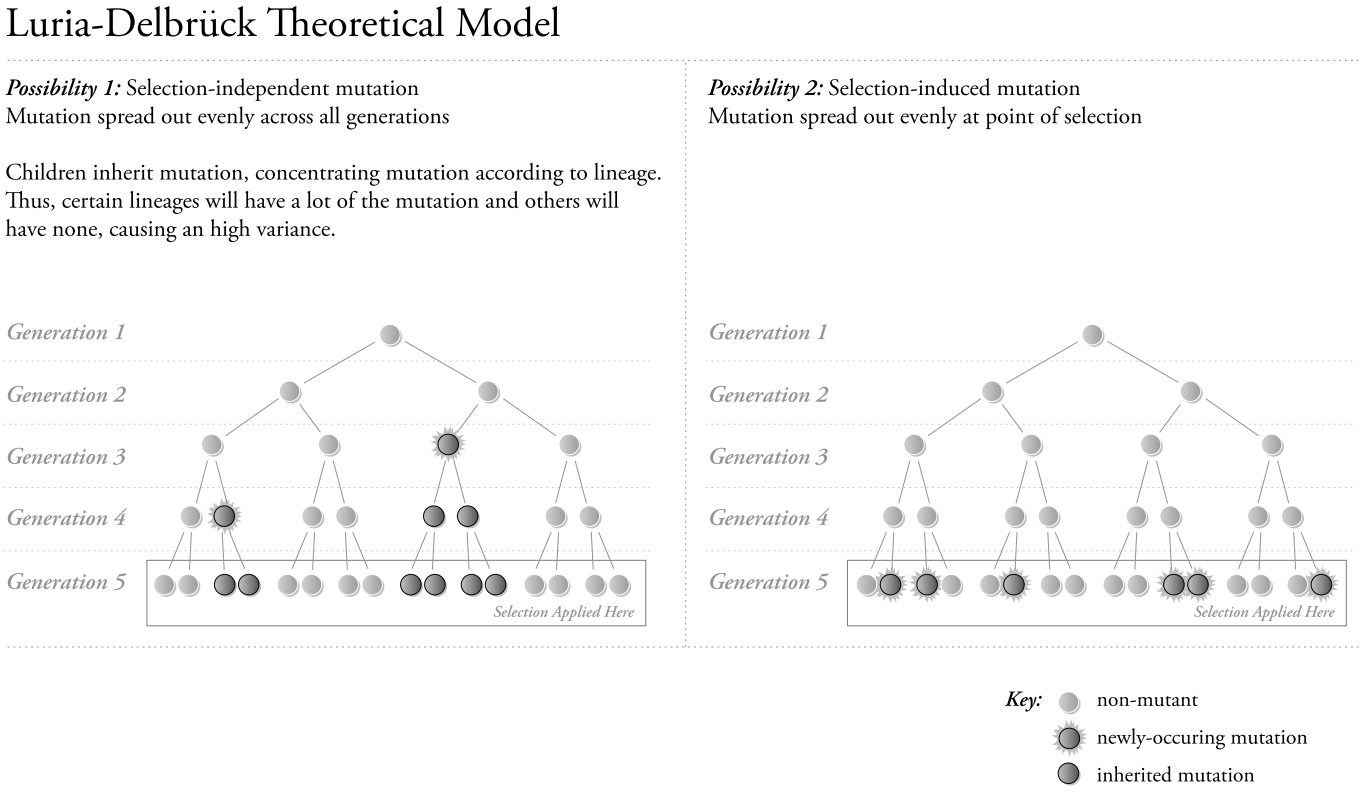


Figure 2. This is a model showing why the distributions are different for selection-induced and non-selection-induced mutations. The light gray dots represent bacteria that do not have the mutation. Black dots represent bacteria that do have the mutation. In this graph, selection is applied at Generation 5. If the mutation was statistically random and noncorrelated with selection, then the mutation could have occurred at any point in the organism's history. The onset of the mutation is not only statistically random but also statistically random in every generation. Since the mutation is genetic, the descendants of the mutated cell will also have the mutation. This gives cells from that lineage a much larger representation in the final population, which will create a large variance. The other possibility is that the mutations are the result of selection. In that case, the lineage will not affect the outcome of the mutation—they will be statistically random at the point of selection, and nonexistent otherwise.

mutation is statistically random, then we can use the second group to tell if the selection event was the cause of the mutation.

In the second group, the bacteria were grown in separate tubes, giving each tube an independent lineage. If the mutation happened because of the selection event, then that would mean that the specific lineage of a cell would not matter in determining survival—it would be the selection event, not the lineage, that was causing the mutation. If separating them by lineage does dramatically affect the distribution (giving it a high variance), then lineage is the primary factor determining survival, and thus the mutation must have occurred beforehand (i.e., independently of the selection event) and been passed on genetically. Figure 2 depicts these two possibilities graphically.

This experiment established that, for a large number of mutations, the produced mutations were both statistically random and noncorrelative with the selection event.

The Lederberg Experiment

A different experimental method known as "replica plating" was developed a decade later. It also tests for the noncorrelation of mutation with the selection event but does not prove whether or not the mutation is statistically random (Lederberg and Lederberg, 1952). The Lederberg experiment is also much more intuitive and requires less mathematics to understand. In the Lederberg experiment, a bacterial culture is spread across a Petri dish. Following incubation, individual colonies form on the surface of the plate. Using a special technique called "replica plating," the colonies on the first plate are sampled and copied to a new plate, where they are in the same position as the original.

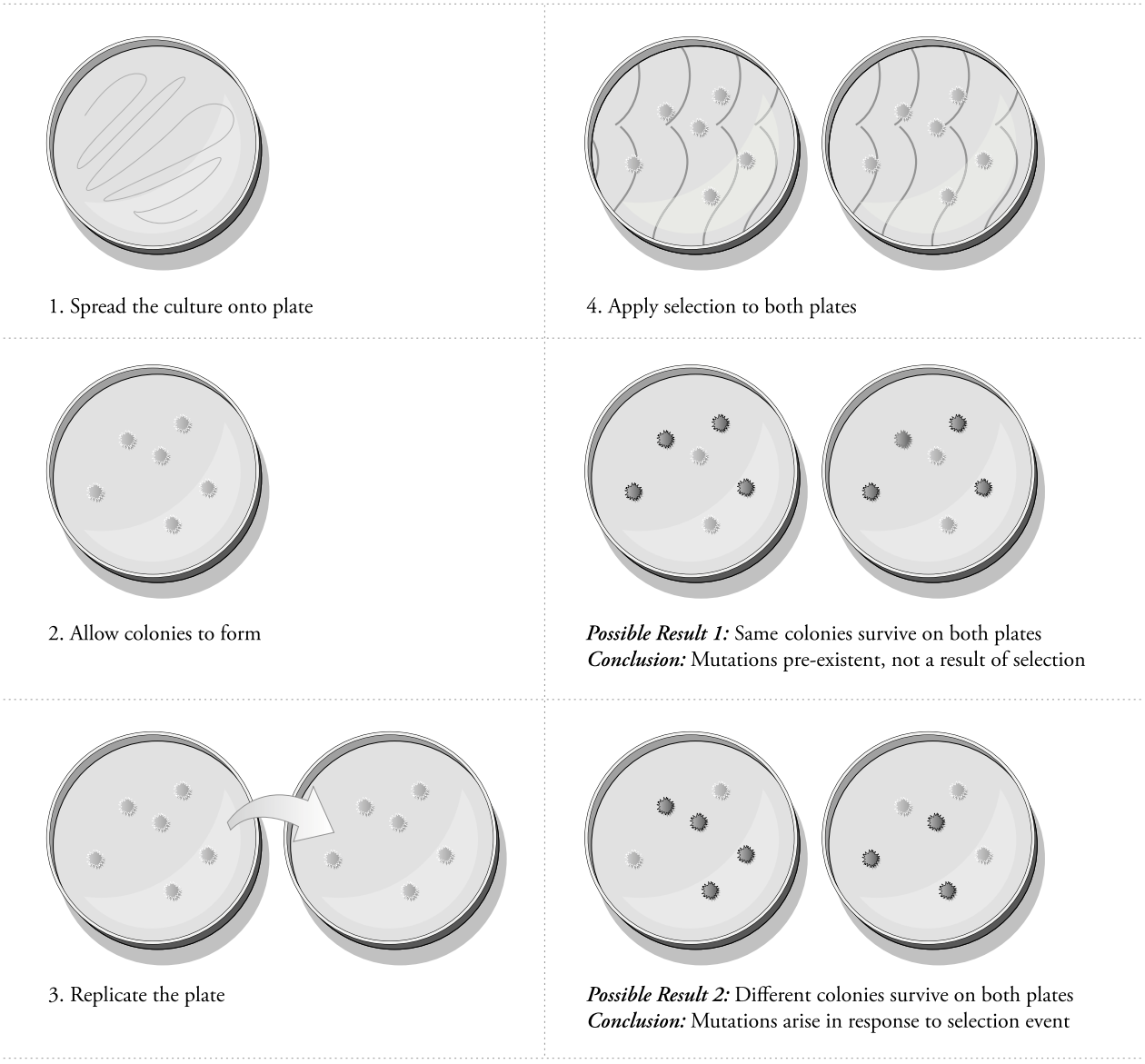


Figure 3. In the Lederberg experiment, a Petri dish is spread with bacteria (Step 1), and individual colonies are form following incubation (Step 2). The Petri dish is then replicated-plated onto another Petri dish in such a way as to preserve the position of the colonies on both plates (Step 3). A selection agent is then applied (the original Lederberg experiment used bacteriophage T-1) to both plates (Step 4). If the selection itself is the cause of the mutation, then there should not be a positional correlation between surviving colonies on each plate. If the mutations were already in the population, however, then the same colonies should survive on both plates.

Then, a selection agent is applied to both plates. If the surviving colonies are in the same location on both plates, then that means that the mutation had occurred prior to the selection event. If the surviving colonies are in different locations on each plate, then that means that the mutation occurred after (and because of) the selection event (see also Figure 3).

Lederberg, Luria-Delbrück, Philosophical Randomness, and Future Fitness

The result of these experiments is that a large class of mutations is both statisti-

cally random and noncorrelated with selection events. For many researchers, this has indicated that the mutation process is, in fact, philosophically random. As stated in the Berkeley evolution web site, "In this respect, mutations are random—whether a particular mutation happens or not is generally unrelated to how useful that mutation would be" (Caldwell et al., 2007). This is a clear articulation of the concept of philosophical randomness, which is directly attributed to such experiments. Another popular articulation of it appeared on Talk Origins' Chance FAQ, where the author stated, "The changes that get encoded in genes occur with no forethought to the eventual needs of the organism (or the species) that carries those genes" (Wilkins, 1997). One more statement comes from the more technical literature: "Although many environmental agents can influence the rate and type of mutation, one of the central tenets of Darwinian evolution is that mutations are random with respect to the needs of the organism in coping with its environment" (Templeton, 2006, p. 3). Again, Lederberg is cited in support.

The common conclusion of philosophical randomness from the Luria-Delbrück and Lederberg experiments is based upon a false dichotomy. The reasoning that mutation in the absence of selection indicates philosophical randomness is unwarranted if design possibilities are included. Specifically, statistically random mutation in the absence of selection makes sense in a design paradigm as a process to maximize future fitness.

The two types of randomness discussed in connection with the Lederberg and Luria-Delbrück experiments are noncorrelation and statistical randomness. Statistical randomness is characterized by processes that lead to a constant percentage of given events no matter what the population size or the method of segregating the population. Therefore, using statistically random mutations, a population of organisms could preserve a steady supply of alternative metabolic configurations in the population in order to prevent selection events from wiping out the entire population. If a statistically random process is used, it guarantees that a certain percentage of mutants will exist in the population. This is like stock market investors who maintain a given percentage of their financial holdings in alternative types of investments (gold and bonds for instance) just in case market conditions cause the primary investments to fail. In the same way, the reason many of these mutations are noncorrelated with the selection event, and thus the organism's *current* fitness, may be because they are instead looking forward to the whole population's *future* fitness.

Like alternative investments, each possible configuration has trade-offs. The alternative configurations for organisms are not as fit as the primary one under standard conditions, but maintaining a certain percentage of organisms in the population of mutant configurations enables them to survive novel or extreme circumstances in which their primary configuration fails them (Anderson, 2003; 2005; Anderson and Purdom, 2008). Thus, with design, we have the additional option of understanding mutation as being teleological, even when it is not related to any present need.

Production of Variability in the Genome

If a population lacks variability, then it is extremely susceptible to selection events. If an organism were engineered to produce variability, however, it would then be much less susceptible to adverse selection. Because of experiments like the fluctuation test and replica plating, it has often been assumed that specific variation-producing mechanisms do not exist, or if they do exist, they are a minor phenomenon in origins. Many recent studies, however, point to existing mechanisms that produce biologically sensible mutations and do not merely select them. This may occur either as a response to selection or independent of selection (Ashcraft, 2004; Caporale, 1999; Caporale, 2000; Henderson et al., 1999; Moxon, 1997; and Rogozin et al., 2005 are just a few examples). If the set of mutations is restricted to those that are biologically meaningful, or at least those likely to be biologically meaningful, then statistical randomization within this set will produce mutants that are biased toward current or future usefulness.

This does not mean that all mutations conform to this pattern—some or many mutations may indeed be philosophically random—but that creation biologists should at least consider the possibility of forward-looking mutations when analyzing mutational patterns.

Phenotypic Versus Genotypic Randomness

When determining whether or not a mutation is philosophically random, one factor that often is not fully considered is the difference between analyzing genotypes and analyzing phenotypes. Many evolutionary experiments are evaluated only on the basis of phenotypes, the genotype being determined only indirectly by the behavior of the phenotype across generations. But when looking at phenotypes, the only possibilities that can be considered are those that can be observed. When examining the genetic makeup of an organism, however, we have access not only to the results of the organism's coding but also to the code itself.

Think of a sentence such as "Eat an orange." If we were to make a statistically random change with uniform probability at the level of the lettering (i.e., the coding), the result would not likely make much sense at all. Performing a random replacement via a simple

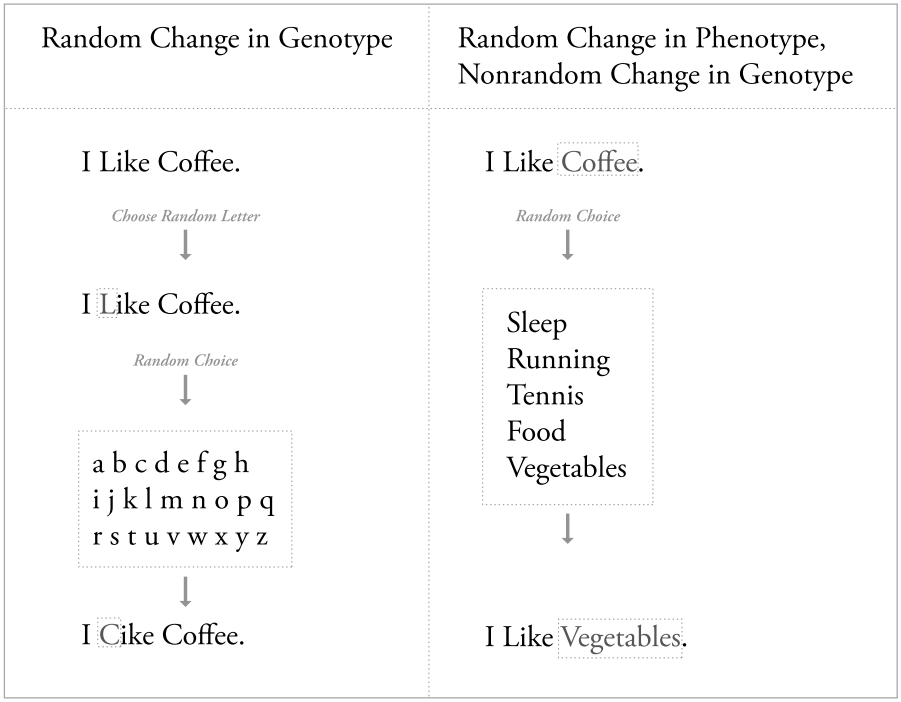


Figure 4. This figure demonstrates what is meant by random phenotype changes being different than random genotype changes. If the random change is on the level of the genotype (as would be expected with "copying errors"), the change itself has no relation to the final meaning of the sentence. In this example we changed one letter at random without any respect to the final meaning of the sentence. The end result made the sentence nonsensical. The second example is a random change in phenotype. In this case, all of the possible choices are at least sensible, whether or not they are true. It is still a genotypic change and is still statistically random, but the randomness has been restricted to possibilities that are at least potentially sensible. In this example we used whole words as the unit of changes, but it could also be done using a unit as small as morphemes (the smallest semantically-meaningful unit) or as large as can be imagined. The main point is that it is still a random choice, but the possible alternatives have been restricted much farther than an arbitrary genotypic change would allow.

computer program produced "Eat an ortnge," which is nonsensical (i.e., it does not produce a valid phenotype). However, if instead we constrained the lettering (genotypic) changes to those that make sense within the context, or those that at least have a chance of making sense within the context, we have much better results. For example, if I chose to replace "orange" with a statistically random member of the set ("apple," "pear," "tomato," "radish"), then all of the resulting phrases (genotypes) would at least make grammatical sense—i.e., we would get a viable phenotype at the end (see Figure 4 for a side-by-side comparison of these ideas).

Therefore, if an evolutionary study were looking at phenotypes alone, such a study would not be able to see the amount of constraints on the underlying genotype that were involved. What might appear to be an unrestrained change may in fact be severely constrained in its production (not just selection) by the cell itself. Merely looking at the phenotypes cannot tell us whether or not the underlying code is changing in a way that is constrained toward biological sense or if the underlying code is changing in an unconstrained way. Philosophical randomness, also called the "evolutionary notion of chance," would require that the mutations occur outside of biological controls. If we instead find that the biology of cells is restricting the mutations in large part to sensible areas, then even if the changes are statistically randomized within these areas, it is a constrained randomness.

What is observed is that genomes do not exhibit statistical randomness across all configuration possibilities, but instead the set of possibilities is restricted by mechanisms within the genome that focus mutations in "hot spots." Two possibilities then occur. Either these regions are hot spots simply because of arbitrary structural occurrences, or they are hot spots for the purpose of constraining mutation to functionally useful areas. One can differentiate between these two possibilities by whether or not the hot spots are more likely to contain beneficial mutations than the rest of the genome (Dembski, 2001; Dembski, 2005).

For instance, in criticizing Behe, Coyne has pointed out that

> what we do not mean by "random" is that all genes are equally likely to mutate (some are more mutable than others) or that all mutations are equally likely (some types of DNA change are more common than others). It is more accurate, then, to call mutations "indifferent" rather than "random": the chance of a mutation happening is indifferent to whether it would be helpful or harmful (Coyne, 2007).

What Coyne has said is that the existence of hot spots in a genome does not invalidate the hypothesis that mutations are philosophically random. In order to maintain that they are "indifferent," however, these hotspots should not be highly correlated with the usefulness of

a change compared to a random one. In this instance, by random I mean a statistically random one with a uniform probability distribution across the genome. If changes in these hot spots are, in fact, more biased toward beneficial or at least biologically sensible changes than arbitrary changes in the rest of the genome, then the idea that they are "indifferent" and merely the result of "errors in DNA replication" falls short.

Rogozin et al. (2005) give a helpful overview of some properties of genomic hot spots, but fall short of categorizing the hot spots according to their occurrence in functionally useful areas. Caporale (1999) is much more concerned with the beneficial nature of what she calls "molecular strategies" and says that genomes

> have evolved mechanisms that generate multiple sequence changes in a single step, allowing them to bypass unselected neutral, and negatively selected, sequences that may lie on point mutation pathways between the current sequence and a more optimal sequence (p. 2).

Thus, according to Caporale, there are many genomic potentials that will never even be attempted because of the molecular strategies in place. It is not that they are selected against, rather the genome focuses its own mutations in potentially beneficial directions while skipping the likely dead ends.

This sort of phenomenon already has been shown in certain cases of somatic hypermutation, which is a related but different type of mutation in which mutations are accelerated in response to specific environmental stresses. For instance, in mutating the immunoglobulin genes for refining binding affinity, the mutations are constrained almost entirely to the V region of the immunoglobulin gene, which is the most likely place for a beneficial mutation to occur (Wagner et al., 1995, Papavasiliou and Schatz 2002). The challenge for future creationist research will be to show quantitatively that hot spots for spontaneous mutations are also more likely than random to produce biologically sensible changes that may be useful in alternate environments.

Conclusion

Randomness is not a singular concept but instead a multiplicity of concepts. Only certain varieties of randomness are at odds with teleology, and it is important for creation biologists to be familiar with how the different types of randomness can be utilized in evaluating biological phenomena. Statistically random mutation, even when noncorrelated with a selection event, is not sufficient evidence for philosophical randomness. Instead, such mutational processes may also suggest that population microevolution "plans ahead" to prepare for future contingencies. The evidence in the current biochemical literature gives support for the idea that the genome is biased toward generating biologically "sensible" mutations. This in turn indicates that such processes are loaded with information about the search space that could not have been obtained solely through stochastic events, and that their ultimate origin "transcends chance and necessity" (Dembski 2005).

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Book Review

In the Name of Education

by Jonas E. Alexis

Xulon Press, Longwood, FL, www.xulonpress.com, 438 pages, $22.00.

Author Jonas Alexis is a high-school math teacher in Avon Park, FL (p. 29). As an "insider," he has much to say about current public education, and it is not positive. Alexis describes declines in the political, social, moral, educational, and spiritual fabric of the U.S. In particular, he identifies educational failure with the "weird – and detrimental idealogies [and spokesmen that] infiltrate our schools" (back cover). The popular, misguided trends include political correctness, "no child left behind" policies (p. 243), affirmative action (p. 265), evolution, secular humanism, entitlements, slavery reparation (p. 281), multiculturalism (p.305), and many others. Regarding multicultural excesses in the classroom, author Alexis is a native Haitian and knows the issues personally. The only solution that Jonas finds is to bring God back into education (p. 321). That is, we need to return to the Biblical, Christian heritage of American public schools.

The book shows familiarity with thousands of named writers and leaders. These include poets, philosophers, and scientists of the past and present. Useful quotes appear on every page. In preparation for this book, the author describes checking out 50 library books at a time, and the scholarly effort is obvious (p. 407). There are 1770 endnotes and a "select" bibliography list with 360 references.

A second edition of this book (which is underway) may correct some shortcomings. Along with numerous typos, the words "macro-" and "microevolution" are somehow reversed in meaning (p. 234). Also, the ten-page index has little value since the page numbers listed do not match the text. Beyond this, author Alexis has written an important book, and further polishing will add to its value.

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