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BACTERIAL ANTIBIOTIC RESISTANCE & EVOLUTION

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On November 24, 1859, Charles Darwin published *The Origin of Species*, popularizing organic evolution. The science of genetics, of course, was completely unknown at the time, and would not come into its own until approximately forty-one years later. Since around 1900, evolutionists have advocated "neo-Darwinism," as opposed to "classical Darwinism." In classical Darwinian thought, natural selection alone served as the mechanism of evolution. In neo-Darwinian thought, natural selection and genetic mutations work together as evolution's mechanism.

Genetics has played an increasingly important role in evolution, especially in regard to mutations that alter the genetic code within each organism. That code is expressed biochemically in deoxyribonucleic acid (DNA). Mutations are "errors" in DNA replication (see Ayala, 1978, pp. 56-69). It is those errors that cause the genetic change necessary for evolution to occur. In 1957, George Gaylord Simpson wrote: "Mutations are the ultimate raw materials for evolution" (1957, p. 430). Twenty-six years later, nothing had changed when Douglas J. Futuyma remarked:

> By far the most important way in which chance influences evolution is the process of mutation. Mutation is, ultimately, the source of new genetic variations, and without genetic variation there cannot be genetic change. Mutation is therefore necessary for evolution (1983, p. 136).

Mutations can occur in several different ways, and can affect individual genes or entire chromosomes (see Futuyma, p. 136). Further, mutations can be placed, theoretically, into at least three categories: (a) bad; (b) neutral; and (c) good.

Some mutations may produce profound effects. They can alter the structure of a critical protein so drastically that the organism becomes severely distorted and unable to survive. Other mutations may cause changes in the protein that do not affect its function at all. Such mutations are adaptively neutral—i.e., they are neither better nor worse than the original form of the gene. Still other mutations are "decidedly advantageous" (Futuyma, p. 136).

Neither bad nor neutral mutations aid evolution, since the bad ones produce effects that are deleterious (and often lethal), and the neutral ones neither help nor hurt an organism. Neo-Darwinian evolution relies entirely on **good** mutations, since they not only alter the genetic material, but should be (to use Dr. Futuyma's words) "decidedly advantageous." Evolutionary progress, then, is dependent upon nature "selecting" the good mutations, resulting in genetic change that ultimately produces new organisms.

BACTERIA AND RESISTANCE TO ANTIBIOTICS

What does this have to do with the resistance of bacteria to antibiotics? Over the past several years, the medical community has become increasingly concerned over the ability of certain bacteria to develop resistance to antibiotics. Undoubtedly this concern is justified. Antibiotics, which usually are substances produced naturally by certain microorganisms, inhibit the growth of other microorganisms. One of the first antibiotics to be discovered (in 1928) was penicillin, produced by the mold *Penicillium chrysogenum*. Since then, more than a thousand similar substances have been isolated. Most people recognize the tremendous impact antibiotics have had in the battle with pathogenic (disease-causing) organisms. Without antibiotics, the death toll from infections and diseases would be much higher than it is.

Today, however, there is compelling evidence that we are in danger of losing our battle against certain pathogens. Bacteria sometimes develop resistance to even powerful antibiotics. As a result, the number of antibiotics that can be used against certain diseases is dwindling rapidly. Both scientific and popular publications have addressed this issue. The cover story of the March 28, 1994 issue of *Newsweek* was titled, "Antibiotics: The End of Miracle Drugs?" (Begley, 1994). Articles in *Scientific American* (Beardsley, 1994), *Science* (Travis, 1994; Davies, 1994), *Discover* (Caldwell, 1994), and *Natural History* (Smith, 1994), all have called attention to the impact on our lives that bacterial resistance to antibiotics is causing.

The phenomenon of bacterial drug resistance was documented initially around 1952 (see Lederberg and Lederberg, 1952). Interest has increased as fewer antibiotics are effective against pathogens, and as deaths from bacterial infections increase. Scientific concerns are both pragmatic and academic. In the pragmatic sense, those working in medical fields (doctors, nurses, pharmacists, researchers, etc.) are interested because lives are at stake. In an academic sense, this issue is of importance to evolutionists because they believe the mutations in bacteria responsible for drug resistance are, from the standpoint of the bacterial population, "good," and thus offer significant proof of evolution. Their point is that the bacteria have adapted so as to "live to fight another day"—an example of "decidedly advantageous" mutations. Evolutionist Colin Patterson of Great Britain commented: "The development of antibiotic-resistant strains of bacteria, and also of insects resistant to DDT and a host of other recently discovered insecticides, **are genuine evolutionary changes** (1978, p. 85, emp. added). But are these mutations sufficient to explain long-term, large-scale evolution (macroevolution)?

AN ALTERNATIVE EXPLANATION

Bacteria do not become resistant to antibiotics merely by experiencing genetic mutations. In fact, there are at least three genetic mechanisms by which resistance may be conferred. First, there are instances where **mutations** produce antibiotic-resistant strains of microorganisms. Second, there is the process of **conjugation**, during which two bacterial cells join and an exchange of genetic material occurs. Inside many bacteria there is a somewhat circular piece of self-replicating DNA known as a plasmid, which codes for enzymes necessary for the bacteria's survival. Certain of these enzymes, coincidentally, assist in the breakdown of antibiotics, thus making the bacteria resistant to antibiotics. During conjugation, plasmids in one organism that are responsible for resistance to antibiotics may be transferred to an organism that previously did not possess such resistance.

Third, bacteria can incorporate into their own genetic machinery foreign pieces of DNA by either of two types of DNA transposition. In **transformation**, DNA from the environment (perhaps from the death of another bacterium) is absorbed into the bacterial cell. In **transduction**, a piece of DNA is transported into the cell by a virus. As a result of incorporating new genetic material, an organism can become resistant to antibiotics. Commenting on these processes, Walter J. ReMine wrote:

> Transformation and transduction occur extremely infrequently, but this rarity can be offset somewhat by the enormous population sizes that bacteria can achieve, especially under laboratory conditions. By those three methods bacteria can acquire DNA that alters their survival.... For example, DNA transposition can result in reduced permeability of the cell wall to certain substances, sometimes providing an increased resistance to antibiotics (1993, p. 404).

The issue is not whether bacteria develop resistance to antibiotics through alterations in their genetic material. They do. The issue is whether or not such resistance helps the evolutionists' case. I suggest that it does not, for the following reasons.

First, the mutations responsible for antibiotic resistance in bacteria do not arise as a result of the "need" of the organisms. Futumya has noted: "...the adaptive 'needs' of the species do not increase the likelihood that an adaptive mutation will occur; mutations are not directed toward the adaptive needs of the moment.... Mutations have causes, but the species' need to adapt isn't one of them" (1983, pp. 137,138). What does this mean? Simply put, bacteria did not "mutate" after being exposed to antibiotics; the mutations conferring the resistance were present in the bacterial population even prior to the discovery or use of the antibiotics. The Lederbergs' experiments in 1952 on streptomycin-resistant bacteria showed that bacteria that never had been exposed to the antibiotic already possessed the mutations responsible for the resistance. Malcolm Bowden observed: "What is interesting is that bacterial cultures from bodies frozen 140 years ago were found to be resistant to antibiotics that were developed 100 years later. Thus the specific chemical needed for resistance was inherent in the bacteria" (1991, p. 56). These bacteria did not mutate to become resistant to antibiotics. Furthermore, the non-resistant varieties did not become resistant due to mutations.

Second, while pre-existing mutations may confer antibiotic resistance, such mutations simultaneously may decrease an organism's viability as well. For example, "the surviving strains are usually less virulent, and have a reduced metabolism and so grow more slowly. This is hardly a recommendation for 'improving the species by competition' (i.e., survival of the fittest)" (Bowden, p. 56). Just because a mutation provides an organism with a certain trait does not mean necessarily that the organism **as a whole** has been helped. For example, in the disease known as sickle-cell anemia (caused by a mutation), people who are "carriers" of the disease do not die from it and are resistant to malaria, which at first glance would appear to be an excellent example of a good mutation. However, that is not the entire story. While such people are indeed resistant to malaria, they do not possess the stamina of, and do not live as long as, their non- carrier counterparts. Bacteria may be resistant to a certain antibiotic, but that resistance comes at a price. Thus, in the grand scheme of things, acquiring resistance does not lead necessarily to the production of new species or types of organisms.

Third, regardless of how bacteria acquired their antibiotic resistance (i.e., by mutation, conjugation, or by transposition), they are still exactly the same bacteria **after** receiving that trait as they were **before** receiving it. The "evolution" is not vertical macroevolution but horizontal microevolution (i.e., adaptation). In other words, these bacteria "...are still the same bacteria and of the same type, being only a variety that differs from the normal in its resistance to the antibiotic. No new 'species' have been produced" (Bowden, p. 56). In commenting on the changing, or sharing, of genetic material, ReMine has suggested: "It has not allowed bacteria to arbitrarily swap major innovations such as the use of chlorophyll or flagella. The major features of microorganisms fall into well-defined groups that seem to have a nested pattern like the rest of life" (1993, p. 404).

In their attempts to understand antibiotic resistance, microbiologists have studied extensively two genera of bacteria—*Escherichia* and *Salmonella*. In speaking about *Escherichia* in an evolutionary context, France's renowned zoologist, Pierre-Paul Grassé, observed:

> ...bacteria, despite their great production of intraspecific varieties, exhibit a great fidelity to their species. The bacillus *Escherichia coli*, whose mutants have been studied very carefully, is the best example. The reader will agree that it is surprising, to say the least, to want to prove evolution and to discover its mechanisms and then to choose as a material for this study a being which practically stabilized a billion years ago (1977, p. 87).

Although *E. coli* allegedly has undergone a billion years' worth of mutations, it nevertheless has remained "stabilized" in its "nested pattern." While mutations and DNA transposition have caused change within the bacterial population, those changes have occurred within narrow limits. No long-term, large- scale evolution has occurred.

CONCLUSION

The suggestion that the development in bacteria of resistance to antibiotics as a result of genetic mutations or DNA transposition somehow "proves" organic evolution is flawed. Macroevolution requires change across phylogenetic boundaries. In the case of antibiotic-resistant bacteria, that has not occurred.

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