Intelligent Design and Evolution Awareness Center

# The Implications of Antibiotic and Antiviral Drug Resistance for the Power of Darwinian Evolution

As found on the IDEA Center website at http://www.ideacenter.org

**By Casey Luskin**

Bacterial antibiotic resistance or viral antiviral drug resistance are often cited to prove the alleged creative power of evolution through mutation and selection. While antibiotic resistance and antiviral drug resistance are very real phenomena, they have no real bearing on the actual ability of evolutionary processes to create new genetic information, and do not give real clues as to the origin of species in the first place. Antibiotic resistance and antiviral drug resistance are excellent examples of microevolution, or minor change within population, and involve the origination of zero to miniscule amounts of information in the genome.

**Drug-Dodging Bugs**

As noted earlier, there are basically two types of "resistance." The first is "antibiotic resistance," which is when microorganisms, such as bacteria, become immune to drugs designed to kill the microorganism. These "antibiotics" are designed to kill off the living microorganisms. The second type is "antiviral drug resistance," occurring when a virus mutates such that antiviral drugs are no longer effective in stopping the spread of a virus. Microorganisms are living cells, complete with chromosomes, metabolism, and reproduction by cell-division, and thus can be "killed." Viruses, on the other hand, are typically not thought to be "alive" and cannot be "killed" in our typical understanding of the word. Viruses spread by hijacking the machinery of a host cell to allow them to replicate, and infect other cells. Viruses become most dangerous when they kill their host cells during as they hijack and use the cell's machinery to replicate more virus copies. Viruses thus differ from "living" cells in that they are always parasites and cannot live apart from the help of living cells. While some true living microorganisms are also parasitic, these living parasitic microorganisms are capable of reproducing through cell division and conduct their own distinct metabolic processes and protein production. In contrast, viruses live completely off of the biochemical parts, molecules, and energy produced by its host cell. In essence, a virus is not alive because it cannot metabolize chemicals in its environment into food for itself, but requires a living organism to help it produce the essential biomolecules it needs to reproduce (see http://www.uq.edu.au/vdu/KidsVirusesAlive.htm for details). Thus, a virus cannot be "killed," but its spreading can be slowed or even stopped through various anti-virus drugs that prevent it from replicating new virus copies. Viruses, which do use small strands of DNA or RNA to replicate, can undergo mutations such that their replication is not stopped by these antiviral drugs, hence producing antiviral drug resistance.

**HIV Antiviral Resistance**

The amount of genetic informational change between rapidly mutating virus strains, such as HIV, is miniscule compared to the type of informational changes required in the wild to create new body plans or biochemical pathways. Anti-HIV drugs typically work to stop virus replication through a number of different ways, such as through protease inhibitors or retrotranscriptase inhibitors. These inhibitor drugs are then mixed together into a "cocktail" to help slow HIV replication. Unfortunately, no complete "cure" for HIV has yet been found.

HIV is particularly prone to "evolution" (i.e. change) because of its high replication and mutation rate--nearly one mutation per replication. In fact, one researcher claims that the high rate of virus replication and small size of their genome would cause any given HIV mutation to exist even before a given drug which might be resisted is introduced. In other words, there's nothing magical about HIV antibiotic resistance. Unfortunately, this fact has made HIV quite difficult to combat, and many have suffered greatly because of difficulties in stopping HIV from becoming resistant to drugs. HIV is an extremely rapidly mutating virus which mutates it's way through human immune systems, making it difficult for the body to combat. If evolution ever had a chance, it had it's easiest and simplest chance with HIV virus anti resistance (see HIV Resistance to Antiretroviral Drugs (*Journal of American Medical Association*, Vol. 4, No. 3, Sept. 1996) for more info.)

**Bacterial Antibiotic Resistance**

Antibiotics are chemicals which retard virus or bacterial proliferation by entering the microbes and interfering with the production components needed to for reproduction. This can include impairing protein manufacture or destroying cell walls. Antibiotic resistance typically involves a simple point mutation which slightly changes the structure of antibiotic target (the cell wall or ribosome (protein factory)) such that the antibiotic is no longer effective against it. It does not involve a change in function, but merely a slight change in structure such that function is maintained and the antibiotic's structural effect upon the target is inhibited. Some resistance occurs if an enzyme the bacteria happens to has interferes with the antibiotic such that it cannot reach it's target. Cell walls which are not affected by antibiotic drugs are easily selectable under a Darwinian scenario. This is real evolution, and because organisms, replication mutations, and gene swapping are so prevalent in bacteria, once one out of countless bacteria becomes resistant, it is quite a quick and simple process for many others to either get selected out or obtain the gene for resistance. However, the processes behind antibiotic resistance do not involve the creation of new real significant information (More information on how bacteria become resistant can be

found at, The Challenge of Antibiotic Resistance by Stuart Levy (Scientific American, March 1998)). Antibiotic (and pesticide) resistance does not tell how new biochemical pathways originate, how complex organ systems develop, or how molecules turned into humans but rather they involve minor benign structural changes in various proteins of target systems and/or chance interaction of target organism enzymes with the antibiotic. One recent review of anti-biotic resistance in by Christopher Walsh (Molecular Mechanisms that Confer Antibacterial Drug Resistance, *Nature*, Vol. 406 (N6797): 775-781 (2000)), cites 3 mechanisms of anti-biotic resistance (see diagrams at right):

1) The overproduction of already-existing protein pumps to export antibiotic drugs (nothing new created here)

2) Minor structural changes in various proteins of target systems (these are functionally-moot and requires only a point mutation, and are not information-building), and

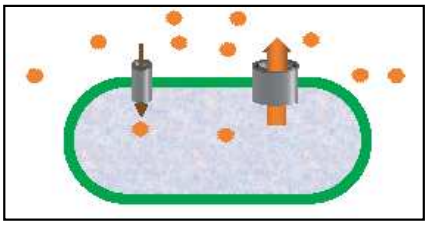
3) Destructive of the antibiotic by interaction with very slightly modified pre-existing enzymes of the organism (again, no new information is created and changes are minor, if any).

Though Walsh shows that bacteria have found innovative ways to survive attacks from antibiotic drugs, Walsh doesn't mention that bacteria, after becoming resistant, often face a "cost", which is a decrease in relative fitness compared to the original strain before introduction of the anti-biotic.

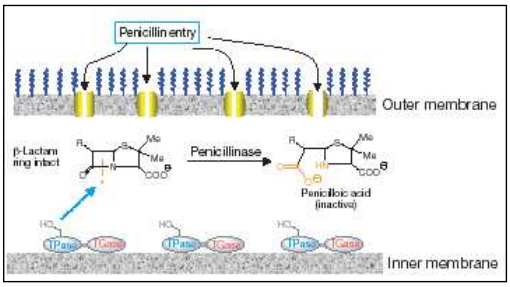
In none of these cases, however, has any significant information, useful for explaining macroevolutionary change, been added to the genome. In fact, Walsh implies that the very short time frame in which antibiotic resistance often develops testifies to the simplicity of the genetic and phenotypic changes entailed therein. Antibiotic resistance thus actually weakens the bacteria, and that in this case, evolution only comes at a fitness cost, and definitely doesn't represent the addition of new genetic information to the genome.

**Antibiotic and Antiviral Drug Resistance is Futile**
The alleged power of evolution through mutation and natural selection is not established by claiming antibiotic or antiviral drug resistance because no new information-rich (or even information-poor) functions appear in the cell and the changes typically involve nothing more than functionally-moot minor structural changes in the cells. In fact, the fitness cost associated with antibiotic resistance indicates that functionality these organisms is not very plastic, as resistant strains are less efficient at performing key cellular functions than the non-resistant strains. If nothing else, this indicates that this form of resistance does NOT include advantageous mutations that create new functions or add to the vitality or vivacity of a cell. When trying to prove macroevolution, or that Darwinian evolution can create new cellular functions, citing to antibiotic and antiviral drug resistance is futile.

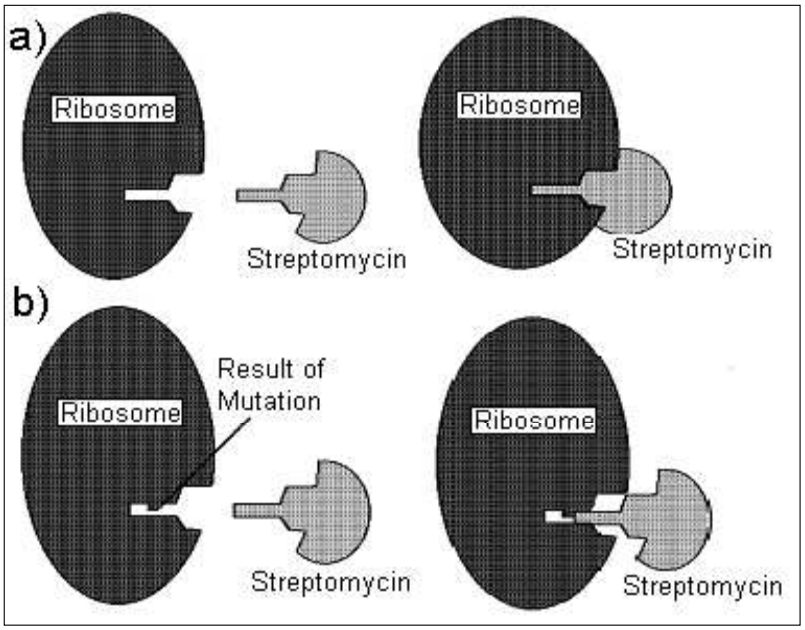
# 3 Common Mechanisms of Antibiotic Resistance:



1. Overproduction of already-existing protein pumps to export antibiotic drugs (nothing new created). From Walsh (2000).




2. Overproduction or slight modification of pre-existing enzymes in the bacteria can destroy antibiotic. (nothing new) From Walsh (2000).




3. Very slight functionally moot change in structure of target causes it to become incompatible with antibiotic drug. From *Not by Chance!* By Lee Spetner.