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|  | Introduction When the availability of penicillin increased dramatically during World War II, it allowed soldiers to combat their most dreaded enemy and biggest wartime killer- infected wounds. Initially, it was discovered by French medical student, Ernest Duchesne in 1896, then later rediscovered by Scottish physician Alexander Fleming in 1928. The product of the mold Penicillium crippled many bacteria that caused disease. However, only four years after drug companies began mass production of penicillin in 1943, microbes, resistant to the drug, began appearing.  The resistance spread quickly. Between 1979 and 1987, only 0.02 percent of *Pneumococcus* strains infected patients surveyed by the Nation Centers for Disease Control and Prevention were penicillin resistant. Today, it is estimated that about 6.60 percent of strains are resistant. The American Medical Association reports that in 1992, 13,300 hospital patients died of bacterial infections resistant to antibiotic treatment.  How did this happen? "There was complacency in the 1980s. The perception was that we had licked the bacterial infection problem. Drug companies weren't working on new agents. They were concentrating on other areas, such as viral infections," says Michael Blum, M.D., medical officer in the Food and Drug Administration's division of anti-infective drug products."  In the meantime, resistance increased to a number of commonly used antibiotics, possibly related to overuse of antibiotics. Recent cases of bacteria resistant to modern antibiotic have been cause of alarm and a great deal of anxiety in our society. In the 1990s, resistant strains of once common bacteria are appearing at an alarming rate. According to a report in the April 28, 1994, New England Journal of Medicine, researchers have identified bacteria in patient samples that resist all currently available antibiotic drugs.  The increased prevalence of antibiotic resistance is an outcome of evolution. Any population of organisms, bacteria included, naturally includes variants with unusual traits--in this case, the ability to withstand an antibiotic's attack on a microbe. When a person takes an antibiotic, the drug kills the defenseless bacteria, leaving behind--or "selecting," in biological terms--those that can resist it. These renegade bacteria then multiply, increasing their numbers a million fold in a day, becoming the predominant microorganism. The antibiotic does not technically cause the resistance, but allows it to happen by creating a situation where an already existing variant can flourish.  A patient can develop a drug-resistant infection either by contracting a resistant bug to begin with, or by having a resistant microbe emerge in the body once antibiotic treatment begins. Drug-resistant infections increase risk of death, and are often associated with prolonged hospital stays, and sometimes complications. These might necessitate removing part of a ravaged lung, or replacing a damaged heart valve.  There have been many specific instances in which common bacteria have become resistant to drugs. For instance, *Staphylococcus aureus*, has become resistant to multiple antibiotics. This type of multiple-drug resistance poses a major threat, both because of infection and because of the harmful side effects often associated with high-end antibiotics.  Other methods of controlling bacteria include the use of acid preservation and antiseptics. The first method, acid preservation, was why most scientists felt that bacterial contamination of such things such as apple juice were highly unlikely. However, when several batches of Odwalla apple juice were found to be contaminated with E. Coli, scientists sought an explanation. As it turned out, the bacteria were able to acquire a stress induced resistance to an acid environment, thus surviving long enough to cause damage.  This stress-induced resistance functions by causing a shift in the cell wall of the organism. When exposed to a sub-lethal concentration, the cell wall is changed to become less permeable to the toxin. Over time, the cell will develop a resistance to increasingly larger doses and eventually will be able to survive for a longer period of time in lethal doses. This change, however is transitory and will be readily reversed if exposure to the toxin ceases.  With this phenomenon in mind, one need but take notice of the increasingly prevalent use of anti-bacterial products. Today's society has become infatuated with killing bacteria. Companies are capitalizing on society's desire to wipe out bacteria, manufacturing every product imaginable into anti-bacterial forms. Soaps, hand sanitizers, lotions, and sprays have been marketed, providing a sense of security to the general public. Are these products going to lose their effectiveness if used continuously? The problem is worsened by the fact that a vast majority of these products all use the same active ingredient: ethyl alcohol. As such, it would be valuable to determine whether bacteria can develop stress-induced resistance to ethyl alcohol.  The format of the acid-induced resistance test was fairly simple. First, a calibration curve for colony survival is determined, so that the threshold for a "lethal" dose can be determined. Then, one exposes one group to a sub-lethal dose of acid and a control group is placed in an optimal pH buffer. The two colonies are then exposed to a lethal dose and then the survival times are measured.  The question, then, is whether this same type of resistance test can be applied to ethyl alcohol. In order to consider this question, it is prudent to look at the way in which ethyl alcohol attacks bacteria. In the seventy percent to the ninety percent range, ethyl alcohol works by dissolving the lipid membrane of the bacterium. This will kill a wide variety of bacteria, but the ethyl alcohol evaporates very quickly, causing it to have very little residual effect. As such, the ethyl alcohol will likely only cause stress-induced resistance if secured in a non-volatile medium, such as a gel.  Antibiotic resistance is inevitable, say scientists, but there are measures we can take to slow it. However, there are simple precautions that can be taken in order to slow the process of antibiotic resistance. These approaches include more frequent hand washing by health-care workers, quick identification and isolation of patients with drug-resistant infections, and improving sewage systems and water purity in developing nations.  Drug manufacturers are once again becoming interested in developing new antibiotics. Efforts are under way on several fronts--improving infection control, developing new antibiotics, and using drugs more appropriately. These efforts have been spurred both by the appearance of new bacterial illnesses, such as Lyme disease and Legionnaire's disease, and resurgences of old foes, such as tuberculosis, due to drug resistance.  FDA is doing all it can to speed development and availability of new antibiotic drugs. "We can't identify new agents--that's the job of the pharmaceutical industry. But once they have identified a promising new drug for resistant infections, what we can do is to meet with the company very early and help design the development plan and clinical trials," says Dr. Robert Glascow of the CDC.  In addition, drugs in development can be used for patients with multi-drug-resistant infections on an emergency IND (compassionate use) basis, if the physician requests this of FDA. This is done for people with AIDS or cancer, for example.  No one really has a good idea of the extent of antibiotic resistance, because it hasn't been monitored in a coordinated fashion. Each hospital monitors its own resistance, but there is no good national system to test for antibiotic resistance.  This may soon change. CDC is encouraging local health officials to track resistance data, and the World Health Organization has initiated a global computer database for physicians to report outbreaks of drug-resistant bacterial infections.  Scientists have already deemed resistance to antibiotics unavoidable. However, with research and care, these effects should be able to be delayed.  Note: I recommend that you read the Word Document file, as converting to HTML butchered the footnotes and seriously messed up the formatting. This file is located in Dan /Genie Web as Biology.doc. | |
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