Antibiotics: A Savior in Need of Saving

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Introduction

There are millions of bacteria around us, some are capable of harming us, while others are beneficial to us. Bacteria that are capable of harming us can cause infections, which can lead to disease and even death. However, there are many methods one can use not only to prevent but cure bacterial infections. One such method is the use of antibiotics. Antibiotics are small, natural chemical substances that are produced and secreted by microorganisms into the environment to control other organisms (Hutchings, Fernández-Martínez, Centre, J & Hoskisson, 2012). Antibiotics have been around since a famous scientist, Alexander Fleming, discovered the origin of one very common antibiotic called Penicillin in 1928. Fleming was working with *Staphylococcal* bacteria and discovered that one of his Petri dishes was contaminated with mold (Tan & Tatsumara, 2015). When Fleming observed the dish, he noticed that staph bacteria were dying around the area where mold was growing and found that a fungus by the name of *Penicillum notatum* was secreting something that was capable of inhibiting the growth of bacteria. This lead to the discovery of Penicillin. When Fleming was awarded the Nobel Prize in Medicine or Physiology for his accidental discovery in 1945, he noted: "One sometimes finds what one is not looking for." The discovery of Penicillin was an important milestone which led to the discovery of many other antibiotics. It was found that fungi were not the only microorganisms that could make antibiotics, as bacteria were also capable of producing these substances. The powerful antibiotic Vancomycin, for instance, is produced by *Actinobacteria*, which are typically found in soil (Hutchings et al, 2012).

The objective of this lab was to test the effect of six different antibiotics: Ampicillin, Penicillin G, Tetracycline, Chloramphenicol, Erythromycin and Clindamycin on bacterial growth and determine if two bacteria, one gram-positive and one gram-negative, were resistant or susceptible to these antibiotics.

Materials & Methods

Gram-negative bacteria: *Escherichia coli,* gram-positive bacteria*: Staphylococcus aureus*, empty sterile nutrient agar plates, sterile swabs, culture tube rack, Bunsen burner, paper towels and soap, bleach bottles, forceps, filter paper disks, antibiotic discs (Ampicillin, Penicillin G, Tetracycline, Chloramphenicol, Erythromycin and Clindamycin), antibiotic dispensers and refills.

Two agar plates were divided into five sections, each section was labeled with the name of one of the antibiotics used. A sterile swab was dipped into a liquid sample of bacterial culture and the agar plate was swabbed everywhere with the corresponding bacterial sample. The plates were left to dry briefly, then, using forceps, an antibiotic disk was taken from the dispenser and placed in the center of the corresponding section on the agar plate. The procedures were repeated for the other antibiotic disks and the other bacterial sample. The agar plates were then placed in an incubator at 37^0C and were later observed for the zone of inhibition.

Results

The observed presence of medium to large zones of growth inhibitions around the antibiotic disks indicated that the bacteria were susceptible to the antibiotic, meaning that the antibiotics were successful in destroying the bacteria. When the zone of growth inhibition was absent or small, it indicated that the bacteria were resistant to the antibiotics, meaning that the antibiotic had no effect on the bacteria. Results indicated that *Escherichia coli* (gram-negative) were susceptible to Ampicillin, Penicillin G, Tetracycline and Chloramphenicol and were resistant to Erythromycin and Clindamycin. Results also indicated that *Staphylococcus aureus* (gram-positive) were susceptible to Ampicillin, Penicillin G, Tetracycline, Chloramphenicol, Erythromycin and Clindamycin.

Conclusion

The test was extremely helpful in studying the effect of antibiotics on two samples of bacteria, gram-negative *Escherichia coli* and gram-positive *Staphylococcus aureus*. In the case of *Escherichia coli,* it was observed that these bacteria were susceptible to all the antibiotics except two. Ampicillin and Penicillin were successful in inhibiting the formation of the cell wall by preventing peptidoglycan linkage therefore killing the bacteria; the effect of these antibiotics on the bacteria was bactericidal. Tetracycline and Chloramphenicol have a bacteriostatic effect on *Escherichia coli.* Tetracycline worked by interfering with transfer RNA attachment to messenger RNA ribosome complex and therefore inhibiting protein synthesis, and Chloramphenicol worked by binding to the 50s ribosomal subunit and preventing peptide bonds formation therefore stopping protein synthesis. It was also found that *Escherichia coli* grew and were resistant to Erythromycin and Clindamycin.

In the case of *Staphylococcus aureus,* it was observed that the bacteria were susceptible to all six of the antibiotics. The mechanism of action for Ampicillin, Penicillin, Tetracycline and Chloramphenicol was the same as described before. Erythromycin and Clindamycin also were bacteriostatic and also inhibited protein synthesis therefore leading to cell death.

It was interesting to note that *Escherichia coli* were found to be susceptible to Ampicillin, which is a semi-synthetic form of Penicillin. Penicillin and its derivative Ampicillin are known to target only gram-positive bacteria by interfering with the formation of the cell wall. *Escherichia coli* are characterized as gram-negative bacteria. It is known that most antibiotics against gram-positive

bacteria do not work against gram-negative bacteria, due to cell wall structure differences. However it was found that the only reason Ampicillin was able to work against *Escherichia coli* was because it contained an amino group that allowed Ampicillin to penetrate the cell wall of gram-negative bacteria (*Escherichia coli)*. Once inside, Ampicillin was able to irreversibly bind to an enzyme transpeptidase and inhibit it from forming the cell wall, which leads to leakage and cell death (Anthony & Lawrence, n.d.). The effect of Ampicillin on *Escherichia coli* was due to specificity, not toxicity, because it had components that allowed it to enter the bacterial cell wall and destroy the bacteria by breaking down the bacteria's cell wall. The mechanism for Ampicillin was bactericidal, meaning that it killed the bacteria.

Another observation that was made was that Erythromycin, an antibiotic that blocks protein synthesis by binding to the 50S ribosomal subunit, was unable to kill *Escherichia coli.* This was due to the fact that *Escherichia coli* were found to have developed a native resistance to the antibiotic. It was found that *Escherichia coli* had ribosomes that did not bind to Erythromycin. This was due to "a single amino acid replacement in the L4 protein of the 50S subunit" (Pratt & Scholar, n.d.). This mutation found in *Escherichia coli* was due to a change in the target molecule, a process that led to resistance.

Antibiotics can be used to classify bacteria on the basis of antibiotic resistance patterns. For example, some bacteria have beta-lactamase, an enzyme that breaks down the beta-lactam ring in Penicillin, thus making the antibiotic less effective. Using tests to detect the presence of beta-lactamase can help identify and classify bacteria on the basis of the presence of the enzyme. Antibiotic susceptibility testing can also be used to detect bacterial resistance and help classify bacteria. Further genomic studies can be done to determine if the resistance is due to enzymes or mutations in genes. These findings can aid in identification. It should be noted that using only antibiotics is not always useful in identification and classification of bacteria. Other tests such as Gram stains, biochemical, immunological and genomic tests should be used in conjunction to positively identify and classify bacteria.

In conclusion, antibiotics are extremely helpful in fighting infections and killing pathogenic microorganisms. However antibiotics can also be very harmful. Antibiotics have different mechanisms of actions and some of them can cause adverse effects on host cells causing toxicity to humans. For example, antibiotics used in protein synthesis work on prokaryotic cells that have 70S ribosomes compared to proteins in eukaryotic cells that have 80S ribosomes. However, mitochondria inside eukaryotic cells contain 70S ribosomes and certain antibiotics can cause problems inside hosts. Other antibiotics work by inhibiting nucleic acid synthesis and they can also interfere with human DNA and RNA leading to problems (Case, Funke & Tortora, 1982, p.563). Antibiotics can also lead to problems such as allergies, nausea, vomiting, skin rashes and diarrhea. Since some antibiotics are broad spectrum they can kill beneficial bacteria found in the gut and disrupt the normal flora leading to other issues.

Finally, the biggest problem is resistance of bacteria to antibiotics. This comes from over prescribing antibiotics by health care providers, consuming dairy, meat, and poultry that were treated with antibiotics and patients not following proper instructions while taking antibiotics. All of these lead to mutations in bacteria and new antibiotics must be sought that are even more potent to kill these resistant bacteria. This is a growing and serious problem in the world. Each day, more and more bacteria become resistant to antibiotics. The most potent infection caused by the Methicillin-Resistant *Staphylococcus aureus* (MRSA) is still not treatable even with the strongest antibiotic Vancomycin. Recently, bacteria that cause Gonorrhea, a sexually transmitted infection, have been found to be resistant to many different antibiotics.

Fixing the problem by synthesizing new antibiotics will only work in the short term. This is because bacteria can pass on resistant genes fairly quickly to other microbes. According to the World Health Organization (WHO), death from drug resistant infections are at 700,000 per year and WHO predicts that this number will reach 10 million by 2050 (Beck, 2015). If things remain the same we will go back to the past when even the smallest injuries were fatal to mankind and life expectancy was very short. Therefore, new policies should be implemented to prevent abuse of antibiotics. Physicians should be careful in prescribing antibiotics and should do it once the specific strand of bacteria has been identified. Further changes should be made in the food industry where animals should not be given antibiotics unless necessary. Antibiotics are extremely important for human survival: they are our saviors who need to be saved so that our future doesn't resemble our past.

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