CALIFORNIA SCIENCE & ENGINEERING FAIR
2018 PROJECT SUMMARY

Name(s)

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Project Number

J1709

Project Title

Unnecessary and Excessive Antibiotic Use: An Uprising of Resistant Bacteria

Abstract

Objectives/Goals

The objective of my project was to determine which bacteria where susceptible or resistant to certain antibiotics and to find out why this occurred. My hypothesis was that bacteria that were susceptible to an antibiotic would have a larger zone of inhibition because the antibiotic prevented it from growing, while bacteria that were resistant would have a smaller zone because the antibiotic could not effectively prevent it from growing.

Methods/Materials

First petri dishes were divided into 4quadrants and the bacteria was loaded on the dish. Next, diffusion disks were soaked in different antibiotics and loaded on the plates. Once finished, the plates were inverted and left to incubate for 16-20 hours.The zone of inhibitions were calculated by measuring the diameter of each zone, using a metric ruler. This was repeated 2 times. The E. Coli and Enterobacter were obtained from carolina.com, a biological supply website.

Results

It was found that E. Coli was susceptible to Amoxicillin, Augmentin, Sulfamethoxazole-trimethoprim, Azithromycin, Cefdinir, and Cephalexin. However, E. Coli was resistant to Clindamycin and Penicillin. It was found that Enterobacter was susceptible to Sulfamethoxazole-trimethoprim, Augmentin, Azithromycin, Cefdinir, and Cephalexin. However, Enterobacter was resistant to Amoxicillin and Penicillin.

Conclusions/Discussion

For both E. Coli and Enterobacter, Sulfamethoxazole-trimethoprim was effective in fighting them because Sulfamethoxazole interferes with the synthesis of folate by competing with p-aminobenzoic acid in the biosynthesis of dihydrofolate. Trimethoprim serves as an inhibitor of dihydrofolate reductase, inhibiting the synthesis of tetrahydrofolate. Finally, when exposed to Cephalexin both E. Coli and Enterobacter were susceptible because Cephalexin acts by inhibiting synthesis of the peptidoglycan layer of the bacterial cell wall; cephalexin closely resembles d-alanyl-d-alanine, an amino acid ending on the peptidoglycan, so it is able to irreversibly bind to the active site of PBP. With this information, it can be proven that undestanding the way antibiotics work could provide us with an insight into the future and how to fight antibiotic-resistant bacteria. This knowledge can help scientists modify antibiotics in order to combat the newly growing population of resistant bacteria.

Summary Statement

Bacteria that were susceptible had a larger zone of inhibition and were prevented from growing; however, bacteria that were resistant had a smaller zone and had mutated over many yers to resist that antibiotic.

Help Received

I was assisted by Lida Gevorkian, school site coordinator, and Dr. Albarez, Glendale Adventist Medical Center immunologist, who verified my results and answered my questions about the function of the antibiotics.