ABSTRACT

Elizabethkingia Anophelis is a rod shaped, Gram-negative, stationary bacteria that is resistant to many antibiotics. It is believed that the protein that creates a antibiotic resistance is created by the Elizabethkingia. To determine this, bacteria was grown inside of two different growth conditions (normal and alternate) and then extracted to test differences in DNA, RNA, and Proteins.

INTRODUCTION

Elizabethkingia anophelis is a gram-negative, rod-shaped bacterium which has resulted in a multistate outbreak of mainly bloodstream infections in the states of Wisconsin, Michigan and Illinois. Affecting primarily those individuals over 65 years of age with at least one serious underlying health condition. The Elizabethkingia bacterial genus was first discovered by Elizabeth O. King, a CDC microbiologist in 1959. Using fluid from the spinal cords of children, many of whom had died of meningitis between 1948 to 1958.(1, 3) In our research, we examined 5 genes from the lipid metabolism pathway in Elizabethkingia anophelis. Our objective was to see how these specific genes helped the bacteria function and how big of an impact each gene has on the overall process of the Elizabethkingia disease. After finding the results, we later looked at how two types of antibiotics affected our 5 genes. Our overall question was how important lipid metabolism is in Elizabethkingia and how susceptible our genes are to antibiotics.

MATERIALS AND METHODS

Using all three databases: RAST, Patric, and UniProtKB, we researched what antibiotics that *Elizabethkingia anophelis* is susceptible to. After looking at five genes contributing to lipid metabolism and by combining information from all three databases, we were able to compare susceptibility when Elizabethkingia anophelis is exposed to various antibiotics. We used computers to research information about five genes related to the lipid metabolism pathway and used the excel data spreadsheet to compare transcripts found after antibiotics were used to test effectiveness.

Exploring Elizabethkingia Anophelis

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|-----------------------|-------------|-------------|-------------|-------------|----------------|--|--|
| | | | | RESULTS | | | |
| Genes by | # | # | # | # Total | Fold | Fold | |
| RAST id | transcripts | transcripts | transcripts | Transcripts | change | change | |
| | control | Cefotax | Imipenem | | Cefotax | Imipenem | |
| | | | | | | | |
| | | | | | | | |
| 1631 | 152 | 218 | 144 | 514 | 1.4 | -1.1 | |
| 106 | 4 | 6 | 5 | 15 | 1.5 | 1.3 | |
| 444 | 3 | 3 | 8 | 14 | -1.0 | 2.7 | |
| 1630 | 12 | 22 | 12 | 46 | 1.8 | -1.0 | |

Gene 106 - 3-hydroxybutyryl-CoA dehydrogenase

E. anophelis R2 (

Gene 444 - Enoyl-[acyl-carrier-protein] reductase E. anophelis R2 (

Gene 1630 - Acyl carrier protein

E. anophelis R2 <

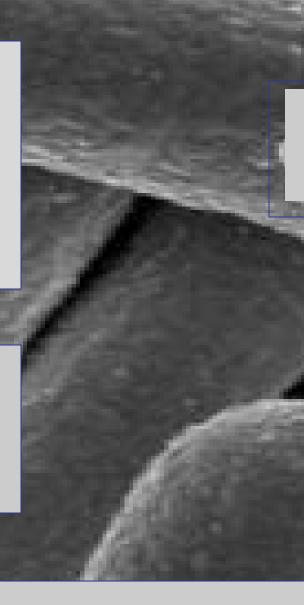
Gene 1631- 3-oxoacyl-[acyl-carrier-protein] synthase 2 and 3

3-hydroxybutyryl-CoA dehydrogenase has very few transcriptions and has very little change when introduced to the antibiotics Cefotax and Imipenem.

Enoyl-[acyl-carrier-protein] reductase also has very few transcriptions and has very little change when introduced to the antibiotic Cefotax but over doubles transcription when introduced to Imipenem.

Acyl carrier protein has very few transcriptions and has no change in transcription when introduced to the antibiotic Imipenem but nearly doubles in transcription when introduced to the antibiotic Cefotax.

3-oxoacyl-[acyl-carrier-protein] synthase 2 and 3 have many transcriptions and when introduced to the antibiotic Cefotax, transcription increased. When the protein was introduced to Imipenem transcription decreased slightly.



DISCUSSION

We questioned how our pathway works in Elizabethkingia and how impactful antibiotics are on it. We found that none of the genes are beside each other on the genome. We came to the conclusion that this means they do not work together as much as we thought they did. Instead, we believe that they are all just of the same kind. We found that our pathway builds up and breaks down proteins. We also believe that the antibiotics do not work very well on the pathway as a whole and therefore they are resistant.



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