Elizabethkingia Metabolism

GROUP #21

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ABSTRACT

In this study we have been looking at genes involved in the metabolism of Ascorbate and Alderate. The genes we have been looking at are involved in the transformation of energy for Elizabethkingia anophelis R26. the genes are Aldehyde Dehydrogenase, 2-hydroxy-6-oxo-6-phenylhexa-2,4-dienoate hydrolase, Multiple polyol-specific dehydrogenase, and Membrane-bound lytic murein transglycosylase D precursor. We were interested in just how important these genes were to the bacteria and if they were affected by antibiotics.

INTRODUCTION

Elizabethkingia anophelis R26 is a bacteria that has shown antibacterial resistances. With the bacteria causing harm in a small outbreak in December of 2015. This outbreak has caused Elizabethkingia to be in the spotlight of the CDC. They are trying to figure out why and how the bacteria is so resistant to antibacterial drugs. With this we decided to look into the genes of the bacteria. When we were looking through the genes we found the pathways for the metabolism of of Ascorbate and Alderate. We decided to follow this pathway to find what all was involved.

MATERIALS AND METHODS

Our group used the PatricDB and Browser to discover a pathway and find five genes associated with that pathway. We went to the Unique Gene Count to find out more information on each individual gene. We used the BLAST site to match our gene's sequences to the RASTdb. After finding our gene's Feature ID we used the RASTdb to look at the Visual Region Information of each gene. From D2L we used an MS Excel spreadsheet named "E.anoR26_RNAseqData_FoldChanges.xls" to find the number of transcripts observed under each growing condition and the fold change in transcription compared to the control.

RESULTS

In the study we looked to find if how Elizabethkingia metabolized certain items. We looked at the genes in the Ascorbate and Alderate metabolism pathway to find information. As we looked through the genes we found that the genes we were looking at coded for proteins used in the production of ATP and Glycolysis. With the genes being apart of the production of energy we could assume that the proteins were necessary to live. When looking at how the genes were affected by antibiotics we learned that they do not affect them on a large scale. We can not deduce if the genes were affected by the antibiotics or if there was just a random change in gene production. With this information we can assume that the genes do not affect the antibacterial resistance of Elizabethkingia.

Gene	Fold Change* (Cefotax/Control)	Fold Change* (Imipenem/control)	TOTAL mRNA ** from all 3 conditions	mRNA Counts Control	mRNA Counts Cefotax	mRNA Counts Imipenem
2-hydroxy-6-oxo-6-phenylhexa-2,4-dienoate hydrolase	-3.0	-1.5	6	3	1	2
Aldehyde dehydrogenase	2.0	-1.0	4	1	2	1
Membrane-bound lytic murein transglycosylase D precursor	-1.0	-1.2	61	22	21	18
Multiple polyol-specific dehydrogenase	-2.0	2.0	7	2	1	4
UDP-glucose dehydrogenase	-1.0	1.1	46	15	15	16

DISCUSSION

Elizabethkingia anophelis is a bacteria that is highly resistant to most antibacterial drugs on the market. Ultimately, or goal in this research class was to try and find a link between the genes we were researching and the antibacterial process of the bacteria. The genes my group researched did not seem to uncover any valuable evidence. Of the five genes that our group studied, only two of them had any effect in Elizabethkingia's antibacterial process; and even then, the fold changes recorded after our genes were tested weren't great enough to consider our genes a liable explanation for the bacteria's resistance against drugs.

REFERENCES

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