Elizabethkingia Anopelis
How does osmoregulation play its role in this specific gene?

GRP# 19

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INTRODUCTION

Elizabethkingia anophelis is a gram negative bacteria. Gram negative bacteria means that it can not hold the crystal violet stain that is used in the Gram staining method. It is a gene that is usually found in the environment in either soil, rivers, or reservoirs. We were to focus on how this disease as a total works and how the genes that make up this disease play their role. The reason for this research was to look up possible reason for this disease, to see how the separate genes made this disease happen. We started off with finding our genes we wanted to research through RAST database or Patric database. When we found our genes, we were to do outside research on the group of genes we picked. Then we were to find its DNA sequence and put it in a different website to see if the databases had it correct. The total point of this research was to figure out what the genes did in Elizabethkingia anophelis. This could have lead to possibly seeing how the disease came along and how to diagnose this disease based how its genes react.

MATERIALS AND METHODS

Use Rast Data Base or Patric database to find your information
- Browse, search and select any pathway or group of genes/proteins that contains more than 4 members that appeal you and briefly describe this pathway, class of proteins, family of protein, or group of interesting genes.
- Record the source and the pathway or group you selected and provide a list of the genes within that pathway or group.
- Click “Stress Response” category after viewing the “seed viewer”
- Click on “Osmoregulation”
- Gene Numbers: fig|1246994.5.peg.1557
fig|1246994.5.peg.258
fig|1246994.5.peg.1555
fig|1246994.5.peg.1794
fig|1246994.5.peg.1992
- Searched “*Osmoregulation* its role is *Outer membrane protein A precursor”* in google then press on images and went to different websites to find more detailed information.
- http://labs.mbi.nus.edu.sg/bpast/research.html
https://www.ebi.ac.uk/interpro/entry/IPR023743
http://www.ncbi.nlm.nih.gov/pubmed/16622204
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3617542/

When doing the research on Elizabethkingia anophelis we were to find a group of five genes that represented each other in a certain way. We had the option of using the RAST database or the Patric database to find the information needed to complete this research. When using either database you were asked to navigate throughout the website and figure out all the parts of your gene. The materials we needed to complete this was the instructions of Dr. Canaan, the website, and to work together as a group to put all the pieces together.
When our group found our group of genes we wanted to research over we were to figure out what that genes job in Elizabethkingia anophelis was, its sequence, how it reacted to the other genes around it, and what it was composed of. When doing the research through the database we had to keep in mind that some of the information could be incorrect or a mess up in the database at that time. This caused us to do more research outside of those databases. We had to take all the parts of everything we had found out and piece it together to find out the results of everything we researched. We used a method of trial and error throughout our research to see what was right and what was wrong. Finally, after we put everything together we had our results to show how osmoregualtion does what job and function towards Elizabethkingia anophelis.

RESULTS

We began by going through the RAST database and we decided to pick of proteins that were all affiliated with Osmoregulation. The RAST numbers for these proteins are 258, 1992, 1794, 1555, 1557. We then used outside sources to do further research on each individual protein. These proteins are predicted to control what comes in and out of the cell. At his point in our research, we did not know whether these proteins actually existed in the Elizabethkingia Anophelis R26. Then we took parts of the DNA sequences given on the RAST database and put it into the BLAST database. We found that all five of our proteins exist in Elizabethkingia Anophelis R26. While looking at our group of proteins on the chart on D2L that showed the number of transcriptions under the different conditions. The genes 258, 1794, and 1557 had a good number of transcribes but genes 1992 and 1555 did not have enough transcribes to be able to confidently use the data. The chart also told us that genes 1992 and 1555 had a significant change in transcriptions from the Control to the Imipenem, but this information cannot be used confidently because there was a very low number of transcription for both of these genes. There was also a significant change for gene 1557 from the Control to the Cefotax and we can be confident in this information. Lastly, we looked up the genes surrounding our genes in the Elizabethkingia Anophelis R26 genome. None of the surrounding genes had predicted functions that had to specifically do with Osmoregulation, but one gene, gene 1558, had to do with transport. The rest of the surrounding genes were either catalyzing proteins or had to do with DNA and the genome.

Results:

Gene 1
Aquaporin Z fig|1246994.5.peg.258

mRNA Count Control	mRNA Count Cefotax	mRNA Count Imipenem	mRNA Count Total	Fold change Cefotax	Fold Change Imipenem
57	72	41	170	1.3	-1.4

Most of the genes surrounding my gene has quite a bit more transcripts than my gene, but it still has a good amount of transcripts. Neither the Cefotax nor the Imipenem had a significant change in expression. Both of them were slightly underneath fifty percent. There were more transcripts in Cefotax than Imipenem but neither of them had a significant enough change to actually count.

Gene 2
Outer membrane protein A precursor fig|1246994.5.peg.1992

mRNA Count Control	mRNA Count Cefotax	mRNA Count Imipenem	mRNA Count Total	Fold change Cefotax	Fold Change Imipenem
1	1	2	4	-1.0	2.0

This gene did transcribe, but it transcribed with very low numbers. With the low numbers, you can see that the gene in Imipenem may have doubled but we cannot be confident with this answer. To figure out if this is true, you would have to do further testing to find the answers.

Gene 3
Outer membrane protein A precursor Fig|1246994.5.peg.1794

mRNA Count Control	mRNA Count Cefotax	mRNA Count Imipenem	mRNA Count Total	Fold change Cefotax	Fold Change Imipenem
539	699	387	1625	1.3	-1.4

When gene 1794 of *E. anophelis* was in the control group it moderately transcribed mRNA (539 times). When exposed to Cefotax the translation grew slightly by 160, totaling at 699 translations. When the gene was exposed to Imipenem the translations decreased by 55% resulting in a total of 387 translations. This is a significant drop in mRNA translation due to the exposure of Imipenem. As a result you could say that Imipenem inhibits the translation of the outer membrane protein A precursor by 55%.

Gene 4
Outer membrane protein A precursor fig|1246994.5.peg.1557

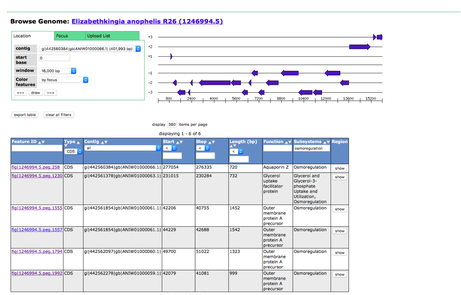
mRNA Count Control	mRNA Count Cefotax	mRNA Count Imipenem	mRNA Count Total	Fold change Cefotax	Fold Change Imipenem
53	77	42	172	1.5	-1.3

Gene 1557 does not have significant changes in the fact of it being less/greater than or equal to 1.5. It is slightly under the fifty percent. The significant changes came from the mRNA counts. The total count came to 172 when Cefotax, Imipenem, and the control count was placed in the significance.

Gene 5
Outer membrane protein A precursor fig|1246994.5.peg.1555

mRNA Count Control	mRNA Count Cefotax	mRNA Count Imipenem	mRNA Count Total	Fold Change Cefotax	Fold Change Imipenem
1	1	2	4	-1.0	2.0

Gene 1555 does not have a significant change for Cefotax, but it does have a significant change for Imipenem. This gene did transcribe but it transcribed with really low numbers. So you cannot be confident with the change given above. Further testing would need to be done to further prove this information.



DISCUSSION

The genes we researched all had a similar specific purpose, and that purpose was to the protect the cell itself. Without these genes the cell’s vulnerability would come from a lack of an outer membrane. These genes produce proteins that maintain the water and other fluids inside and out of the cell membrane (osmoregulation). This protein precursor A helps fight against “infection” within the cell; as in protection from antibiotics and environmental inhibitors like a change in temperature or a lack of oxygen.

One of our genes (1794) transcribes 55% more when exposed to Cefotax. Most of our other genes transcription went up, but not by more than 50%. The more they transcribed the more they “protected” the cell from the antibiotics.

So, the genes that produce protein precursor A are vital to the protection of Elizabethkingia anophelis, therefore these genes are vital to the existenece of this bacterium.

REFERENCES

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ABSTRACT

Group 19 researched genes 258, 1992, 1794, 1557, 1557. We found that most of our genes contribute to the transcription of the protein precursor A. This protein aids in the overall protection of the elizabethkingia anophelis cell wall.