Hormogonia Mutation of Touch Sensitive With the Removal of Pili in Cyanobacteria

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1 Introduction

Nitrogen fixing is a vital process to plants that helps promote growth so it can out produce other plants. Cyanobacteria is a particular set of bacteria that helps promote nitrogen-fixing. Cyanobacteria like Nostoc punctiforme, will produce a certain subtype of cells that allows the nitrogen-fixing increments called heterocysts. Cyanobacteria also has another photosensitive cell type that creates sugars called a vegetative cell[5]. Once these cells are produced the cell loses it ability mobilize, unless it differentiates back into hormogonia again. There are two ways of getting into hormogonia.One way is being activated with Hormogonia-inducing factors (HIF), in which a plant will secrete a certain chemical substance. This process is not well known.The other way is by depletion of nitrogen. When a severe enough depletion of a resource occur then a cyanobacteria has a mechanism to survive in the form of a blob-like spore called akinetes. Hormogonia is a linear structure that is used for mobility of the cyanobacteria. How it mutates can help us understand whether or not the hormogonia has a touch sensitive mechanism that drives it to go back to normal.

These genes were found by using a microarray, and then read to be either up-regulated or down-regulated during a twenty four hour time interval when compared to other species of bacteria that had similar chemotaxis on a genomic level. The six types of chemotaxis input sensation that the scientist looked at were Methyl-accepting chemotaxis proteins(MCP), chemotaxis A(CheA), chemotaxis B(CheB), chemotaxis R(cheR),chemotaxis w(CheW), chemotaxis Y(CheY)[2]. All these proteins together help with the the bacteria's ability to find the source of food. This is to be considered when it starts smelling the food and goes into a tumble[1]. In the previous experiments there were several genes that caused hormogonia to move very little or became immobile towards light. Then in the same experiment they also had one go all over the place with light. They were all a part of the MCP mutation that had occurred and one of them had a special interaction. The experiment had some cyanobacteria had moved closer while some of them had not moved at all and others had some weird changes in their functionality. One of the weirder changes to function was hormogonia differentiated itself out into any S-shaped with the mutation of MCP.

The genes were compared to other bacterias with similar pilius IV motors because they looked similar in genomic. The scientist assumed a similar functionality. The genes in which the experiment was based upon was Prepilin (pilA), Prepilin peptidase (pilD) , pilus retraction (pilT) .When removing pili it does hamper movement, as well as the cyanobacteria in the form of hormogonia has a lose of symbioses[4].If there are too many pili then there is also a lose in mobility and a lost of symbiose. The pili also influence the mechanism behind the hormogonia movement.

2 Experiment

We are going to have to make AA/-N plates and add streptomycin on the plates [3].We are going to buy some of the old Nostoc from meeks lab with the omega mutation in the site with that exact site of mutation into the MCP[2]. Here is a way for us to make an E.coli k-12 into an resistant stranded of Ampicillin that has a sucrose and a streptomycin as way to promote the primer region of hind-III[6] . Once the ampicillin resistance have been placed into a plasmid it should be able to want to conjugation itself into the Nostoc punctiforme with the MCP mutation. We would have to test to see how many mutates had survived by add sugar onto the new plates after a numbers of hours in which should cause a majority of them to explode. My hope is to select for the mutation that has only ampicillin resistances . We could test this by having a Southern blot to verify that these genes have been implanted properly.

3 Discussion

The realization of this mechanism may not change a thing and instead may create several more questions . Like for instance this gene might actually be an initiation for the akinetes and that could be the reason why forms into any S -shape. It could also be that there is no touch mechanism in cyanobacteria. There could be a number of other variables I have not accounted for. If mutation does go back to normal it will not be able to expand because of the removal of pilius and that will cause it to be immobilized. The variability of the two genes being able to be switched on and cut out at the right points may cause it die immediately.

4 Reference

References

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