Lab Report 6

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1

The ubiquitous cyanobacterium *Prochlorococcus marinus* SS120 is missing a gene ketopantoate reductase, which is involved in pantothenate synthesis (Figure 1). However, *Prochlorococcus marinus* is known to grow in pure culture without the addition of pantothenate, so another gene must be replacing its function. I looked up ketopantoate reductase on EcoCyc and found that acetohydroxy acid isomeroreductase can replace its function. To check if acetohydroxy acid isomeroreductase is present in the *Prochlorococcus marinus* genome, I looked up the gene on MicrobesOnline and found that it was present in every Prochlorococcus genome on the website. This suggests that acetohydroxy acid isomeroreductase replaces the function of ketopantoate reductase in *Prochlorococcus marinus* SS120.

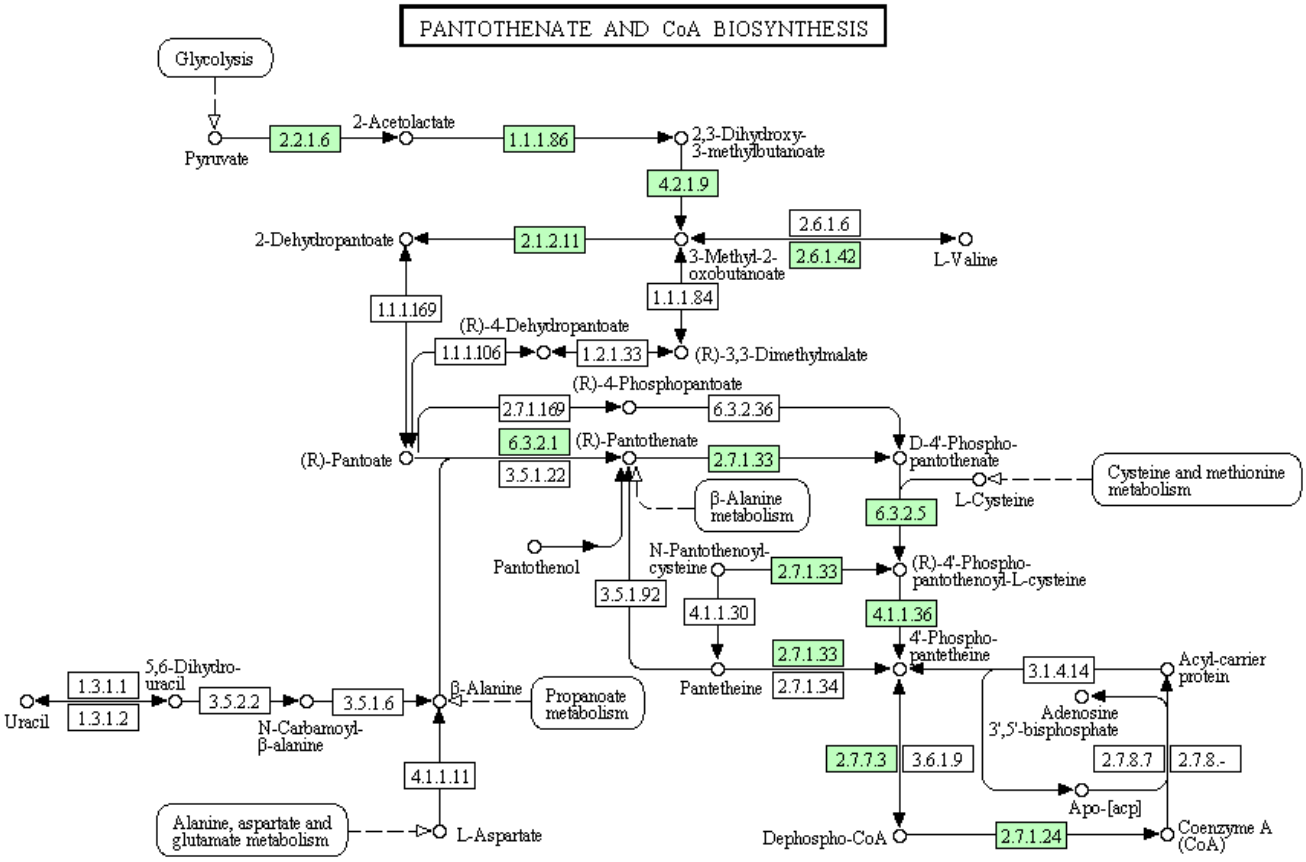

Figure 1: KEGG pathway for pantothenate synthesis

2

Pelagibacter HTCC1062 is missing the thiC gene involved in the synthesis of thiamine. thiC allows purine metabolism end products to be converted to a precursor of thiamine, 4-amino-5-hydroxymethyl-2-methylpyrimidine. However, *Pelagibacter* HTCC1062 has the enzymes to convert 4-amino-5-hydroxymethyl-2-methylpyrimidine to thiamine 2. In such a reduced genome, it is not likely that these genes downstream to thiC are not in use do to the lack of substrate, so thiC must be being circumvented somehow. One possibility is that the product of thiC, 4-amino-5-hydroxymethyl-2-methylpyrimidine, is being absorbed directly from the ocean water. When searching the literature for the presence of 4-amino-5-hydroxymethyl-2-methylpyrimidine in the ocean, I found a paper out of the glorious and infallible Giovannoni lab, that confirmed my suspicion [1]. Carini et al. 2014 state that 4-amino-5-hydroxymethyl-2-methylpyrimidine is necessary for the growth of textitPelagibacter in small amounts and confirmed its presence in the open ocean.

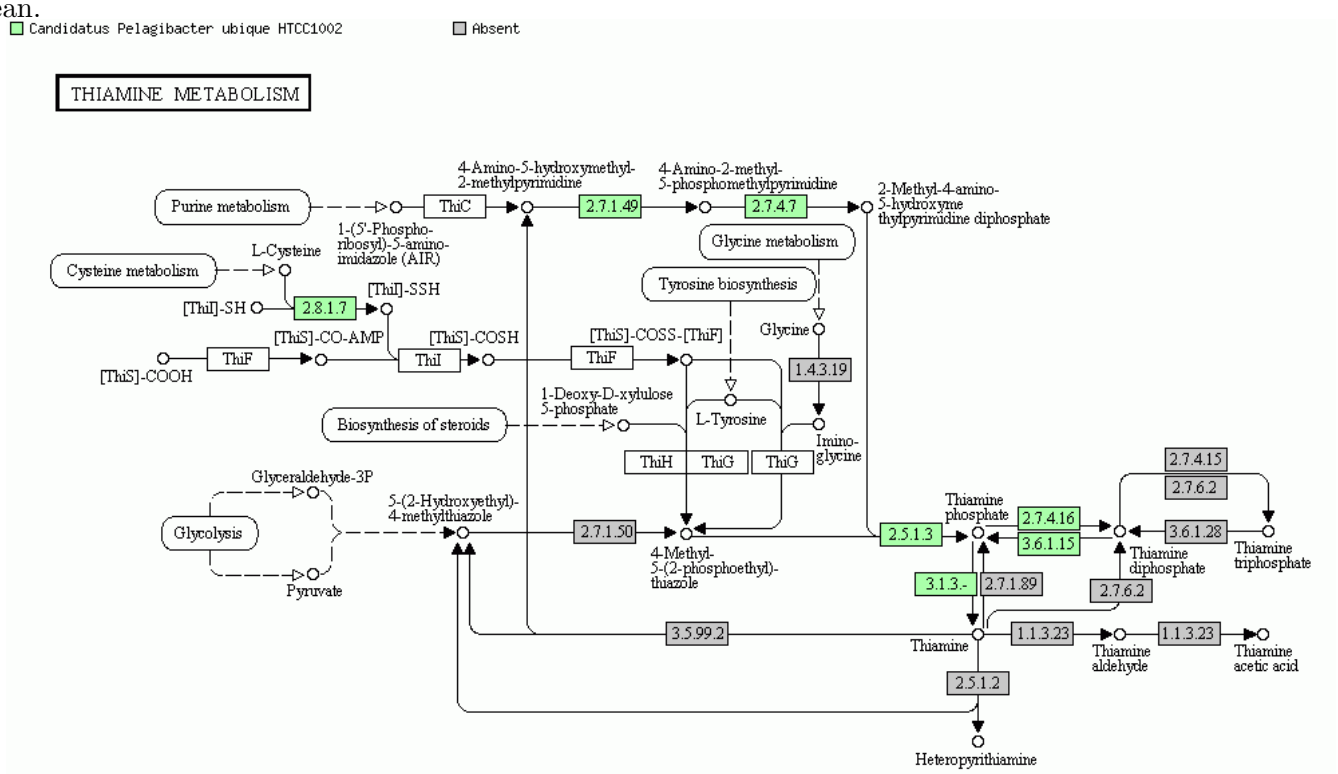

Figure 2: KEGG pathway for thiamine synthesis in *Pelagibacter*

3

The putative carbon monoxide dehydrogenase gene in Janibacter strain HTCC2649 is thought to be a misannotation, since there is no evidence that Janibacter strain HTCC2649 can use carbon monoxide as a substrate. I searched for the carbon monoxide dehydrogenase gene in Janibacter strain HTCC2649 on MicrobesOnline and found another annotation as an xanthine dehydrogenase. xanthine dehydrogenase is involved in the degradation of xanthine and hypoxanthine to urate, a process which reduces two NAD+ to NADH (Figure 3). In the same operon of the putative monoxide dehydrogenase gene in Janibacter strain HTCC2649 there is a urate permiase (Figure 4). This suggests that the misannotated gene could in fact be a xanthine dehydrogenase that allows for the breakdown of nucleic acid via xanthine and hypoxanthine. In this case, the associated urate permiase could function to remove the urate from the cell as a waste product. This would imply that Janibacter strain HTCC2649 can derive energy from the breakdown of nucleic acids, excreting urate in the process. This could be tested by comparing the growth rate of

Janibacter strain HTCC2649 in pure culture on minimal media with differing amounts of nucleic acids as substrates. Increased growth in response to higher levels of nucleic acid and a corresponding increase in urate concentration in the media would support this hypothesis.

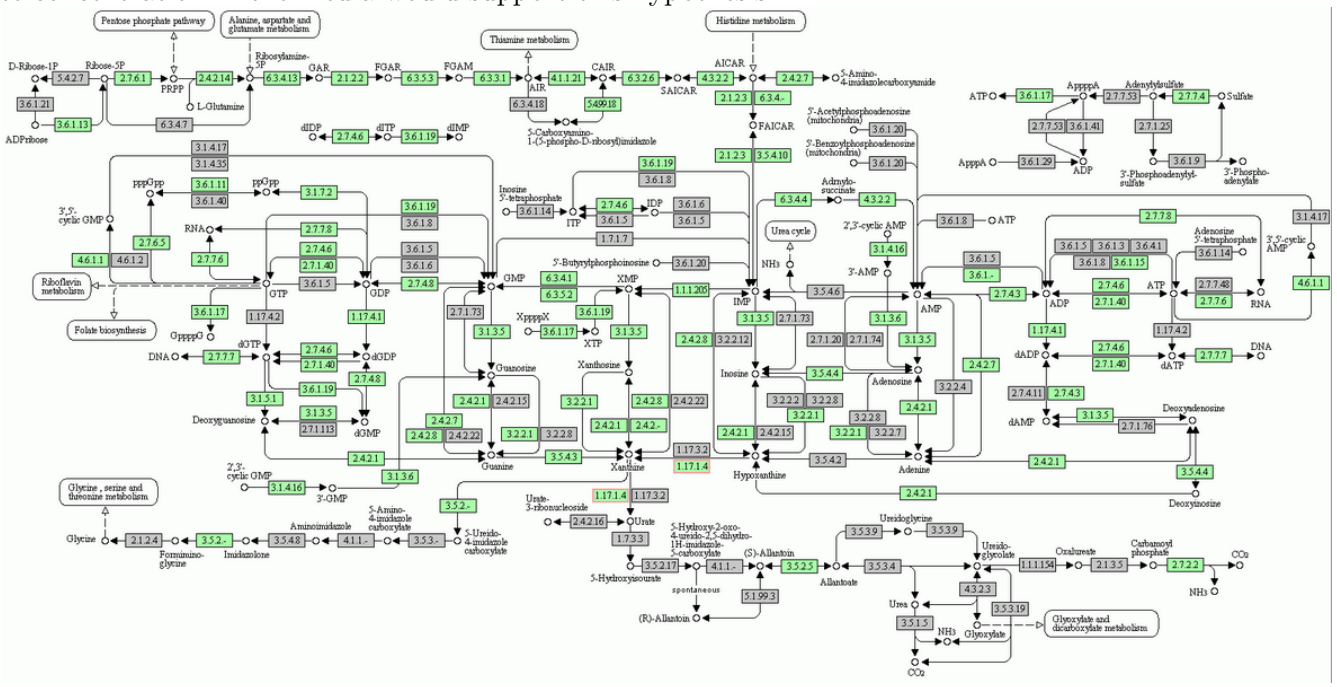

Figure 3: KEGG pathway for Purine metabolism in *Janibacter*

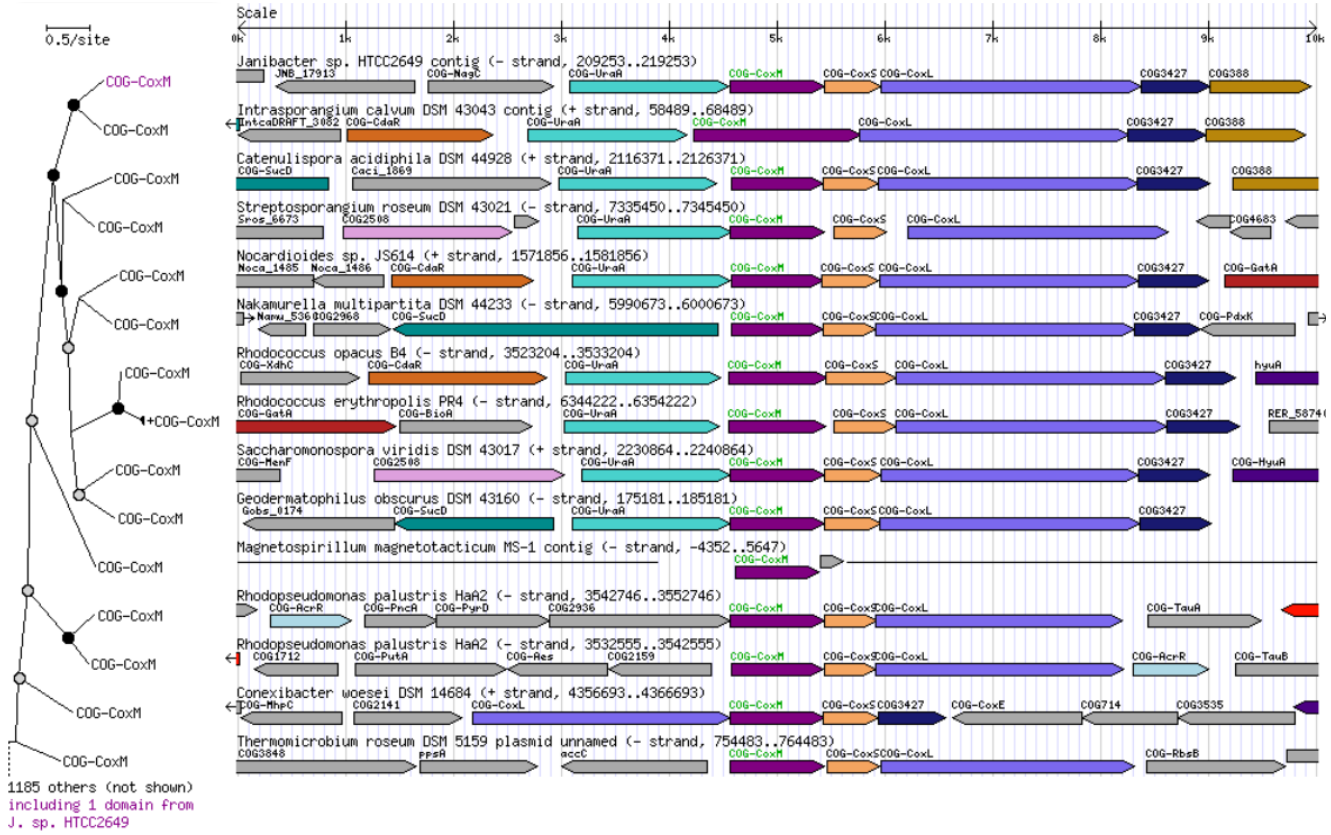

Figure 4: MicrobesOnline tree view for genomic regions of organisms closely related to *Janibacter*

References

[1] Paul Carini et al. "Discovery of a SAR11 growth requirement for thiamins pyrimidine precursor and its distribution in the Sargasso Sea". en. In: *The ISME Journal* (Apr. 2014). ISSN: 1751-7362. DOI: 10.1038/ismej.2014.61. URL: http://www.nature.com/ismej/journal/vaop/ncurrent/full/ismej201461a.html (visited on 05/16/2014).