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Identification of potential dna elements that may regulate sphingolipid metabolizing enzymes and ceramide transport protein using bioinformatics tools

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Sphingolipid pathway plays a major role in anti-cancer drugs and radiation induced cell death. Ceramide, a pro-apoptotic molecule is central to sphingolipid metabolism. Ceramide released from sphingomyelin by sphingomyelinase in response to various stimuli, has been shown to induce cell death in variety of cells, including those of cancer. However, the presence of intra-cellular ceramide is transient as the ceramide gets metabolically converted in to various other sphingolipids that are either pro or anti-apoptotic. This suggests the importance ofmaintaining the ratio of sphingolipids in inducing cell death.

We hypothesize that down regulating the enzymes that convert ceramide in to either C-1-P and / or S-1-P keep the intracellular ceramide levels high thus increasing the sensitivity to anti-cancer treatment modalities. To test the hypothesis, we have been looking at various factors like DNA elements, mutations in the genes, small molecule inhibitors and activators, and ceramide transfer protein (CERT) that may contribute to upregulation or down regulation of these enzymes.

In this regard, we are investigating for the possible presence of DNA elements that may regulate expression of genes involved in ceramide synthesis and its metabolism. Investigations of the present study included genes which encode ceramide kinase (CERK), sphingosine kinases (SPHK1&2), sphingosine-1-phosphate lyase (SGPL1), ceramidases (1,2 &3), glucosidase-β- acid (GBA) and galactocerebrosidase (GALC).

The data obtained so far reveals the presence of DNA regulatory elements in ceramide synthases and enzymes involved in ceramide conversion but not in ceramide transfer protein.

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