

## Research Proposal Abstract:

Vitamin B9 (Folate) is an important component of normal metabolism, involved in the methylation of DNA. Methylation or Demethylation of DNA is used to turn genes on or off, thereby regulating the expression of certain proteins. Alterations in DNA methylation can lead to certain conditions, such as Alzheimer's disease. Because various disease states and cognitive deficits have been linked to impaired folate metabolism, we suspected that gene activity is altered in animals based on the timing and amount of dietary folate intake. We used a murine model to test this hypothesis, by administering differing amounts of folate to each group in our study. We discovered that vitamin B9 deficiency correlates to short-term memory deficiency and vitamin B9 causes specific genes to be activated or repressed in the brain. Microarray analysis showed that certain genes were activated or repressed in folate deficient diets. We were also able to confirm the results of the microarray analysis in the lab through qPCR analysis. We then analyzed gene ontologies to investigate the possible biological pathways that these genes may be involved in. This study is an important because it builds a strong case for epigenetic basis of biological disorders, suggesting that disease conditions may not only result from mutational abnormalities only, but acquired environmental traits as well.