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Improving mouse-DMH screening capability by adding 2000 mouse CpG islands

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DNA methylation alteration, in correlation with gene expression, is involved in development and progression of many cancers. Using a microarray based method, mouse-DMH (Differential Methylation Hybridization), our lab is able to study DNA methylation changes during prostate cancer progression in the TRAMP (Transgenic Adenocarcinoma of Mouse Prostate) mouse model. Currently, there are about 3000 CpG islands on the microarray, which were used as probes to detect DNA methylation changes.

In order to improve our ability to screen for a larger number of CpG island methylation changes, we are working on adding about 2000 more mouse CpG islands onto the array. In addition, we have successfully designed primers and PCR amplified CpG islands for tumor suppressor genes and proto-oncogenes which have been previously reported in literature to be differentially methylated during development of human prostate cancer. These genes include AR (Androgen Receptor), ER α (Estrogen Receptor alpha), ER β (Estrogen Receptor beta) and GSTP1 (Glutathione S-Transferase PI). Primer design and PCR amplification for other known tumor suppressors/oncogenes is still in process.

The microarray-based mouse-DMH is a tool of great potential. It can easily be adapted to screen for DNA methylation changes in other mouse cancer models and generate valuable data leading to understanding of the molecular mechanism behind cancer development, which will in turn contribute to treatment of human cancers.