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Longer-term Cigarette Smoke Extract Exposure-Induced Methylation and Expression Changes in GSTP1, in Human Lung Cells

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Methylation at the 5'-position of cytosine in CpG dinucleotides is a common modification of DNA in vertebrate genomes. In many types of human cancers, aberrant promoter hypermethylation is a major mechanism for silencing tumor suppressor or other cancer-associated genes, such as genes involved in carcinogen activation or detoxification, DNA repair, metastasis and invasion. Glutathione S-transferase Pi (GSTP1) is the major phase II detoxification enzyme in non-malignant human lung and it is found to be frequently hypermethylated in various cancers. In a previous study, a method using tag-modified bisulfite genomic sequencing (tBGS) was developed in our lab, and we found the methylation maps for the GSTP1 promoter diverged between NHBE (normal human bronchial cells, unmethylated) and A549 (lung carcinoma cells, completely methylated). We now continue to use tBGS to measure the methylation status of GSTP1 in cultured bronchial cells, both primary normal, and immortalized normal cells, which are continuously exposed to cigarette smoke extract (CSE) up to 3 months. Results of methylation changes will be presented, as will progressive expression changes, for phase II gene GSTP1.