Commentary

# Environmental genetic diseases

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Several studies have shown that utero-environment is associated with disease risk, including coronary heart disease type 2 diabetes, childhood obesity as well as psychiatric problems and disorders. Smoking during pregnancy influences widespread and highly reproducible differences in DNA methylation at birth. Less dramatic effects have been reported for maternal body mass index (BMI) pre-eclampsia and gestational diabetes possible epigenetic changes as a consequence of prenatal stress are less well established. Some of these early differences in DNA methylation persist, although attenuated, through childhood and might be related to later symptom s and indicators of disease risk, including BMI during childhood or substance use in adolescence. Methylation quantitative trait loci (meQTLs, i.e., SNPs significantly associated with DNA methylation status the association of meQTLs with DNA methylation is relatively stable throughout the life course.

**Key Words:** Utero-Environment; DNA Methylation; Coronary Heart Disease

## DESCRIPTION

In addition, SNPs within meQTLs are strongly enriched for genetic variants associated with common disease in large genome-wide association studies (GWAS) such as BMI, inflammatory bowel disease, type 2 diabetes or major depressive disorder. With trauma associated changes only observed in carriers of the rare allele. The most comprehensive study of integrated genetic and environmental contributions to DNA methylation so far was performed by The et al. This study examined variably methylated regions (VMRs), defined as regions of consecutive CpG-sites showing the highest variability across all methylation sights assessed on the Illumina Infinium HumanMethylation450 Bead Chip array. Prenatal environmental factors such as maternal BMI, maternal glucose tolerance and maternal smoking on DNA methylation at VMRs. They found that 75% of the VMRs were best explained by the interaction between genotype and environmental factors (GxE) whereas 25% were best explained by SNP genotype and none by environmental factors alone. Specifically, this is addressed by evaluating the relevance of genetic variants that interact with the environment to shape the methylome for their contribution to genetic disease risk. Finally the enrichment of genetic variants within additive as well as interactive models in GWAS for complex disorders supports the importance of these environmentally modified methylation quantitative trait loci for disease risk.

## CONCLUSION

The levels of urinary DNA damage markers Guam and NNAL may be an appropriate exposure marker for evaluating the smoking status of patients in a smoking cessation program. Decreased with the duration of smoking cessation, in the same manner as the smoking exposure markers (nicotine, cotinine, and NNAL). The urinary levels of cotinine and NNAL positively correlated with the Guam levels.

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