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Gene by Prenatal Alcohol Exposure Interaction Effects on Growth and Cognition in Mother-Child Dyads in a South African Birth Cohort

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Abstract:
Background: Fetal Alcohol Spectrum Disorders (FASD), caused by prenatal alcohol exposure (PAE), are a leading, preventable cause of neurodevelopmental delay. Despite phenotypic variability in FASD children, maternal and child genetic influences in FASD remain largely unknown. This study employs comprehensive single-marker and gene-level analyses to identify FASD-associated risk markers and loci utilizing mother-child dyad data from two longitudinal birth cohorts in South Africa, where the prevalence of FASD is among the highest in the world. **Methods:** Illumina MegaEX genotype data from mother-child dyads were analyzed in single-marker analysis was using Mixed Model Association to study gene-environment interactions (alcohol consumption during pregnancy) on FASD outcomes, including diagnosis, working memory, recognition discrimination, and height. Covariates included maternal age, socioeconomic status, choline supplementation, and prenatal cigarette exposure. Subsequently, gene-based analysis was performed using GCTA based on single-marker summary statistics. **Results:** Separate gxe analyses of mother and child genotype data identified unique genetic biomarkers in both groups. In the single marker analysis for reduced child height, the top SNPs identified were *rs61852292* and *rs10782983* from child and maternal genotype data, respectively (beta = 4.13, p = 1.1e-6; beta = 1.19, p = 2.9e-8). In the gene-based analysis for child height, 150 genes were nominally significant in both mothers and children, including *STX6* (p = 2.06e-6/6.9e-4 (child/mother)) and *MR1* (p = 3.31e-4/9.3e-3). For recognition discrimination, 107 genes were nominally significant in both strata, including *FOXD3* (p = 3.3e-5/0.023) and *IQGAP3* (p = 1.9e-3/1.4e-5). Additionally, several gene markers in candidate methyl donor metabolism pathway genes were significant for child height reductions: *FADS1*, *FADS2* (p = 0.012), *ACTR8* (p = 5.4e-3), and *MAT2A* (p = 0.03) in children; in the mothers, *MTHFR* (p = 0.015) and *MTHFD2* (p = 0.033). **Discussion:** These findings identify novel genetic markers associated with FASD, suggesting mechanistic roles and potential therapeutic targets. Some markers are linked to neurological disorders. Methyl donor metabolism-related genes, notably affected, suggest that choline treatment could mitigate prenatal alcohol exposure effects, highlighting clinical relevance from both animal and human studies. **Conclusion:** Comprehensive single-marker and gene-based analyses of mother and child genotype data revealed statistically significant markers demonstrating interaction effects with prenatal alcohol exposure on FASD outcomes.

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