

Association of School Connectedness with Suicidal Ideation Resistance Among Youth at Elevated Polygenic Risk

Substance use and suicidal ideation (SI) frequently occur during adolescence, with SI representing a major risk factor for substance use onset and progression. Although SI is heritable, not all individuals at elevated genetic risk for SI will express this phenotype. Thus, identifying protective factors that explain discrepancies between genetic liability and observed SI (hereafter, SI resistance) is a priority, which may have downstream consequences for reducing substance use and other negative sequelae. We tested associations between SI resistance and positive childhood experiences (PCEs) in the Adolescent Brain Cognitive Development Study (ABCD) (N=3,212) using multiple waves of data (M age=12.03-16.01). SI polygenic scores (PGS) were derived from a GWAS of SI in the Million Veterans Program (Neff=213,029). PCEs were derived from a prior ABCD factor analysis, yielding three factors: neighborhood safety, parental attachment, and school connectedness. Active SI (thoughts of suicide with a specific plan) and passive SI (thoughts of dying without a plan) were assessed at three time points based on parent- and child-reports. A growth model was fit to SI resistance to model developmental changes with SI at each wave was regressed on SI PGS. The scale of the residuals was inverted, such that larger latent scores index greater SI resistance (i.e., high genetic risk paired with low observed SI). We observed significant associations of school connectedness with the intercepts of passive SI resistance ($\beta=0.12$, 95% CI [0.07, 0.18]) and active SI resistance ($\beta=0.08$, 95% CI [0.02, 0.14]). Findings indicate that school connection in early adolescence contributes to SI resistance among youth at higher genetic risk, but does not predict changes in SI resistance over time. School-based interventions may promote social integration and safety in school settings, thereby buffering genetic liability for SI and disrupting developmental pathways to substance use.

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