TITLE: Long-Term Effects of Polygenic Propensity for Educational Attainment on Educational Attainment Mediated by Inhibitory Control and Variation by a Family-Based Intervention

FULL AUTHOR LIST: Giulia A. Borriello¹, Kit K. Elam², Gianna Rea-Sandin³, Kathryn Lemery-Chalfant³, Fazil Aliev⁴,⁵, Danielle Dick⁵, and Thao Ha³

AFFILIATIONS: ¹ Department of Psychological and Behavioral Sciences, Indiana University Bloomington, IN, USA; ² Department of Applied Health Sciences, Indiana University Bloomington, IN, USA; ³ Department of Psychology, Arizona State University, Tempe, AZ, USA; ⁴ Faculty of Business, Karabuk University, Turkey; ⁵ Department of Psychology, Virginia Commonwealth University, Richmond, VA, USA

KEYWORDS: Educational attainment; Continuous shrinkage; Polygenic risk score; Inhibitory control; Adolescence

ABSTRACT:

The majority of studies on educational attainment (EA) focus on direct genetic effects rather than on how etiological influences unfold across development. The current study investigates genetic influences in developmental pathways to EA over a 12-year period in a sample of at-risk youth who participated in a family-based intervention program. We tested for direct effects of a polygenic score (PS) for EA on adulthood EA, mediated by adolescent inhibitory control, and for differences in effects based on intervention status.

Data were from the Project Alliance 1 study (PAL1), a longitudinal randomized trial of 999 adolescents (51% male) and their families. Participants were randomly assigned to intervention or control conditions of the Family Check-Up (FCU), designed to reduce adolescent problem behavior. Genetic propensity for EA was captured using a continuous
shrinkage PS based on a recent GWAS of EA. Adolescent inhibitory control was assessed at age 17 and EA at age 27. All analyses controlled for parents’ EA, ethnicity, age, and the first two ancestry principal components.

The PS directly predicted EA ($B = .15, SE = .04, p < .001$). In the mediated model the PS predicted inhibitory control ($B = .14, SE = .05, p = .002$), inhibitory control predicted EA ($B = .21, SE = .04, p < .001$; indirect effect $B = .03, SE = .01, p = .02$), and a significant direct effect remained ($B = .12, SE = .04, p = .001$). The association between the PS and EA was significantly different [$\chi^2_{\text{diff}}(1) = 5.68, p = .017$] in intervention ($B = .04, SE = .06, p = .56$) and control conditions ($B = .20, SE = .06, p < .001$). Results highlight individual and FCU mechanisms in pathways to EA.

GRANT SUPPORT: R01 DA007031, R01 DA036832, Grant DA07031 from the National Institute on Drug Abuse, Grant AA022071 from the National Institute on Alcoholism and Alcohol Abuse