

NAME OF PRESENTING AUTHOR: Amanda Shrewsbury

EMAIL ADDRESS OF PRESENTING AUTHOR: ashrews1@asu.edu

LOCATION OF PRESENTING AUTHOR:  
America (North)

TIME ZONE OF PRESENTING AUTHOR: USA, Pacific Standard Time (PST)

TYPE OF SUBMISSION: Poster

MEMBER STATUS:  
Associate

ELIGIBLE FOR THOMPSON AWARD: No  
ELIGIBLE FOR ROWEWARD: Yes

---

TITLE:

Genetic and environmental influences on externalizing symptoms and aspects of executive function in middle childhood

FULL AUTHOR LIST:

Amanda M Shrewsbury<sup>1</sup>, Gianna Rea-Sandin<sup>1</sup>, Sierra Clifford<sup>1</sup>, Leah D. Doane<sup>1</sup>, Kathryn Lemery-Chalfant<sup>1</sup>

AFFILIATIONS:

<sup>1</sup>Department of Psychology, Arizona State University, Tempe, Arizona, USA

KEYWORDS:

executive function, externalizing symptoms, genetics, childhood, twin

ABSTRACT:

Research has linked deficits in executive function (EF), such as shortfalls in inhibition and cognitive flexibility, with externalizing problems in childhood (e.g., Schoemaker et al., 2013, *Journal of Abnormal Child Psychology*, 41, 457-471). However, quantitative genetic research examining associations between EF and externalizing problems in middle childhood is scarce. This study examined the genetic and environmental variation in externalizing symptoms and EF in a middle childhood sample. The sample comprised 710 twins (55.6% White, 28.3% Hispanic, 3.9% Black; 51.5% female; *M* age = 8.42 years, *SD* = 0.68) from the Arizona Twin Project (Lemery-Chalfant et al., 2019, *Twin Research and Human Genetics*, 22, 681-685). Externalizing symptoms were assessed with

the Health and Behavior Questionnaire, and EF was assessed with the Conner's Continuous Performance Task (CPT; Conners, 2000, *Multi-Health Systems*) and the Eriksen Flanker Task (Eriksen & Eriksen, 1974, *Perception & Psychophysics*, 16, 143-149). Univariate ACE models indicated that additive genetics ( $A=.57$ ), shared ( $C=.32$ ), and non-shared ( $E=.11$ ) environmental influences explained variation in externalizing symptoms, whereas an AE model best represented CPT ( $A=.49$ ,  $E=.51$ ) and Flanker ( $A=.48$ ,  $E=.52$ ) data. Our findings suggest the importance of additive genetic influences for these traits. However, phenotypic correlations were low for externalizing symptoms with CPT data ( $r=.14$ ) and with Flanker data ( $r=.145$ ), resulting in errors when attempting to fit bivariate models to examine covariation. Given robust associations between EF and ADHD in the literature, future directions include fitting a univariate model for ADHD symptoms and bivariate models of EF and ADHD symptoms in this sample.

GRANT SUPPORT:

R01HD079520

R01HD086085

---

---