

Concept Paper Form

Provisional Paper Title: Etiology of low self-control across two cohorts: the moderating effect of socio-economic status
Proposing Author: Langevin, Stephanie
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P.I. Sponsor: Boivin, Michel; Caspi, Avshalom; Moffitt, Terrie* (if the proposing author is a student or colleague of an original PI) *In alphabetical order
Today's Date: 3/24/2021

Objective of the study:

Childhood low self-control is a major risk factor for a wide variety of adverse developmental outcomes. Associations have been reported between low self-control and externalizing (e.g., antisocial, criminal behaviors) and internalizing (e.g., self-harm) behaviors, poor physical health (e.g., smoking, substance abuse, weight gain, lower academic achievement and wealth) and poorer social functioning (e.g., prosocial peer rejection, association with antisocial peers, unplanned teenage pregnancy, sexual infidelity, unsafe sex) in adolescent, adult and elderly participants (Chapple, 2005; Moffitt et al., 2011; Moffitt et al., 2013; Piquero, MacDonald, Dobrin, Daigle, & Cullen, 2005; Pratt & Cullen, 2000; Richmond-Rakerd et al., 2019; Tangney, Baumeister, & Boone, 2004; Wolfe, 2015; Wolfe, Reisig, & Holtfreter, 2016).

Since the publication of Gottfredson and Hirschi's (1990) self-control theory, in which variability in antisocial behaviors is attributed to variability in self-control, self-control has become a key feature in criminological research. However, at least two features of the Self-control theory have been disputed. The first widely disputed postulate is that self-control acquisition (and lack thereof) is wholly attributable to parental and school socialization in infancy and childhood. Indeed, results from twin studies have reported that the heritability of self-control is of approximately 60%, with unique environmental contributions accounting for the remaining 40% of the variance (Willems, Boesen, Li, Finkenauer & Bartels, 2019). However, these studies have mostly been conducted on samples of adolescent and adult participants. Consequently, it remains unclear whether the heritability estimates drawn from these studies generalize to younger children.

The second disputed postulate claims that the etiology of self-control is unaffected by individual or structural characteristic other than early parental and school socialization, such as family

socioeconomic status (SES) and neighborhood SES. Contrasting with this postulate, scientific evidence linking socioeconomic strains and executive functions and self-control in childhood have reported that lower SES was associated with lower executive functions and lower levels of self-control (Lawson, Hook, & Farah, 2018; Vrantsidis, Clark, Chevalier, Espy & Wiebe, 2020). To date, however, it remains unclear whether distinct genetic and environmental contributions to youth's low self-control emerges according to family and neighborhood SES. To test this hypothesis, we propose to investigate the etiology of self-control in two twin cohort studies: the Quebec Newborn Twin Study (QNTS; Boivin et al., 2019) and the E-Risk study.

This study has three primary aims:

Aim 1) To investigate the genetic and environmental contributions to individual variability in childhood low self-control (age 5-10 years) in the E-Risk study. A previously published measure of self-control will be used (Moffitt et al., 2011; Richmond-Rakerd, et al., 2019).

Building on previous meta-analyses of self-control in order participants as well as meta-analyses of phenotypes closely related to self-control (e.g., antisocial behaviors), we hypothesize the heritability estimates to account for 50-70% of the variance in childhood self-control (Lubke et al., 2018; Polderman et al., 2015; Porsch et al., 2016; Willems et al., 2019).

Aim 2) To test whether the relative contribution of genetic and environmental factors to individual differences in childhood low self-control (age 5-10 years) varies according to family SES and neighborhood SES in the E-Risk cohort (gene-environment interaction, GxE). To do so, we plan to use the self-control and SES measures previously published (Belsky et al., 2019; Moffitt et al., 2011; Newbury et al., 2018; Odgers, Donley, Caspi, Bates, & Moffitt, 2015; Richmond-Rakerd, et al., 2019).

We hypothesize that similarly to previous GxE investigations of SES and antisocial behaviors (Tuvblad, Grann & Lichtenstein, 2005), higher heritability estimates will be noted in the context of lower environmental adversity whereas stronger environmental contributions are expected in the context of higher environmental adversity. Such findings would fall in line with Raine's social push model, which posits that highly adverse environments may mask biological risk factors that would have been otherwise more likely to be detected in the absence of these adverse environments (Raine et al., 2002).

Aim 3) To test whether the GxE findings from the E-Risk cohort replicate in early childhood (age 18 months to 5 years) in an independent study, i.e. the QNST cohort. To do so, we will investigate the moderating effect of family SES on the etiology of low self-control. The consideration of two independent twin cohorts provides two clear advantages: the possibility of replicating and generalizing results across two cohorts, and the capacity to test whether there are developmentally sensitive periods over the course of childhood (18 months to 5 years VS. 5-10 years) with regards to the etiology of low self-control.

Data analysis methods:

Firstly, univariate genetic modeling analyses will be conducted to examine the genetic and environmental etiology of twin participants of the E-Risk and QNTS studies. Structural equation modeling will be conducted to estimate the relative contribution of genetic (A), shared (C) and unique (E) environments. Secondly, univariate models testing the genetic and environmental interaction between low self-control's latent factors (A, C and E) and socio-economic status in childhood. The models will be assessed using χ^2 -difference tests, AIC, BIC, and RMSEA, whereby the full ACE model will be compared to more restrictive models. Analyses will be conducted using Mplus.

Variables needed at which ages:

Study	Variable label	Variable description
E-Risk	FamilyID	Family ID
	Rorder	Random twin selection
	ATWINID	Twin A ID
	BTWINID	Twin B ID
	SAMPSEX	Sex of child
	Zygosity	Zygosity of child
	Seswq35	Family SES
	ACORN	Neighborhood SES
	IMD	UK government index of deprivation
	ECORISK	Individual indicators of ecorisk: deprived, dirty, dangerous, disorganized, until age 10 (from Belsky et al., 2019)
	Lowsc510e	Low self-control, ages 5-10
QNTS	As prescribed by the Research Unit on Children's psychosocial maladjustment, a separate concept paper for the QNTS data has been submitted.	

Significance of the Study (for theory, research methods or clinical practice):

Results from twin studies have estimated that heritability accounts for ~60% of the variance in self-control among adolescents and adults. Previous studies of singletons have further reported the association between low self-control and individual and structural adversity, such as low family and neighborhood socioeconomic status. Although these studies have provided important insight into the etiology of low self-control, they have overlooked a possible gene-environment interplay in those associations. The contributions of the current study to the field of criminology are both theoretical and potentially practical. First, the study will provide a test of two core postulates of Gottfredson and Hirschi's self-control theory by investigating the genetic and environmental etiology of low self-control in early childhood (18 months to 5 years) and childhood (5 to 10 years), and by investigating whether heritability and environmental estimates are conditional upon socioeconomic status. Second, findings could have potential implications for clinical research and practice, whereby a better understanding of the roots and the factors influencing the acquisition (and lack thereof) of self-control among children may provide more insight to help bolster self-control among at-risk children and promote healthy and successful like trajectories in these children. Indeed, if gene-environment

interactions are identified, then different prevention and intervention approaches could be considered according to different socioeconomic areas.

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Data Security Agreement

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<input checked="" type="checkbox"/>	I am current on Human Subjects Training (CITI (www.citiprogram.org) or equivalent)
<input checked="" type="checkbox"/>	My project is covered by the Duke ethics committee OR I have /will obtain ethical approval from my home institution.
<input checked="" type="checkbox"/>	I will treat all data as "restricted" and store in a secure fashion. My computer or laptop is: a) encrypted (recommended programs are FileVault2 for Macs, and Bitlocker for Windows machines) b) password-protected c) configured to lock-out after 15 minutes of inactivity AND d) has an antivirus client installed as well as being patched regularly.
<input checked="" type="checkbox"/>	I will not "sync" the data to a mobile device.
<input checked="" type="checkbox"/>	In the event that my laptop with data on it is lost, stolen or hacked, I will immediately contact Moffitt or Caspi.
<input checked="" type="checkbox"/>	I will not share the data with anyone, including my students or other collaborators not specifically listed on this concept paper.
<input checked="" type="checkbox"/>	I will not post data online or submit the data file to a journal for them to post. <i>Some journals are now requesting the data file as part of the manuscript submission process. Study participants have not given informed consent for unrestricted open access, so we have a managed-access process. Speak to Temi or Avshalom for strategies for achieving compliance with data-sharing policies of journals.</i>
<input checked="" type="checkbox"/>	I will delete all data files from my computer after the project is complete. Collaborators and trainees may not take a data file away from the office. This data remains the property of the Study and cannot be used for further analyses without an approved concept paper for new analyses.
<input checked="" type="checkbox"/>	I have read the Data Use Guidelines and agree to follow the instructions.

Signature: Stephanie Langevin

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Proposing Author	Langevin, Stephanie
Other Contributors* *in alphabetical order	Arseneault, Louise; Boivin, Michel; Brendgen, Mara; Caspi, Avshalom; Dionne, Ginette; Moffitt, Terrie; Ouellet-Morin, Isabelle; Vitaro, Frank
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Please check your contribution(s) for authorship:

<input type="checkbox"/>	Conceptualizing and designing the longitudinal cohort study
<input type="checkbox"/>	Conceptualizing data collection protocols and creating variables
<input type="checkbox"/>	Data collection
<input type="checkbox"/>	Conceptualizing and designing this specific paper project
<input type="checkbox"/>	Statistical analyses and interpretation (or reproducibility check)
<input type="checkbox"/>	Writing
<input type="checkbox"/>	Reviewing manuscript drafts
<input type="checkbox"/>	Final approval before submission for publication
<input type="checkbox"/>	Agreement to be accountable for the work
<input type="checkbox"/>	Acknowledgment only, I will not be a co-author

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