



Published in final edited form as:

*Alcohol Clin Exp Res.* 2021 June ; 45(6): 1249–1264. doi:10.1111/acer.14604.

## Associations of developmental imbalance between sensation seeking and premeditation in adolescence and heavy episodic drinking in emerging adulthood

Connor J. McCabe<sup>1</sup>, Tamara L. Wall<sup>1</sup>, Marybel R. Gonzalez<sup>1</sup>, Alejandro D. Meruelo<sup>1</sup>, Sonja C. Ebersson-Shumate<sup>1</sup>, Duncan B. Clark<sup>2</sup>, Kate B. Nooner<sup>3</sup>, Sandra Ann Brown<sup>1</sup>, Susan F. Tapert<sup>1</sup>

<sup>1</sup>University of California, San Diego, San Diego, CA, USA

<sup>2</sup>University of Pittsburgh, Pittsburgh, PA, USA

<sup>3</sup>University of North Carolina Wilmington, Wilmington, NC, USA

### Abstract

**Background:** Dual systems theories suggest that greater imbalance between higher reward sensitivity and lower cognitive control across adolescence conveys risk for behaviors such as heavy episodic drinking (HED). Prior research demonstrated that psychological analogues of these systems, sensation seeking and premeditation, change from childhood through emerging adulthood, and each has been independently linked with HED. However, few studies have assessed whether change over time in these developing analogues is prospectively associated with HED. Moreover, we know of no research that has shown whether within-person differences between higher sensation seeking and relatively lower premeditation across the adolescent period predict HED in emerging adulthood.

**Methods:** Prospective data from the National Consortium on Alcohol and NeuroDevelopment in Adolescence study ( $n = 715$ ) were used to examine the association of sensation seeking and premeditation with HED among adolescents ages 16 to 20 years. We used novel applications of latent difference score modeling and growth curve analysis to test whether increasing sensation seeking, premeditation, and their imbalance over time are associated with HED across the study period, and whether these associations differed by sex.

**Results:** Whereas premeditation increased linearly from adolescence through emerging adulthood across sexes, males reported growth and females reported decline in sensation seeking. Sensation seeking in adolescence (and not premeditation) was associated with higher levels of HED by emerging adulthood. Importantly, greater imbalance between sensation seeking and premeditation was associated with higher levels of HED by emerging adulthood though we note

---

**Correspondence** Connor J. McCabe, Department of Psychiatry, University of California, San Diego, San Diego, CA, USA. [cjmccabe@health.ucsd.edu](mailto:cjmccabe@health.ucsd.edu).

**CONFLICT OF INTEREST**

The authors declare no conflicts of interest.

**SUPPORTING INFORMATION**

Additional supporting information may be found online in the Supporting Information section.

that variability capturing this imbalance correlated highly ( $r = 0.86$ ) with baseline levels of sensation seeking.

**Conclusions:** Developmental imbalance between higher sensation seeking and lower premeditation in late adolescence may be a risk factor for greater HED in emerging adulthood.

### Keywords

adolescence; alcohol use; dual systems; impulse control; sensation seeking

---

## INTRODUCTION

Adolescence through emerging adulthood is a developmental period characterized by the emergence and escalation of problem substance use, including heavy episodic (i.e., binge) drinking (HED; Casey et al., 2008; Reyna & Farley, 2006; Stagman et al., 2011). For instance, national estimates in the United States indicated that young adults between ages 18 and 25 years (approximately 11.9 million people) report the highest rate of past month HED (34.9%) relative to both children under age 18 years (4.7%) and adults over age 25 years (25.1%; SAMHSA, 2019). HED during adolescence and emerging adulthood is a robust predictor of continued and/or escalating HED and alcohol use disorder (Chassin et al., 2002; McGue & Iacono, 2005), other drug use behaviors (McCarty et al., 2004; Viner & Taylor, 2007), and numerous other problem health behaviors (Hingson et al., 2006; Miller et al., 2007) into later adulthood. Understanding risk factors associated with HED during this period is therefore essential in designing prevention and intervention targets to mitigate risk for alcohol-related dysfunction throughout the lifespan.

Several heuristic models of adolescent risk-taking behavior, collectively referred to as dual systems models (e.g., Shulman et al., 2016; Steinberg et al., 2008), suggest that the interplay between sensation seeking and cognitive control during adolescence may predict individual differences in risk for HED. Sensation seeking is defined as the tendency toward novelty and excitement and has been hypothesized to increase rapidly at puberty and decline slowly thereafter (Ernst, 2014; Shulman et al., 2016; Spear, 2000), predisposing adolescents toward greater sensitivity to reward relative to younger children and adults. At the same time, the cognitive control systems reflects the capacity to regulate reward-driven impulses, which may mature relatively later and more slowly across adolescence (Gogtay et al., 2004). Together, these models imply that each of these systems (on average) should exhibit distinct patterns of change across development, such that sensation seeking will progress curvilinearly and cognitive control either linearly (Steinberg, 2010) or generally more slowly (Casey et al., 2008; Luna & Wright, 2015) from adolescence through emerging adulthood. They also suggest an “imbalance hypothesis” that explains how the asymmetrical development between systems may be a critical determinant of risky behaviors and alcohol use in particular (Shulman et al., 2016). Specifically, larger within-person differences between higher sensation seeking and lesser-developed cognitive control should in turn characterize risk for higher levels of problem substance use behaviors (Casey & Jones, 2010).

Evidence has supported that sensation seeking and cognitive control progress along their purported developmental trajectories (e.g., Harden & Tucker-Drob, 2011; Quinn & Harden, 2013; Romer et al., 2010) with sex differences observed in these growth processes (e.g., Shulman et al., 2014), and meta-analysis has shown that measures of both sensation seeking ( $r = 0.26$ ) and cognitive control (i.e., premeditation;  $r = 0.22$ ) are moderately associated with HED during adolescence (Stautz & Cooper, 2013). However, several methodological limitations have impeded examinations of whether within-person imbalance between these systems predicts real-world drinking behaviors (Johnson et al., 2009; Meisel et al., 2019). First, few studies have utilized an appropriate longitudinal design that can characterize the within-person developmental change hypothesized in dual systems theory (King et al., 2018; Lydon-Staley & Bassett, 2018), requiring concurrent assessment of sensation seeking, premeditation, and drinking behavior spanning the course of the adolescent and young adult period. Second, few researchers have used analytic practices that directly quantify within-person differences between these developing systems. For instance, a review of dual-systems methodologies noted that although many studies tested imbalance hypotheses using either additive or interactive models of sensation seeking and cognitive control, difference score approaches may instead be most optimal in characterizing within-person imbalances between these constructs (Meisel et al., 2019). Methodological limitations in the design and analyses typically employed have thus restricted the impact of dual systems theories on understanding developmental risk for adolescent drinking behavior.

The current study addresses these limitations by longitudinally examining whether sensation seeking, premeditation, and within-person imbalances between these systems are associated with HED from adolescence through emerging adulthood. Using data from the National Consortium on Alcohol and NeuroDevelopment in Adolescence (NCANDA), we used growth curve modeling to characterize change over time in both sensation seeking and premeditation and to link these growth factors with level and change in HED. We then characterized the within-person imbalance between sensation seeking and premeditation using a novel application of latent difference score (LDS) growth modeling (McArdle, 2009; Meisel et al., 2019) and tested whether this imbalance was associated with level and growth in HED. Consistent with prior work (Stautz & Cooper, 2013), we hypothesized that greater sensation seeking and lower premeditation in adolescence would independently predict HED in emerging adulthood. Addressing dual systems theory, we further hypothesized that within-person differences (i.e., imbalance) between higher sensation seeking and lower premeditation would predict higher levels and faster escalation of HED in adolescence through emerging adulthood. Given some prior work demonstrating greater peaks in sensation seeking and slower growth in impulse control over time among males (Shulman et al., 2014), we further explored whether these effects were stronger among males than females.

## MATERIALS AND METHODS

### Participants

Data are from NCANDA, a longitudinal multisite study of 831 healthy developing youth that aims to identify the effects of alcohol use on the developing brain and examine

characteristics that predict alcohol use and related problems, based on the public data release NCANDA\_PUBLIC\_4Y\_REDCAP\_V02 (<https://doi.org/10.7303/syn24226662>; distributed to the public according to the NCANDA Data Distribution agreement <https://www.niaaa.nih.gov/research/major-initiatives/national-consortium-alcohol-and-neurodevelopment-adolescence/ncanda-data>). Participants were recruited at ages 12 to 21 years from five sites (University of California, San Diego, SRI International, Duke University Medical Center, University of Pittsburgh Medical Center, and Oregon Health and Sciences University), and data were collected using an accelerated longitudinal design (described in greater detail in the Analytic Strategy Section below; Duncan et al., 1996; Miyazaki & Raudenbush, 2000). The majority of the sample (83%) had limited or no history of alcohol or drug use at baseline, though 50% were oversampled to include youth at elevated risk for alcohol use disorder due to endorsement of 1 or more externalizing symptoms or 2 or more internalizing symptoms, family history of alcohol use disorder, or having tried alcohol before age 15 (Brown et al., 2015). Key exclusion criteria were English nonfluency, contraindications for neuroimaging, early neurodevelopmental problems, current major Axis I psychiatric disorder, uncorrected sensory impairment, known prenatal drug or alcohol exposure, history of serious medical problems, medication use affecting brain function or blood flow, and pervasive developmental or severe learning disorder (see Brown et al., 2015 for further detail).

### Informed consent

All participants underwent informed consent procedures at study entry with a research associate trained in human subject research protocols. Adult participants or parents of minor participants provided written informed consent prior to study participation, and minor participants provided written assent. The Institutional Review Board at each data collection site approved this study, and each site followed the above procedures to obtain voluntary informed consent or assent depending on the age of participant.

### Measures

**Demographics**—Self-report measures of race, biological sex, and socioeconomic status (SES) were included as covariates. Approximately three-quarters (72%) of the sample identified as White, 11% identified as Black, and 7% identified as Asian. Fewer than 1% identified as Native American/American Indian or Pacific Islander, and the remainder (9%) identified as another or more than one race. A total of 12% identified as Hispanic. Half of the participants (49%) were male. SES was measured as the total combined family income over the past year, measured on a Likert scale ranging from 1 (“Less than \$5000”) to 10 (“\$200,000 or greater”). Responses were averaged from age 16 to 20 and treated as a time-invariant covariate. The median combined household income was between \$100,000 and \$199,999 in the sample.

**Sensation seeking and premeditation**—Indicators of sensation seeking and premeditation were assessed using an abbreviated version of the Urgency, Premeditation, Perseverance, and Sensation Seeking (UPPS) Impulsive Behavior scale (Whiteside & Lynam, 2001). Participants self-reported on a 4-Likert scale ranging from “not at all” to “very much” the degree to which each statement was true of them. Sensation seeking (four

items) measured the tendency to pursue novelty and excitement, and premeditation (four items) measured the predisposition toward thinking before acting. Example sensation-seeking items were “I welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional” and “I will try anything once.” Premeditation items included “My thinking is usually careful and purposeful” and “I usually think carefully before doing anything.” Items from each of the facets were computed as mean scores and standardized prior to analyses to facilitate interpretation. Reliability measures for each subscale across ages were acceptable ( $\alpha$ 's ranging from 0.68 to 0.72 for sensation seeking and 0.80 to 0.83 for premeditation). Prior work has demonstrated adequate psychometric proprieties of the short-form version used in the current study (Cyders et al., 2014).

**HED**—Past year HED was assessed using the item “during the past year, how many times have you consumed 4+ (females)/5+ (males) drinks within an occasion? (0–365)” from the Customary Drinking and Drug use Record (Brown et al., 1998). As this variable was zero-inflated and positively skewed, it was converted to an ordinal scale according to percentiles reported on this measure to help mitigate potential violations of parametric assumptions. Namely, the 25th percentile value was 2 HED episodes, the 50th percentile (i.e., median) value was five episodes, and the 75th percentile value was 17 episodes. As such, past year HED was coded such that 0 = “0 episodes,” 1 = “1–2 episodes,” 2 = “3–5 episodes,” 3 = “6–17 episodes,” and 4 = “18 or more episodes.” Sensitivity analyses were conducted by comparing results to a log-plus-1 transformation of HED, noting the caveat that log-transformations have been shown to produce bias and inefficiency of model parameters when applied to count-distributed variables (O’Hara & Kotze, 2010). Approximately one-quarter of the sample reported at least 1 HED episode at the earliest timepoint of data analysis for HED (age 17 years), and the majority (59.4%) reported at least 1 episode at age 20.

## Procedures

Enrolled participants completed a baseline assessment including detailed interview, neuropsychological assessment, and neuroimaging, which was repeated annually thereafter. Data used for the current study included a subset of NCANDA participants providing data between age 16 years and age 20 years, resulting in a total of 715 adolescents included in analyses. Timepoints of data collection for the sample were from baseline through the fourth annual wave. Retention rates were good across all five research project sites overall through year 4 (78%), with follow-up rates as high as 97% among sites. Data collection was conducted by research associates trained with annual reliability evaluations to criterion and calibrated annually by a centrally trained psychometrician using procedures established by the NCANDA Coordinating Center and the Data Analysis and Administrative Resources. Tests were administered in the identical order across data collection sites and completed in approximately 3 h.

## Analytic strategy

Data were collected using an accelerated longitudinal design (Duncan et al., 2006; Miyazaki & Raudenbush, 2000) in which participants were sampled from a span of baseline ages and

were assessed annually thereafter for up to four annual follow-up assessments. Although participants differed in age at baseline, repeated annual assessments resulted in measurements that overlapped in age, as well as missing data by design (i.e., “planned missingness”) for participants who were not assessed at a given age. For instance, measurements at age 16 years comprised of  $n = 423$ <sup>1</sup> observations, which reflected a combination of participants who were aged 16 at baseline ( $n = 92$ )<sup>2</sup> as well as participants who were 16 years at follow-ups at year 1 ( $n = 101$ ), year 2 ( $n = 99$ ), year 3 ( $n = 94$ ), and year 4 ( $n = 37$ ). Similarly, observations at age 17 included those who were aged 17 years at baseline, those who were aged 17 years at follow-up year 1 (i.e., those who were aged 16 years at baseline); and so on. Missing data under this design can be considered missing completely at random (Baraldi & Enders, 2010), allowing the appropriate use of missing data methods that can preserve a larger sample size available at each age and characterize developmental processes along the full time course of data collection. In other words, despite that only a subset of the sample was represented at any given age, information from the full sample used in the current study ( $n = 715$ ) was used to estimate trajectories of study measures with minimal loss of power. Given this design, we used full information maximum likelihood as an estimator for all specified models to appropriately handle these missing data patterns (Baraldi & Enders, 2010).

Primary analyses were conducted across multiple steps. First, we assessed longitudinal measurement invariance (Haan et al., 2018; Vandenberg & Lance, 2000) for sensation seeking and premeditation to ensure any changes over time in these constructs (and their LDS) were not attributable to age-related changes in the measurement model for either construct. Second, we used unconditional growth curve models (GCMs; Preacher et al., 2008; Singer & Willett, 2009) to characterize growth trajectories for sensation seeking and premeditation from age 16 years to age 20 years. In each model, we included both linear and quadratic terms at the outset to assess whether linear or nonlinear specifications were most optimal in characterizing growth processes for each. Third, we assessed correlations among growth factors for sensation seeking, premeditation, and HED using parallel process GCMs to describe whether and how these were correlated over time from age 16 to 20 years. This was done to assess whether sensation seeking or premeditation was uniquely associated with HED, which was not possible to assess directly using a LDS approach (Meisel et al., 2019). Finally, we extended this model to address key study hypotheses via LDS GCM (McArdle, 2009; Meisel et al., 2019) from age 17 years to age 20 years.<sup>3</sup> In this model, we assessed correlations between growth factors for the dual systems imbalance (i.e., latent difference between sensation seeking and premeditation) and HED. In all GCMs, additional covariates included a dummy race variable (0 = non-White, 1 = White) and SES. Sex was also included as a covariate when estimating models across the full sample (i.e., when models were not stratified by sex, see below).

---

<sup>1</sup>Total sample sizes at ages 17 through 20 were  $n = 470$ ,  $n = 462$ ,  $n = 381$ , and  $n = 381$ , respectively.

<sup>2</sup>Sample sizes for baseline ages were  $n = 92$  for age 16 years;  $n = 114$  for age 17 years;  $n = 93$  for age 18 years;  $n = 54$  for age 19 years; and  $n = 66$  for age 20 years.

<sup>3</sup>Inclusion of age 16 year HED resulted in nonconvergence of this model and was thus removed from analysis.

We explored differences across males and females in key parameter estimates by testing moderation by biological sex throughout analyses. This was done by first stratifying a given model by biological sex using a multigroup framework in which all parameters were estimated freely across groups. Then, we constrained (i.e., fixed) parameters of interest to equivalence across males and females and assessed whether we observed a significant decrement in model fit as a result. A significant decrement in model fit provided evidence that the given parameter estimate was of differing magnitude for males and females. Significance testing was conducted using either a chi-square difference test or its Satorra-Bentler scaled variant (Satorra & Bentler, 2010) when a robust estimator was employed. When chi-square tests suggested no differences across groups on a given parameter, they were fixed to equivalence for reporting purposes below.

In all GCMs, intercepts for HED were set at age 20 years and sensation seeking, premeditation, and their developmental imbalance were specified at the earliest timepoint used in the model to establish appropriate temporal precedence. We evaluated model fit using the adjusted chi-square difference test, where a nonsignificant result was an indication of adequate model fit. We supplemented this test with a number of alternative fit indices (Chen, 2007; Cheung & Rensvold, 2002; Meade et al., 2008), including the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA), and the standardized root-mean square residual (SRMR). Evaluating model fit was guided by approximate recommendations provided in prior work (Chen, 2007; Hu & Bentler, 1999; Yu, 2002). Noting nonnormality in our transformed HED variable, we used maximum likelihood with robust standard errors (MLR) in all parallel process GCMs.<sup>4</sup> This provided more accurate and robust parameter estimation for discrete outcomes compared with the standard maximum-likelihood estimation and alternatives (Méndril, 2010; Rhemtulla, Brosseau-Liard, & Savalei, 2012; Schumacker & Beyerlein, 2000). All analyses were conducted in R (R Core Team, 2020) using the “lavaan” package (Rosseel, 2012). Code for building our LDS model in “lavaan” was guided by Kievit et al. (2018) and Mplus code provided by Meisel et al. (2019). We provide reproducible code for all models described below in a public GitHub repository, available at [https://github.com/connorjmccabe/Dev\\_Imbalance\\_Code](https://github.com/connorjmccabe/Dev_Imbalance_Code).

## RESULTS

Descriptive statistics are provided in Table 1. Below, we describe general trends based on these observed values, though we provide more detail based on standardized estimates from our growth curve models reported in the sections that follow.

Overall, sensation seeking appeared to change minimally over time, whereas premeditation increased modestly. As expected, HED tended to increase from age 17 to age 20.

Correlations indicated moderate stability over time in sensation seeking, premeditation, and

---

<sup>4</sup>We attempted several categorical data models (e.g., zero-inflated negative binomial and ordinal models) to best accommodate the discrete nature of this measure. Unfortunately, none of these models provided converging solutions. Nonetheless, MLR provides estimates of chi-square statistics and standard errors that are each robust to nonnormality in the outcome variable while uniquely retaining the capacity to accommodate data with missingness designs relative to alternatives (Lai, 2018; Lei and Shiverdecker, 2020). We thus considered MLR as a suitable alternative to a discrete data analysis approach.

HED. Moreover, being male was correlated with higher sensation seeking, and higher SES was correlated with more HED across the study period. Sensation seeking and premeditation were modestly and negatively correlated across ages.

### Measurement invariance

We began by examining measurement invariance of sensation seeking and premeditation across ages to ensure that factor loadings and latent intercepts were the same at each age for both constructs (i.e., metric and scalar invariance; Vandenberg & Lance, 2000). This was established by first estimating factor loadings freely across ages, then by examining whether fixing the factor loadings and intercepts to equivalence across ages worsened model fit. If fit did not worsen as a result, this was taken as evidence favoring equivalence of these respective parameters across age. Results supported that metric and scalar invariance across ages was met for both sensation seeking (metric:  $\Delta\chi^2_{12} = 7.10$ ,  $p = 0.85$ ; scalar:  $\Delta\chi^2_{12} = 18.8$ ,  $p = 0.09$ ) and premeditation (metric:  $\Delta\chi^2_{12} = 14.60$ ,  $p = 0.26$ ; scalar:  $\Delta\chi^2_{12} = 15.20$ ,  $p = 0.23$ ). This implied that any changes observed in the means and latent differences between sensation seeking and premeditation over time were unlikely the result of statistical measurement artifacts.

### Unconditional growth curve models

We examined unconditional growth curve models for sensation seeking and premeditation to describe: (1) whether and how each changed over time, (2) whether level and change in each were moderated by sex, and (3) whether growth patterns were correlated with each other. This was achieved by estimating growth in each construct from age 16 years to age 20 years simultaneously for each sex.

We began by exploring linear versus quadratic growth in sensation seeking and premeditation across sex. There was no evidence of quadratic growth in sensation seeking ( $B = 0.01$ , 95% CI = [0.00, 0.01]) or premeditation ( $B = 0.00$ , 95% CI = [-0.01, 0.02]) from age 16 years to age 20 years in the whole sample. Thus, quadratic terms were omitted from the model for parsimony. The resulting model provided excellent fit in the whole sample ( $\chi^2_{41} = 55.41$ ,  $p = 0.07$ , RMSEA = 0.02, CFI = 0.99, TLI = 0.98, SRMR = 0.05) and when stratified by sex ( $\chi^2_{82} = 91.50$ ,  $p = 0.22$ , RMSEA = 0.02, CFI = 0.99, TLI = 0.99, SRMR = 0.06).

Full sample results from this GCM are provided in Table 2, with estimates separated by sex provided in Table S1. Consistent with study hypotheses, premeditation increased linearly by approximately 0.29 standardized units each year (95% CI = [0.06, 0.52], see Figure 1B). There was no evidence that level ( $\Delta\chi^2_1 = 0.44$ ,  $p = 0.51$ ) or growth ( $\Delta\chi^2_1 = 0.10$ ,  $p = 0.75$ ) in premeditation significantly differed by sex. However, noninvariance of the growth parameter for sensation seeking ( $\Delta\chi^2_1 = 11.10$ ,  $p < 0.001$ ) suggested that while females decreased in sensation seeking over time ( $B = -0.04$ , 95% CI = [-0.08, -0.01],  $\beta = -0.41$ ), males increased in sensation seeking by 0.30 standardized units each year from age 16 years to age 20 years (95% CI = [0.01, 0.59]). Males also reported approximately 0.40 standard



deviations higher on sensation seeking at age 16 years compared with females ( $\Delta\chi^2_1 = 4.54$ ,  $p = 0.03$ ). Finally, consistent with hypotheses, growth in sensation seeking was not significantly associated with growth in premeditation across sexes (although level of sensation seeking and premeditation was correlated at age 16 years;  $r = -0.26$ ,  $p < 0.001$ ). This suggested that age-related changes in premeditation and sensation seeking were largely independent growth processes from adolescence through emerging adulthood.

### Parallel process growth curve model

Next, HED growth was regressed on growth for sensation seeking and premeditation to examine whether and how each were related over time across sexes. Model fit was adequate when analyzing the full sample ( $\chi^2_{132} = 247.78$ ,  $p < 0.001$ , RMSEA = 0.04, CFI = 0.94, TLI = 0.93, SRMR = 0.06) and the sample stratified by sex ( $\chi^2_{238} = 305.79$ ,  $p < 0.001$ , RMSEA = 0.026, CFI = 0.96, TLI = 0.96, SRMR = 0.07).

Results are summarized in Table 3.<sup>5</sup> Although HED did not change significantly over time on average, there was substantial variation in change over time ( $B = 0.099$ , 95% CI = [0.071, 0.128]), and higher SES was associated with greater level ( $\beta = 0.271$ , 95% CI = [0.181, 0.361]) and growth ( $\beta = 0.277$ , 95% CI = [0.160, 0.393]) in HED. While premeditation was unrelated to level and growth in HED, sensation seeking at age 16 years predicted greater HED by age 20 years ( $\beta = 0.188$ , 95% CI = [0.061, 0.315]). White versus non-White race was associated with an approximately 0.11 standardized unit increase in HED at age 20 (95% CI = [0.022, 0.200]). Invariance testing indicated that no effects differed significantly between males and females. Estimates were similar using a log-transformation of HED (see Table S3).

### LDS growth model

To examine imbalance hypotheses, we specified a GCM in which the imbalance between sensation seeking and premeditation was quantified via LDS at each study age (e.g., Meisel et al., 2019). Mathematically, these represented an adolescent's latent score on sensation seeking at a given age minus their latent score on premeditation at this same age. Therefore, referring to Figure 1, latent factors DI<sub>17</sub>, DI<sub>18</sub>, DI<sub>19</sub>, and DI<sub>20</sub> reflected developmental imbalances between systems at each age, accounting for measurement error. These factors were then used as latent indicators of slope and intercept growth factors. Germane to dual systems hypotheses, specifying latent intercepts and slopes in this model was critical to (1) quantify within-person variation in level (intercept) and change (slope) in the imbalance over time and (2) assess whether these factors were associated with level and change in HED.

We made several model specification choices to facilitate estimation and sensible inference in our results. First, factor loadings for the developmental imbalance were fixed to linear growth beginning at age 17 years (i.e., factor loadings were [0 1 2 3]) while loadings for HED were [-3 -2 -1 0] to set its intercept at age 20 years. This was done to retain strong temporal precedence that reflected our association of interest (i.e., developmental imbalance

<sup>5</sup>Results were replicated omitting observations at age 16 years to match the developmental period of the latent difference score GCM. Model estimates were essentially identical (see Table S2).

in adolescence predicting HED in emerging adulthood). Second, intercepts and variances for item-level indicators of sensation seeking and premeditation were, respectively, fixed to equivalence across time to facilitate model estimation. We provide further detail and code for reproducing this LDS model directly at the GitHub web page provided above.

Results are summarized in Table 4 and Figures 1 and 2. Whereas RMSEA values were in the acceptable range for the full sample (0.063) and the sex-stratified sample (0.057), there was evidence of suboptimal model fit when examining relative fit indicators in the full ( $\chi^2_{736} = 2874.00$ ,  $p < 0.001$ , CFI = 0.67, TLI = 0.67, SRMR = 0.17) and stratified samples ( $\chi^2_{1438} = 3161.08$ ,  $p < 0.001$ , CFI = 0.72, TLI = 0.73, SRMR = 0.10).<sup>6</sup>

Above and beyond the effects of race, SES, and sex, evidence indicated that greater within-person imbalance between sensation seeking and premeditation in adolescence (age 17 years) was associated with more HED by emerging adulthood (age 20 years). Namely, each standard deviation increase in the developmental imbalance reported at age 17 years was associated with a 0.16 standardized unit increase in HED at age 20 years (95% CI = [0.027, 0.287]; see Figure 2). Although developmental imbalance between sensation seeking and premeditation decreased over the study period, variation in change in the imbalance did not subsequently predict level or change in HED. We found no differences when testing invariance of parameters across sex. Estimates were similar using a log-transformation of HED (see Table S4).

As a final check our results, we supplemented analyses above with two additional steps. First, we examined the correlations between latent measures of sensation seeking, premeditation, and the imbalance at age 17 to determine the degree to which variance in the imbalance correlated with its constituent measures. This demonstrated that sensation seeking ( $r = 0.864$ ) and premeditation ( $r = -0.727$ ) were each highly correlated with the developmental imbalance. Then, noting that our additive parallel process growth curve model suggested that sensation seeking at age 16 predicted HED at age 20, we specified an additional path in our LDS model that regressed the age 20 HED intercept on sensation seeking at age 17. Although issues of collinearity were anticipated given high correlation between sensation seeking and the imbalance, this was done to evaluate whether the effect of the imbalance at age 17 persisted when controlling for concurrent levels of sensation seeking. Neither sensation seeking ( $\beta = 0.278$ , 95% CI = [-0.338, 0.893]) nor the developmental imbalance ( $\beta = -0.147$ , 95% CI = [-0.782, 0.487]) was associated with HED level in this model, due in large part to the highly inflated standard errors of these effects under this specification (0.31 and 0.32 for standardized values, respectively).

---

<sup>6</sup>The RMSEA value corresponding with the null model was 0.112, indicating a reasonably well-fitting baseline model where paths were constrained to zero. Because incremental fit measures (e.g., CFI and TLI) compare a null model (i.e., a “worst-fitting” model where paths are zero) to the specified model, these measures may be untenably small when the null model fits reasonably well (e.g., when RMSEA values for the null model fall below 0.158; Kenny et al., 2015). This likely explains the poor fit implied by these values, which some have suggested are likely not informative in this model scenario (Kenny, 2012). Code for reproducing these supplemental analyses and viewing all model fit measures for this LDS model produced by “lavaan” is available in the GitHub repository URL provided in the Methods section above.

## DISCUSSION

Dual systems accounts of adolescent risk behavior provide a generative framework for understanding whether imbalances between sensation seeking and cognitive control are associated with adolescent risk for HED. However, no studies to our knowledge have provided direct evidence that such imbalances predict risky alcohol behaviors. The goal of this study was to address this gap by examining the association between developmental imbalances in sensation seeking, premeditation, and HED in a prospectively followed sample of youth, using statistical approaches that directly test dual-systems theory (Meisel et al., 2019). Three key findings emerged. First, consistent with prior work (Harden & Tucker-Drob, 2011; Quinn & Harden, 2013; Romer et al., 2010; Shulman et al., 2014), sensation seeking increased from age 16 to age 20 years among males and decreased among females, and premeditation increased modestly over time for both sexes. Second, higher sensation seeking at age 16 years was associated with higher levels of HED by age 20 years. However, we found no relation between premeditation and HED; thus, while premeditation may be an important factor at other ages, it did not independently drive HED during this age range. Third, these results suggested that higher sensation seeking *relative to* premeditation represented an additional risk marker for later HED. That is, consistent with dual systems theory, greater within-person imbalance between sensation seeking and premeditation in adolescence predicated higher levels of HED by emerging adulthood. To our knowledge, the current study is the first to provide direct evidence that within-person desynchrony between sensation seeking and premeditation may represent a distinct individual difference factor in youth that confers risk for subsequent HED.

We observed age-related changes in sensation seeking and premeditation between late adolescence and emerging adulthood and observed differences between sexes. However, although we observed growth in the expected directions for both sensation seeking and premeditation, we did not observe a curvilinear growth pattern for sensation seeking during this age period as might be expected from some dual systems theories (Steinberg, 2010). Nonetheless, prior work using more extensive study periods (i.e., beginning from age 10 years [Shulman et al., 2014] or 12 years [Harden & Tucker-Drob, 2011] through age 24 years) has shown that sensation seeking peaks in mid-to-late adolescence, with slowed growth thereafter through emerging adulthood. It is possible that the items used in the current study, which measure general tendencies corresponding with these measures, may have been less sensitive to detecting interindividual variability across annual measurements relative to prior work (Hertzog & Nesselroade, 2003). Moreover, because the current study focused on a later and more narrow age range (age 16 to 20 years), it is likely that we observed sensation seeking at the latter range of this developmental process, during which curvilinear change in sensation seeking was likely more modest. Relatedly, prior work has shown that females in particular exhibited little age-related change in sensation seeking in late adolescence while sensation seeking may be still increasing during this period among males (Shulman et al., 2014). Given that we observed growth in sensation seeking for only males using the current sample, our findings are consistent with this prior work, adding that sensation seeking may in fact be declining for females in late adolescence and emerging adulthood. This may mirror findings suggesting that females may peak earlier in sensation

seeking (at approximately age 16 years) relative to males (at approximately age 18 years; Shulman et al., 2014): Given the age range of 16 to 20 years in the current sample, males may have been generally *growing toward* their developmental peak in sensation seeking, whereas females may have been *declining from* their peak. Future work may focus on whether differences in growth rates over time between sexes may be explained by differences in maturational processes across sex, such as relative earlier pubertal onset among females (Forbes & Dahl, 2012; Smith, Chein, & Steinberg, 2013).

Prior studies have reported that sensation seeking and premeditation may each be independently linked with HED in adolescent samples either cross-sectionally (e.g., Stautz & Cooper, 2013) or longitudinally (e.g., Ellingson et al., 2019; Quinn & Harden, 2013), yet our findings indicated that only sensation seeking in adolescence was prospectively linked with HED in emerging adulthood. These findings are consistent with the notion that sensation seeking in particular may be linked with greater alcohol consumption among adolescents (e.g., Stautz & Cooper, 2013), and imply that interventions that may help mitigate reward-driven impulses during adolescence may reduce risk for HED in later life. For instance, engaging in protective behavioral strategies (such as making concrete plans to stop or limit drinking) has been shown to buffer the effect of sensation seeking on HED among adolescents (Dumas et al., 2017). Others have shown that sensation-seeking youth may also improve more quickly in delaying gratification as they experience typical risk behaviors in adolescence, which might in turn mitigate their substance use risk (Romer et al., 2010). As such, teaching protective behavioral strategies and affording adolescent experiences that promote delayed gratification may be particularly amenable for reducing risk for HED among sensation-seeking youth. By contrast, we were surprised that we observed no association of premeditation with HED. Some meta-analytic findings have suggested that a lack of premeditation is more uniquely associated with problematic alcohol use than drinking quantity (Coskunpinar et al., 2013), such that the influence of premeditation on alcohol quantity above and beyond sensation seeking may not have been observable in the current sample. Future work may find effects of premeditation on alcohol behaviors that are more directly indicative of problem drinking, such as alcohol misuse or experiencing negative alcohol consequences (e.g., McCabe, et al., 2015).

We built upon these additive findings to show that within-person differences between sensation seeking and premeditation in adolescence predicted HED by emerging adulthood. We achieved this by using difference score approaches explicitly identified as an optimal strategy for testing dual systems theory (Meisel et al., 2019) and by applying these methods in the relatively large and longitudinal NCANDA sample. We contrast this approach with other dual systems research utilizing interaction models that can characterize whether sensation seeking and a lack of premeditation synergistically predict substance use behaviors (e.g., McCabe et al., 2015). Although interaction models are useful in characterizing how premeditation may moderate the effect of sensation seeking on substance use at the between-person level, difference score approaches add to our understanding of developmental relevant processes that are uniquely able to characterize within-person differences between these constructs. However, we note that our hypotheses were only partially supported in that growth in the imbalance was not associated with level or change in HED. This was likely a reflection of the more limited age span that was examined in the present study, during which

we observed relatively modest change in both sensation seeking and premeditation (see discussion above).

Meisel et al. (2019) were the first to introduce difference score models as a means of characterizing disparities between dual systems constructs, and this work was foundational in informing the approach of the current paper. However, in contrast to the present results, their work found no evidence suggesting that the difference between the dual systems constructs examined, reward sensitivity and inhibitory control, was related to alcohol use. We note several methodological differences that may explain this disparity. First, we used measures of dual systems constructs from late adolescence to emerging adulthood (i.e., ages 17 to 20 years), whereas Meisel and colleagues measured these constructs during the middle adolescent period (i.e., ages 12 to 14 years). Given sensation seeking generally peaks and remains relatively high in mid-to-late adolescence (approximately age 16 for females and age 18 for males; Shulman et al., 2014), it is likely that imbalance between systems is greater during this later period. Our study may have therefore benefitted from additional power to detect associations with alcohol-related outcomes given that the imbalance was likely larger during this age period. Second, whereas Meisel and colleagues used behavioral measures to represent reward sensitivity and inhibitory control, we used measures from subscales of the UPPS that were explicitly designed to measure distinct, modestly correlated aspects of impulsivity (Whiteside & Lynam, 2009). Several studies have suggested that self-report measures perform better than behavioral task measures in predicting real-world behavior due to generally greater reliability relative to task measures (Dang et al., 2020; King et al., 2014). Given that the UPPS has fairly well-established psychometric properties (e.g., Smith et al., 2007), subscales from this instrument may be more reliable psychological analogues for testing dual systems theory relative to behavioral task measures.

### Limitations and future directions

We note several limitations in the current study that we hope will be addressed in future research. The focus of this study was on HED, yet whether these findings generalize to other substances (e.g., nicotine and marijuana use) or other forms of alcohol use (e.g., disordered use) should be addressed in future work. Relatedly, given dual systems frameworks seek to explain a broad range of risk-taking behaviors, findings should be extended to other forms of adolescent risk behaviors, including risky sexual conduct (e.g., Charnigo et al., 2013) or risky driving and other reckless behaviors (e.g., Pharo et al., 2011). Additionally, the study sample was predominantly White and reported generally higher household income relative to the national average. Replication of findings in other racially and economically diverse samples will enhance the generalizability of study results, who may report generally less HED relative to White and higher SES young adults (e.g., Humensky, 2010; McCabe et al., 2007).

We acknowledge further that, despite having chosen our model specification *a priori* based on established work (Meisel et al., 2019), fit indicators for our LDS model provided mixed evidence that our theoretical model optimally characterized our data. In particular, whereas RMSEA values were adequate for our LDS model, several relative fit indicators (e.g., CFI and TLI) were quite poor by contrast. We note that incremental fit measures may be

minimally informative when baseline (i.e., null) models have relatively low RMSEA values, which were the case in our specified LDS model (Kenny et al., 2015; see also Footnote 6). In short, this may reflect that our baseline model itself provided a reasonable fit to the data, in turn causing our proposed model to appear less of an improvement by comparison despite absolute fit indicators (i.e., RMSEA) suggesting adequate fit. Nonetheless, future work may obtain better-fitting models using other modeling approaches, including (for instance) count data models that may better suit discrete substance use outcomes (Atkins et al., 2013) rather than our robust linear approach. Indeed, we note that despite trends in the current sample of mean increases in HED over time, the imprecision (i.e., standard error) of this slope estimate was substantial (see, e.g., Tables 3 and 4), which we suspected was a reflection of our robust estimator producing a highly conservative linear estimate of this effect.

Although we consider the application of LDS models a novel and important extension of dual systems theory, we acknowledge these models also have several limitations. First, these models can be difficult to specify, often requiring somewhat strict parameter constraints and large sample sizes to be estimable and identified (Haan et al., 2018). Second, given the LDS approach employed in the current study represents an instance of growth curve models, they are also subject to the concerns raised in growth curve models more broadly, such as the assumption that the growth process is appropriately specified (Curran et al., 2010).

Finally, in contrast to other between-person methods such as the interaction approach in testing dual systems theories (e.g., McCabe et al., 2015), we note in particular that difference score approaches cannot test whether the difference explains alcohol use above and beyond the influence sensation seeking and impulse control alone (Meisel et al., 2019). Put differently, this difference score approach implied that sensation seeking and impulse control influence alcohol use *only* through their within-person difference. By nature of how they are quantified, difference scores will often covary strongly with the measures used to compute them (Edwards, 1994, 2001); for this reason, constituent variables involved in the difference score are typically omitted as covariates in a specified difference score model to avoid issues of collinearity. Consistent with this, our supplemental LDS analyses showed that factor scores representing developmental imbalance and sensation seeking were correlated strongly at age 17. Consequently, and perhaps unsurprisingly, precision of the estimates was compromised for both the sensation seeking and the developmental imbalance when both were regressed on age 20 HED. Given especially that our parallel process model showed sensation seeking at age 17 predicted HED at age 20, we emphasize that we cannot definitively conclude using this modeling approach that variation in the imbalance—and not sensation seeking or premeditation on their own—was driving the imbalance effects. Nonetheless, difference score approaches remain a within-person method that is well-matched as a test of dual systems hypotheses of HED risk despite some of its limitations.

We note that relations between dual systems processes and substance use are likely bi-directional. For instance, several studies have shown structural and functional changes in brain regions implicated in self-regulation resulting from heavy drinking in adolescence (Squeglia et al., 2009), suggesting HED behaviors may conversely shape dual systems imbalance over time. Although many participants in the NCANDA sample had not initiated alcohol use at baseline, analyses in the current study were not isolated to those individuals,

and it is difficult to rule out the effect of HED on developing dual systems given the correlational nature of the current study. However, the Adolescent Brain Cognitive Development (ABCD) study represents an unprecedented opportunity for examining the development of alcohol use behaviors among children with almost no prior alcohol use exposure by design (Lisdahl et al., 2018). Replication of the current study's findings in the ABCD sample is a clear extension of this work as participants in the ABCD sample mature into late adolescence and emerging adulthood, during which many individuals initiate and escalate in alcohol use.

We consider this a first step in applying novel methodological approaches that appropriately match theoretical models of adolescent risk behavior. In particular, whereas LDS modeling allows the examination of *growth in the imbalance* between sensation seeking and premeditation over time, latent growth interactions may provide a methodology for examining *imbalance in growth* between sensation seeking and premeditation across development (Li et al., 2000). Applying this or a related methodology may directly address whether more-rapid growth in sensation seeking combined with less-rapid growth in premeditation may be a marker of HED in adolescence and emerging adulthood. We encourage that future work apply such theory-driven statistical approaches to test dual systems hypotheses, ideally using a large sample size and more protracted study period such as in the ABCD study sample.

The overarching goal of this manuscript was to provide a direct test of dual systems hypotheses of real-world HED in a longitudinal community-based sample of adolescents. Nonetheless, we acknowledge that dual systems theories likely reflect an overly parsimonious account of adolescent risk behavior (Gladwin & Figner, 2014; Pfeifer & Allen, 2012, 2016), such that other developmental factors play crucial roles in characterizing adolescent risk for HED. For instance, it is well-established that social and contextual aspects of adolescent development confer risk for alcohol involvement and problem use during this period, with peer substance use norms in particular among the most robust predictors of alcohol use engagement and misuse (Chassin, Sher, Hussong, & Curran, 2013; King & Chassin, 2004). As such, future research may examine whether developmental imbalance is moderated by such social influences in producing or reducing risk for HED. Moreover, several extensions of dual systems theories have suggested that adolescent hyperresponsivity to negative emotion-based contexts may represent a third systems integral to risky adolescent decision-making (Ernst, 2014). We note that this theoretical framework mirrors the conceptual distinction made in the personality literature between emotion-based rash action (i.e., positive and negative urgency; Cyders & Smith, 2008) compared with other dispositions toward impulsive action (e.g., sensation seeking and premeditation). Given that negative urgency in particular is among the most robust predictors of problem levels of alcohol use (Stautz & Cooper, 2013), examining the influence of these emotion-based constructs on adolescent drinking behaviors may add critically to dual systems theory and research.

## Summary

The current findings suggested that within-person differences between sensation seeking and premeditation in adolescence may characterize a unique indicator of HED risk during the transition to emerging adulthood. This work was designed to explicitly test dual systems theory using a methodological approach and sample that was particularly well-suited as a test of this theory. Nonetheless, we consider this work a step forward in providing a comprehensive understanding of adolescent HED risk. We hope that this work stimulates future study of within- and between-person factors developing crucially during this period that shape HED and alcohol use disorder risk across the lifespan.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

### Funding information

This research was supported by the National Institute on Alcohol Abuse and Alcoholism of the National Institutes of Health (T32AA013525). Reported data were obtained from the multisite National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA) study, which is supported by the U.S. National Institute on Alcohol Abuse and Alcoholism with cofunding from the National Institute on Drug Abuse, the National Institute of Mental Health, and the National Institute of Child Health and Human Development grant numbers: AA021697 (Pfefferbaum and Pohl), AA021695 (Brown and Tapert), AA021692 (Tapert), AA021696 (Colrain and Baker), AA021681 (Goldston and Nooner), AA021690 (Clark), and AA021691 (Nagel). This work was also made possible by the National Institute on Alcohol Abuse and Alcoholism grant K23 AA026869 that supports Alejandro Meruelo, MD, PhD.

## REFERENCES

- Atkins DC, Baldwin SA, Zheng C, Gallop RJ & Neighbors C (2013) A tutorial on count regression and zero-altered count models for longitudinal substance use data. *Psychology of Addictive Behaviors*, 27, 166–177. [PubMed: 22905895]
- Baraldi AN & Enders CK (2010) An introduction to modern missing data analyses. *Journal of School Psychology*, 48, 5–37. [PubMed: 20006986]
- Brown SA, Brumback T, Tomlinson K, Cummins K, Thompson WK, Nagel BJ et al. (2015) The national consortium on alcohol and neuro-development in adolescence (NCANDA): a multisite study of adolescent development and substance use. *Journal of Studies on Alcohol and Drugs*, 76, 895–908. [PubMed: 26562597]
- Brown SA, Myers MG, Lippke L, Tapert SF, Stewart DG & Vik PW (1998) Psychometric evaluation of the customary drinking and drug use record (CDDR): a measure of adolescent alcohol and drug involvement. *Journal of Studies on Alcohol*, 59, 427–438. [PubMed: 9647425]
- Casey BJ, Jones RM & Hare TA (2008) The adolescent brain. *Annals of the New York Academy of Sciences*, 1124, 111–126. [PubMed: 18400927]
- Casey BJ & Jones RM (2010) Neurobiology of the adolescent brain and behavior: implications for substance use disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49, 1189–1201. [PubMed: 21093769]
- Charnigo R, Noar SM, Garnett C, Crosby R, Palmgreen P & Zimmerman RS (2013) Sensation seeking and impulsivity: combined associations with risky sexual behavior in a large sample of young adults. *The Journal of Sex Research*, 50, 480–488. [PubMed: 22456443]
- Chassin L, Pitts SC & Prost J (2002) Binge drinking trajectories from adolescence to emerging adulthood in a high-risk sample: predictors and substance abuse outcomes. *Journal of Consulting and Clinical Psychology*, 70, 67–78. [PubMed: 11860058]

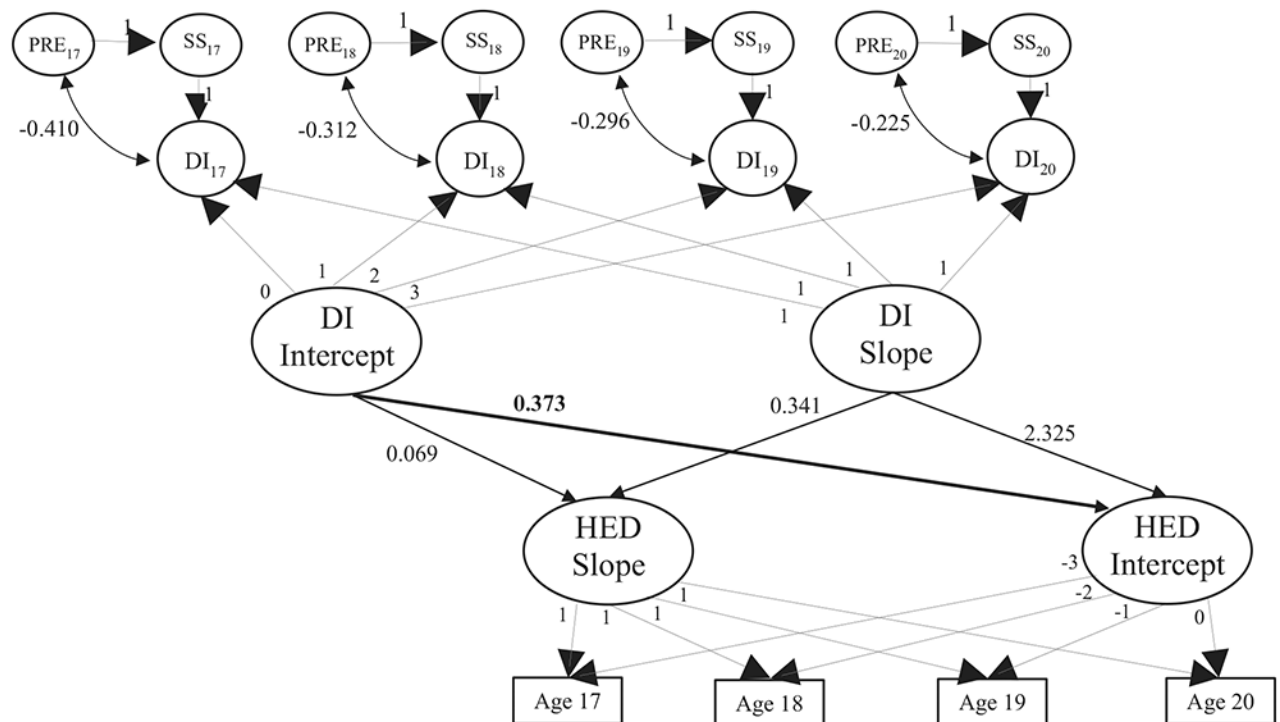


- Chassin L, Sher KJ, Hussong A & Curran P (2013) The developmental psychopathology of alcohol use and alcohol disorders: research achievements and future directions. *Development and Psychopathology*, 25, 1567–1584. [PubMed: 24342856]
- Chen FF (2007) Sensitivity of goodness of fit indexes to lack of measurement invariance. *Structural Equation Modeling*, 14, 464–504.
- Cheung GW & Rensvold RB (2002) Evaluating goodness-of-fit indexes for testing measurement invariance. *Structural Equation Modeling*, 9, 233–255.
- Coskunpinar A, Dir AL & Cyders MA (2013) Multidimensionality in impulsivity and alcohol use: a meta-analysis using the UPPS model of impulsivity. *Alcoholism, Clinical and Experimental Research*, 37, 1441–1450.
- Curran PJ, Obeidat K & Losardo D (2010) Twelve frequently asked questions about growth curve modeling. *Journal of Cognition and Development*, 11, 121–136. [PubMed: 21743795]
- Cyders MA, Littlefield AK, Coffey S & Karyadi KA (2014) Examination of a short English version of the UPPS-P Impulsive Behavior Scale. *Addictive Behaviors*, 39, 1372–1376. [PubMed: 24636739]
- Cyders MA & Smith GT (2008) Emotion-based dispositions to rash action: positive and negative urgency. *Psychological Bulletin*, 134, 807–828. [PubMed: 18954158]
- Dang J, King KM & Inzlicht M (2020) Why are self-report and behavioral measures weakly correlated? *Trends in Cognitive Sciences*, 24, 267–269. [PubMed: 32160564]
- de Haan A, Prinzie P, Sentse M & Jongerling J (2018) Supplemental material for latent difference score modeling: a flexible approach for studying informant discrepancies. *Psychological Assessment*, 30, 358–369. [PubMed: 28406670]
- Doumas DM, Miller R & Esp S (2017) Impulsive sensation seeking, binge drinking, and alcohol-related consequences: do protective behavioral strategies help high risk adolescents? *Addictive Behaviors*, 64, 6–12. [PubMed: 27533076]
- Duncan SC, Duncan TE & Hops H (1996) Analysis of longitudinal data within accelerated longitudinal designs. *Psychological Methods*, 1, 236–248.
- Duncan SC, Duncan TE & Strycker LA (2006) Alcohol use from ages 9 to 16: a cohort-sequential latent growth model. *Drug and Alcohol Dependence*, 81, 71–81. [PubMed: 16006054]
- Edwards JR (1994) The study of congruence in organizational behavior research: critique and a proposed alternative. *Organizational Behavior and Human Decision Processes*, 58, 51–100.
- Edwards JR (2001) Ten difference score myths. *Organizational Research Methods*, 4, 265–287.
- Ellingson JM, Corley R, Hewitt JK & Friedman NP (2019) A prospective study of alcohol involvement and the dual-systems model of adolescent risk-taking during late adolescence and emerging adulthood. *Addiction*, 114, 653–661. [PubMed: 30398695]
- Ernst M (2014) The triadic model perspective for the study of adolescent motivated behavior. *Brain and Cognition*, 89, 104–111. [PubMed: 24556507]
- Forbes EE & Dahl RE (2012) Research review: altered reward function in adolescent depression: what, when and how? *Journal of Child Psychology and Psychiatry*, 53, 3–15. [PubMed: 22117893]
- Gladwin TE & Figner B (2014) ‘Hot’ cognition and dual systems: Introduction, criticisms, and ways forward. In: Cowan N & Balota D (Eds.) *Frontiers of cognitive psychology series: neuroeconomics, judgment and decision making*. New York, NY: Psychology Press, pp. 157–180.
- Gogtay N, Giedd JN, Lusk L, Hayashi KM, Greenstein D, Vaituzis AC et al. (2004) Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences*, 101, 8174–8179.
- Hallgren KA, McCabe CJ, King KM, & Atkins DC (2019) Beyond path diagrams: Enhancing applied structural equation modeling research through data visualization. *Addictive behaviors*, 94, 74–82. [PubMed: 30219251]
- Harden KP & Tucker-Drob EM (2011) Individual differences in the development of sensation seeking and impulsivity during adolescence: further evidence for a dual systems model. *Developmental Psychology*, 47, 739–746. [PubMed: 21534657]
- Hertzog C & Nesselroade JR (2003) Assessing psychological change in adulthood: an overview of methodological issues. *Psychology and Aging*, 18, 639–657. [PubMed: 14692854]

- Hingson RW, Heeren T & Winter MR (2006) Age at drinking onset and alcohol dependence: age at onset, duration, and severity. *Archives of Pediatrics and Adolescent Medicine*, 160, 739–746. [PubMed: 16818840]
- Hu LT & Bentler PM (1999) Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Structural Equation Modeling*, 6, 1–55.
- Humensky JL (2010) Are adolescents with high socioeconomic status more likely to engage in alcohol and illicit drug use in early adulthood? *Substance Abuse Treatment, Prevention, and Policy*, 5, 1–10.
- Johnson SB, Blum RW & Giedd JN (2009) Adolescent maturity and the brain: the promise and pitfalls of neuroscience research in adolescent health policy. *Journal of Adolescent Health*, 45, 216–221.
- Kenny DA (2012) Measuring model fit. *DavidakennyNet*.
- Kenny DA, Kaniskan B & McCoach DB (2015) The performance of RMSEA in models with small degrees of freedom. *Sociological Methods & Research*, 44, 486–507.
- Kievit RA, Brandmaier AM, Ziegler G, van Harmelen AL, de Mooij SMM, Moutoussis M et al. (2018) Developmental cognitive neuroscience using latent change score models: a tutorial and applications. *Developmental Cognitive Neuroscience*, 33, 99–117. [PubMed: 29325701]
- King KM & Chassin L (2004) Mediating and moderated effects of adolescent behavioral undercontrol and parenting in the prediction of drug use disorders in emerging adulthood. *Psychology of Addictive Behaviors*, 18, 239–249. [PubMed: 15482079]
- King KM, Littlefield AK, McCabe CJ, Mills KL, Flournoy J & Chassin L (2018) Longitudinal modeling in developmental neuroimaging research: common challenges, and solutions from developmental psychology. *Developmental Cognitive Neuroscience*, 33, 54–72. [PubMed: 29395939]
- King KM, Patock-Peckham JA, Dager AD, Thimm K & Gates JR (2014) On the mismeasurement of impulsivity: trait, behavioral, and neural models in alcohol research among adolescents and young adults. *Current Addiction Reports*, 1, 19–32.
- King G, Tomz M, & Wittenberg J (2000) Making the most of statistical analyses: Improving interpretation and presentation. *American journal of political science*, 347–361.
- Lai K (2018) Estimating standardized SEM parameters given nonnormal data and incorrect model: Methods and comparison. *Structural Equation Modeling*, 25, 600–620.
- Lei PW & Shiverdecker LK (2020) Performance of estimators for confirmatory factor analysis of ordinal variables with missing data. *Structural Equation Modeling*, 27, 584–601.
- Li F, Duncan TE & Acock A (2000) Modeling interaction effects in latent growth curve models. *Structural Equation Modeling*, 7, 497–533.
- Lisdahl KM, Sher KJ, Conway KP, Gonzalez R, Feldstein Ewing SW, Nixon SJ et al. (2018) Adolescent brain cognitive development (ABCD) study: overview of substance use assessment methods. *Developmental Cognitive Neuroscience*, 32, 80–96. [PubMed: 29559216]
- Luna B & Wright C (2015) Adolescent brain development: implications for the juvenile criminal justice systems. In: Heilbrun K, DeMatteo D & Godstein N (Eds.) *APA handbook of psychology and juvenile justice*. Washington, DC: American Psychological Association.
- Lydon-Staley DM & Bassett DS (2018) The promise and challenges of intensive longitudinal designs for imbalance models of adolescent substance use. *Frontiers in Psychology*, 9, 1–9. [PubMed: 29410639]
- McArdle JJ (2009) Latent variable modeling of differences and changes with longitudinal data. *Annual Review of Psychology*, 60, 577–605.
- McCabe CJ, Louie KA & King KM (2015) Premeditation moderates the relation between sensation seeking and risky substance use among young adults. *Psychology of Addictive Behaviors*, 29, 753–765. [PubMed: 26415063]
- McCabe SE, Morales M, Cranford JA, Delva J, McPherson MD & Boyd CJ (2007) Race/ethnicity and gender differences in drug use and abuse among college students. *Journal of Ethnicity in Substance Abuse*, 6, 75–95. [PubMed: 18192205]
- McCarty CA, Ebel BE, Garrison MM, DiGiuseppe DL, Christakis DA & Rivara FP (2004) Continuity of binge and harmful drinking from late adolescence to early adulthood. *Pediatrics*, 114, 714–719. [PubMed: 15342844]

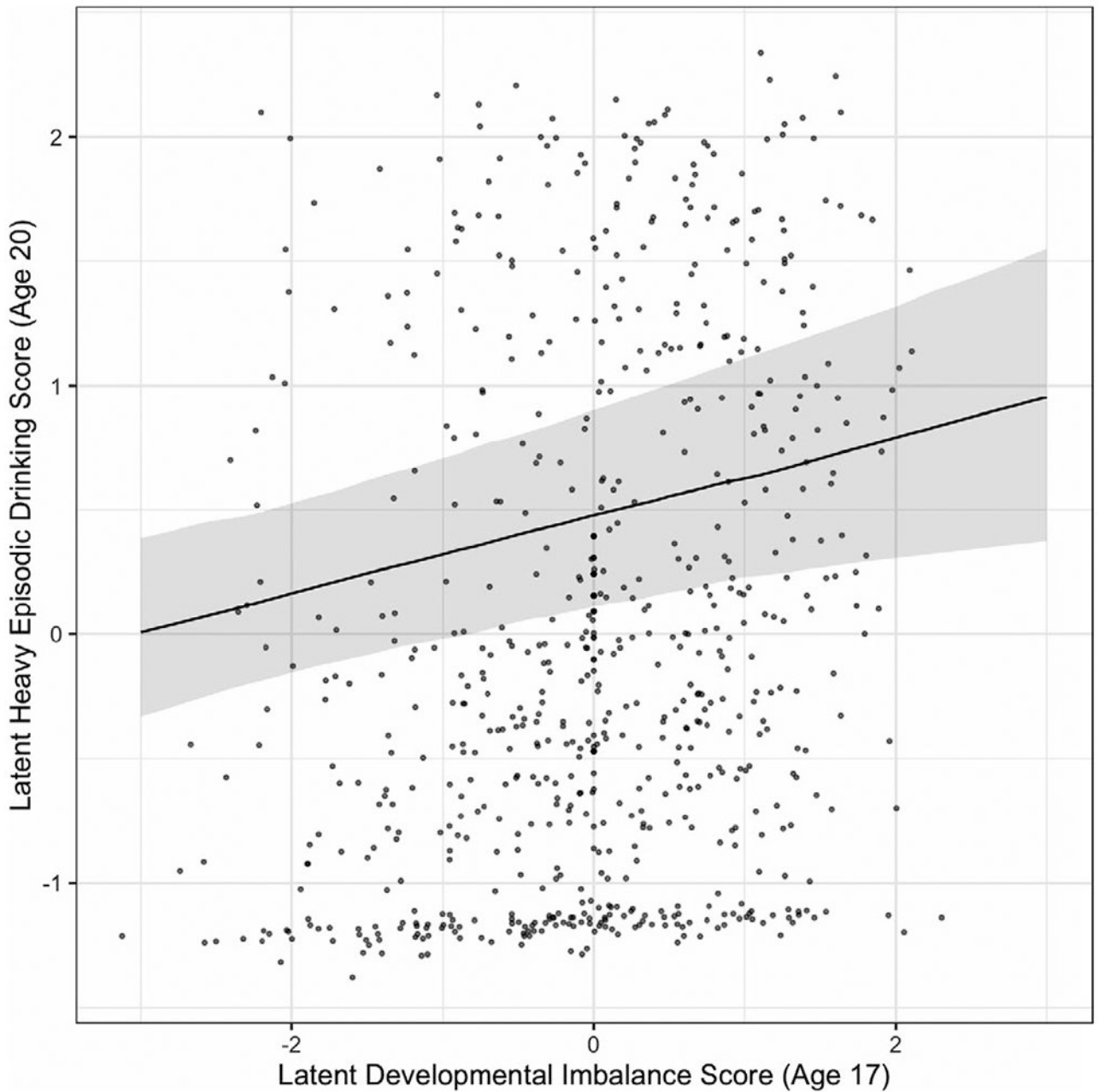
- McGue M & Iacono WG (2005) The association of early adolescent problem behavior with adult psychopathology. *American Journal of Psychiatry*, 162, 1118–1124.
- Meade AW, Johnson EC & Braddy PW (2008) Power and sensitivity of alternative fit indices in tests of measurement invariance. *Journal of Applied Psychology*, 93, 568–592.
- Meisel SN, Fosco WD, Hawk LW & Colder CR (2019) Mind the gap: a review and recommendations for statistically evaluating Dual Systems models of adolescent risk behavior. *Developmental Cognitive Neuroscience*, 39, 100681. [PubMed: 31404858]
- Miller JW, Naimi TS, Brewer RD & Jones SE (2007) Binge drinking and associated health risk behaviors among high school students. *Pediatrics*, 119, 76–85. [PubMed: 17200273]
- Mindrill D (2010) Maximum likelihood (ML) and diagonally weighted least squares (DWLS) estimation procedures: a comparison of estimation bias with ordinal and multivariate non-normal data. *International Journal for Digital Society*, 1, 60–66.
- Miyazaki Y & Raudenbush SW (2000) Tests for linkage of multiple cohorts in an accelerated longitudinal design. *Psychological Methods*, 5, 44–63. [PubMed: 10937322]
- O’Hara RB & Kotze DJ (2010) Do not log-transform count data. *Methods in Ecology and Evolution*, 1, 118–122.
- Pfeifer JH & Allen NB (2012) Arrested development? Reconsidering dual-systems models of brain function in adolescence and disorders. *Trends in Cognitive Sciences*, 16, 322–329. [PubMed: 22613872]
- Pfeifer JH & Allen NB (2016) The audacity of specificity: moving adolescent developmental neuroscience towards more powerful scientific paradigms and translatable models. *Developmental Cognitive Neuroscience*, 17, 131–137. [PubMed: 26754460]
- Pharo H, Sim C, Graham M, Gross J & Hayne H (2011) Risky business: executive function, personality, and reckless behavior during adolescence and emerging adulthood. *Behavioral Neuroscience*, 125, 970–978. [PubMed: 22004262]
- Preacher K, Wichman A, MacCallum R & Briggs N (2008) *Latent growth curve modeling*. Thousand Oaks, CA: Sage.
- Quinn PD & Harden KP (2013) Differential changes in impulsivity and sensation seeking and the escalation of substance use from adolescence to early adulthood. *Development and Psychopathology*, 25, 223–239. [PubMed: 22824055]
- R Core Team (2020) R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. <https://www.R-project.org/>
- Reyna VF & Farley F (2006) Risk and rationality in adolescent decision making: implications for theory, practice, and public policy. *Psychological Science in the Public Interest*, 7, 1–44. [PubMed: 26158695]
- Rhemtulla M, Brosseau-Liard PÉ & Savalei V (2012) When can categorical variables be treated as continuous? A comparison of robust continuous and categorical SEM estimation methods under suboptimal conditions. *Psychological Methods*, 17, 354–373. [PubMed: 22799625]
- Romer D, Duckworth AL, Sznitman S & Park S (2010) Can adolescents learn self-control? Delay of gratification in the development of control over risk taking. *Prevention Science*, 11, 319–330. [PubMed: 20306298]
- Rosseel Y (2012) Lavaan: An R package for structural equation modeling. *Journal of Statistical Software*, 48(2), 1–36.
- SAMHSA. (2019) Key substance use and mental health indicators in the United States: Results from the 2018 National Survey on Drug Use and Health. HHS Publ No PEP19-5068, NSDUH Ser H-54.
- Satorra A & Bentler PM (2010) Ensuring positiveness of the scaled difference chi-square test statistic. *Psychometrika*, 75, 243–248. [PubMed: 20640194]
- Schumacker RE & Beyerlein ST (2000) Confirmatory factor analysis with different correlation types and estimation methods. *Structural Equation Modeling*, 7, 629–636.
- Shulman EP, Harden KP, Chein JM & Steinberg L (2014) Sex differences in the developmental trajectories of impulse control and sensation-seeking from early adolescence to early adulthood. *Journal of Youth and Adolescence*, 44, 1–17. [PubMed: 24682958]

- Shulman EP, Smith AR, Silva K, Icenogle G, Duell N, Chein J et al. (2016) The dual systems model: review, reappraisal, and reaffirmation. *Developmental Cognitive Neuroscience*, 17, 103–117. [PubMed: 26774291]
- Singer JD & Willett JB (2009) *Applied longitudinal data analysis: modeling change and event occurrence*. New York, NY: Oxford University Press.
- Smith AR, Chein J & Steinberg L (2013) Impact of socio-emotional context, brain development, and pubertal maturation on adolescent risk-taking. *Hormones and Behavior*, 64, 323–332. [PubMed: 23998675]
- Smith GT, Fischer S, Cyders MA, Annus AM, Spillane NS & McCarthy DM (2007) On the validity and utility of discriminating among impulsivity-like traits. *Assessment*, 14, 155–170. [PubMed: 17504888]
- Spear LP (2000) The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews*, 24, 417–463. [PubMed: 10817843]
- Squeglia LM, Jacobus J & Tapert SF (2009) The influence of substance use on adolescent brain development. *Clinical EEG and Neuroscience*, 40, 31–38. [PubMed: 19278130]
- Stagman S, Wile S & Schwarz DP (2011) NCCP | Adolescent substance use in the U.S. [http://www.nccp.org/publications/pub\\_1008.html](http://www.nccp.org/publications/pub_1008.html)
- Stautz K & Cooper A (2013) Impulsivity-related personality traits and adolescent alcohol use: a meta-analytic review. *Clinical Psychology Review*, 33, 574–592. [PubMed: 23563081]
- Steinberg L (2010) A dual systems model of adolescent risk-taking. *Developmental Psychobiology*, 52, 216–224. [PubMed: 20213754]
- Steinberg L, Albert D, Cauffman E, Banich M, Graham S & Woolard J (2008) Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: evidence for a dual systems model. *Developmental Psychology*, 44, 1764–1778. [PubMed: 18999337]
- Vandenberg RJ & Lance CE (2000) A review and synthesis of the measurement invariance literature: suggestions, practices, and recommendations for organizational research. *Organizational Research Methods*, 3, 4–69.
- Viner RM & Taylor B (2007) Adult outcomes of binge drinking in adolescence: findings from a UK national birth cohort. *Journal of Epidemiology and Community Health*, 61, 902–907. [PubMed: 17873228]
- Whiteside SP & Lynam DR (2001) The Five Factor Model and impulsivity: using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, 30, 669–689.
- Whiteside SP & Lynam DR (2009) Understanding the role of impulsivity and externalizing psychopathology in alcohol abuse: application of the UPPS Impulsive Behavior Scale. *Personality Disorders: Theory, Research, and Treatment*, 11(3), 69–79.
- Yu CY (2002) Evaluating cutoff criteria of model fit indices for latent variable models with binary and continuous outcomes.



**FIGURE 1.**

Latent difference score growth model. Note. PRE, UPPS Premeditation; SS, UPPS Sensation Seeking; DI, Developmental Imbalance; HED, Past Year Heavy Episodic Drinking Frequency. All coefficients presented indicate unstandardized values. Bolded values and arrows indicate significant path coefficients. Factor loadings for the developmental imbalance slope factor were [0 1 2 3] (i.e., the intercept was set to age 17 years). Loading for the HED slope factor was [3 -2 -1 0] (i.e., the intercept was set to age 20 years). Covariate effects (SES, sex, and race) and factor loadings for manifest indicators of sensation seeking and premeditation at each age were omitted for parsimony



**FIGURE 2.**

Relation between latent measures of adolescent developmental imbalance and young adult heavy episodic drinking. *Note.* Latent heavy episodic drinking and developmental imbalance measures are unit standardized. Developmental imbalance scores reflect latent within-person differences in sensation seeking relative to one's level of premeditation. Latent variable point values for each observation were generated using the "lavPredict" function in "lavaan" following guidelines provided by Hallgren et al. (2019). Predicted values (i.e., black line) and their 95% confidence regions were generated via parametric bootstrap (i.e., Monte Carlo simulation; King, Tomz, & Wittenberg, 2000)

TABLE 1

Descriptive statistics and correlation matrix of study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
<i>Covariates</i>																	
1. Race (1 = White)	-	0.06	<b>0.20</b>	-0.05	-0.02	-0.03	0.01	-0.10	-0.02	-0.04	0.05	-0.09	0.01	<b>0.13</b>	<b>0.11</b>	<b>0.18</b>	<b>0.13</b>
2. Sex (1 = Male)	-	-	0.06	0.08	<b>0.21</b>	<b>0.22</b>	<b>0.28</b>	<b>0.22</b>	0.00	0.03	0.08	0.04	0.01	0.04	0.05	0.08	0.03
3. Socioeconomic status	-	-	-	0.03	0.05	0.03	0.01	0.08	0.03	-0.02	0.02	-0.07	0.00	<b>0.18</b>	<b>0.24</b>	<b>0.30</b>	<b>0.21</b>
<i>Sensation seeking</i>																	
4. Age 16	-	-	-	-	<b>0.52</b>	<b>0.55</b>	<b>0.49</b>	<b>0.26</b>	<b>-0.10</b>	<b>-0.23</b>	<b>-0.17</b>	<b>-0.29</b>	<b>-0.20</b>	<b>0.14</b>	<b>0.17</b>	0.14	-0.04
5. Age 17	-	-	-	-	-	<b>0.63</b>	<b>0.60</b>	<b>0.49</b>	<b>-0.14</b>	<b>-0.17</b>	-0.08	-0.06	-0.05	<b>0.17</b>	<b>0.20</b>	0.09	0.13
6. Age 18	-	-	-	-	-	-	<b>0.67</b>	<b>0.58</b>	<b>-0.14</b>	<b>-0.15</b>	-0.06	<b>-0.14</b>	-0.06	0.07	<b>0.15</b>	0.09	0.09
7. Age 19	-	-	-	-	-	-	-	<b>0.64</b>	-0.16	-0.12	0.00	<b>-0.11</b>	-0.08	<b>0.16</b>	<b>0.18</b>	<b>0.16</b>	<b>0.17</b>
8. Age 20	-	-	-	-	-	-	-	-	-0.06	-0.04	0.02	-0.12	-0.05	0.07	0.10	<b>0.14</b>	<b>0.14</b>
<i>Premeditation</i>																	
9. Age 16	-	-	-	-	-	-	-	-	-	<b>0.46</b>	<b>0.45</b>	<b>0.30</b>	<b>0.55</b>	<b>-0.13</b>	<b>-0.13</b>	-0.03	0.10
10. Age 17	-	-	-	-	-	-	-	-	-	-	<b>0.50</b>	<b>0.36</b>	<b>0.43</b>	<b>-0.10</b>	-0.09	0.03	0.10
11. Age 18	-	-	-	-	-	-	-	-	-	-	-	<b>0.44</b>	<b>0.47</b>	-0.09	-0.10	-0.04	0.07
12. Age 19	-	-	-	-	-	-	-	-	-	-	-	-	<b>0.53</b>	<b>-0.19</b>	-0.10	-0.09	-0.05
13. Age 20	-	-	-	-	-	-	-	-	-	-	-	-	-	0.01	-0.10	-0.06	-0.04
<i>Heavy episodic drinking</i>																	
14. Age 17	-	-	-	-	-	-	-	-	-	-	-	-	-	-	<b>0.67</b>	<b>0.51</b>	<b>0.49</b>
15. Age 18	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	<b>0.63</b>	<b>0.52</b>
16. Age 19	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	<b>0.76</b>
17. Age 20	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mean	0.72	0.49	8.20	2.56	2.58	2.62	2.47	2.51	3.35	3.39	3.41	3.45	3.48	0.49	0.78	1.29	1.52
Standard deviation	0.45	0.50	1.78	0.92	0.91	0.90	0.93	0.92	0.65	0.65	0.64	0.62	0.60	1.01	1.22	1.54	1.58
Skew	-0.96	0.04	-1.19	-0.24	-0.20	-0.18	-0.04	-0.22	-0.70	-0.73	-0.77	-0.71	-0.76	2.08	1.38	0.68	0.47

Note: Bolded values indicate significant correlations at the  $p < 0.05$  level.

TABLE 2

Summary of unconditional growth curve models

	Est.	SE	95% CI	Standardized estimate
<b>Sensation seeking</b>				
Growth parameters				
Age 16 intercept	2.542	0.038	[2.468, 2.616]	3.544
Slope	0.001	0.013	[-0.025, 0.027]	0.006
Intercept variance	<b>0.515</b>	<b>0.055</b>	<b>[0.407, 0.622]</b>	<b>1.000</b>
Slope variance	<b>0.021</b>	<b>0.007</b>	<b>[0.006, 0.035]</b>	<b>1.000</b>
Residual variances				
Age 16	0.376	0.047	[0.284, 0.469]	0.422
Age 17	0.344	0.033	[0.279, 0.409]	0.420
Age 18	0.287	0.028	[0.232, 0.343]	0.376
Age 19	0.289	0.032	[0.226, 0.351]	0.357
Age 20	0.302	0.046	[0.211, 0.393]	0.334
<b>Premeditation</b>				
Growth parameters				
Age 16 intercept	3.375	0.026	[3.324, 3.425]	7.916
Slope	<b>0.027</b>	<b>0.009</b>	<b>[0.008, 0.045]</b>	<b>0.290</b>
Intercept variance	<b>0.182</b>	<b>0.026</b>	<b>[0.130, 0.233]</b>	<b>1.000</b>
Slope variance	<b>0.008</b>	<b>0.004</b>	<b>[0.001, 0.016]</b>	<b>1.000</b>
Residual variances				
Age 16	0.231	0.027	[0.178, 0.285]	0.560
Age 17	0.230	0.021	[0.190, 0.270]	0.578
Age 18	0.229	0.020	[0.189, 0.268]	0.573
Age 19	0.202	0.020	[0.163, 0.242]	0.515
Age 20	0.135	0.023	[0.089, 0.181]	0.373
<b>Growth factor covariances</b>				
Age 16 sensation seeking ~ sensation-seeking change	-0.030	0.017	[-0.064, 0.003]	-0.293
Age 16 premeditation ~ premeditation change	-0.011	0.009	[-0.028, 0.006]	-0.287
Age 16 sensation seeking ~ Age 16 premeditation	<b>-0.081</b>	<b>0.024</b>	<b>[-0.128, -0.033]</b>	<b>-0.264</b>



Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

	<b>Est.</b>	<b>SE</b>	<b>95% CI</b>	<b>Standardized estimate</b>
Sensation-seeking change ~~ premeditation change	0.000	0.003	[-0.006, 0.006]	-0.013
Age 16 sensation seeking ~~ premeditation change	0.002	0.009	[-0.017, 0.020]	0.023
Age 16 premeditation ~~ sensation-seeking change	0.009	0.009	[-0.008, 0.027]	0.152

*Note:* Bolded values indicate significance of parameters of interest at the 95% level of confidence. Abbreviations: 95% CI, 95% confidence interval; Est., estimate; SE, standard error.

**TABLE 3**

Summary of parallel process growth curve model

	Est.	SE	95% CI	Standardized estimate
<b>Intercepts</b>				
Age 16 premeditation	3.372	0.027	[3.320, 3.424]	8.020
Age 16 sensation seeking	2.528	0.039	[2.451, 2.606]	3.508
Age 20 HED	-0.868	1.101	[-3.025, 1.289]	-0.575
<b>Growth rates</b>				
Premeditation slope	<b>0.027</b>	<b>0.009</b>	<b>[0.009, 0.045]</b>	<b>0.302</b>
Sensation-seeking slope	0.005	0.013	[-0.021, 0.032]	0.038
HED slope	-0.254	0.306	[-0.854, 0.345]	-0.731
<b>Fixed effects</b>				
Age 16 premeditation → Age 20 HED	-0.135	0.266	[-0.656, 0.386]	-0.038
Age 16 sensation seeking → Age 20 HED	<b>0.394</b>	<b>0.130</b>	<b>[0.138, 0.650]</b>	<b>0.188</b>
Premeditation change → Age 20 HED	-2.431	2.291	[-6.920, 2.059]	-0.144
Sensation-seeking change → Age 20 HED	1.455	1.181	[-0.859, 3.770]	0.138
White → Age 20 HED	<b>0.375</b>	<b>0.153</b>	<b>[0.076, 0.675]</b>	<b>0.111</b>
SES → Age 20 HED	<b>0.201</b>	<b>0.035</b>	<b>[0.133, 0.269]</b>	<b>0.271</b>
Male → Age 20 HED	0.036	0.142	[-0.243, 0.314]	0.012
Age 16 premeditation → HED change	0.006	0.075	[-0.140, 0.153]	0.008
Age 16 sensation seeking → HED change	0.064	0.039	[-0.011, 0.140]	0.133
Premeditation change → HED change	-0.992	0.720	[-2.404, 0.419]	-0.254
Sensation-seeking change → HED change	0.312	0.330	[-0.334, 0.958]	0.128
White → HED change	0.057	0.042	[-0.025, 0.139]	0.073
SES → HED change	<b>0.047</b>	<b>0.010</b>	<b>[0.027, 0.068]</b>	<b>0.277</b>
Male → HED change	0.037	0.039	[-0.039, 0.114]	0.054
<b>Growth factor variances</b>				
Premeditation intercept	<b>0.177</b>	<b>0.03</b>	<b>[0.117, 0.236]</b>	<b>1.000</b>
Premeditation slope	0.008	0.005	[-0.001, 0.017]	1.000
Sensation-seeking intercept	<b>0.519</b>	<b>0.054</b>	<b>[0.413, 0.625]</b>	<b>1.000</b>
Sensation-seeking slope	<b>0.020</b>	<b>0.008</b>	<b>[0.006, 0.035]</b>	<b>1.000</b>

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

	Est.	SE	95% CI	Standardized estimate
HED intercept	<b>1.911</b>	<b>0.171</b>	<b>[1.575, 2.247]</b>	<b>0.841</b>
HED slope	<b>0.099</b>	<b>0.015</b>	<b>[0.069, 0.129]</b>	<b>0.815</b>

Note: Bolded values indicate significance of parameters of interest at the 95% level of confidence.

Abbreviations: Est., estimate; SE, standard error; SES, socioeconomic status; 95% CI, 95% confidence interval.

TABLE 4

Summary of latent difference score growth curve model

	Est.	SE	95% CI	Standardized estimate
<b>Intercepts</b>				
Age 17 imbalance	-1.699	0.390	[-2.464, -0.935]	-2.616
Age 20 HED	0.731	0.473	[-0.196, 1.659]	0.474
<b>Growth rates</b>				
Imbalance slope	<b>-0.077</b>	<b>0.024</b>	<b>[-0.124, -0.030]</b>	<b>-0.725</b>
HED slope	0.235	0.162	[-0.083, 0.553]	0.521
<b>Fixed effects</b>				
Age 17 imbalance → Age 20 HED	<b>0.373</b>	<b>0.156</b>	<b>[0.067, 0.679]</b>	<b>0.157</b>
Imbalance change → Age 20 HED	2.325	2.050	[-1.693, 6.342]	0.161
White → Age 20 HED	0.322	0.171	[-0.013, 0.657]	0.093
SES → Age 20 HED	<b>0.189</b>	<b>0.037</b>	<b>[0.117, 0.262]</b>	<b>0.250</b>
Male → Age 20 HED	-0.109	0.156	[-0.414, 0.195]	-0.035
Age 17 imbalance → HED change	0.069	0.053	[-0.035, 0.172]	0.099
Imbalance change → HED change	0.341	0.625	[-0.884, 1.567]	0.081
White → HED change	0.042	0.061	[-0.078, 0.162]	0.042
SES → HED change	<b>0.039</b>	<b>0.014</b>	<b>[0.012, 0.066]</b>	<b>0.177</b>
Male → HED change	-0.029	0.056	[-0.139, 0.082]	-0.032
<b>Growth factor variances</b>				
Imbalance intercept	<b>0.422</b>	<b>0.038</b>	<b>[0.347, 0.497]</b>	<b>1.000</b>
Imbalance slope	0.011	0.007	[-0.002, 0.025]	1.000
HED intercept	<b>2.070</b>	<b>0.172</b>	<b>[1.733, 2.407]</b>	<b>0.869</b>
HED slope	<b>0.193</b>	<b>0.030</b>	<b>[0.134, 0.251]</b>	<b>0.947</b>

Note: Bolded values indicate significance of parameters of interest at the 95% level of confidence.

Abbreviations: Est., estimate; SE, standard error; SES, socioeconomic status; 95% CI, 95% confidence interval.