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Genetically Influenced Change in Sensation Seeking Drives the Rise of Delinquent Behavior during Adolescence

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Abstract

Sensation seeking is associated with an increased propensity for delinquency, and emerging research on personality change suggests that mean-levels of sensation seeking increase substantially from childhood to adolescence. The current study tested whether individual differences in the rate of change of sensation seeking predicted within-person change in delinquent behavior and whether genetically influenced differences in rate of personality change accounted for this association. Sensation seeking and delinquent behavior were assessed biennially between ages 10–11 and 16–17 in a nationally representative sample of 7,675 youths from the National Longitudinal Study of Youth: Children and Young Adults (CNLSY). Analyses using latent growth curve modeling found that within-person change in sensation seeking was significantly and positively correlated with within-person change in delinquency from childhood to adolescence. Furthermore, behavioral genetic analyses of a subset of 2,562 sibling pairs indicated that there were substantial genetic influences on both initial levels of sensation seeking and change in sensation seeking during early adolescence, with over 80% of individual differences in change due to genetic factors. Finally, these genetically driven increases in sensation seeking were most important for predicting increases in delinquency, whereas environmental paths between sensation seeking and delinquency were not significant. These results suggest that developmental changes in delinquent behaviors during adolescence are driven by a genetically governed process of personality change.

Keywords

Sensation Seeking; Delinquency; Antisocial Behavior; Adolescence; Behavior Genetics; Family Design; Personality

Adolescence is a peak period of risk for delinquent behavior. While a small subset of delinquent youth have histories of serious behavioral problems dating back to early childhood, much delinquency arises *de novo* in adolescence, as part of a constellation of socially problematic risk-taking behaviors (such as binge drinking, risky sex, and reckless driving) that also increase dramatically during this developmental period (Jessor & Jessor, 1977; Moffitt, 1993). Unfortunately, despite concerted efforts to reduce delinquency and other forms of adolescent risk-taking, prominent reviews have found that even rigorous and well-designed prevention programs result in only modest benefits (Steinberg, 2004), leading to the conclusion that “the causes of antisocial behavior are not yet well enough understood to prevent it” (Moffitt, 2005, p. 533). Thus, better understanding of the developmental processes that underlie increases in delinquency during adolescence continues to be a

priority for research. *Sensation seeking* – a personality trait that predisposes people to select and prefer experiences that are novel, rewarding, or exciting – is a risk factor for adolescent delinquency (Cooper, Wood, Orcutt, & Albino, 2003; Horvath & Zuckerman, 1993; Newcomb & McGee, 1991; Sijtsema et al., 2010; Zuckerman, 2007). The current paper describes how emerging research on *change* in sensation seeking from late childhood to adolescence can provide new insights on the origins of the adolescent increase in delinquent behavior.

Contemporary research on personality has shown that sensation seeking – like many aspects of personality – exhibits substantial developmental change over the course of the lifespan (Caspi, Roberts, & Shiner, 2005; Roberts, Walton, & Viechtbauer, 2006). In particular, both cross-sectional and longitudinal studies have shown that mean levels of sensation seeking increase from childhood through mid-adolescence, peaking around age 16 (Cauffman et al., 2010; Harden & Tucker-Drob, 2011; Lynne-Landsman, Graber, Nichols, & Botvin, 2011; MacPherson, Magidson, Reynolds, Kahler, & Lejuez, 2010; Romer & Hennessy, 2007; Steinberg et al., 2008). Interestingly, increases in behavioral indices of sensation and novelty seeking are also evident in rodents (Adriani, Chairottie, & Laviola, 1998; Laviola, Macri, Morley-Fletcher, & Adriani, 2003; Stansfield & Kirstein, 2006), suggesting that primitive biological processes play an important role in this developmental change. In humans, mean-level changes in sensation seeking have been hypothesized to be due to maturational changes in the adolescent brain (Casey, Getz, & Galvan, 2008; Somerville, Jones, & Casey, 2010; Steinberg et al., 2009; Steinberg, 2008). As Somerville et al. (2010) note, subcortical brain regions involved in responding to novel, rewarding, or emotional stimuli – including the amygdala and the nucleus accumbens – “show an exaggerated response profile in adolescents” (p. 131). Specifically, adolescents, relative to children and adults, exhibit a stronger response to large monetary rewards in the nucleus accumbens (Ernst et al., 2005; Galvan et al., 2006), and show a prolonged timecourse of striatal responding to reward (Fareri, Martin, & Delgado, 2008). These changes in brain activity may be related to pubertal development, which has been linked to changes in neural circuits involved in reward-motivated behavior, particularly in the nucleus accumbens and in dopaminergic pathways to the prefrontal cortex (Blakemore, Burnett, & Dahl, 2010; Forbes & Dahl, 2010; Kuhn et al., 2010).

While there is clear evidence for mean-level changes in sensation seeking, these mean age-trends belie substantial individual differences in the magnitude and alacrity of personality development (Roberts & Mroczek, 2008). Previous longitudinal research has found that youth differ in the extent to which they exhibit age-related change in sensation seeking (Harden & Tucker-Drob, 2011; Quinn & Harden, In Press). It is likely that these individual differences in change in sensation seeking are governed by genetic factors. In particular, recent molecular genetic work has found cross-sectional associations between sensation seeking and genetic polymorphisms related to the dopamine system (Derringer et al., 2010; Lang, Bajbouj, Sander, & Gallinat, 2007; Laucht, Becker, & Schmidt, 2006; Ray et al., 2009; but see Powell & Zeitsch, 2011 for a critique). More generally, genetic factors have been found to account for 34% – 69% of the variance in sensation seeking in both adults (Eysenck, 1983; Fulker, Eysenck, & Zuckerman, 1980; Hur & Bouchard, 1997; Stoel, De Geus, & Boomsma, 2006) and adolescents (Koopmans, Boomsma, Heath, & van Doornen, 1995; Miles et al., 2001; Mustanski, Viken, Kaprio, & Rose, 2003), with minimal sex differences in heritability (Koopmans et al., 2005; Stoel et al., 2006). The hypothesis that change in sensation seeking is due to genetic factors is consistent with the conceptualization of personality change as due to “biologically based intrinsic maturation” (Costa & McCrae, 2006, p. 26). In contrast, other personality theorists have emphasized the role of shifting environmental experiences in determining personality change (Roberts, Walton, & Viechtbauer, 2006a; Roberts, Walton, & Viechtbauer, 2006b). However, this debate has

largely focused on personality change during the transition to adulthood; no previous genetically informed study has specifically tested the extent to which genetic factors account for individual differences in *change* in personality from late childhood to mid-adolescence.

Understanding the origins of individual differences in personality change during adolescence is particularly important in light of recent theoretical work that implies that these age-related increases in sensation seeking drive age-related increases in delinquent behavior during early adolescence. According to the *dual systems theory* of adolescent risk-taking (Casey et al., 2008; Casey, Jones, & Somerville, 2011; Ernst, Pine, & Hardin, 2006; Somerville et al., 2010; Steinberg, 2008), the developmental “spike” in risk-taking and delinquent behavior is due to the rapid increases in sensitivity to novelty and rewards that occur in early adolescence (particularly when paired with the relatively slow maturational timecourse of cortical structures that govern inhibition, planning, and executive control). Although this neurodevelopmental account of adolescent risk-taking has gained popularity, the behavioral predictions of the dual systems model have not been widely tested using longitudinal data, which are necessary to examine hypotheses concerning the mechanisms of developmental change. Rather, the preponderance of evidence for the dual systems model has come from cross-sectional research showing that mean levels of sensation seeking, reward dependence, and neural activity in response to reward are higher in adolescents relative to either children or adults – paralleling the age-trends evident for delinquency and risk-taking (e.g., Cauffman et al., 2010; Galvan et al., 2006, 2007; Hare et al., 2008; Steinberg et al., 2008). Yet, it is obviously possible for two constructs to have coincident mean age-trends without one being a mechanism for the other. (For example, adolescents also spend more time studying geometry than either children or adults, resulting in an inverted U-shaped curve that parallels the age-trends seen for delinquent behavior, but it would be implausible to claim that the emergence of geometry-studying accounts for the rise of delinquent behavior.) A more rigorous test of the interdependency of sensation seeking and delinquency is whether individual differences in change in sensation seeking predict individual differences in change in delinquent behavior. That is, does the adolescent who experiences the most rapid increase in sensation seeking also demonstrate the most rapid increase in delinquent behavior? Previous research on substance use has found evidence for longitudinal associations with sensation seeking (Crawford, Pentz, Chou, Li, & Dwyer, 2003; MacPherson, Magidson, Reynolds, Kahler, & Lejeuz, 2010; Quinn & Harden, In Press), but to our knowledge, this test has not been applied to delinquent behavior. While correlations between individual differences in longitudinal change are not, of course, conclusive evidence of a causal relationship, it is nevertheless valuable to assess the predictions of the dual system model using longitudinal methods that can discriminate whether *within-person* change in sensation seeking is accompanied by within-person change in delinquency. Moreover, it is also necessary to test the directionality of the association between sensation seeking and delinquency, as it is also possible that involvement in delinquent behavior (and the environmental contexts that accompany such involvement) drive personality change, rather than the reverse.

To address these gaps in previous research, the current study uses longitudinal, genetically informed data on the development of sensation seeking and delinquent behavior between age 10–11 and age 16–17 in a nationally-representative sample of 7,675 youth, including 2,562 sibling pairs of varying degrees of genetic relatedness. Our analyses focused on three research questions. First, to what extent do within-person increases in sensation seeking predict within-person increases in delinquent behavior, as predicted by the dual systems model of adolescent risk-taking? Second, to what extent are individual differences in change in sensation seeking attributable to genetic influences? Third, to what extent do these genetic influences on change in sensation seeking drive increases in delinquency?

Method

Participants

Mother Generation: The National Longitudinal Survey of Youth (NLSY79)—The Bureau of Labor Statistics designed and funded the NLSY79 in order to study workforce participation in the U.S. A complex survey design was used to select a nationally-representative sample of 3,000 households containing 6,111 youth, plus an additional oversample of 3,652 African-American and Hispanic youth, aged 14–21 years as of December 31, 1978. The response rate for the initial NLSY79 survey was over 90% of the eligible sample, and participants have been re-interviewed annually from 1979 to 1994 and biennially since 1994. Retention rates for follow-up assessments of the NLSY79 sample were greater than 90% for the first 16 waves and greater than 80% for subsequent waves.

Adolescent Generation: The NLSY79 Children and Young Adults (CNLSY)—Beginning in 1986, the biological children of the NLSY79 women were assessed biennially (Chase-Lansdale, Mott, Brooks-Gunn, & Phillips, 1991). The initial participation rate was 95%, and the average retention rate through 2006 was approximately 90%. Beginning in 1988, children over the age of 10 completed individual supplemental interviews that assessed their attitudes and behaviors. Finally, beginning in 1994, older children who were age 15 by the end of the survey calendar year (termed “young adults” by the NLSY survey administrators) were administered a separate interview. As of 2006, 11,466 children were identified as having been born to 6,283 NLSY79 women. After weighting for sample selection, the average NLSY79 woman has had 1.9 children, which is more than 90% of their ultimate predicted childbearing. The current study uses data from $N = 7,675$ youth who reported on their delinquent behavior at least once between the ages 10–17.

Sibling Pairs Sub-Sample—The original purpose of the NLSY data collection was not behavioral genetic analyses, and the genetic relatedness of siblings residing in the same household was not specifically assessed. Rodgers and colleagues (Rodgers, Johnson, & Bard, 2005) have, however, developed an algorithm that classifies CNLSY participants into kinship pairs.¹ Using these kinship links, genetic relatedness (i.e., the correlation between additive genes) between pairs was assigned according to genetic theory: 0.25 for half-siblings, 0.375 for ambiguous siblings, and 0.50 for full siblings. The only difference between these coefficients and the standard measures of relatedness derived from a quantitative genetic model is that the algorithm assigned a value midway between half- and full-sibling values when it could not be ascertained whether siblings shared a father. An extensive series of validation analyses have compared the biometrical composition of well-characterized phenotypes, such as adult height, obtained using the CNLSY kinship links with results obtained in other studies. The links have been used in a number of published behavioral genetic studies (e.g., D’Onofrio et al., 2008; Mendle et al., 2009; Van Hulle, Rodgers, D’Onofrio, Waldman, & Lahey, 2007). For behavioral genetic analyses, the current study makes use of a sibling pairs subsample comprising $N = 2,562$ sibling pairs (778 half-siblings, 267 ambiguous siblings, and 1,517 full siblings), where one sibling pair was randomly selected per nuclear family.

¹All CNLSY siblings in a household were unequivocally the biological children of a single mother; classifying children as full versus half siblings depended on the determination of whether they shared a biological father. This determination was based on maternal report of “Is the biological father of this child currently living in the household?” (assessed annually 1986–2000) and child report of “How far away does your biological father live from your home?”. A detailed description of the kinship classification algorithm can be obtained from the first author upon request.

Measures

Maternal characteristics—*Socioeconomic status* (SES) was measured using self-reported total family income (median = \$22,500 per year), including government support and food stamps but excluding income received by unmarried cohabiting partners, when the mother was 30 years old. (Because cohabiting relationships are at higher risk of dissolution, and mothers who end cohabiting relationships are typically not entitled to a portion of the former partner's earnings, income from cohabiting partners is less reflective of relatively stable differences in socioeconomic status; Kalil & Ryan, 2010.) *Maternal cognitive ability* was measured in the 1980 assessment using composite scores on the word knowledge, paragraph comprehension, math knowledge, and arithmetic reasoning subtests of the Armed Services Vocational Aptitude Battery (ASVAB). *Maternal education* was measured using maternal report of the number of years of school they had completed ($M = 13.4$ years, $SD = 2.50$ years; approximately 9% of the sample reported 11 years or less). Finally, maternal age at first birth was calculated using the date of birth for the mother and her first child ($M = 21.9$, $SD = 4.52$, range = 11.7 – 38.3 years).

Sensation seeking—*Sensation-seeking* was measured by youth self-report on the following three items: (1) “I enjoy taking risks”; (2) “I enjoy new and exciting experiences, even if they are a little frightening or unusual”; and (3) “Life with no danger in it would be too dull for me.” These items comprised a scale intended to measure propensity for risk-taking; items were drawn from multiple inventories (NLSY79 Children and Young Adults, 2009). All items were rated on a 4-point scale ranging from *Strongly Disagree* to *Strongly Agree*. Internal reliability for these items was good (Cronbach's $\alpha = .69$, aggregating across ages). Moreover, as previously reported in Harden & Tucker-Drob (2011), the pattern of correlations between sensation seeking and Big Five personality traits in the full CNLSY sample were consistent with previous empirical research, with significant and positive correlations with concurrent measures of Extraversion and Openness. Sensation-seeking sum scores were residualized for all demographic and maternal characteristics and then standardized to z-scores ($M=0$, $SD=1$). The right panel of Figure 1 illustrates the mean-level age-trends in sensation seeking.

Delinquency—Beginning in 1988, children who were at least 10 years old but not yet 15 were administered the “Child Self Administered Supplement” (CSAS), which included 6 items from the Self-Report of Delinquency (SRD; Elliott & Huizinga, 1983): (1) hurt someone bad enough to need bandages or a doctor; (2) lied to a parent about something important; (3) took something from a store without paying for it; (4) intentionally damaged or destroyed property that did not belong to you; (5) had to bring your parent(s) to school because of something you did wrong; and (6) skipped a day of school without permission. Beginning in 1994, youth who were 15 years old or older were administered the “Young Adult” assessment, which also included the 6 SRD items. Because of budgetary constraints, the SRD items were dropped from the YA assessment (but not CSAS) in 2000 only. Each of the SRD items was dichotomized as “never” versus “at least once or more” and summed. Symptom counts thus ranged from 0 to 6 (median=1, $M=1.29$, $SD=1.39$). Previous analyses of CNLSY data (described in Harden et al., 2009) have tested the criterion validity of the SRD items and found that these items significantly predict, for both males and females, the odds of being convicted for a non-trivial criminal offense (excluding drug possession), controlling for a broad variety of demographic and contextual background variables. Cronbach's α for the 6 SRD items ranged from 0.64 to 0.68 across assessment waves. The left panel of Figure 1 illustrates the mean-level age-trends in delinquent behavior.

Analytic Methods

Analyses focused on three major questions: (1) do within-person increases in sensation seeking predict within-person increases in delinquent behavior?; (2) to what extent are individual differences in change in sensation seeking due to genetic influences; and (3) to what extent do these genetic influences on change in sensation seeking drive increases in delinquency? For Question #1, we examined whether change in sensation seeking significantly predicted change in delinquency, using latent growth curve modeling (LGM; McArdle & Nesselrode, 2003) and data from the full CNLSY sample (illustrated in Figure 2). The basic LGM can be written as follows:

$$Y[t] = y_L + A[t] \times y_S + e[t]$$

where $Y[t]$ is the observed score at each age t , y_L is a latent score representing the initial level at age 10, y_S is a latent score representing the magnitude of change over time, $A[t]$ is a vector of time-specific ‘basis’ coefficients representing the shape of change over time, and $e[t]$ is a vector of time-specific residual errors. The latent level and change factors, in turn, are assumed to be multivariate normal. Constraining the values of $A[t]$ constitutes a test of specific hypotheses regarding the shape of change. In the current analyses, both sensation seeking and delinquency were modeled using a “latent basis” LGM, in which the basis coefficients were fixed to equal 0 and 1 for the first two timepoints and freely estimated from the data for the remaining timepoints. Preliminary analyses suggested that a latent basis model of change in sensation seeking fit significantly better than a linear model (linear model: $\chi^2 = 51.08$, $df = 5$, CFI = .905, TLI = .886, RMSEA = .057; latent basis model: $\chi^2 = 15.51$, $df = 3$, CFI = .974, TLI = .949, RMSEA = .038; model fit comparison: $\Delta\chi^2 = 35.53$, $\Delta df = 2$, $p < .001$.) This was also true for change in delinquency (linear model: $\chi^2 = 109.28$, $df = 5$, CFI = .927, TLI = .913, RMSEA = .054; latent basis model: $\chi^2 = 53.79$, $df = 3$, $p < .001$, CFI = .965, TLI = .929, RMSEA = .048; model fit comparison: $\Delta\chi^2 = 55.49$, $\Delta df = 2$, $p < .001$.) The key parameter of interest for the LGM of delinquency and sensation seeking was the correlation between the slope factors: is within-person change in sensation seeking significantly and positively correlated with within-person change in delinquency?

As a follow-up analysis related to Question #1, we also modeled longitudinal data on sensation seeking and delinquency using an auto-regressive, cross-lagged analysis (illustrated in Figure 3). Cross-lagged models and latent growth curve models have been described as complementary methods for the analysis of longitudinal data. The LGM approach models *intra-individual change* as a random effect (i.e., does an individual adolescent increase in his or her own delinquent behavior from age 10 to age 12); in contrast, the cross-lagged analysis models *change relative to the group* as a fixed effect (i.e., do adolescents who are higher than their peers at age 10 remain higher than their peers at age 12)? For this reason, the LGM approach is “better suited for studying individual differences in development and change”, which is the focus of the current investigation (Curran, 2000); however, these models are ambiguous regarding the direction of the association between correlated changes. One advantage of the cross-lagged analysis over the LGM approach is that the former specifies *directional* relationships between the constructs of interest, i.e., does X predict future levels of Y, and does Y predict future levels of X, after accounting for the temporal stability of both X and Y. Thus, we also fit a cross-lagged analysis of the longitudinal data, in order to distinguish between alternative hypotheses regarding the direction of the relation between sensation seeking and delinquency – does sensation seeking predict future levels of delinquent behavior, and/or does involvement in delinquent behavior predict future levels of sensation seeking? The finding that levels of delinquency have prospective associations with subsequent levels of sensation seeking

would suggest that personality change, rather than driving increases in delinquent behavior, is instead an effect of behavioral change.

To address Question #2, we estimated the extent to which individual differences in change in sensation seeking were due to genetic influences, using data from the sibling pairs subsample. As shown in Figure 4, we used a classical behavioral genetics model combined with LGM, in which the variance in each growth factor (i.e., level of sensation seeking and change in sensation seeking) was decomposed into three latent factors: additive genetic influences (*A*); environmental influences that make siblings in the same home similar to each other (shared environment, or *C*); and environmental influences that make siblings different from each other, plus measurement error (non-shared environment, or *E*). The correlation between the *A* factors in the first and second sibling in each pair are fixed according to genetic theory (.5 for full siblings, .375 for ambiguous siblings, .25 for half siblings); the correlation between the *C* factors were fixed to 1.0. In this case, the *A*, *C*, and *E* components were standardized; the square of the standardized path from *A* equals the familiar heritability coefficient. The key parameter of interest for the behavioral genetic model of sensation seeking was the *A* component of the slope factor, which reflects whether individual differences in change in sensation seeking are due to genetic factors.

Finally, to address Question #3, we estimated the genetic and environmental association between sensation seeking and delinquency, again using data from the sibling pairs subsample. This model (for one sibling per pair) is shown in Figure 5. The key parameters of interest in this model are the genetic and environmental cross-paths from the *A*, *C*, and *E* components of sensation seeking to the growth factors of delinquency. These paths test the extent to which the association between sensation seeking and delinquency is due to common genetic versus environmental influences. In particular, the path from the *A* component of change in sensation seeking to change in delinquency tests the extent to which genetic differences in the magnitude of personality change during adolescence account for the coincident increase in delinquent behavior.

All models were estimated in the software program Mplus (Muthen & Muthen, 1998–2011) using full information maximum likelihood (FIML), which has been recommended as the preferred method for accounting for missing data (Schafer & Graham, 2002). In addition, standard errors and model fit statistics were adjusted for non-independence of data from children from the same family (i.e., sibling clusters; Asparouhov & Muthén, 2006). For the phenotypic models, model fit was evaluated using the root mean square error of approximation (RMSEA; Steiger, 1990), comparative fit index (CFI; Bentler, 1990), and the standardized root mean square residual (SRMR; Bentler, 1995). Based on Hu & Bentler (1999), cut-offs values for acceptable model fit were $RMSEA \leq .06$, $CFI \geq .95$, and $SRMR \leq .08$. The only index of absolute model fit available for the behavioral genetic models was SRMR; relative fit of behavioral genetic models were compared using the Bayesian Information Criterion (BIC; Raftery, 1993), where smaller values indicate a more parsimonious representation of the data. All models included youth race/ethnicity (coded as African-American, Hispanic/Latino, or White) and gender as statistical covariates.

Results

Question #1: Are Within-Person Increases in Sensation Seeking Associated with Within-Person Increases in Delinquent Behavior?

Standardized estimates for key parameters from the LGM of delinquency and sensation seeking are shown in Figure 2. The overall fit of this model was good ($CFI = .97$; $RMSEA = .02$, $SRMR = .02$). There were significant individual differences in initial level of delinquency ($Var = .641$, $SE = .130$, $P < .01$), change in delinquency ($Var = .206$, $SE = .041$,

$P < .01$), initial level of sensation seeking ($\text{Var} = .684$, $SE = .122$, $P < .01$), and change in sensation seeking ($\text{Var} = .103$, $SE = .025$, $P < .01$). As is often observed in LGM analyses, there was a significant and negative association between initial levels of sensation seeking and delinquency and subsequent change in these constructs: Youth who were highest in sensation seeking and delinquency at age 10 demonstrated the least change during adolescence. Consistent with previous research on this topic, there was a significant and positive correlation ($r = .36$) between level of sensation seeking and level of delinquency. Moreover, after controlling for initial level, there was a significant and positive correlation ($r = .42$) between *change* in sensation seeking and change in delinquency. Finally, residual variance in sensation seeking at each age was modestly correlated with residual variance in delinquency ($r_s .11$ to $.16$, $P < .05$). *Overall, results from analyses of Question #1 indicated that within-person increases in sensation seeking were significantly and positively associated with within-person increases in delinquent behavior between the ages of 10–11 and 16–17.*

Question #1B: Does Involvement in Delinquent Behavior Predict Future Levels of Sensation Seeking?

The finding of significant correlated change between sensation and delinquency is ambiguous regarding the direction of the association. Correlated change could result from sensation seeking influencing subsequent delinquency or from involvement in delinquent behavior influencing subsequent personality. To test the directionality of this association, we next fit an autoregressive cross-lagged analysis.

Standardized parameter estimates from the autoregressive cross-lagged analysis are shown in Figure 3. The overall fit of this model was good ($\text{CFI} = .97$, $\text{RMSEA} = .03$, $\text{SRMR} = .03$). As expected, there was moderate stability in both delinquent behavior (stabilities = $.32$ – $.39$) and sensation seeking (stabilities = $.28$ – $.37$): Youth who were above the population mean in each construct at an earlier age were likely to be above the mean at later ages. At each age, there was a significant and positive concurrent association between delinquency and sensation seeking ($r_s = .17$ – $.22$, $P_s < .01$). Moreover, sensation seeking significantly predicted subsequent involvement in delinquent behavior two years later across ages ($\beta_s = .09$ – $.11$, $P_s < .01$). In contrast, the associations between delinquent behavior and subsequent levels of sensation seeking were more modest ($\beta_s = .02$ – $.05$) and were not significant at any age. *Overall, results from analyses of Question #1B indicated that the longitudinal association between sensation seeking and delinquency cannot be attributed to an effect of involvement in delinquent behavior on subsequent levels of sensation seeking; rather, evidence from cross-lagged analysis indicated that sensation seeking predicted future levels of delinquency.*

Question #2: How Much Variance in Change in Sensation Seeking is Due to Genetic Differences?

Having established the direction of the association between sensation seeking and delinquency, we then returned to a LGM framework, in order to estimate the relative contribution of genetic and environmental factors to individual differences in change in sensation seeking. Unstandardized parameter estimates for the behavior genetic model of growth in sensation seeking are summarized in Table 1 (model illustrated in Figure 4). Overall model fit was acceptable ($\text{SRMR} = .08$). Sixty-two percent of the variance in initial level of sensation seeking was due to genetic factors; the remaining variance (38%) was due to environmental influences that differed between siblings. Shared environmental influences on initial level of sensation seeking were negligible. As mentioned above, a substantial portion of the variation in change in sensation seeking was shared with initial level of sensation seeking. Of the remaining unique variance in change in sensation seeking, 83%

was due to genetic influences $[\text{.24}^2/(\text{.24}^2 + \text{.00}^2 + \text{.11}^2)]$. The remaining variance was due to modest and non-significant non-shared environmental factors, and shared environmental influence on change in sensation seeking was negligible. Because shared environmental influences on level and change were negligible, these shared environmental influences were fixed to zero for subsequent models. *Overall, results from analyses of Question #2 indicate that individual differences in change in sensation seeking from ages 10–11 to 16–17 are primarily due (83%) to genetic differences.*

Question #3: Do Genetic Influences on Change In Sensation Seeking Drive Increases in Delinquent Behavior in Adolescence?

Finally, having established that (1) within-person change in sensation seeking is significantly associated with within-person change in delinquency, and (2) within-person change in sensation seeking is substantially influenced by genetic factors, we fit a multivariate behavioral genetic model to test the extent to which genetic influences on sensation seeking change account for the rise of delinquency in early adolescence (illustrated in Figure 5). Unstandardized parameter estimates for this model are summarized in Table 2. Overall model fit was fair (SRMR = .08; BIC = 35282.50). The genetic and environmental cross-paths, which are of primary theoretical interest in this model, are in the bottom half of Table 2. First, the association between initial level of sensation seeking and level of delinquency was primarily due to common genetic influences (SS-level_A → DEL-level = 0.44), whereas the non-shared environmental path was not significantly different from zero (SS-level_E → DEL-level = -.22). Second, neither the genetic nor the non-shared environmental component of initial level of sensation seeking predicted change in delinquency; this is consistent with the non-significant phenotypic correlation between SS-Level and DEL-change (reported above). Third, the association between change in sensation seeking and change in delinquency was also due to common genetic influences (SS-change_A → DEL-change = 0.14), whereas the non-shared environmental path was again not significant (SS-change_E → DEL-change = 0.09).

We then fit a trimmed model in which the non-significant cross-paths (all non-shared environmental cross-paths, and the genetic path between SS-level and DEL-change) were fixed to zero. Model fit for the trimmed model was marginally improved (SRMR = .08, BIC = 35263.72), indicating that the trimmed model was a more parsimonious representation of the associations between sensation seeking and delinquency. Standardized parameter estimates from the final trimmed model are shown in Figure 6. Of the total variance in initial level of delinquency (controlling for gender and race/ethnicity), 35% was due to genetic influences on level of sensation seeking, 46% was due to genetic influences independent of sensation seeking, and 19% was due to shared environmental influences independent of sensation seeking. Put differently, of the total *genetic* variance in initial levels of delinquency, 43% were accounted for by genes influencing initial levels of sensation seeking.

Of the variance in change in delinquency (unique of initial levels), 30% was due to genetic influences on change in sensation seeking, 6% was due to genetic influences independent of sensation seeking, and the remaining 64% was due to non-shared environmental influences independent of sensation seeking. Put differently, of the *genetic* variance in change in delinquency, unique of initial levels, 84% was due to genes influencing the increase in sensation seeking. *Overall, results from analyses of Question #3 indicate that genes influencing change in sensation seeking are significantly associated with increases in delinquency during adolescence ($R^2 = 30\%$) and account for over 80% of genetic variance in delinquency change.*

Discussion

Adolescence is a unique transitional period in the human lifespan, characterized by rapid gains in physical ability, cognitive skills, and emotional sophistication, but also characterized by escalating vulnerabilities to diverse forms of psychopathology and behavioral problems. Stemming from imaging research on adolescent brain development, the dual systems model (Casey et al., 2007; Steinberg, 2008) has posited that adolescents' increasing vulnerability to externalizing behavior, in particular, is driven by maturational changes in the responsiveness of subcortical systems to emotional and motivational cues. Consistent with this model, previous research has found that adolescents exhibit a marked increase in sensation seeking between the ages of 10 and 16. The current study used longitudinal, behavioral genetic data to further investigate the relation between these personality changes and increases in delinquent behavior in adolescence. Results from latent growth curve models indicated that within-person change in sensation seeking was significantly associated with within-person change in delinquency. Moreover, follow-up analyses using cross-lagged models found that a reverse causal effect of involvement in delinquent behavior on subsequent personality cannot account for this pattern of correlated change. Sensation seeking consistently predicted future delinquency, but delinquency did not significantly predict future sensation seeking. This constitutes some of the first evidence to derive from longitudinal data that personality change during early adolescence drives increases in delinquent behavior.

While research on personality change during the transition *out of* adolescence and into adulthood has largely emphasized the changing social demands imposed by the assumption of adult social roles (e.g., Roberts, Caspi, & Moffitt, 2003; Roberts, Walton, Vichtbauer, 2006), there has been less theoretical and empirical work on the causes of personality change during the transition *into* adolescence, with the few studies focusing on the early adolescent transition emphasizing the role of maturational changes in the adolescent brain (e.g., Steinberg et al., 2008). No previous genetically informed study has tested the extent to which individual differences in personality change from childhood to adolescence can be accounted for by differences in genes versus differences in environmental experiences. In fact, to our knowledge there have been only two previous studies – of any age – that have used a behavioral genetic, multiple-wave design and latent growth curve modeling to examine genetic and environmental contributions to within-person personality change (Bleidorn, Kandler, Reimann, Angleitner, & Spinath, 2009; Hopwood et al., 2010). These studies – plus a handful of others not using latent growth curve modeling approaches (e.g., Blonigen, Carlson, Hicks, Krueger, & Iacono, 2008; Kandler et al., 2010; McGue, Bacon, Lykken, 1993) – have found evidence for substantial environmental influence on personality change in late adolescence and adulthood. In contrast, the current analyses found that change in sensation seeking from late childhood to mid-adolescence was due almost entirely to genetic factors ($h^2 = 83\%$), with negligible shared environmental influence and modest (and non-significant) non-shared environmental influence. Our estimates of genetic influence on personality change may be much higher than estimates from previous studies because we focused on personality change during early adolescence, a developmental period characterized by rapid and extensive biological change, rather than during adulthood, when the individual has already attained biological maturity. In addition, we examined a more specific facet of personality, rather than a broad-band personality dimension, which may partially account for the different pattern of results. Further genetically-informed, longitudinal research is necessary to test whether genetic influences on personality change are similarly high for other traits measured in adolescence, or for sensation seeking measured at other points in the lifespan.

Given the conceptual link between sensation seeking and mesocorticolimbic dopamine pathways responsive to reward and novelty, these genetic differences in change may be due to polymorphisms in genes related to the dopamine system, which have been linked to levels of sensation seeking in previous cross-sectional research (e.g., Derringer et al., 2010). A more speculative hypothesis, stemming from new research on the impact of pubertal development on the structure and function of dopaminergic neural circuits (Blakemore et al., 2010; Forbes & Dahl, 2010; Kuhn et al., 2010), is that genetic differences in sensation seeking change are due, in part, to genetic differences in the timing or rate of puberty. Certainly, pubertal timing is strongly heritable (e.g., Ge, Natsuaki, Neiderhiser, & Reiss, 2008), and a handful of previous studies have found that higher pubertal status or earlier pubertal timing predicts elevated sensation seeking, above and beyond chronological age (Martin et al., 2001, Martin et al., 2002; Steinberg et al., 2008). Moreover, youth differ in the rapidity of pubertal development (i.e., pubertal tempo; Mendle, Harden, Brooks-Gunn, & Graber, 2010), although no genetically informed studies of pubertal tempo have been conducted to-date.

Additionally, we found evidence for substantial genetic influence on delinquent behavior in adolescence. This is consistent with a large body of previous behavioral genetic research (Rhee & Waldman, 2002), including previous analyses of NLSY data (Rodgers, Buster, & Rowe, 2001; Van Hulle, et al., 2007). Moreover, these heritability estimates are consistent with research showing intergenerational transmission of antisocial behavior (D'Onofrio et al., 2007; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003). Interestingly, previous research has found the genetic variance in antisocial behavior increases substantially over the course of adolescence (Bergen, Gardner, & Kendler, 2007; Burt & Neiderhiser, 2009; Eley, Lichtenstein, & Moffitt, 2003). For example, Burt and Neiderhiser (2009) found that genetic influences accounted for 64% of the variance in delinquent behavior in early adolescence (M age = 11.4), but accounted for over 80% of the variance just 3 years later (M age = 14.0 years). In the current analyses, over 80% of the unique genetic variance in change in delinquency was accounted for by genetic influences on change in sensation seeking. Given that (a) the association between change in sensation seeking and delinquency is clearly mediated via genetic pathways, and (b) change in sensation seeking is itself highly heritable, future research should examine the hypothesized neural underpinnings of increases in sensation seeking as endophenotypes for the emergence of genetic variation in delinquent behavior (Gottesman & Gould, 2003).

It is important to note that our results, which indicate that genes account for very large portions of variation in the development of sensation seeking, do not mean that personality development is deterministic or that environmental inputs are inconsequential. It is well-established that behavioral genetic estimates of heritability include both the direct effect of genes and the effects of gene-environment correlation. That is, to the extent that an individual is systematically selected into environmental experiences as a function of their genetic predispositions, and these environmental experiences influence a given phenotype, this process of gene-environment correlation will serve to increase estimates of heritability. Caspi et al. (2005) posited that environmental experiences tend to reinforce the personality characteristics that led individuals to be selected into these experiences, resulting in both increasing stability and increasing heritability of individual differences in personality with age. For example, an adolescent may choose certain peer groups based on his or her initial levels of sensation seeking, and these peer socialization experiences, in turn, may reciprocally influence sensation seeking. This reciprocal process will result in a net increase in both the “match” between the adolescent’s genotype and his or her environment *and* in the estimate of the heritability of sensation seeking. It is also likely that heritable variation in personality change represents genetic differences in children’s responses to the unique social

challenges of adolescents. In both scenarios, genetic variance in sensation seeking can be quite large, while at the same time being dependent on the environment.

Better understanding of the ways in which an individual's genetic predispositions are translated into personality change – and ultimately into delinquent behavior – may offer new insights for intervention programs: In what environmental contexts is the link broken between sensation seeking, which is not, in and of itself, a harmful personality trait, and delinquency, which is “one of the most costly and vexing problems” faced by society (Dodge, 2003, p. 8)? Previous behavioral genetics research has suggested that heritable variation in delinquent behavior is attenuated in adolescents from families characterized by low levels of dysfunction (Button, Scourfield, Martin, Purcell, & McGuffin, 2005), by warm, positive parenting (Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007), and by few parent-child relationship problems (Hicks, South, DiRago, Iacono, & McGue, 2009). It is possible that these environmental advantages minimize genetic variation by disrupting the association between sensation seeking and delinquency, perhaps by providing external controls for adolescents' impulses. In addition, previous authors have suggested providing adolescents with alternative outlets for sensation seeking; adventure sports, for example, provide highly stimulating experiences (and a certain level of risk) but are not deviant or antisocial (White, Labouvie, & Bates, 1985). To our knowledge, however, no research has tested whether socially sanctioned risk-taking activities mitigate adolescents' propensities for delinquent behavior, or whether such activities only serve to reinforce sensation seeking tendencies. Research directly addressing environmental modifiers of the sensation seeking-delinquency link will prove important for examining these issues.

Limitations

The current study has two limitations that are worth noting. First, the sensation seeking assessment used in the CNLSY is very brief. While unfortunate, brevity of measurement is a typical and necessary trade-off in population-based studies with a broad scope and a large sample size (in this case, over 7,000 individuals). The three items used here map most closely to the Thrill and Adventure Seeking dimension of sensation seeking (Zuckerman, Eysenck, & Eysenck, 1978). Notably, multivariate behavioral genetic analyses have demonstrated that the various dimensions of sensation seeking have correlated but distinct genetic influences (Koopmans et al., 1995; Stoel et al., 2006), thus it remains unclear whether genetically-driven change in other facets of sensation seeking (Boredom Susceptibility, Experience Seeking, or Disinhibition) would account for additional variance in change in delinquency. Nevertheless, several pieces of evidence support our contention that, despite its brevity, the CNLSY sensation seeking measure is valid and reliable. As previously noted in Harden & Tucker-Drob (2011), the mean age-trends in the current study are highly consistent with previous studies using more extensive self-report and behavioral measures of sensation seeking (e.g., Steinberg et al., 2008). In addition, our measure of sensation seeking is significantly related in the CNLSY sample with Big Five personality traits (extraversion and openness) that have been previously identified as theoretically and empirically related to sensation seeking. Most importantly, our sensation seeking measure accounted for substantial variance in change in delinquent behavior, indicating that is tapping a developmentally meaningful construct. Generally, we believe that results from longitudinal data, particularly from a nationally-representative sample, provide an important complement to cross-sectional results derived from more detailed assessment of convenience samples.

Second, the current study assessed only delinquent behaviors that were non-violent, rule-breaking activities, which previous research has shown to be etiologically and developmentally distinct from aggressive behaviors (Achenbach, 1991; Burt, 2009). Aggressive behavior is more developmentally stable than rule-breaking (Stanger,

Achenbach, & Verhulst, 1997; Tremblay, 2003; Verhulst, Koot, & Berden, 1990). Not only do aggressive behaviors have an earlier median age-at-onset, with the highest rates of aggression seen in youth with onset before age 10 (Lahey et al., 1998; Lahey, Loeber, Quay, Frick, & Grimm, 1992), adolescents who exhibit aggressive behaviors are also more likely to persist in antisocial behavior across the transition to adulthood (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). Moreover, aggressive behavior also shows greater *genetic* stability. For example, Eley et al. (2003) found a near perfect correlation ($r_A = .99$) between genetic influences on aggression in childhood and aggression in adolescence. Burt & Neiderhiser (2009) have similarly reported that genetic influences on aggression, as distinct from delinquency, remained stable from childhood through adolescence. Thus, the current results should be considered relevant for the increases in non-aggressive delinquency that typify the “adolescent-limited” or “adolescent-onset” trajectories of antisocial behavior. Future research will be needed to examine the relation between sensation seeking and aggressive, “life-course-persistent” trajectories of antisocial behavior. It is possible that, for aggressive outcomes, other facets of personality (e.g., fearlessness, callous-unemotionality; Raine, Reynolds, Venables, Mednick, & Farrington, 1998; Frick, Cornell, Barry, Bodin, & Dane, 2003) may be more important.

Conclusion

Sensation seeking has long been known as a risk factor for delinquent behavior in adolescence and adulthood, but little research has examined the role of change in sensation seeking during the transition to adolescence. The current study, using longitudinal, behavioral genetic data from a nationally representative sample of youth, suggests that the predominant influence on change in sensation seeking is genetic factors, and that these genetically-influenced changes in sensation seeking drive increases in delinquent behavior from childhood to mid-adolescence.

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Figure 1.

Mean Levels of Sensation Seeking and Delinquent Behavior by Age.

Note. Bands represent SEs. Sensation seeking on standardized (z-score) scale. Delinquency measured as symptom count (0–7).

Figure 2.

Latent Growth Curve Model of Delinquency and Sensation Seeking, with Standardized Parameter Estimates.

Note. Intercept factor loadings fixed to 1.0 across all ages; slope factor loadings fixed to 1 at age 12–13 and estimated freely from data for subsequent ages. *SEs* are in parentheses.

Analysis controlled for youth gender and race/ethnicity (not shown). Parameters with * are significant at $P < .05$.

Figure 3.

Cross-Lagged Analysis of Delinquency and Sensation Seeking, with Standardized Parameter Estimates.

Note. Parameters with * are significant at $P < .05$. Analysis controlled for youth gender and race/ethnicity (not shown).

Figure 4.

Behavioral Genetic Model of Change in Sensation Seeking.

Note. Only one sibling per pair is shown. A=Additive Genetic, C=Shared Environmental, E=Non-shared Environmental. Correlations between A components fixed to .5 in full siblings, .375 in ambiguous siblings, and .25 in half siblings; correlations between C components fixed to 1.0 in all sibling pair types. Analysis controlled for youth gender and race/ethnicity (not shown).

Figure 5. Full Behavioral Genetic Model of Change in Sensation Seeking and Change in Delinquency. *Note.* Only one sibling per pair is shown. A=Additive Genetic, C=Shared Environmental, E=Non-shared Environmental. Correlations between A components fixed to .5 in full siblings, .375 in ambiguous siblings, and .25 in half siblings; correlations between C components fixed to 1.0 in all sibling pair types. Analysis controlled for youth gender and race/ethnicity (not shown).

Figure 6.

Final Trimmed Behavioral Genetic Model of Change in Sensation Seeking and Change in Delinquency, with Standardized Parameter Estimates.

Note. Only one sibling per pair is shown. A=Additive Genetic, C=Shared Environmental, E=Non-shared Environmental. Correlations between A components fixed to .5 in full siblings, .375 in ambiguous siblings, and .25 in half siblings; correlations between C components fixed to 1.0 in all sibling pair types. Analysis controlled for youth gender and race/ethnicity (not shown).

Table 1

Unstandardized Parameter Estimates from Behavioral Genetic Model of Sensation Seeking.

Parameter	Estimate (<i>SE</i>)	Heritabilities/Environmentalities
SS-Level		
Additive Genetic (A)	.58* (.16)	$h^2 = 62\%$
Shared Environmental (C)	.00 (.01)	$c^2 = 0\%$
Non-Shared Environmental (E)	.45* (.19)	$e^2 = 38\%$
SS-Change[†]		
Additive Genetic (A)	.24* (.06)	$h^2 = 83\%$
Shared Environmental (C)	.00 (.03)	$c^2 = 0\%$
Non-Shared Environmental (E)	.11 (.12)	$e^2 = 17\%$
SS-Level → SS-Change	-.30* (.10)	

[†] Genetic and environmental influences on SS-Change are unique of genetic and environmental variance shared with SS-Level.

* Parameter significant at $P < .05$.

Table 2

Unstandardized Parameter Estimates from Full Behavioral Genetic Model of Sensation Seeking and Delinquency.

Genetic and Environmental Influences [†]			
	A	C	E
SS-Level	.62* (.13)	[0]	.43* (.19)
SS-Change	.25* (.06)	[0]	.12 (.10)
DEL-Level	.26 (.25)	.24* (.11)	.00 (.01)
DEL-Change	.08 (.31)	.00 (.01)	.27* (.08)
Genetic and Environmental Cross-Paths			
	A	C	E
SS-Level → DEL-Level	.44* (.13)	[0]	-.22 (.13)
SS-Level → DEL-Change	-.10 (.18)	[0]	.14 (.18)
SS-Change → DEL-Change	.14* (.07)	[0]	.09 (.15)

Note. SEs are in parentheses.

[†] Genetic and environmental influences on SS-Change, DEL-Level, and DEL-Change are unique of genetic and environmental variance shared with predictor variables.

* Parameter significant at $P < .05$.