Developmental Science

Developmental Science 15:1 (2012), pp 150–163 DOI: 10.1111/j.1467-7687.2011.01115.x

PAPER

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 7675 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 2562 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.

Introduction

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, Veenstra, Lindenberg, van Roon, Verhulst, Ormel & Riese, 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, 2006b). 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, Shulman, Steinberg, Claus, Banich, Graham & Woolard, 2010; Harden & Tucker-Drob, 2011; Lynne-Landsman, Graber, Nichols & Botvin, 2011; MacPherson, Magidson, Reynolds, Kahler & Lejuez, 2010; Romer & Hennessy, 2007; Steinberg, Albert, Cauffman, Banich, Graham & Woolard, 2008). Interestingly, increases in behavioral indices of sensation and novelty seeking are also evident in rodents (Adriani, Chairotti & Laviola, 1998; Laviola, Macri, Morley-Fletcher & Adriani, 2003; Stansfield & Kirstein, 2006), suggesting that primitive biological processes play an important role in this

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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.*, 2008; 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, Jazbec, McClure, Monk, Blair, Leibenluft & Pine, 2005; Galvan, Hare, Parra, Penn, Voss, Glover & Casey, 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, Johnson, Thomae, Luo, Simon, Zhou & Walker, 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, Krueger, Dick, Saccone, Grucza, Agrawal *et al.*, 2010; Lang, Bajbouj, Sander & Gallinat, 2007; Laucht, Becker & Schmidt, 2006; Ray, Bryan, MacKillop, McGeary, Hesterberg & Hutchison, 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, van den Bree, Gupman, Newlin, Glantz & Pickens, 2001; Mustanski, Viken, Kaprio & Rose, 2003), with minimal sex differences in heritability (Koopmans *et al.*, 1995; 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, 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; Galvan, Hare, Voss, Glover & Casey, 2007; Hare, Tottenham, Galvan, Voss, Glover & Casey, 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 *et al.*, 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 7675 youth, including 2562 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 US. A complex survey design was used to select a nationally representative sample of 3000 households containing 6111 youth, plus an additional oversample of 3652 African-American and Hispanic youth, aged 14–21 years as of 31 December 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 6283 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 $N = 7675$ youth who reported on their delinquent behavior at least once between the ages of 10 and 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, Van Hulle, Waldman, Rodgers, Harden, Rathouz & Lahey, 2008; Mendle, Harden, Turkheimer, van Hulle, D'Onofrio, Brooks-Gunn, Rodgers, Emery & Lahey, 2009; Van Hulle, Rodgers, D'Onofrio, Waldman & Lahey, 2007). For behavioral genetic analyses, the current study makes use of a sibling pairs subsample comprising $N = 2562$ sibling pairs (778 half-siblings, 267 ambiguous siblings, and 1517 full siblings), where one sibling pair was randomly selected per nuclear family.

Measures

Maternal characteristics

Socioeconomic status (SES) was measured using self-reported total family income (median = $22,500 per

[1] All 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.

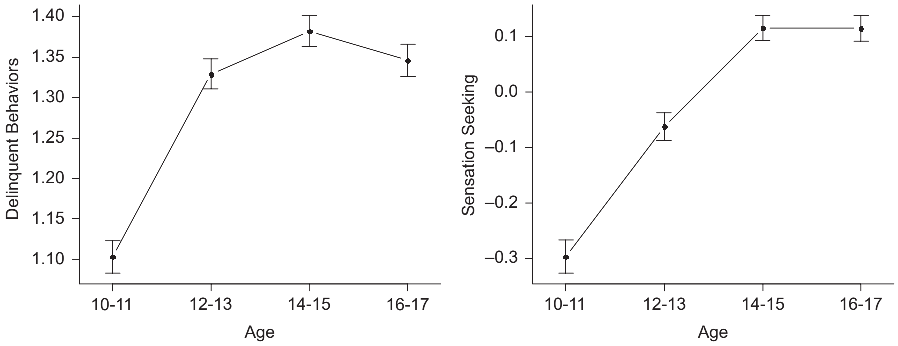

Figure 1 *Mean levels of sensation seeking and delinquent behavior by age. Note: Bands represent SEs. Sensation seeking on standardized (z-score) scale. Delinquency measured as symptom count (0–7).*

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 and Tucker-Drob (2011), the pattern of correlations between sensation seeking and Big Five personality traits in the full CNLSY sample was 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 six 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 six SRD items. Because of budgetary constraints, the SRD items were dropped from the YA assessment (but not the 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, D'Onofrio, van Hulle, Turkheimer, Rodgers, Waldman & Lahey, 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 alpha for the six 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

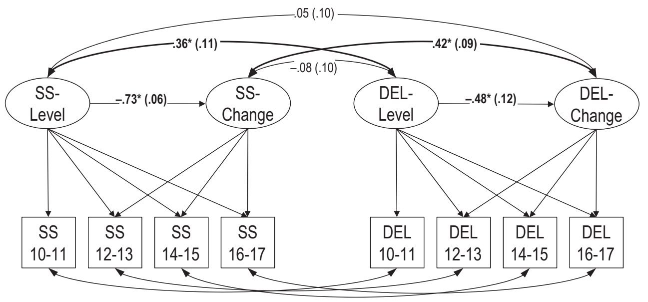

Figure 2 *Latent growth curve model of delinquency and sensation seeking, with standardized parameter estimates. Note: Intercept factor loadings fixed to 1.0 across all ages; slope factor loadings fixed to 1 at age 12–13 and estimated freely from data for subsequent ages. SEs are in parentheses. Analysis controlled for youth gender and race/ethnicity (not shown). Parameters with * are significant at* p *< .05.*

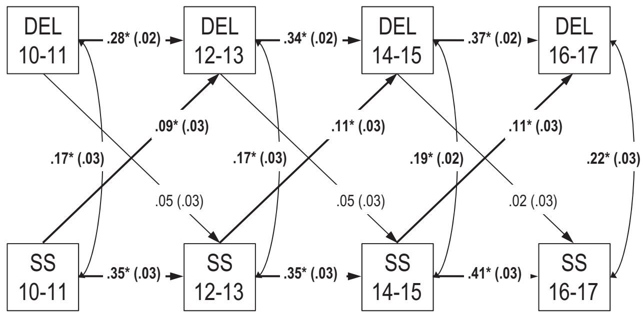

Figure 3 *Cross-lagged analysis of delinquency and sensation seeking, with standardized parameter estimates. Note: Parameters with * are significant at* p *< .05. Analysis controlled for youth gender and race/ethnicity (not shown).*

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 & Nesselroade, 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

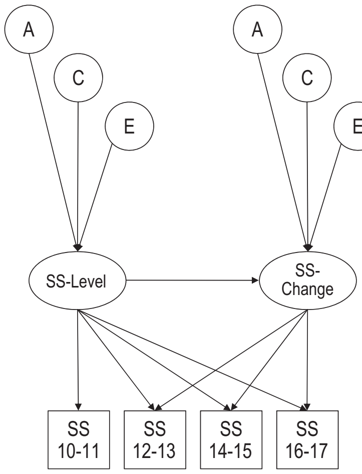

Figure 4 *Behavioral genetic model of change in sensation seeking. Note: Only one sibling per pair is shown. A = Additive Genetic, C = Shared Environmental, E = Non-shared Environmental. Correlations between A components fixed to .5 in full siblings, .375 in ambiguous siblings, and .25 in half siblings; correlations between C components fixed to 1.0 in all sibling pair types. Analysis controlled for youth gender and race/ethnicity (not shown).*

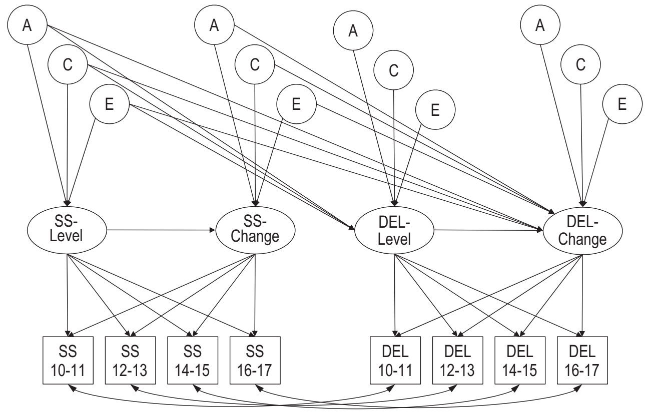

Figure 5 *Full behavioral genetic model of change in sensation seeking and change in delinquency. Note: Only one sibling per pair is shown. A = Additive Genetic, C = Shared Environmental, E = Non-shared Environmental. Correlations between A components fixed to .5 in full siblings, .375 in ambiguous siblings, 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).*

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 is fixed according to genetic theory (.5 for full siblings, .375 for ambiguous siblings, .25 for half siblings); the correlation between the *C* factors was 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 (Muthén & Muthén, 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 and Bentler (1999), cut-off values for acceptable model fit were RMSEA ≤ .06, CFI ≥ .95, and SRMR ≤ .08. The only index of absolute model fit available for the behavioral genetic models was SRMR; relative fit of behavioral genetic models was 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 (Var = .684, *SE* = .122, $p < .01$), and change in sensation seeking (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 (rs .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 (CFI = .97, RMSEA = .03, 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 (rs = .17–.22, ps < .01). Moreover, sensation seeking significantly predicted subsequent involvement in delinquent behavior two years later across ages (βs = .09–.11, ps < .01). In contrast, the associations between delinquent behavior and subsequent levels of sensation seeking were more modest (βs = .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 an 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 (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 $[.24^2/(.24^2 + .00^2 + .11^2)]$. 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

Table 1 *Unstandardized parameter estimates from behavioral genetic model of sensation seeking*

Parameter	Estimate (*SE*)	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: *SE*s 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 $p < .05$.

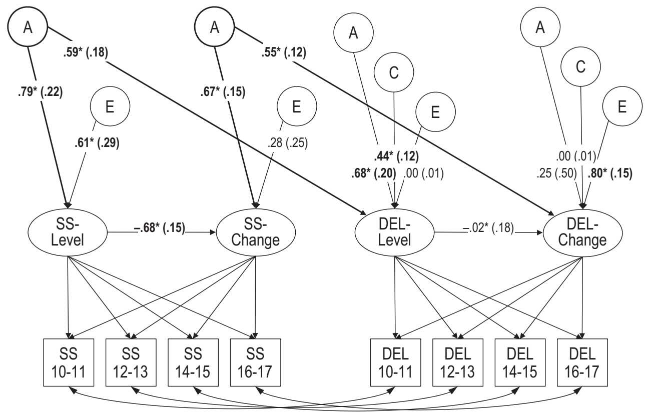

Figure 6 *Final trimmed behavioral genetic model of change in sensation seeking and change in delinquency, with standardized parameter estimates. Note: Only one sibling per pair is shown. A = Additive Genetic, C = Shared Environmental, E = Non-shared Environmental. Correlations between A components fixed 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).*

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% was accounted for by genes influencing initial levels of sensation seeking.

Of the variance in change in delinquency (unique to 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 to 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*., 2008;

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, 2001; Roberts *et al.*, 2006b), 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, Donnellan, Blonigen, Krueger, McGue, Iacono & Burt, 2011). These studies – plus a handful of others not using latent growth curve modeling approaches (e.g. Blonigen, Carlson, Hicks, Krueger & Iacono, 2008; Kandler, Bleidorn, Reimann, Spinath, Thiel & Angleitner, 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, Logan, Leukefeld, Milich, Omar & Clayton, 2001; Martin, Kelly, Rayens, Brogli, Brenzel, Smith & Omar, 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.

In addition, 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, Slutske, Turkheimer, Emery, Harden, Heath, Madden & Martin, 2007; Thornberry, Freeman-Gallant, Lizotte, Krohn & Smith, 2003). Interestingly, previous research has found that 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 adolescence. 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 7000 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 and 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 it 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, Loeber, Quay, Applegate, Shaffer, Waldman, Hart, McBurnett, Frick, Jensen, Dulcan, Canino & Bird, 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 and 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.

Acknowledgements

Paige Harden and Elliot Tucker-Drob are Faculty Research Associates of the Population Research Center at the University of Texas at Austin, which is supported by a center grant from the National Institute of Child Health and Human Development (5-R24-HD042849). Research by Patrick Quinn is supported by National Institute on Alcohol Abuse and Alcoholism Grant 5T32-AA07471 and by the Waggoner Center for Alcohol and Addiction Research. The NLSY kinship links were provided by Dr Joe Rodgers, Department of Psychology, University of Oklahoma. The creation of the links occurred through the support of research grants from NIH/NICHD on which Dr Rodgers was PI. Others who wish to use the NLSY kinship links can obtain copies of these kinship links by writing to Dr Rodgers at jrodgers@ou.edu.

References

Achenbach, T.M. (1991). *Manual for the Child Behavior Checklist and 1991 Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.

Adriani, W., Chiarotti, F., & Laviola, G. (1998). Elevated novelty seeking and peculiar d-amphetamine sensitization in periadolescent mice compared with adult mice. *Behavioral Neuroscience*, **112**, 1152–1166.

Asparouhov, T., & Muthén, B. (2006). Multilevel modeling of complex survey data. *Proceedings of the American Statistical Association*. Seattle, WA: American Statistical Association.

Bentler, P.M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, **107**, 238–246.

Bentler, P.M. (1995). *EQS structural equations program manual*. Encino, CA: Mulivariate Software.

Bergen, S.E., Gardner, C.O., & Kendler, K.S. (2007). Age-related change in heritability of behavioral phenotypes over adolescence and young adulthood: a meta-analysis. *Twin Research and Human Genetics*, **10**, 423–433.

Blakemore, S.J., Burnett, S., & Dahl, R. (2010). The role of puberty in the developing adolescent brain. *Human Brain Mapping*, **31**, 926–933.

Bleidorn, W., Kandler, C., Reimann, R., Angleitner, A., & Spinath, F.M. (2009). Patterns and sources of adult personality development: growth curve analyses of the NEO PI-R scales in a longitudinal twin study. *Journal of Personality and Social Psychology*, **97**, 142–155.

Blonigen, D.M., Carlson, M.D., Hicks, B.M., Krueger, R.F., & Iacono, W.G. (2008). Stability and change in personality traits from late adolescence to early adulthood: a longitudinal twin study. *Journal of Personality*, **76**, 229–266.

Burt, S.A. (2009). Are there meaningful etiological differences within antisocial behavior? *Clinical Psychology Review*, **29**, 163–178.

Burt, S.A., & Neiderhiser, J.M. (2009). Aggressive versus nonaggressive antisocial behavior: distinctive etiological moderation by age. *Developmental Psychology*, **45**, 1164–1176.

Button, T.M.M., Scourfield, J., Martin, N., Purcell, S., & McGuffin, P. (2005). Family dysfunction interacts with genes in the causation of antisocial symptoms. *Behavior Genetics*, **35**, 115–120.

Casey, B.J., Getz, S., & Galvan, A. (2008). The adolescent brain. *Developmental Review*, **28**, 62–77.

Casey, B.J., Jones, R.M., & Somerville, L.H. (2011). Braking and accelerating of the adolescent brain. *Journal of Research on Adolescence*, **21**, 21–33.

Caspi, A., Roberts, B.W., & Shiner, R.L. (2005). Personality development: stability and change. *Annual Review of Psychology*, **56**, 17.1–17.32.

Cauffman, E., Shulman, E.P., Steinberg, L., Claus, E., Banich, M.T., Graham, S., & Woolard, J. (2010). Age differences in affective decision making as indexed by performance on the Iowa Gambling Task. *Developmental Psychology*, **46**, 193–207.

Chase-Lansdale, P.L., Mott, F.L., Brooks-Gunn, J., & Phillips, D.A. (1991). Children of the National Longitudinal Survey of Youth: a unique research opportunity. *Developmental Psychology*, **27**, 918–931.

Cooper, M.L., Wood, P.K., Orcutt, H.K., & Albino, A. (2003). Personality and the predisposition to engage in risky or problem behaviors in adolescence. *Journal of Personality and Social Psychology*, **84**, 390–410.

Costa, P.T. Jr., & McCrae, R.R. (2006). Age changes in personality and their origins: comment on Roberts, Walton, and Viechtbauer (2006). *Psychological Bulletin*, **132**, 26–28.

Crawford, A.M., Pentz, M.A., Chou, C.-P., Li, C., & Dwyer, J.H. (2003). Parallel developmental trajectories of sensation seeking and regular substance use in adolescents. *Psychology of Addictive Behaviors*, **17**, 179–192.

Curran, P.J. (2000). A latent curve framework for studying developmental trajectories of adolescent substance use. In J. Rose, L. Chassin, C. Presson, & J. Sherman (Eds.), *Multivariate applications in substance use research* (pp. 1–42). Hillsdale, NJ: Erlbaum.

Derringer, J., Krueger, R.F., Dick, D.M., Saccone, S., Grucza, R.A., Agrawal, A., *et al.* (2010). Predicting sensation seeking from dopamine genes: a candidate system approach. *Psychological Science*, **21**, 1282–1290.

Dodge, K.A. (2003). Investing in the prevention of youth violence. *International Society for the Study of Behavioral Development Newsletter*, **2**, 8–10.

D'Onofrio, B.M., Slutske, W.S., Turkheimer, E., Emery, R.E., Harden, K.P., Heath, A.C., Madden, P.A.F., & Martin, N.G. (2007). Intergenerational transmission of childhood conduct problems: a children of twins study. *Archives of General Psychiatry*, **64**, 820–829.

D'Onofrio, B.M., Van Hulle, C.A., Waldman, I.D., Rodgers, J.L., Harden, K.P., Rathouz, P.J., & Lahey, B.B. (2008). Smoking during pregnancy and offspring externalizing problems: an exploration of genetic and environmental confounds. *Development and Psychopathology*, **20**, 139–164.

Eley, T.C., Lichtenstein, P., & Moffitt, T.E. (2003). A longitudinal behavioral genetic analysis of the etiology of aggressive and nonaggressive antisocial behavior. *Development and Psychopathology*, **15**, 383–402.

Elliott, D.S., & Huizinga, D. (1983). Social class and delinquent behavior in a national youth panel. *Criminology: An Interdisciplinary Journal*, **21**, 149–177.

Ernst, M., Jazbec, S., McClure, E.B., Monk, C.S., Blair, R.J.R., Leibenluft, E., & Pine, D.S. (2005). Amygdala and nucleus accumbens activation in response to receipt and omission of gains in adults and adolescents. *NeuroImage*, **25**, 1279–1291.

Ernst, M., Pine, D., & Hardin, M. (2006). Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine*, **36**, 299–312.

Eysenck, H.J. (1983). A biometrical-genetical analysis of impulsive and sensation seeking behavior. In M. Zuckerman (Ed.), *Biological bases of sensation seeking, impulsivity, and anxiety* (pp. 1–27). Hillsdale, NJ: Erlbaum.

Fareri, D.S., Martin, L.N., & Delgado, M.R. (2008). Reward-related processing in the human brain: developmental considerations. *Development and Psychopathology*, **20**, 1191–1211.

Feinberg, M.E., Button, T.M.M., Neiderhiser, J.M., Reiss, D., & Hetherington, E.M. (2007). Parenting and adolescent antisocial behavior and depression. *Archives of General Psychiatry*, **64**, 457–465.

Forbes, E.E., & Dahl, R.E. (2010). Pubertal development and behavior: hormonal activation of social and motivational tendencies. *Brain and Cognition*, **72**, 66–72.

Frick, P.J., Cornell, A.H., Barry, C.T., Bodin, S.D., & Dane, H.A. (2003). Callous-unemotional traits and conduct problems in the prediction of conduct problem severity, aggression, and self-report of delinquency. *Journal of Abnormal Child Psychology*, **31**, 457–470.

Fulker, D.W., Eysenck, S.B.G., & Zuckerman, M. (1980). A genetic and environmental analysis of sensation seeking. *Journal of Research on Personality*, **14**, 261–281.

Galvan, A., Hare, T.A., Parra, C.E., Penn, J., Voss, H., Glover, G., & Casey, B.J. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience*, **26**, 6885–6892.

Galvan, A., Hare, T., Voss, H., Glover, G., & Casey, B.J. (2007). Risk-taking and the adolescent brain: who is at risk? *Developmental Science*, **10**, F8–F14.

Ge, X., Natsuaki, M.N., Neiderhiser, J.M., & Reiss, D. (2007). Genetic and environmental influences on pubertal timing: results from two national sibling studies. *Journal of Research on Adolescence*, **17**, 767–788.

Gottesman, I.I., & Gould, T.D. (2003). The endophenotype concept in psychiatry: etymology and strategic intentions. *American Journal of Psychiatry*, **160**, 636–645.

Harden, K.P., D'Onofrio, B.M., van Hulle, C., Turkheimer, E., Rodgers, J.L., Waldman, I.D., & Lahey, B.B. (2009). Population density and youth antisocial behavior. *Journal of Child Psychology and Psychiatry*, **50**, 999–1008.

Harden, K.P., & Tucker-Drob, E.M. (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.

Hare, T.A., Tottenham, N., Galvan, A., Voss, H.U., Glover, G.H., & Casey, B.J. (2008). Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. *Biological Psychiatry*, **63**, 927–934.

Hicks, B.M., South, S.C., DiRago, A.C., Iacono, W.G., & McGue, M. (2009). Environmental adversity and increasing genetic risk for externalizing disorders. *Archives of General Psychiatry*, **66**, 640–648.

Hopwood, C.J., Donnellan, M.B., Blonigen, D.M., Krueger, R.F., McGue, M., Iacono, W.G., & Burt, S.A. (2011). Genetic and environmental influences on personality trait stability and growth during the transition to adulthood: a three-wave longitudinal study. *Journal of Personality and Social Psychology*, **100**, 545–556.

Horvath, P., & Zuckerman, M. (1993). Sensation seeking, risk appraisal, and risky behavior. *Personality and Individual Differences*, **14**, 41–52.

Hu, L., & Bentler, P.M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Structural Equation Modeling*, **6**, 1–55.

Hur, Y.M., & Bouchard, T.J. (1997). The genetic correlation between impulsivity and sensation seeking traits. *Behavior Genetics*, **27**, 455–463.

Jessor, R., & Jessor, S.L. (1977). *Problem behavior and psychosocial development: A longitudinal study of youth*. New York: Academic Press.

Kalil, A., & Ryan, R.M. (2010). Mothers' economic conditions and sources of support in fragile families. *Future of Children*, **20**, 39–61.

Kandler, C., Bleidorn, W., Reimann, R., Spinath, F.M., Thiel, W., & Angleitner, A. (2010). Sources of cumulative continuity in personality: a longitudinal multiple-rater twin study. *Journal of Personality and Social Psychology*, **98**, 995–1008.

Koopmans, J.R., Boomsma, D.I., Heath, A.C., & van Doornen, L.J.P. (1995). A multivariate genetic analysis of sensation seeking. *Behavior Genetics*, **25**, 349–356.

Kuhn, C., Johnson, M., Thomae, A., Luo, B., Simon, S.A., Zhou, G., & Walker, Q.D. (2010). The emergence of gonadal hormone influences on dopaminergic function during puberty. *Hormones and Behavior*, **58**, 122–137.

Lahey, B.B., Loeber, R., Quay, H.C., Applegate, B., Shaffer, D., Waldman, I., Hart, E.L., McBurnett, K., Frick, P.J., Jensen, P.S., Dulcan, M.K, Canino, G., & Bird, H.R. (1998). Validity of DSM-IV subtypes of conduct disorder based on age of onset. *Journal of the American Academy of Child and Adolescent Psychiatry*, **37**, 435–442.

Lahey, B.B., Loeber, R., Quay, H.C., Frick, P.J., & Grimm, J. (1992). Oppositional defiant disorder and conduct disorders: issues to be resolved for the DSM-IV. *Journal of the American Academy of Child and Adolescent Psychiatry*, **31**, 539–546.

Lang, U.E., Bajbouj, M., Sander, R., & Gallinat, J. (2007). Gender-dependent association of the functional catechol-O-methyltransferase Val158Met genotype with sensation seeking personality trait. *Neuropsychopharmacology*, **32**, 1950–1955.

Laucht, M., Becker, K., & Schmidt, M.H. (2006). Visual exploratory behavior in infancy and novelty seeking in adolescence: two developmentally specific phenotypes of DRD4? *Journal of Child Psychology and Psychiatry*, **47**, 1143–1151.

Laviola, G., Macri, S., Morley-Fletcher, S., & Adriani, W. (2003). Risk-taking behavior in adolescent mice: psychobiological determinants and early epigenetic influence. *Neuroscience and Biobehavioral Reviews*, **27**, 19–31.

Lynne-Landsman, S.D., Graber, J.A., Nichols, T.R., & Botvin, G.J. (2011). Is sensation seeking a stable trait or does it change over time? *Journal of Youth and Adolescence*, **40**, 48–58.

McArdle, J.J., & Nesselroade, J.R. (2003). Growth curve analysis in contemporary psychological research. In J. Schinka & W. Velicer (Eds.), *Comprehensive handbook of psychology, Volume II: Research methods in psychology* (pp. 447–480). New York: Pergamon Press.

McGue, M., Bacon, S., & Lykken, D.T. (1993). Personality stability and change in early adulthood: a behavioral genetic analysis. *Developmental Psychology*, **29**, 96–109.

MacPherson, L., Magidson, J.F., Reynolds, E.K., Kahler, C.W., & Lejuez, C.W. (2010). Changes in sensation seeking and risk-taking propensity predict increases in alcohol use among early adolescents. *Alcoholism: Clinical and Experimental Research*, **34**, 1400–1408.

Martin, C.A., Kelly, T., Rayens, M., Brogli, B., Brenzel, A., Smith, W., & Omar, H.A. (2002). Sensation seeking, puberty and nicotine, alcohol and marijuana use in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, **41**, 1495–1502.

Martin, C.A., Logan, T.K., Leukefeld, C., Milich, R., Omar, H., & Clayton, R. (2001). Adolescent and young adult substance use: association with sensation seeking, self-esteem and retrospective report of early pubertal onset. A preliminary examination. *International Journal of Adolescent Medicine and Health*, **13**, 211–219.

Mendle, J., Harden, K.P., Brooks-Gunn, J., & Graber, J.A. (2010). Development's tortoise and hare: pubertal timing, pubertal tempo, and depressive symptoms in boys and girls. *Developmental Psychology*, **46**, 1341–1353.

Mendle, J., Harden, K.P., Turkheimer, E., van Hulle, C., D'Onofrio, B.M., Brooks-Gunn, J., Rodgers, J.L., Emery, R.E., & Lahey, B.B. (2009). Associations between father absence and age of first sexual intercourse. *Child Development*, **80**, 1463–1480.

Miles, D.R., van den Bree, M.B.M., Gupman, A.E., Newlin, D.B., Glantz, M.D., & Pickens, R.W. (2001). A twin study of sensation seeking, risk taking behavior, and marijuana use. *Drug and Alcohol Dependence*, **62**, 57–68.

Moffitt, T.E. (1993). Life-course-persistent and adolescent-limited antisocial behavior: a developmental taxonomy. *Psychological Review*, **100**, 674–701.

Moffitt, T.E. (2005). The new look of behavioral genetics in developmental psychopathology: gene–environment interplay in antisocial behaviors. *Psychological Bulletin*, **131**, 533–554.

Moffitt, T.E., Caspi, A., Dickson, N., Silva, P.A., & Stanton, W. (1996). Childhood-onset versus adolescent-onset antisocial conduct in males: natural history from age 3 to 18. *Development and Psychopathology*, **8**, 399–424.

Mustanski, B.M., Viken, R.J., Kaprio, J., & Rose, R.J. (2003). Genetic influences on the association between personality risk factors and alcohol use and abuse. *Journal of Abnormal Psychology*, **112**, 282–289.

Muthén, L.K., & Muthén, B.O. (1998–2011). *Mplus user's guide* (6th edn.). Los Angeles, CA: Muthén & Muthén.

Newcomb, M.D., & McGee, L. (1991). Influence of sensation seeking on general deviance and specific problem behaviors from adolescence to young adulthood. *Journal of Personality and Social Psychology*, **61**, 614–628.

Powell, J.E., & Zeitsch, B.P. (2011). Predicting sensation seeking from dopamine genes: use and misuse of genetic prediction. *Psychological Science*, **22**, 413–415.

Quinn, P.D., & Harden, K.P. (in press). Differential changes in impulsivity and sensation seeking and the escalation of substance use from adolescence to early adulthood. *Development and Psychopathology.*

Raftery, A.E. (1993). Bayesian model selection in structural equation models. In K.A. Bollen & J.S. Long (Eds.), *Testing structural equation models* (pp. 163–180). Newbury Park, CA: Sage.

Raine, A., Reynolds, C., Venables, P.H., Mednick, S.A., & Farrington, D.P. (1998). Fearlessness, stimulation seeking and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. *Archives of General Psychiatry*, **55**, 745–751.

Ray, L.A., Bryan, A., MacKillop, J., McGeary, J., Hesterberg, K., & Hutchison, K.E. (2009). The dopamine D4 receptor (DRD4) gene exon III polymorphism, problematic alcohol use and novelty seeking: direct and mediated genetic effects. *Addiction Biology*, **14**, 238–244.

Rhee, S.H., & Waldman, I.D. (2002). Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. *Psychological Bulletin*, **128**, 490–529.

Roberts, B.W., Caspi, A., & Moffitt, T.E. (2001). The kids are alright: growth and stability in personality development from adolescence to adulthood. *Journal of Personality and Social Psychology*, **81**, 670–683.

Roberts, B.W., & Mroczek, D. (2008). Personality trait change in adulthood. *Current Directions in Psychological Science*, **17**, 31–35.

Roberts, B.W., Walton, K.E., & Viechtbauer, W. (2006a). Patterns of mean-level change in personality traits across the life course: a meta-analysis of longitudinal studies. *Psychological Bulletin*, **132**, 1–25.

Roberts, B.W., Walton, K.E., & Viechtbauer, W. (2006b). Personality traits change in adulthood: reply to Costa and McCrae (2006). *Psychological Bulletin*, **132**, 29–32.

Rodgers, J.L., Buster, M., & Rowe, D.C. (2001). Genetic and environmental influences on delinquency: DF analysis of NLSY kinship data. *Journal of Quantitative Criminology*, **2**, 145–168.

Rodgers, J.L., Johnson, A.B., & Bard, D.E. (2005). NLSY-Children/Young Adult (1986–2000) kinship linking algorithm. Unpublished manuscript.

Romer, D., & Hennessy, M. (2007). A biosocial-affect model of adolescent sensation seeking: the role of affect evaluation and peer-group influence in adolescent drug use. *Prevention Science*, **8**, 89–101.

Schafer, J.L., & Graham, J.W. (2002). Missing data: our view of the state of the art. *Psychological Methods*, **7**, 147–177.

Sijtsema, J.J., Veenstra, R., Lindenberg, S., van Roon, A.M., Verhulst, F.C., Ormel, J., & Riese, H. (2010). Mediation of sensation seeking and behavioral inhibition on the relationship between heart rate and antisocial behavior: the TRAILS study. *Journal of the American Academy of Child and Adolescent Psychiatry*, **49**, 493–502.

Somerville, L.H., Jones, R.M., & Casey, B.J. (2010). A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain and Cognition*, **72**, 124–133.

Stanger, C., Achenbach, T.M., & Verhulst, F.C. (1997). Accelerated longitudinal comparisons of aggressive versus delinquent syndromes. *Development and Psychopathology*, **9**, 43–58.

Stansfield, K.H., & Kirstein, C.L. (2006). Effects of novelty on behavior in the adolescent and adult rat. *Developmental Psychobiology*, **48**, 10–15.

Steiger, H. (1990). Structural model evaluation and modification. *Multivariate Behavioral Research*, **25**, 173–180.

Steinberg, L. (2004). Risk-taking in adolescence: what changes, and why? *Annals of the New York Academy of Sciences*, **1021**, 51–58.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, **28**, 78–106.

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**, 1764–1777.

Stoel, R.D., De Geus, E.J.C., & Boomsma, D.I. (2006). Genetic analysis of sensation seeking with an extended twin design. *Behavior Genetics*, **36**, 229–237.

Thornberry, T.P., Freeman-Gallant, A., Lizotte, A.J., Krohn, M.D., & Smith, C.A. (2003). Linked lives: the intergenerational transmission of antisocial behavior. *Journal of Abnormal Child Psychology*, **31**, 171–184.

Tremblay, R.E. (2003). Why socialization fails: the case of chronic physical aggression. In B.B. Lahey, T.E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 182–224). New York: Guilford Press.

Van Hulle, C.A., Rodgers, J.L., D'Onofrio, B.M., Waldman, I.D., & Lahey, B.B. (2007). Sex differences in the causes of self-reported adolescent delinquency. *Journal of Abnormal Psychology*, **116**, 236–248.

Verhulst, F.C., Koot, H.M., & Berden, G.F.M.G. (1990). Four-year follow-up of an epidemiological sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, **29**, 440–448.

White, H.R., Labouvie, E.W., & Bates, M.E. (1985). The relationship between sensation seeking and delinquency: a longitudinal analysis. *Journal of Research in Crime and Delinquency*, **22**, 197–211.

Zuckerman, M. (2007). Sensation seeking and crime, antisocial behavior, and delinquency. In M. Zuckerman, *Sensation seeking and risky behavior*. Washington, DC: American Psychological Association.

Zuckerman, M., Eysenck, S.B.G., & Eysenck, H.J. (1978). Sensation seeking in England and America: cross-cultural, age and sex comparisons. *Journal of Consulting and Clinical Psychology*, **46**, 139–149.

Received: 31 March 2011
Accepted: 28 August 2011