THE DISSERTATION COMMITTEE FOR MARIE DANIELLE CARLSON CERTIFIES THAT THIS IS THE APPROVED VERSION OF THE FOLLOWING DISSERTATION:

GENE-ENVIRONMENT INTERPLAY IN ADOLESCENT SEXUAL HEALTH AND EXTERNALIZING BEHAVIORS: A CRITICAL EXAMINATION OF CONTEXT

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Gene-Environment Interplay in Adolescent Sexual Health and Externalizing Behaviors: A Critical Examination of Context

BY

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Gene-Environment Interplay in Adolescent Sexual Health and Externalizing Behaviors: A Critical Examination of Context

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The University of Texas at Austin, 2018

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The correlates of adolescent sexual behavior and externalizing spectrum behaviors have been documented across a range of disciplines and include both genetic and environmental factors. Over the last 15 years, the dynamic interplay between genes and the environment has garnered increasing interest among researchers who study risk-taking behavior. In spite of this popularity, the racial and socioeconomic composition of much behavioral genetic research to date has been largely homogenous, based on middle- to upper-middle-class Caucasian samples. Consequently, the universality of many findings remains unclear, and the roles of key contextual factors related to race and social privilege remain largely unexamined. To address these gaps, my dissertation will include three empirical studies, leveraging a range of biometric and structural equation modeling techniques to address three research questions. Each question builds in succession toward the overarching objective to better understand the contextual roles of racial stratification, social class, and chronic stress and trauma in relation to individual differences in adolescent sexual health and externalizing spectrum behaviors.

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Introduction

Many forms of disadvantage have been linked to higher externalizing behaviors and earlier onset of sexual activity (e.g., Belsky et al., 2010; Coley & Chase-Lansdale, 1998). In contrast, contextual advantage and privilege have been linked to the opposite pattern. Further traction in the etiology of individual differences in adolescent sexual activity and externalizing behavior may be gained by reference points of context. The epidemiological and sociological literatures have historically emphasized the importance of context in relation to behavior and have documented several predictors of early sexual behavior including low socioeconomic status, racial/ethnic minority status, childhood adversity, substance abuse, and externalizing more broadly (Shoefield et al., 2008).

In contrast, the behavioral genetic literature has traditionally emphasized the role of heritable variation in behavior. The heritability statistic provides an index of total phenotypic variance associated with genetic variance, and has historically been the dominant focus of this line of research. What is less appreciated is that the derivation of this statistic is fundamentally dependent upon the average contextual variation of the sample. Formally defined, heritability refers to "the proportion of the total phenotypic variance that is associated with genetic variance in a specific sample with a specific genetic composition and environmental context" (Vitzthum, 2003, p. 541). Thus, environmental context is an integral, if under-recognized, component in calculating heritability and key for understanding its variation.

This is a critical point of consideration for the present series of studies, in particular, because heritability estimates have traditionally been derived in Caucasian middle-class samples, most prominently from the United States, Australia, New Zealand, Denmark, Finland, and Sweden. Notably, these latter five countries are highly socially, culturally, and racially

homogenous, and most provide universal healthcare and postsecondary education. Consequently, explanatory power beyond the Caucasian middle class is limited.

Over the last several decades, researchers have advanced explanatory theories to account for the association between contextual adversity and adolescent health-risking social behaviors such as early sexual behavior and externalizing. Traditionally, some of these perspectives have focused more heavily on the environmental antecedents of adolescent risk behavior, while others have placed a larger focus on the role of common genetic vulnerability. Although both genetic and environmental risk factors have been linked to adolescent health-risking social behaviors, examining these etiological components in isolation excludes a more nuanced, ecologically valid understanding of their dynamic interplay—that is, whether the relative contributions of genetic and environmental factors underlying variation in an outcome might differ depending on social context.

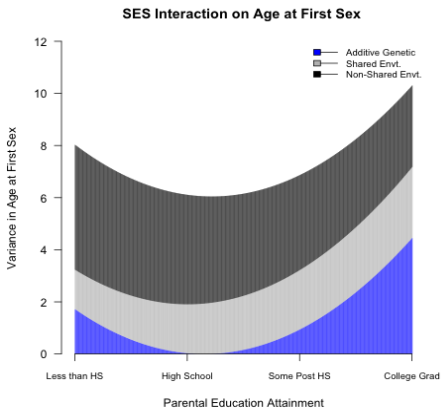
Fortunately, in recent years, technical advancements in modeling have enabled more nuanced examination of gene-environment interplay (Purcell, 2002). Consequently, over the last 10–15 years, there has been a proliferation of behavioral genetic studies examining the dynamic interplay between genes and measured environmental contexts. In spite of this popularity, however, the racial and socioeconomic composition of much behavioral genetic research has remained largely uniform, based on environmentally homogenous, middle-class, Caucasian samples. Consequently, the universality of many findings remains unclear, and the role of key contextual factors related to racism and social privilege are not well understood.

To address these gaps, my dissertation includes three empirical studies, leveraging a series of biometric and structural equation modeling techniques to address three research questions. Each question builds in succession toward the overarching objective to better

understand the contextual roles of racial stratification, social class, and chronic stress and trauma in relation to individual differences in adolescent sexual health and externalizing spectrum behaviors.

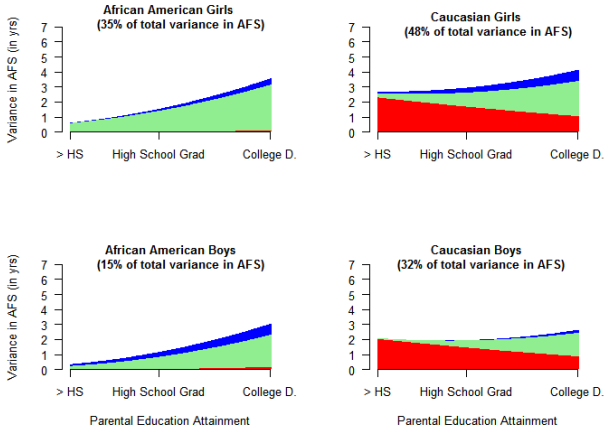
Study 1: Do genetic and environmental influences on timing of first sexual intercourse vary as a function of environmental context?

Youth who experience adverse environments in early life initiate sexual activity at a younger age, on average, than those from more advantaged circumstances. Evolutionary theorists have posited that ecological stress precipitates earlier reproductive and sexual onset, but it is unclear how stressful environments interact with genetic influences on age at first sex. Using a sample of 1,244 pairs of twins and non-twin full siblings from the National Longitudinal Study of Adolescent Health, Study 1 tested for gene-by-environment interactions (G×E) on age at first sex (AFS). Multivariate interaction models indicated that genetic influences on AFS were suppressed among low-socioeconomic-status (SES) and ethnic-minority (African American and Hispanic) youth compared with higher SES and ethnic-majority (non-Hispanic White) youth. Father absence did not uniquely moderate genetic influences on AFS. These results suggest that the relative contributions of genetic and environmental influences in the etiology of first sexual intercourse are contingent upon context such that genetic influences are suppressed in contexts marked by social and economic disadvantage and amplified in contexts marked by social and economic privilege.



Study 2: Are predominant etiological models for understanding adolescent sexual behavior truly universal? Specifically, are patterns of association between sexual behavior and externalizing behavior seen in African American youth and youth from lower SES backgrounds similar to patterns previously observed in Caucasian youth?

Despite global reductions in teen pregnancy and STI infections over the last decade, the *gap* between sexual health outcomes as a function of race/ethnicity and social class in America remain large. Population-wide correlates of adolescent sexual activity, such as substance use and delinquency, have given rise to a conceptualization of adolescent sexual activity as a manifestation of a genetically influenced propensity to externalizing behaviors more generally. To date, however, empirical evidence for this perspective has been largely based upon Caucasian middle class samples. Indeed, despite the magnitude of these sociodemographic disparities in sexual health, little research has examined whether predominant etiological models for understanding sexual health behavior generalize beyond Caucasian American youth to include African American youth. Therefore, it is unclear whether the associations between adolescent sexual behavior and social deviance more broadly are moderated by race. Using twin data from the National Longitudinal Study of Adolescent Health, Study 2 tested whether these patterns apply to African American youth and youth from lower SES contexts. Results indicated that for all youth, higher externalizing (a general factor comprising involvement in alcohol use, drug use, nonviolent crime, and fighting) is correlated with earlier first sex and more sexual partners. These associations, however, were



appreciably attenuated among African American youth, as were mean levels of externalizing. For Caucasian youth, the link between age at first sex and externalizing was attributable to both common genetic influences and shared environmental influences, whereas for African American youth the link was exclusively attributable to shared environmental influences. The link between number of sexual partners and externalizing was attributable to common genetic influences for all adolescent groups except African American girls, in which the shared environment predominated. Furthermore, for Caucasian boys, externalizing accounted for 41% of the variation in number of sexual partners, whereas for African American boys, it only accounted for 20%. At this point in time, genetic predispositions toward externalizing do not appear to contribute to the etiology of sexual behavior equivalently in race/ethnic majority versus minority youth.

Study 3: To what extent does early chronic stress and trauma have a role in the etiology of externalizing spectrum behaviors and adolescent sexual behavior?

Early chronic stress (including poverty, neglect, and emotional abuse) is a risk factor for a range of adverse health and psychosocial outcomes across the life-span (Lupien, McEwen, Gunnar, & Hein, 2009; Felitti et al., 1998). During adolescence, stress is highly correlated with a variety of externalizing spectrum behaviors including fighting, delinquency, and substance abuse, as well as with sexual behaviors including earlier first sexual intercourse and more sexual partners. The goal of Study 3 was to better understand the role of chronic stress and trauma in relation to global externalizing behavior (EXT), specific rule-breaking behaviors unique from global EXT (such as alcohol use, fighting, delinquency, and illegal drug use), and adolescent sexual behavior.

Participants

Participants were drawn from the National Longitudinal Study of Adolescent Health (Add Health; Udry, 2003). Add Health data were collected in four waves between 1994 and 2008. Sampling for Add Health began with identification of all high schools in the United States that had at least 30 enrollees (*N*=26,666). Schools were stratified by geographic region, urbanicity, school size or type, and racial composition. A random sample of schools, ranging from 7th to 12th grades and 9th to 12th grades, were then collected from these strata. Seventy-nine percent of contacted schools agreed to participate, and 90,118 students completed a confidential in-school survey during the 1994–1995 academic year. From school rosters a subset of 20,745 randomly selected students, ages 11 to 21 (*M* = 16 years, 25th percentile = 14 years, 75th percentile = 17 years), were selected to complete a 90-minute in-home interview between April and December of 1995 (Wave 1 interview: 10,480 female; 10,264 male). The study followed up with a series of in-home interviews conducted in 1996 (Wave II), August 2001-2002 (Wave III), and 2007-8 (Wave IV).

Add Health deliberately oversampled adolescent sibling pairs (sibling sample described in Harris, Halpern, Smolen, & Haberstick, 2006). The breakdown of siblings by type includes 307 monozygotic (MZ) twin pairs, 452 dizygotic (DZ) twin pairs, 1,251 non-twin full biological sibling pairs, 442 half-sibling pairs, and 662 non-biological sibling pairs. Twin zygosity was determined on the basis of twin reports, frequency of being mistaken for one's co-twin, and responses to a four-item questionnaire on similarity of appearance—measures that have been cross validated with zygosity determinations based upon DNA samples and are widely used to determine zygosity in twin research (Loehlin & Nichols, 1970; Spitz et al., 1996). Jacobson and Rowe (1999) found negligible differences for sociodemographic variables (e.g., age, ethnicity, and maternal education) between Add Health sibling pairs and the full Add Health sample.

A subsample of 2,612 participants submitted saliva samples for genotyping. Zygosity status was confirmed using 11 polymorphic, unlinked short tandem repeat markers, and twin pairs were classified as MZ only if they were 100% concordant on all genotypes.

Each study provides further detail on the demographic composition of the specific subsample used.

Study 1: Early Adverse Environments and Genetic Influences on Age at First Sex: Evidence for Gene × Environment Interaction[1]

Youth who experience adverse childhood environments initiate sexual activity earlier, on average, than youth from more advantaged circumstances (e.g., Belsky, Steinberg, Houts, Halpern-Felsher, & NICHD Early Child Care Research Network (2010); Coley & Chase-Lansdale, 1998). During the last three decades, researchers have advanced several explanatory theories for this association. Most prominently, evolutionary perspectives draw from the meta-theoretical *life history* framework (Charnov, 1993; Stearns, 1992), which emphasizes a tradeoff between an organism's allocation of resources to physical growth versus the production of offspring. According to life history theory, organisms in environments with abundant and dependable resources bias the allocation of resources toward a slower, "quality-oriented" reproductive strategy characterized by delayed reproduction and greater investment in fewer offspring. In contrast, organisms in environments with scarce or unstable resources bias the allocation of resources toward a faster, "quantity-oriented" reproductive strategy characterized by early reproduction and limited parental investment in a greater number of offspring.

Although life history theory was originally developed to explain *inter*-species differences in average time to sexual maturity, Belsky, Steinberg, and Draper (1991), in their highly cited *psychosocial acceleration theory*, applied the life history framework to individual differences in human sexual development, including differences in pubertal timing, age at first sex, and age at childbearing. They posited that a principal function of the first 5–7 years of life is to provide a

[1] Study published in peer reviewed journal. Carlson, M.D., Mendle, J., & Harden, K.P. (2014). Early adverse environments and genetic influences on age at first sex: Evidence for gene x environment interaction. *Developmental Psychology*. *15(5),* 1532. I served as primary author and my role included data preparation and analysis and manuscript writing.

child with a sense of the availability and predictability of resources and of the trustworthiness and dependability of others. Children from environmentally disadvantaged backgrounds are hypothesized to develop behavior patterns that accelerate reproduction. That is, early rearing environments "set" an individual's reproductive behavior.

Belsky et al. (1991) defined environmental disadvantage rather broadly, including factors such as poverty, father absence, parental fighting, and harsh or inconsistent parent-child relations. Other theorists have emphasized the role of the father as a key determinant of the association between early sexual onset and familial ecological stress. Draper and Harpending (1982, 1988) first posited that father absence played a particularly important role in female sexual behavior. Ellis (2003, 2004) subsequently developed *paternal investment theory*, which emphasizes the quality of paternal caregiving as a key regulator of pubertal timing and onset of reproductive behavior in young girls. Consistent with this theory, several studies have found that father absence, one indicator of low paternal investment, uniquely predicts early onset of sexual activity in girls (e.g., Devine, Long, & Forehand, 1993; Ellis et al., 2003). More recent studies of pubertal timing (Tither & Ellis, 2008) and risky sexual behavior (Ellis, Schlomer, Tilley, & Butler, 2012) have found that variation in the low end of paternal investment appears to be most relevant for regulation of pubertal timing and risky sexual behavior in young girls.

A complicating factor in any theory of environmental mechanisms is the role of genes. Previous behavioral genetic research indicates that age at first sexual intercourse (AFS) is partially heritable, meaning that a proportion of the observable differences in AFS between individuals within a population can be attributed to genetic differences (see Harden, 2013, for a review). The magnitude of heritability estimates for AFS has varied, ranging from relatively modest (.24-.36; e.g., Lyons et al. 2004; Waldron, 2007; Segal & Stohs, 2009) to quite

substantial (.49-.72; e.g., Mustanski, Viken, Kaprio, Winter, & Rose, 2007; Dunne et al. 1997a). There is also evidence for both cohort and gender effects. Most notably, Dunne and colleagues (1997a) found that heritability for AFS was substantially higher for males (.72) and females (.49) born in the late 1950s to 1960s than for males (.00) and females (.32) born between the 1920s and early 1950s. These findings underscore that heritability estimates are inherently time and population specific, and are thus expected to vary as a function of sample characteristics. Indeed, such variability might provide clues with regard to the interplay between environmental context and genetic influences on AFS: Dunne et al. (1997a) proposed that, as social mores proscribing premarital sex became less culturally salient over successive generations, individual differences in AFS became increasingly a function of genetically influenced characteristics.

Behavioral genetic studies of sexual phenotypes have documented heritable variation in AFS, but have not typically considered the interplay between genetic influences and environmental regulators of sexual development. Evolutionary researchers acknowledge the existence of genetic influences on reproductive phenotypes, and have convincingly argued that non-zero heritability estimates in industrialized Western populations do not necessarily invalidate evolutionary arguments (e.g., Ellis, 2004). Nevertheless, this stream of research has primarily focused on how early environments might instigate a cascade of social and psychological outcomes that in turn regulate reproductive strategy, including timing of AFS, and few studies have specifically described how these environmental experiences might interact with genetic influences. The goal of the current paper, then, is to incorporate evolutionary thinking regarding the environmental antecedents of sexual timing into behavioral genetic research on age at first sex. Specifically, we investigate *gene-by-environment interaction* (G×E).

In a G×E interaction, genetic influences on the phenotype depend on environmental context, *and* an organism's response to the environment depends on genotype. In quantitative genetic studies, such as those described in the current paper, G×E interactions are most often reported in terms of how environmental context moderates genetic influences (although see Harden, Hill, Turkheimer, & Emery, 2008), while candidate gene × E interactions are typically reported in terms of how genetic influences moderate the effect of environmental context. These parameterizations are two sides of the same coin. Throughout the current paper, we will emphasize both sides of the interaction—how environment depends on genotype and how genotype depends on environment.

Although any finding of significant moderation is generally termed a G×E effect, there are a number of distinct patterns of G×E results, each of which is consistent with a different underlying mechanism. First, as predicted by a *diathesis-stress* model, individuals might differ in their genetic vulnerability to adverse environments. Put differently, adverse environments might activate or accentuate genetic vulnerabilities. Consequently, genetic variance—which refers to the variance in a phenotype accounted for by differences in genotype—will be higher under conditions of increased environmental adversity and minimized in high quality environments, as illustrated in the first panel of Figure 1.1 Second, individuals might differ in their genetic predispositions to profit from advantageous environmental contexts, as predicted by the *bioecological model* (Bronfenbrenner & Ceci, 1994) and more recently by the *vantage sensitivity* model (Pluess & Belsky, 2013). Accordingly, genetic variance will be increased under conditions of high environmental quality but suppressed under conditions of low environmental quality, as illustrated in the second panel of Figure 1.1 (labeled "genetic suppression"). Third, a *differential susceptibility* model posits that people differ in their susceptibility, or plasticity, to

environmental influence, such that those with greater plasticity are more sensitive to environments that are marked by both enrichment (leading to outcomes more positive than their less sensitive counterparts) and deprivation (leading to outcomes more negative than their less sensitive counterparts (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Ellis, Essex, & Boyce, 2005). Because differential susceptibility predicts that heritable polygenic variation contributes to differences in responses to the environment (Ellis et al., 2011), and that susceptible individuals will differ from non-susceptible individuals most markedly in both very good and very bad environments, genetic variance will be maximized at opposing extremes of an environmental moderator (both very low and very high environmental quality) and show negligible influence in "average" environments (e.g., South & Krueger, 2013). This is illustrated in the third panel of Figure 1.1.

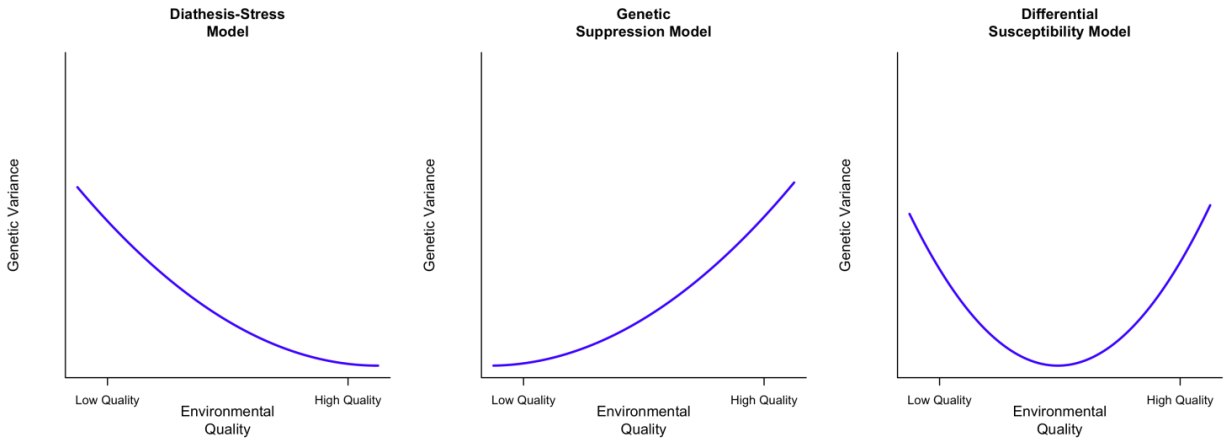


Figure 1.1 Descriptive patterns of G×E results

Evolutionary theorists have generally made no specific predictions regarding the expected pattern of interaction between ecological stress and genetic influences for outcomes such as AFS. Moreover, very few behavioral genetic studies have examined G×E in AFS. The

few twin studies that have been conducted, however, have found diminished genetic influences on sexual behavior and related phenotypes in more adverse or socially constraining environments (consistent with the genetic suppression pattern outlined above). For example, Waldron and colleagues (2009) found that additive genetic effects accounted for 0% of the variance in AFS for women who had experiences childhood sexual abuse, in contrast to 39% for non-abused women. Although childhood sexual abuse is qualitatively distinct from the risk factors examined in the present study, Waldron et al.'s (2009) results are broadly consistent with the hypothesis that genetic influences on age at first sex might be suppressed in adverse contexts. Similarly, Rodgers et al. (1999) found evidence for racial differences in the heritability of AFS: among African American adolescents (who experience, on average, lower average socioeconomic status and higher rates of father absence), the heritability of AFS approached zero, as compared to approximately 50% in Caucasians.

In addition, two candidate gene studies (Gibbons et al., 2012; Manuck, Craig, Flory, Halder, & Ferrell, 2011) have examined the association between life-history-relevant phenotypes and specific genetic variants as a function of ecological stress. Manuck et al. (2011) found a significant interaction between a polymorphism of the estrogen receptor-α gene, *ESR1*, and the quality of the family environment in predicting age at menarche. Consistent with the twin model results described above, in which genetic differences were strongest among advantaged populations, the difference among *ESR1* genotypes with respect to age at menarche was largest in high quality family environments. Finally, Gibbons et al. (2012) reported that polymorphisms in the serotonin transporter gene and dopamine D4 gene interacted with racial discrimination to predict "life history strategy cognitions" in African American adolescents; results were consistent with a differential susceptibility pattern. As many results from candidate G×E studies

are likely to be false positives (Duncan & Keller, 2011), and neither interaction result has been independently replicated, these results should be interpreted as preliminary.

Goals of the Current Study

The goal of the current study was to test whether three broad markers of environmental risk—low socioeconomic status, biological father absence in childhood, racial/ethnic minority status—moderate the heritability of AFS. Following previous theoretical and empirical work, we hypothesize that genetic influences on AFS will be minimized for youth who experience each of these environmental risks. In addition, because much research on reproductive timing has focused specifically on early reproductive timing in girls, we include both male and female adolescents in our sample, and examine gender differences in the magnitude of genetic influences on AFS. We test our hypotheses using a nationally representative sample of twins and non-twin full siblings from the National Longitudinal Study of Adolescent Health.

Method

Participants

Participants comprised a subsample of 1,244 same-sex twin and non-twin full-sibling pairs (281 monozygotic (MZ) pairs, 246 dizygotic (DZ) pairs, 717 non-twin full-sibling pairs) from the National Longitudinal Study of Adolescent Health (Add Health; Udry 2003a). In order to maximize power to detect interactive effects, all same-sex sibling pairs that shared both biological parents were included (Posthuma & Boomsma, 2000). Forty-nine percent of the sample was male and the remainder (51%) was female.

Add Health is a nationally representative, longitudinal study targeting adolescent health and risk behaviors. Data was collected in four waves between 1994 and 2008. Details of the

study design and sampling procedure may be found in Bearman, Jones, & Udry (1997) and Harris (2009). Add Health deliberately oversampled adolescent sibling pairs initially identified through school rosters and adolescent self-report on an in-school questionnaire completed by 90,000 students just prior to Wave I. From this point, twin pair zygosity was diagnosed by matching 11 molecular genetic markers and by twins' responses to four questionnaire items concerning similarity of appearance (Harris, 2006). Similar self-report measures are widely used to determine zygosity in twin research and have been cross-validated with zygosity determinations based upon DNA samples (Loehlin & Nichols 1970; Spitz et al., 1996). Jacobson and Rowe (1999) found negligible differences for sociodemographic variables (e.g., age, ethnicity, and maternal education) between Add Health sibling pairs and the full Add Health sample.

Measures

Age at first sex (AFS). At Waves I and II, participants reported whether they had ever had vaginal intercourse, and if so, in what month and year they had sex for the first time. From these reports, AFS (in years) was calculated. At Waves III and IV, participants were asked whether they had ever had vaginal intercourse, and if so, their age (in years) when they first had sex. As in previous studies with this data set (e.g., Harden, Mendle, Hill, Turkheimer, & Emery, 2008), analyses used the age at first sex from the earliest wave in which the participant reported having had sex, in order to minimize telescoping. For example, if an adolescent reported having had sex at age 13 at Wave I and at age 14 at Wave II, the Wave I report was used. Because we were interested in voluntary first sex, when non-virgin participants reported an AFS that was likely prepubertal and possibly nonconsensual (< 11 years), they were coded as missing (N = 104 individuals), resulting in a measure of AFS ranging from 11-30 years (M = 17.16, SD = 2.88).

Participants who did not endorse an age at first sex by the last reporting wave were also coded as missing (N = 336 individuals). The correlation between AFS in the first and second sibling of each pair was 0.33 in DZ twins, 0.40 in non-twin full sibling pairs, and 0.56 in MZ twins. The correlations in AFS across study waves ranged from .42 to .85. Reliability of reports of AFS across waves have been extensively studied in the Add Health data, and reporting errors tend to be largely random and have little impact on the conclusions drawn from the estimated ages at first sex (e.g., Upchurch et al. 2002).[2]

Biological father absence. At Wave I participants were asked whether they were living with their biological father and, if not, to indicate at what age they had they last lived with him. From this information a variable was created to index biological father absence at or before the age of 10. (This cut-off was chosen to ensure that father absence temporally preceded AFS; below, we report results from post-hoc sensitivity analyses which varied the cut-off age for father absence.) In the rare instance in which siblings living in the same household were discordant in their endorsement of biological father absence, we coded the pair father absent. Of the 1,244 sibling pairs, 361 (29%) reported father absence at or before the age of 10.

Socioeconomic status (SES). Socioeconomic status was measured using residential parent's mean level of educational attainment. Educational attainment is a commonly used index of socioeconomic status (Bradley & Corwyn, 2002), which might be more stable than family income (U.S. Treasury Department, 2008) and has been used in previous G×E analyses (e.g., Harden, Turkheimer, & Loehlin, 2007). Educational attainment was coded on a 9-point ordinal scale ranging from "8th grade or less" to "professional training beyond a four-year degree." The

[2]Using the Add Health data, Upchurch and colleagues (2002) evaluated the conclusions from seven analyses of age at first sex, each based upon a separate assumption for coding reported age of first intercourse, and found that all seven analyses reached very similar conclusions.

median level of SES in the study sample was a score of 5 (equivalent to a GED or high school graduate), and the mean score was 5.25 (*SD* = 2.14).

Race/ethnicity. In terms of racial/ethnic identity, 38% of our sample identified as either African American or Hispanic (among this 38%, 56% identified as African American and 44% Hispanic) and the remainder (62%) identified as Caucasian. Race was dummy-coded such that 1 corresponded to either African American or Hispanic, and 0 corresponded to Caucasian.

Gender. Gender was coded such that 1 corresponded to males and 2 to females. Table 1.1 summarizes the relations among AFS and the four moderating variables. Consistent with prior epidemiological literature, adolescents from higher SES homes reported a later AFS, on average, whereas adolescents from father-absent and racial/ethnic minority homes reported earlier AFS. Moreover, racial/ethnic minority adolescents were more likely to experience father absence and had lower SES.

Table 1.1 Correlations among study variables

Variables	Age at First Sex	SES	Father Abs.	Race	Gender
Age at First Sex	1.00				
SES	**.14**	1.00			
Father Absence	**-.13**	-.05	1.00		
Race	**-.10**	**-.28**	**.06**	1.00	
Gender	.04	**-.07**	-.02	.03	1.00

***Note*. Correlations based on one twin per pair, selected at random. Pearson correlations are presented for continuous variables; phi coefficients for the associations among dichotomous variables. Values significantly different from zero at $p < .05$ are in bold.**

Analyses

Data were analyzed using a series of structural equation models (SEM) using the software program Mplus (Muthen & Muthen 1998–2007). Model fit was evaluated using

differences in model log-likelihood and RMSEA. RMSEA values less than 0.05 indicate good model fit (Browne & Cudeck, 1993).

First, we estimated genetic and environmental influences on age at first sex using a univariate biometric model (Neale & Maes, 2007). This model partitions the variance of a phenotype (here, AFS) into additive genetic effects (*A*), shared environmental effects (*C*; family-level experiences that serve to make siblings more similar), and non-shared environmental effects (*E*; environmental experiences that are uncorrelated between twins, plus measurement error)[3]. This methodology capitalizes on the difference in genetic similarity between MZ and DZ twins to make inferences about the relative contributions of genes and environments to a given phenotype. The correlation between the *A* components in the first and second sibling in each pair is fixed to 1.0 in MZ twins and 0.5 in DZ twins and non-twin full siblings. In the context of the model MZ and DZ twins and non-twin full siblings share 100% of their common, or shared, environment and 0% of their unique, or non-shared environment. Thus the correlation between the *C* component in the first and second sibling is fixed to 1.0 in all pair types, whereas the *E* correlation is fixed to 0 in all pair types.

[3] Although conventionally labeled the non-shared environmental factor, this factor represents variation due to factors that differ within MZ twin pairs. To the extent that MZ twins are not, in fact, perfectly genetically identical (Charney, 2012), the effects of that within-MZ variation will be reflected in *E*.

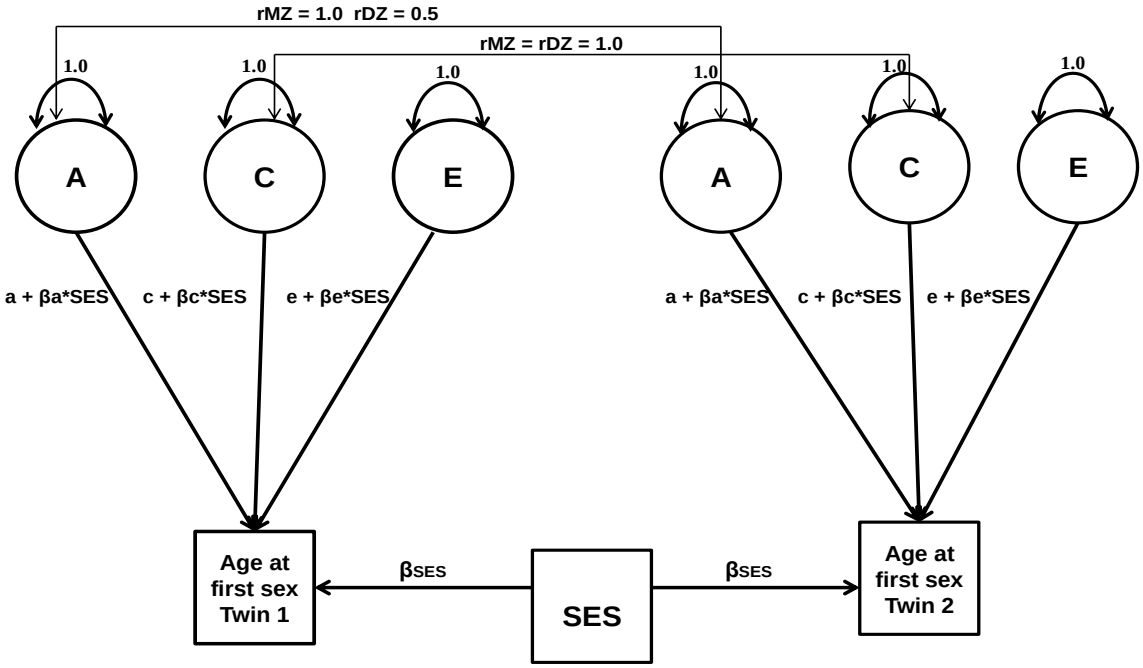


Figure 1.2 Path diagram of G×E interaction model.

***Note*. A, C, and E represent the univariate additive genetic, shared environmental, and non-shared environmental variance components; βa, βc, βe represent the moderated components of a, c, and e; and βses represents the main effect of SES on age at first sex.**

To test for G×E effects, we used a model that was designed to test interactions between a measured environmental moderator and the paths from the latent genetic and environmental factors (Purcell, 2002); see Figure 1.2 for an example using SES as the moderator. First, the main effect of the moderator variable, SES, on AFS is estimated as β_{SES}. The variance in the outcome variable (i.e., AFS) that is *unique* of the moderator is divided into latent *A*, *C*, and *E* components. In addition, the paths from the *A*, *C*, and *E* components to AFS are allowed to interact with the moderator variable (e.g., path labeled a + βa*SES). Thus, for the interaction model using SES as a moderator, AFS was modeled as follows:

(1) $$AFS = \beta ses + (a + \beta a*SES)A + (c + \beta c*SES)C + (e + \beta e*SES)E$$

The presence of moderation can be inferred when an interaction term, βa, βc, or βe is significantly different from zero. In the case of *gene*-environment interaction in particular, this would refer to a significant βa term. For example, a significant and positive βa term would indicate that as SES increases, the genetic variance in AFS also increases. Conversely, a negative βa term would indicate that as SES increases, the genetic variance in AFS decreases. To address concerns about gene-environment correlation (e.g., Mendle et al., 2006; Mendle et al., 2009), we controlled for gene-environment correlation by including the main effects of each moderator.

Results

Genetic and Environmental Influences on Age at First Sex: Population Averages

The first column in the top of Table 1.2 ("Main Effects Only") shows the parameter estimates from the univariate ACE model for AFS, without any moderator effects. These results represent the average contribution of genetic and environmental variation in the sample as a whole. Additive genetic effects accounted for 38% of the variance in AFS [1.74/(1.74+1.29+1.84)], shared environmental effects for 21%, and unique environmental effects for 42%.

Moderation by Socioeconomic Status

Model 2 tested whether SES moderated the magnitude of genetic and environmental influences on AFS. Parameter estimates for Model 2 are summarized in Table 1.2 ("SES Interaction" univariate model). Overall, the interaction model fit the data better than a reduced "main effect only" model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 52.88$, $\Delta df = 3$, $p < .001$). There was a significant main effect of SES, whereby each unit increase in SES

corresponded to just under a 2.5-month increase in AFS. Neither *C* nor *E* showed any significant interaction effects with SES, but there was a significant G×E interaction, illustrated in Figure 1.3A. Visual representation of the interaction reveals a U-shaped curve suggestive of a differential-susceptibility effect. Among adolescents whose parents had only a high school education, additive genetic effects accounted for no variation in AFS, whereas among adolescents whose parents had graduated from college, additive genetic effects accounted for 43%.

Table 1.2 Unstandardized ACE variance for age at first sex

Univariate Models					
Parameters	Main Effects Only	SES Interaction	Father Absence Interaction	Race/ Ethnicity Interaction	Gender Interaction
a	**1.74 (.22)**	.40 (.33)	**1.90 (.22)**	**2.09 (.23)**	1.97 (.77)
c	**1.29 (.22)**	**1.44 (.10)**	**1.18 (.26)**	.79 (.45)	.64 (1.08)
e	**1.84 (.08)**	**1.98 (.05)**	**1.83 (.10)**	**1.69 (.10)**	**2.46 (.29)**
b_M		**.20 (.04)**	**-.74 (.16)**	**-.64 (.14)**	.19 (.14)
b_a		**.57 (.05)**	**-1.35 (.45)**	-1.34 (1.02)	-.09 (.44)
b_c		.07 (.10)	.35 (.31)	.87 (.54)	.32 (.59)
b_e		-.07 (.04)	-.09 (.15)	**.41(.18)**	**-.41 (.17)**
Multivariate Model					
Parameters	Main Effects	SES Interaction	Father Absence Interaction	Race/ Ethnicity Interaction	Gender Interaction
a	0.41 (.61)				
c	**1.55 (.40)**				
e	**2.56 (.21)**				
b_M	--	**.19 (.04)**	**-.57 (.16)**	**-.30(.15)**	.17 (.14)
b_a	--	**.44 (.08)**	.21 (.29)	**-.74(.35)**	**.52(.24)**
b_c	--	-.05 (.11)	-.63 (.59)	.47 (.44)	-.31 (.34)
b_e	--	**-.07(.03)**	-.28 (.17)	.12 (.14)	**-.40 (.12)**

***Note*. All estimates unstandardized (in units of years). Univariate models estimated interactions with each moderator separately; multivariate model estimated interactions with all moderators simultaneously. Abbreviations: *a* = additive genetic effects, *c* = shared environmental effects, *e* = non-shared environmental effects; b_M = main effect of moderator on age at first sex; b_a, b_c, and b_e = interactions between the moderator and the A, C, and E**

components, respectively. Parameters significantly different than zero at $p < .05$ are in bold.

In addition, there also appeared to be an uptick in genetic variance at very low levels of SES. Based on the 95% confidence intervals around the estimated genetic variance at each level of parental education, however, genetic influences on AFS at the low end of parental education were significantly different from zero only at the very lowest level of parental education (less than 8th grade). Only 3.4% of twin pairs had this level of parental education. Roisman et al. (2012) argued that the *proportion affected* "offers a pragmatic way of evaluating evidence for differential susceptibility," as "the model is of limited use if only a small number of individuals experience the theorized [effects]" (p. 396). They suggested a cut-off of proportion affected > 16%; based on this recommendation, we "question whether [our] data are consistent with differential susceptibility theory" (p. 396).

An alternative way to represent this interaction, more directly parallel to how results from candidate gene × environment studies are typically presented, is to plot the predicted relationship between SES and AFS for two values on the latent "A" factor, which represents genetic predispositions for later versus earlier age at first sex (shown in Figure 1.3B). Higher socioeconomic advantage was positively associated with later AFS for youth with higher scores on the latent *A* factor (+1 SD above the mean). In addition, consistent with the U-shaped curve for genetic variance, there was a crossover effect potentially suggestive of differential susceptibility, as youth with higher scores on the latent *A* factor showed the earliest AFS at low levels of SES. As discussed above, however, the difference between genotypes (i.e., the genetic variance) was not significant at the low end of SES except for the few pairs whose parents had less than an 8th grade education.

Overall, results from the SES moderation models suggest that genetic influences on AFS are actuated in high SES environments but minimal in low SES environments. Put differently, high SES environments facilitate a later AFS, but only in those individuals with particular genetic predispositions.

Moderation by Biological Father Absence

Model 3 tested for moderation effects of father absence. Parameter estimates from Model 3 are summarized in the third column of Table 1.2 ("Father Absence Interaction" univariate model). There was a significant main effect of father absence, whereby children who experienced father absence at or before age 10 experienced AFS nearly 9 months earlier, on average, than their father-present counterparts. There was also a significant G×E interaction. For individuals who did not experience father absence at or before age 10, additive genetic effects accounted for 43% of the variation in AFS; in contrast, for individuals whose biological fathers were absent at or prior to 10, additive genetic effects accounted for only 5% of the variation in AFS. Neither *C* nor *E* showed any significant interaction effects with early father absence. Overall, the interaction model fit the data better than a reduced "main effect only" model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 20.98$, $\Delta\, df = 3$, $p < .001$).

Moderation by Race/Ethnicity

Model 4 tested for moderation effects of race/ethnicity. Parameter estimates for Model 4 are summarized in the fourth column of Table 1.2 ("Race/ Ethnicity Interaction" univariate model). There was a significant main effect of race/ethnicity. Children who identified as African American or Hispanic tended, on average, to experience AFS just over 7.5 months earlier than Caucasians. In terms of the moderation model, neither *A* nor *C* showed any significant moderation. There was, however, a significant *E* by race/ethnicity interaction. Unique

environmental variance accounted for roughly 36% of the variance in AFS for Caucasian youth, as compared to 60% for African American/Hispanic youth. The pattern for additive genetic variance mirrored the G×E effects observed for SES and father absence, in that the additive genetic variance in AFS tended to be suppressed for African American/Hispanic individuals and amplified for Caucasian individuals, although this interaction did not reach customary significance thresholds (p <.05). In addition, the interaction model overall did not fit the data significantly better than a reduced "main effect only" model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 5.82$, $\Delta df = 3$, $p = .12$).

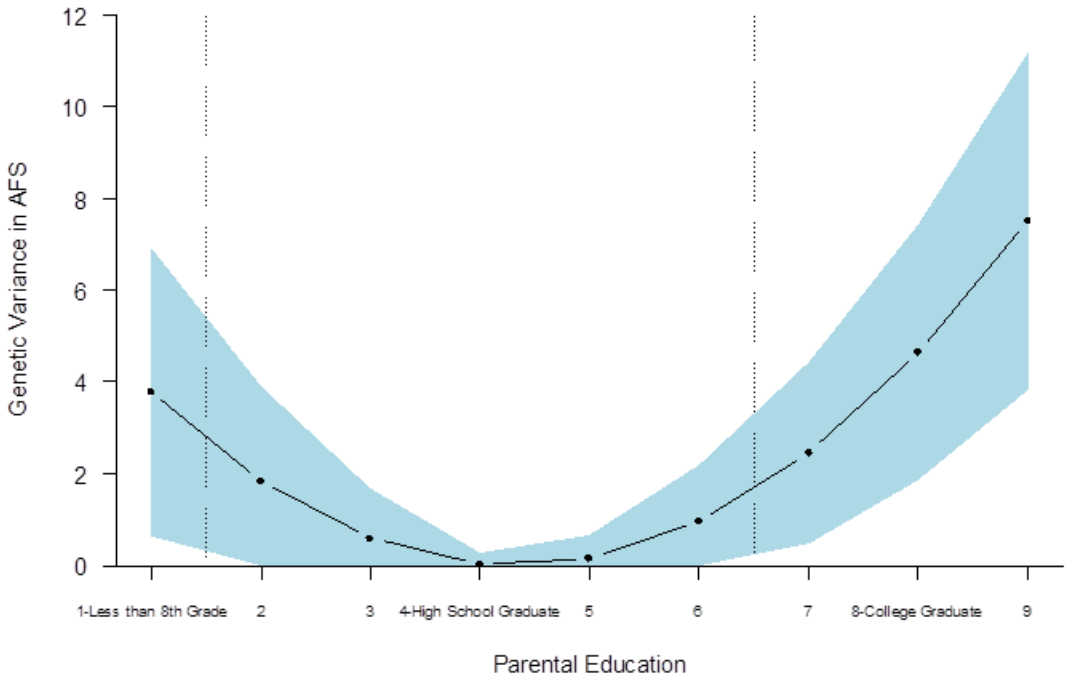


Figure 1.3A Gene × SES interaction on age at first sex

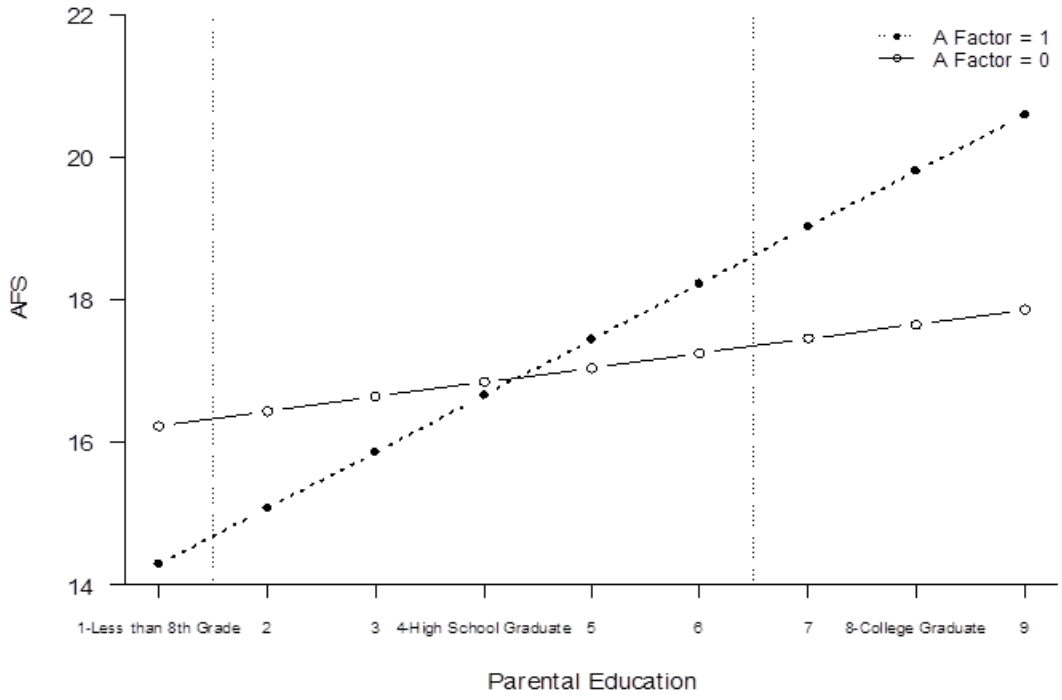


Figure 1.3B Gene × SES interaction on age at first sex

Note. **Figures 1.3A and 1.3B based on parameters from univariate model of SES interaction (Table 2). "AFS" = Age at first sex. Vertical dashed lines delineate regions of significance; genetic variance (i.e., differences between genotypes) is significantly different from zero to the left of the first line and the right of the second line. (a) 95% confidence interval around estimate for genetic variance is shown in blue. Genetic variance is plotted in unstandardized form (in units of years). (b) "A Factor" = additive genetic factor illustrated in Figure 2. Values represent the mean and +1 *SD* above the mean on the latent factor.**

Moderation by Gender

Model 5 tested for moderation effects of gender, as summarized in the final column of Table 1.2 ("Gender Interaction" univariate model). There was no significant main effect for gender. Neither *A* nor *C* showed any significant moderation; however, there was a significant *E* by gender interaction. For females, unique environmental variance accounted for 51% of the variance in AFS, whereas for males, unique environmental effects accounted for 60% of the

variance. Overall, the interaction model fit the data better than a reduced "main effect only" model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 16.82$, Δ *df* = 3, $p < .001$).

Multivariate Interaction Model

Because of the overlap between SES, racial/ethnic minority status, and father absence, our final model tested all interactions simultaneously in a multivariate interaction model. This model tests whether each moderator uniquely interacts with genetic and environmental influences on AFS, above and beyond its relation with the other moderators. The bottom half of Table 1.2 summarizes the parameter estimates from the multivariate interaction model. Six results were notable. First, SES, father absence, and race/ethnicity all had significant unique main effects on AFS. Second, the interaction between SES and additive genetic variance in AFS remained significant in the full model. As was observed when SES was entered as the only moderator, additive genetic effects accounted for greater variation in AFS among youth from more advantaged backgrounds. Third, a non-shared environmental interaction with SES emerged as significant, suggesting that as SES increased, the non-shared environment became less influential on AFS. Fourth, the interaction between father absence and genetic variance in AFS was no longer statistically significant in the full model. Fifth, the interaction between race/ethnicity and non-shared environmental variance was also no longer significant once entered into the full model. However, race/ethnicity did moderate the additive genetic variance for AFS in the full model, with genetic variation suppressed among Black/Hispanic youth compared to Caucasian youth. Sixth, the interaction between gender and non-shared environmental variance maintained its significance once entered into the full model. In addition, the interaction between gender and the latent additive genetic variance became statistically significant, with females showing greater additive genetic influence on AFS than males. Overall,

the fit of the full multivariate interaction model was significantly better than the fit of a reduced model, in which all interaction effects were fixed to zero ($\Delta\chi^2 = 75.06$, $\Delta\ df = 12$, $p < .0001$).

Post-Hoc Sensitivity Analyses

Because evolutionary-developmental theory emphasizes the first 5–7 years of life as particularly sensitive to environmental input, we conducted a series of post-hoc sensitivity analyses assessing father absence using alternate age cutoffs (ages 5, 6, 7) and as a continuous measure (number of years of father absence before age 10). In none of these scenarios was father absence a statistically significant moderator of genetic and environmental influences on AFS. Full results of these sensitivity analyses may be obtained upon request.

Discussion

Youth who experience environmental adversity tend to initiate sexual intercourse at an earlier age. The present study tested whether three broad markers of environmental risk—low socioeconomic status, biological father absence in childhood, and racial/ethnic minority status—moderated the heritability of AFS. Our results suggest that genetic influences for age at first sex are greater in contexts of relative social advantage and suppressed in more adverse conditions. In particular, genes are a stronger predictor of timing of first sex among high-SES and Caucasian individuals and contribute negligibly to AFS among low-SES and ethnic minority adolescents. These findings are consistent with previous G×E interaction studies of environmental adversity both for AFS (Waldron et al., 2008) and for other phenotypes (e.g., intelligence, Turkheimer et al., 2003). In addition, father absence did not uniquely moderate genetic influences on AFS in a full model that simultaneously controlled for SES and race/ethnicity, suggesting that father

absence, *per se*, might not be the most potent environmental precursor to early sexual activity, but rather a "proxy" for a larger matrix of social disadvantage.

In addition, although we obtained significant shared environmental variance in AFS in the sample as a whole (21% in a model with no moderation), it should be noted that mean differences between race/ethnic groups in age at first sex will lead to higher estimates of shared environmental variance. This finding might be attributable to the high levels of racial and ethnic diversity of the Add Health sample. In support of this interpretation, the estimate of shared environmental variance from a model that included the main effect of race/ethnicity ("Race Interaction Model" in Table 1.2) was smaller (.79 versus 1.29) and no longer significantly different than zero.

To make sense of our results, it is important to remember that there is not a single gene "for" age at first sex; rather, genetic influences on sexual timing are likely mediated through a complex constellation of physiological (e.g., pubertal timing, physical attractiveness), motivational (e.g., sexual drive), and behavioral (e.g., sensation seeking, substance use, religiosity) traits. As such, the finding of higher genetic variance in advantaged populations indicates that these "embodied characteristics matter strongly and pervasively as causes" of individual sexual behavior (Freese, 2008, p. S20), but *only* for individuals who occupy positions of relative social privilege and economic security. The relevant question, then, is how these links between embodied characteristics and sexual behavior are disrupted under conditions of social disadvantage. One explanation that could account for both the "main effects" of adverse environments on the average age at first sex and the moderating effects on genetic variance in age at first sex is that individuals who would otherwise be genetically predisposed towards later sexual intercourse (via, for example, later pubertal timing, greater religiosity, reduced sensation

seeking, or greater anxiety) are shaped by the social context in which they live to initiate sexual intercourse earlier. For example, several studies have shown that media consumption, which tends to correlate with riskier sexual attitudes, is greater among low SES youth (e.g., Blosser, 1988; Ward et al., 2005). This might result both in reduced genetic variation in age at first sex and earlier mean ages at first sex—consistent with our findings.

Previous research on racial differences in the relation between pubertal timing and initiation of sexual activity in adolescent girls would also be consistent with this explanation. Large, epidemiological samples have shown non-trivial heritability estimates for pubertal timing (e.g., Harden, Mendle, & Kretsch, 2012), and genetic influences on age at menarche have been found to overlap with genetic influences on age at first sex (Rowe, 2002). That is, part of the genetic influence on age at first sex—at least in girls—can be accounted for by heritable differences in the onset of puberty. However, after controlling for mean group differences in pubertal timing as a function of race, Cavanagh (2004) found that the phenotypic association between pubertal timing and age at first sex was moderated by race: later pubertal timing was associated with delayed sexual initiation among Caucasian but *not* among African American girls. In explaining her results, Cavanagh (2004) noted that "differences in the social construction of girlhood must be taken into account when examining the pathways that make up the human lifecourse" (p. 306). Although puberty might be the time in which many Caucasians first become aware of themselves as sexually mature, African American girls tend to be overly sexualized in American culture (hooks, 1992). Consequently the pubertal transition might not hold the same significance for African American girls, both in terms of how they view themselves and how they are viewed by others (Cavanagh, 2004).

Another consideration is the role of childhood sexual abuse, an established risk factor for earlier AFS. Using a larger sample of female adolescents from the Add Health study, Mendle, Ryan, & McKone (2016) found that childhood sexual abuse (prior to age 6) predicted earlier menarche (as did African American and Latina ethnicity), while other forms of childhood adversity included in the model simultaneously, including physical abuse, physical neglect, and father absence, did not. As timing of menarche also predicts onset of sexual intercourse due in part to common genetic mechanisms (Rowe, 2002), then the accelerating effects of CSA on pubertal timing could be one mechanism whereby genetically mediated differences in pubertal timing are suppressed from manifesting phenotypically, thus resulting in earlier pubertal timing and correspondingly earlier onset of sexual intercourse.

In a post-hoc analysis we examined the correlations between childhood sexual abuse (CSA) and each of our moderators. For these analyses we operationalized CSA as any affirmative endorsement at either wave 3 or wave 4 (ever = 1, never = 0) to the question: "How often had one of your parents or other adult care-givers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?" Results revealed statistically significant associations between CSA and each of the moderators suggesting that CSA will be a promising consideration for future studies on the mechanisms underlying this pattern of results (*r* with SES = -.12, Father Absence = .25, Race/Ethnic Minority Status = .12, Gender = .21; all *p*'s < .01).

Earlier sexual intercourse has sometimes been conceptualized under a higher-order domain of externalizing (or disinhibited) behavior (Jessor & Jessor, 1977). In contrast to our finding of decreased heritability of sexual behavior in disadvantaged contexts, Hicks et al. (2009) found that the heritability of adolescent antisocial behavior increased in the context of multiple

indicators of environmental adversity (deviant peer relations, poor parent-child relations, and poor academic engagement). Although precocious sexual activity is correlated with externalizing behaviors, it is also qualitatively unique in important ways. For instance, although earlier intercourse in some individuals is likely influenced by the hallmark characteristics of externalizing behavior, such as sensation seeking and impulsivity, it might also be part of an integrated life-history strategy (Ellis, 2004). In addition, recent research has shown that early sexual activity within the context of a long-term monogamous relationship might actually be associated with decreased levels of delinquent behavior (Harden et al. 2008; McCarthy & Grodsky, 2011).

Limitations and Future Directions

There are a number of methodological considerations that are important to note. First, although we have interpreted SES as an index of differences in environmental advantage, parental educational attainment also reflects genetic differences between parents (Rowe, Vesterdal, & Rodgers, 1998). This occurs because educational attainment is partially contingent on heritable traits such as intelligence, conscientiousness, and attentional capacity. Although the biometric model controlled for genetic variance common to educational attainment and AFS, we were unable to rule out the possibility that increased heritability in AFS might not be better accounted for by a gene × gene interaction rather than a gene × environment interaction. In addition, the magnitude of the genetic correlation between AFS and SES remains unknown. Because raised-together biological sibling pairs are necessarily identical for parental characteristics such as SES, however, twin modeling is not genetically informative in this regard.

Second, while we were interested in obtaining reports on voluntary AFS, the limitations of the AFS definition preclude our ability to ascertain with 100% certainty that all sexual experiences were indeed voluntary.

Third, given the well documented links between childhood sexual abuse (CSA) and earlier AFS, and the links between CSA and each of our moderators (Waldron et al., 2008; Bachmann, Moeller, & Benett, 1988; Ryan, Mendle, & Markowitz, 2015), our omission of CSA as a covariate in this study is a limitation and should be included in future research.

Fourth, like many other researchers, we use father absence as an indicator of paternal investment. Although father absence is highly correlated with factors broadly related to low paternal investment, such as diminished relationship quality and emotional distance (e.g., Cooksey & Craig, 1998; Gorvine, 2010), recent studies have shown that alternative indicators of (low) paternal investment, such as paternal psychopathology, substance abuse and legal troubles, might be better predictors of daughters' development than a dichotomous father present-absent distinction (e.g., Ellis & Essex, 2007; Ellis et al., 2012). It will be important for future studies to assess alternative indicators of paternal investment, including indicators indexing the positive end of the spectrum, before an unequivocal interpretation can be made for its role in moderating the heritability of AFS.

Fifth, although African Americans and Hispanics both show earlier age at first sex relative to Caucasians and both endure the effects of racism in American culture, there are important sociocultural differences between these two groups. Unfortunately, due to sample size, we did not have adequate power to estimate differences between these minority groups. Finally, more generally, quantitative genetic models require very large numbers of participants to distinguish between different patterns of G×E (e.g., differential susceptibility versus genetic

suppression). In the case of the present study, the results of our SES moderation models and post-hoc sensitivity analyses appear to be consistent with genetic suppression. However, because of the comparatively small number of families at the very low end of the SES spectrum (i.e., less than high school education), it is worth being cautious about whether our results reflect a differential susceptibility versus genetic suppression effect. This ambiguity underscores the need for behavioral genetic research to include adequate numbers of socioeconomically disadvantaged and minority families, who are currently underrepresented in the majority of twin samples.

Conclusion

The present study used behavioral genetic methodology to investigate the genetic and environmental etiology of individual differences in AFS. We tested for the presence of gene × environment interaction using three broad indices of environmental risk. Individuals whose backgrounds were characterized by relative advantage showed greater genetic influence in AFS. Conversely, genetic effects were suppressed for individuals whose backgrounds were characterized by relative disadvantage. These results suggest that among adolescents who have fewer social and economic resources to draw upon, AFS is increasingly influenced by family-level environmental circumstances rather than genetic propensities.

Study 2: Racial/Ethnic Differences in the Genetic and Environmental Links between Adolescent Sexual Behavior and Externalizing Behaviors[4]

Over the last 15 years the rates of teen pregnancy and sexually transmitted infections (STI) in the United States have steadily declined. However, U.S. rates of teen pregnancy, births, abortion, and STIs remain well above other Western industrialized countries (Barbieri, 2012). Moreover, across all of these outcomes, the gap in sexual health disparities between Caucasian youth and African American youth remain large. In 2009, the rate of new HIV infections was 15 times higher among African American women than Caucasian women (Hamilton, Martin, & Ventura, 2012). African American women are also 3-4 times more likely to die during pregnancy or childbirth than Caucasian American women—a disparity that has endured for over 50 years (Center for Reproductive Rights, 2014). Moreover, in certain regions of the United States such as Mississippi and Washington D.C., the rates of pregnancy related maternal death for African American mothers are on par with rates in non-industrialized countries, while rates for Caucasian American mothers in these same regions remain on par with the national average (Center for Reproductive Rights, 2014). Despite the magnitude and persistence of these race/ethnic disparities, surprisingly little research has examined whether predominant etiological models for understanding adolescent sexual behavior generalize to African American youth.

Adolescent Sexual Behavior and Externalizing

Greater number of sexual partners and earlier sexual intercourse are correlated with externalizing behavior problems, such as substance use and delinquency. These associations have prompted theorists, beginning with Reiss (1970), to conceptualize early sexual activity as

[4] This study has not been published yet. As primary author my role on this study has involved conceptualization, the data preparation and analysis, and writing it up and getting feedback and input from my advisor, Dr. Paige Harden who is co-author.

an indicator of a generalized propensity toward deviance. Jessor and Jessor (1977) advanced *problem behavior theory,* which posits that behaviors such as adolescent delinquency, substance use, problems at school and precocious sexual activity are manifestations of a single syndrome. Similarly, others have hypothesized an underlying predisposition towards sensation seeking, risk taking, and impulsivity to account for the co-occurrence between behaviors such as unprotected sex and substance abuse (Deas-Nesmith, Brady, White, & Campbell, 1999).

Behavioral genetic research has shown that antisocial behaviors, substance use behaviors, and personality traits such as impulsivity and sensation seeking can be modeled as manifestations of a heritable externalizing dimension (EXT) (Krueger, Markon, Patrick, Benning, & Kramer, 2007). Although indicators of sexual activity *per se* are not typically included in behavioral genetic research on EXT, the positive correlations between earlier sexual behaviors (such as first sexual intercourse and number of sexual partners) and delinquency and substance use suggest that early sexual behavior may also be a manifestation of genetic vulnerabilities to EXT. Indeed, using a sample of US adolescent twins, Harden et al. (2009) and Harden and Mendle (2011) found evidence for common genetic vulnerability accounting for both delinquency and early age at first sex. Likewise, common genetic influences largely accounted for the association between sexual health-risk related behaviors and EXT behaviors in two nationally representative twin samples from Australia (Verweij, Zietsch, Bailey, & Martin, 2009) and Sweden (Donahue, D'Onofrio, Lichtenstein, & Langstrom, 2013).

Race/Ethnic Differences in the Links between Sexual Behavior and EXT

Empirical evidence supporting a problem behavior perspective, as well as heritability estimates for externalizing, have been derived from predominantly middle-class Caucasian samples. Even among the most diverse behavioral genetic samples, African Americans typically

represent a relatively small proportion of participants, and population-level findings could obfuscate important sub-population differences. The absence of African American samples informing theory in this domain is problematic, particularly in light of the sexual health disparities that remain between Caucasian and African Americans in the United States.

A body of literature suggests that problem behavior models may not explain sexual activity in African American adolescents, among whom associations between EXT and sexual behavior are attenuated. For instance, Miller-Johnson et al. (1999) found that 6th grade self- and parent-reports of externalizing did not predict early childbearing among African American females. Similarly, Bachanas et al. (2002) found that conduct disorder was not significantly associated with unprotected sex among female African American teens, and Black, Ricardo, and Stanton (1997) failed to find an association between sexual activity and substance use and delinquency in a sample of urban African American teens (Black et al., 1997). Finally, Doljanac and Zimmerman (1998) found that substance use and delinquency better predicted high-risk sexual behavior for Caucasians than African American youth, leading the authors to conclude that other models may be necessary to explain sexual behavior among African American adolescents.

Preliminary evidence further questions whether the association between early sexual behavior and EXT are due to shared genetic mechanisms in ethnic minority youth, as demonstrated in previous findings with Caucasians. For instance, impulsivity—a heritable trait that confers risk for EXT behaviors– did *not* predict engaging in (pre-sexual) moderate intimate behaviors at age 12 for African American girls but did for Caucasian girls (Hipwell et al. 2010). Additionally, early menarche, another heritable individual difference factor, did not predict onset of sexual intercourse in African American girls as it did in Caucasians (Cavanagh, 2004).

Theoretically consistent with these findings, recent behavioral genetic work showed that genetic influences on age at first sex were lower among African American and Hispanic adolescents and youth from lower SES backgrounds relative to Caucasian adolescents and youth from higher SES backgrounds (Carlson, Mendle, & Harden, 2014). Together, these studies question whether shared genetic vulnerabilities account for the association between EXT and sexual behavior among African American youth and youth from lower socioeconomic households more broadly.

Goals of the Current Study

Our goals were two-fold. First, we used a large sample of U.S. adolescents to examine phenotypic associations between EXT and two indices of sexual behavior, age at first sex (AFS) and number of sexual partners. Second, we examined the genetic and environmental underpinnings of the association between EXT and adolescent sexual behavior as a function of race/ethnicity, using the twin and sibling participants from this sample. Our aim was to determine whether the conceptualization of adolescent sexual behavior as a manifestation of EXT, which emphasizes the role of genetic predispositions linking alternate manifestations of deviance, extends to African American youth and youth from lower SES backgrounds.

Method

Participants

Participants were drawn from Wave 1 of the National Longitudinal Study of Adolescent Health (Add Health; Udry 2003A). Add Health is a nationally representative longitudinal study, collected in four waves between 1994 and 2008, that targets adolescent health and risk-taking behaviors. The full sample, which includes over 20,000 participants, was selected through a stratified, school-based, cluster sampling design (see Chantala & Tabor, 1999, for an elaborated

description of the study design). Wave 1 participant data were collected in 1995, when participants ranged from 12 to 20 years of age (Wave 2 in 1996, Wave 3 in 2001-02, & Wave 4 in 2007-08). Because our first set of analyses (factor analyses of the EXT spectrum) did not require genetically informative data, we capitalized on the full sample of Caucasian (70%, *n* = 10,001) and African American (30%, *n* = 4,286) adolescents using data from 14,287 participants total (49% male, 51% female). Because we were interested in adolescents, we limited our sample to individuals between the ages of 12-18. Participant mean age was 15.85 years. For the biometric analyses, participants included 879 pairs of same-sex twin and non-twin full biological siblings, 197 monozygotic [MZ] twin pairs, 184 dizygotic [DZ] twin pairs, and 498 full sibling [FS] pairs; 49% male, 51% female). All analyses controlled for the main effects of gender.

The racial/ethnic composition of the sibling pairs sample was as follows: 70% non-Hispanic Caucasian and 30% African American. The gender break-down by sibling pair type for Caucasian and African American adolescents was as follows: Caucasian boys/girls: MZ pairs = 72/68, DZ pairs = 54/75, FS pairs =204/183; African American boys/girls: MZ pairs =30/27, DZ pairs = 24/31, FS pairs = 47/64.

Measures

The Add Health interviews measured a wide array of health-relevant domains, including physical, mental, emotional, and sexual health. Items included in the current analysis were drawn from Wave 1. Indicators of EXT were based on theoretical consistency with the EXT factor as previously described in the literature (e.g., Krueger et al., 2002; Krueger et al., 2007). See Table 2.1 for descriptive statistics for the full sample.

Table 2.1 Descriptive statistics for measurement model indicators as a function of race/ethnicity and biological sex for full sample

	Caucasian			African American		
Items	Mean (SD) / %	Range	N	Mean (SD) / %	Range	N
Tobacco						
M	1.42 (2.13)	0-5.48	4351	.49 (1.28)	0-5.48	1635
F	1.51 (2.18)	0-5.48	4461	.32 (.98)	0-5.48	1807
Binge drinking past 2 weeks						
M	0.55 (0.79)	0-2.45	4940	0.25 (0.62)	0-2.45	1979
F	0.41 (0.68)	0-2.45	5003	0.18 (0.51)	0-2.45	2249
Intoxication frequency						
M	0.53 (0.72)	0-2.45	4938	0.28 (0.63)	0-2.45	1986
F	0.45 (0.68)	0-2.45	4940	0.22 (0.53)	0-2.45	2250
Inhalant use						
M	8%	0-1	4933	3%	0-1	1971
F	6%	0-1	5004	3%	0-1	2249
Marijuana use						
M	31%	0-1	4940	29%	0-1	1975
F	28%	0-1	5003	22%	0-1	2244
Other illegal drug use*						
M	11%	0-1	4929	3%	0-1	1974
F	11%	0-1	5009	2%	0-1	2246
Physical fight						
M	0.78 (.90)	0-3.87	4967	0.94 (.93)	0-3.87	2022
F	0.36 (0.66)	0-3.87	5023	.64 (.80)	0-3.61	2252
Delinquency						
M	1.53(1.18)	0-5.74	4966	1.34 (1.14)	0-5.74	2021
F	1.31(1.06)	0-5.29	5023	1.31 (0.99)	0-4.69	2254
Sex in exchange for drugs						
M	2%	0-1	4955	2%	0-1	2018
F	1%	0-1	5015	1%	0-1	2255

***Note*. Tobacco = square root transformation of number of days smoked in last 30; Intoxication frequency = square root transformation of total intoxication events over last 12 months; Inhalant use = dichotomous use/no use over last 12 months; Marijuana use = dichotomous use/no use over lifetime; Other illegal drug use = drug use outside of inhalants and marijuana; Physical fights = physical violence composite (square root transformed); Delinquency = rule breaking composite (square root transformed, detailed in methods); Sex in exchange for drugs = dichotomous yes/no lifetime measure.**

Substance use. *Drinking* items included number of occasions in which participants endorsed having five or more drinks in one sitting over the previous two weeks and the number of occasions over the last 12 months in which participants drank to the point of intoxication. Individuals reporting no endorsement of smoking or drinking were coded as 0. Items were square root transformed to reduce non-normality. *Smoking* was measured as the number of days cigarettes were smoked over the last 30 days and did not uniquely load onto any one specific factor and thus loaded exclusively on the general EXT factor.

Illegal drugs. Use of illegal drugs was measured using four dichotomized items indexing any use of any *inhalant* or *other illegal drug* use (excluding marijuana) over the last 12 months, any endorsement of lifetime marijuana use, and any lifetime endorsement of *exchanged sex for drugs*.

Antisocial behavior. *Fighting* was measured using 8 items (e.g., participated in a group fight, carried a gun to school) rated on a 4-point scale [Frequency of occurrence over last 12 months: 0=*Never*, 1=*One or two times*, 2=*Three or four times*, 3=*Five or more times*] except for three serious items (shooting or stabbing someone, getting into a serious physical fight; pulling a knife or gun on someone) which were coded as 0 = *Never* or 3 = *One or more times*; items were summed to produce a composite tapping violent aggression (alpha = .75). *Delinquency* was measured using 11 items (e.g., number of times lied to parents, stole something worth more than $50) rated on the same 4-point scale (alpha = .80); items were again summed to produce a composite tapping non-violent antisocial behavior such as stealing, vandalizing, and general rule breaking. Both the *fighting* and *delinquency* composite scores were square root transformed to reduce skewness.

Age at first sexual intercourse (AFS). At each wave of data collection participants were asked whether they had ever had vaginal intercourse, and if so, were directed to specify the month and year (Waves 1 and 2), or their age in years (Waves 3 and 4), that they first has sex. To minimize telescoping we used reports from the earliest wave in which sexual intercourse was reported, a method that has been done in previous studies with this data set (e.g., Harden, Mendle, Turkheimer, & Emery, 2008). Participants reporting an age at first sexual intercourse before age 11 were coded as missing due to the greater possibility of sexual intercourse in this age range being non-consensual. Thus, our measure of age at first sex ranged from 11-30 years (M = 17.16, SD = 2.88). Individuals who did not report an age at first sex by Wave 4 were coded as missing.

Number of sexual partners. Number of sexual partners was obtained from via self-report at Wave 3 in response to a computer administered interview in which participants were asked, "With how many partners have you ever had vaginal intercourse, even if only once?" Participants that reported no sexual partners were assigned a 0. Because a minority of individuals reported exceedingly high numbers we log-transformed this variable to reduce positive skew.

Socioeconomic status (SES). Socioeconomic status was controlled for in all analyses and indexed using mean level of residential parent's education. Parent education was coded on a 9-point scale ranging from "8th grade or less" to "professional training beyond a 4-year degree" and standardized in analyses. Mean level of SES corresponded to high school graduate.

Racial/ethnic minority status. Race/ethnicity (Caucasian or African American) was based on adolescent self-report. Race/ethnicity was dummy-coded, such that Caucasians = 0 and African Americans = 1.

Analyses

All analyses were conducted in M*plus* (Muthen & Muthen, 1998–2007). Model fit was assessed using the chi-squared fit statistic and root mean square error of approximation (RMSEA). RMSEA values below .08 are considered good fit and below .05 are considered very good fit.

Results

Confirmatory Factor Analytic Model of the Externalizing Spectrum

We conducted a confirmatory factor analysis (CFA) that tested a bifactor model of EXT behaviors (Figure 2.1) addressing nonindependence of data from siblings using the TYPE = COMPLEX command in M*plus* and clustering by family. Specifically, all EXT behaviors were modeled as loading onto a general *EXT* factor while controlling for gender, race/ethnicity, and SES on *EXT*. Because of the well documented developmental changes in EXT over the course of adolescence, it was necessary to control for age at the indicator level. Likewise, gender, race/ethnicity, and SES were also controlled at the indicator level for delinquency and fighting items, as these characteristics have shown diverging trends across these traits that could be obscured at the factor level. In addition, residual covariance among theoretically related variables was modeled with three specific factors on which subsets of items loaded: *drinking*, *illegal drugs*, and *antisocial behavior*. Based on preliminary analyses, smoking cigarettes did not have unique loadings on any of the specific factors and thus loaded exclusively on the general *EXT* factor. The fit of this CFA model was good (RMSEA = .05, CFI = .97, TLI = .94). We used this CFA model to estimate individual factor scores for EXT; these factor scores were saved and used as the key variables in all subsequent analyses.

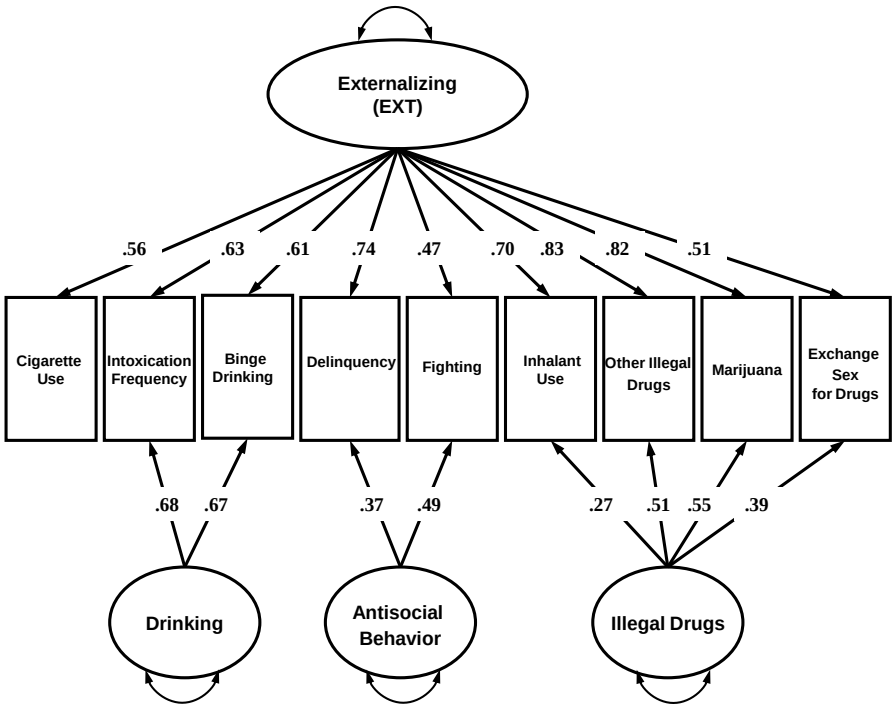


Figure 2.1 Confirmatory factor analytic model of the EXT spectrum

***Note*. All factor loadings are presented in standardized form and significant at $p < .0005$. Pearson correlations between study variables broken down by race/ethnicity and gender are presented in Table 2.2. EXT factor scores and each sexual phenotype indicated that on average, higher EXT was associated with earlier age at first sex across race/ethnicity, though attenuated among African Americans ($r_{male\_t1} = -.25$, $r_{female\_t1} = -.28$) relative to Caucasians ($r_{male\_t1} = -.41$, $r_{female\_t1} = -.47$). Likewise, higher EXT was associated with more sexual partners but this association was also attenuated for African American adolescents ($r_{male\_s1} = .22$, $r_{female\_s1} = .18$) relative to Caucasian adolescents ($r_{male\_s1} = .36$, $r_{female\_s1} = .37$). (Values above are presented for one sibling per pair, all *p*-values < .05.) Values for other half of sibling pair presented below in Table 2.2 for all study variables (partitioned by race/ethnicity and biological sex.)**

Table 2.2 Zero-order correlations between study variables with standard errors as a function of race/ethnicity and biological sex within the biometric sample

	African American Adolescents							Caucasian Adolescents						
Composites	**EXT**	**F/D**	**Alc.**	**Drg.**	**AFS**	**NSX**	**SES**	**EXT**	**F/D**	**Alc.**	**Drg.**	**AFS**	**NSX**	**SES**
EXT	1.00	0.57	0.29	0.15	-0.30	0.19	-0.18	1.00	0.36	0.36	-0.01	-0.47	0.33	-0.27
SE		0.08	0.10	0.10	0.10	0.11	0.10		0.05	0.05	0.06	0.05	0.06	0.06
n		101	101	101	94	82	101		304	304	304	279	246	304
F/D	0.52	1.00	-0.14	-0.19	-0.38	0.06	-0.01	0.34	1.00	-0.19	-0.13	-0.04	0.03	-0.01
SE	0.08		0.10	0.10	0.10	0.11	0.10	0.05		0.06	0.06	0.06	0.06	0.06
n	120		101	101	94	82	101	314		304	304	279	246	304
Alc.	0.18	-0.10	1.00	-0.06	0.02	0.00	0.16	0.35	-0.18	1.00	0.00	-0.22	0.20	0.18
SE	0.09	0.09		0.10	0.10	0.11	0.10	0.05	0.06		0.06	0.06	0.06	0.06
n	120	120		101	94	82	101	314	314		304	279	246	304
Drg.	0.07	-0.13	-0.04	1.00	0.06	0.08	0.52	-0.06	-0.34	-0.15	1.00	0.07	-0.08	0.58
SE	0.09	0.09	0.09		0.10	0.11	0.09	0.06	0.05	0.06		0.06	0.06	0.05
n	120	120	120		94	82	101	314	314	314		279	246	304
AFS	-0.29	-0.20	-0.13	0.00	1.00	-0.27	0.07	-0.42	-0.09	-0.13	0.05	1.00	-0.50	0.25
SE	0.09	0.09	0.10	0.10		0.11	0.10	0.05	0.06	0.06	0.06		0.06	0.06
n	110	110	110	110		84	94	278	278	278	278		248	283
NSX	0.26	0.18	0.10	0.11	-0.46	1.00	-0.12	0.36	0.04	0.12	-0.02	-0.55	1.00	-0.16
SE	0.10	0.10	0.10	0.10	0.09		0.11	0.06	0.07	0.06	0.07	0.06		0.06
n	95	95	95	95	94		82	238	238	238	238	224		250
SES	-0.10	0.13	0.37	0.58	0.01	0.07	1.00	-0.21	0.02	0.16	0.48	0.27	-0.15	1.00
SE	0.09	0.09	0.09	0.07	0.10	0.10		0.06	0.06	0.06	0.05	0.06	0.06	
n	120	120	120	120	110	95		314	314	314	314	280	240	

Note. **Only one twin shown per pair. Girls are presented below the diagonal and boys are presented above the diagonal. EXT = global externalizing factor, F/D = domain specific Fighting/Delinquency, Alc. = domain specific alcohol use, Drg. = domain specific drug use, AFS = age at first sexual intercourse, NSX = Number of sexual partners, SES = socioeconomic status.**

Means and standard deviations for EXT and sexual phenotypes broken down by race/ethnicity and biological sex are summarized in Table 2.3. On average, African American adolescents reported earlier AFS than Caucasian adolescents (p <.001), and within race/ethnicity timing of AFS was similar. Comparable numbers of sexual partners were reported across race/ethnicity and gender with the exception of African American boys reporting approximately one more partner, on average, than the other three groups (all p's < .01). Externalizing was lower among African American adolescents relative to Caucasian adolescents (p < .0001), corresponding to approximately one standard deviation below the sample mean.

Table 2.3 Means and standard deviations for sexual phenotypes and externalizing factor from measurement model broken down by race/ethnicity and gender

Variable	Caucasian	African American
Age at First Sex		
M	17.40 (2.89)	15.80 (2.94)
F	17.32 (2.78)	16.31 (2.35)
Number of Sexual Partners		
M	2.87 (1.19)	3.71 (2.36)
F	2.74 (1.80)	3.03 (1.98)
Externalizing factor		
M	-.11 (.90)	-1.05 (.84)
F	-.16 (.83)	-.95 (.62)

***Note*. One sibling shown per pair. Age at first sex is in units of years. Number of sexual partners is presented in non-transformed person units for interpretive clarity. EXT factor scores presented in Z-scale.**

Biometric Analyses: Main Effects

For each sexual phenotype (age at first sex and number of sexual partners), we fit a classical biometric model to examine the genetic and environmental contributions to its association with EXT. Variance in each phenotype was decomposed into three latent factors, labeled *A* for additive genetic effects, *C* for shared environmental effects (experiences that make

siblings more similar to one another), and *E* for non-shared environmental effects (experiences that make siblings more dissimilar from one another, plus measurement error). *A* factors were correlated 1.0 for MZ twins and 0.5 for DZ and FS pairs. Each sexual phenotype was regressed on the *A*, *C*, and *E* components of EXT. These cross paths (labeled a_c, c_c, e_c in Table 2.4 and Figure 2.2) are of particular importance because they represent the degree to which each sexual phenotype and EXT overlap due to shared genetic or environmental influences. Both models controlled for the main effects of ethnicity, gender, SES and age on the phenotypes.

Parameter estimates (unstandardized regression coefficients) for the main effect models are displayed in Table 2.4. The total variance in EXT, the variance in each sexual phenotype that overlaps with EXT, and the variance in each sexual phenotype that is unique from EXT can be calculated by summing the squares of their corresponding coefficients (labeled a, c, e, for the EXT estimates, a_c, c_c, e_c for estimates common to both EXT and sexual outcome, and a_u , c_u , e_u for estimates unique from EXT). The *proportional* contributions of genetic and environmental influences can be calculated by squaring the parameter coefficient of interest and dividing it by the total variance in that domain. For example the heritability of AFS that is unique from EXT can be computed as follows:

$$a_u^2 / (a_u^2 + c_u^2 + e_u^2) = 1.45^2 / (1.45^2 + 0.00^2 + 1.77^2) = .40$$

The heritability statistic for the overall variance in each sexual phenotype can also be attained from these parameters by dividing the *total* additive genetic variance for a given sexual outcome by the total variance for that outcome: e.g., $(a_u^2 + a_c^2) / (a_u^2 + c_u^2 + e_u^2 + a_c^2 + c_c^2 + e_c^2)$. Across the sample as a whole, AFS and number of sexual partners showed comparable genetic influences, accounting for 38% of the total variance in AFS and 40% of the total variance in number of sexual partners. The unique environment accounted for moderate variance in both

phenotypes (46% for AFS; 50% for number of sexual partners) and the shared environment accounted for modest variance (15% for AFS; 10% for number of sexual partners). For EXT additive genetic influences accounted for approximately 44% of the total variance, followed by non-shared environmental influences (40%) and shared environmental influences (16%).

For both AFS and number of sexual partners, the covariance with EXT was attributable to both common genetic and shared environmental factors. At the population-average level, 23% of the total variance in AFS, and 20% of the total variance in number of sexual partners, could be accounted for by variance in EXT. For AFS, the shared environment accounted for 64% of the covariance followed by additive genetic effects which accounted for 31% and the non-shared environment which accounted for 5%. For number of sexual partners, additive genetics accounted for 53% of the covariance followed by shared environmental effects which accounted for 46%. The unique environment did not account for any appreciable covariance (1%).

For both AFS and number of sexual partners, the variance that was unique from EXT was primarily attributable to non-shared environmental influences (59% for AFS; 59% for number of sexual partners) followed by additive genetic influences (40% for AFS; 36% for number of sexual partners) and negligible to modest shared environmental influences (1% for AFS; 15% for number of sexual partners).

Table 2.4 Parameter estimates from bivariate main effect models for each sexual phenotype and externalizing

	EXT			Common with EXT			Unique from EXT		
Sexual Outcome	**a**	**c**	**e**	**a_C**	**c_C**	**e_C**	**a_U**	**c_U**	**e_U**
Age at First Sex	**0.53***** (.41, .65)	**0.32***** (.18, .47)	**0.51***** (.46, .56)	**-0.70**** (-1.19, -.20)	**-1.01*** (-1.54, -.47)	**-0.28** (-.52, -.03)	**1.45***** (1.08, 1.82)	0.00 (-2.52, 2.53)	**1.77***** (1.60, 1.95)
No. of Sexual Partners	**0.53***** (.40, .66)	**0.32***** (.17, .48)	**0.51***** (.45, .56)	**0.31**** (.04, .58)	0.29 (-.03, .60)	0.04 (-.08, .15)	**0.54***** (.26, .81)	0.08 (-1.29, 1.46)	**0.69***** (.61, .78)

***Note*. Parameter estimates and 95% confidence intervals are unstandardized. a = additive genetic, c = shared environment, e = non-shared environment. a, c, e = parameter estimates pertain to externalizing (EXT); a_C, c_C, e_C = parameter estimates for covariance between sexual phenotypes and externalizing; a_U , c_U, e_U = parameter estimates for variance in sexual phenotypes unique from externalizing. All estimates control for the main effects of ethnicity, gender, SES, and age. The general externalizing factor was transformed to Z-scale prior to analyses. AFS is in metric of years; number of sexual partners was log transformed to reduce skewness and transformed to Z-scale. Values in bold indicate statistical significance and astrices specify the degree of significance as follows, * $p < .05$, ** $p < .01$, *** = $p < .001$.**

Biometric Analyses: Race/Ethnic Interaction Effects

For the general EXT factor and each sexual phenotype, we next fit an interaction model (illustrated in Figure 2.2) in which each of the genetic and environmental parameters were allowed to differ by racial/ethnic minority status. Following the recommendations of Keller (2014), this model also controlled for interactions with relevant covariates (gender, SES, and age; interactions with age and SES are omitted from Figure 2.2 for ease in presentation). Interaction models also controlled for main effects of ethnicity, gender, SES and age, as well as two-way interactions between the moderator of primary interest (ethnicity) with each covariate control (i.e., gender, SES, & age for a total of three observed two-way interaction terms).

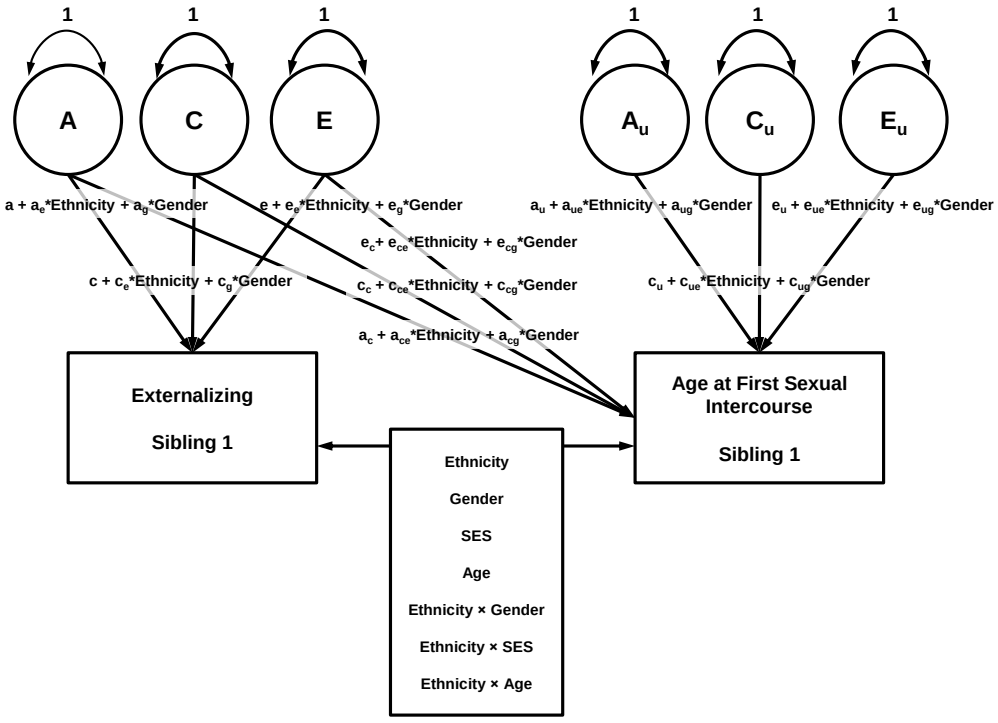


Figure 2.2 Path diagram of bivariate interaction model for EXT and age at first sex

Note. **Only interactions with ethnicity and gender are shown, but the complete models also controlled for interactions with SES and age. A = additive genetic, C = shared environment, E = non-shared environment. Only one twin per pair is shown.**

Parameter estimates for the interaction models are displayed in Table 2.5 and key results are illustrated in Figures 2.4-2.6. Dummy coding was used for ethnicity [0 = Caucasians, 1=African Americans] and gender [0 = males, 1 = females]. Caucasian males thus served as the intercept when all moderators were assigned 0 (labeled "reference" in Table 2.5). In the case of SES and age, values were mean centered such that 0 corresponded to average SES and average age of the sample. To the extent that any of the nine *a*, *c*, and *e* parameter estimates show statistically reliable deviation from the reference group as a function of the moderator (or other covariates included in the model), this would constitute a significant interaction. The path coefficients for each of the non-reference groups can be derived by summing the value of the reference group and interaction terms. Variance components can then be derived by squaring this value (and standardized by dividing this value by the sum of the total variance). In the case of the cross paths, the sign of the coefficient is of particular importance because it provides information about the directionality of the relationship between the predictor and the outcome. Using AFS as an example, if the a_C coefficient is negative, this means that the genetic variance associated with high externalizing is predictive of earlier AFS.

We present results broken down by race/ethnicity and gender (i.e., Caucasian boys, Caucasian girls, African American boys, African American girls). However, when interpreting these findings we remind the reader that these estimates represent variation due to *A*, *C*, and *E* as a function of ethnicity and/or gender at mean levels of SES and age. Both interaction models fit the data better than their respective main effects models (AFS: $\Delta\chi^2 = 864$ Δ df = 40, $p < .0005$; number of sexual partners: $\Delta\chi^2 = 849$, Δ df = 42, $p < .0005$).

Interaction results for externalizing. Table 2.6 shows the parameter estimates that correspond to our two interaction models. Overall, ethnicity and gender moderated the variance

in EXT such that the shared environment accounted for more variance in EXT for girls (28% for Caucasian girls; 20% for African American girls) than boys. This interaction appeared to be driven by Caucasian boys as the standardized squared summation of the individualized parameter estimates revealed that only 7% of the variation (non-significant) in EXT could be accounted for by shared environmental factors in Caucasian boys versus 25% among African American boys ($p = .003$).

Interaction results for the association between EXT and sexual behaviors. Results are illustrated in Figures 2.3-2.6, with parameter estimates reported in Table 2.5. Covariance between EXT and AFS emerged for all groups. Among Caucasian girls, 48% of the total variance in AFS could be accounted for by variation in EXT, followed by 32% for Caucasian boys, 35% for African American girls, and 15% for African American boys. Most prominently, ethnicity moderated the genetic covariance between EXT and AFS such that additive genetic influences accounted for substantially more of the covariation between AFS and EXT among Caucasian adolescents than among African American adolescents ($p < .01$). Specifically, among Caucasian boys and girls respectively, 72% and 57% of the total covariance between AFS and EXT could be accounted for by additive genetic effects. The remaining covariance was primarily accounted for by the shared environment (24% and 32% for Caucasian boys and girls, respectively). In contrast, for African American adolescents, additive genetic influences did not account for *any* of the overlap between EXT and AFS. Rather, the shared environment was most prominent, accounting for 71% and 92% of the covariance for African American boys and girls, respectively. The remainder of the covariance between AFS and EXT was accounted for by non-shared environmental influences (8% of total covariance for African American girls and 29% of the covariance for African American boys).

Additionally, the magnitude of overlap between EXT and AFS showed a differential pattern across SES for African American adolescents and Caucasian adolescents. For Caucasian adolescents the overlap between EXT and AFS was moderate and uniform irrespective of mean parent education level. In contrast, for African American adolescents, the overlap between EXT and AFS appeared to be greater at higher levels of SES such that it was negligible among adolescents whose mean parent education was 1SD below the sample average ("some high school"; for African American boys and girls respectively, variation in EXT accounted for 5% and 11% of the variance in AFS), modest at the sample average ("high school graduate"; reported above), and modest-to-moderate at just above 1SD of the sample average ("college graduate"; for African American boys and girls respectively, variation in EXT accounted for 28% and 45% of the variance in AFS). See Figure 2.3.

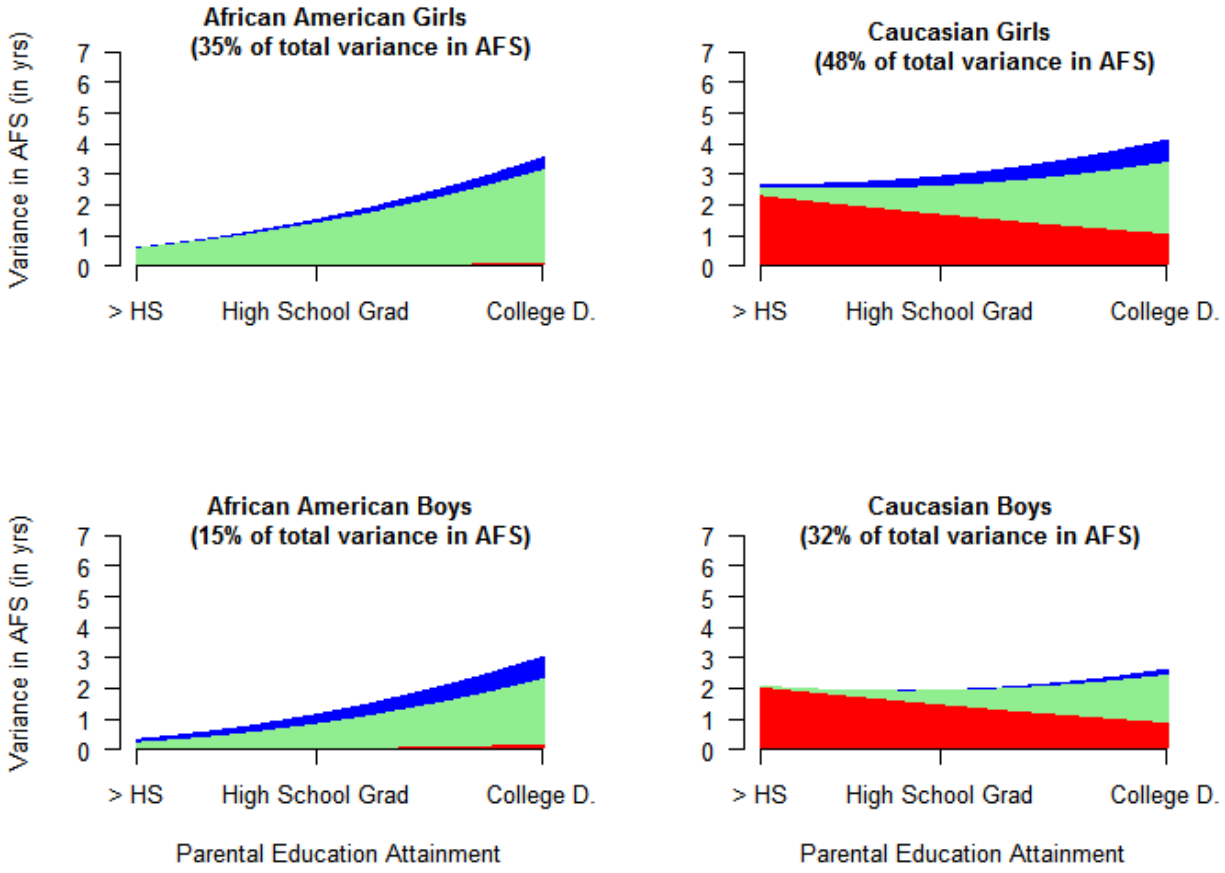


Figure 2.3 Decomposition of total variance in age at first sex predicted by variance in EXT across parent education broken down by race/ethnicity and gender

Note. **Percentages for each group refer to the percentage of total variance in age at first sex accounted for by variance in externalizing at average levels of parent education. AFS = age at first sex; EXT = externalizing; A = additive genetic, C = shared environment, E = non-shared environment. Figure based on model parameters presented in Table 2.4.**

For Caucasian adolescents, lower mean parent education corresponded to greater genetic mediation of the overlap between EXT and AFS. Specifically, mean parent education 1SD below the sample mean corresponded to additive genetic variation accounting for 97% and 86% of the total covariance between EXT and AFS among Caucasian boys and Caucasian girls, respectively. In comparison, at average levels of SES (i.e., mean parent education equivalent to

high school diploma), additive genetic effects accounted for 74% and 57% of the total covariance between AFS and EXT among Caucasian boys and Caucasian girls respectively. And for Caucasian boys whose mean parent education was just beyond 1SD above the sample mean (Z = 1.26; equivalent to mean parent education of "college graduate"), additive genetic influences accounted for 40% of the covariance between AFS and EXT, and shared environmental influences accounted for 54% of the covariance. A similar pattern emerged for Caucasian girls such that additive genetics accounted for 30% of the total covariance between EXT and AFS and the shared environment accounted for 53% of the covariance when mean parent education corresponded to college graduate.

As for African American adolescents, while the magnitude of the covariance between AFS and EXT was minimal at lower levels of parent SES and greater at higher levels, the source of covariation remained the same—attributed to shared environmental factors. This differential pattern may be a byproduct of differences in sample size between Caucasian and African American subsamples which could impact the stability of heritability estimates for the smaller African American subsample. At opposing ends of the SES distribution depicted on the x-axes in Figures 2.3-2.6, these differences should be kept in mind as fewer adolescents populate the outer regions of the distribution, which will be magnified with smaller subsample of African Americans. See Figure 2.3.

Like AFS, covariance between EXT and number of sexual partners emerged for all groups. Among Caucasian boys, 41% of the total variation in number of sexual partners could be accounted for by variation in EXT, followed by 25% for Caucasian girls, 22% for African American boys, and 20% for African American girls. The covariance between EXT and number of sexual partners was primarily genetically mediated among boys and Caucasian girls. Among

African American girls, in contrast, the covariance between EXT and number of sexual partners was primarily attributable to common shared environmental mechanisms (72%). Finally, although there were no statistically significant interactions by ethnicity or gender, the individual summation of parameter estimates for the genetic covariance between EXT and number of sexual partners failed to reach statistical significance for African American girls (b = .17, p = .26). Instead, summation of the individual parameter estimates for shared environmental covariance between EXT and number of partners did attain statistical significance for African American girls (b = -.32, p < .05). Additionally, for Caucasian girls, individual parameter estimates revealed modest but statistically significant shared environmental covariance between EXT and number of sexual partners (b = -.20, p < .05) though less appreciable and in the opposite direction than the common genetic variance (b = .34, p < .05). The directionality of the shared environmental covariance was consistent with that which also emerged among African American girls. These results are illustrated in Figure 2.4.

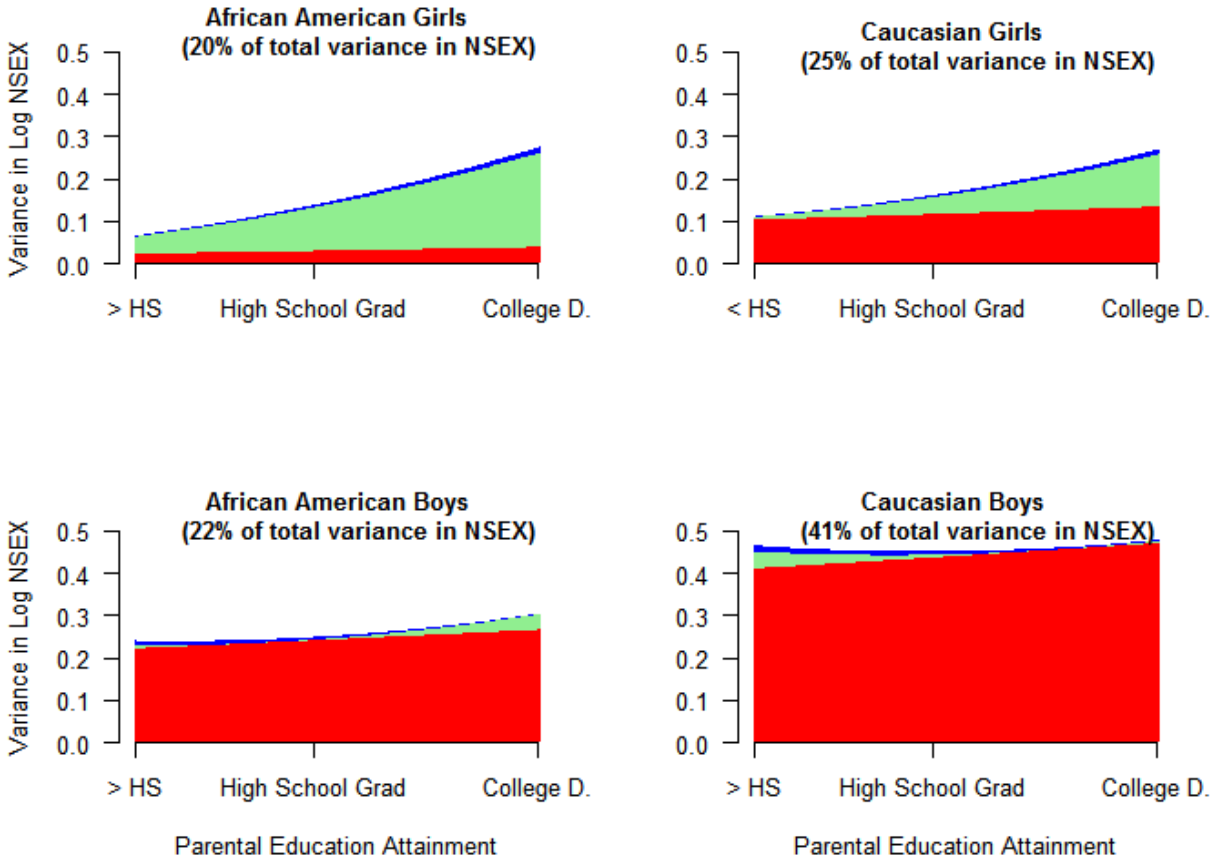


Figure 2.4 Decomposition of total variance in number of sexual partners predicted by variance in EXT across parent education broken down by race/ethnicity and gender

Note. **Percentages for each group refer to the percentage of total variance number of sexual partners accounted for by variance in externalizing at average levels of parent education. NSEX = number of sexual partners; EXT = externalizing; A = additive genetic, C = shared environment, E = non-shared environment. Figure based on model parameters presented in Table 2.4.**

Finally, a negative C × SES interaction emerged on the covariance of EXT and number of sexual partners such that as SES increased the shared environmental variance associated with higher EXT increased as well and contributed to fewer sexual partners. Upon further inspection of these data, this effect appeared to pertain to girls as described above and depicted in the plots in Figure 2.4.

Interaction results for unique variance in sexual phenotypes. Results are illustrated in Figures 2.5 and 2.6, with parameter estimates reported in Table 2.5. Consistent with previous findings from a larger sample of Add Health siblings (e.g., Carlson et al., 2014; a sample that also included Hispanic ethnicity and ages into young adulthood), SES moderated the heritability of AFS such that higher SES corresponded to greater unique genetic variance in age at first sexual intercourse (independent of EXT).

Upon further inspection of these data, this pattern appeared to be driven by Caucasian adolescents, which is consistent with multivariate results from our previous study (2014) in which African American (and Hispanic) ethnicity was also associated with the suppression of additive genetic effects underlying AFS. African American ethnicity moderated the shared environmental variance in AFS such that African American ethnicity corresponded to greater shared environmental variance in AFS unique from EXT. Upon further examination of the individual parameter estimates, this effect appeared to be driven by African American boys (b = 1.5, $p < .01$) as the estimates for African American girls did not attain statistical significance (b = .65, p = .26). Finally, although genetic variance appears to decrease as a function of SES for African American adolescents in Figure 2.5, these estimates did not attain statistical significance, nor appreciable magnitude, and likely reflect instability due to smaller sample size for African American adolescents.

Among Caucasian adolescents the shared environment did not account for any of the variation in AFS that was unique from EXT. Finally, there was also a significant E × Gender interaction such that the unique environment accounted for greater variation in AFS for boys relative to girls ($p < .01$). Nevertheless, the unique environment (which also includes

measurement error) was an appreciable source of variation in the AFS unique from EXT for all adolescents. Results illustrated in Figure 2.5.

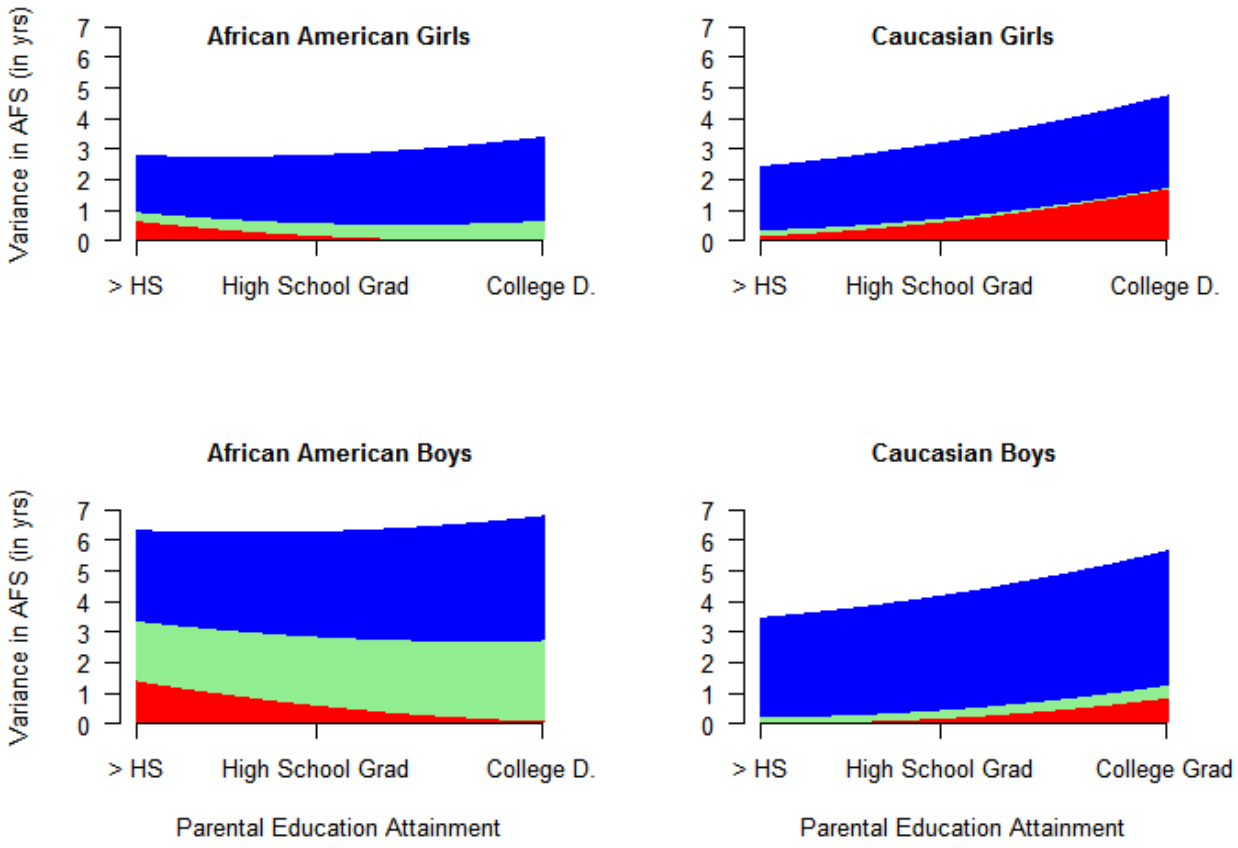


Figure 2.5 Decomposition of total variance in age at first sex unique from EXT across parent education broken down by race/ethnicity and gender

Note. **AFS = age at first sex; EXT = externalizing; A = additive genetic, C = shared environment, E = non-shared environment. Figure based on model parameters presented in Table 2.5.**

For number of sexual partners, no significant interactions emerged for the variance unique from EXT. However, the individual parameter estimates for the shared environmental variance did emerge as statistically significant for African American adolescents (African American boys: $b = .45$, $p < .05$; African American girls: $b = .46$, $p < .01$). Similar to the case for

AFS, non-shared environmental factors were the most prominent source of variation in the etiology of number of sexual partners unique from EXT. Results illustrated in Figure 2.6.

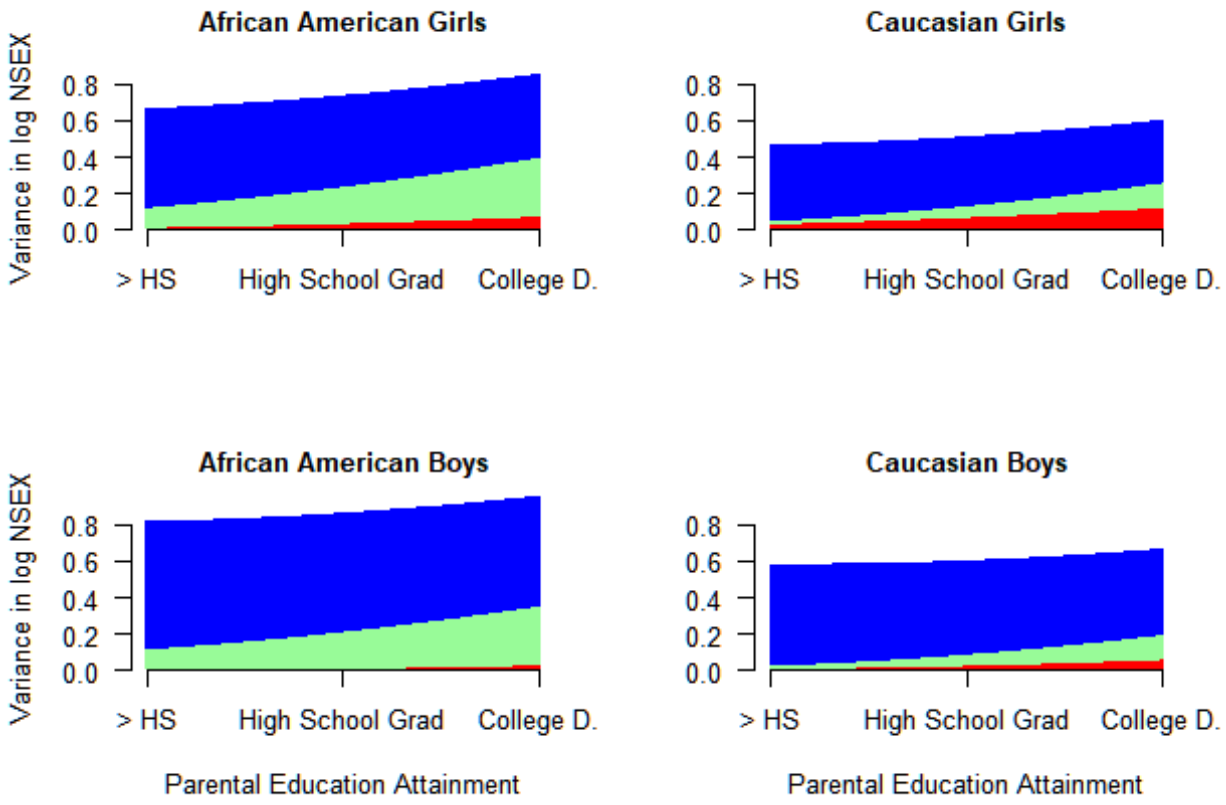


Figure 2.6 Decomposition of total variance in number of sexual partners unique from EXT broken down by race/ethnicity and gender

Note. **NSEX = number of sexual partners; EXT = externalizing; A = additive genetic, C = shared environment, E = non-shared environment. Figure based on model parameters presented in Table 2.5.**

Table 2.5 Parameter estimates from bivariate interaction models for each sexual phenotype and externalizing

		EXT			Common with EXT			Unique from EXT		
Age at First Sex		**a**	**c**	**e**	**a_C**	**c_C**	**e_C**	**a_U**	**c_U**	**e_U**
Reference	(*Cauc. Male*)	**0.64*** (.49, .79)	0.24 (-.08, .55)	**0.54*** (.47, .62)	**-1.20*** (-1.85, -.58)	-0.69 (-1.66, .27)	-0.15 (-.53, .22)	0.37 (-.38, 1.11)	0.52 (-.30, 1.14)	**1.94***** (1.72, 2.16)
Interaction coefficients	Ethnicity ($-_e$) *(African Am)*	**-0.20*** (-.38, -.03)	0.16 (-.11, .42)	-0.08 (-.18, .02)	**1.31**** (.50, 2.10)	-0.21 (-1.36, .93)	***-0.42*** (-.89, .05)	-1.12 (-2.37, .14)	**.98*** (.06, 1.91)	-0.08 (-.44, .29)
	Gender ($-_g$) *(Female)*	-0.03 (-.20, .14)	***0.25*** (.50, .00	-0.07 (-.16, .02)	-0.09 (-.74, .22)	-.28 (-1.1, .55)	-0.02 (-.36, .41)	0.39 (-.38, 1.16)	***-0.86*** (-1.73, .02)	**-0.36**** (-.60, -.12)
	SES ($-_{ses}$) *(Mean)*	-0.04 (-.12, .04)	0.10 (.00, .20)	-0.02 (-.06, .03)	0.22 (-.16, .14)	**-.45*** (-.79, -.11)	**-0.23*** (-.46, -.01)	**0.42*** (.03, .82)	0.10 (-.38, .59)	0.13 (-.03, .29)
	Age ($-_{age}$)	0.01 (-.04, .06)	0.04 (.00, .09)	0.00 (-.03, .04)	0.00 (-.22, .24)	**0.23*** (.06, .41)	-0.08 (-.22, .07)	**0.25*** (.04, .47)	0.04 (-.31, .40)	0.06 (-.04, .17)
Number of Sexual Partners		**a**	**c**	**e**	**a_C**	**c_C**	**e_C**	**a_U**	**c_U**	**e_U**
Reference *(Cauc. Male)*		**.60***** (.42, .78)	0.23 (-.19, .55)	**0.57***** (.48, .65)	**0.66***** (.33, .90)	0.08 (-.36, .59)	-0.09 (-.25, .06)	.14 (-.53, .81	0.25 (-.05, .55)	**0.72***** (.59, .85)
Interaction coefficients	Ethnicity ($-_e$) *(African Am)*	-0.10 (-.26, .06)	.16 (-.02, .30)	**-0.12*** (-.21, -.02)	-0.17 (-.54, .20)	-0.12 (-.38, .16)	0.01 (-.18, .20)	-0.08 (-.57, .42)	.20 (-.10, .50)	0.09 (-.03, .23)
	Gender ($-_g$) *(Female)*	-0.20 (-.47, .08)	**-.64***** (-1.08, .34))	-0.05 (-.15, .05)	-0.32 (-.80, .18)	-0.28 (-.71, .13)	0.16 (-.03, .35)	.11 (-.43, .66)	.00 (-.33, .33)	-0.10 (-.24, .04)
	SES ($-_{ses}$) *(Mean)*	-0.01 (-.07, .06)	0.02 (-.06, .09)	-0.02 (-.07, .02)	0.02 (-.13, .03)	**-0.12*** (-.23, -.01)	0.03 (-.04, .11)	0.09 (-.05, .23)	0.12 (-.01, .24)	-0.03 (-.09, .03)
	Age ($-_{age}$)	0.04 (.00, .09)	0.03 (-.03, .08)	-0.01 (-.04, .03)	0.02 (-.10, .11)	0.01 (-.05, .09)	0.01 (-.06, .08)	-0.09 (-.18, .01)	0.01 (-.07, .10)	0.03 (-.01, .08)

***Note*. Model estimates and 95% confidence intervals are presented in raw metric form (unstandardized variance components can be derived by squaring these values). a = additive genetic, c = s hared environment, e = non-shared environment. a, c, e = parameter estimates pertaining to externalizing; a_C, c_C, e_C = parameter estimates for covariance between sexual phenotypes and externalizing; a_U , c_U, e_U = parameter estimates for variance in sexual phenotypes unique from externalizing. EXT = Externalizing; ($_{-e}$), ($_{-g}$), ($_{-SES}$), ($_{-age}$) specify the suffix for the interaction coefficients (e.g., for ethnicity: a_e, c_e, e_e, a_{Ce}, c_{Ce}, e_{Ce}, a_{Ue}, c_{Ue}, e_{Ue}) All estimates control for the main effects of the moderator (ethnicity), gender, SES, and age as well as observed moderator × control variable interactions and latent A, C, and E × control variable interactions. The general externalizing factor was transformed to Z-scale prior to analyses. AFS is in metric of years; number of sexual partners was log transformed to reduce skewness and transformed to Z-scale. Statistical significance is denoted as follows: italic bold $p < .10$, * $p < .05$, ** $p < .01$, *** = $p < .001$.**

Discussion

Population-wide correlates of adolescent sexual activity, such as substance use and delinquency, have given rise to a conceptualization of adolescent sexual activity as a manifestation of a genetically influenced propensity to externalizing behaviors more generally. To date, empirical evidence for this perspective, however, has been largely based upon middle class Caucasian samples. Using a subsample of African American and Caucasian same-sex twins and siblings, our aims were twofold: (1) examine the source and magnitude of the associations between EXT and two sexual behaviors, age at first sex (AFS) and number of sexual partners, and (2) examine the genetic and environmental links between EXT and sexual behavior to determine whether patterns for the racial and middle class majority extend to African American adolescents and youths from lower SES backgrounds more broadly.

Biometric analyses indicated important sociodemographic differences with respect to sexual behavior in adolescence and its relation to EXT behaviors. Specifically, although externalizing was correlated with AFS and number of sexual partners across race/ethnicity and SES, the magnitude of these associations and the extent to which they reflected common genetic influences substantially differed in accordance with the intersectionality of racial classification, gender and SES.

Overall, EXT accounted for more variance in sexual behavior for Caucasian youth than for African American youth. For Caucasian adolescents, the overlap between AFS and EXT was accounted for by common genetic mechanisms and, to a lesser extent, shared environmental mechanisms. In contrast, for African American youth, the overlap between AFS and EXT was exclusively accounted for by shared environmental mechanisms. Across all adolescents, the

magnitude of shared environmental mechanisms on the overlap between EXT and AFS was greater at higher levels of SES.

Genetic variance mediated the association between number of sexual partners and EXT for boys and accounted for the majority of the covariance for Caucasian girls. However, for African American boys, the amount of the overlap between EXT and number of sexual partners was modest overall, accounting for a minimal amount of the total variation in number of sexual partners, whereas among Caucasian boys, it was quite moderate (accounting for over 40% of the total variance in number of sexual partners). Among African American girls, the magnitude of the overlap between EXT and number of sexual partners was also modest and was fully accounted for by shared environmental mechanisms.

Finally, like the pattern for AFS, the magnitude of shared environmental influences on the overlap between EXT and number of sexual partners was greater at higher levels of SES, but only for adolescent girls. For adolescent boys the overlap between EXT and number of sexual partners remained constant across SES.

Adolescents in the United States receive strong social and cultural messages that emphasize delaying sexual intercourse as a means of delaying childbearing and focusing on educational and occupational goals. It is perhaps not surprising, then, that Caucasian youth who violate this social proscription may be more likely to also violate other social norms, including respect for other persons/property and taboos regarding substance use. Genetically influenced vulnerabilities—such as high impulsivity and sensation seeking—that underlie one form of social deviance also underlie other forms of social deviance.

In contrast, genetically mediated vulnerabilities to externalizing are largely uncoupled from sexual behavior among African American youth. Specifically, timing of first sexual

intercourse or number of sexual partners has minimal relation on one's liability towards externalizing behavior, and genetic mechanisms underlying externalizing liability do not necessarily manifest through sexual behavior.

Historical Context

In addition to disrupting dominant racial and gendered narratives about Black sexuality as "deviant" behavior, the current findings call into question the validity of a problem behavior framework for research that aims to understand the factors that influence sexual behavior among African American adolescents. Prior to contrasting these racial and gendered narratives with the data from the present study, it will be useful to provide some historical background from which these dominant sexual narratives can be contextualized, and from which the construct of race and its intersectionality with gender can be understood as representing dynamic social processes (rather than a static individual- or group-based characteristic) (Zuberi, Patterson, & Stewart, 2015).

> *"Today, as in 1962, there is a critical need for a deeper understanding of the role of the Afro-American in American history and culture. For it is becoming increasingly evident that Santayana was right when he said that men who cannot remember the past are condemned to repeat it."*
>
> -Lerone Bennett Jr.,
> in the preface to his book *Before the Mayflower* (1968)

The religious doctrine of White colonizers, which equated sexual feelings with sin and threat of eternal damnation, proved problematic for biologically normative feelings of sexual arousal. As the patriarchal structure of society conferred women subordinate to men, personal responsibility for the moral conflict and dissonance posed by feelings of sexual attraction was

expropriated onto the female sex (Rogers, 1966). Consequently, up until the 19th century, women and girls were regarded as immoral temptresses who were sexually impure and untrustworthy.

A confluence of changes during the 19th century, including growing economic prosperity among White property-holding men, a coinciding shift away from the stringent religious teachings of prior generations, and the mass sexual exploitation and commodification of enslaved Black women's bodies through the sexual economy of the slave trade, converged to dramatically transform the image of White female sexuality while further degrading the image of Black female sexuality (hooks, 1981).

In her classic scholarship, *Ain't I a Woman*, bell hooks describes the new White womanhood that emerged in the 19th century:

> The new image of white womanhood was diametrically opposed to the old image. She was depicted as a goddess rather than a sinner; she was virtuous, pure, innocent, not sexual and worldly.… The message of the idealization was this: as long as white women possessed sexual feeling they would be seen as degraded immoral creatures; remove those sexual feelings and they become beings worthy of love, consideration, and respect. Once the white female was mythologized as pure and virtuous, a symbolic Virgin Mary, white men could see her as exempt from negative sexist stereotypes of the female. The price she had to pay was the suppression of natural sexual impulses. Given the strains of endless pregnancies and the hardships of childbirth, it is understandable that 19th century women felt no great attachment to their sexuality and gladly accepted the new, glorified de-sexualized identity white men imposed upon them. (hooks, 1981, p. 81)

Only three to five generations removed for many Americans today, the institution of chattel slavery was also instrumental in constructing dominant sexual narratives about Black womanhood. Despite the inherently coercive nature of sexual encounters between enslavers and their enslaved (Davis, 1981), the common narrative framed by the slaveholders (to include the Christian wives who had to reconcile the conduct of their husbands) promoted the depiction of Black women and girls as lustful, hypersexual, and manipulative "sexual savages" and

"Jezebels" capable of corrupting good Christian men (Smith, 2012; Eaves, 2015). Some enslaved women and girls were also sold into sex trafficking (forced prostitution or forced concubinage), promoting victim-blaming sexual narratives that portrayed Black women and girls as materialistic tramps and prostitutes with insatiable sexual desire. As slaveholders gained economically from the sexual reproduction of enslaved women, forced reproduction (with a partner of the slaveholder's choosing) was commonplace (Foster, 2011; Morgan, 2004). Testimonials from formerly enslaved women (and men) compared their treatment to that of livestock (Eaves, 2015). Sexual narratives stemming from these practices portrayed Black women (and men) as hypersexual, "bestial," "animalistic," and "uncivilized" (Smith, 2012).

During the post-Civil War, Black Reconstruction era, sexual narratives that idealized White womanhood, devalued Black womanhood, and demonized Black manhood gained prominence for their utility as potent mechanisms of social control (in addition to the passage of anti-miscegenation laws) to deter interracial marriage and relationships and to retain the pre-Civil War social order. As such, the degradation of Black women and girls helped to ensure that White men (and White women) would perceive them as morally and sexually loose—not as proper "ladies" suitable for formal relationships or marriage. The myth of the Black male rapist/predator narrative rose to prominence during this period as well, in service of instilling fear that would deter White women and girls from interacting with Black men and boys (Smith, 2012). Through popular media, politics, and material culture, these distorted depictions of Black womanhood have remained salient within American culture and psyche.

During the post-Civil War, post-Black Reconstruction era, Black manhood became further demonized in service of establishing support for America's first iteration of mass incarceration, the convict-lease system (Alexander, 2012). This system, which served to

mitigate the loss of slave labor to the southern economy following emancipation and the start of the Great Migration from the Deep South to the Northeast and Midwest, was accomplished through highly arbitrary and subjective racially enforced laws (e.g., "Black codes," vagrancy, "mischief" and "insulting gestures" laws) that ultimately served to ensure an adequate supply of labor (i.e., "convicts") to meet harvest time demands (Alexander, 2012; Zuberi et al., 2015).

These narratives emphasized the Black man as a predatory threat to White society—one whose supposed laziness and lack of self-constraint made him unfit to handle the responsibilities of freedom, thus necessitating supervision "for his own well-being," the law and moral order of society, and the protection of White women (Eaves, 2015; Zuberi et al., 2015). The effectiveness of this system and its fear-mongering propaganda campaign is apparent from records showing strikingly similar economic productivity in the pre-emancipation South as in the post-emancipation South under the convict-lease system, as well as from seasonal arrest patterns corresponding with harvest time (Blackmon, 2009, and Oshinsky, 1997, as cited in Zuberi et al., 2015).

At the turn of the 20th century, notions of genetic determinism were bolstered by the advent of social statistics and biometrics pioneered by Francis Galton and other prominent statisticians and the application of psychometric testing appropriated from Western Europe (Lombardo, 2011; Zuberi et al., 2015). Both played a central role in providing a scientific veneer to Anglican beliefs about the genetic inferiority of people of color, immigrants from Southern and Eastern Europe, and working-class and impoverished populations broadly (Lombardo, 2011).

Hereditary-based explanations for social stratification would prompt a eugenics movement in the United States that would linger deep into the 1970s (Allen, 1995; Lombardo,

2011). With notable class, gendered, and racial undertones, the first several decades of the movement "emphasized the need to identify and segregate feebleminded people living in the community" (Lombardo, 2011, p.51). The conventional wisdom at the time was that "feebleminded girls were the source of venereal disease and illegitimacy" (Lombardo, 2011, p.51).

An essay series published in the *Atlanta Constitution* during the 1920s reflects reasoning that garnered wide appeal among the middle and upper classes, namely that "money was wasted on trying to educate the defective in schools, yet too little money had been spent to maintain or expand the state facility that would—by quarantining defectives and preventing their mating—represent a step in preventing those costs" (Lombardo, 2011, p.51). A similar sentiment was reflected in Buck v. Bell (1927) which upheld, by an 8-to-1 Supreme Court vote, the state's right to forcibly sterilize individuals deemed unfit to procreate. The majority opinion read, "It is better for all the world, if instead of waiting to execute degenerate offspring for crime, or to let them starve for their imbecility, society can prevent those who are manifestly unfit from continuing their kind … Three generations of imbeciles are enough" (as cited in Lombardo, 2011, p. 21).

Predating formalized problem behavior theories in psychology, renowned biologist and eugenicist Charles Davenport promoted the argument that "sexually immoral people were also afflicted with criminality and feeblemindedness." And "Prostitutes, criminals, and tramps" he claimed, "lacked the genes that allowed modern human beings to control their primitive and antisocial instincts …" (Lombardo, 2011, p. 146).

The outset of the 20th century was also notable for its creation of a separate juvenile justice system for youths. Instrumental in shaping the transition to the new system was the role

of White middle-class Protestant women who, as part of the progressive "child savers" movement, were concerned with what they perceived as the "social evils" of the day, which chiefly referred to the "sexual morality" of young girls and women (Pasko, 2010). In her review on girls' sexuality and the juvenile justice system from its origins to the present, Pasko described the gendered transformations that accompanied the shift to the new system:

> Whereas the first juvenile court originally defined "delinquent" as those under sixteen who had violated a city ordinance or law, when the definition was applied to girls, the court included incorrigibility, associations with immoral persons, vagrancy, frequent attendance at pool halls or saloons, other debauched conduct, and use of profane language in its definition. Ultimately, many of the activities of the early child savers and juvenile courts revolved around monitoring the behavior of young girls, particularly immigrant girls and girls of color, to prevent their straying from the path of sexual purity. (Pasko, 2010, p. 1100)

As such, with the implementation of the juvenile courts, adolescents' sexuality became subject to state control in the form of status offenses for "sexual misconduct" and "sexual immorality." In practice, these charges were nearly exclusively applied to girls, and were defined primarily by having sex as evidenced through routinely ordered gynecological exams or in some cases interrogation of the suspected male partner (Oden & Schlossman, 1991 as cited in Pasko, 2010). During this time, girls were routinely blamed for their own victimization, with psychiatrists even going so far as to deem "weakness for the uniform" as a causal factor in the disproportionate numbers of military men associated with adolescent girls' "sexual immorality" (Pasko, 2010). Despite the severely gendered double standard in enforcement of status offenses for sexual activity, it would not be until the 1974 Juvenile Justice and Delinquency Prevention Act (JJDPA) that the institutionalization of adolescents for status offenses would be prohibited (although with caveats, revisitations and reversals that extend into the present).

Public perceptions of race- and class-based disparities in sexual restraint and irresponsibility were further distorted by the fact that throughout the extended period of legislating female sexuality, middle- and upper-middle-class White women and girls who conceived out of wedlock were routinely sent away under secrecy to maternity homes, where they remained during the visible stages of pregnancy until giving birth and giving up their babies (a decision that was often coerced by parents and maternity home staff). As these homes excluded girls and women of color and were financially prohibitive for poor and working-class Whites, single parenthood and its affiliation with sexual immorality and irresponsibility became further associated with women from these subgroups, as it was quite literally unseen (and often officially undocumented; Geary, 2016) among girls and women whose families had the financial means to cover it up and avoid the shame and stigma of a "wayward" daughter (Lombardo, 2011).

Although eugenic science was debunked following World War II and its rhetoric became socially taboo, the underlying class, racial, and gendered prejudices that propelled its wide support and policy objectives prior to World War II remained largely intact as terminology and rhetoric evolved.

Prior to World War II, forced sterilization focused primarily on first- and second-generation immigrants and institutionalized populations where sterilization often factored into treatment duration, sentencing, and parole decisions (e.g., those deemed mentally ill, certain classes of criminals, the physically handicapped, developmentally disabled, individuals with epilepsy, sexual minorities, and poor and working-class women and girls deemed sexually promiscuous or irresponsible) (Lombardo, 2011).

With the resumption of mass African American migration out of the Deep South (the second Great Migration, 1940–1970, which added westward migration into California), as well as welfare reform policies implemented as a response to the Depression, including Aid to Families with Dependent Children (which included Black Americans starting in the 1960s), the sterilization focus shifted from institutionalized populations to the non-institutionalized poor receiving government assistance. Coinciding with the new focus on the non-institutionalized poor and working class, the hereditarian-focused ideology of the pre-WWII era was supplanted with a "culture of poverty" focus (Lombardo, 2011).

The 1960s–1970s struggle for civil rights and the Black and women's liberation movements faced formidable pushback and an institutionalized backlash that would increasingly come into focus as birth control, welfare reform, and law-and-order policy initiatives (and scandals[5,6]) seeded during the Nixon and Reagan administrations began to take

[5] During the Relf v. Weinberger (1974) Supreme Court sterilization case, an investigation into further sterilization abuses led by Dr. Bernard Rosenfeld of Health Research Group concluded, "It is probable that of the 2 million people who undergo surgical sterilization each year, at least several hundred thousand are considerably less than well informed about the irreversibility, risks and alternative methods of family planning when the[y] 'decide' to have these operations" (Health Research Group, 1973, as cited in Lombardo, 2011, p. 176). This figure is similar to that referenced in the initial opinion in the Relf v. Weinberger sterilization case. US Federal Judge Gerhard Gesell concluded that between 100,000 and 150,000 annual cases of sterilization of poor people had taken place, and in his opinion he criticized the "drift into a policy which has unfathomed implications and which permanently deprives unwilling or immature citizens of their ability to procreate without adequate legal safeguards and a legislative determination of the appropriate standards in light of the general welfare and of individual rights" (Relf v. Weinberger 372 F. Supp 1196 (1974), 1204; Lombardo, 2011 p.180–181).

[6] The national security crimes of the 1980s involving the collateral impact invoked by the CIA complicity in a spin-off scandal from the Iran-Contra crimes, in which CIA and US government officials turned a blind eye to Contra involvement in large-scale cocaine and arms smuggling into the United States to support the overthrow the Sandinista National Liberation Front, which the Reagan administration viewed as a communist threat (Drugs, Law Enforcement and Foreign Policy, a Report of the Senate Committee on Foreign Relations, Subcommittee on Terrorism, Narcotics and International Operations, 1989). In spite of the drug war that had just been declared in 1982, a senate subcommittee investigation discovered, "There was substantial evidence of drug smuggling through the war zones on the part of individual Contras, Contra suppliers, Contra pilots mercenaries who worked with the Contras, and Contra supporters throughout the region…. U.S. officials involved in Central America failed to address the drug issue for fear of jeopardizing the war efforts against Nicaragua…. In each case, one or another agency of the U.S. government had information regarding the involvement either while it was occurring, or immediately thereafter…. Senior U S policy

shape and compound over subsequent administrations and across party lines. The "culture of poverty" focus increasingly assumed racial and gendered undertones, with a new emphasis on "family structure"—a concept propelled into the mainstream by the widely publicized Moynihan Report, published in 1965 and heavily recirculated in the 1980s (Geary, 2015), originally titled "The Negro American Family: The Case for National Action."

Described by civil rights leader James Farmer as "the most serious threat to the ultimate freedom of American Negroes to appear in print in recent memory" (Geary, 2015, p.95), the Moynihan Report—the objective, intent, and impact of which continues to be debated—would focus extensively on Black male unemployment and out-of-wedlock birth rates among working-class Black women. Conflating Black single-mother, working-class households with a "black matriarchal family structure," Moynihan, an advisor in the Johnson administration (and thereafter the Nixon administration), contended that Black family structure lay at the crux of what he termed a self-perpetuating "tangle of pathology" afflicting working class Black Americans (Moynihan, 1965).

Asserting that Black population growth, especially among working-class Blacks, "must inevitably lead to an unconcealable crisis in Negro unemployment," Moynihan's report effectively enabled narratives about "black family structure" to overshadow the realities of mass deindustrialization and the tangled web of interpersonal and institutional racial discrimination that compounded the impact on Black Americans. That is, the erosion of income- and property-

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makers were not immune to the idea that drug money was a perfect solution to the Contras' funding problems." (Drugs, Law Enforcement and Foreign Policy, a Report of the Senate Committee on Foreign Relations, Subcommittee on Terrorism, Narcotics and International Operations, 1989). The downstream distribution networks from the large-scale cocaine trafficking during the 1980s disproportionately impacted racially isolated Black and Brown communities with high concentrated poverty, most notoriously South-Central Los Angeles and Compton, CA, but also pockets within large cities elsewhere in the nation including New York, Miami, New Orleans, and beyond; the same communities then disproportionately targeted in the war on drugs (Tonry, 1995; Nunn, 2002). (Gary Webb, San Jose Mercury News, "Dark Alliance" series, August 1996; Weinberg, Steve [Nov 17, 1996]).

tax revenue for basic public infrastructure and services that followed widescale closures, outsourcing, and relocations in manufacturing (which markedly escalated in the 1970s and 1980s) was compounded by housing discrimination, White flight/suburbanization, political negligence, and limited reserves of wealth, effectively segregating large swaths of Black Americans into isolated pockets of concentrated poverty (Taylor, 2016).

As overt displays of racial discrimination and prejudice became increasingly taboo in the decades following Civil Rights legislation, including anti-discrimination laws and the formal abolishment of Jim Crow era "separate but equal" laws, the White establishment would also adapt. Throughout the 1980s and 1990s, racialized imagery pairing Black women with welfare and the quintessential image of the "welfare queen" became widespread.

The "welfare queen" assumed the long-standing sexist and White supremacist narratives associated with Black female sexuality. She was portrayed as sexually promiscuous, irresponsible, materialistic, manipulative, and lacking in self-constraint. She was also portrayed as demasculinizing and domineering—a contrast to the dominant sexual narratives depicting White women as passive. The effectiveness of the "welfare queen" mythology is suggested by the swell of public support for sweeping welfare reforms and cuts to public services throughout the 1980s and 1990s.

Likewise, beginning in the 1980s racialized imagery pairing Black males with crack cocaine and the quintessential image of the "drug addict" (and drug dealer) became widespread, fostering a swell of public support for "tough on crime" drug laws that would fuel America's second iteration of mass incarceration through a racially enforced War on Drugs (Tonry, 1995; Alexander, 2012).

As in the drug war in the 1920s that coincided with prohibition, the 1980s and '90s "drug addict" assumed the longstanding White supremacist narratives associated with Black manhood. As such, the new "drug addict" was portrayed as a predatory threat to society, whose poor choices, preference for instant gratification, and failure to take responsibility for family and employment obligations justified a need for control over the law and order of society and public safety. The effectiveness of this system and its racialized propaganda campaign is apparent from records showing dramatically disproportionate policing, enforcement, and incarceration of Blacks and Latinos for drug-related infractions despite committing drug offenses at rates no greater (and in some cases less) than White Americans (Tonry, 1995; Nunn, 2002; Alexander, 2012).

Amid the drug war, unprecedented overhauls were also made to the juvenile justice system beginning in the 1990s, making it easier to try and sentence minors as adults and retain minors in secure detention for status offenses (Moriearty & Carson, 2012). As it pertains to status offenses, girls continue to be disproportionately represented relative to their representation within the juvenile justice system for criminal offenses (Pasko, 2010). And from 2003 to 2013, secure confinement has only grown for African American and Native girls, who were already disproportionately represented among girls in confinement (Rovner, Sentencing Project, 2016).

In her comprehensive review of girls' sexuality as conceptualized throughout the last 100 years within the juvenile justice system, Pasko (2010) concludes that today, "the correctional focus—through one definition or another—continues to be on girls' sexual behavior as cause for legal response, detention, and commitment" (p. 1129). She further notes:

> There still exist the underlying assumptions that girls are sexually manipulative and that there is a singular accepted sexual path for young women to take: heterosexual propriety. When a girl deviates from such a path, the source of the problem lies with her flawed choices, damaged personality, and inability to take responsibility, rather than the structural conditions that shape her life or the men who are counterparts in such activities. Such inability to control sexual impulses and to avoid risky sexual behavior is often viewed as cause for further detention and commitment. (Pasko, 2010, p.1116)

A shift from moralization to medicalization has also taken place in contemporary times. As such, girls' sexual behavior is often conceptualized as symptomology or a marker of a broader syndrome of behaviors—substance use, impulsivity, deceitfulness, irresponsibility—that lead to "poor choices." The influence of problem behavior ideology seems apparent.

Although the professionals and juvenile justice officials interviewed by Pasko (2010) often acknowledged girls' complex histories, including prior trauma, such factors were not integrated into the conceptualization of current challenges. Rather, decontextualized "concepts of choice and responsibility" prevailed, and "similar to the early eras that concentrated on immigrant girls, such inability to take responsibility was racialized" (p.1116).

The problem behavior ideology and continued focus on "the control and micro-management of girls' bodies and sexuality" (p.1129), is reflected in many of the statements from Pasko's (2010) interviewees, including the following excerpt from one therapist:

> All of our girls are on birth control. Because if they do weekend furloughs, we cannot have them getting pregnant. Even if they say they are gay, who knows what they will get into. We have them sign forms that they will not have sex or do drugs or drink and when they get back after the weekend, we give them drug and pregnancy tests and occasionally do [gynecological] exams. If they want a furlough, they have to agree to this. These girls can be very manipulative and while we do want to trust them, well, having birth control, tests, gyno exam … they know they cannot get away with it. We have to have these measures of control. (Pasko, 2010, p.1119)

Another interviewee reflected similar attitudes, stating: “We don’t want any incidents and when you give girls some freedom, with freedom comes mistakes and bad choices, and well, incidents” (p. 1128).

As this abbreviated history illustrates, racialized and gendered sexual narratives have been monopolized by those who hold positions of power within society, with the means to project and institutionalize their voice and will as truth and prescribe the parameters of normality. In the United States, this has traditionally been the purview of White propertied men. However, as it pertains to female sexual propriety, White middle- and upper-class Christian women have been complicit in promoting these narratives as well, and have been instrumental in institutionalizing female sexual purity as “normative” and departures as “wayward.” Thus, through the systems and institutions to which middle- and upper-class Whites have been exclusively privy, they have been conferred with the power to define the sexual narrative of the “other”—including people of color, working-class White women, sexual minorities, and adolescents.

What becomes increasingly clear when viewed all together is that despite the evolution of terminology and constructs, the same racial, class, and gendered sexual narratives continue to undergird successive waves of fear-mongering that galvanize support for enhanced systems of social control that seek solution through separation—the removal of an “other” from an “us.”

Disrupting Racialized Narratives about Deviance and Unconstrained Sexuality

There is a long-standing history linking female sexual behavior with deviance and portraying (White) male sexual behavior as normative, “boys will be boys” behavior. The fact that sexual stereotypes that characterize White male sexuality are less prominent than those of other intersecting identities might reflect the very sexual agency, institutional power, and

gender and racial privilege that has historically characterized White heterosexual male sexuality. As it pertains to the present study, this comparative lack of external constraints imposed on the expression of one's sexuality would be consistent with a closer correspondence between one's level of externalizing and the timing of first sexual intercourse and number of sexual partners. This is the pattern that emerged among White boys in the present study, with common genetic factors underlying the overlap between externalizing and sexual behavior.

For White girls, a broadly similar pattern emerged as for White boys, except for less total overlap between externalizing and number of sexual partners for White girls. Overall, this pattern of results for White girls may serve to signal the greater sexual agency that White adolescent girls have experienced in more recent decades—a by-product, perhaps, of the greater representation of White middle-class women within the women's liberation movement of the 1960s.

Nevertheless, there was still a notable gendered pattern to the findings wherein for *all* girls, shared environmental variance accounted for a portion of the overlap between externalizing and number of sexual partners, and greater externalizing was less strongly linked to number of sexual partners. One possibility to explain this pattern of results is that the societal norms that led to the gendered enforcement of status offenses and compelled White middle- and upper-class families to send their daughters away at great financial cost and secrecy to spare them the shame of unwed motherhood have likely played a role in transmitting parental anxieties about daughters' sexual activity in particular. These worries in turn might lead to more protective and restrictive parenting approaches with girls, which would be consistent with data showing that parents tend to provide closer supervision and tracking of whereabouts for girls relative to boys (e.g., Block, 1979; Morrongiello & Dawber, 1998; Morrongiello et al., 2006). These additional

constraints on girls' behavior would be consistent with shared environmental variance playing a role in the overlap between externalizing and number of sexual partners for girls, but not boys, as well as the pattern in which it was greatest at higher levels of SES, where resources to secure supervision are more readily available.

Conversely, correspondingly lower parental supervision would be consistent with an exclusively genetically mediated correspondence between externalizing and number of sexual partners, the pattern that emerged overall for boys (albeit with less total overlap between externalizing and number of sexual partners for African American boys). Within the context of a patriarchal society, it is possible that this pattern might reflect the greater sexual agency and independence that our culture confers to the male gender, which generally encourages the expression of male heterosexual sexuality.

As reviewed earlier, dominant narratives have depicted African American men and boys as behaviorally deviant and sexually unconstrained. The data from the current study do not accord with the long-standing narratives imposed on Black male sexuality, nor do they conform well with the expectations of a problem behavior framework.

Overall, African American boys exhibited substantially lower externalizing on average relative to Caucasian boys. The magnitude of the overlap between externalizing and both sexual outcomes for African American boys was also modest overall, consistent with a general uncoupling between externalizing disposition and sexual behavior—a pattern of findings counter to predictions of a problem behavior model. The variance in sexual behavior unique from externalizing was sizable and attributed to both shared and non-shared environmental factors. Since non-shared environmental variance also includes measurement error, we focus on

the possible interpretations of the shared environmental variance underlying each sexual phenotype.

Given the phenotypic pattern of earlier age at first sexual intercourse and one greater partner on average at the aggregate, potential explanatory mechanisms for this pattern of findings among African American boys might be informed by the unique convergence of patriarchy and racial oppression.

Within American society, Black males have been subject to a long-standing history of exclusion from avenues of power and status that have been afforded to White males. As such, within the context of a racially oppressive and patriarchal milieu, heterosexual male sexuality may constitute one of the few remaining avenues in which displays of dominance and patriarchal entitlement to male privilege might be exercised or affirmed. To the extent that these dynamics might explain expressions of sexuality relevant to timing of first intercourse or number of sexual partners, such mechanisms would be consistent with a decoupling of externalizing disposition from sexual behavior, as well as shared environmental mechanisms accounting for the variation unique to timing of sexual intercourse and unique to number of sexual partners.

This is but one of several potential explanations. We expand on more general possibilities in the subsequent section. What appears to be clearer is that the pattern that arises for African American boys from the current data does not accord with problem behavior models (nor the implications of dominant sexual narratives) that have paired behavioral deviance and impulsivity with sexual behavior, linked through common genetic mechanisms. Specifically, although the overlap between externalizing and number of sexual partners could be attributed to common genetic variance, the degree of overlap between EXT and number of sexual partners

was only modest, explaining minimal variation in number of sexual partners. The overlap between EXT and age at first sexual intercourse was only modest as well and was accounted for by shared environment mechanisms, not common genetic mechanisms as would be predicted by a problem behavior model. Finally, for each sexual phenotype, the majority of the variation was explained by environmentally mediated mechanisms wholly distinct from externalizing. Together, these findings call into question the validity of a problem behavior model for conceptualizing sexual behavior among African American boys.

African American women and girls have also been subject to longstanding sexual narratives that have devalued their personhood, depicting them as morally loose and sexually unrestrained (e.g., Wilson & Huntington, 2006; Smith, 2012; hooks, 1981)—stereotypes that might appear to suggest heightened sexual autonomy or liberation. The data depict a starkly distinct portrait, disrupting the dominant sexual narratives that continue to be imposed on Black female sexuality and casting doubt on the validity of a problem behavior framework for conceptualizing sexual behavior among African American girls.

Overall, African American girls exhibited substantially lower externalizing on average relative to Caucasian girls (and Caucasian boys). African American girls reported earlier average AFS relative to Caucasian girls, and a comparable number of partners. The magnitude of the overlap between externalizing and sexual outcomes for African American girls was modest overall, consistent with a general uncoupling between externalizing disposition and sexual behavior—a pattern counter to predictions of a problem behavior model. Furthermore, to the extent that externalizing and sexual behavior did show modest overlap it was accounted for by shared environmental mechanisms—a pattern counter to the problem behavior model, which

posits a common genetically based “syndrome” linking behavioral deviance and early sexual activity.

One potential mechanism to account for the shared environmental variance linking externalizing and sexual behavior might be related to the fact that African American girls have traditionally received strong messages from within their religious and spiritual communities, reinforced by their elders, concerning sexual abstinence and discouragement from alcohol and drug use (e.g., Meier, 2002; Miller et al., 1997). To the extent that family-level factors such as these might serve to dissuade young girls from engaging in externalizing spectrum behaviors while also promoting delayed sexual activity, this would be consistent with shared environmental variance underlying externalizing and sexual behavior among African American girls. However, there is also substantial intra-ethnic variability in religiosity and spirituality among African American girls, and this mechanism would not inform the finding of earlier average timing of first sexual intercourse.

Additional Candidate Mechanisms

In addition to the possibilities considered so far, the results from the current study are complex and are likely to be explained by multiple mechanisms. One possibility to account for the genetic mediation between externalizing and AFS is that genetic influences on pubertal timing might be one mechanism underlying individual differences in susceptibility to both externalizing behaviors and earlier sexual behavior.

Specifically, as earlier age at pubertal onset would be expected to correspond with less advanced maturation of the cognitive control systems governing premeditation and constraint, then the normative increases in reward sensitivity, sensation seeking, peer salience, and interest in sex that accompany neurobiological and hormonal changes of pubertal onset (Kretsch &

Harden, 2013; Forbes et al., 2010; Martin, Kelly, Rayens, Brogli, Brenzel et al., 2002; Steinberg et al., 2008) would be expected to increase susceptibility to both externalizing behaviors and earlier sexual behavior among early onset adolescents relative to their same-aged peers whose pubertal onset occurs at a later age (see review in Smith et al., 2013). This would be potentially consistent with common genetic mechanisms underlying the association between externalizing and sexual behavior—the pattern that emerged most clearly among Caucasian adolescents.

If this is the case, the remaining question is how this process might be disrupted or distinct among African American adolescents who showed less overall coupling between externalizing and sexual behavior and greater shared environmental versus genetic mediation underlying the coupling that did emerge, particularly among African American girls.

One explanatory mechanism might be informed by previous research conducted with the Add Health sample which found that later pubertal timing was predictive of later sexual intercourse among Caucasian girls but not among African American girls (Cavanagh, 2004). This suggests that, at least among African American girls, genetically mediated variation in pubertal timing would be uncorrelated with phenotypic differences in timing of first sexual intercourse. To the extent that genetic variation underlying age at first sexual intercourse might be accounted for by its correspondence with genetic variation in pubertal timing, then it might be expected that heritability in age at first sex would be suppressed among African American girls. This would be broadly consistent with multivariate results from our previous work with the Add Health data in which heritability of AFS was suppressed among African American and Latinx adolescents (and White adolescents from lower SES backgrounds; Carlson et al., 2014).

Thus, if genetic influences underlying timing of first sexual intercourse are suppressed among African American adolescents, then genetic variation underlying externalizing could not

account for any genetic variation underlying age at first sexual intercourse. This possibility would appear to be consistent with the present pattern of findings for AFS among African American adolescents. The uncoupling between externalizing and sexual behavior among African American girls would also be consistent with prior research findings in which impulsivity, a key component of externalizing and risky behavior more broadly, did *not* predict early sexual intercourse for African American girls (Hipwell, Keenan, Loeber, & Battista, 2010), as well as findings from a mixed-sex sample of African American teens wherein sexual intercourse was not associated with either substance use or delinquency (Black et al., 1997).

One implication of an uncoupling between pubertal timing and AFS, and specifically in the case of later pubertal timing and earlier AFS, would be that the neurobiological and hormonal aspects of pubertal onset that are posited to increase individual motivation to engage in sexual behavior (e.g., elevations in sensation seeking, reward sensitivity, greater salience of peers and sex) would be less salient prior to pubertal onset. In this case, the relevant question becomes, What sorts of factors might override individual differences in pubertal onset to account for earlier average age at first sexual intercourse among African American adolescents?

One possibility might be related to the impact of the dominant sexual narratives and stereotypes about Black sexuality that pervade mainstream American media in shaping younger Black adolescents' perceptions about the sexual experience/norms among their intra-racial peers.

The impact of perceptions about peer behaviors/norms in shaping behavior has precedent in the substance use literature wherein college student misperceptions (overestimations) of peer drinking norms—ascribed to media portrayals of exaggerated college drinking—have been shown to increase alcohol consumption among college students, due to desire to "fit in" and adopt normal behavior (e.g., Hingson & White, 2012; Perkins, Haines, & Rice, 2005; Perkins,

1997). Similar mechanisms have been posited for adolescent sexual behavior, with evidence suggesting that younger adolescents are more prone to overestimating the sexual experience of their peers (e.g., Kinsman, Romer, Furstenberg, & Schwarz, 1998; Rogers & Rowe, 1993; Zimmer-Gembeck & Helfand, 2007).

Applied to the present study, such mechanisms could be consistent with both an uncoupling pattern between externalizing and sexual behavior and greater shared environmental mechanisms underlying variation in sexual behavior unique from externalizing—the same patterns that emerged among African American adolescents. This mechanism could also potentially account for the phenotypic pattern of earlier average AFS among African American adolescents and the higher average Wave 1 reports for sexual partners among African American boys, the subset of adolescents for whom manhood is often depicted in mainstream media through the narrow terms of female sexual conquest.

Of further consideration, the uncoupling between pubertal onset and onset of sexual behavior also raises questions about the possibility of coercive sexual encounters. Childhood sexual abuse has been robustly linked to earlier first intercourse, more sexual partners, less consistent use of condoms (e.g., Beitchman et al., 1992; Kendall-Tackett, Williams, & Finkelhor, 1993; Senn et al., 2008; Trickett & Putnam, 1998), and elevated risk for STI contraction and unintended pregnancy (e.g., Clum et al., 2009; Mugavero, et al., 2007; Senn et al., 2008). Moreover, childhood sexual abuse also places female adolescents at heightened risk for physical and sexual re-victimization (see Classen, Palesh, & Aggarwal, 2005 for a review), greater severity of later victimizations, and victimizations perpetrated by non-peers (i.e., individuals four or more years older than victims) (Barnes, Noll, Putnam, & Trickett, 2009; Boney-McCoy &

Finkelhor, 1995a; Gidycz, Coble, Latham, & Layman, 1993; Krahe, Scheinberger-Olwig, Waizenhofer, & Kolpin, 1999; Wekerle & Avgoustis, 2003).

Of further relevance to the present study, across gender/sex and clinical/nonclinical samples, CSA has also been linked to a broad range of psychological and affective disturbance, with some research suggesting that women may be more likely to develop a broader array of internalizing symptoms, while men may be more likely to develop substance use disorders (Stein, Golding, Siegel, Burnam, & Sorenson, 1988) and externalized aggression including rage and attempts to reassert their masculinity, which can include displays of hypermasculinity (Kaufman, 1984).

In light of these considerations, we conducted a set of follow up phenotypic analyses on the prevalence of childhood sexual abuse partitioned by race/ethnicity, biological sex, and zygosity to examine whether CSA might be a promising mechanism to make sense of our results. For these analyses we drew on data from Waves 3 and 4 and operationalized CSA as any affirmative endorsement at either wave (ever = 1, never = 0) to the question, "How often had one of your parents or other adult care-givers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?" These results are displayed in Table 2.6.

Table 2.6 Prevalence of childhood sexual abuse endorsed by study participants at wave 3 by zygosity, race/ethnicity, and biological sex

	MZ	DZ	FS
Caucasian Adolescents			
Girls	3.8%	3.8%	9.1%
Boys	0.0%	1.0%	6.1%
African American Adolescents			
Girls	14.0%	9.3%	14.0%
Boys	11.1%	18.4%	4.0%

Note. **MZ = monozygotic twins, DZ = dizygotic twins, FS = full biological non-twin siblings (same sex).**

Overall, the greater prevalence of sexual abuse among African American adolescents[7] relative to Caucasian adolescents, and among Caucasian girls relative to Caucasian boys, suggests that childhood sexual abuse merits further consideration as a potentially relevant mechanism for both the potential uncoupling between genetic variance in externalizing and sexual behavior, as well as an environmental mechanism which might jointly impact externalizing and sexual behavior and/or impact sexual behavior independent from externalizing.

A final consideration, given increasing evidence that early sexual abuse often co-occurs with multiple other forms of stress and adversity (Cook et al., 2005; DeJong, 2010; Dong et al., 2004; Finklehor, Turner, Ormrod, & Hamby, 2009; Streeck-Fischer & van der Kolk, 2000), is the potential influence of trauma exposure on sexual behavior more broadly. A recent study may shed important mechanistic clues about the role of traumatic stress exposures on the sexual behavior of young women. This study examined the link between post-traumatic stress and sexual risk behavior within a sample of sexually active African American undergraduate women. Consistent with findings from the broader literature, post-traumatic stress was linked to higher number of sexual partners and greater prevalence of unprotected vaginal intercourse and intercourse under the influence of a substance. This study further found that sexual compulsivity and sensation seeking were unrelated to post-traumatic stress among this sample of young women, suggestive perhaps of an uncoupling between externalizing and sexual behavior.

[7] The estimates for African American boys may be unstable due to disproportionate sample attrition among African American boys by Waves 3 and 4 when the sexual abuse questions were administered (Mullan-Harris, 2013).

Instead, post-traumatic stress was strongly associated with lower perceived sexual control. Furthermore, lower perceived sexual control was associated with higher frequency of unprotected sex and sex under the influence of substances (Munroe et al., 2010). This could be consistent with common environmental mechanisms undergirding the link between EXT behaviors such as substance use and sexual behavior, as well as unique environmental mechanisms accounting for variation in sexual behavior that is independent from EXT.

Taken together, possibilities such as these would be consistent with earlier sexual intercourse and more sexual partners while at the same time seriously calling into question the assumed sexual agency, intrinsic preference, and unfettered choice that the widespread stereotypes and risk-based language that commonly characterize early sexual activity—especially among girls of color—would imply.

Further Considerations

On the other hand, we do not wish to imply a uniform lack of agency among the sexual expression of adolescent girls. Indeed, sexual activity including first sexual intercourse is a normative part of adolescence (Carver, Joyner, & Udry, 2004). Furthermore, AFS between ages 16 and 18 is associated with markers of social adjustment and school connectedness (Zimmer-Gembeck & Helfand, 2007). And within the context of a long-term monogamous relationship, adolescent sexual activity has also been linked with lower levels of delinquency (Harden et al., 2011).

Additionally, there is an inherent classism in reproductive messages that define waiting for motherhood until after completing higher education and establishing one's career as the single ideal or socially responsible course of action. This notion is powerfully affirmed in SmithBattle's (2013) discussion of Geronimus' (2003) idea that: "Wide disparities in status and

power insulate professionals from the circumstances that predispose to early parenting and reinforce erroneous assumptions that middle-class norms regarding family formation and parenting are 'natural' rather than contingent upon resources and opportunities that are largely unavailable to disadvantaged groups" (p. 237). Moreover, delaying reproductive timing until higher education is completed and career goals are stabilized might be conceived as a sexual risk behavior itself, as this can pose its own problems for childbearing, as evidenced by the recent explosion in IVF offerings for upper-middle-class professionals.

Relatedly, another possible explanation for the pattern of results among African American adolescents is that earlier sexual activity may not be uniformly considered a "deviant" behavior (Edin & Kefalas, 2005). In fact, early initiation of reproductive behavior, in conjunction with multigenerational family structures, may be adaptive in some social contexts, particularly high-poverty, politically marginalized neighborhoods. Among African American women, who experience declines in physical health even in their 20s and 30s, teenage childbearing is associated with benefits to infant health (as measured by birth weight and mortality rates) compared to childbearing that is delayed into adulthood (Geronimus, 2003). Put slightly differently, race/ethnic minority youth who live in socially and politically disenfranchised contexts may be shaped by the structure or impact of these same environments to initiate sexual intercourse earlier. Thus, genetically mediated factors that may be relevant in shaping the types of behaviors that tend to correlate with greater violation of social norms may not be manifest in earlier sexual intercourse among African American adolescents for whom earlier sexual activity may hold a qualitatively distinct significance relative to adolescents from racially privileged White middle-class backgrounds.

Adolescent Sexual Behavior and the Problem Behavior Perspective

Although the findings for Caucasian adolescents were broadly consistent with the predictions of a problem behavior perspective, the merits of a problem behavior framework may benefit from further scrutiny for several reasons.

First, even among Caucasian adolescents who conformed most closely with the predictions posited by a problem behavior theory, the overlap between externalizing and each of the sexual phenotypes was still only moderate at best. Indeed, for all adolescents, irrespective of race/ethnicity and gender, the majority of variance in each sexual behavior was accounted for by factors unique from externalizing. Although some of this is undoubtedly measurement error (and as such we did not interpret the E component underlying variance in sexual behavior unique from EXT) there is substantive theoretical precedent for expecting that mechanisms apart from EXT would also account for variance in sexual behavior (e.g., Leigh, 1989; Hill & Preston, 1996; Buss & Shackelford, 1997; Buss, 2003). In fact, Meston & Buss (2007) collected 237 reasons for why people have sex and developed hierarchical taxonomy of motivations comprising four broad factors (Emotional, Physical, Goal Attainment, Insecurity) and 13 subfactors (e.g., stress reduction, pleasure, social status, duty/pressure, mate guarding, love/commitment). This suggests that additional mechanisms independent from a problem behavior perspective (and externalizing) merit consideration for a more complete understanding of adolescent sexual health and behavior.

Second, although common genetic mechanisms accounting for both externalizing spectrum behaviors and adolescent sexual behavior align with the predictions of a problem behavior model, this concordance does not preclude the possibility that the mechanistic processes that link these behaviors might be wholly distinct from an underlying “syndrome”

posited by the original problem behavior framework (Jessor & Jessor, 1977). Indeed, the genetically based and developmentally normative mechanistic process that we propose herein could serve as one such example.

Third, problem behavior theory offers no clear explanatory framework from which the present pattern of moderation can be clearly conceptualized. It makes no a priori prediction about the role of contextual mechanisms in disrupting the putative genetically based "syndrome" from manifesting phenotypically.

Fourth, from a conceptual standpoint, there is nothing inherently "deviant" about adolescent sexual behavior; it is in fact statistically normative (Guttmacher Institute, 2006), and within certain relationship contexts even linked with lower delinquency and social adjustment (Harden et al., 2011). Moreover, as suggested by the work of Meston & Buss (2007) highlighted above, sexual behavior is likely to be influenced by a complex array of social and motivational factors.

Thus, it is possible that the problem behavior conceptualization itself may be problematic—a by-product of the racial, gendered, class, and Puritan-based narratives that have persisted overtime and in service not so much of describing sexual behavior for the purposes of health, understanding, and individual empowerment, but perhaps more so in service of controlling it and controlling others.

Conclusion

We examined the genetic and environmental links between EXT and adolescent sexual behavior to see whether results observed using predominantly Caucasian middle-class adolescents extend to African American adolescents. Although EXT was correlated with AFS

and number of sexual partners across all groups of youth, the mechanisms underlying these associations were distinct between Caucasian racial majority and African American racial minority youth. Genetic variation in EXT spectrum behaviors—including fighting, delinquency, drinking, smoking, and drug use—correspond to earlier AFS but only for Caucasian adolescents. And in the case of number of sexual partners, this genetically mediated link extended to Caucasian boys and to a lesser extent Caucasian girls and African American boys. Genes did not mediate this link for African American girls; rather, shared environmental factors did. Finally, for African American boys and African American girls, significant variation in sexual behavior was also accounted for by shared environmental factors unique from EXT.

These results are consistent with epidemiological and sociological studies that suggest problem behavior models may not adequately explain individual differences in sexual activity in African American adolescents. Future research should seek to identify the ways in which intersecting systems of power, privilege, oppression and identity serve to impact and maintain sexual health disparities and contribute to the destructive sexual narratives that continue to shape policy and the lens through which we see young people.

Study 3: Early Chronic Stress and Trauma in Association with Adolescent Sexual Health and Global and Specific Externalizing Spectrum Behaviors[8]

During adolescence, rates of morbidity and mortality increase by 200% (Forbes & Dahl, 2010; CDC 2009). Behavioral risk-taking, which increases at puberty and throughout adolescence, is posited to be a central mechanism. Consistent with this idea, adolescence is associated with substantial increases in rates of accidents, homicide, suicide, depression, substance abuse, eating disorders, sexually transmitted infection, and unintended pregnancy (Force, 1996; Ozer et al., 2002; Resnick et al., 1997). Aside from "risk taking," many of these behaviors are linked to emotional distress, including pervasive feelings of anger, inadequacy, shame, fear, uncertainty, helplessness and hopelessness—hallmark indicators of early chronic stress and trauma.

The teenage years can be both exciting and challenging to navigate. On the one hand it is a pivotal period of developmental transitions including pubertal maturation and increased orientation to peers, yet on the other hand, adolescents continue to lack the full rights, autonomy, and status of legal adults. Frustration stemming from this *maturity gap* has been posited as a key mechanism undergirding developmentally normative increases in externalizing behaviors such as substance use and delinquency during adolescence (Moffitt, 1993).

For adolescents whose developmental histories have included multiple, prolonged, and severe forms of chronic stress and trauma, this pivotal stage of development may be particularly trying. Indeed, early chronic stress (including poverty, violence exposure, and mistreatment) is a risk factor for a range of adverse health and psychosocial outcomes across the life-span (Davidson, Hughes, Blazer, & George, 1991; Davidson, 2001). During adolescence, stress is

[8] This study has not been published yet. I am the sole author on this study.

highly correlated with a variety of externalizing spectrum behaviors including fighting, delinquency, and substance abuse, as well as with sexual behaviors including earlier first sexual intercourse and more sexual partners.

In spite of these links, the extent to which chronic stress and trauma is linked to adolescent externalizing broadly—that is, the co-occurrence of multiple forms of disinhibited behaviors—or to specific types of behaviors, such as fighting or substance abuse, independent from a global externalizing pattern, is unclear. Moreover, the extent to which early exposure to chronic stress and trauma may exert a causal role in the emergence of more pronounced externalizing spectrum psychopathology during the adolescent period is also unclear.

This is a key question, because more severe presentations of externalizing underlie a range of diagnoses that co-occur across development, including attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), learning disability diagnoses, anxiety, disruptive mood dysregulation disorder (DMDD), depression, conduct disorder (CD), antisocial personality disorder (ASPD), and borderline personality disorder (BPD) (reviewed in van der Kolk et al., 2005). To be sure, the rate of clustering among these disorders exceeds the expectation given their individual base rates (e.g., Krueger et al., 2002, 2005; van der Kolk et al., 2005). One possibility that remains under-explored is that there is in fact a causal link between exposure to multiple types of severe or persistent stress and trauma exposures over the course of development (infancy into childhood) and adolescent externalizing behaviors that exceed the developmentally normative range. To the extent that early chronic stress and trauma may account for more severe presentations of externalizing during adolescence, trauma-informed interventions may hold the key to helping children, adolescents, and adults with a range of behavioral patterns that have been traditionally under-recognized as indicators or adaptations to

chronic or severe traumatic stress and classified as constitutionally based co-occurring characterological, impulse, attentional, oppositional, and antisocial disorders and deficits.

Post-Traumatic Stress Disorder: A Historical Context

Commonly linked to war, the concept of stress-related syndromes dates back several centuries. In the advent of World Wars I and II, terms such as "shell shock," "combat stress," and "battle fatigue" became common nomenclature to reference the patterns of stress-related maladjustment among combat veterans returning from deployment. In spite of the emergent patterns rendered by these large-scale exposures to horrific events, the notion that an external event could have a role in the onset of a psychological disorder remained controversial. And it was not academics, but rather Vietnam veterans, service members' families, and a few psychoanalysts who championed the need for a diagnosis that linked symptoms to a set of certain types of traumatizing experiences such as war (van der Kolk & Najavits, 2013). In spite of this intense outside pressure, post-traumatic stress disorder (PTSD) did not become an official diagnosis until 1980, when it was introduced in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III). In this iteration PSTD was classified as an anxiety disorder and conceived as a fear-based response to a stressor of magnitude that exceeded the range of our adaptive capacity to cope. In 1994, DSM-IV criteria emphasized exposure to a catastrophic event in conjunction with symptom endorsement from three symptom clusters (intrusive recollections, hyperarousal, and numbing/avoidance) along with duration and functional impairment criteria.

In 2013, DSM-5 introduced several evidence-based changes to PTSD diagnostic criteria. It was reclassified from an anxiety disorder to a trauma- and stress-related disorder and symptom criteria were expanded to recognize the prominence of dysphoric and anhedonic symptom

presentations. Additionally, a preschool subtype for children under six was added with avoidance-related symptom criteria relaxed as deemed developmentally appropriate. Despite these accommodations, the application of PTSD criteria to adolescents has been criticized for inadequately capturing the complete scope of traumatic stress symptomology exhibited during this stage of development (e.g., Cook et al., 2005; Herman, 1992; Garvert, Brewin, Bryant, & Maercker, 2013; Terren-Sweeny, 2013; van der Kolk et al., 2005; van der Kolk & Najavits, 2013).

Beyond PTSD: The Complexity of Early Chronic Stress and Trauma on Post-Traumatic Stress Response

One implication of the centrality of combat veterans in shaping original formulations of PTSD is that our broad notions of this disorder have been fundamentally informed by traumatic stress reactions in which both the index event and onset of stress response occur during adulthood. This is a key consideration since developmental timing is relevant for understanding and predicting the impact of stress over the course of development. For instance, prenatal stress affects the development of the hypothalamic–pituitary–adrenal (HPA) system (Lupien, McEwen, Gunnar, & Heim, 2009) such that it is adaptively upregulated to equip the organism to be highly perceptive to early signs of stress in its environment. Postnatal early childhood stress, in contrast, exerts its effects most prominently on the hippocampus (Lupien et al., 2009). And the prefrontal cortex, a brain region involved in regulation of cognitive and emotional processes, is particularly sensitive to stress across later childhood to mid-adolescence (ages 7–16) (Sowell et al., 1999). Additionally, the impact of prolonged or severe trauma exposure in childhood may not emerge phenotypically until later adolescence after formation of synaptic connections and pruning has stabilized relative to early childhood. Indeed, this could be the mechanism underlying findings

that severity of traumatic stress symptoms secondary to childhood trauma prominently increase over the course of adolescence (Denton, Frogley, Jackson, John, & Querstret, 2016).

The interplay of developmental timing, severity, and duration of trauma exposure are key predictors of impairment and distress as well as symptom sequelae beyond PTSD (e.g., Pearlman, 2001; Terr, 1991; Andersen, 2003; van der Kolk & Courtois, 2005). Among individuals with extensive trauma in their backgrounds, there is evidence for substantive departures in symptom profile from prototypical PTSD to include relational, affective, and self-concept components (Cloitre et al., 2011; Cloitre, Garvert, Brewin, Bryant & Maercker, 2013; Elklit, Hyland, & Shevlin, 2014; Roth, Newman, Pelovitz, van der Kolk, & Mandel, 1997; van der Kolk et al., 1996). These response profiles may reflect the complexity of psychological sequelae when chronic stress and trauma meet the attachment system (van der Kolk & Najavits, 2013; Tarren-Sweeny, 2013).

Consistent with this idea, Ahmad, Sundelin-Wahlsten, Sofi, Qahar, and Knorring (2000) examined the psychometric properties of PTSD and other post-trauma symptoms in childhood and found that among the children whose trauma events were categorized as *least severe*, the intercorrelations between PTSD items were greater than the intercorrelations among the non-PTSD-related items. In contrast, among the children who had experienced the most frequent, chronic, and severe traumatic events, there was no distinction in intercorrelations between PTSD and other post-traumatic symptoms in childhood. These authors concluded that a broader range of symptoms beyond the "classic" PTSD symptoms should be used when investigating childhood trauma and post-traumatic psychopathology (Ahmad et al., 2000).

These distinctions in traumatic response are posited to be in part because of key differences in the nature of the antecedent traumatic stressors. Judith Herman, an early advocate for recognizing complex PTSD apart from traditional PTSD, highlighted this distinction:

> The child trapped in an abusive environment is faced with the formidable task of adaptation. She must find a way to preserve a sense of trust in people who are untrustworthy, safety in a situation that is unsafe, control in a situation that is terrifyingly unpredictable, power in a situation of helplessness. Unable to care for or protect herself, she must compensate for the failures of adult care and protection with the only means at her disposal. (Herman, 1992, p. 96)

In consideration of this possibility, the working group for DSM-IV classified such cases under "disorders of extreme stress not otherwise specified" (DESNOS) (Friedman, Resick, Bryant, & Brewin, 2011) but discarded the idea of a separate diagnosis in light of field trials showing that 92% of individuals that met for DENOS also met for PTSD.

Similarly, for DSM-5 (2013), van der Kolk et al. (2005) championed inclusion of a new *developmental trauma disorder* (DTD) classification that would capture the distress and functional impairment associated with frequent multi-type interpersonal violence or other trauma exposures in childhood that show lasting impact on somatic, attentional, affective, interpersonal, and self-perception domains into adulthood (Roth et al., 1997; van der Kolk et al., 1996; van der Kolk et al., 2009). Ultimately this proposal was discarded in favor of conserving the current system of classification (Denton et al., 2016).

Failure to recognize the full scope of traumatic stress symptoms and developmental sequelae presents several problems (van der Kolk, 2005). To start, a proportion of severely traumatized individuals will fall through the diagnostic cracks. In addition, the symptom features absent from current criteria and the functional impairment and distress that accompanies them, are unlikely to be measured, assessed, targeted and tracked over the course of treatment for

PTSD. Furthermore, without recognizing these behaviors as signs of traumatic stress they could be inappropriately categorized as personality pathology or another comorbid disorder or willful oppositionality—labels that further contribute to negative self-concept and social alienation (indeed, some of the core cognitions that serve to *maintain* post-traumatic stress symptomology (Foa, 1998).

Moreover, affectively charged terms such as "traumatic" or a "catastrophic event" may be less likely to resonate with survivors of multiple adversities or trauma that occurred early in development, especially if prolonged or prevalent across many domains, relative to someone whose exposure occurred in late adolescence or early adulthood or was a circumscribed event (e.g., Ford et al., 2008, 2010). This has implications for both seeking help and endorsing trauma symptoms in the context of a clinical setting, including a structured clinical interview. In fact, there is evidence that PTSD is vastly under-recognized in academic and community mental health settings, with estimates suggesting that as few as 4% of individuals with the disorder receive the diagnosis (Davidson et al., 1991; Davidson, 2001; Amaya-Jackson et al., 1999; Switzer et al., 2001).

Adolescent Survivors of Complex Trauma: Under-Recognized and Over-Diagnosed?

If traumatic stress responses in childhood and adolescence manifest more broadly than traditional PTSD symptomology, then it might be expected that children and adolescents who meet for PTSD will have a greater likelihood to meet for additional disorders. Consistent with this, data from the NCS showed that 44% of women and 59% of men with PTS/D (post-traumatic stress disorder or sub-diagnostic threshold post-traumatic stress) also met criteria for three or more other psychiatric diagnoses. Additionally, adolescents who have been exposed to extreme stress who do not meet full PTSD criteria, yet exhibit additional traumatic stress

sequelae, may either fail to qualify for mental health services or be assigned a pathological disorder apart from trauma altogether.

Data from the National Child Traumatic Stress Network (NCTSN) revealed that children abused at home tend to meet criteria for 4–7 diagnoses (as cited in van der Kolk & Najavits, 2013). A 2007 study published in the *Annals of General Psychiatry* found that over 90% of psychiatric inpatient admits in their sample (N = 139) endorsed at least one traumatic experience and nearly 70% endorsed multiple. In spite of the high prevalence of multiple traumas and clear indicators of functional impairment and distress in this sample (e.g., inpatient hospitalization, suicidality), only 7% had a diagnosis of PTSD (Floen & Elklit, 2007). Similarly, a study based on a representative sample of 50 community mental health service recipients with dual mental health and substance abuse diagnoses found that although the majority of individuals had documentation of exposure to one or more significant physical or sexual traumas in their charts, not a single individual was diagnosed with PTSD, nor were their treatment plans providing trauma-informed care (Wiland, 1999). Findings such as these suggest the possibility that traumatic stress and its impairment on functioning may be both vastly under-recognized and widely pathologized with non-trauma-based diagnoses.

Indeed, childhood exposure to multiple and/or prolonged maltreatment is associated with a host of DSM disorders, psychosocial maladjustment, and neurobiological adaptations across the life-span. In adulthood, survivors of childhood trauma have elevated rates of major depressive disorder (MDD), personality disorders, self-harming behavior, suicidal behavior (e.g., Brown, Cohen, Johnson, & Smailes, 1999) and interpersonal difficulties (Tyler, Allison, & Winsler, 2006). Among children, overly compliant, withdrawn, or explosive behavior, as well as inappropriately sexual, reckless, or defiant behaviors are also common (e.g., Stubblefield-Tave et

al., 2005), and child and adolescent survivors of complex trauma receive diagnoses of attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) at elevated rates (Famularo, Kinscherff, & Fenton, 1992; Cook et al., 2005). Additionally, children subjected to multiple maltreatments commonly show some divergences in neurobiological profile from that of their non-maltreated counterparts, including in cognitive, language, motor, and social domains, and in identifying their emotions (Streeck-Fischer & van der Kolk, 2000). Notably, similar neurobiological profiles have also been linked to high externalizing.

Externalizing Psychopathology as an Adolescent Indicator of Exposure (and Adaptation) to Early Chronic Stress and Trauma?

Multivariate studies have shown that comorbidity among a spectrum of behaviors such as substance dependence, antisocial behavior, and disinhibited personality traits can be modeled hierarchically with a global latent factor (labeled externalizing, EXT) that captures the common variance linking each phenotype within the spectrum, and specific factors that capture the distinctions among phenotypes within the spectrum (Krueger et al., 2002, 2005, 2007). Biometric analyses of these multivariate models suggest that variation in the EXT factor corresponds primarily to additive genetic variation, while both genetic and environmental factors underlie distinctions between specific forms of EXT spectrum phenotypes (for example, fighting only or illicit drug use only).

Evidence for the existence of a coherent externalizing liability has been well replicated. However, the mechanistic origins of this liability remain unclear. Perhaps externalizing disorders (and commonly comorbid attentional, learning, and personality disorders) have been notoriously challenging to treat because we have fundamentally conceptualized and studied this end of the spectrum as an entity independent of its roots. In this study I explore the possibility that the

impact and accompanying adaptations of early and prolonged stress and trauma exposure exert a fundamental role in elevations of global externalizing behavior that exceed the adolescent population-normative range. In addition, I also examine the links between domain-specific externalizing behaviors and sexual behaviors that have also been linked to childhood stress and trauma.

Early Chronic Stress and Trauma and Externalizing: The Possibility of a Link

If early chronic stress and trauma has a role in the downstream expression of adolescent externalizing, then we should expect to find elevated prevalence rates of childhood trauma exposure among adolescent samples who are elevated on externalizing. Consistent with this basic expectation, trauma exposure is highly prevalent among youth within the juvenile justice system, a group that tends to be elevated on externalizing (e.g., Ford, Chapman, Hawke, & Albert, 2007; Smith, Leve, & Chamberlain, 2006; Lederman, Dakof, Larrea, & Li, 2004). According to a study at a large detention center in a big city, more than 90% of youth endorsed at least one—often multiple—qualifying traumatic events according to DSM-IV criteria (Abram et al., 2004). In comparison, an epidemiological study of trauma exposure prevalence in a representative sample of youth in the community was 25% (Costello, Erkanli, Fairbank, & Angold, 2002). In further support of a link, studies of childhood disaster victims and witnesses to violence have reported fighting and aggression at school to be the most common behavioral problem among children exposed to these events (Terr, 1979; Eth & Pynoos,1985). Similarly, externalizing and attention problems were the most prevalent types of behavioral problems to emerge in a sample of 342 adolescents (ages 6–18) adopted from large institutional settings in Russia where infants were deprived of consistent, emotionally responsive caregiving (Merz & McCall, 2010). Infants adopted in early infancy fared better in both of these domains relative to

infants who were adopted after 18 months. And children adopted from severely depriving Romanian institutions that lacked both adequate psychosocial and physical resources showed greater magnitude of externalizing and attentional problems, and within a shorter duration of time spent in the institutional setting (Merz & McCall, 2010).

Additionally, a study of 311 children, ages 3–15 years, who had witnessed violent events in their home country prior to attaining refuge in a new country (e.g., taking shelter from bombing, witnessing street shooting, death in the family, father detained, witnessing arrest of family member, father tortured, mother tortured, father disappeared), found that across the re-experience, arousal, and avoidance items, the single most common symptom endorsement was "gets upset easily" (53%) followed by "is easily aroused" (44%). And after conducting extensive confounder correction of the trauma events, "mother tortured" and "father disappeared" showed significant and independent effects on magnitude of post-trauma symptoms. The strongest correlate of "mother tortured" was "destroys things" (OR 9.9, $p < .0005$); the strongest correlate of "father disappeared" was "disobeys parents" (OR 3.1, $p < .01$); "mother tortured and/or father disappeared" was most strongly related to "fears the future" (OR = 4.7, $p < .0005$) (Montgomery & Foldspang, 2006).

In contrast to adult PTSD presentations, in which re-experiencing, arousal, and avoidance symptoms are prominent, factor analyses revealed that a two-factor solution comprising arousal and sleep disturbance best captured these children's symptoms. In spite of the cultural differences that might distinguish a sample of refugee children (in this case from a range of Middle Eastern countries) from a sample of American children, this scenario is uniquely apt to examine the main effect of prolonged exposure to severe traumatic stress early in development—

indeed, youth at the extreme end of the traumatic stress exposure continuum—with minimal caveats about the potential confounding role of gene-environment correlation.

In addition to aggression, post-traumatic stress sequelae are also linked to other prominent domains of externalizing. Among patients seeking treatment for PTSD, for instance, rates of alcohol or drug abuse/dependence have been documented in upward of 60–80% (Branchey, Davis, & Lieber, 1984; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Khouzam & Donnelly, 2001; Perkonigg, Kessler, Storz, & Wittchen, 2000). Early chronic stress and trauma—particularly of an interpersonal nature—is also a risk factor for a range of adolescent health-risking behaviors. Among adolescent girls cumulative trauma exposure has been found to be even more predictive than PTSD of sexually risky behaviors (Smith, Leve, & Chamberlain, 2006). Greater rates of alcohol and tobacco use, driving while intoxicated, and sexual health-risking behaviors have also been documented among women who have experienced multiple types of abuse (Rodgers et al., 2004). Chronic and/or severe childhood trauma is also a robust risk factor for suicide among both sexes (Dube et al., 2001; Green et al., 2005; Roy, 2005; Thompson et al., 2005; Ullman & Breckman, 2002) and substance abuse for both sexes (e.g., Breslau, Davis, & Shultz, 2003; Hien, Cohen, & Campbell, 2005). Given the link between one's thoughts and behavior, it is conceivable that suicidal cognitions such as hopelessness, foreshortened future, and the desire to end one's life might contribute to apathy about long-term health and safety and in turn influence implicit cost-benefit calculations pertaining to drug and alcohol use and decisions about sex.

Finally, if complex traumatic stress symptomology has a causal role in EXT-related symptomology/impairments, then we should expect to see improvements in EXT-related symptomology after treating traumatic stress symptomology. While no such study has explicitly

tested this hypothesis to my knowledge, there are two clinical trials that are consistent with a potential role of traumatic stress symptomology underlying elevations on adolescent externalizing and aggression. The first comes from a clinical control trial that administered divalproex sodium to adolescents who were diagnosed with both PTSD and conduct disorder. Relative to the low-dose control group, the treatment group showed a reduction in intrusion, avoidance, and hyperarousal symptoms. Notably, these reductions in traumatic stress symptomology were also accompanied by reductions in aggressive behavior (Steiner et al., 2007). The second study, a recent randomized clinical trial conducted with veterans through the VA San Diego Healthcare System, documented elevations on Wechsler Adult Intelligence Scale (WAIS) performance in areas of processing speed, working memory, and verbal memory after treating PTSD (In press, 2018). This is notable, given that a similar neurobiological profile has been shown to accompany more severe presentations of externalizing. Although not conclusive, these findings are consistent with the possibility for a role of post-traumatic stress sequelae underlying elevations in externalizing spectrum behavior.

Adolescence: An Information Gap

There is a notable lack of developmentally informed data that addresses how early exposure to chronic and/or severe traumatic stress manifests—or might be expected to manifest—during the developmental period of adolescence (ages 12–18). Empirically validated measures of trauma symptomatology that extend beyond PTSD criteria have received scant attention in younger demographics, and among adolescents in particular.

A recent review by Denton et al. (2016) identified 40 papers evaluating such assessment instruments since 2005. Of these 40, nine measures were designed and validated for children (ages 2–12 years) and only two were designed and validated for adolescents (ages 12–18)

(Assessment Checklist for Adolescents [ACA], Tarren-Sweeney, 2013a; Brief Assessment Checklist for Adolescents [BAC-A], Tarren-Sweeney, 2013b). Denton et al. concluded:

> Few assessment measures have been robustly investigated to generate confidence in their use with children and adolescents who have suffered developmental trauma. … The lack of attention to demographic information suggests that researchers have not engaged sufficiently with the evidence that abuse rarely occurs in isolation from other adversities (Dong et al., 2004)." (Denton et al., p. 279)

Denton and colleagues went on to identify one of the fundamental challenges in this domain as such:

> … the lack of consistency in the theoretical understanding of these children and adolescents' difficulties. There has been an emphasis on trying to conform to a diagnostic classification system, for example, DSM-IV PTSD, but failure to recognize the complexity of presenting difficulties. … The challenge for researchers is to remain focused on the unique impact of cumulative trauma (Briere, Kaltman, & Green, 2008) and the research suggesting multiple- and single-trauma experiences result in different trauma symptomatology profiles (Green et al., 2000; Jonkman, Verlinden, Bolle, Boer, & Lindauer, 2013)." (Denton et al., 2016, p. 280)

Clearly, much remains to be learned about the biopsychosocial manifestations of early chronic stress and trauma during the adolescent period.

Prevalence of Victimization Among Children and Adolescents

The lack of validated tools to assess developmentally sensitive indicators of traumatic stress during adolescence is further concerning because when it comes to serious crime, adolescents are actually at greater risk for victimization than adults (Song, Singer, & Anglin, 1998). Aggregating across the United States, adolescents ages 12–19 are victims of three times as many rapes, three times as many assaults, and twice as many robberies than those over age 20. A national telephone survey conducted in 1994 of randomly selected adolescents ages 10–16 (N = 2,000) found that 41% of the sample endorsed some form of victimization from simple and aggravated assault to sexual abuse (Boney-McCoy & Finkelhor, 1995).

Exposure to violence at school is also disturbingly common, particularly in large city public schools. A survey of over 3,700 high school students in the Cleveland area (Song et al., 1998) found that among students attending large city schools, 75% had witnessed at least one person being physically beaten at school over the past year. Also alarmingly, 45% of these students endorsed witnessing someone being either shot or shot at over the past year. Another study found that 47% of first graders and 31% of second graders in large city schools had witnessed a shooting and a stabbing respectively (Song et al., 1998).

These findings are not unique to Cleveland. There are similar findings in other big city public schools. In several Chicago public elementary schools, for instance, surveys of over 1,000 students revealed that 75% of the young boys and 10% of the young girls had witnessed either a robbing, shooting, stabbing, or killing of another individual in their lifetime. A closer look at the data revealed that 26% of 10-year-olds and 30% of 19-year-olds had witnessed murders. Among 10-year-olds, 41% had witnessed stabbings—slightly higher than that endorsed by the 19-year-olds. A community mental health center on the south side of Chicago conducted a survey of 536 African American second graders, fourth graders, sixth graders, and eighth graders and found that 26% of the children had seen a person get shot and 29% had seen a stabbing take place (Bambade, Shakoo, & Chalmer, 1991). Of the 2,000 homicides on record in Los Angeles county in 1982, an estimated 10–20% were witnessed by a dependent child. Similar percentages were reported in Detroit as well.

Patterns of Victimization Among Children and Adolescents

Although much research has focused on specific forms of childhood maltreatment, most maltreated children experience more than one form of abuse (Kinard, 1994; Cook et al., 2005; DeJong, 2010; Dong et al., 2004; Finkelhor, Turner, Ormrod, & Hamby, 2009; Streeck-Fischer

& van der Kolk, 2000). In a nationwide study, Turner, Finkelhor, & Ormrod (2010) found that among adolescents who endorsed childhood sexual abuse, 50% of them were victims of multiple types of traumas. This is consistent with findings that adolescents are more likely to have experienced multiple life-threatening traumatic exposures than an isolated life-threatening trauma (Suliman et al., 2009). This may account in part for why distinct types of childhood maltreatment have been linked to general rather than specific effects on psychological functioning in adulthood (e.g., Varia, Abidin, & Dass; Widom & Ames, 1994). Consistent with this idea, number of traumatic events has shown a cumulative linear correspondence with severity of PTSD and depression symptomology in adulthood (Suliman et al., 2009).

Additionally, some research suggests that traumatic events that take place in early childhood manifest most strongly in later childhood in psychosomatic form. Specifically, youth who have experienced high levels of trauma in their backgrounds show higher levels of trauma stress symptoms, and these trauma stress symptoms tend to increase over the course of adolescence. Also, the impact of prior trauma on trauma stress symptoms tends to be more pronounced among older adolescents relative to children (Lam, Lyons, Griffin, & Kisiel, 2015). Interestingly, however, there is also evidence that in spite of more pronounced somatic symptoms, adolescents may report lower subjective severity relative to younger children. This might be a potential consequence of the age-related increases in basal cortisol that correspond with increased HPA activity between pre-adolescence and adulthood (reviewed in Gunnar & Vazquez, 2006), which might impact subjective salience by way of reduced interoceptive awareness.

Nevertheless, these patterns, in conjunction with the data showing higher rates of exposure to potentially traumatic events among children and adolescents relative to adults, raise the question: How are children and adolescents coping?

Are they *more* resilient than adults? Are they falling under the radar? Or might developmentally normative adaptations for surviving non-population-normative levels of early, sustained, and severe traumatic stressors be mistaken for behavioral or characterological pathologies and predispositions divorced from their roots as a traumatic stress response, altogether?

This possibility, and its potential implications for the infrastructure and institutionalized norms that shape the treatment and life trajectories of adolescent survivors of complex trauma, merit scrutiny. One of the barriers to gaining traction in understanding the link between early complex trauma and disruptive adolescent behaviors such as fighting, delinquency, and more severe presentations of EXT in general, has been attributed to a general lack of clarity on the mechanistic underpinnings that link the two (Ardino, 2012; Kerig, 2012a; Dierkhising et al., 2013).

Integrating clinical science on fear, arousal, chronic stress, and adaptation with principles from cognitive behavioral theory, I outline a biologically plausible mechanism for the role of early complex trauma underlying externalizing behaviors that exceed (population-level) developmental normativity in severity and persistence.

A Biologically Plausible Mechanism: Threat and the Alarm System—A Convergence of Internalizing in Externalizing

From a factor analytic framework, and traditional conceptualizations of externalizing and internalizing as two etiologically distinct and generally opposing dimensions, it may seem

counterintuitive to account for variation in externalizing with internalizing symptoms (e.g., somatic, cognitive, affective states). However, this is consistent with the complex trauma phenotype (Herman, 1992; Cook et al., 2005; van der Kolk et al., 2005, 2009; Garvert, Brewin, Bryant, & Maercker, 2013) and might be understood mechanistically from a functional or adaptive perspective of fear- and stress-based responses and the behavioral action tendencies and self-regulatory based coping behaviors that accompany them. Because this is a critical point for the current study, it merits further elaboration. In doing so, I will also provide some mechanistic insight into how complex traumatic stress reactivity might be expected to manifest as (or through) behaviors or symptom profiles traditionally conceived or interpreted as more pronounced and enduring presentations of externalizing.

Rather than simply "symptoms" of a disorder, anxiety and panic responses serve as a protective alarm system to facilitate survival under conditions of threat or danger (Telch, 1992). Anxiety orients attention toward future-oriented threat, whereas panic orients attention to immediate threat. As such, panic is characterized by an acute surge in anxiety that is far more intense but shorter acting than anxiety arising from a future-oriented threat. Both responses manifest through a complex system of interactions involving the hypothalamic-pituitary-adrenal (HPA) system, the prefrontal cortex, the hippocampus, and the amygdala.

When a threat is perceived, the amygdala sends a signal to the hypothalamus, which relays a signal through the autonomic nervous system to the adrenal glands, which activate the sympathetic nervous system. As stress hormones (epinephrine, norepinephrine, cortisol) secreted by the adrenal glands enter the bloodstream, a cascade of coordinated effects within cognitive, physical, and behavioral systems are activated, all in service to promote survival in the context of perceived threat or danger.

The cognitive system prompts shifts in attention and orienting response that promote vigilance and monitoring/scanning the environment for potential threat and narrowing the field of consciousness in response to stimuli that are perceived to resemble potential threat. The cognitive, or mental system, also forms protective cognitions ("core beliefs") about safety from threat and ability to cope with threat, informed from prior experiences. Because survival of the organism is at stake, threat-likelihood *overestimations* (i.e., erring on the side of caution when appraising likelihood of threat) have conferred survival benefits over precision, as underestimation is costly.

The physical response system includes nervous and chemical effects, cardiovascular effects, and respiratory effects modulated by the sympathetic and parasympathetic nervous systems. Blood from the digestive system is diverted to large muscle groups in preparation for fighting off or fleeing threat. Rapid heartrate and increased blood pressure facilitate release of adrenaline throughout the body and increase oxygen supply to large muscles. Blood vessels in the skin, intestines, and extremities constrict, an adaptation to protect from bleeding to death from potential laceration, and glucose is secreted by the liver into the bloodstream to facilitate stamina for extended exertion. Release of analgesic neurochemicals buffer against pain from injuries that may be incurred during fight or flight that could hinder escape from danger.

As noted above, core beliefs are often altered in the wake of trauma; generally, the more previous exposure to traumatic events and/or chronic stressors, the more ingrained these core beliefs tend to be. These alterations tend to take reliable forms (Foa, 1998), including beliefs about the safety of the world (e.g., "The world is a dangerous place"; "You never know when something terrible will happen"; "I have to be on guard all of the time"), the meaningfulness of life ("I have no future"; "I feel dead inside"), the trustworthiness of others ("I can't trust

anyone"; "People are not what they seem") the worthiness of self ("I am bad"; "I have permanently changed for the worse"), a sense of incompetence and beliefs about one's ability to cope ("I can't rely on myself"; "If I think about the event I will not be able to handle it") (Agar, Kennedy, & King, 2006; Dunmore, Clark, & Ehlers, 1999; Epstein, 1991).

Within a cognitive behavioral framework, these changes in cognition are conceptualized as maintaining factors for recurring-traumatic stress reactivity (post-traumatic stress symptoms) because of their role in perpetuating perceptions of current or imminent threat after the threatening period or event has passed and is unlikely to return (Foa, 1998). (In the case of ongoing threat or potential for danger, however, these cognitions would not be considered "maintaining factors" but rather protective and adaptive in relation to the current environment.)

This perception of current or imminent threat can manifest as chronic hyperarousal and hypervigilance, intrusive thoughts and imagery (memories, nightmares, flashbacks, paranoia, hallucinations), panic attacks, heightened emotional reactivity (e.g., affective dysregulation), elevated startle, impulsivity, lowered distress tolerance, chronic feelings of anger, episodes of rage, pervasive shame, suicidality, foreshortened sense of future, impaired concentration, sleep disruption, chronic hypoarousal, emotional numbing, and dissociation (Ogden, Minton, & Pain, 2006; Kendall-Tackett, 2000).

These responses are interrelated and reinforce one another. The markedly unpleasant sensations that characterize the alarm response serve to motivate activation of survival promoting behavioral action tendencies (fight/flight/freeze) which are naturally reinforced by the negative feedback loop of the HPA system that returns the body to homeostasis after the perceived threat subsides (Levine, 1997). The behavioral action tendency associated with panic is escape oriented and, depending on the nature of the threat (and trauma history and resources of the individual

such as size, strength, etc.), will generally fall within the fight, flight, or freeze (dissociative/numbing) response domains, promoting the response that will optimize survival. The behavioral action tendency associated with anxiety is avoidance, as anxiety orients attention toward an impending or future-oriented threat (Telch, 1992).

The action tendencies that accompany the alarm response, such as behavioral avoidance or physical escape through fight or flight, may be unfeasible depending on the nature of the threat. Such is often the case for infants and children who lack the physical strength of an adult. Moreover, the sources of threat (and by extension trauma-related reminders or triggers) are often embedded within domains of a child's normative environment (e.g., home, neighborhood/community, school). Thus, when severe imminent threat is physically inescapable (i.e., neither fighting off nor fleeing danger is physically viable) the behavioral action tendencies associated with mental forms of escape by way of a dissociative or numbing (hypoarousal/"freeze") response can be most adaptive—a mechanism to numb the pain (and conscious awareness) when physical escape is not viable. This is described by Ogden:

> In contrast to the energy consuming processes mediated by the sympathetic nervous system, increased dorsal vagal tone is associated with energy conservation: Many functions of the body begin to slow down, leading to "a relative decrease in heart rate and respiration and accompanied by a sense of 'numbness,' 'shutting down within the mind,' and separation from the sense of self (Siegel, 1999, p. 254)." (Ogden, 2006, p. 31)

This line of defense is triggered into action by a lack of oxygen in the tissues of the body (hypoxia) and functions to decrease arousal into the *hypo*arousal zone. The emotional blunting and cognitive effects associated with the hypoarousal state can pose problems for exercising judgement about danger and personal safety and consequently may increase vulnerability for further traumatization (e.g., taking a shortcut through a dangerous park alone at night). Emotional processing is often impacted too as hypoarousal "can reduce the capacity to sense

emotions and experience emotional reactions to significant events, [and] thus diminish effective emotional processing" (Ogden, 2006, p. 35).

People who have experienced chronic and/or severe trauma, such as characteristic of early complex trauma, often vacillate between both *hyperarousal* and *hypoarousal* (Siegel, 1999). As described by Ogden (2006),

> When traumatic experiences are chronic, the most adaptive survival responses for a specific set of circumstances are repeatedly activated, either as a result of actual threat or in preparation for anticipated threat triggered by traumatic reminders. Traumatized people are usually so sensitized by past traumatic events that they have very low thresholds for relatively minor stressors, responding with the extreme arousal adaptive in the past, either becoming hyperaroused or becoming hypoaroused. In either case, because the window of tolerance has become functionally narrowed by repeated traumatic responses, the individual is now increasingly more vulnerable to perceived traumatic triggers. Many traumatized individuals are unable to prevent wide swings of dysregulated arousal, fluctuating between the extreme zones of hyperarousal and hypoarousal. This recurring "bottom-up hijacking" is experienced as sudden ruptures in the window of tolerance, after which the individual is unable to easily or quickly return to the optimal arousal zone (Siegel, 1999). (Ogden, 2006, p. 34).

From what has been described so far, the function of the externalizing domain pertaining to domain-specific fighting emerges most clearly from the standpoint that: (1) it is one of the core behavioral action tendencies for escape from perceived threat; (2) through mechanisms of sensitization, repeated exposures to physical abuse or assault (or witnessing violence) predicts increasing activation of the alarm response at increasingly lower thresholds of perceived (interpersonal) threat, leaving adolescents susceptible to engaging in (pre-emptive) reactive aggression (Dodge & Schwartz, 1997; Schultz, Izard, & Bear, 2004; Pollak & Tolley-Schell, 2003; Ford, Fraleigh, & Connor, 2010; Maughan & Cicchetti, 2002; McCrory et al., 2011).

However, the prolonged state of stress activation that characterizes chronic hyperarousal and can be triggered by trauma-related cues or reminders (e.g., intrusive thoughts/memories/sensations, re-experiencing-spectrum features), might be one mechanism that

motivates the use of external means to facilitate internal regulation of the stress response system (to provide a sense of reprieve from a highly sensitized and persisting activation of alarm). Such may be the case for chronic *hypo*arousal too, though the accompanying coping mechanisms/behaviors would be in service of *increasing* arousal.

Thus, in service of modulating or regulating extended aversive states of arousal, a range of behaviors characteristic of the externalizing spectrum might prove highly reinforcing by way of negative reinforcement (i.e., reducing an aversive state). Extended states of *hyper*arousal might be expected to reinforce behaviors that produce a dampening or nervous system depressant effect (e.g., alcohol, marijuana, opioids, benzodiazepines, barbiturates, or other anxiolytic drugs) or facilitate the discharge of intense stress-related hyperarousal; e.g., nicotine (Kassel et al., 2007), verbal outbursts or physical fights (e.g., Graham-Bermann & Levendosky, 1998; Ford et al., 2006, 2012), or sex (McKusick, Horstman, & Coates, 1985; Lyle, 2003; Schuster, Mermelstein, & Wakschlag, 2013).

In cases of extended *hypo*arousal this might reinforce behaviors that increase arousal and disrupt aversive states of numbness (e.g., amphetamines, cocaine, reckless/bold behaviors, new sexual/romantic partners, or self-harm [self-cutting, self-burning, or initiating fights against a larger opponent]) or provide a grounding mechanism to interrupt distressing autonomic states of dissociation and/or re-experiencing (Corrigan, Fisher, & Nutt, 2011; van der Kolk, Perry, & Herman, 1991; Klonsky, 2007). Finally, food and sex may also be used for regulatory functions by way of releasing neurotransmitters (e.g., dopamine, oxytocin, serotonin) that can provide a sense of reprieve from aversive mood/affective states (Lee, O'Riordan, & Lazebnik, 2009; Brady et al., 2010; Cortoni & Marshall, 2001).

Complex Traumatic Stress Exposure and Sequelae: A Developmentally Informed Approach

In summary, a number of research groups have proposed that current DSM diagnostic categories may be incomplete for individuals who have undergone early, multi-type, chronic, and/or severe traumatic stress exposures (Cook et al., 2007; van der Kolk et al., 2009). To date, much of the empirically based research and validated measurement tools have been derived with adult populations and are based upon a narrower construct of traditional PTSD. Extending these constructs and assessment measures to adolescents does not fully capture the complete spectrum of biopsychosocial indicators of traumatic stress response as manifested during this period of development. Moreover, there is evidence that standard PTSD measures may be particularly inadequate for adolescents who have endured the most severe and/or prolonged exposures to traumatic stress beginning early in life (Turner, Finkelhor, & Ormrod, 2010) and is often social and/or interpersonal in nature, as they do not capture the accompanied disturbances in attachment-related behaviors (van der Kolk et al., 2013; Terren-Sweeny, 2013) and self-organization (Herman, 1992) including key affective, negative self-concept, and social/interpersonal domains (Cloitre et al., 2013).

Operationalizing Early Chronic Stress and Trauma

To date, there has been no established consensus or recommended "best practice" for the operationalization of early chronic stress and trauma. As more research has gotten underway, however, several investigators have provided recommendations for future research efforts in this area. Greeson and colleagues have noted:

> One issue is how to best define different types of trauma exposure … Currently, the child trauma field uses multiple terms for similar phenomena, including complex trauma, polyvictimization, and cumulative risk/adversity. Therefore, it will be important to unpack the conceptual similarities and differences inherent in these phenomena in an

> effort to reach consensus about terminology and meanings. Establishing a common language in turn will provide greater clarity for researchers, clinicians, and policymakers, which can facilitate greater synergy across professional discourses. (Greeson et al., 2011, p. 105)

After reviewing the child trauma literature with these considerations in mind, I elected to adopt the label *complex trauma* to be consistent with the terminology and definition set forth by the National Child Traumatic Stress Network (NCTSN, Spinazzola et al., 2005). According to the NCTSN, complex trauma exposure refers to "the experience of multiple or chronic and prolonged, developmentally adverse traumatic events, most often of an interpersonal nature and early-life onset" (Spinazzola et al., 2005, p. 5). According to the NCTSN, complex trauma commonly comprises a combination of physical, emotional, and educational neglect and mistreatment and is also common among children exposed to war and chronic community violence. The ubiquity and chronicity of developmentally adverse traumatic events "situates complex trauma as a dual problem of exposure and adaptation" (p. 5).

Thus, complex trauma differs from some of the other maltreatment composite indices in the literature in that it is a term that encompasses both the *exposures* to developmentally adverse experiences as well as the *adaptations* that are posited to accompany these long-term exposures and impact an array of affective, somatic, interpersonal, cognitive, and behavioral sequelae (Cook et al., 2005; van der Kolk, 2005).

Following from the example of Kisiel and colleagues (2009), the NCTSN definition of complex trauma has been operationalized in recent empirical studies as having two or more of five trauma experiences, rated moderate to severe: sexual abuse, physical abuse, emotional abuse, neglect, or domestic violence (Kisiel, Fehrenbach, Small, & Lyons 2009; Greeson et al., 2011). However, as the original authors noted, given their focus on a child welfare population,

their operationalization did not capture non-caregiver sources of interpersonal trauma that can also be relevant such as exposure to community violence, traumatic loss, racism or racial discrimination, school violence or harassment, and sexual assault or rape (Finkelhor, Ormrod, & Turner, 2007; Carter, 2007;Bryant-Davis & Ocampo, 2005).

A further consideration is that to date, the studies that have examined complex trauma using the NCTSN definition have included samples drawn from either the child welfare system or children and adolescents referred for trauma-related treatment to a NCTSN-affiliated clinic. One consequence of this is that exposure to severe trauma within these samples is virtually a qualifying criterion. In contrast, the prevalence of complex trauma within a population-representative sample such as Add Health is far rarer. Moreover, with a population-representative sample it is necessary to contend with the reality that many forms of abuse, possibly as a function of chronicity as some studies have indicated, go unreported altogether (e.g., Fergusson, Horwood, & Woodward, 2000; Widom & Morris, 1997; Finkelhor, 1984; Watkins & Bentovim, 1992), resulting in contamination and undermining the magnitude and specificity of stress and trauma-related effects.

Further problematic in the case of the current study is that exposures to caregiver abuse are not formally inquired of participants until the third study wave. Consequently, sample attrition becomes a serious concern, and in this case, the attrition disproportionately impacts the subset of participants who have experienced caregiver mistreatment and complex trauma, resulting in valuable loss of information.

Additionally, with a population representative sample in particular, a further area that is important to include, and which the above operationalization does not capture, are indices of the broader contexts that the child encounters on a routine basis. The biopsychosocial recovery that

marks the healing process following exposure to a traumatic event (or exposure to a chronic stressor that might originate from a particular context) takes place over time and across the range of settings in which children spend their time. Most commonly this consists of home, school, and neighborhood. The nature of these settings then, becomes highly relevant to resilience, recuperation, and recovery processes as each setting impacts this process whether it be through facilitation of a rapid recovery or conversely, by exacerbating or even adding new injuries—and everything in between (Gudino, 2013; MacMillan, Tanaka, Duku, Vaillancourt, & Boyle, 2013; Fremont, 2004; Gustafsson, Larsson, Nelson, & Gustafsson, 2009; Osofsky, 1999; Power, 2004; Proctor et al., 2007; Maniglio, 2009).

This is consistent with increasing recognition in the child trauma literature that a broad and diverse array of adverse experiences are predictive of poorer outcomes and that the relationship between contextual risk factors and poor outcomes appears to take the form of a cumulative dose-response relationship (e.g., Felitti et al., 1998; Araya et al., 2009; Chipman et al., 2010; Greeson, 2011, 2014; Layne et al., 2014). Moreover, non-interpersonal adverse family circumstances (e.g., poverty, poor parental health, parental loss) have been shown to predict trauma-related symptoms independent from interpersonal and non-interpersonal traumatic life events, and for boys has been shown to magnify the mental health impact of interpersonal and non-interpersonal traumatic life events (Nilsson, Gustafsson, & Svedin, 2012).

Thus, to mitigate these problems the current study adopts a unique approach of indexing adversity indirectly, capitalizing on the empirical evidence that severe maltreatment rarely occurs in the absence of other types of stressors (e.g., Kinard, 1994; Suliman et al., 2009; DeJong, 2010; Turner et al., 2010; Finkelhor, Turner, Ormrod, & Hamby, 2009), that more stressors confer poorer prognosis (Briere, Kaltman, & Green, 2008; Suliman et al., 2009; Greeson et al., 2014),

and that ubiquity of stress across multiple life domains characterizes the typical profile of childhood complex trauma (Spinazzola et al., 2005; Layne et al., 2014). Obviating problems of sample attrition or dependency on self-reports of highly sensitive caregiver-perpetrated traumatic events, this approach will instead capture a comprehensive range of exposures, experiences, and perceptions that combined, comprise an index of adversity experiences and indicators that commonly accompany more severe/chronic stress and trauma exposures (Herman, 1992; Cook et al., 2005; Cloitre et al., 2013; van der Kolk et al., 2013; Terren-Sweeny, 2013; Layne et al., 2014).

Drawing from the conceptual and empirical frameworks delineated in the childhood complex trauma literature, and integrating the recommendations and research insights delineated above, I embrace a comprehensive conceptualization of complex trauma that indexes both cumulative and co-occurring exposure to multiple types of stressors and trauma-related exposures beginning in childhood and manifesting in adolescence. Consistent with the data that most maltreated children experience multiple types of abuse and adversities, across multiple settings, and on multiple occasions (Kinard, 1994; Cook et al., 2005; DeJong, 2010; Dong et al., 2004; Finkelhor, Turner, Ormrod, & Hamby, 2009), and that contextual factors relating to home, neighborhood, and school can have independent and exacerbating effects on trauma-related symptomology and global stress (e.g., Freisthler et al., 2008; Brydon et al., 2004; Gruenewald et al., 2006), the present study will draw on a comprehensive array of exposures across key life domains in conjunction with adolescent perceptions of safety and security within each of them to define a comprehensive index of *global life strain*.

In addition, I also incorporate recent (past 12 months) exposures to non-caregiver forms of interpersonal violence that were reported at Wave 1 to comprise an index of *recent violent*

victimization. Events in this index (such as having witnessed a shooting or having been physically attacked / jumped) constitute qualifying criterion A events for PTSD and are also predictive of more pronounced profiles of arousal (e.g., hyperarousal and/or hypoarousal, depending in part on the nature of previous stress/trauma-related exposures) (e.g, Song, Singer, & Anglin, 1998; Schwarz & Perry, 1994). Together, these indices will capture the array of key features that are posited to distinguish more severe and complex forms of traumatic stress exposures by including information pertaining to type, severity, ubiquity, developmental timing, and context.

I accommodate the second part of the *dual problem* of complex trauma (exposure and *adaptation*) using factor scores derived from a measurement model composed of an array of indicators that have been implicated as markers of adaptation imprinted from sustained states of stress-related autonomic arousal (Adkins et al., 2009), in conjunction with disturbances in mood, affective, self-concept, and interpersonal domains; that is, the domains that are posited to converge to comprise the defining profile of adaptive sequalae characteristic of childhood complex trauma as manifested during the developmental period of adolescence (Turner, Findelhor, & Ormrod, 2010; Terren-Sweeny, 2013; van der Kolk et al., 2013).

As Krueger & Markon (2011) have noted, a symptom-level approach such as this can be particularly useful for helping to "unpack" lower prevalence conditions and facilitate the emergence of new dimensions. As childhood complex trauma and its sequela is expected to have a low base-rate within the general population (prevalence estimates are generally < 5%), this approach constitutes one of the strengths of the current study. Nevertheless, as many of these domains can also arise independent of traumatic stress exposures and adaptations, and the present study was not equipped to distinguish trauma-based adaptations from individual

differences arising from other disorders or domains, I refer to this global symptom domain as global *internalizing*.

The Present Study

The present study aims to better understand the role of chronic stress and trauma in the etiology of adolescent externalizing behavior (EXT), specific rule-breaking behaviors unique from global EXT (alcohol use, fighting, illegal drug use), and adolescent sexual behavior, using a large sample of adolescents from the National Study of Adolescent Health (Add Health; Udry 2003a).

Guided by the literature on adolescent complex trauma, and the mechanistic framework described earlier, I predict the following:

1. Global life strain, recent violent victimization, and *internalizing* (the focal stress/trauma-related predictors) will each show positive associations with externalizing and with the specific domain of fighting (Terr, 1979; Dodge, 1980; Ford, 2012; Green and Berkowitz, 1976; Song, Singer, and Anglin, 1998).
2. When combined into a single multivariate main effect model, global life strain, recent violent victimization, and the *internalizing* factor will each retain statistical significance and account for unique variance in externalizing and domain-specific fighting.
3. Global life strain, recent violent victimization, and to a lesser degree the *internalizing* factor will each show positive, independent, main effects on number of Wave 1 sexual partners and negative main effects on age at first sexual intercourse (AFS).
4. Due to the elevated threat sensitization posited to follow a violent interpersonal assault and prime anticipation and preparedness for future interpersonal threat (e.g., Dodge, 1980; Ford, 2012; Graham-Bermann & Levendosky, 1998), I predict that recent violent

victimization will be most strongly associated with domain-specific fighting. I predict that violent victimization will also be associated with elevated externalizing primarily by way of the variation in global externalizing behavior accounted for by fighting (i.e., non-domain-specific fighting). Thus, I predict that the association between violent victimization and externalizing will be significant and moderate but lower in magnitude relative to its association with domain-specific fighting.

5. I test whether the associations between high global life strain and the global *internalizing* factor will combine in an additive manner or interact synergistically in the prediction of adolescent externalizing and domain-specific fighting. Given the mechanistic framework outlined earlier, I predict that increased global *internalizing* will moderate (amplify) the impact of global life strain in the prediction of global externalizing and domain-specific fighting.
6. Consistent with conceptualizations of complex trauma as a dual problem of exposure and adaptation, I also predict that the *internalizing* factor will at least partially mediate the association between global life strain and outcomes of externalizing and domain-specific fighting.
7. Finally, given the racial/ethnic diversity of the Add Health sample, I also examine the extent to which exposures to different types of stress/strain experiences may diverge in accordance with the intersectionality of gender and race/ethnicity (African American, Latinx, European American).

Method

Participants

Participants were drawn from Wave 1 of the National Longitudinal Study of Adolescent Health (Add Health; Udry 2003A), a multi-wave study of over 20,000 US adolescents collected in four waves between 1994 and 2008, selected through a stratified, school-based, cluster sampling design. Wave 1 data were collected in 1995 when adolescents were between the ages of 12 to 20 years of age (Wave 2 in 1996, Wave 3 in 2001-02, and Wave 4 in 2007-08). For the purposes of this study, data included a subset of population representative African American, Latinx and European American adolescents between the ages of 12 and 18 who also had complete data on the parent questionnaire completed by the head of household at Wave 1 (usually resident mother). The response rate for the Wave 1 parent questionnaire was 98.5% for the child-specific data. Design features including an oversample of African American adolescents with highly educated parents (operationalized as at least one parent holding a college degree), as well as population stratification and Add Health's school based sampling design, were accommodated using the sampling weights provided by Add Health and clustering by school. In total the sample included 14,941 adolescents (males = 49%, females = 51%; European American = 58%, African American = 25%, Latinx/non-White Hispanic = 17%). In addition to adolescent self-report at Wave 1, head-of-household report and interviewer report were also used for select items as noted below.

Measures

Complex trauma. The operationalization for complex trauma was guided by the definition provided by the National Child Traumatic Stress Network (NCTSN, Spinazzola et al., 2005), in which complex trauma exposure refers to "the experience of multiple or chronic and prolonged, developmentally adverse traumatic events, most often of an interpersonal nature and early-life onset" (p. 5) and in which the ubiquity and chronicity of developmentally adverse

traumatic events "situates complex trauma as a dual problem of exposure and adaptation" (p.5). Through collaborative efforts between clinical research institutions and community mental health providers, the NCTSN has identified six key areas of disruption beyond the prototypical PTSD that is common for more complex trauma. These domains include affective, negative self-concept, perceptual (e.g., attention/concentration), behavioral, attachment (interpersonal/relational problems), and autonomic/physiological (somatization) (Spinazzola et al., 2002). These domains integrated with recommendations from previous research on complex trauma comprised the guiding framework for constructing the focal three complex trauma related predictors for the current study.

Complex trauma (part 1 of 2): Exposures. As a starting point for operationalizing the exposure component of complex trauma, items were drawn from a list of Wave 1 Stressful Life Events (SLE) adapted from Ge et al. (1994) for use in Add Health by Adkins, Wang, Dupre, van den Oord, & Elder (2009). The original 25-item index comprised an array of events across many domains (e.g., family, school, finances, relationships, physical health, violence exposure, tragic loss, accidents) and was adapted by Adkins et al. (2009) using criteria established by Turner & Wheaton (1995) that defined SLEs as acute events of sudden onset and limited duration that occurred within the previous 12 months.

One consequence of these selection criteria for the present study is that key components of the complex trauma construct are missing (Spinazzola et al., 2005; Cook et al., 2005). As Adkins et al. (2009) acknowledged in their study, "other aspects of the stress process-including chronic stressors and buffering resources-are also important components … We encourage future research to improve upon the current analyses with more exhaustive mode that integrate chronic

stressors and buffering psychosocial resources as predictors and mediators in the stress process" (p. 54–55).

As such, select SLE items were combined into a composite that would tap *recent* exposures to interpersonal trauma. These items were selected based upon meeting DSM-5 criterion A threshold for a stressor, defined as exposure to actual or threatened death, serious injury, or sexual violence (American Psychiatric Association, 2013, p. 271). Based on the extracted items, this index most closely reflected recent exposure to violent victimization and was labeled accordingly.

Because this composite was limited to events within the previous 12 months, it excluded many characteristic features of the complex trauma construct (e.g., stressors that occur early in life, are chronic or prolonged, occur across multiple domains, and include school and/or caregiver related forms of interpersonal strain) (Terr, 1991; Pearlman, 2001; Spinazzola et al., 2005; van der Kolk et al., 2005; Cook et al., 2005). In light of these considerations and of recent calls for adolescent trauma research to include an expanded range of life domains, stressors, and symptoms/behavior problems (e.g., Layne et al., 2014; Sweeny, 2013; Burgermeister, 2007), I constructed a second exposure related composite to capture more global life strain.

Item selection and compilation for this composite proceeded in accordance with theory on complex trauma with an emphasis on adolescent perceptions (Rutter, 2016; Cohen, Alper, Adler, Treanor, & Turner, 2008; Singh-Manoux, Marmot, & Adler, 2005; Mankowski & Wyer, 1997) and a holistic sampling of adversities and contextual/psychosocial factors empirically linked with elevated stress-vulnerability, particularly within the context of other stressful/traumatic life events (e.g., Cohen & Wills, 1985; Stachour, 1998; Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Schilling, Aseltine, & Gore, 2007, 2008; Gustafsson, Larsson,

Nelson, & Gustafsson, 2009; Maniglio, 2009; Taylor, 2011; Wang, Cai, & Peng, 2014). In accordance with recommendations for inclusion of multiple informants in research on stress processes (Kovacs, 1989; Compas, 1987), resident caregiver and Add Health interviewer reports were included in measures as available and described below.

Complex trauma (part 2 of 2): Adaptation. The "dual problem" of complex trauma refers to the stressor/trauma-related exposures *and* the adaptations that accompany extended exposures to developmentally adverse events. Adaptations that uniquely characterize the complex trauma profile cluster together across affective, self-concept, perceptual (e.g., attention/concentration), behavioral, attachment (interpersonal/relational), and autonomic/physiological (somatization) domains (Spinazzola et al., 2002; Cook et al., 2005; van der Kolk et al., 2005; Cloitre et al., 2009). Items for the adaptation composite were sampled from each of these domains and are described in further detail below (key predictor variable #3).

Individual items, coding, and construction of the three composite variables that comprise the focal indices of complex trauma in the current study are detailed below, numbered 1–3.

Key Predictor Variables

1. Recent exposure to violent victimization (past 12 months). Summation of the following Wave 1 items comprised the composite index of recent violent victimization exposure. This composite, which ranged from 0 – 8 (*Mode* = 0), was coded from *0–2* corresponding to zero- (76.6%), one- (12.2%), and two or more (11.2%) victimizations within the previous 12 months. Items included:

a. ***Jumped or physically assaulted.*** Participant responses to being jumped or beaten up were coded on a 3-point scale (0 = "no times" [88.52%], 1 = "one time" [8.92%], 2 = "two times" [2.56%]).

b. *Knife or gun pulled.* Participant responses to having a knife or gun pulled on them were coded on a 3-point scale, like the one above (zero = 87.24%, once =10.17%, twice = 2.59%).

c. *Shot at or stabbed.* Participant responses to being shot at or stabbed were coded on a 3-point scale (zero = 98.69%, once = 1.14%, twice = 0.17%).

d. *Witnessed a shooting or stabbing.* Participant responses to witnessing a shooting or stabbing were coded on a 3-point scale (zero = 87.72%, once = 8.92%, twice = 3.36%).

2. Global life strain. This composite consists of four broad life domains that are commonly disrupted among adolescents with complex trauma (Spinazzola et al., 2005; Cook et al., 2005). Composite scales indexing hardship within each of the four life domains (*a-d* below) were standardized (z-scale) and then combined and averaged to form a single composite variable representing Global Life Strain. These scales, described in detail below, included: a) *attachment and acceptance at home*, b) *family health problems*, c) *economic and residential safety and security*, and d) *adolescent perceptions of safety and acceptance at school*. All scales were coded such that greater numeric values corresponded to greater strain. Descriptive statistics broken down by biological sex and race/ethnicity for all predictors and outcomes are presented in Table 3.1.

a. *Attachment and acceptance at home.* This domain combined two facets of attachment related indicators that are characteristically disrupted among children/adolescents with complex trauma (van der Kolk et al., 2013; Layne et al., 2014), *i*) *early disruptions and loss in caregiving* and *ii) adolescent perceptions of emotional safety and security within their family system*. Items were drawn from a combination of adolescent and parental questionnaire report. The sum of the dichotomous items comprising the *disruptions and*

loss in caregiving subscale were summed with the mean of the *adolescent perceptions of emotional safety and security* subscale to comprise a composite index of *Attachment and Acceptance at Home* (*Mean* = .92, SD =.73, *Mode* = 0). The final index of *Attachment and Acceptance at Home* was then transformed to z-scale.

i. *Disruptions and loss in caregiving.* The following four items were summed to comprise the *disruptions in loss and caregiving* subscale (*Mode* = 0, [88.8%], *Range* = 0 - 4).

*Extended caregiver separation (*e.g., Surtees et al., 2006). Parental questionnaire responses to "Was there ever a period of at least six months when [Child] did not live with you?" were coded such that any positive endorsement within the first 10 years of the adolescent's life was coded 1 (6.3%) and all other responses were coded 0. This cut-off was chosen to correspond to caregiver separation that took place during childhood, prior to adolescent onset.

Disruption or loss of primary caregiver (e.g., Bet et al., 2009). Adolescents whose primary caregiver(s) did not include at least one biological or adoptive parent were coded as 1 (3.3%) and all else were coded as 0.

Death of mother (e.g., Layne et al., 2014). Adolescents who experienced the death of their mother during their lifetime were coded as 1 (1.2%), adolescents who did not were coded 0.

Death of father (e.g., Layne et al., 2014). Adolescents who experienced the death of their father during their lifetime were coded as 1 (3.1%), adolescents who did not were coded 0.

ii. ***Adolescent perceptions of emotional safety and security.*** Adolescent responses to the following statements were coded on 5-point scale (0 = *Very much / Strongly agree* to 4 = *Not at all / Strongly disagree*) and then averaged to comprise the *adolescent perceptions of emotional safety and security* subscale (*Mean* = .78, *SD* = .56): "How much do you feel that your family pays attention to you?" / "How much do you feel that people in your family understand you?" / "How much do you feel that your parents care about you?" / "Most of the time your mother is warm and loving toward you."/ "You feel loved and wanted."

b. ***Family health problems.*** This domain is broadly relevant to stress within the home environment and was chosen based on research linking poor parental health both directly (e.g., Layne et al., 2014) and indirectly to child/adolescent stress by way of the physical and psychological impact of parental health problems influencing caregiving resources and practices such as consistency, attentiveness, harshness, etc. (e.g., Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Schilling, Aseltine, & Gore, 2007, 2008). Resident caregiver responses to the Wave 1 parent questionnaire were combined for a cumulative index of family health problems (*Mean* = 1.88, *SD* = 1.40, *Median* = 1.5, *Range* = 0 – 10). Items were selected on the basis of their status as a chronic health condition or health complaint linked to chronic stress (e.g., Surtees et al 2006; Richie et al 2009) and/or empirical evidence suggesting greater probability of impaired/disrupted caregiving in association with the condition:

Chronic Obstructive Pulmonary Disease (COPD)- biological mother. Parent report on adolescent's biological mother (*yes = 1, no = 0).*

Diabetes-biological mother. Parent report on adolescent's biological mother (*yes = 1, no = 0).*

Alcoholism-biological mother. Parent report on adolescent's biological mother (*yes = 1, no = 0).*

Alcoholism-biological father. Parent report on adolescent's biological father *(yes = 1, no = 0).*

Physical disability-resident mother. Parent report on adolescent's *resident mother* (*yes = 1, no = 0).*

Physical disability-resident father. Parent report on adolescent's *resident father* (*yes = 1, no = 0).*

Migraine headaches-biological mother. Parent report on adolescent's biological mother (*yes = 1, no = 0).*

Migraine headaches-adolescent. Parent-report on adolescent (*yes = 1, no = 0).*

Resident caregiver perception of physical health. Responses to "How is your general physical health?" were coded on a 5-point scale (0 = "excellent," to 4 = "very poor").

Adolescent physical health (parent report). Responses to "How would you rate [*Adolescent*]'s physical health?" were coded using the same scale as above.

Prior to summing items for an overall index of family health problems, resident mother physical health and adolescent physical health were recoded such that only endorsements of "poor" (= 1) and "very poor" (= 2) counted toward the final summation of physical health problems. Endorsements below this threshold were

coded 0. All *family health problem* items were summed and then transformed to z-scale.

c. *Residential safety and economic security.* This domain is relevant to the stress process as it both structures exposure to stressful life events and elevates vulnerability to their impact through access to resources and coping mechanisms (Pearlin, 1989; Link & Phelan, 1995; Dodge & Pettit, 2003). Items for this composite (*Mean* = 3.94, *SD* = 2.15, *Median* = 4.0, *Range* = 0 – 12) were drawn from Wave 1 Parent Survey and Add Health Interviewer report as specified below, consistent with indices many others have used in Add Health to index similar domains:

Neighborhood drug use (parent report). Wave 1 parent questionnaire responses to the question, "In this neighborhood, how big a problem are drug dealers and drug users?" were coded on a 3-point scale (0 = "no problem at all," 1 = "a small problem," and 2 = "a big problem").

Financial strain (parent report). Wave 1 parent questionnaire responses to the question, "Do you have enough money to pay your bills?" was coded on a 2-point scale, and reverse coded to conform to higher scores corresponding to greater adversity (0 = yes, 1 = no).

Unemployed resident mother (parent report). Endorsement of current unemployment = 1, otherwise coded 0.

Unemployed resident father (parent report). Endorsement of current unemployment = 1, otherwise coded 0.

Parental educational attainment. Adolescents whose residential parents' mean education was equivalent or below "more than eighth grade but did not graduate

from high school" (and whom did not obtain a GED) were coded as 1 and all other levels of parental educational attainment were assigned 0. This cutoff point was chosen in accordance with research indicating that the distinction in education level between high school graduate (or equivalent) versus less than a high school graduate is particularly impactful in terms of distinguishing levels of perceived stress and exposure to stressful life events (Down, Palermo, Chyu, Adam, & McDade, 2014).

Desire to move (adolescent report). "If, for any reason, you had to move from here to some other neighborhood, how happy or unhappy would you be?" Item responses ranged from 0 = "not at all happy," up to 4 = "very much [happy]." For this composite, only unequivocal endorsements of "very happy" (= 1) counted towards the overall index, all other responses were coded 0.

Interviewers' safety perception (interviewer report). Add Health interviewers who participated in the At-home interview were asked "When you went to the respondent's home, did you feel concerned for your safety?" Positive endorsements were coded 1 and negative endorsements were coded 0.

Perception of safety in current neighborhood (adolescent report). "How strongly do you agree or disagree with the following statement? I feel safe in my neighborhood." (0 = strongly agree / very much to 4 = strongly disagree / not at all). Participants who responded "strongly disagree" were coded 2 and participants who responded "disagree" were coded 1, all other responses were coded 0.

Neighborhood & dwelling repair (interviewer report). At wave 1 Add Health interviewer responses to, "How well kept is the building in which the respondent lives?" and "How well kept are most of the buildings on the street?" were each coded on a 4-point scale (3 = "very poorly kept (needs major repairs)," 2 = "poorly kept (needs minor repairs)," 1 = "fairly well kept," 0 = "very well kept"). Because adolescents from rural areas were not asked the second question there was sizable missing data on this variable. Therefore, the average of the non-missing values were used for this composite. In this way, adolescents from rural areas were assigned the value they received on the dwelling repair item. Additionally, items were rescaled such that only endorsements of "very poorly kept" (= 2) and "poorly kept" (=1) counted towards the final scale. The *residential safety and economic security* scale was transformed to z-scale.

d. *Perceptions of safety and acceptance at school.* Perceptions of safety and acceptance at school were indexed using responses to the following statements coded on a 5-point scale (0 = strongly agree / very much to 4 = strongly disagree / not at all) and then averaged.

"You feel safe in your school" / "You feel you are a part of your school" / "How much do you feel that your teachers care about you?"

In addition, as a more distal index of school climate, adolescents who responded, "strongly agree" to the statement, "Students at your school are prejudice" were assigned an additional point on this scale (*Mean* = 1.49, *SD* = .89, *Range* = 0 - 5). The *perceptions of safety and acceptance at school* scale was transformed to z-scale.

Finally, all four scales *a-d* above were transformed to z-scale prior to summing and then averaging for the composite index of *Global Life Strain*.

3. Adaptation: Global *Internalizing* Factor. At Wave 1 adolescents were asked about the frequency in which they experienced a broad array of cognitive, affective, interpersonal, and somatic concerns/perturbances. Drawing from the literature on childhood complex trauma, I sampled a broad range of theoretically relevant markers of adaptation to prolonged stress and trauma exposure. Because Add Health does not have predefined traumatic stress related symptom scales to guide combining items, theoretically relevant items were subjected to a bifactor confirmatory factor analysis. This model allows for the measurement of a single common latent factor (global *internalizing*) while also modeling, and controlling for, item covariation unique to subsets of items that hang together. The bifactor structure was the conceptually favored choice to model the data because it allows for the simultaneous modeling of a broad global factor encompassing the range of cognitive, affective, somatic, and interpersonal states that have been described in the child and adolescent trauma literature as clustering together to characterize the complex trauma adaptations, including the fluctuation between states of *hypo*arousal and *hyper*arousal that characterizes complex trauma (Ogden, 2006), while also allowing for more narrow factors that tap interoceptive sensitivity (*hyper*arousal specific), an energy-intensive liability dimension for the expression of PTSD in the context of trauma exposure, and a numbing specific factor (*hypo*arousal specific), an energy-conserving adaptation posited to accompany more prolonged, uncontrollable/unpredictable, or inescapable chronic stress or trauma-related exposures (e.g. Siegel, 1999; Ogden, 2006).

The global factor, labeled hereafter as global *internalizing*, corresponded to covariation among an array of mood, somatic arousal, affective, self-concept and interpersonal states. The

residual covariance from the global factor cohered into two domain-specific factors reflecting subsets of items that covaried independent from the global factor and from one another. These included a domain-specific *Somatic Sensitivity* factor, in which items pertaining to headaches, stomachaches and trouble relaxing loaded most highly, and a domain-specific *Negative Self-Concept/Numbing* factor, in which items pertaining to self-concept (e.g., "You felt just as good as other people"; "You thought your life had been a failure"; "You felt life was not worth living") and affective anhedonia (e.g., items indexing inability to experience feelings of happiness, enjoyment) loaded most highly. Factor scores from this model were computed by *M*plus and saved for use in subsequent analyses. A list of individual items and factor loadings are displayed in Table 3.1.

Table 3.1 Factor loadings for global and specific stress-related adaptation factors

Items (drawn from Add Health at wave 1)	Global Factor "Internalizing" Factor	Specific Factor 1 "Somatic Sensitivity"	Specific Factor 2 "Neg. Self-Concept / Numbing"
"You felt fearful." *	0.60		
"You had trouble keeping your mind on what you were doing." *	0.64		
"You felt life was not worth living." *	0.65		0.53
"You were happy." *	0.42		0.43
"You enjoyed life." *	0.42		0.55
"You felt you were just as good as other people." *	0.24		0.57
"You felt hopeful about the future." *	0.22		0.54
"You were bothered by things that don't normally bother you." *	0.64		
"You thought your life had been a failure." *	0.67		0.42
"You felt that people disliked you." *	0.57		0.17
"You felt that you were too tired to do things." *	0.60		
"In the past 12 months, how often have you had trouble relaxing?"	0.50	0.45	
"In the past 12 months, how often have you had trouble falling asleep or staying asleep?"	0.44	0.38	
"You felt depressed." *	0.72		0.25
"During the past 12 months, did you ever seriously think about committing suicide?"	0.52		0.28
It was hard to get started doing things." *	0.57		
"Since school started this year how often have you had trouble paying attention?"	0.48		
"You didn't feel like eating; your appetite was poor." *	0.54		
"People were unfriendly to you." *	0.49		*0.02*
"In the past 12 months, how often have you been moody?"	0.48	0.34	
"Since school started this year, how often have you had trouble getting along with other students?"	0.45		*-0.04*
"In the past 12 months, how often have you had a poor appetite?"	0.38	0.39	
"In the past 12 months, how often have you had chest pain?"	0.37	0.39	
"In the past 12 months, how often have you felt hot all over suddenly, for no reason?"	0.40	0.32	
"In the past 12 months, how often have you felt dizzy?"	0.41	0.42	
"In the past 12 months, how often have you had a stomachache or upset stomachache?"	0.31	0.43	
"In the past 12 months, how often have you had a headache?"	0.24	0.51	
"You talked less than usual." *	0.40		0.15
"What do you think are the chances you will live to 35?"	0.22		0.32
"I usually go out of my way to avoid my problems."	0.07		0.10

***Note*. Items are coded such that greater distress is associated with higher scores. Items with "*" are responses that correspond to reporting on the previous week. Factor loadings are presented in standardized form. All factor loadings are statistically significant at $p < .001$ except for italicized loadings which demarcate non-statistical significance.**

Outcome Variables

Externalizing spectrum behaviors. Items for EXT were selected based on theoretical consistency with the EXT factor as described in the literature (e.g., Krueger et al., 2002; 2007). Due to diverging developmental trends among select indicators, age was controlled at the item level prior to model fitting. Based on theory and prior research, I fit a bifactor model to the data in which EXT was indexed by a global factor that accounted for common variance among all items, and three domain-specific factors indexing variation in adolescent *drinking*, *drug use*, and *fighting* behavior unique from global EXT. Model-derived factor scores were computed in *M*plus for use as the EXT spectrum outcomes in model testing.

Age at first sexual intercourse (AFS). Participants reported on whether they had ever had vaginal intercourse and their age at first sexual intercourse at Waves 1–4. Consistent with previous studies with this sample (e.g., Harden, Mendle, Hill, Turkheimer, & Emery, 2008), to reduce telescoping of retrospective reports, we extracted participant reports from the earliest wave in which they endorsed having had sexual intercourse. Participants who reported sexual intercourse before age 11 were coded as missing due to the greater possibility of these encounters being nonconsensual. Participants who did not endorse sexual intercourse by Wave 4 were coded as missing. Additionally, lifetime forcible rape was included as a covariate for females in all sexual outcome analyses. Because males were not asked this question, we were unable to control for this variable among male adolescents. Among our sample of adolescents, AFS ranged from 11 to 30 years old ($M = 16.58$, $SD = 2.70$).

Number of sexual partners. Wave 1 self-report responses to a computer-administered question, "With how many partners have you ever had vaginal intercourse, even if only once?" was used to index number of sexual partners. Participants who reported no sexual partners were assigned a 0. Reports ranged from 0 to 900 partners (M = 1.34, SD = 10.48), resulting in significant over-dispersion. To address this, partner counts beyond 25 (corresponding to the 97.8th percentile among adolescents who endorsed at least one lifetime sexual partner) were assigned a value of 26. Although this did not fully correct the over-dispersion for number sexual partners, it enabled employment of a zero-inflated negative binomial regression. This model estimates two separate models and then combines them: first, a logit model predicts membership in the 0 group; second, a negative binomial model predicts counts for individuals who do not always have 0s.

Analyses

Analyses were conducted using structural equation modeling software *M*plus (Muthen & Muthen, 1998–2007). I assessed model fit using root mean square error of approximation (RMSEA; values below .08 and .05 are considered adequate and excellent respectively), the Comparative Fit Index (CFI), the Tucker-Lewis Index (TLI) (for both indices values of .90 and .95 are considered adequate and excellent, respectively), and the chi-squared fit statistic. Mediation analyses were conducted in *M*plus using MODEL INDIRECT.

For the bifactor model of stress response indicators, all cross loadings were constrained to zero as specified by the model. Missing values for continuous variables were handled using full-information maximum likelihood and for categorical variables using pairwise present. For items that were ordered-categorical indicators with five or fewer response categories, we used robust weighted least squares (WLSMV) estimator (Beauducel & Herzbert, 2006), and for continuous

items we used maximum likelihood with robust standard errors, MLR. For all models, non-independence of data due to Add Health's school-based sampling design was handled using the TYPE = COMPLEX command in *M*plus and clustering by school. Sampling weights provided by Add Health were used to adjust for population stratification. Prior to all analyses and composite creation, items were recoded so that higher scores corresponded to greater adversity/distress. All models covary biological sex and race/ethnicity, pubertal timing, and chronological age unless noted otherwise.

Additionally, in line with recent calls for the wider adoption of an intersectionality approach in research (Mullings & Schulz, 2006; Warner & Brown, 2011), race/ethnicity and gender were dummy coded to be consistent with this approach, with White males as the reference point. An intersectionality approach assumes that race/ethnicity and gender constitute identities that are greater than the sum of their parts. This approach "is centered on structural inequality (Thornton, Dill & Zambrana, 2009) and stipulates that because race/ethnicity and gender are fundamental determinants of opportunity structure, defining access to both the resources that promote health and exposure to the risks that undermine health, their effects cannot be disaggregated or understood separately" (Warner & Brown, 2011, p. 1236).

Results

Pearson's correlations between complex trauma-related predictors, global and specific EXT behaviors, and sexual activity outcomes are displayed in Table 3.2. Overall, global life strain, recent violent victimization, and the global *internalizing* factor showed a similar pattern of correlates. Each stress/trauma-related predictor was associated with greater global externalizing, greater domain-specific *fighting*, and earlier age at first sexual intercourse. Additionally, each

stress/trauma-related composite showed modest to minimal negative associations with domain-specific *illegal drug use,* and modest positive associations with total number of sexual partners at wave 1. None of the three stress/trauma-related composites were appreciably associated with domain-specific *alcohol use*.

Table 3.2 Correlations among study variables

Variable	GS	*Attch*	*Sch*	*Nhd*	*Hlth*	Vic	Int.	SS	Numb	EXT	Fight	Alc	Drug	AFS	Ptr	Pub	Age
Global Strain	1.00																
Attach	0.64	1.00															
School	0.63	0.31	1.00														
Nhood	0.63	0.15	0.15	1.00													
Health	0.60	0.13	0.10	0.26	1.00												
Recent Victim	0.25	0.15	0.20	0.19	0.10	1.00											
Internalizing	0.44	0.37	0.36	0.18	0.18	0.21	1.00										
Somatic-S	*0.00*	0.03	0.03	-0.11	0.04	-0.09	0.14	1.00									
Numb-S	0.27	0.23	0.14	0.18	0.12	0.09	0.16	-0.18	1.00								
EXT	0.26	0.25	0.30	0.06	0.05	0.33	0.32	0.08	0.06	1.00							
Fighting-S	0.22	0.14	0.16	0.17	0.09	0.46	0.20	-0.19	0.07	0.21	1.00						
Alcohol-S	0.02	0.00	0.09	-0.07	0.02	-0.03	0.02	0.14	-0.01	0.30	-0.20	1.00					
Drugs-S	-0.06	*-0.01*	-0.02	-0.09	-0.02	-0.11	*-0.01*	0.17	*0.01*	-0.17	-0.06	-0.14	1.00				
AFS	-0.24	-0.15	-0.16	-0.15	-0.12	-0.22	-0.14	0.06	-0.09	-0.34	-0.18	-0.05	0.22	1.00			
N. Partners	0.07	0.04	0.06	0.04	0.02	0.10	0.05	-0.02	*0.01*	0.12	0.10	0.03	-0.08	-0.12	1.00		
Puberty	-0.04	*-0.01*	0.03	-0.07	-0.06	0.04	0.02	0.11	-0.20	0.15	0.03	0.12	*0.01*	-0.05	-0.01	1.00	
Age	0.14	0.11	0.13	0.07	0.04	0.06	0.14	-0.10	-0.02	0.03	0.04	0.03	*0.01*	0.08	0.07	0.07	1.00

***Note*. All values significant at $p < .05$ unless noted by italics which denotes non-statistical significance. Variables coded such that higher numbers correspond to greater adversity / distress. Global strain = Global life strain, a composite comprised of the mean of four z-scored life domain composites: *Attach/Attch* = Acceptance and attachment at home, *School/Sch* = Perceptions of safety and acceptance at school, *Nhood/Nhd* = Residential safety and economic security, *Health/Hlth* = Family health problems; Recent Victim/Vic = Violent victimization within previous 12 months; *Internalizing*/Int = global *internalizing* factor score, Somatic-S/SS = somatic sensitivity specific factor, Numb-S/Numb = numbing/negative self-concept specific factor; EXT = global externalizing, Fighting-S/Fight = fighting specific factor that is unique from global EXT, Alcohol-S/Alc = alcohol use unique from global EXT,**

Drugs-S/Drug = Illicit drug use unique from global EXT; AFS = age at first sexual intercourse, N. Partners/Ptr = total number of sexual partners reported at wave 1; Puberty/Pub = relative pubertal timing, Age = age at wave 1.

Stress and Trauma-Related Exposures by Gender and Race/Ethnicity

On average, global life strain was comparable though slightly higher among girls than boys (d = .08, *CI* = .05, .11), and global *internalizing* was modestly higher for girls (*d* = .21, *p* < .001). Consistent with findings from the broader literature, boys endorsed greater prevalence of recent (non-sexual) violent victimization as girls (boys 34% versus girls 18%, z =2.58, *p* < .001).

Among the domain-specific factors, girls showed slightly elevated though generally comparable levels to boys on the *negative self-concept/numbing* factor (*d* = .09, *CI* = *.09, .12*). Sex differences emerged most prominently for the domain-specific *somatic sensitivity* factor where female sex was associated with elevated *somatic sensitivity* relative to males (*d* = .76, *CI* = .73, .79).

Consistent with the broader literature (Adkins et al., 2009), African American and Latinx adolescents showed higher levels of stressor/trauma-related exposures relative to European American adolescents including higher global life strain (African American: *d* = .39, *CI* = .35, .42; Latinx: *d* = .35, CI = .31, .40), nearly twice the relative risk of recent violent victimization (African American: 35%, RR (z=2.47) = 1.84, *CI* = 1.13, 2.99, *p* < .05; Latinx 34%, RR (z=2.34) = 1.79, *CI* = 1.10, 2.91 versus Caucasian 18%), and slightly higher global *internalizing* (African American *d* = .12, *CI* = .08, .16; Latinx *d* = .12, *CI* = .08, .16).

Examination of the constituent components of global life strain revealed that racial/ethnic differences were driven most prominently by racial/ethnic disparities in residential safety and economic security (European American vs. African American: *d* = .64, *CI* = .61, .68; Latinx: *d* = .58, *CI* = .53, .62) followed by family health problems (European American vs. African American: *d* = .22, *CI* = .18, .26; Latinx: *d* = .22, *CI* = .18, .27), and modest differences in disrupted attachment (European American vs. African American adolescents *d* = .14, *CI* = .10,

.18; Latinx: d = .11, CI = .06, .15). The nature of the racial/ethnic disparities on residential insecurity and economic strain were such that on average African American and Latinx adolescents emerged as moderately above the sample average (African American: M = .36, SD = 1.08; Latinx: M = .29, SD = 1.05), corresponding to greater endorsements of neighborhood unsafety and economic insecurity while European American adolescents emerged as slightly below the sample average (European American: M = -.25, SD = .88), corresponding to a general sense of neighborhood safety and adequate economic resources, on average. Additionally, African American girls endorsed greater feelings of unsafety in their neighborhood relative to African American boys, which appeared to drive the gender differences between African American adolescents on the unsafe neighborhood and economic strain composite (d = .11, p < .001).

Finally, there were notable differences in the domain-specific internalizing factors as a function of racial majority (European American adolescents) versus minority status (African American adolescents, Latinx adolescents). On average, European American adolescents were substantially higher on domain-specific *somatic sensitivity* unique from global *internalizing* relative to their same-sex African American and Latinx counterparts *(*d = .60, CI = .56, .63*)*. In contrast, African American and Latinx adolescents were significantly higher on domain-specific *negative self-concept / numbing* unique from global *internalizing* relative to their same-sex European American counterparts *(*d = .43, CI = .39, .46*)*. All results are displayed in Table 3.3.

Table 3.3 Descriptive statistics for complex trauma-related composites and constituent components by biological sex and race/ethnicity

	European American					African American					Latino/a				
Variable	M / %	SD	Min	Max	n	M / %	SD	Min	Max	n	M / %	SD	Min	Max	n
Global Life Strain															
M	-0.11	0.60	-1.45	2.98	4402	0.07	0.60	-1.32	2.33	1753	0.09	0.61	-1.45	2.43	1264
F	-.09	0.63	-1.45	3.23	4445	0.17	0.62	-1.38	2.49	1872	0.13	0.61	-1.28	2.35	1259
Disrupted Attachment															
M	-0.10	0.90	-1.27	5.93		-0.01	1.06	-1.27	5.93		-0.05	0.95	-1.27	4.64	
F	-0.05	0.94	-1.27	5.80		0.12	1.12	-1.27	7.73		0.10	1.06	-1.27	6.44	
Unsafe at School															
M	0.03	1.02	-1.71	3.89		-0.08	0.97	-1.70	3.52		0.06	1.00	-1.71	3.52	
F	0.00	1.01	-1.71	3.89		-0.02	0.96	-1.70	3.89		-0.01	0.96	-1.71	3.89	
Family Health Problems															
M	-0.14	0.96	-1.37	4.75		0.08	1.01	-1.37	5.11		0.08	1.00	-1.37	5.11	
F	-0.05	1.00	-1.37	5.47		0.18	1.03	-1.37	4.75		0.16	1.00	-1.37	3.67	
Residential Insecurity & Economic Strain															
M	-0.25	0.87	-1.91	4.12		0.30	1.06	-1.91	4.58		0.30	1.04	-1.68	3.89	
F	-0.25	0.90	-1.91	3.65		0.42	1.10	-1.91	5.05		0.28	1.06	-1.91	3.89	
Recent Violent Victimization*															
M	26.3	12.8	0	2		43.7	25.0	0	2		43.8	25.4	0	2	
F	12.1	4.2	0	2		27.5	11.2	0	2		24.4	11.4	0	2	
Internalizing Factor															
M	0.05	0.87	-2.33	3.29		0.12	0.90	-2.14	3.02		0.11	0.87	-2.16	3.06	
F	0.21	0.92	-2.15	4.17		0.33	0.97	-2.30	3.90		0.35	0.99	-2.20	4.36	
Somatic Sensitivity															
M	0.00	0.67	-2.84	2.77		-0.50	0.74	-3.24	1.92		-0.37	0.74	-2.96	1.98	

Table 3.3, cont.

F	0.33	0.67	-2.09	2.97	0.06	0.75	-2.30	2.67	0.18	0.73	-2.32	3.45
Negative self-concept / Numbing												
M	-0.02	0.63	-1.54	3.49	0.26	0.86	-1.75	3.11	0.48	0.79	-1.81	3.01
F	0.06	0.66	-1.85	2.88	0.24	0.83	-1.67	3.06	0.60	0.72	-1.75	2.45

***Note*. Higher values indicate greater strain. Non-bold italicized variable names represent composite subcomponents; Recent Violent Victimization corresponds to victimization within previous 12 months; *All variables are presented in Z-scale with the exception of Recent Violent Victimization which is presented in the first column as the percentage of adolescents who endorsed *any* victimization over the previous 12 months, and in the second column as the percentage of adolescents who endorsed two or more victimizations over the past 12 months.**

Consistent with the conceptualization of the global *internalizing* factor serving as an index of adaptation to cumulative and/or chronic stress/strain, this factor was positively associated with the global life strain composite (r = .44, $p < .0001$). The *negative self-concept/numbing* factor also showed moderate associations with global life strain (r = .27, $p < .0001$). In contrast, the domain-specific *somatic sensitivity* factor was uncorrelated with global life strain (r = -.001, $p = .85$).

Consistent with prior research that has suggested stress/trauma-related symptoms from early childhood manifest most strongly during adolescence, age was positively associated with the global *internalizing* factor (r = .14, $p < .0001$). Pubertal timing showed minimal association with the global *internalizing* factor (r = .02, $p < .05$). However, earlier pubertal timing was associated with higher scores on the *negative self-concept/numbing* factor (r = -.20, $p < .0001$), which showed minimal relation with age (r = -.02, $p < .05$). The domain-specific *somatic sensitivity* factor, in contrast, was associated with younger age (r = -.10, $p < .0001$) and later pubertal timing (r = .11, $p < .0001$).

Individual Main Effect Models of Stress and Trauma-Related Predictors on Adolescent Behavioral Outcomes

A series of multivariate multiple regression models were tested to examine the links between the stress and trauma-related constructs and adolescent sexual activity and externalizing spectrum behaviors. To control for modest residual interrelatedness among the observed factor score composites derived from the measurement model of externalizing, all externalizing

spectrum outcomes were tested simultaneously in all models and their residuals were allowed to correlate.[9]

Global and specific externalizing spectrum behaviors. Model testing began by examining a series of main effect multivariate multiple regression models beginning with a baseline model in which only the demographic covariates (gender/ethnicity, age, pubertal timing) were regressed on the general and domain-specific externalizing outcomes (global EXT, fighting-specific, alcohol-specific, and drug-use-specific behavior). Each of the three stress/trauma-related predictor domains (i.e., global life strain, recent violent victimization, *internalizing*) were then modeled individually (with demographic covariates). Parameter estimates for these models are displayed in the first four panels of Table 3.4.

Baseline demographic differences emerged most prominently on the domain-specific alcohol and drug use outcomes. Specifically, African American adolescents, on average, were more than one standard deviation lower than their European American and Latinx adolescent counterparts on the domain-specific alcohol and drug use factors. This suggests that alcohol and drug use among African American adolescents tends to occur primarily in association with externalizing whereas among European American and Latinx adolescents, it also occurs independent from externalizing. Mean-level differences in drug use were driven primarily by endorsement of inhalants (d = -.49, CI = -.60, -.38) and drug use excluding marijuana (d = -.93, CI = -1.07, -.80). African American adolescents also endorsed lower frequency of intoxication and binge drinking relative to their European American and Latinx counterparts.

[9] Results were highly similar between models that tested outcomes individually versus simultaneously. For ease in presentation we present results from models in which externalizing outcomes were tested simultaneously. Individual outcome results are available from first author.

Consistent with predictions, global life strain, recent violent victimization and the global *internalizing* factor all showed statistically significant main effects of moderate magnitude on global externalizing and domain-specific fighting (β range = .21 to .54; *p's* < .001). Additionally, the domain-specific internalizing factors, *negative self-concept/numbing* and *somatic sensitivity*, each showed modest main effects on global externalizing (β = .06 and β = .06, respectively, *p's* <.001) and the *negative self-concept/numbing* factor also showed a modest main effect on domain-specific fighting (β = .05, *p* <.001).

Each focal predictor also showed statistically significant, though modest, main effects on domain-specific alcohol use (β range = .04 to .11, *p's* < .01), as did the domain-specific *negative self-concept/numbing* factor (β = .04, *p* < .01). There were no appreciable main effects for any of the focal or domain-specific stress/trauma-related predictors on drug use behavior independent from global externalizing.

Table 3.4 Multivariate multiple regression results for individual and combined main effects models of global life strain, recent violent victimization, & global internalizing predicting global and domain-specific externalizing outcomes during adolescence

	Baseline Model				Global Life Strain				Violent Victimization (12 mo.)				Internalizing Predictors*				Full Model			
EXT Spectrum Outcomes	*EXT*	*Fight*	*Drink*	*Drug*	*EXT*	*Fight*	*Drink*	*Drug*	*EXT*	*Fight*	*Drink*	*Drug*	*EXT*	*Fight*	*Drink*	*Drug*	*EXT*	*Fight*	*Drink*	*Drug*
Constant (WM)	*.06*	**.34**	**.19**	**.08**	**.11**	**.37**	**.21**	**.08**	**-.13**	**.14**	**.16**	**.08**	**.13**	**.38**	**.20**	**.08**	-.00	**.20**	**.20**	**.08**
WF	**-.10**	**-.96**	.05	**.29**	**-.12**	**-.98**	.04	**.29**	.00	**-.85**	.06	**.29**	**-.22**	**-1.02**	.03	**.30**	**-.12**	**-.90**	.05	**.30**
BM	.01	**.52**	**-1.25**	**-1.72**	*-.12*	**.43**	**-1.29**	**-1.72**	***-.17***	**.33**	**-1.27**	**-1.72**	-.02	**.47**	**-1.26**	**-1.73**	***-.18***	**.30**	**-1.31**	**-1.73**
BF	**-.22**	**-.27**	**-1.38**	**-1.30**	**-.39**	**-.38**	**-1.43**	**-1.30**	**-.26**	**-.31**	**-1.38**	**-1.30**	**-.38**	**-.38**	**-1.40**	**-1.30**	**-.43**	**-.40**	**-1.44**	**-1.30**
LM	-.04	**.34**	-.05	**.27**	*-.14*	**.27**	-.09	**.27**	***-.19***	**.19**	-.08	**.27**	-.09	**.30**	-.08	**.26**	**-.21**	.17	*-.11*	**.25**
LF	-.08	**-.48**	-.04	**.57**	***-.22***	**-.57**	*-.09*	**.57**	-.10	**-.50**	-.05	**.57**	***-.25***	**-.60**	-.09	**.57**	**-.26**	**-.59**	*-.10*	**.57**
Age	*.04*	*-.02*	*.04*	*-.02*	.00	**-.04**	*.03*	*-.02*	*.03*	*-.02*	***.04***	*-.02*	*-.00*	**-.05**	*.03*	*-.02*	-.01	**-.05**	*.03*	-.02
Puberty	**.16**	**.06**	**.06**	**-.08**	**.16**	**.06**	**.06**	**-.08**	**.13**	**.04**	**.06**	**-.08**	**.15**	**.06**	**.07**	**-.07**	**.13**	***.04***	**.07**	**-.07**
Global Life Strain	--	--	--	--	**.32**	**.21**	**.11**	.00	--	--	--	--	--	--	--	--	**.15**	**.07**	**.10**	.00
Victimized (12mo)	--	--	--	--	--	--	--	--	**.52**	**.54**	**.08**	-.01	--	--	--	--	**.38**	**.47**	.04	-.01
Internalizing factor	--	--	--	--	--	--	--	--	--	--	--	--	**.33**	**.23**	***.04***	-.01	**.22**	**.13**	.00	-.01
NSC/Numbing-S	--	--	--	--	--	--	--	--	--	--	--	--	**.06**	**.05**	***.04***	.02	.02	*.02*	.02	.02
Somatic Sensitivity-S	--	--	--	--	--	--	--	--	--	--	--	--	**.06**	.01	.00	-.01	**.06**	.00	-.00	-.01
ΔR^2 (from baseline model)	--	--	--	--	**.10**	**.04**	**.02**	.00	**.11**	**.12**	**.01**	.00	**.13**	**.05**	**.01**	.00	**.21**	**.15**	.02	.00
Total R^2	**.03**	**.27**	**.24**	**.40**	**.13**	**.31**	**.26**	**.40**	**.14**	**.39**	**.25**	**.40**	**.16**	**.32**	**.25**	**.40**	**.24**	**.42**	**.26**	**.41**

***Note*. Values presented in Z-scores and standardized with respect to the outcome. Age and pubertal timing were standardized prior to model testing. Dummy coding was used for intersectionality of biological sex and race/ethnicity, "Constant" corresponds to White male (WM). Gender/ethnicity specific coefficients are denoted WF = White female, BM= Black male, BF = Black female, LM = Latino male, LF = Latina female, and can be calculated by adding the value of the respective coefficient to the value of the constant (For example, calculating the intercept specific to Black females for the externalizing outcome in the baseline model, located in the far left panel, would entail: *Constant* + β_{BF}**BF* => *.06 + (-.22)*1 = -.16*). All continuous variables were mean centered prior to analyses. Externalizing spectrum outcomes denoted: EXT = externalizing, Fight = domain-specific fighting (unique from global EXT), Drink = domain-specific alcohol use, Drug = domain-specific drug use. Victimized (12 mo.) = violent victimization within previous 12 months (this coefficient is multiplied by its frequency corresponding to: 0 = none, 1= once, or 2 = two or more victimizations), *Internalizing* factor = global internalizing factor, NSC/Numbing-S = domain-specific negative self-concept / numbing, Somatic Sensitivity-S = domain-specific somatic sensitivity; Numeric values in bold = $p < .001$;**

bold, italics = *p* <.01; non-bold, italics = *p* < .05; non-bold, non-italics = not statistically significant. *This *Internalizing* model presented here is distinguished from the global strain and recent violent victimization models in its inclusion of the domain-specific *negative self concept/numbing* and *somatic sensitivity* predictors in addition to the focal predictor of global *internalizing*. This was done for ease in presentation given the minimal differences between the global internalizing only model (not pictured) and the combined global and specific internalizing model (presented in the fourth column); i.e., the main effects of global *internalizing* on EXT (*β* = .35 vs. *β* = *.33*) and on domain-specific fighting (*β* = .24 vs. *β* = *.23)* were comparable between models. However, full results can be made available for the interested reader by request.

Sexual behavior outcomes. For the sexual behavior outcomes (age at first sexual intercourse and number of sexual partners) model testing proceeded parallel to the procedure for the externalizing spectrum outcomes except that each sexual outcome was modeled separately. Parameter estimates for the baseline models and the individual stress/trauma-related domains are displayed in the first four panels of Table 3.5.

Baseline demographic differences emerged most prominently for timing of first sexual intercourse such that European American and Latina adolescents on average reported later timing of first sexual intercourse than African American and Latino adolescents. Among adolescents who were sexually active at Wave 1, number of sexual partners were generally comparable except for African American adolescents in which girls endorsed approximately one fewer partner than the sample average of 4.7 partners ($CI_{Constant}$ = 3.17, 6.24; CI_{bBF} = -1.66, -.06; CI_{bBM} = -1.03, 2.82) and boys endorsed approximately two more. Of note, after controlling for the stress/trauma-related predictors, the number of partners for African American boys who were sexually active at Wave 1 reduced to an average of 4.31 ($p < .001$).

Consistent with predictions, global life strain, recent violent victimization, and global *internalizing* all showed statistically significant main effects, of moderate magnitude, on age at first sexual intercourse (AFS: *b* range = -.36 to -.78, in units of years; all *p's* < .001) and number of sexual partners (*b* range = .23 to .75 in units of people; all *p's* < .05).

As previously noted, these models also controlled for lifetime forcible rape among females (Add Health did not collect this information for males at Wave 1). The magnitude of the coefficients for forcible rape on the sexual health outcomes were substantial and are displayed in Table 3.5. Overall, forcible rape was associated with an age at first sexual intercourse between 1.5 to 2 years earlier than girls who endorsed no lifetime forcible rape (all *p's* < .001).

Endorsement of previous forcible rape was also associated with greater number of partners at Wave 1 relative to girls who endorsed no lifetime forcible rape by Wave 1 ($p < .001$). Due to limitations of the Wave 1 questionnaire phrasing, the extent to which girls counted nonconsensual sexual partners in their total sexual partner count was unable to be ascertained with certainty.

Table 3.5 Unstandardized multivariate regression models of adolescent sexual behavior as predicted by the individual and combined effects of three traumatic stress related composites: global life strain, recent violent victimization, and global internalizing

	Baseline Models		Global Life Strain		Violent Victimization		Internalizing Predictors		Full Models	
Sexual Activity Outcomes	Age at First Sexual Intercourse	Total Sexual Partners (at wave 1)	Age at First Sexual Intercourse	Total Sexual Partners (at wave 1)	Age at First Sexual Intercourse	Total Sexual Partners (at wave 1)	Age at First Sexual Intercourse	Total Sexual Partners (at wave 1)	Age at First Sexual Intercourse	Total Sexual Partners (at wave 1)
Constant (WM)	**16.94**	**4.7**	**16.83**	**3.38**	**17.23**	**3.47**	**16.87**	**3.71**	**17.07**	**3.12**
WF	-.13	-.65	.03	**-.86**	*-.18*	*-.60*	**.00**	**-1.13**	*-.18*	**-.71**
BM	**-2.1**	**1.92**	**-1.82**	**1.34**	**-1.82**	**1.22**	**-1.94**	**1.58**	**-1.59**	**1.19**
BF	**-1.04**	*-.86*	**-.53**	**-1.28**	**-.84**	**-1.20**	**-.69**	**-1.39**	**-.55**	**-1.25**
HM	**-.84**	.64	**-.62**	**.31**	**-.60**	.27	**-.65**	.36	***-.41***	.13
HF	-.08	-.44	.31	-.76	.06	-.56	.26	-.95	.31	**-.73**
Age	**.16**	**.09**	**.38**	**.27**	**.31**	***.13***	**.34**	**.09**	**.39**	***.41***
Puberty	**-.30**	*.32*	**-.29**	*.24*	**-.25**	.20	**-.32**	*.23*	**-.28**	.19
Lifetime forcible rape*	--	--	**-1.64**	**1.42**	**-1.90**	**1.44**	**-1.87**	**1.49**	**-1.49**	**1.23**
Global Life Strain	--	--	**-.69**	**.40**	--	--	--	--	**-.56**	*.23*
Victimized (12 mo.)	--	--	--	--	**-.78**	**.75**	--	--	**-.54**	**.60**
Internalizing Factor	--	--	--	--	--	--	**-.36**	*.23*	*-.08*	.06
Neg. Self-Concept / Numbing	--	--	--	--	--	--	**-.17**	.08	-.05	.01
Somatic Sensitivity	--	--	--	--	--	--	***.10***	***.24***	***.12***	*.20*
ΔR^2 (from baseline model)	--	--	**.08**	**.12**	**.06**	**.09**	**.04**	**.11**	**.10**	**.15**
R^2	**.06**	**.21**	**.14**	**.33**	**.12**	**.30**	**.10**	**.32**	**.16**	**.36**

***Note*. Continuous covariates (puberty, global life strain, victimization, *internalizing* factor) were standardized prior to estimating models with the exception of age, which was left in units of years and mean centered. Parameter estimates displayed in units of the outcome (i.e., units of years for age at first sexual intercourse and units of person for number of sexual partners). Dummy coding was used for intersectionality of race/ethnicity and biological sex, "Constant" corresponds to White male. For number of sexual partner composite virgins were coded as 0 and a zero-inflated negative binomial regression was used to accommodate over-dispersion (further detail in method section). All continuous covariates were mean centered unless otherwise specified. Numeric values in bold = $p < .001$; bold, italics = $p < .01$; non-bold, italics = $p < .05$; non-bold, non-italics = not statistically significant. *Lifetime forcible rape was only collected for girls in the Add Health wave 1 data set. This variable was not included in the baseline models.**

Full Multivariate Main Effect Models

Because of the moderate intercorrelations among the stress/trauma-related predictor variables, the next step was to combine the stress/trauma-related predictors into a single model to test their effects simultaneously. This model provides information about the extent to which each of the individual stress/trauma-related predictors contributes unique information towards the prediction of a given outcome (e.g., externalizing, domain-specific fighting) above and beyond its association with the other stress/trauma-related predictors in the model.

Global and specific externalizing spectrum behaviors. Consistent with predictions, each of the focal three stress/trauma-related predictors retained statistical significance in the full multivariate main effect model, accounting for unique variance in both EXT and fighting unique from EXT. The modest main effects of the stress/trauma variables on domain-specific alcohol use reduced to zero in the full model with the exception of the modest main effect for global life strain which retained its significance. Parameter estimates for the full model are presented in the last panel of Table 3.5.

The effects for each stress/trauma-related predictor remained fairly consistent between the individual main effect models and the full multivariate main effect model, with a couple exceptions. First, for both EXT and domain-specific fighting, the global life strain composite showed the greatest single decrease in magnitude between the individual models and the full multivariate model, decreasing by approximately one half and two thirds, respectively, of its original effect size (EXT: $\beta_{univariate}$ = .32 vs. $\beta_{multivariate}$ = .15; domain-specific fighting: $\beta_{univariate}$ = .21 vs. $\beta_{multivariate}$ = .07). Second, for EXT and domain-specific fighting, the magnitude of global *internalizing* decreased by approximately one third and one half, respectively, of their original

effects after being combined in a single model with the other stress/trauma-related predictors (EXT: $\beta_{univariate}$ = .33 vs. $\beta_{multivariate}$ = .22; domain-specific fighting: $\beta_{univariate}$ = .23 vs. $\beta_{multivariate}$ = .13).

The effect sizes for recent violent victimization on EXT and adolescent fighting unique from externalizing remained substantial across models and accounted for more total variance in the externalizing spectrum outcomes than any of the other individual stress/trauma-related predictors, although for externalizing, the effect size for victimization was somewhat attenuated in the full multivariate model (EXT: $\beta_{univariate}$ = .52 vs. $\beta_{multivariate}$ = .38; domain-specific fighting: $\beta_{univariate}$ = .54 vs. $\beta_{multivariate}$ = .47). After recent violent victimization, the global *internalizing* factor accounted for the second most variance in EXT and domain-specific fighting. Global life strain accounted for the third most unique variance.

None of the modest main effects of the domain-specific *negative self-concept/numbing* factor retained statistical significance for any of the externalizing outcomes in the full model. However, the significance and magnitude for the domain-specific s*omatic sensitivity* factor on EXT was retained in the full model (β_{full} = .06 versus $\beta_{individual}$ = .10).[10]

Overall, results from the full multivariate main effect model were consistent with an additive, dose-response effect of the stress/trauma-related variables on global EXT and domain-specific fighting. The full model accounted for 24% of the total variance in global externalizing. In total, 88% of the explained variance in externalizing was accounted for by the stress/trauma-related predictors. In addition, the full model accounted for 42% of the total variance in domain-

[10] Note that this estimate is not displayed in Table 3.5, as this space was used to present estimates from a multivariate model of the global and domain specific internalizing factors. This was a pragmatic choice since there were minimal appreciable differences in effect estimations between the individual main effect models of the internalizing variables and a multivariate model that combined the global and domain specific internalizing variables.

specific fighting. And of this variance, 36% was accounted for by the stress/trauma-related predictors. Parameter estimates from the full multivariate main effect model are presented in the last panel of Table 3.4.

Sexual behavior outcomes. Each of the focal stress/trauma-related predictors retained statistical significance in the full multivariate main effect models accounting for unique variance in age at first sexual intercourse and number of sexual partners at Wave 1.

Overall, the effects for each stress/trauma-related predictor remained fairly consistent between the individual and the full multivariate models for some domains but not others. For both sexual outcomes the effect of the global *internalizing* factor was substantially reduced when combined with the other stress/trauma-related predictors in the full multivariate models ($AFS_{internalizing}$: $b_{univariate}$ = -.36 vs. $b_{multivariate}$ = -.08; Partners: $b_{univariate}$ = .23 vs. $b_{multivariate}$ = .06). For AFS, global life strain retained its significance and much of its original magnitude in the full model (e.g., $AFS_{global\ life\ strain}$: $\beta_{univariate}$ = -.69 vs. $\beta_{multivariate}$ = -.56, in units of year). For number of sexual partners, the effect of global life strain also retained its significance in the full model but was moderately attenuated in magnitude ($Partners_{global\ life\ strain}$: $\beta_{univariate}$ = .40 vs. $\beta_{multivariate}$ = .23, in units of year). Similar to the pattern for externalizing and domain-specific fighting outcomes, the effects associated with recent violent victimization remained fairly consistent between the individual and combined predictor models, particularly for number of sexual partners (Partners: $\beta_{univariate}$ = .75 vs. $\beta_{multivariate}$ = .60).

Finally, one unexpected outcome to emerge was a diverging pattern between the sexual behavior outcomes and the domain-specific *somatic sensitivity* factor such that higher *somatic sensitivity* was associated with later age at first sexual intercourse ($\beta_{multivariate}$ = .12, $p < .01$) yet also associated with higher number of sexual partners ($\beta_{multivariate}$ = .20, $p < .05$). Furthermore

between the internalizing predictor models and the full multivariate models the effects of global *internalizing* substantially diminished while the effects of the *somatic sensitivity* factor remained stable accounting for more variation than either the global *internalizing* factor or the *negative self-concept/numbing* specific factor. Parameter estimates for the full multivariate main effects models of the sexual outcomes are displayed in the last panel of Table 3.5.

Multivariate Interaction Models

Results from the full multivariate main effect models were consistent with an additive relationship between stress and trauma-related exposures and global externalizing and domain-specific fighting. However, because the multivariate main effect model did not test any interactive associations between the trauma-related predictors and externalizing spectrum outcomes, the nature of the interplay between the complex trauma-related predictors and the externalizing spectrum outcomes is unclear. Thus, the next step was to test a multivariate interaction model to examine whether the relations between the core component indices of complex stress and trauma were in fact additive in nature or whether their convergence might operate synergistically in the prediction of externalizing and domain-specific fighting during adolescence.

Guided by the definitions of childhood complex trauma delineated by the NCTSN, two 2-way interaction models were tested in which the key constituent components of complex trauma (e.g., exposure [global life strain, recent victimization] *and* adaptation [global *internalizing*]) were specified to converge interactively. Although model testing included domain-specific alcohol and drug use, the hypothesis-driven testing was primarily formulated on the externalizing and domain-specific fighting outcomes, which will be the focus of discussion here. Nevertheless, all model testing results are presented below in Table 3.6.

Table 3.6 Multivariate interaction results for global life strain, recent violent victimization, & and the global internalizing factor on global and domain-specific externalizing spectrum behaviors

	Model 1 Internalizing × Global Strain				Model 2 Internalizing × Victim			
	EXT	*Fight*	*Drink*	*Drugs*	*EXT*	*Fight*	*Drink*	*Drugs*
Constant (WM)	**.14**	**.33**	**.34**	**.23**	-.02	**.14**	**.32**	**.22**
WF	**-.21**	**-1.01**	*.04*	**.30**	**-.12**	**-.90**	.05	**.30**
BM	-.08	**.44**	**-1.29**	**-1.73**	***-.15***	**.32**	**-1.28**	**-1.73**
BF	**-.44**	**-.41**	**-1.43**	**-1.29**	**-.38**	**-.38**	**-1.40**	**-1.29**
LM	*-.12*	**.28**	-.09	**.26**	***-.19***	**.18**	-.10	**.26**
LF	**-.28**	**-.60**	*-.10*	**.58**	***-.24***	**-.58**	-.08	**.57**
Age	-.01	**-.06**	*.03*	*-.03*	.00	**-.03**	***.02***	-.02
Puberty	**.15**	**.06**	**.07**	**-.08**	**.13**	***.04***	**.07**	**-.07**
Global Life Strain	**.19**	**.14**	**.10**	.01	--	--	--	--
Victimized (12mo)	--	--	--	--	**.42**	**.48**	*.06*	-.01
Global Internalizing	**.26**	**.18**	.01	-.01	**.27**	**.16**	*.03*	-.00
Domain-Specific Negative Self-Concept Numbing	.03	*.03*	.02	.02	**.05**	**.04**	***.04***	.02
Domain Specific Somatic Sensitivity	**.06**	.00	-.01	-.01	**.06**	.01	.02	-.01
Internalizing × Global Life Strain	*.05*	-.00	***.04***	*-.04*				
Internalizing × Recent Violent Victimization	--	--	--	--	-.00	.00	.02	-.01
R^2	**.19**	**.34**	**.26**	**.40**	**.22**	**.42**	**.25**	**.40**

***Note*. Values are in z-scores, standardized with respect to the outcome. Dummy coding was used for coding intersectionality of race/ethnicity and biological sex, "Constant" corresponds to White male (WF = White female, BM = Black male, BF= Black female, HM = Hispanic male, HF = Hispanic female). Calculations for the other ethnicity/gender specific intercepts can be computed by adding their respective coefficient to the reference value. All continuous covariates were mean centered prior to analyses. EXT = global externalizing factor, Fight = domain specific fighting factor, Drink = domain specific alcohol use factor, Drugs = domain specific drug use factor. Numeric values in bold = $p < .001$; bold, italics = $p < .01$; non-bold, italics = $p < .05$; non-bold, non-italics = not statistically significant.**

The first model (labeled "Model 1" in Table 3.6) tested for a 2-way interaction between the global *internalizing* factor and global life strain while controlling for the domain-specific internalizing factors. Model testing revealed a significant interactive effect on global externalizing ($\beta = .05$, $CI_{95\%} = .01, .08$) such that within the population average range of global

life strain, level of global *internalizing* showed no systematic correspondence with externalizing. However, as global life strain departed from the average range into the elevated range, elevations in global *internalizing* increasingly corresponded to elevations in externalizing above and beyond its additive effect with global life strain (results for the externalizing outcome are depicted in Figure 3.1A). Overall the interaction model fit the data better than a main effect-only model in which interaction effects were fixed to zero ($\Delta\chi^2$ (TRd) = 12.07, Δ *df* = 4, CD = 1.82, $p < .05$).

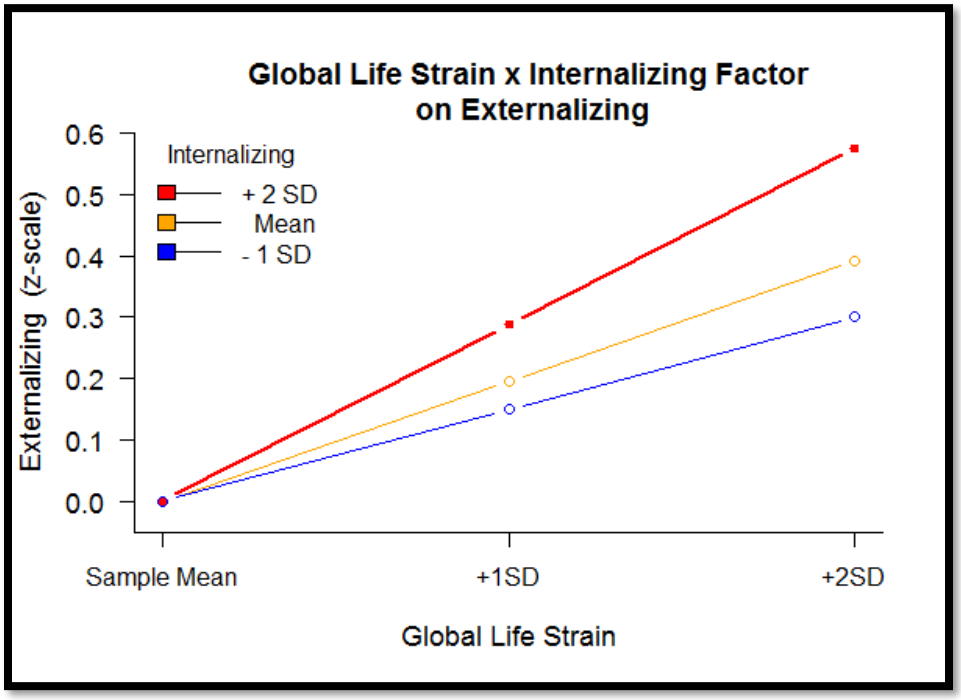

Figure 3.1A Global life strain interacts with internalizing in amplification of global externalizing behavior among adolescents

Contrary to prediction, Model 1 showed no significant interactive effect on domain-specific fighting (β = -.00, $CI_{95\%}$ = -.03, .02). Instead, statistically significant interaction effects emerged for domain-specific drinking (β = .04, $CI_{95\%}$ = .01, .08) and for domain-specific drug

use (β = -.04, $CI_{95\%}$ = -.07, -.01). The effect for domain-specific drinking was similar to the effect for global externalizing and is presented below in Figure 3.1B.

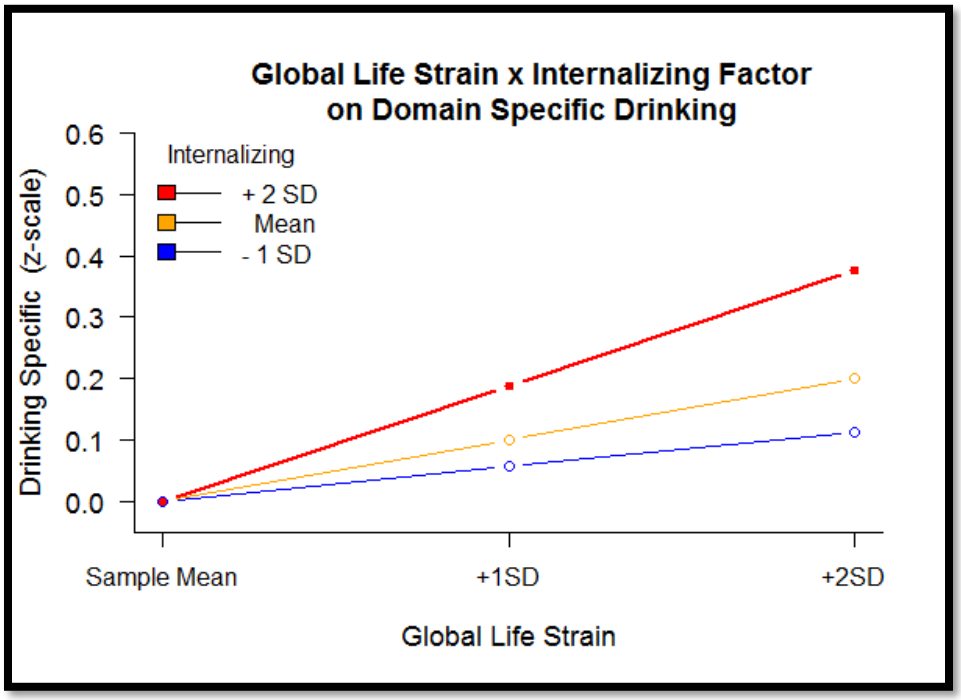

Figure 3.1B Global life strain interacts with internalizing to amplify adolescent alcohol use behavior unique from externalizing

In contrast, the interaction effect for domain-specific drug use was such that elevated *internalizing* was actually associated with lower domain-specific drug use at higher levels of global life strain. This interaction effect is displayed in Figure 3.1C.

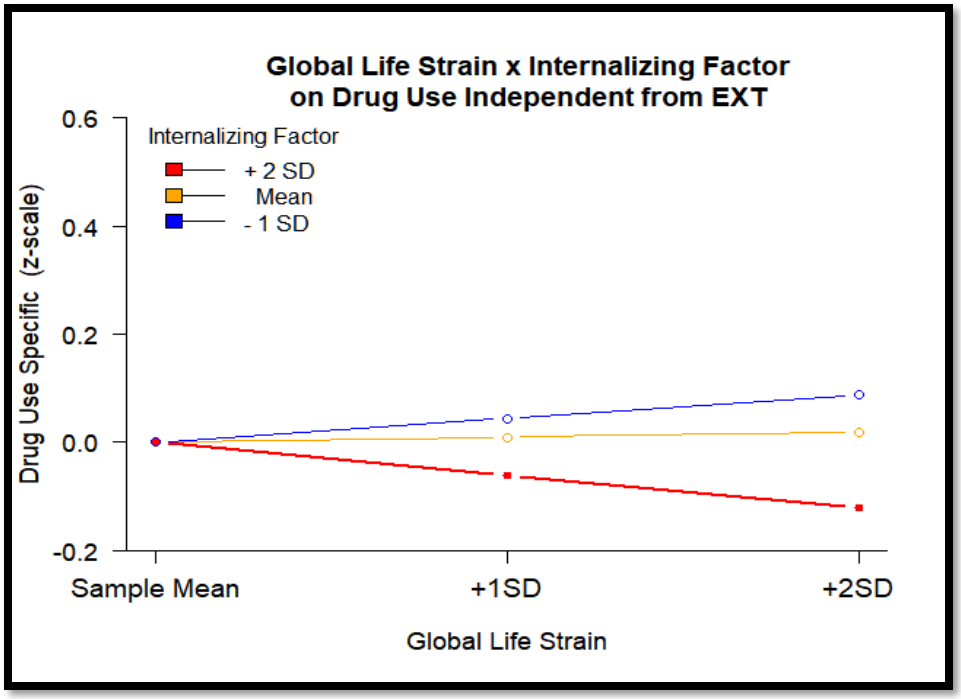

Figure 3.1C Global life strain interaction with internalizing on adolescent drug use independent from externalizing

The second model ("Model 2" in Table 3.6) tested for a 2-way interaction between the global *internalizing* factor and violent victimization on each of the externalizing outcomes. This model tested whether the effect of global *internalizing* would interact synergistically with recent violent victimization in the prediction of the externalizing spectrum outcomes. Results showed no significant interactive effects on any of the externalizing spectrum outcomes (results for the domain-specific fighting outcome are displayed in Figure 3.2). This suggests that the association between global *internalizing* and violent victimization on externalizing spectrum outcomes is most consistent with a cumulative-linear (dose-response) effect.

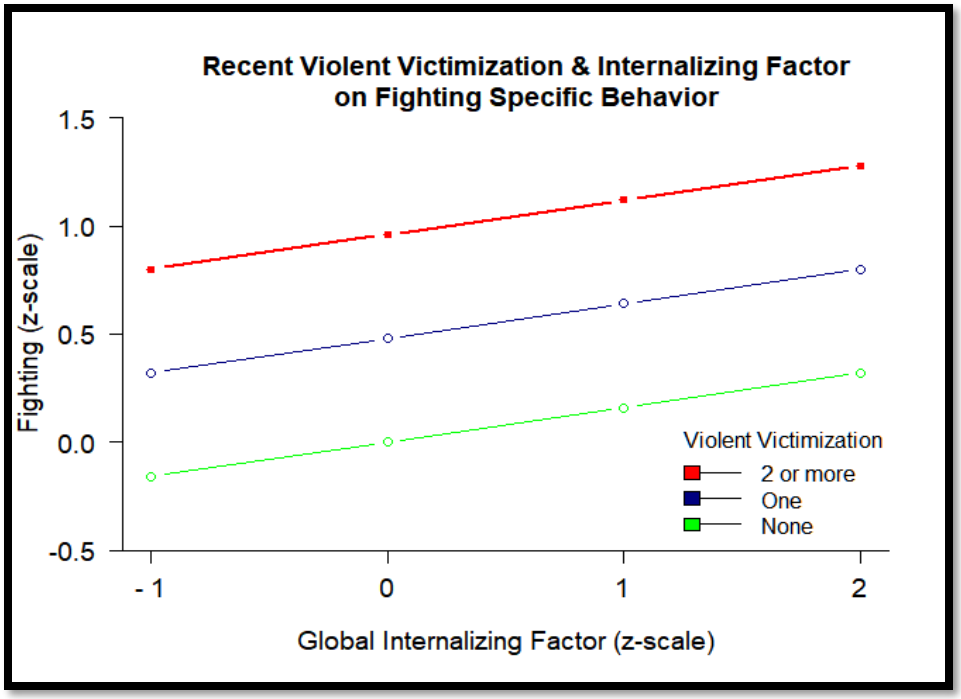

Figure 3.2 Additive effects of global internalizing and violent victimization predicting fighting unique from global externalizing behavior

Mediation Analyses

The final question pertaining to the nature of the interplay between the stress and trauma-related predictors and externalizing and domain-specific fighting was to examine the extent to which global *internalizing* might account for, or partially explain, the link between stress and trauma-related exposures and adolescent externalizing spectrum behaviors.

Results from initial model testing, in which the main effects of global life strain on externalizing and domain-specific fighting behavior were reduced after entering into the full multivariate model, suggest the possibility that part of the effect of global life strain might be operating by way of shaping more proximal beliefs, feelings, inferences, and behaviors over time. Mediation through the global *internalizing* factor, which in this study prominently captures an array of mood, somatic, arousal, affective, attentional, and self-concept domains, would be

broadly consistent with conceptualizations of complex trauma as a dual problem of exposure *and* adaptation. To explore this possibility, the next step was to apply a mediation model (displayed in Figures 3.3A-C) to test the following hypotheses:

1. The global *internalizing* factor will partially mediate the association between global life strain and externalizing.
2. The global *internalizing* factor will partially mediate the association between global life strain and fighting unique from externalizing.
3. Given the recent time frame of the violent victimization index (i.e., previous 12 months) and its temporally circumscribed nature relative to the global life strain composite, I predict that recent violent victimization will exert its effects on adolescent externalizing and domain-specific fighting more directly as compared to global life strain.

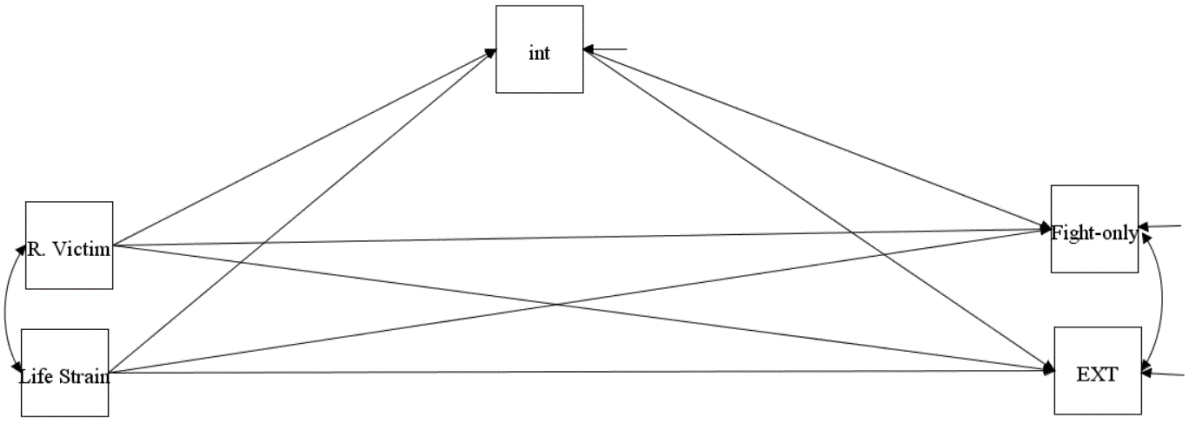

Figure 3.3A Hypothesized partial mediation model of the global internalizing factor mediating the association between the stress and trauma-related predictors and adolescent fighting and global externalizing behavior

***Note*. Int = *Internalizing* predictor, R. Victim = Recent violent victimization (occurring within the previous 12 months), Life Strain = Global life strain composite, EXT = global externalizing, Fight-only = fighting unique from global externalizing. Model controls for biological sex and age (covariates not pictured for presentation clarity).**

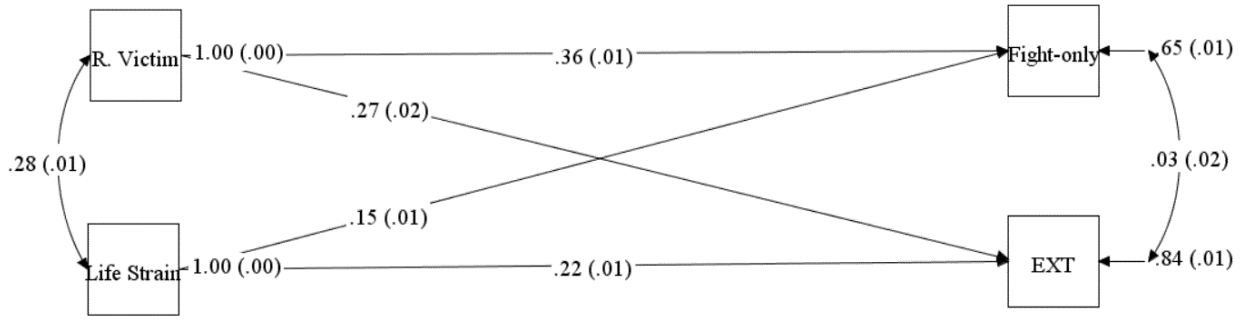

Figure 3.3B Estimates excluding indirect effect of global internalizing factor

***Note*. Estimates are presented in standardized form with standard errors in parentheses.**

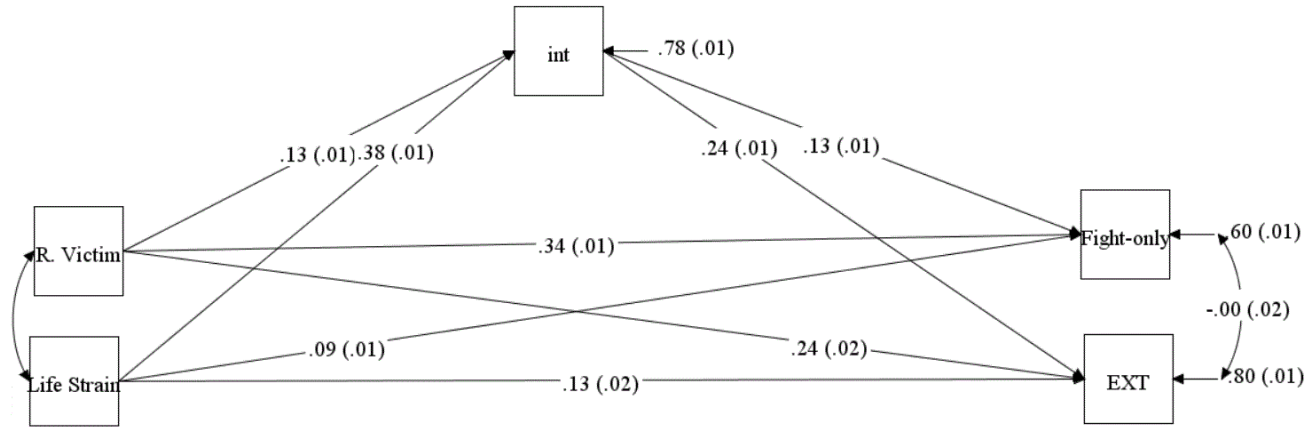

Figure 3.3C Mediation model results

***Note*. Estimates are presented in standardized form with standard errors in parentheses. Int. = Global *Internalizing*, R. Victim = Recent violent victimization (occurring within the previous 12 months), Life Strain = Global life strain composite, EXT = global externalizing, Fight-only = fighting unique from global externalizing. Model controls for biological sex and age (covariates not pictured for presentation clarity).**

The model fit the data well (RMSEA = .00, CFI = 1.0, TLI = 1.0, SRMR = .00).

Global life strain through internalizing factor. Consistent with hypotheses, global life strain showed statistically significant indirect effects on global externalizing (indirect = .09, *SE*

= .003, $p < .0001$) and domain-specific fighting (indirect = .05, *SE* = .004, $p < .0001$) by way of the global *internalizing* factor.

Recent violent victimization through internalizing factor. The indirect effect of violent victimization by way of the *internalizing* factor was modest for global EXT (indirect = .05, *SE* = .002, $p < .0001$) and for domain-specific fighting (indirect = .03, *SE* =.02, $p < .0001$).

Discussion

Recent reviews of the traumatic stress literature have concluded that more research is needed on complex trauma presentations in childhood and adolescence (Sweeny, 2013). To date, data are particularly sparse during the adolescent years between ages 12 and 18. This is a critical gap as this period marks the onset of puberty, which coincides with an array of normative biopsychosocial changes including increased orientation to social cognitions, salience of peers, and identity formation, as well as a period in which the amygdala and hypothalamus—key brain regions implicated in fear and stress related processes— are going through significant changes.

The ubiquity of normative biopsychosocial changes that coincide with the adolescent period, including greater impulsivity, risk-taking, and sensation seeking, may contribute to under-recognition of traumatic stress symptomology as manifested during adolescence. Although childhood and adolescent traumatic stress responses share common features with adult syndromes like post-traumatic stress disorder (DSM-5 PTSD), the application of adult PTSD criteria to adolescents fails to capture the full array of traumatic stress symptoms that adolescents are more likely to exhibit, including anxiety, depression, anger, and aggression. This is a key consideration as the transition to adolescence is also marked by an onslaught of mood and anxiety-related psychopathology and marks a stage of development in which misbehavior begins

to be more heavily sanctioned by society (e.g., increased suspensions, expulsions, enforcement of status offenses/arrests, detainment).

Thus, disentangling responses to traumatic stress from developmentally normative changes in risk taking during adolescence is critical for ensuring the long-term health and wellbeing of adolescent survivors of complex stress and trauma. Failure to do so places the victimized adolescent at disproportionate risk for punishment and stigmatization. Socially sanctioned consequences, discipline, and reinforcement strategies that may be effective for curbing disruptive behavior secondary to normative adolescent development may be ineffective or exacerbate the behaviors they are intended to curb among traumatized adolescents, further compounding feelings of social alienation, hopelessness, anger, and cognitions about competence, safety, and trust.

Using a large epidemiological sample of US adolescents ages 12–18, I sought to further understand the role of early complex trauma in the etiology of global externalizing behavior, specific rule-breaking behaviors unique from global externalizing (e.g., fighting- drinking-, drug use-specific behaviors), and adolescent sexual behavior. Focal predictors included an omnibus index of global life strain, an index of recent violent victimization, and a global adaptation index (global internalizing) comprising an array of mood, somatic/hyperarousal, affective, interpersonal, cognitive, and self-concept symptoms. These indices were informed by the broader clinical literature on chronic and complex post-traumatic stress during the adolescent period. Consistent with prior research (e.g., Suliman, et al., 2009; Foy, Ritchie, & Conway, 2012), findings revealed a cumulative, dose-response relationship between number and magnitude of stress- and trauma-related exposures/adaptations and levels of global externalizing, domain-specific fighting, earlier age at first sexual intercourse, and number of sexual partners.

In addition, although the global internalizing factor and global life strain were each important, adolescents who exhibited high elevations on *both* showed elevations in EXT (and in drinking unique from EXT), above and beyond their additive effects. One potential interpretation of this interaction is that elevated responsivity (sensitization) to context, when one's normative contexts have been characterized by strain across multiple levels, is a metabolically (energetically) taxing combination. With few or limited resources within one's ecological milieu (e.g., social supports, sense of security at home, school, and community) to buffer or protect against recurring environmental insult or challenge, this strain is more likely to exceed one's self-regulatory capacity. As such, adolescents contending with high global life strain, coupled with high responsivity (sensitization) to it, might do the best they can by drawing on what they can—acting out to bring attention to adults who might be able to help them, discharging negative affect, and/or otherwise leveraging means at their disposal, such as alcohol or marijuana in an effort to dampen or otherwise regulate their metabolically taxing and subjectively aversive reactivity to frequent and extended activations of the alarm system. Put another way, this interactive effect might reflect the downstream manifestation of experience-dependent sensitization to threat that undergirds the behavioral dysregulation (and regulatory based coping strategies) characteristic of more pronounced and enduring presentations of externalizing and alcohol use behavior. And the adolescent period might be a particularly susceptible stage of development for the reinforcement of coping behaviors related to the externalizing spectrum of behaviors, given the ongoing maturation of the socioemotional and cognitive control systems and the increased responsivity to dopamine.

The domain-specific alcohol and drug use behaviors showed minimal associations with any of the stress/trauma-related variables, suggesting that the elevated prevalence of substance

dependence associated with complex trauma in adolescence is likely subsumed by externalizing more broadly. However, the interaction results suggest one caveat, in which conjoint elevations on global life strain and global internalizing were associated with elevated domain-specific alcohol use, above and beyond EXT. This finding is consistent with the elevated rates of alcohol use associated with trauma-based disorders among adolescents (and adults).

The lack of association between the domain-specific substance use factors and stress/trauma-related variables suggests that these factors might be capturing recreational drinking and drug use behavior stemming from peer-influenced motivations. The impact of peer-based norms might also account for the mean-level differences that emerge between Caucasian adolescents and African American adolescents on the domain-specific drinking and drug use factors, wherein Caucasian adolescents showed significant elevations in these domains relative to African American adolescents. These differences are consistent with previous findings suggesting that Caucasian adolescents tend to drink more heavily and frequently and especially so among Caucasian adolescents from more socioeconomically advantaged backgrounds (Bleich et al., 2012; Keys et al., 2012).

Finally, consistent with mechanistic theory, mediation analyses suggested that part of the effects of global life strain on externalizing and domain-specific fighting operated indirectly by way of the global internalizing factor. In contrast, the effects of recent violent victimization on externalizing and domain-specific fighting were direct (i.e., not statistically mediated). However, recent violent victimization was measured by asking participants about events in the last 12 months. If effects of victimization on sensitization to threat depend on time since the event (blunted sensitivity followed by heightened sensitization), then aggregating across people who experienced an event within a one year time frame might lead to an inability to detect effects. On

the other hand, if victimization (or threat of victimization) happens on a routine basis, then circulating cortisol would be expected to be elevated and remain elevated (e.g., Friedman et al., 2007; Inslicht et al., 2006), consistent with a general hypoarousal. In this case, the individual might be less prone to endorse internalizing symptoms.

Although not conclusive, these findings are consistent with a potential role of childhood complex trauma sequalae underlying more pronounced presentations of externalizing and domain-specific fighting and alcohol use behaviors among adolescents.

Complex Trauma and Externalizing Psychopathology

Multivariate studies have shown that comorbidity among a spectrum of behaviors such as substance dependence, fighting, delinquency, and disinhibited personality traits can be modeled hierarchically with a global latent factor (labeled externalizing, EXT) that captures the common variance linking each phenotype within the spectrum, and specific factors that capture the distinctions among phenotypes within the spectrum (Krueger et al., 2002, 2005, 2007). Biometric analyses of these multivariate models suggest that variation in the EXT factor corresponds primarily to additive genetic variation, while both genetic and environmental variance underlie distinctions between specific forms of EXT spectrum behaviors (e.g., fighting only).

The costly and conspicuous nature of externalizing spectrum disorders has drawn considerable research interest in further delineation of the mechanisms and distinguishing features that underlie more severe presentations of externalizing psychopathology. Several prominent theories of externalizing psychopathology, and related constructs by other names (e.g., DSM-5 disruptive disorders: oppositional defiant disorder, disruptive mood dysregulation disorder, childhood onset conduct disorder with limited prosocial emotions, antisocial personality disorder, “life-course persistent,” psychopathy constructs) have emphasized trait-

level factors such as impulsivity, sensation seeking, callousness, unemotionality, and an array of neurocognitive and attentional correlates posited to be more genetically influenced and to distinguish more severe and persistent externalizing profiles from transient and developmentally normative rule-breaking behaviors during adolescence (e.g., Moffitt, 1993, 2006; Moffitt et al., 2008).

Children who are referred for mental health services to address pronounced EXT behaviors (or behaviors such as fighting unique from global EXT) are often assigned a progression of externalizing diagnoses over the course of development including attention deficit hyperactivity disorder, oppositional defiant disorder or disruptive mood dysregulation disorder, and childhood-onset conduct disorder with limited prosocial emotions (American Psychiatric Association, 2000; Cook et al., 2003; Ackerman et al., 1998; Copeland, Keeler, Angold, & Costello, 2007; Moffitt, 2006; Pardini & Frick, 2013). From this point, the groundwork is paved for a diagnosis of antisocial personality disorder (ASPD) or borderline personality disorder after the adolescent reaches age 18. Aside from the EXT spectrum, another common factor that links these diagnoses is poor treatment prognosis and stigmatization. Underscoring this point, many insurance providers, including Medicaid, will not cover mental health services for a diagnosis of ASPD.

Thus, as it stands currently, evidence for the existence of a coherent externalizing liability has been well replicated. However, the mechanistic origins of this liability remain unclear. Perhaps externalizing disorders and commonly comorbid attentional, learning, and personality disorders have been notoriously challenging to treat because we have fundamentally conceptualized, studied, and treated this end of the spectrum as an entity removed from its roots: a survival promoting adaptation to early, prolonged, and repeated overwhelming stress and

fear/threat based-activation (interpersonal and/or uncontrollable and developmentally sensitive survival-relevant threat/alarm activation).

Conflating a Correlate with the Core?

A potentially complicating factor for the continuum approach to externalizing is the differential pattern of correlates that have been linked to more severe and persisting versus developmentally normative EXT presentations (e.g., Moffitt, 1990, 1993). These are posited to include differences pertaining to onset of EXT behaviors, duration, severity, neurobiological correlates, psychiatric and medical comorbidities, responsivity to threat cues, attachment/interpersonal relationships, and academic performance among others. Consequently, some theorists have posited that there might be important qualitative distinctions between adolescents who populate the more normative versus elevated range of the EXT curve.

This general idea is most clearly exemplified by Terrie Moffitt's now classic developmental taxonomy of antisocial behavior, which posits that juvenile delinquency "conceals two qualitatively distinct categories of individuals, each in need of its own distinct theoretical explanation" (Moffitt, 1993, p. 674).[11] These hypothetical subtypes are labeled *Adolescence-Limited* (AL) and *Life-Course Persistent* (LCP).

According to this taxonomy, the *Adolescence-Limited* subtype is posited to be more socially influenced, arising from a "maturity gap" between pubertal and social maturation creating a sense of frustration that "encourages teens to mimic antisocial behavior in ways that

[11] Although many theories also attempt to explain EXT related behaviors, particularly within the domain of criminology, I focus on Moffitt's taxonomy due to its influential role in shaping the conceptualization and classification of DSM externalizing disorders (disruptive disorders and accompanying comorbidity), particularly subtypes of conduct disorder, the precursor diagnosis for Antisocial Personality Disorder.

are normative and adjustive" (Moffitt, 1993, p. 674). As adolescents attain the full rights and responsibilities of adults this subtype is posited to desist from antisocial behavior.

In contrast, the L*ife-Course Persistent* subtype is posited to be more genetically influenced, with childhood onset, more aggressive behavior and personality traits, distinct neurobiological correlates, and persistence into adulthood. The LCP subtype is also distinguished from the AL subtype by its greater correspondence with a range of early risk factors including harsh and inconsistent parenting practices and poverty (e.g., Moffitt, 1993, 2003, 2006). In sum, for the LCP subtype this theory posits that "children's neuropsychological problems interact cumulatively with their criminogenic environments across development, culminating in a pathological personality" (Moffitt, 1993, p. 674).

At the outset of her exposition Moffitt acknowledged that classification schemes of "antisocial persons" were not new but that "none of these classifications has acquired the ascendancy necessary to guide mainstream criminology and psychopathology research" (Moffitt, 1993, p. 674). Over the last 25 years, the LCP/AL classification scheme has accomplished this. However, I suggest that this might also be related to some of the longstanding challenges to gaining traction on outstanding questions, explanatory gaps, and improving prognosis and quality of life for adolescents and adults who exhibit more severe and persisting EXT presentations.

Integrating results from the present study with existing research and concepts from the child and adolescent complex trauma literature and basic science on stress response, threat and the adaptive alarm system, I offer a distinct theoretical explanation to account for the patterns of EXT (and domain-specific fighting/drinking behaviors) among individuals who have

traditionally been grouped into the "LCP" labeled category and clinical derivatives (and commonly accompanying comorbidity) of this framework.

I suggest that an interaction between the historical context of the 1990s moral panic over adolescent delinquency and the alliance between criminology and psychopathology perspectives served to shape the conceptualization and operationalization of the LCP construct such that its core ("offending") and its correlates (early macro- and micro-level adversity) have been transposed, and survival-promoting, experience-dependent adaptations (neurobiological and attentional differences, resting cortisol, responsivity to threat-related cues) have been conflated with causal deficits and dysfunction. Specifically, in the theory that I propose, "offending" is a *correlate*, not the "LCP" core, and chronic stress and trauma is the "LCP" *core*, not a correlate.

In this way I offer a response to Moffitt's call that the LCP construct is in need of a theoretical explanation. However, in doing so I offer a theoretical explanation that accounts for the LCP observations yet is fundamentally incompatible with the LCP construct as conceptualized currently.

An Alternative Theoretical Framework: Life-Course Persistent Oppressive Stress and Trauma

Rather than qualitatively distinct *types of persons*, I posit that developmentally transitory versus more severe and persisting externalizing behavior is rooted in qualitatively distinct developmental profiles of *exposure* (and experience-dependent adaptations) to stress and fear/threat-based activation: one that is linked to population normative range stress and fear activation and adaptations across the balance of key stress-sensitive stages of early development (including adolescence) and one that is linked to highly elevated, non-population normative levels of overwhelming and oppressive stress and fear/threat-based activation (specifically,

developmentally sensitive survival-based threat activations) and adaptations across successive key stages of early development and into adolescence.

This distinction is qualitative in recognition that the same event can exert a differential impact on one's stress response and recovery/repair processes depending on developmental timing, previous stress and trauma exposure history, and the extent that one's routine ecological milieus (home, school, neighborhood, community) and primary socioemotional supports serve to facilitate, prolong, or impede healing/recovery processes. As such, the features that are predictive of trauma-stress severity and complexity: onset, frequency, magnitude, duration, controllability, social-evaluative/interpersonal threat (post-puberty), and availability/access to coping resources, will likewise be relevant in estimating the severity and impact of any given series or configuration of chronic stress and trauma-related events and exposures. These factors will also be relevant to predicting adaptations, coping behavior patterns/tendencies, and the types of behavioral action tendencies most likely to be drawn on (e.g., *avoid*: anxiety; *escape*: fight, physical flee, mental flee via dissociation; *shut down*: reduce pain under context where threat is omnipresent or unavoidable and uncontrollable).

In short, after taking the above features into account, then all else being equal, those who are exposed to the greatest number of adversities in conjunction with the fewest buffering or mitigating factors will be expected to more prominently manifest an array of developmentally sensitive adaptations to optimize survival in the context of the input from their accumulated lived experience—with greater weight given to survival-relevant threats that elicit the alarm response early in development (as systems of attachment, stress responsivity, circadian rhythm, and other autonomic processes are being calibrated) and proceed across successive key developmental stages, including early childhood when the hippocampus is rapidly developing, and adolescence,

a key stage for identity formation and a period in which socioemotional, cognitive control, and neurobiological and hormonal systems, including the HPA system, are experiencing significant reorganization and maturation (Sisk & Zehr, 2005; Steinberg, 2010).

However, because the stress/trauma exposure histories between these subgroups will generally differ in quantity of adverse experiences, quantitative scales that encompass a wide and diverse range of both proximal and distal stress/trauma-related exposures and experiences (to include normative ecological milieus of relevance for coping) might be expected, in many cases (though not always), to serve as an adequate proxy to differentiate between more normative range exposure histories from the more elevated range characteristic of complex trauma.

Clinical Implications

Defining and labeling more severe presentations of externalizing under an extreme stress- and trauma-related disorder would have significant implications for access to health services and facilitate the dismantling of practices that disproportionately stigmatize and criminalize these adolescents, placing them in secure detention facilities that compound traumatic stress, reinforce trauma cognitions about safety, trust, and competence, add to family strain, and have been shown to produce iatrogenic effects (e.g., DeVeaux, 2013; Gatti, Tremblay, & Vitaro, 2009; Mendel, 2011; Vieraitis, Kovandzic, & Marvell, 2007), and compound their hardship and barriers following release (e.g., Buffington, Dierkhising, & Marsh, 2010; Kubiak, 2004; Mendel, 2011). ASPD, which is far more likely to be diagnosed when preceded by a CD diagnosis, is not covered by many insurance companies, including Medicare, suggesting, if the present theory has merit, that the clinical status quo effectively excludes among the most victimized individuals who have among the fewest resources to draw on to cope (e.g., family, social network, financial, academic record, work history) from receiving clinical services. In many cases then, this leaves

the criminal justice system to repeat at the institutional level a troubling pattern of which many survivors of complex trauma have been well acquainted: a relationship in which the same entity that they depend on to procure their life-sustaining needs is also a source of fear, chronic stress, and trauma, both during detainment and the detainment-related hardships that follow release (DeVeaux, 2013; Mendel, 2011).

Additional Considerations and Limitations

There are several methodological and conceptual considerations and limitations that merit comment. Foremost, the present study is based on cross-sectional data. Although I have conceptualized complex trauma-related variables as "predictors" and sexual activity and externalizing spectrum variables as "outcomes," these designations are arbitrary as temporal inferences cannot be established with correlational data. Consequently, this study cannot discern whether complex trauma-related factors precede onset of externalizing spectrum pathology or whether this relationship may operate in the reverse direction (e.g., elevated externalizing behaviors predisposing an individual to elevated risk for adverse outcomes across a range of contexts and situations). From a conceptual standpoint, this relational ambiguity is not particularly problematic for the present study. Indeed, there is no clear "starting point" for complex trauma; rather, it is a process that evolves over the course of development. The same can be said for externalizing pathology. Moreover, it is quite likely this relationship is bidirectional, operating in a reinforcing feedback loop. Indeed, the presence of gene-environment correlation and interaction does not diminish the compatibility nor potential utility of a trauma informed approach.

Another consideration with cross-sectional data is that it precludes establishing temporal precedence for testing mediation. While mediation in the classical sense requires temporal

precedence to be established, in this case that was not desirable since the objective was to examine the extent to which contributions from global life strain might have been subsumed by the *internalizing* factor in our model. Moreover, from a conceptual standpoint, it is not my contention that the variation captured by the *internalizing* factor is a meaningfully separable component of liability; rather, I conceptualize it as an accompanying consequence (adaptation) of prolonged exposure to chronically stressful situations. This interpretation would also be consistent with research findings that internalizing rarely precedes externalizing (e.g., Fergusson & Horwood 2002; Rutter et al. 2006).

The current study relies on self-report data. The limitations of self-report have been widely recognized, and to mitigate this limitation we incorporated parent and interviewer report as relevant. However, we also note that the participants' perception is of fundamental importance when they are reporting on domains related to their personal sense of safety, perceptions of acceptance and belonging, and beliefs about themselves, others, and various somatic and interoceptive perturbances. Moreover, research has also shown that when it comes to reporting on stress, subjective reports are more predictive of long-term health outcomes and biological embedding (Cohen, Alper, Adler, Treanor, & Turner, 2008; Gianrosetal, 2007; Singh-Manoux, Marmot, & Adler, 2005; Rutter, 2016). Nevertheless, some of the domains in which adolescents were asked to report on were sensitive such as lifetime forcible rape (which was only asked of girls at Wave 1) and the externalizing spectrum outcomes which included questions about illegal drug use, alcohol use, fighting, and delinquent behaviors. The Add Health at-home interview took measures to mitigate privacy concerns and demand characteristics by using a computer based interface for potentially sensitive questions rather than a face-to-face interview.

Although experiences of physical and sexual abuse are often core components of complex trauma, I nevertheless opted to omit this information from the composite construction for the following reasons: (1) self-report abuse information was not provided until Wave 3, at which point many of our original sample participants, and disproportionately those with greater global life strain, were missing on these data; (2) a core feature of post-traumatic stress is avoidance of reminders or cues of the traumatic event, and this is especially so as a function of severity and psychological trauma (Holahan & Moos, 1987). To the extent individuals who are more severely traumatized are less likely to disclose their experiences with abuse, this poses problems of contamination. In support of these concerns, the correlations between child protective services visit and an abuse composite that included physical, emotional, and sexual abuse, was only r = .07 ($p <$ .001); in contrast, the global *internalizing* factor and the global life strain composite were both correlated upward of r = .15 ($p <$ .001) with child protective service involvement—more than any combination of the self-report abuse composites. Finally, many studies, particularly with the Add Health data, have examined the self-report abuse composites but few have examined life strain and traumatic stress by indexing the key features that commonly co-occur with complex trauma. Given the overwhelming evidence that (1) abuse does not occur in isolation, and (2) prognosis is predicted by cumulative stress/adversity, often across multiple domains (i.e., number of risk factors in the absence of buffering or protective factors), this methodological choice is, in fact, one of the unique strengths of the present study. Thus, for these reasons I opted to omit explicit self-report abuse items from this study and index these adversities indirectly.

The composite index of recent violent victimization was limited to endorsement from a predefined list of events. This is a noteworthy limitation, as violent victimization types outside of

these events—such as intimate partner violence and sexual violence, which disproportionately targets girls or witnessing of domestic violence—were not included in this index.

Limitations of the question wording in the Add Health data as it pertains to age at first sexual intercourse and number of sexual partners precluded our ability to ascertain with 100% certainty that all reported first sexual encounters and sexual partner count were in fact consensual encounters. To reduce the likelihood of including nonconsensual AFS encounters, adolescents who reported an age at first sex before the age of 11 were coded as missing. Although we controlled for forcible rape in the analyses of our sexual activity outcomes, this definition of rape excludes non-forcible rape encounters which is a limitation of the Wave 1 data. Finally, forcible rape was not asked of male adolescents at Wave 1, which is a limitation of the Add Health data.

The composite index of early disruptions and loss of a primary caregiver did not include parental incarceration, which is a limitation, particularly as it relates to understanding strain among racial and ethnic minority youth and youth from lower socioeconomic backgrounds as the War on Drugs and associated mass incarceration constitutes a source of caregiver loss that has disproportionately affected adolescents within in these communities.

Finally, Add Health participants are drawn from a school-based design and as such these data excluded adolescents who withdrew from school by Wave 1, an unfortunate limitation for the present study given the higher prevalence of high school non-completion among youth with complex trauma in their backgrounds (Rumberger, 2011).

Future Directions

In the closing words to the seminal introduction of her theory, Moffitt (1993) wrote, "Delinquency theories are woefully ill-informed about the phenomenology of modern teenagers

from their own perspective. I fear that we cannot understand adolescence-limited delinquency without first understanding adolescents" (p.696).

In a parallel vein, I suggest that disruptive disorder and externalizing theories are woefully ill-informed about childhood complex trauma both from the perspective of the child trauma literature and the phenomenology and insights from survivors of complex trauma from their own perspective (and across the lifespan). I fear we cannot understand more severe and persisting forms of externalizing psychopathology without first understanding adolescents, and the interface between childhood complex trauma and the developmental period of adolescence. Given the heterogenous developmental profiles that can comprise complex trauma and its low base rate within the general population, future research may benefit from conducting within person (N=1) studies to improve our clinical understanding of this important phenomenon.

Conclusion

In spite of the phenotypic overlap between indicators of normative adolescent development and many of the behaviors traditionally conceived as externalizing (impulsivity, aggression, risk-taking, substance use), the interface between traumatic stress sequalae from complex trauma and the biopsychosocial changes that characterize onset and course of adolescence (ages 12–18) has received minimal attention.

This is a critical gap as adolescence marks a period of development in which childhood trauma experiences begin to manifest more prominently in psychosomatic form (e.g., hyperarousal, hypervigilance, sleep disruption, irritability, anger, depressed mood, self-reproach, impaired concentration). At the same time, adolescence is also a notable stage of development wherein disruptive behaviors begin to be sanctioned more heavily in society, particularly within

the school context where police officers ("school resource officers") are increasingly on hand, and traditionally administrative punishments have been increasingly replaced by criminal citations and arrests.

Summary

This study used a large US-population-based sample of over 14,000 adolescents ages 12–18 to examine the interplay between an array of composite variables indexing core features of complex trauma in predicting global externalizing behavior, domain-specific fighting, alcohol use, and drug use behaviors and adolescent sexual behavior. Results from multivariate testing revealed a cumulative dose-response association between the complex trauma-related predictors and global externalizing, fighting behavior unique from externalizing, timing of first sexual intercourse, and number of sexual partners. In addition, significant interaction effects emerged such that global life strain and high global *internalizing* interacted synergistically to predict elevated externalizing and domain-specific drinking among adolescents.

Mediation analyses revealed significant indirect effects from global life strain to global externalizing and domain-specific fighting by way of the *global internalizing* index. These results suggest that childhood complex trauma may be relevant for an array of adolescent health behaviors particularly global externalizing and alcohol use and fighting apart from externalizing. Trauma-informed treatment approaches may have utility for a range of externalizing spectrum disorders that to date have been largely refractory to treatment intervention. This possibility awaits future testing.

Grand Discussion

Advances in statistical modeling at the outset of the millennium ushered a proliferation of behavioral genetic studies examining the dynamic interplay between genes and measured environmental contexts. Despite this popularity, however, the sample composition of much behavioral genetic research to date has been comprised of largely homogenous middle-class, European American samples. Consequently, the universality of many findings remains unclear, and the role of contextual factors related to intersecting systems of privilege and oppression remain poorly understood in relation to the etiology of adolescent sexual health and externalizing spectrum behaviors. Thus, the overarching objective of the present series of studies was to better understand the contextual roles of several markers of privilege and oppression (racial stratification, social class, and chronic stress and trauma) in relation to adolescent sexual health and externalizing spectrum behaviors.

In Study 1 we examined whether genetic and environmental influences on timing of first sexual intercourse varied as a function of two broad markers of social stratification (African American and Hispanic minority status versus Caucasian majority status; socioeconomic status as indexed by mean parent educational attainment) and a narrower marker of intrafamilial strain (early father absence). Findings from the multivariate interaction model revealed significant gene × environment interaction for socioeconomic status and racial/ethnic minority status such that genes were a moderate predictor of timing of first sexual intercourse among youth from high-SES households and racially privileged backgrounds (Caucasian adolescents) but were not predictive of individual differences in sexual timing among adolescents from lower-SES households and racially marginalized backgrounds (African American and Hispanic youth).

Ideologies of individualism have a longstanding precedent in undergirding discourse on sexual behavior and reproductive health. Findings from Study 1, however, underscore the point that the utility of an approach to adolescent sexual behavior that focuses on genes and biological mechanisms to explain individual differences may critically hinge upon the sociohistorical context of the population of interest. Failure to consider this possibility can pave the way for practices that systematically undervalue/under-attend to social or structural environmental mechanisms that shape sexual behavior, while systematically over attending to the role of genes and individual-level explanatory factors that presuppose as normative, the nebulous constellation of benefits and freedoms conferred by structural and institutional privilege.

In Study 2 we examined the universality of problem behavior models that conceptualize early sexual activity as an indicator of a more generalized propensity toward deviant behavior. Specifically, we examined whether patterns of association between adolescent sexual activity and externalizing behavior previously observed in Caucasian samples extend to African American youth. Findings showed that the associations between externalizing and earlier timing of first sexual intercourse and more sexual partners were attenuated among African American adolescents relative to Caucasian adolescents, and to the extent that they overlapped, the mechanisms underlying their associations were distinct. Genetic predispositions toward EXT spectrum behaviors accounted for earlier AFS among Caucasian youth but not African American youth. For African American youth, the overlap was fully accounted for by shared environmental factors. For number of sexual partners, genes played a more prominent role in the overlap with EXT among all adolescents except for African American girls, in which shared environmental factors fully mediated the link. Overall, findings from Study 2 suggest that problem-behavior models do not adequately explain individual differences in sexual activity among African

American youth. These findings highlight how erroneous inferences can be drawn when findings from studies conducted on White and predominantly middle-class samples are presented (explicitly or implicitly) as universal discoveries.

Taken together, Studies 1 and 2 underscore the important role that intersecting systems of privilege and oppression can have in shaping the relative contribution of genetic and environmental mechanisms underlying individual differences in timing of sexual behavior and the correspondence between externalizing behavior and sexual behavior during adolescence. Additionally, in both studies a similar pattern emerged in which genetic differences account for a greater proportion of individual differences in adolescent sexual behavior and its overlap with externalizing behaviors for adolescents from more socially privileged backgrounds. In contrast, for adolescents from more socially marginalized backgrounds, individual differences in these behaviors were almost exclusively accounted for by environmental mechanisms.

Together, these studies underscore the larger point that the failure to appreciate the boundaries of inference can have major social and ethical consequences by shaping perceptions (researchers and broader public alike) that extend beyond the data and, as emphasized in Study 2, can perpetuate longstanding White supremacist narratives and racial stereotypes that can pose harm in very tangible ways and at scale through policy.

Although Studies 1 and 2 did not identify the environmental mechanisms that account for individual differences in adolescent sexual behavior among racial and ethnic minority youth and youth from lower-SES households, ecological theories have posited early life stress to exert a causal role on earlier sexual onset and reproductive behavior. Notably, aside from earlier sexual onset, childhood exposure to chronic stress and trauma is also linked to greater engagement in a range of health-risk behaviors including substance dependence, impulsivity, aggression,

delinquency, and attentional problems—externalizing behaviors. Complicating matters, these behaviors are also commonly associated with normative adolescent development.

One possibility is that adolescent risk taking that exceeds developmental normativity could be a byproduct indicator of adaptation to a chronically harsh and stressful environment. This is an important consideration since adolescence marks an important turning point in development wherein earlier childhood trauma experiences begin to manifest more prominently in trauma stress symptoms. At the same time, adolescence is a notable stage of development in which disruptive behaviors begin to be sanctioned more heavily in society.

Thus, in Study 3, I examined these associations more closely, advancing the possibility that externalizing spectrum behaviors that exceed the population-normative developmental range might be more parsimoniously accounted for as an entity contextualized by its roots, which I posit to be complex trauma and survival prioritizing adaptations and trauma-stress sequalae.

In consideration of the adult narratives that have conceptualized adolescent behavior from a problem perspective, in the remaining discussion I more critically consider how these perspectives and historical context more broadly might inform contemporary conceptualizations of EXT spectrum behaviors ("disruptive disorders" in DSM vernacular). In accordance with my overarching objective to apply a critical examination of context, I consider more fundamentally how it might be that rule breaking behaviors came to define the core pathology of externalizing behaviors more broadly as opposed to the psychological pain that underlies these visible manifestations of distress. To this end, I examine how the potential roles of power and privilege, historical context, dominant narratives, implicit biases, language and the tools of science might contribute to leading us astray from wider consideration of the possibility of complex trauma and

related adaptations undergirding more severe forms of externalizing spectrum behaviors (DSM disruptive disorders).

Shaping the Narrative: The Role of Power and Privilege in Defining Psychological Distress

The question of how to define psychological trauma has been a source of long-standing academic debate and is an issue that at its heart is intimately related to power and privilege: the power and privilege to be heard, the power and privilege to impact one's own narrative, and the power and privilege for one's psychological pain to be recognized and validated.

The progression of revisions to PTSD within the American Psychiatric Association's Diagnostic and Statistical Manual (DSM) is illustrative of the impact of critical mass advocacy in wielding influence over the recognition of certain presentations of psychological distress as valid. This was exemplified most clearly with the role of combat veterans and their family members in advocating for a diagnosis that linked service members' traumatic stress symptomology to their wartime combat. The women's liberation movement also had a role in catalyzing changes to PTSD criteria, through increasing public consciousness that sexual violence could also result in traumatization. And more recently PTSD criteria for DSM-5 was revised to recognize the impact of vicarious trauma increasingly noticed among first responders, who, within the course of their work can accumulate multiple vicarious exposures to potentially traumatic events.

The history of critical mass in impacting recognition of psychological trauma and shaping the boundaries of its precise classification leads one to wonder about the voices that have been systematically missing or marginalized. Who might these be? And how might the nuances of their psychological distress be conceptualized or addressed within our current institutions?

Many voices have been missing, marginalized, or otherwise underrepresented, and more research will be needed to competently address this diversity. In the present series of studies, I focused on adolescents. In Study 3, my aim was to focus on that subset of adolescents who have been failed on many levels, and whose voices are often marginalized, devalued, or silenced altogether through respected systems and trusted institutions that combined, wield the power and authority to communicate messages directly to the adolescent—and to others about the adolescent—that weave a highly convincing narrative that they are the source of the core problem, that their voices are untrustworthy or unreliable, their intellect is deficient, their capacity to contribute to society is minimal, and that resources would therefore be better spent trying to contain their risk to others.

These narratives should give us pause to consider the following: Where did they come from? Who wrote them? Who is missing? What is the relationship between the author and the subject? And finally, how and in what way might these factors be relevant in shaping the contours and contemporary conceptualizations of externalizing?

Shaping the Narrative: Historical Context, Overview, and Role of an Influential Developmental Taxonomy in the Conceptualization and Classification of Adolescent Externalizing

In 1993, crime became the most covered topic on the US national evening news as moral panic about increasing crime swept the nation (Moriearty & Carson, 2012). Television programming and popular news magazines "created a veritable onslaught of information that both implicitly and explicitly linked adolescents, and African American adolescents in particular, with violent crime" (Moriearty & Carson, 2012, p. 307). Legal scholars Moriearty and Carson (2012) described the cultural climate leading into the 1990s including the shift in focus onto adolescents:

> … deep into America's Wars on Crime and Drugs, an incursion commenced against a target that had, to that point, remained largely outside the crosshairs. Prompted by rising crime rates and a handful of high-profile incidents, politicians, the media, and much of the public became consumed by what they characterized as a looming threat. … This incursion bore many of the classic features of other modern American social wars: rhetorical excess, political extremism, graphic media, punitive policies, and, perhaps most critically, the casting of the enemy as a moral reprobate. To this end, the image of the adolescent "super-predator," a term a Princeton professor coined in 1995, was a particularly salient symbol.
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> … During the 1990s, nearly every state in the country enacted laws that made it easier to try kids as adults, expanded criminal court sentencing authority over juvenile offenders, and modified or eliminated juvenile court confidentiality laws. These changes have been called the "broadest and most sustained legislative crackdown ever on serious offenses committed by youth within the jurisdictional ages of American Juvenile Courts." (p. 281–282)

It is within this historical milieu that Terrie Moffitt's highly influential developmental taxonomy of antisocial behavior emerged (Moffitt, 1993), introducing the idea that the classic age-crime curve[12] (Farrington, 1986) and, more specifically juvenile delinquency, "conceals two qualitatively distinct categories of individuals, each in need of its own distinct theoretical explanation" (p. 674).

In this taxonomy, Moffitt distinguishes between two hypothetical subtypes labeled "life-course persistent" (LCP) and "adolescent limited" (AL) "antisocial" behavior. The *adolescent-limited* subtype begins in adolescence, posited to arise from a "maturity gap" between pubertal maturation and social maturation and that "encourages teens to mimic antisocial behavior in ways that are normative and adjustive" (Moffitt, 1993, p. 674). Accordingly, "instead of a biological basis in the nervous system, the origins of adolescence-limited delinquency lie in youngsters' best efforts to cope with the widening gap between biological and social maturity"

[12] The age-crime curve refers to population level patterns in which prevalence of offending increases over adolescence, peaking in the late teens and sharply declining thereafter (Farrington, 1986; Piquero, Farrington, & Blumstein, 2007).

(p. 689). As such, Moffitt regards the AL subtype of delinquency as an "adaptive response to contextual circumstances" (p. 689). As adolescents attain the rights and responsibilities of adults, antisocial behavior is posited to resolve.

In contrast, the *life-course persistent* subtype is posited to be more genetically influenced, with childhood onset, more aggressive behavior and personality traits, neurobiological "deficits," and persistence into adulthood. This subtype is also distinguished from the AL subtype by its lower base rate (estimated population prevalence is approximately 5-8% and predominantly male) and greater correspondence with early risk factors including harsh and inconsistent parenting practices and poverty (Moffitt, 1993, 2003, 2006).

Moffitt elaborates on her image of the LCP subtype:

> Already adept at deviance, lifecourse-persistent youths are able to obtain possessions by theft or vice that are otherwise inaccessible to teens who have no independent incomes (e.g., cars, clothes, drugs, or entry into adults-only leisure settings). Life-course-persistent boys are more sexually experienced and have already initiated relationships with the opposite sex. Life-course-persistent boys appear relatively free of their families of origin; they seem to go their own way, making their own rules. As evidence that they make their own decisions, they take risks and do dangerous things that parents could not possibly endorse. As evidence that they have social consequence in the adult world, they have personal attorneys, social workers, and probation officers; they operate small businesses in the underground economy; and they have fathered children (Weiher, Huizinga, Lizotte, & Van Kammen, 1991). Viewed from within contemporary adolescent culture, the antisocial precocity of life-course-persistent youths becomes a coveted social asset (cf. Finnegan, 1990a, 1990b; lessor & Jessor, 1977; Silbereisen & Noack, 1988). Like the aforementioned bird calls that were mimicked by hungry tamarin monkeys, antisocial behavior becomes a valuable technique that is demonstrated by life-course-persistents and imitated carefully by adolescence-limiteds. (Moffitt, 1993, p. 687)

In sum, this theory postulates that for the LCP subtype, "children's neuropsychological problems interact cumulatively with their criminogenic environments across development, culminating in a pathological personality" (p. 674).

At the outset of her exposition, Moffitt acknowledged that classification schemes of "antisocial persons" were not new but noted that "none of these classifications has acquired the ascendancy necessary to guide mainstream criminology and psychopathology research" (p. 674). In the 25 years since its debut a prolific body of work has developed from this framework influencing criminology, forensic risk assessment, and diagnostic classification schemes for two revisions of the DSM disruptive disorders[13]. Across each of these domains the life course persistent prototype has remained a central focus.

Despite its multidisciplinary influence, however, the taxonomy has also faced criticism related to its semi-parametric group-based modeling (SPGM) methodology, the generous bandwidth for what constitutes a reproduction of subtypes, and the overall strength of the data supporting the taxonomy (e.g., Bauer & Curran, 2004; Erosheva, Matsueda, & Telesca, 2014; Sampson & Laub, 2003; Skardhamar, 2009, 2010), particularly among more recent meta-analyses of prospective longitudinal studies (Odgers et al., 2008; Jollifee, Farrington, Piquero, MacLeod, & van de Weijer, 2017; Jolliffee, Farrington, Piquero, Loeber, & Hill, 2017). Furthermore, even with amendments to accommodate empirical inconsistencies[14], basic

[13] For DSM-IV a childhood onset subtyping scheme was added to the diagnosis of Conduct Disorder (CD) to distinguish a form of the disorder posited to persist into young adulthood as compared to adolescent onset forms that were posited to be developmentally transitory diminishing by the end of adolescence. In DSM-5 a further specifier was added arising from research in the child psychopathy literature on a construct labeled 'callous-unemotionality,' (CU) in order to improve specificity in distinguishing a more severe or "life-course persistent" subgroup of individuals within the childhood-onset subtype of CD (Frick, Cornell, Barry, Bodin, & Dane, 2003; McMahon, Witkiewitz, Kotler, & Conduct Problems Prevention Research Group, 2010; Frick & Viding, 2009; Pardini & Fite, 2010). As such, it was agreed upon to add a specifier labeled "with limited prosocial emotions" to DSM-5 Conduct Disorder to distinguish a more severe and persistent subtype within the childhood-onset subgroup that is posited to persist into adulthood.

[14] For example, adding intermediary subtypes such as 'low-level chronics' [Moffitt, 2006a], an adult onset subtype (Moffitt, 2006b); a childhood limited classification to distinguish early onset youth who do not continue into adulthood [Moffitt, 2006]; extending the adolescent-limited subtype into emerging adulthood, a "prolonged" adolescent offender (Salvatore, Taniguchi, & Welsh, 2015). An example pertaining to the Diagnostic and Statistical Manual of Mental Disorders includes revising the DSM-IV (1994) classification for Conduct Disorder for DSM-5 (2013) to add the specifier "with limited prosocial emotions" to the childhood-onset subtyping scheme for CD to address the heterogeneity in etiology of the childhood-onset subtype of CD (Frick & Viding, 2009) including findings that a

questions remain unanswered[15], explanatory gaps continue to arise, and treatment prognosis for older adolescents and adults remains poor.

Additionally, the interrelations among many of the externalizing spectrum disorders (ADHD, ODD, DMDR, CD, ASPD, SUD) with one another and with symptoms or disorders outside the EXT spectrum remain poorly conceptualized and inferences are complicated by changing diagnostic schemes across revisions of the DSM that impact content and rule-based constraints on comorbidity among individual disorders.

Moreover, in spite of the routine appeals to the clinical utility of distinguishing more severe subtypes of EXT using the LCP-inspired frameworks—that is, so that those most in need of treatment/services can be prioritized—evidence suggests that in practice the opposite seems to be happening (e.g., Mansion & Chassin, 2015). According to recent data, young people within the juvenile justice system, and particularly adolescents of color, who are either perceived to have more severe EXT (e.g., earlier age at first arrest, prior contact with the juvenile system, an aggression related offense) or are simply older adolescents, are actually *less* likely to receive treatment services during incarceration or to be connected with services within the community upon release. Mansion and Chassin noted, "This finding is consistent with previous literature that suggests juvenile courts are less likely to find older juveniles amenable to treatment. One reason

substantial proportion of youth who meet childhood onset criteria do not persist with conduct problems beyond the adolescent period (Odgers et al., 2008).

15 For example, upon reviewing the state of the research on offending careers, a study group concluded that "Surprisingly little is known even about the most basic questions of how many juvenile offenders (ages 15-17) persist into adult offending (at ages 18 or later), and what factors in the juvenile years predict persistence into the adult years. More needs to be known about processes that may influence offending between ages 15 and 29, especially individual factors (including those that tend to develop with age, such as psychosocial maturity, impulse control, cognitive decision-making, executive functioning, risk taking, emotion regulation, and other factors that tend to emerge with age such as mental health problems). More needs to be known about how life circumstances, such as education, employment, romantic relationships and cohabitation, substance use, and peer relationships) influence the development of offending. In addition, there is a need to better understand how individuals' routine activities and their neighborhood and situational contexts influence offending" (Farrington & Laub, 2013, p.18).

for this finding may be that the court believes older offenders are likely not able to be rehabilitated before adulthood (Gee, 1983; Slobogin, 2013)" (2015, p. 241).

Finally, despite the abundance of studies inspired by the developmental taxonomy, the overrepresentation of individuals from poor and working class backgrounds and among racial and ethnic minority adolescents—particularly Black adolescents—remain under-conceptualized, often unreported, unaddressed all together, or minimized through statistical controls—a practice which incidentally does nothing to control these disparities from arising in a justice system that is disproportionately populated by the factors controlled for in research—age, sex, race, income, education, untreated severe mental illness, substance dependence.

In sum, despite the prolific and multidisciplinary body of research that has been inspired by this developmental taxonomic framework over the last 25 years, fundamental questions remain and the central tenant of the typology, that "temporary versus persistent antisocial persons constitute two qualitatively distinct types of persons" (Moffitt, 1993, p. 673) is poorly substantiated by a full accounting of the data. Nevertheless, this line of theory remains highly influential within the criminology and psychopathology literature including DSM diagnostic schemes for disruptive disorders.

In the next section I consider why this might be, exploring the potential role of mainstream social cognitions and dominant racialized narratives in shaping the conceptual contours of the LCP and AL categories of classification. More fundamentally, beyond any single diagnostic category or theoretical construct I also explore how it might be that rule-breaking and defiant behavior came to define the core pathology of externalizing disorders (or disruptive disorders) in the first place rather than the psychological distress that undergirds these emergent behavioral manifestations including, in many cases, clear indicators of trauma.

A Critical Examination of Context: Integrating Insights from History and Critical Race Theory to Guide a Critical Examination of Constructs

Critical Race Theory (CRT) is a school of thought that originated in legal scholarship (as cited in Obasogie, Headen, & Mujahid, 2017). It provides an analytical framework that moves race and racism from the periphery to the center in the analysis of existing power structures, recognizing race as a dynamic product of social forces (e.g., economic, political, legal, interpersonal) (Obasogie, et al., 2017). This framework recognizes that intersecting identities (e.g., race, gender, class) conjointly shape the contours of social experience, including opportunities, benefits, and burdens. CRT challenges positivist assumptions about objectivity, encouraging methodological innovation and the incorporation of narratives or counterstories to give voice to those who are silenced within current systems of knowledge production (Williams, 1991, as cited in Obasogie et al., 2017).

Within the field of psychology, Adams and Salter (2011) note that there has been "little consideration towards the disciplinary conventions … that constitute racial power in psychological science" (Adams & Salter, 2011, p. 1355). They elaborate on this point, suggesting that "the development of a Critical Race Psychology requires a greater degree of identity consciousness—and critical reflexivity regarding the role of racial identity in the knowledge construction process—than has been typical in psychological science. … the rationale is to illuminate the typically obscured role of racial identity and racialized subjectivity in the production of conventional scientific wisdom" (p. 1362).

As applied to psychology, Adams and Salter describe that "CRP emphasizes a self-critical, identity-conscious, reflexive form of inquiry that illuminates the operation of racial power and ideology in theory, application, and method" (2013, p. 790). As such, researchers are

challenged “to consider the ways in which their own identity positions afford some understandings and constrain alternative understandings” (2013, p. 786).

In service of this objective, I will focus on White middle- to upper-middle class identity, which aligns with the majority identity in clinical psychology as well as my own identity position. Among the salient features of this identity position are power and privilege. Thus, to begin I consider some of the ways in which power and privilege might be expected to impact the lens through which information is attended to, organized, and interpreted broadly. Next, I draw on these ideas and the CRT framework as I consider (1) how rule-breaking behaviors came to define the core pathology of externalizing disorders in the first place, as opposed to the psychological distress that undergirds them, and (2) the potential influence of historical context, dominant racialized narratives, and White middle- and upper-middle class identity position in shaping the conceptual contours of the LCP and AL categories of classification (and analogous constructs).

Power and Information Processing

Power has been shown to impact cognition in several ways. For instance, research has shown that power facilitates access to goal facilitating information while constraining attention to goal-constraining information (Miyamoto & Ji, 2011). Greater social power is also associated with a reduced tendency to take the perspective of another person, rendering reduced sensitivity to the wants and needs of others (Blader, Shirako, & Chen, 2016). People in positions of relative power have also been shown to attend more to stereotype-confirming information of people in lower power positions (Fiske & Depret, 1996; Overbeck & Park, 2001; Vescio, Snyder, & Butz, 2003).

Greater social power promotes a context-independent cognitive processing style in which a focal object and its features are processed independent from context. In contrast, lower power promotes a more holistic cognitive processing style in which information is processed relationally or in a context-dependent fashion (Nisbett, 2003; Nisbett, Peng, Choi, & Norenzayan, 2001). Consistent with this, individuals with relatively higher power show greater use of adjectives (information that describes the features of the individual) and taxonomic categorization when describing others whereas individuals with lower power show greater use of verbs and thematic categorization (Miyamoto & Ji, 2011).

Power and Perspective: Defining and Framing the Problem

Psychiatric labels for oppositional and defiant behavior have a lineage dating back to the 1850s, when the American psychiatrist Dr. Samuel Cartwright devised two disorders specific to Black people, labeled *Drapetomania* and *Dysaesthesia Aethiopica* (as cited in Myers, 2014). The former was defined as a madness that caused Black people to run away from the enslavement of their White captors. The latter was defined by acts of oppositionality and resistance to enslaved labor such as breaking tools, destroying crops, and holding strikes. In an interview Cartwright elaborated, "they wander about at night, and keep in a half nodding state by day. They slight their work—cut up corn, cane, cotton and tobacco, when hoeing it, as if for pure mischief. They raise disturbances with their overseers, and among their fellow-servants, without cause or motive, and seem to be insensible to pain when subjected to punishment" (as cited in Myers, 2014, p. 372). Incidentally, Cartwright is also credited with branding a condition of a "deficit in attention" of mind which chiefly concerned the inculcation of new rules or details as deemed important by the White slaveholders (Myers, 2014, p. 374).

In the 1960s, as the civil rights and the Black liberation movements gained momentum, and uprisings culminating from years of racial discrimination and injustice arose in cities across the United States, the medicalization of Black oppositionality resurfaced. In 1968, with the second revision of DSM (DSM-II), the diagnostic criteria for schizophrenia underwent marked transformation to include criteria such as "frequently hostile and aggressive." Black men with active involvement in the Black liberation and civil rights movements became increasingly labeled with paranoid and delusional subtypes of schizophrenia, which at the time was grounds for indefinite psychiatric institutionalization (Metzl, 2010). Some psychiatrists went so far as to attribute Black protest and uprisings as behavior stemming from a dysfunction in the brain, for which they recommended psychosurgery as treatment (Metzl, 2010).

From here the mainstream narrative and accompanying imagery becomes focused on the visible behaviors and discharge of negative affect that combined frame the emergent product of longstanding mistreatment and oppression as stemming from a baseless and irrational propensity toward oppositionality, defiance, and violence. As a phenomenon detached from its roots, the focus becomes on a threat posed to others and society at large.

The pairing of Blackness and threat would re-emerge in the 1980s in service of garnering public support for a war on drugs declared in 1982, ultimately paving the way for the downward extension of these associations to Black adolescents the following decade. By this time Black males were so tightly coupled with social cognitions of threat and criminality, that adolescent hardship and the reality of compounding oppressions confronting young people in highly racially and economically segregated and neglected regions became overshadowed by distorted perceptions of personal threat among the mainstream public that both promoted and reinforced

dehumanizing and antagonistic attitudes and social cognitions towards adolescents in general and Black adolescent males in particular.

This is perhaps most strikingly exemplified by the public's receptivity to former Princeton University political science professor, John Dilulio's, piece titled "The Coming of the Super-Predators," in which, with noted racial undertones, he "warned of an oncoming tsunami of adolescent 'super-predators,' 'morally-impoverished' youth who had grown up 'surrounded by deviant, delinquent, and criminal adults in abusive, violence-ridden, fatherless, Godless, and jobless settings.' These were 'kids who have absolutely no respect for human life and no sense of the future … stone-cold predators'" (Moriearty & Carson, 2012, p. 296).

It is instructive that although Dilulio's description of childhood is broadly consistent with complex trauma, the conclusion that follows contains no trace of concern for the adolescent and the childhood conditions described. To the contrary, it proceeds to condemn and malign the adolescent, portraying a qualitatively distinct type of human being: a "stone-cold predator."

This example illustrates the power of implicit biases (and perceptions of threat) to shape the way information is attended to, perceived, and processed, facilitating certain interpretations and conclusions (e.g., super predator; society is at risk and must be protected) while constraining the consideration of others (e.g., trauma-stress responses and survival prioritizing adaptations to environmental contingencies; invest in systems and infrastructure to promote healing and empowerment and to support the health, and psychological and economic well-being of children and their families and communities).

In these historical examples it is instructive that the objection, or behavioral response, to mistreatment and injustice comes to define the problem behavior. In the case of *Drapetomania*, running away defines the problem behavior. The context of forced enslavement is absent.

Likewise, for *Dysaesthesia Aethiopica*, defiance and destruction of property define the problem behavior. The context of forced enslavement is absent. During the Civil Rights era, Black demonstration and militant protest define the problem behavior. The context of longstanding oppression, terror, violation of human rights, and White supremacy are absent—a pattern that extends downward to adolescents in the 1990s.

To what extent might these historical patterns, biases, and the role of power differentials more broadly (e.g., White supremacy, child versus adult status) have implications for contemporary conceptualization of the contours of trauma and externalizing disorders? To the extent that the imprint of more severe and sustained forms of stress and trauma—more complex types of trauma—manifest in ways that systematically depart from the prototypical profile of PTSD, it is conceivable that it might be interpreted very differently: not as psychological or emotional pain but as a problem, a behavior problem that poses disruption to others.

Perhaps in this way implicit biases combined with the impact of power on cognition have posed a formidable barrier to conceptualizing young person's expressions of anger, rule breaking, and defiance as responses to extended injustice, traumatization, and fear/threat-related adaptations. Consequently, it might be within the contours of discretion that rule breaking and oppositionality have come to define the core pathology of externalizing or disruptive disorders instead of the underlying psychological distress that has been disrupting to the individual.

Consistent with this possibility it is instructive to consider a key question that has traditionally been missing in defining more severe forms of externalizing psychopathology and associated disruptive disorders. That is, what does normative adaptation to extremely non-population normative circumstances look like? What does normative adaptation to prolonged mistreatment and frequent threat to survival look like? Similarly, what does "good" adaptation to

prolonged mistreatment and frequent threat to survival look like? Failure to systematically address these questions implies an assumption that either egregious and sustained adversity is simply not a reality for any segment of children and adolescents within the population or that children and adolescents who sustain these experiences are not entitled to express their anger, opposition, defiance, and coping through the avenues at their disposal.

Alternatively, a further possibility is that we have been misled by conceptualizing survivors of more complex forms of trauma as somehow qualitatively distinct, and consequently have perceived them to be impervious to the associated psychological pain and distress of complex trauma.

Conceptualizing the Contours of Externalizing: Context, Identity Position, and Social Cognitions

In 1899 the juvenile justice system was established apart from the adult system based on the philosophy that young people deserve treatment and rehabilitation. By the 1990s, however, this philosophy would markedly transform to prioritize community protection and retribution as public fear about adolescent delinquency consumed the nation. Studies of network television news conducted from 1990 to 1991 documented an explicit framing of an "us" (White, middle class suburban Americans) versus "them" (Black Americans and a few corrupted Whites) in stories pertaining to crime, drug use, and delinquency (Jernigan & Dorfman, 1996, as cited in Alexander, 2012, p. 105). Consequently, in the post-civil rights "color-blind" era, crime and "crime control" became code words for talking about race—and Black males in particular (Alexander, 2012).

Although psychopathy research had been around since the early 20th century, "until 1990, few works about child psychopathy were published, and little attention was given to

psychopathic traits in children and adolescents (Salekin, 2006; Salekin & Lynam, 2010)" (Ribeiro da Silva et al., 2013, p. 72). This would dramatically change as an "exponential increase" in research interest and publications about child and adolescent psychopathy ensued (Ribeiro da Silva et al., 2013, p. 72).

Of pertinence to the present discussion, many of the descriptors used to define the more severe presentations of EXT, such as the "life course persistent" and juvenile psychopathy constructs (e.g., callous-unemotionality), are reflected in the descriptions cited by legal scholars Moriearty & Carson (2012) commonly ascribed to Black adolescent boys:

> In the collective subconscious of these decision-makers and, the evidence suggests, in our collective American subconscious, young black males are often associated with "adult qualit[ies]," little "desire to change," "no … remorse," and "no moral content." We perceive them as "less immature," "more violent," "more culpable, more likely to reoffend, and more "deserving of punishment" than their white counterparts. While these associations may also be the product of the history of race relations in this country, strategic politics, and crime rates inasmuch as they are the product of the "super-predator" war, we cannot ignore the role of the "super-predator" war, and social wars in general, in shaping our social cognition. (p. 312)

Through the impact of power on cognition and implicit threat related biases I posit that, at scale, middle-class White identity positions might serve to foster the systematic over-attending to incidents of disruptive or externalizing behavior (or fighting or alcohol use independent from broader EXT) and ascribing them to dispositional factors while systematically under-attending to contextual information including individual-specific histories and the contingencies under which "disruptive" behaviors and associated cognitions and emotions unfold.

As explained by Bridges and Steen (1998), "Perceptions shape diagnostic and treatment processes by forming the base of information professionals use to classify clients into meaningful categories (Farrell and Swigert 1978; Scull 1975; Sudnow 1965)" (p. 554). As such, one possibility is that the social cognitions bolstered by long-standing racialized narratives and

the public fear surrounding adolescent delinquency converged to drive increasing interest and motivation toward discerning "normative" adolescent delinquency from that arising from a "qualitatively distinct type" of adolescent.

Race and Class Disparities in LCP Categorization and Related Constructs

Current evidence suggests that African American adolescents from poor/working-class households are disproportionately represented among juvenile psychopathy diagnoses (Sitney, Caldwell, & Caldwell, 2016)—a diagnosis that notably is absent from the DSM and the ICD but is widely used to inform judges in sentencing decisions. African American adolescents from poor/working-class neighborhoods (and males in particular) are also more likely to be diagnosed with characterological based disorders, including conduct disorder (Baglivio, Wolff, Piquero, Greenwald, & Epps, 2017), and to be disproportionately classified into the "LCP" subtype (or trajectory) (Elliot, 1994; Haynie, Weiss, & Piquero, 2008; Vazsonyi & Keiley, 2007; Maldonado-Molina, Jennings, & Komro, 2010; Lynne-Landsman, Graber, Nichols, & Botvin, 2011; Markowitz & Salvatore, 2012; Markowitz, 2015; Farrington et al., 2017). At the same time, these adolescents are far less likely to receive trauma-based diagnoses, as poverty/low socioeconomic status, African American ethnicity, youth, and male status are uniquely predictive of lower likelihood of receiving a trauma-based diagnosis (Borowsky et al., 2000; Liebschutz et al., 2007). And these disparities persist despite the evidence that African Americans, on average, (and particularly among lower income residentially segregated neighborhoods) tend to experience higher rates of chronic stress and trauma across the life-span relative to most other demographics, with the exception of Native Americans, who likewise have been subject to intergenerational trauma stemming from colonization and oppression (e.g., Carter, 2007; Franko et al., Geronimus, 2002; Jackson & Cummings, 2011; Jasienska, 2009; McCabe & Gregory,

1998; Turner & Avison, 2003; Turner & Lloyd, 2004; Zlotnick et al. 2003). Finally, research also shows that even among low income African Americans who have been diagnosed with PTSD, only a fraction receives PTSD focused treatment or referral for therapeutic services (Davis et al., 2011).

Racialized Subjectivity in the Construction of Constructs

One way that identity positions might be relevant to the construction of EXT-related theory, particularly as it pertains to the LCP/AL developmental taxonomic framework, is that White middle- and upper-middle class identity and affiliated privilege might promote a greater identification with the pattern of behaviors assigned to the AL subtype. Consequently they might be more inclined to ascribe the behaviors designated to this subtype as developmentally normative experimentation and rebellion—a normative response to contextual circumstances, which in this case refers to the gap between one's pubertal maturation and their social status as a minor (Moffitt, 1993).

With a lack of identity consciousness, the dominant racial and class identity defines normativity in relation to the legacy and constellation of privileges, resources, and benefits aligned with their identity position. Accordingly, deviations from this might be more likely to be tallied towards an index of abnormality or severity of deviance. Thus, events, contexts, and person-environment transactions that would be rarer on average, among the identity position of a relatively socially and economically privileged middle-to upper-middle class White majority, might be perceived and consequently operationally defined as more deviant.

This might be particularly relevant as it pertains to neighborhood context, such as the conditions of the built environment and overall sense of safety/security that distinguish racially segregated neighborhoods with high concentrated wealth versus high concentrated poverty. In

the latter context, children and adolescents are more likely to witness violence, learn about victimization of others, or be victimized themselves (Attar, Guerra, & Tolan, 1994). Consequently, behaviors secondary to threat-related hypervigilance (or hypoarousal/emotional numbing in more severe or prolonged exposure to community violence) might be more common (e.g., carrying a gun for personal safety; fighting that stems from sensitization to early signs of potential threat). Furthermore, certain infractions, most notably, drug possession and status offenses tend to be under-enforced within middle class White neighborhoods but aggressively enforced within middle- and poor/working class Black and Latino neighborhoods (e.g., Tonry, 1995; Alexander, 2012).

Consequently, from a predominantly White upper-middle-class identity position, these events, infractions, and accompanying emotional responsivity/survival promoting adaptations and coping strategies might be perceived as originating from "qualitatively distinct types of individuals" as opposed to qualitatively distinct experiences conferred by differences in safety, privilege, resources, law enforcement practices and zero tolerance environments such as continues in many schools across the nation. As such, it is conceivable that these are the lines that begin to delineate what is perceived by this identify position to be a qualitative distinction between "developmentally normative" adolescent behavior from the behavior of an "other."

In this way, I suggest that because of the manner in which the LCP construct has been operationalized, there is good reason to expect that it would produce racially and class biased classification, not because poor and working-class Black adolescents are inherently more "deviant" but perhaps because racial classification (explicitly or implicitly) has been instrumental in organizing its contours, ascribing special weight to behaviors that most distinguished between those behavioral response profiles stemming from the aggregate patterns

among middle-class White adolescents from those that depart from them, to include those profiles characterized by compounding oppressions (e.g., the maturity gap, racial, SES, age, gender) and adversities across multiple proximal and distal domains (school, home, neighborhood).

Likewise, I also suggest that for the same reason that we should expect the LCP construct (and its derivatives) to include disproportionate adolescents of color and disproportionate adolescents from poor and working-class households (and particularly the intersection between the two), we should likewise understand this construct as encompassing not antisocial deviance but rather a reliable constellation of emergent behaviors and adaptations in response to converging and compounding layers of long-standing oppression, chronic stress, and fear/threat-based adaptations—a complex type of stress and trauma.

In summary, evidence to substantiate the proposition that "temporary versus persistent antisocial persons constitute two qualitatively distinct categories of individuals" is weak, yet this line of theory remains highly influential within criminology and psychology diagnostic schemes for disruptive disorders. One reason for this continued influence might be the role of confirmation biases stemming from implicit class and race-based biases, reinforced by the omnipresent class and racialized imagery of "criminality" in American culture and media. Perhaps in this way, the race- and class-based composition of the adolescents disproportionately categorized into the LCP subgroup (or analogous constructs such as the youth versions of psychopathy, ODD, CD with limited prosocial emotions, ASPD) were more likely to register in the minds of adults physically and psychologically removed from the realities faced by the adolescent, as adolescent sociopaths versus survivors.

Next, I consider the ways in which language and the tools of science might also contribute to posing barriers to the wider consideration of more severe forms of externalizing (and fighting/drinking-specific behaviors) as rooted in experience-dependent and survival prioritizing adaptations to chronic stress and trauma.

Potential Barriers to Linking More Severe Externalizing Presentations as Rooted in Complex Forms of Trauma

Although the possibility of a link between early adversity and EXT is by no means a novel concept within the EXT literature, to date there has been little traction in this domain. This raises the question about potential barriers to gaining further traction. Below I offer additional interrelated possibilities:

(1) Reifying language. There is a propensity in social and clinical science research for descriptive constructs such an externalizing (or "life-course persistent" or similar constructs/classifications) to gradually transition from *describing* variation to *explaining* variation. Likewise, there is a tendency for theoretical constructs to become solidified as causal entities (Sampson & Laub, 2003; Laub & Sampson, 2005). However, just as schizophrenia does not *cause* one to experience auditory hallucinations, elevations on externalizing do not *cause* one to exhibit disinhibited behavior, nor provide an explanation for it.

(2) Misconceptions about heritability. Within the literature, externalizing and similar constructs (particularly those indexing the higher end of EXT, e.g., youth versions of psychopathy; life-course persistent inspired constructs including early onset CD with limited prosocial emotions or analogously, early onset antisocial behavior with callous unemotionality,

(CU) have frequently been described as "highly heritable."[16] These descriptions, combined with common misconceptions about what the heritability statistic tells us and what it does not tell us, can leave an erroneous impression about the degree to which genetic differences as indexed through a heritability statistic matter—particularly in an instrumental, intervention/malleability oriented sense—for a complex phenotype.

Critically, estimations of heritability derived from twin-family designs are distinct from "heritability" as commonly used in the English language: "the quality of being heritable, or capable of being inherited' (Oxford English Dictionary, cited by Stolenberg, 1997). The term is also reminiscent of heredity, inherited, and heritable, common words all implying the passing of something from parent to offspring. Thus, to nongeneticists, high values of heritability seem to imply that much more (in this case, genes) is transmitted than would be the case if the values were low. However … this is a fallacious interpretation" (Vitzthum, 2003, p. 542). Rather, in statistics, heritability refers to "the ratio of the observed phenotypic correlation to the theoretical genotypic correlation" (Weiss, 1993, as cited in Vitzthum, 2003). A key implication of this is

[16] For example: "…the remarkably high heritability for CU [callous unemotional] and for AB [antisocial behavior] in children with CU suggests that molecular genetic research on antisocial behavior should focus on the callous-unemotional core of psychopathy. Finally, combining neuroscience and genetic methodologies should be at the forefront of future research on psychopathy (…) Finally, with regard to public policy, these results confirm the notion that prevention efforts need to begin in the preschool years. As the large genetic component to psychopathic antisocial behavior is likely to reflect not only the direct effects of genes, but also gene–environment interaction (Moffitt, 2003), preventative efforts for psychopathy will benefit from developmental investigations of this interaction using measured genes and environments. Finding a large shared and non-shared environmental influence on the AB of children without psychopathic tendencies suggests that this subgroup of children with early onset AB is probably amenable to traditional interventions aimed at improving family, school and neighbourhood conditions" (Viding, Blair, Moffitt, & Plomin, 2005, p. 592). / "Our results indicate that exhibiting high levels of callous-unemotional traits (CU) at 7 years, as assessed by teachers at the end of the first year of school, is under strong genetic influence. Minimal shared environmental influences on callous-unemotional traits were detected, suggesting that at the age of 7, environmental factors common to both members of the twin pair (such as socio-economic status, school and neighbourhood) do not account for extreme CU. Moreover, antisocial behavior (AB) for children who are high on CU (i.e., children with psychopathic tendencies) is highly heritable. In contrast, the extreme AB of those without psychopathic traits was under strong environmental influence—shared as well as non-shared" (Viding, Blair, Moffitt, & Plomin, 2005, p.596-597).

that the etiological importance of context should not be reflexively discarded in the face of so called "large" heritability estimates—a point I will return to shortly for further elaboration. And a point that I suggest might be particularly key for understanding stress-related phenomenology.

Misconceptions about heritability also undergird the notion that a construct is a more valid, cohesive, or meaningful entity because it has been documented as heritable. Vitzthum (2003) summarizes this point, cautioning:

> Even if a phenotype is easily defined and measurable, it does not necessarily mean that the trait is biologically meaningful or that it is a unit coded in some way by DNA. For example, in the 19th century the "science" of phrenology considered head bumps to be reliable biological markers. One could calculate a heritability estimate for head bump number and size from pedigrees if one were so inclined; the interpretation could be written in a scientific manner. Of course, no one would undertake such an effort today, but it would be hubris to assume we are incapable of making such errors" (Vitzthum, 2003, p. 544).

There is good reason to expect that complex trauma-related processes that originate from multiple and varied external insults over the course of development might nevertheless be expected to show relatively "high heritability." This is on account of the multiple interacting and developmentally sensitive systems undergirding stress related processes.

One of the notable findings to emerge in the present study is that relative to the domain-specific externalizing behaviors, global externalizing showed higher associations across the board with each of the *internalizing* factors (i.e., global *internalizing*, domain-specific negative self-concept/numbing, domain-specific somatic sensitivity). This is noteworthy given the patterns of divergence in heritability estimates that traditionally arise between the global (relatively higher estimates) and specific (relatively lower estimates) externalizing spectrum outcomes.

It is possible that in this case the "high" heritability of externalizing may be indexing/detecting the individual differences within personalized reaction norms (which could reasonably be expected, particularly early in the life-course, to be more similar between twins who share 100% of their genetic material compared to 50%) to external input (consistent with the concept of sensitization which is calibrated based upon prior exposure). In this case, a heritability statistic could be very "high" in the sense of genetic differences between individuals accounting for individual gradations of difference in phenotypic expression of externalizing between people but that would not diminish the role of the context or environmental precipitant in effectively "causing" the stress response activation (hypervigilant response) among those who have experienced greater environmental insult throughout the course of development.

Indeed, stress is posited to have development-dependent effects on anatomy, behavior, and cognition as brain structures experience rapid growth and decline over the course of development (Lupien et al., 2009). Coupled with the sheer complexity of interacting systems that work in concert to prepare an organism to respond to a perceived threat (e.g., attentional systems, autobiographical memory, respiratory systems, psychological systems, cardiovascular system, chemical and hormonal messenger systems), it is not surprising that genes would have a role in modulating aspects of these interrelated processes and that genetically identical twins would likely show greater correspondence on the developmental growth and decline of these stress-sensitive systems relative to fraternal twins (or full biological siblings) who share only half of their segregating DNA on average. In the context of a twin study this greater relative similarity between MZ twins would generally be indexed by greater heritability, as would the interaction between additive genetic mechanisms with shared environmental influences such as mechanisms pertaining to SES, racism, climate in the home, neighborhood, and school (Caspi et al., 2002). As

such, genetically mediated differences in stress-sensitive processes between people would correspond, perhaps quite closely, with individual differences in expression of phenotypes undergirded by complex arrays of interacting developmentally sensitive networks; however, this close correspondence, which would translate to "high heritability," would not negate the critical importance of the external precipitants in "causing" the traumatic stress condition/adaptation.

Put differently, the built-in alarm which is likely under genetic control is not the core of the problem, and its experience-dependent sensitization (gene x environment transactions) does not implicate vulnerable or "bad" genes. The original environmental precipitant(s) (unconditioned stimuli) that served to calibrate it are the core problem, not the survival promoting mechanisms that alert (trigger) the individual to take action to restore a sense of safety when threat is perceived.

(3) Methodological norms: the relative absence of idiographic approaches and the misapplication of population-level data to make inferences about an individual. This is particularly relevant to the study of phenotypes for which environmental factors are posited to be influential yet data at the aggregate fail to bear this out, since much individual nuance is lost in aggregation and important sources of heterogeneity can be missed altogether. As such, normative adaptations to extremely non-population-normative experiences or events might appear abnormal or dysfunctional in the context of population-normative environments.

Speaking to the gravity of this issue in psychology, the statistician Molenaar wrote, "the published literature on ideography occupies only a vanishingly small proportion of our scientific journals, which is an indefensible and unjustified neglect of the facts … To convey this point to the audience I do not need a subtle argument, but a manifesto" (p. 204).

Among the implications of this is that to date, many of the quasi-causal methods used to examine the link between early maltreatment and subsequent antisocial behaviors may be underestimating the true effects of environmental antecedents on account of applying nomothetic methodological frameworks that may underestimate the true effects at the individual level as well as perpetuate overly narrow and ecologically (biologically/developmentally) unrealistic conceptualizations and operationalization of causality.

After delineating his proofs, Molenaar's manifesto concluded that "psychometrics and statistical modeling as we now know it in psychology are incomplete. What is lacking is the scientific study of the individual, his or her structure of IAV [intra-individual variation], for its own sake. Scientific psychology can only become complete if it includes the idiographic point of view (p. 216).

Molenaar further noted that for non-stationary or *nonergodic* processes such as habituation, learning, and development—processes that are highly relevant to the hypothesized mechanisms underlying elevated externalizing behavior in the present study— "there is no scientifically respectable alternative but to study the structures if IAV [intra-individual variation] and IEV [inter-individual variation] for their own sakes" (p. 215). Among the key implications of this is that to date, even among designs such as longitudinal frameworks including latent-growth trajectory class modeling, risk or cognitive assessments undergirded by classical test theory, the resulting findings may have little if any correspondence to the case for any single individual or patterns among single individuals.

(4) Language and labels. Specifically, labels—diagnostic and otherwise—that prime implicit biases, conjure criminality, unduly shape expectancies, impact interpretation of data, and

sow indifference through "othering," to norms, policies, practices, analyses, and conclusions that might otherwise draw wider scrutiny or demand a greater burden of proof or rigor.

To emphasize the importance of labeling and language, I draw on an excerpt from Haig A. Bosmajian, professor emeritus from Stanford University, from *The Language of Oppression*:

> Just as our thoughts affect our language, so does our language affect our thoughts and eventually our actions and behavior. As Edward Sapir has observed, we are all "at mercy of the particular language which has become the medium of expression" in our society. The "real world," he points out, "is to a large extent unconsciously built up on the language habits of the group. ... We see and hear and otherwise experience very largely as we do because the language habits of our community predispose certain choices of interpretation. …
>
> George Orwell has written in his famous essay "Politics and the English Language" … that "silly words and expressions have often disappeared, not through any evolutionary process but owing to the conscious action of a minority." Wilma Scott Heide, speaking as president of the National Organization for Women several years ago, indicated that feminists were undertaking this conscious action: "In any social movement, when changes are effected, the language sooner or later reflects the change. Our approach is different. Instead of passively noting the change, we are changing language patterns to actively effect the changes …"
>
> This then is our task—to identify the decadence in our language, the inhumane uses of language, the "silly words and expressions" which have been used to justify the unjustifiable, to make palatable the unpalatable, to make reasonable the unreasonable, to make decent the indecent. (Bosmajian, 1974)

Classifying individuals as "life-course persistent" or with diagnostic or descriptive derivatives of this framework (e.g., childhood onset conduct disorder with limited prosocial emotions; callous-unemotionality; youth assessments of psychopathy; referring to adolescents as "offenders," "antisocial," "delinquents") provides a similar language of "othering" that facilitates the transformation of multidimensional children, adolescents, and adults with complex individual histories into a monolithic one-dimensional problem to control or liability to contain.

Viewed in this way, prior victimization becomes overshadowed by a victimizer narrative that brings to the forefront a one-sided story centered on individuals' behavior (devoid of the

individualized context in which it unfolded) highlighting the fact that they have thrown punches, raised their voice, stolen or destroyed another's property, lied, ingested alcohol, smoked or sometimes sold marijuana, carried a weapon, missed class, violated curfew, or run away from home—behaviors that viewed through a lens of a disruptive disorder enables a status quo of zero tolerance and disciplinary approaches, including suspensions, expulsions, and graduation into the criminal justice system. Actions which can further compound overwhelming stress and trauma as they simultaneously diminish perceptions of its core relevance in the etiology and progression of present behavioral patterns, core beliefs, interpretations, emotions, coping strategies, and physiological responsivity to the outer world.

In this way, early and multiple trauma exposures, although virtually ubiquitous among adolescents in the juvenile justice system (Abram et al., 2004; Arroyo, 2001; Garland et al., 2001), can more easily be relegated (and effectively dismissed) as an unfortunate but commonplace norm within the justice system that is ancillary to understanding an individual's current problems and patterns of relating to others and interpreting and coping with the outside world.

In conjunction with the intersectionality of implicit race-, class-, and adolescent-based biases, I suggest that these language habits unduly predispose adults in positions of power to systematically over-interpret behaviors such as school absence, staying out past curfew, anger and irritability, displays of toughness, fighting, running away from home, or carrying a weapon as "offenses" rather than defenses motivated by fear for personal safety and survival-promoting adaptations, which in many cases undergirds these behaviors among children and adolescents (e.g., Ford et al., 2012; Schwartz, Jackson-Beeck, & Anderson, 1984; Saada Saar et al. 2015).

In conclusion, these barriers might be relevant for the fact that despite the striking parallels between adolescent complex trauma and more severe presentations of EXT the etiology of traumatic stress and fear/threat-based adaptations have garnered minimal consideration within the EXT and disruptive disorder literature.

An Alternative Theoretical Framework

Rather than qualitatively distinct subtypes of individuals, I propose that temporary versus more severe and persisting EXT behavior arises from two qualitatively distinct experiences of early stress and trauma (and survival prioritizing adaptations). Much like Moffitt conceptualized the AL subtype of delinquent behavior as an "adaptive response to contextual circumstances" (p. 689), I posit that the LCP subtype of behavior is likewise an adaptive response to contextual circumstances.

I suggest that early chronic stress and trauma and the resulting traumatic stress sequela provide a potentially more fruitful and parsimonious explanatory framework to account for the substantial overlap among externalizing spectrum disorders over the course of development including ADHD, ODD/DMDD, CD, SUD, and ASPD (Ford et al., 2012; Lahey et al. 1997; Loeber, Burke, & Lahey, 2002; Rowe et al., 2002), selective links to elevations in internalizing symptomology over adolescence, and the broader constellation of contextual correlates, and neurobiological patterns, substrates, and postulated biomarkers that have been reported for more severe presentations of externalizing, including the prototypical profile of the so called "life-course persistent" subtype and related constructs based off of this or similar taxonomy[17].

[17] For example, PTSD, chronic- and complex-PTSD, and childhood complex trauma and related constructs such as DENOS and DTD; in DSM-5 diagnostic terms this would most closely correspond to individuals assigned a progression of diagnoses including ADHD, Disruptive Mood Dysregulation Disorder (or ODD), early onset conduct disorder with limited prosocial emotions, ASPD.

I posit that this framework can also (1) provide a more coherent explanatory framework for understanding more severe and persisting forms of EXT psychopathology, including recently proposed subtyping schemes for conduct disorder, and (2) account for explanatory gaps in the EXT-related frameworks (DSM disruptive disorders, dual taxonomy related constructs and psychopathy related constructs) including patterns of results that have been mixed, inconclusive, and unexpected.

As much of these concepts and ideas already exist within the childhood complex and developmental trauma literature, in the sections that follow I draw from these areas and the adult literature on fear and stress-based disorders (PTSD, complex PTSD) to demonstrate how many of the key findings in the externalizing literature that distinguish more severe and persisting profiles of EXT from more developmentally normative adolescent EXT presentations (namely, the "LCP" construct and its DSM derivatives), can be accounted for and more parsimoniously organized from a framework of experience-dependent and functionally adaptive response to complex traumatic stress.

Prevalence Course and Onset

At an estimated 5-13% prevalence (complex trauma: 5% Costello et al., 2002; polyvictimization: 10% [Finkelhor, Ormrod, & Turner, 2009], 13% [Ford et al., 2010]), rates of complex trauma are broadly consistent with documented estimates for more pronounced EXT-related psychopathology[18]. Additionally, complex trauma and its related adaptations and trauma-stress sequalae can have an adverse impact across the lifespan as its influence is unlikely to

[18] "LCP": 5% Robins, 1985; Moffitt, 1993; 5-8% Piquero, Daigle, Leeper Piquero & Tibbetts, 2007 5-10% Wolfgang, Figlio, & Sellin,1972; CD: 5-8% Piquero, Daigle, Leeper Piquero & Tibbetts, 2007; CD with limited prosocial emotions (measured as CP-CU): Approximately one third of children who meet criteria for CD also demonstrate the CU pattern Mills-Koonce, Wagner, Willoughby, Stifter, Blair, & Granger, 2015; CD-CU 2-4% (Frick. 2009).

spontaneously remit without effective intervention (Streeck-Fischer & van der Kolk, 2000). As such, to the extent that (unresolved) complex trauma may underlie more pronounced forms of EXT, this would be consistent with a developmental progression of EXT behavior that does not correspond (is not limited to) the developmentally normative uptick during adolescence and decline with emerging adulthood.

Consistent with earlier onset of EXT predicting a more severe and persisting course of EXT behaviors, earlier developmental timing of trauma onset likewise predicts more pronounced complex trauma and related adaptations and trauma-stress sequela. Although child recall (and caregiver awareness or reticence to disclose) can pose barriers to ascertaining information about very early events, what aggregate data have shown is that children under age three are victimized at greater rates than children over age three, and children under age one are victimized at the highest rates (US Department of Health and Human Services, 2016). Age five was the mean age of trauma onset based on data extracted from 1,699 child trauma caseloads (Spinazzola et al., 2003); however, of note, these cases were not limited to complex trauma and so this figure is likely inflated relative to what it would be were the sample limited to complex trauma. Though not conclusive, this pattern is nevertheless compatible with the possibility of early exposure to traumatic stress undergirding an earlier onset of EXT behaviors. Early complex trauma could also potentially account for the links between earlier onset of EXT and elevated risk for future trauma exposures as youth who experience interpersonal trauma early in life are at substantially elevated risk for future re-victimization both in adolescence and adulthood (Finkelhor, Ormrod, & Turner, 2007).

Posited Pathways to Childhood Onset Conduct Disorder (CD): Parallels with Complex Trauma-Related Hyperarousal and Hypoarousal

In DSM-5 the specifier "with limited prosocial emotions" was added to the childhood onset CD subtype to improve identification of individuals with a more severe and persisting course of conduct related problems. The evidence base for this change came from the child psychopathy literature on a construct labeled *callous unemotionality* (CU). As described by Pardini and Frick (2013), "consistent with the affective dimension of adult psychopathy, CU traits include a lack of concern for others' feelings, deficient guilt and remorse, and shallow affect" (p. 21). Low temperamental fear is posited to be a unique causal factor for the CU-inspired subtype within the childhood onset CD (labeled in DSM-5 "with limited prosocial emotions") which is thought to lead to the development of early conduct problems "because it reduces the effectiveness of punishment-oriented socialization techniques and fosters the development of CU traits (Pardini, 2006)" (Pardini & Frick, 2013, p. 21).

Further research into the childhood onset subtype of CD has revealed a subset of individuals who do not exhibit CU features but instead exhibit high negative emotionality and high internalizing (Hipwell et al., 2007; Pardini, Lochman, & Frick, 2003). From these findings another causal pathway to childhood onset CD has been advanced: a pathway characterized by *severe anger dysregulation* (Pardini & Frick, 2013). As summarized by Pardini and Frick, "children with anger regulation problems often exhibit early oppositional/defiant behaviors, which tend to precede the development of CD in childhood. Youth with high levels of anger also tend to have a hostile attribution bias when encoding cues of potential threat, which can perpetuate interpersonal conflicts with others" (Pardini & Frick, 2013, p. 22).

Although severe anger dysregulation was not added to DSM-5 as a specifier for childhood onset CD, a new disruptive disorder was added to DSM-5, called Disruptive Mood Dysregulation Disorder, which captures more pronounced presentations of anger. Added in part to address concerns about over-diagnosis of bipolar disorder in children, DMDD is in essence a more severe form of ODD. Unlike ODD however, diagnostic rules allow clinicians to diagnose both DMDD and CD at the same time (as distinct, comorbid disorders). Diagnostically speaking, comorbid DMDD and CD would be the closest proxy to a childhood onset CD with severe anger dysregulation.

Regardless of naming conventions these divergent profiles within the childhood onset CD subtype are consistent with a traumatic stress etiology and trauma-stress symptoms and survival prioritizing adaptations.

In DSM-5, a subtype was added to the PTSD diagnosis in recognition that approximately 12-30% of individuals with PTSD exhibit prominent dissociative and numbing related symptoms (Friedman, Resick, Bryant, & Brewin, 2011; Miller, Wolf, & Keane, 2014). The dissociative subtype of PTSD is usually associated with greater severity and chronicity of trauma exposures and is linked to a unique pattern of brain activation in response to trauma cues, which can include a hypo-aroused/emotionally over-modulated (dissociative/numbing) presentation, apart from the more traditional hyper-aroused/emotional under-modulated reactivity more traditionally associated with PTSD (Miller, Wolf & Keane, 2014). In fact, there is evidence to suggest that elevated startle response in the face of threat cues tends to be more predictive of a single trauma PTSD presentation/response pattern whereas multiple chronic and severe trauma exposure histories, those which characterize complex trauma, are more likely to exhibit a pattern of diminished arousal, consistent with a hypo-aroused dissociative state.

As described by Ogden (2006),

> People with trauma-related disorders are characteristically vulnerable to hyperarousal (i.e., experiencing "too much" activation) and/or hypoarousal (i.e., experiencing "too little" activation) and often oscillate between these two extremes (Post et al., 1997; Van der Hart, Nijenhuis, & Steel, 2006; Van der Kolk et al., 1996). Triggered by traumatic reminders, both autonomic tendencies leave clients at the mercy of dysregulated arousal. When hyperaroused, clients experience too much arousal to process information effectively and are tormented by intrusive images, affects, and body sensations. But when hypoaroused, clients suffer another kind of torment stemming from a dearth of emotion and sensation—a numbing, a sense of deadness or emptiness, passivity, possibly paralysis (Bremner & Brett, 1997; Spiegel, 1997; Van der Hart et al., 2004), and/or may be too distanced from the experience to be able to process information effectively. In both cases top-down regulation is compromised and meaning making becomes biased by the perceived danger signals. Whereas these extremes of arousal may be adaptive in certain traumatic situations, they become maladaptive when they persist in nonthreatening contexts. (Ogden, 2006, p. 26).

Thus, the CD subtype "with limited prosocial emotions" would correspond most closely with a dominant hypoarousal presentation. Hypoarousal can also produce re-experiencing symptoms but instead of the panic-like physiological reactivity characterized by hyperarousal related re-experiencing, hypoarousal re-experiencing, according to Ogden, "causes similar losses in memory, motor or affective functions, and somatosensory awareness as those that occurred during the trauma (Van der Hart et al., 2004). Chronic hypoarousal frequently involves somatoform dissociative symptoms such as motor weakness, paralysis, ataxia, and numbing of inner-body sensation, as well as psychoform dissociative symptoms such as cognitive abnormalities, amnesia, fugue states, confusional states, and deficits in attention (Nijenhuis & Van der Hart, 1999; Van der Hart et al., 2004a and 2004b)" (p. 35).

Body and sensory perception, which is necessary for recognizing, processing, and expressing emotions (Schmid, 2013) is often impacted by chronic exposure to trauma (Sack, Boroske-Leiner, & Lahmann, 2010). Among chronically traumatized individuals, studies have documented diminished perception of pain (Ludäscher et al., 2007; Klossika et al., 2006) and

diminished auditory perception (Maercker & Karl, 2013) during tense conditions (as reviewed in Schmid et al., 2013). Likewise, under emotional situations involving pressure, individuals with extensive trauma and severe neglect in their backgrounds have shown reduced regulation of emotion and perspective taking ability (as reviewed in Schmid, Petermann, & Fegert, 2013; Pears & Fisher, 2005; Fonagy, Gergely, Jursit, & Target, 2002), with diminished perspective taking increasing as a function of duration of prior deprivation and/or neglect (Colvert et al., 2008).

Beyond descriptive similarity, the neurobiological underpinnings that distinguish between these posited subtypes, correspond with the patterns of findings that underlie hypoarousal and hyperarousal.

Neuroendocrinology

The neuroendocrinology associated with more severe forms of EXT can also be accounted for by stress/trauma-related adaptations. PTSD, and particularly more chronic forms (as would be characteristic of complex trauma), are posited to be a dysregulation of a normal stress response (Yehuda, 1999). As such, PTSD is most commonly associated with abnormally low levels of resting cortisol (Yehuda et al., 1996; Yehuda, 1999). Likewise, more severe and persisting profiles of EXT have also been linked to low resting cortisol[19].

Some of the cortisol findings in relation to EXT have been mixed however (Alink et al., 2008). A trauma-related etiology could potentially account for discrepancies in this domain as

[19] Studies reporting low resting cortisol: childhood antisocial behavior (Hawes et al., 2009); psychopathic personality (Gao, Schug, Yang, & Raine, 2009); youth with most severe and persistent conduct disorder presentations / youth high in callous-unemotionality (Loney, Butler, Lima, Counts, & Eckel, 2006); persistent aggressive behavior (McBurnett, Lahey, Rathouz, & Loeber, 2000; van Goozen et al., 1998); children, adolescents, and adults with conduct problems (Susman, 2006; van Goozen, Fairchild, Snoek, & Harold, 2007); psychopathy levels among prison inmates (Cima, Smeets, Jelicic, 2008).

well because elevated cortisol levels are expected among both recently traumatized individuals and individuals who are still living under or are in frequent contact with chronically threatening and unsafe circumstances (Friedman et al., 2007; Inslicht et al., 2006). As cortisol reactivity is related to baseline level, sample variation depending on recent and ongoing trauma (or even contact with trauma-related cues that trigger elevated trauma-stress symptomology among individuals who have been highly trauma-stress sensitized) would likely further complicate clear interpretation of results if these factors were not considered (along with adherence to best practices for measurement of cortisol).

Risk Factors

Many of the early life risk factors that distinguish more pronounced and persistent forms of EXT are commonplace among individuals with histories of complex trauma. For instance, among a range of risk factors, psychopathy, as inferred by total scores on the Psychopathy Checklist-Youth Version (PCL-YV) scale, and its sub-scale dimension antisocial behaviors, were prominently predicted by foster home placement, which is virtually synonymous with childhood complex trauma (Campbell et al., 2004; Krischer & Seveke, 2008). Consistent with this, adolescents with abuse in their backgrounds score higher on the PCL:YV (Campbell et al., 2004; Cauffman, 2008; Krischer & Sevecke, 2008). And this subscale was shown to increase as a function of both severity and repeated exposure to family violence (Ireland & Smith, 2009), consistent with recent evidence that family maltreatment is causal in CD, as opposed to the child contributing an evocative role, as some have posited (Smith, Dishion, Shaw, Wilson, Winter, & Patterson, 2014).

Other forms of trauma exposure, beyond child maltreatment, have also been linked to elevated EXT and fighting related behavior including community violence, witnessing domestic

violence, and traumatic loss (Foy, Ritchie, & Conway, 2012; Kerig, Ward, Vanderzee, & Moeddel, 2009; Wood et al., 2002). Within the psychopathy literature exposure to community violence has been associated with elevated callous-unemotionality in adolescents, and in conjunction with community violence those with the most extensive history of abuse showed the greatest elevations in so called callous unemotional traits as well as the greatest desensitization to distressing images (Kimonis, Frick, Munoz, & Aucoin, 2008). Finally, consistent with the role of poverty in elevating risk for complex trauma, individuals were shown to have 3.5 times higher odds of being categorised into the LCP versus AL subgroup if their family's household received government assistance for six-months or more while growing up (Moore, Silbert, Roberson-Nay & Mazuk, 2017).

To be clear, the take-home message is not that trauma makes someone violent; the range of responsivity to chronic or repeated trauma and fear/threat-based activation should be expected to vary depending on a confluence of factors as reviewed earlier (developmental timing, nature of the threat, frequency, controllability, supports, etc.). But rather to highlight concern with the possibility that we might be systematically labelling a normative response to an inherently unviable situation, the problem behavior.

Comorbidity

In addition to risk factors, comorbidity is remarkably parallel too. Disorders found in association with PTSD for youth include Oppositional Defiant Disorders (ODD), Attention Deficit Hyperactivity Disorder (ADHD), Conduct Disorder (CD), depressive disorders, phobic and panic disorders, Generalized Anxiety Disorder, and substance use disorders (American Psychiatric Association, 2000; Carrion et al., 2002; Cicchetti, 2003; Cook et al., 2005; Ford, 2002; Ford et al., 2012; Greenwald, 2002; van der Kolk et al., 2005; Weinstein, Staffelbach, &

Biaggio, 2000). Finally, just like disruptive behavior disorder symptoms are associated with the development of internalizing problems and social problems over time (Pardini, & Fite, 2010), trauma-stress symptoms increase over the course of adolescence (Sweeny, 2013), and it is well established that the experience of multiple trauma types is associated with increased post-traumatic stress reactions, difficulties in emotion regulation, and internalizing problems (Finkelhor, Turner, Hamby, & Ormrod, 2011). And although substance dependence has been suggested as a potentially promising biomarker for vulnerability to callous-unemotionality (Moffitt et al., 2008), substance use disorders are highly comorbid with PTSD, as high as 85% in clinical samples (Baker et al., 2009; Kessler et al., 1995, 2005; Mills et al., 2006; Petrakis et al., 2002). Moreover, consistent with a trauma etiology, common genetic mechanisms have been shown to account for the overlap between PTSD and substance dependence (Sartor et al., 2011).

Although mid-life physical health comorbidity has not been as widely studied in association with more severe forms of EXT, Moffitt would subsequently add to her dual taxonomy the hypothesis that the LCP subtype would be at elevated risk for poor physical health outcomes at midlife, including cardiovascular disease, and early disease morbidity and mortality (Moffitt, 2003, 2006). While to my knowledge this has yet to be examined at midlife, these outcomes would further be consistent with a complex trauma etiology as overactivation of the stress systems contributes to the suppression of the immune system, leaving individuals with higher rates of infectious disease for up to 20 years following exposure to major trauma (Kendall-Tackett, 2000; Watts-English et al., 2006). Childhood exposure to multiple and/or prolonged maltreatment is also linked to subsequent development of more serious health problems including higher rates of hypertension, cardiovascular, digestive, endocrine, nervous

system, liver, and respiratory diseases (Kendall-Tackett, 2000; Watts-English et al., 2006; Davidson, Hughes, Blazer, and George, 1991; Davidson, 2001).

Neurobiology

Beyond comorbidity many of the neurobiological patterns, substrates, and postulated biomarkers that have been reported for more severe presentations of externalizing, including the prototypical profile of the so-called LCP subtype, are in fact well established neurobiological features of a traumatic stress response and reactivity. As such, unresolved complex traumatic stress can account for many of the findings observed in association with more severe profiles of externalizing, including patterns of neurobiological findings from infancy over the lifecourse as well as academic and interpersonal challenges.

Many of the brain regions posited to have particular relevance to elevated externalizing presentations, such as the prefrontal cortex, hippocampus, amygdala, and corpus callosum, have all been identified within the childhood traumatic stress literature as the key brain regions most sensitive to early stress (Teicher et al., 1997) and most influenced by biological stress systems including the HPA system (De Bellis, 2001). Among children, the stress systems have also been noted to specifically impact attentional systems that influence recall and discrimination or filtering between focal and ancillary information (Lupien et al., 2005). Compared to non-abused controls, experimental work has shown that children with abuse histories and diagnoses of DSM-IV PTSD, have shown poorer performance on attention and other executive functioning tasks (Beers & De Bellis, 2002). Consistent with this, pediatric maltreatment-related PTSD is linked to poorer neurocognitive performance in stress related brain regions impacting measures of executive function, language, memory, and learning (De Bellis et al., 2002). As such, more severe and/or chronic stress has been posited to impact school-based learning in particular

(McGloin & Widom, 2001). Consistent with this, among a sample of first graders exposure to community violence and magnitude of trauma-related distress combined additively to predict reductions in overall IQ scores and reading achievement, leading researchers to suggest that the combination of violence exposure and its traumatic stress sequela may pose particular challenges for academic achievement (Delaney-Black et al., 2002; Saltzman, Pynoos, Layne, Steinberg, & Aisenberg, 2001).

In another study using a sample of individuals with trauma in their backgrounds that lead to PTSD, the duration of years that the index trauma was experienced was inversely related to measured IQ scores (DeBellis et al., 1999). Similar findings have also been shown for maltreatment severity and measured IQ scores (Carret et al., 1995) as well as for indices including lack of supervision, witnessing family violence, and emotional abuse, each of which were also linked to lower reading and math achievement (Salzman et al., 2006; Terr et al., 1991). Another study found that both number of trauma exposures and re-experiencing symptoms (e.g., intrusive thoughts, imagery, nightmares) were associated with lower measured global and verbal IQ scores (Saltzman et al., 2001; Saltzman et al., 2006). During prolonged stress, dopamine and norepinephrine levels are raised which might be one mechanism impacting prefrontal cortex related processes (Arnsten & Goldman-Rakic, 1998). Additionally, the chronic scanning for threat which is characteristic of chronic hyperarousal may draw on a distinct allocation of attentional resources to facilitate a rapid orienting response to potential threats in the environment.

Notably, the neurobiological profiles that characterize major depression, a common accompaniment (or perhaps progression) of more severe and/or prolonged traumatic stress exposures that characterize complex trauma, implicate many of the domains relevant to

performance on these types of measures including attention, concentration, declarative or episodic memory (Zakzanis et al., 1999; Kampf-Shert et al., 2004) and processing speed (Nebes et al., 2004).

Although far fewer studies have examined this domain, there is evidence of normalization of neurobiological divergences upon remission of depression and effective trauma oriented therapeutic intervention. For instance, Trichard et al. (1995) found that verbal fluency performance normalized in depressed patients upon remission of their depressive episode (scores during the depressive episode averaged 2 standard deviations below their remission level performance). Trauma informed therapeutic interventions have also shown post-intervention improvements in academic performance domains of math and reading (Saltzman et al., 2001), and in full scale IQ scores in a Veteran population, as measured by the Weschler Adult Intelligence Scale (VA Healthcare System, San Diego, in progress). These findings may be relevant for externalizing as it is the more severe profiles of EXT that have been linked to performance divergences in these same domains.

Attention to Threat

From a trauma-informed perspective, hypervigilance and attentional orienting to potential threat is not simply a social information processing deficit or spontaneous neurobiological error or bias but rather a response pattern rooted in an adaptive (survival promoting) orienting response conditioned through experience to attend to stimuli that preceded or accompanied prior danger (e.g., anger based facial cues such as the case in caregiver maltreatment or fear-based cues such as the case in witnessing intimate partner violence perpetrated against one's primary caregiver). Severe trauma, particularly if prolonged, can alter brain functioning to prioritize allocation of resources towards survival. Depending on the nature of the trauma, alterations

might promote a state of chronic hyperarousal, drawing on limbic and prefrontal cortical circuitry to promote hypervigilance that enables a heightened scanning for potential threat in one's environment (Williams et al., 2009). Through these mechanisms, individuals who have a history of prior trauma become more easily primed to react to subsequent threat or stressors, a cycle in which further trauma contributes to greater sensitization and more pronounced reactivity to subsequent early signs of threat, effectively narrowing one's "window of tolerance."

However, studies have documented that far from being deficient, children who have experienced maltreatment are in fact highly attuned to early signs of danger, and compared to non-maltreated children even display heightened amygdala reactivity to threat cues presented preconsciously (McCrory, De Brito, Sebastian, Mechelli, Bird et al. 2011; McCrory, De Brito, Kelly, Bird, & Sebastian, 2013; Tottenham et al., 2011). Notably, these alterations in social information processing are consistent with those associated with more severe and persisting EXT presentations.

Attachment Patterns

Disorganized attachment has been identified as the pattern most common among youth who exhibit early onset conduct problems with "callous-unemotional" features (a construct analogous to DSM early onset CD with limited prosocial emotions). Notably, this is consistent with the attachment pattern common among early complex trauma. Consistent with trauma-stress-related origins, a key role for frightening or frightened maternal behavior is posited to undergird disorganized attachment (Main & Hesse, 1990; Schuengel, Bakermans-Kranenburg, & van IJzendoorn, 1999; True, Pisani, & Oumar, 2001). Parental history of unresolved loss or trauma is also predictive of disorganized attachment (Lyons-Ruth & Jacobvitz, 2008; Lyons-Ruth, Repacholi, McLeod, & Silva, 1991; Main & Hesse, 1990; van IJzendoorn, 1995) as well as

severe forms of early adversity including neglect, separation from caregivers, and exposure to domestic violence, all of which pose survival-threat to an infant (Carlson, 1998; van IJzendoorn, Schuengel, & Bakermans Kranenburg, 1999; Lyons-Ruth, Connell, Zoll, & Stahl, 1987; Stovall-McClough & Dozier, 2004).

In contrast to infants with secure attachments, within in the context of stress, infants with disorganized attachment exhibit increased sympathetic nervous system reactivity (Oosterman, De Schipper, Fisher, Dozier, & Schuengel, 2010) and elevations in cortisol (Bernard & Dozier, 2010; Hertsgaard, Gunnar, Farrell, Erickson, & Nachmias, 1995; Spangler & Grossmann, 1993). Research has also found the disorganized attachment pattern to mediate the association between disruptive maternal behavior and toddler behavioral problems (Madigan, Moran, Schuengel, Pederson, & Otten, 2007).

Interventions

Among children and adolescents, the most promising interventions for more severe EXT psychopathology (DSM disruptive disorders; ODD, CD) are those that involve multiple systems of engagement within the child's normative milieu (parent training, teacher training, child advocating) or for adolescents, resemble trauma-informed cognitive-behavioral therapy approaches that emphasize support and establishing safety, skill building, and eventually trauma-related processing (e.g., Borduin et al., 1995; Greenwood, 1994; Greenwald, 2002; Henggeler, Melton, & Smith, 1992; McCart, Priester, Davies, & Azen, 2006; Serketich & Dumas, 1996). In contrast, zero-tolerance, authoritarian, institutional, and punitive approaches show iatrogenic effects (e.g., Brestan & Eyberg, 1998; Henggler & Schoenwald, 1994). For children, parent-interventions are considered first-line treatment options. Parent interventions alone or combined with child or teacher interventions are effective for treating early-onset conduct problems, but

not child interventions alone (e.g., Webster-Stratton, Reid, & Hammond, 2004). Pharmacological approaches generally show minimal effects and pose major risks in terms of side effects, particularly among the antipsychotic approaches that have been increasingly used for aggression, and are more widely used for youth who receive Medicare.

A Potentially More Cohesive Explanatory Framework

Increasing evidence suggests that many of the factors that have been presumed within the literature to be "protective factors" are themselves a marker of severity of the original event. For example, in reviewing the implications of their studies findings on morbidity of holocaust survivors, Yehuda et al. (1998) remarked that it was notable that in these severe cases putative risk and protective factors such as axis I psychopathology and education level served no predictive value. Thus, one possibility is that putative neurocognitive risk factors that are linked to externalizing psychopathology might actually be a consequence rather than an independent genetically based antecedent to pathology.

Others have cited protective factors for more severe EXT to include qualities such as "good intelligence," "low impulsivity and easy temperament," "non-aggression prone social cognitions and beliefs," and "intensive involvement in family activities," (Loesel & Farrington, 2012, p.S18). These are noteworthy for their alignment with interpersonal trauma-stress symptomology particularly of a more complex nature, suggesting that "protection" is in this case simply a marker of interpersonal stress and trauma exposure severity.

Low baseline cortisol has garnered much attention in the EXT literature as potential biomarker for more severe forms of externalizing. As articulated by Loney and colleagues (2006), "Low cortisol does not appear to be a general correlate of antisocial behavior or conduct disorder. Rather, it seems to be a unique feature of a small subgroup exhibiting the most

persistent and severe conduct disorder presentations" (Loney, Butler, Lima, Counts & Eckel, 2006, p. 30). Studies that have examined the offspring of Holocaust survivors, however, cast doubt on this interpretation. Among Holocaust survivors, maternal PTSD conferred risk for attenuated baseline cortisol levels among their non-exposed offspring (Yehuda et al., 2008). Thus, rather reflecting a biological deficit or vulnerability to antisocial behavior, it is more likely serving as an adaptive epigenetic mechanism to enable the individual to be more perceptive to their environment. This would be consistent with the concept of an alternative stress responsive pathway, that some researchers have posited (Lupien et al., 2009).

Furthermore, recent psychobiological findings that tested predictions of the conduct disorder/callous unemotionality (CD-CU) framework (analogous to DSM-5 childhood-onset CD with limited prosocial emotions) in young infants found results counter to predictions of blunted psychophysiological activity and a fearless temperament (e.g., Hawes et al., 2009; Barker et al., 2011), but fully consistent with an experience-dependent functional-adaptation perspective. Specifically, they found that infants who were later characterized as CD-CU as children exhibited a pattern of hyperactivity of the HPA axis and autonomic nervous system, and exhibited high intensity fear reactivity at 15 months old. In discussing possible explanations for their results, Mills-Koonce et al. noted the potential role of allostatic load, stating, "For those young children who are particularly sensitive to environmental conditions, harsh and unpredictable environments may lead to severe and chronic emotional and psychophysiological dysregulation. The effects of such an allostatic load (McEwen, 1998) may result in the overall down-regulation of the biobehavioral stress response system and a multi-system transition from hyper- to hyporeactivity over time" (Mills-Koonce et al., 2015, p.152).

Finally, I suggest that the LCP framework is further flawed in a number of ways, which I suggest is the byproduct of misconceptualizing and mislabeling the LCP construct, and by extension, mistreating the subset of individuals who have been assigned this label and neglecting a subset who have not. Specifically, (1) it continues to ignore the subset of individuals who do not persist in acting out or breaking laws in response to chronic oppression and mistreatment, but who still suffer from complex trauma; (2) among those it does detect, it does so only at the point where their behavior impacts another person or property; (3) even though trauma, fear, and chronic invalidation constitute what I posit to be the *actual* life-course persistent features in one's life, the focus of individuals' diagnoses and treatment centers around their disruptive, defiant, and oppositional behavior or disorderly conduct; and (4) it pits the product of collective adult failures across institutions, policies, planning, practices, investment, and priorities onto the conduct of a mistreated child.

In contrast, the alternative theoretical framework and conceptualization proposed herein accords with the significant overlap between (unresolved) complex trauma and the LCP construct, while at the same time, EXT as a correlate but not the core of complex trauma is also consistent with the fact that not all complex trauma manifests in pronounced EXT spectrum presentations.

Conclusion

To date, theories of externalizing (and similar constructs by different names) have generally drawn upon trait level individual differences to explain variation, most commonly characterological differences pertaining to impulsivity, constraint, disinhibition, sensation seeking, and attentional differences. There is good evidence to suspect that the full story may be

more complex, and particularly so for more pronounced presentations of externalizing. One possibility is that externalizing behaviors that exceed the normative population range might be more fruitfully understood by individual differences in cumulative life adversity and resulting complex traumatic stress presentations. I propose that this possibility merits serious consideration.

As Moffitt noted herself in the debut of her typology in 1993, a taxonomy for classifying antisocial behavior was not new. This may be fundamentally part of the problem. I suggest that a paradigm shift in the way more severe adolescent EXT is conceptualized might be overdue, and should start first and foremost with fundamentally changing the language used to describe these adolescents (and later adults)—a language that is informed by their voices, insights, and perspective, rather than by a narrative that to date has been drafted by adults whose power, positionality, and individual life experiences are, in most cases, far removed from the adolescents they speak for.

One of the goals of the current discussion was to introduce the possibility that the more severe forms of externalizing behavior, including the LCP profile that has been posited to constitute a qualitatively distinct type of person, to encourage further reflection, particularly within the criminal justice, forensic risk assessment, and clinical diagnostic classification, assessment, and treatment domains, on the possibility that predominant approaches to adolescent "deviance" might benefit from a fundamental reconceptualization of the lens through which disruptive behavior in conjunction with implicit class, racial, and adolescent-related biases might be a matter of perception that needs to change within the realms of criminal justice, education, and psychology, starting at the level of the individuals who work within systems that have not traditionally reflected on their individual role and collective identity

position in what is disproportionately viewed as young people's problems or the problems of their immediate family—the problems of an "other." I believe much can be gained within clinical psychology and the study of adolescent behavior with a more critical examination of context, to include greater use of historical frames of reference, narrative, and identity position of the researchers. In sum, I agree with Adams and Salter (2011, 2013) who proposed that "the time is ripe for development of a Critical Race Psychology" (2011, p. 1377).

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Study 2:

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