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Antisocial Behavior from Adolescence to Early Adulthood: Heritability, Stability, and Correlates using a Longitudinal Twin Sample

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ANTISOCIAL BEHAVIOR FROM ADOLESCENCE TO EARLY ADULTHOOD: HERITABILITY, STABILITY, AND CORRELATES USING A LONGITUDINAL TWIN SAMPLE

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University.

by

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Abstract

ANTISOCIAL BEHAVIOR FROM ADOLESCENCE TO EARLY ADULTHOOD: HERITABILITY, STABILITY, AND CORRELATES USING A LONGITUDINAL TWIN SAMPLE

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The purpose of this study was to examine the heritability, stability, and outcomes of antisocial behavior from adolescence into adulthood in a longitudinal twin sample. Specifically, the genetic and environmental influences on conduct disorder, adult antisocial behavior, and alcohol dependence were examined. The influence of genes and environment on the relationship between these disorders was also examined. The study utilized a subset of FinnTwin12, a population-based twin study that consists of five consecutive birth cohorts. The subsample consisted of 1035 twin pairs (N = 2070) and of that 2070, 1854 completed the intensive interview at age 14. At age 22, 1345 twins completed the interview. Participants in the study completed age-appropriate variations of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA). Analyses were run separately by gender. Results provide support for the significant influence of genetic factors on the development and persistence of antisocial behavior. For both males and females, model fitting indicated that genetic influences are the most influential contributor to the association between conduct disorder and adult antisocial

behavior and its stability across time. Additionally, there were no age specific genetic effects suggesting that the genes influencing conduct disorder are the same as those influencing adult antisocial behavior. Results for the relationship between conduct disorder and alcohol dependence differed by gender. For females, insufficient power made it difficult for the model to discriminate between the effects of genetics and shared environment, but the full model suggested that shared environmental influences explained the greatest proportion of variance in the relationship. For males, genetic influences were primarily responsible for the relationship between conduct disorder and alcohol dependence. Similar results were found for males when the relationship between alcohol dependence and adult antisocial behavior was explored. For females, genetic and nonshared environmental influences were the primary source of covariation between these two disorders. The data suggest that the etiology of conduct disorder, adult antisocial behavior, and alcohol dependence vary by gender.

Antisocial Behavior from Adolescence to Early Adulthood: Heritability, Stability, and Correlates Using a Longitudinal Twin Sample

The study of antisocial behavior in childhood, adolescence, and adulthood has yielded equivocal results in several domains. One consistency across findings is that antisocial behavior has short and long-term consequences that impact the individual, those close to them, and society as a whole. Adolescents engaging in antisocial behavior are more likely to experience difficulties in academic achievement, peer relationships, depression, anxiety, and social competence (Kazdin, 1987; Asarnow, 1988; Zoccolillo, 1992; Renouf, Kovacs, & Mukerji, 1997). These antisocial adolescents may become antisocial adults who face higher rates of divorce, unemployment, substance abuse, criminality, psychiatric disorders, and physical illness (Caspi, Elder, & Bern, 1987; Rose, Dick, Viken, Pulkkinen, & Kaprio, 2004; Fergusson, Horwood, & Ridder, 2005; Colman et al., 2009). The tremendous negative impact, to the individual and society that is associated with antisocial behavior underscores the need for research aimed at understanding the causes of this behavior. Understanding the genetic and environmental influences that facilitate the development and maintenance of antisocial behavior could provide insight for intervention and prevention throughout the life course. This review of the literature will provide a glimpse into the variability in definitions of antisocial behavior, perspectives on antisocial behavior across the lifespan, and genetic and environmental influences on the development of these behaviors, while also demonstrating the need for additional research in these areas.

The Heterogeneity of Antisocial Behavior

Antisocial behavior encompasses behaviors that violate social norms and/or impact personal and property rights of others (Simcha-Fagan, Langner, Gersten, & Eisenberg, 1975;

Burt & Donnellan, 2009). This definition provides commonality to a set of behaviors that are often quite individualized. The heterogeneity of these behaviors is evident in varied definitions in clinical and research arenas.

DSM Diagnoses of Antisocial Behavior. The Diagnostic and Statistical Manual (DSM) published by the American Psychiatric Association is the go-to guide for clinicians working with individuals in treatment settings. The DSM outlines symptoms for disorders that occur across the lifespan and provides information relevant to treatment including etiology, course, prevalence, and comorbidity with other diagnostic categories. The DSM-III-R includes two diagnoses for antisocial behavior: Conduct Disorder and Antisocial Personality Disorder (American Psychiatric Association, 1987). The manual also recognizes that individuals may engage in these behaviors at a subclinical level and includes "V-Codes" for use when information is insufficient to attribute the problem to one of the formal diagnoses (American Psychiatric Association, 1987). The DSM-III-R has two V-codes related to antisocial behavior: Childhood or Adolescent Antisocial Behavior and Adult Antisocial Behavior.

Conduct disorder (CD) is a disorder evident in childhood or adolescence. The DSM-III-R criteria for a diagnosis of CD are listed in Table 1. It is one of the most frequently diagnosed childhood disorders by mental health facilities (American Psychiatric Association, 2000; Ingersoll & Previts, 2001). A population study of children age 9-16 estimated that 9.0% of the children would meet criteria for conduct disorder by age 16 (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Children and adolescents who engage in antisocial behaviors, but the behaviors are less persistent (i.e. lasts less than six months) and more isolated can be given a V-code classification of Childhood or Adolescent Antisocial Behavior.

Table 1.

DSM-III-R Criteria for Conduct Disorder.

A. A disturbance of conduct lasting at least six months, during which at least three of the following have been present:

    1. Has stolen without confrontation of a victim on more than one occasion (including forgery)
    2. Has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning)
    3. Often lies (other than to avoid physical or sexual abuse)
    4. Has deliberately engaged in fire-setting
    5. Is often truant from school (for older person, absent from work)
    6. Has broken into someone else's house, building, or car
    7. Has deliberately destroyed others' property (other than fire-setting)
    8. Has been physically cruel to animals
    9. Has forced someone into sexual activity with him or her
    10. Has used a weapon in more than one fight
    11. Often initiates physical fights
    12. Has stolen with confrontation of a victim (e.g. mugging, purse-snatching, extortion, armed robbery)
    13. Has been physically cruel to people

B. If 18 or older, does not meet criteria for Antisocial Personality Disorder

In adulthood, individuals engaging in antisocial behaviors may be given a diagnosis of Antisocial Personality Disorder (APD; Table 2). A survey using a nationally representative sample of adults aged 15 to 54 found lifetime prevalence rates to be 3.5% (Kessler et al., 1994). Although, it is estimated that approximately 4% of those who engage in antisocial behavior as adults committed their first criminal offense after the age of 17, the DSM diagnostic criteria requires evidence of CD before age 15 in order to be diagnosed APD (Elkins, Iacono, Doyle, & McGue, 1997). However, late-onset does not make these individuals any less antisocial and if they are in treatment for such behaviors, it should be acknowledged. Those individuals could receive the V-code of Adult Antisocial Behavior.

Table 2.

DSM-IV Criteria for Antisocial Personality Disorder.

A. There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by three (or more) of the following:
   1) Failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest
   2) Deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure
   3) Impulsivity or failure to plan ahead
   4) Irritability and aggressiveness, as indicated by repeated physical fights or assaults
   5) Reckless disregard for safety of self or others
   6) Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations
   7) Lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another
B. The individual is at least age 18 years.
C. There is evidence of Conduct Disorder with onset before age 15 years.
D. Occurrence of antisocial behavior not exclusively during the course of Schizophrenia or Manic Episodes.

Additional Definitions of Antisocial Behavior. Although the DSM may be the primary resource for clinicians assessing for psychopathology, there are a number of other ways to operationalize antisocial behavior. There are several structured instruments often used to monitor behaviors in treatment or for research purposes. Additionally, distinctions have been drawn based on types of antisocial behavior.

Meta-analyses examining research on antisocial behavior list multiple methods for assessing the behaviors. Some techniques include observation of behaviors as home, observation of physical aggression in a laboratory setting, or official records of criminal histories (Moffitt, 2005). Other techniques involve more standardized instruments such as the Child Behavior Checklist (CBCL; Achenbach, 1991), Multidimensional Peer Nomination Inventory (MPNI; Pulkkinen, Kaprio, & Rose, 1999), Minnesota Multiphasic Personality Inventory-2 (MMPI-2;

Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer), Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982), and the Basic Personality Inventory (BPI; Jackson, 1989). Several of these instruments have multiple forms allowing for self-report, parent report, peer report, or teacher report. Two of these instruments are relevant to the current study and will now be discussed in more detail to further explain the differences in operationalization.

The Multidimensional Peer Nomination Inventory (MPNI; Pulkkinen et al., 1999) was designed to assess behavioral problems, emotional problems, adjustment, and social activity. The measure involves peer nomination where classmates were asked to nominate three female and three male classmates who best fit the behavior described in each of 30 items. Additionally, a 37-item measure assesses these, plus some additional behaviors, based on parent and teacher report (Pulkkinen et al., 1999). Behavioral problems were defined by items assessing hyperactivity-impulsivity, inattention, and aggression and were based on a behavioral and emotional regulation model. Items within these categories were similar to those of the DSM-III-R, but outline a different operationalization of antisocial behavior. For example, behaviors such as stealing, running away, fire setting, cruelty to animals and destruction of property were not included. However, hyperactivity-inattention was included as a behavioral problem with aggression. Hyperactivity and inattention are often associated with antisocial children but diagnostically these behaviors are part of a separate childhood diagnosis, Attention-Deficit Hyperactivity Disorder. It appears the problem behavior scale is broader than other definitions of antisocial behavior.

Another frequently utilized measure is the Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL is a standardized instrument with two versions: one for toddlers (ages 2-3) and one for early childhood through adolescence (ages 4-18). The measure addresses a broad

spectrum of behavioral and emotional problems, including both internalizing and externalizing behaviors. As with the MPNI, there are multiple forms including forms to be completed by the parent and teacher. Additionally, the CBCL has a Youth Self-Report form that can be completed by adolescents age 11-18. The externalizing scale most closely parallels antisocial behavior and encompasses most of the behaviors in the DSM criteria for CD. Additionally, it divides externalizing behavior into two domains: aggression and delinquency (Achenbach & Ruffle, 2000). This distinction implies that antisocial behavior may not be a unitary construct.

Research supports the idea that there are two distinct factors (i.e. aggressive and non-aggressive or rule-breaking) within the broader construct of antisocial behavior. Aggressive antisocial behavior is characterized by acts like fighting, arguing, and confrontation, while the nonaggressive/rule-breaking dimension encompasses behaviors that are non-confrontational like stealing, lying, and vandalism (Fergusson, Horwood, & Lynskey, 1994; Frick et al., 1993; Loeber & Schmaling, 1985). It is posited that these subfactors may provide a more accurate picture of childhood and adolescent antisocial behavior than the unitary presentation in the DSM criteria for CD (Tackett, Krueger, Sawyer, & Graetz, 2003). These dimensions have implications for the course of the behavior, treatment, and associated outcomes (Burt, 2009; Frick et al., 1993; Gelhorn et al., 2006; Loeber & Schmaling, 1985). While individuals often engage in behaviors that fall in one domain or the other, it is also possible for them to engage in behaviors across both domains.

Research has repeatedly demonstrated the presence of these domains within antisocial behavior and several meta-analyses have confirmed their presence across studies utilizing different measures. Loeber and Schmaling (1985) reviewed twenty-eight studies related to childhood antisocial behavior. The majority of these studies used a clinical sample. They found

that antisocial behaviors, as rated by parents and clinicians, resulted in a unidimensional model with covert (i.e. nonaggressive/rule-breaking) behaviors at one end, and overt (aggressive) acts at the other. A subsequent meta-analysis examining 61 studies confirmed this distinction between aggressive and non-aggressive behaviors within childhood and adolescent antisocial behavior (Quay, 1986). The studies spanned almost 40 years and included behavior ratings, behavior observations, peer ratings, self-reports, and reports by other informants like parents, teachers, and clinicians.

While these previous studies have examined the domains within the broader spectrum of antisocial behavior, additional studies focusing on DSM criteria have also supported the two factor distinction. Fergusson and colleagues (1994) conducted a confirmatory factor analysis of the DSM-III-R criteria for conduct disorder. Utilizing parent report, self-report, and police records to ascertain diagnoses of CD, researchers demonstrated that CD may not be a unitary construct and that a distinction should be made between overt and covert CD in order to better understand the disorder (Fergusson et al., 1994). Based on the study by Fergusson et al. (1994), symptoms of the aggressive and nonaggressive domains are distinguished in Table 3.

Using the more recent, DSM-IV CD criteria, Tackett and colleagues (2003) conducted exploratory factor analysis to examine these two domains and also examine the relationship between the domains of CD and the CBCL aggression and delinquent scales. They not only demonstrated support for the distinction within DSM criteria, but also found that the two factors within CD were best predicted by similar types of antisocial behavior from the CBCL. The Aggressive behavior scale of the CBCL predicted the overt (i.e. aggressive) dimension and the Delinquent behavior scale predicted the covert (i.e. nonaggressive/rule breaking) dimension (Tackett et al., 2003). However, factor loadings of several CD items between domains were

different than that found by Fergusson and colleagues (1994). Specifically, "stolen with confrontation" had higher loading on the covert factor, while "often tells lies" loaded higher on the overt factor. Additionally, the loadings for "destroyed others' property" loaded moderately on both factors (overt = .54, covert =.47). The reasons for these changes are not entirely clear, and warrant further exploration.

Table 3.

Aggressive and Nonaggressive Symptoms in the DSM-III-R.

Aggressive

1. Has deliberately engaged in fire-setting
2. Has deliberately destroyed others' property (other than fire-setting)
3. Has been physically cruel to animals
4. Has forced someone into sexual activity with him or her
5. Has used a weapon in more than one fight
6. Often initiates physical fights
7. Has stolen with confrontation of a victim (e.g. mugging, purse-snatching, extortion, armed robbery)
8. Has been physically cruel to people

Nonaggressive

1. Has stolen without confrontation of a victim on more than one occasion (including forgery)
2. Has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning)
3. Often lies (other than to avoid physical or sexual abuse)
4. Is often truant from school (for older person, absent from work)
5. Has broken into someone else's house, building, or car

With all the evidence suggesting the existence of these domains, it seems researchers would be remiss to exclude them when operationalizing antisocial behavior. Like the design of the CBCL, a more effective way of measuring the behavior may be to look at the overall level of antisocial behavior as well as the domains where those behaviors lie. This could provide a more informative picture for treatment purposes. These domains have not been examined within an

adult sample. It would be informative to know if the domains still exist in adulthood or if new domains emerge.

Gender Differences in Antisocial Behavior. Both the prevalence rates and developmental course of the antisocial behavior differ depending on whether the individual is male or female. However, the nature and magnitude of these differences is still not fully understood. Prevalence rates of CD vary significantly between gender, with estimates that 14.1% of boys and only 3.8% of girls will meet criteria by age 16 (Costello et al., 2003). Differences by gender also extend to adult antisocial behavior. Lifetime prevalence rates for adults age 15 to 54 approximate that 5.8% of males and 1.2% of females will meet criteria for APD (Kessler et al., 1994).

Differences in the ratio of male to female antisocial behaviors decrease over time. In early childhood, males are more likely to display antisocial behaviors, while females more often display internalizing behaviors. In adolescence the rates of antisocial behavior are more equal (Cale & Lilienfeld, 2002). It is possible that the dramatic difference in prevalence is due to the way in which females engage in antisocial behaviors. Females may engage in more covert/nonaggressive behaviors, which can be harder to measure when collecting reports of behavior from parents or teachers and may result in lower estimates (Cale & Lilienfeld, 2002; Loeber & Hay, 1997).

Despite displaying less severe or less frequent antisocial behavior, females are still at risk for negative outcomes associated with antisocial behavior. Additional research is needed that includes females, as historically they have often been overlooked in studies of antisocial behavior.

Developmental Trajectories of Antisocial Behavior

Antisocial Behavior Across the Lifespan. The age of onset of antisocial behavior can be a marker for the pathway towards continuity or desistence of the behaviors. While it is true that antisocial behaviors do not persist across the lifespan for everyone, for those who do continue into adulthood the behaviors most often begin during childhood or adolescence (Loeber, 1982; Robins, 1978). Individuals who display more frequent and severe antisocial behaviors in early childhood are at the highest risk for continued antisocial behavior in adulthood. Speaking only diagnostically, it has been estimated that individuals with CD who began the behaviors in early childhood, are almost twice as likely to meet criteria for APD as adults (Robins, Tipp, & Pryzbeck., 1991). A recent study examining the predictability of APD based on retrospective report of CD symptoms found that progression to APD was the norm (Gelhorn, Sakai, Price, & Crowley, 2007). Using an epidemiological sample that assessed various psychiatric and substance use disorders, researchers determined that 79% of men and 75% of women who met criteria for a CD diagnosis went on to meet criteria for APD.

Aside from the diagnostic categories previously discussed, researchers have established a number of titles that classify antisocial individuals based on age of onset and the potential trajectory for these behaviors. Antisocial individuals have been classified as "adolescent-limited" and "life-course persistent", "early starters" and "late starters", and "continuous" and "transitory" (DiLalla & Gottesman, 1989; Moffitt, 1993; Taylor, Iacono, & McGue, 2000). Although the labels vary, the definitions are similar and relate to the point at which an individual begins and desists engaging in antisocial behavior. The life-course persistent individuals are those who participate in antisocial behavior during every stage of their life (Moffitt, 1993). They begin engaging in these behaviors in early childhood, transition to delinquent behaviors in

adolescence, and maintain some pattern of these behaviors in adulthood (Brennan, Hall, Bor, Najman, & Williams, 2003). In contrast, adolescent limited individuals engage in antisocial behavior from their teens into their mid-20s and then desist. It is believed that these individuals are trying to assert the independence they feel ready for but society does not recognize (Moffitt, 1993). These categories are consistent with the findings that the earlier the start of the behavior, the more likely it is to persist into adulthood. Additionally, they reflect the different diagnostic categories of the DSM (e.g. life-course persistent is likely those individuals who meet CD and later meet criteria for APD, adolescent-limited meet criteria for CD or even just a V-code of Adolescent Antisocial Behavior).

DiLalla and Gottesman (1989) proposed a third group they labeled "late bloomers" who do not engage in antisocial behaviors until adulthood. This developmental trajectory for this category has been less frequently researched. Some researchers propose that those who engage in these behaviors later in life do so because of environmental influences (i.e. unemployment, change in life circumstances, or lack of social controls that were present while living with parents or in school) (McGee & Farrington, 2010). However, other researchers hypothesize that individuals in this category may have engaged in antisocial behaviors prior to adulthood but remained undetected or underreported these behaviors (McGee & Farrington, 2010). Late bloomers are most likely to receive a V-Code of Adult Antisocial Behavior.

Aside from age of onset, stability can also be dependent upon the type of behaviors of the adolescent. Children who display nonaggressive behaviors are more likely to desist than those displaying aggressive behaviors (Loeber, 1991; Eley, Lichtenstein, & Moffitt, 2003). Examining the individual DSM-IV criteria for CD as predictors of APD, researchers explored which criteria significantly predicted sub-clinical versus clinical antisocial behavior, and transient versus

persistent antisocial behavior (Gelhorn et al., 2007). It was concluded that the aggressive, victim-oriented items (i.e. "weapons," "cruel to people," "steal with confrontation," and "lies") were the best predictors of adult ASB, while nonaggressive criteria (i.e. "out late," and "truancy") showed limited utility in predicting persistence. These findings provide additional justification for the need to explore the domains of antisocial behavior. It is possible the domains can serve as markers for who is at the highest risk for persistence into adulthood.

Substance Use Disorders and Antisocial Behavior. Antisocial behavior is often associated with other psychological disorders and negative outcomes. Substance use disorders and antisocial behavior have consistently been linked together, but the nature of the relationship is complex. While there is debate over whether substance use leads to antisocial behavior or vice versa, it is clear that substance use disorders are highly comorbid with antisocial behavior in both adolescence and adulthood.

In a study of adolescents engaged in inpatient and outpatient substance abuse treatment programs, 82% of boys and 63% of girls met diagnostic criteria for CD (Molina, Bukstein, & Lynch, 2002). Additionally, participants with comorbid CD had the most severe substance use profiles. These findings also have been supported in a population-based sample. Using a twin sample, researchers found that participants with CD or combined ADHD-CD had the highest rates of substance use and abuse as measured by a structured clinical interview (Disney, Elkins, McGue, & Iacono, 1999). Even after controlling for gender and ADHD, CD was associated with current use of tobacco, alcohol, marijuana, and any substance, frequency of smoking and alcohol use, and the diagnoses of nicotine, alcohol, and cannabis dependences, and any substance use disorder (Disney et al., 1999). Therefore, adolescents with CD are not only at greater risk for

developing substance use disorders, but these disorders are often more severe than in individuals without CD.

The association between antisocial behavior and substance use problems extends into adulthood. In studies of individuals diagnosed with APD, the most prevalent secondary diagnosis was alcoholism and drug addiction (Bartol & Bartol, 2005). A national epidemiological survey found that alcohol and drug use disorders were most strongly associated with APD in comparison to other PDs (Grant et al., 2004). Similar to children and adolescents with a diagnosis of CD, research suggests that individuals with substance use disorders and APD report more substance-related problems and higher lifetime use of tobacco and most illicit drugs (Westermeyer & Thuras, 2005). Individuals in substance abuse treatment who had comorbid APD were on average younger, but had experienced more admissions to treatment, more days in treatment, and greater cost of treatment than those with a substance use disorder alone.

Like with stability of antisocial behavior, earlier age of onset of antisocial behaviors has been associated with higher rates of substance use disorders (McGue & Iacono, 2005). However, even individuals who begin engaging in antisocial behaviors later in adolescence are still at high risk for developing nicotine, alcohol, and cannabis dependence years later (Marmorstein & Iacono, 2005).

Causes of Antisocial Behavior

Genetic and Environmental Influences on Antisocial Behavior. Antisocial children are more likely to have a parent who is antisocial (Blazei, Iacono, & Krueger, 2006; Blazei, Iacono, & McGue, 2008; Jaffee, Moffitt, Caspi, & Taylor, 2003), indicating that the behavior runs in families. Adoption studies are able to tease apart whether familial resemblance is due to genetic and/or environmental influence because they can demonstrate a link to the biological

parent (genetics), despite the parent playing no role in the child's life (environment). Children adopted at birth and reared away from their antisocial biological parent have been found to exhibit higher externalizing behaviors than adopted children without an antisocial biological parent (Cadoret & Cain, 1980; Cadoret, Troughton, & O'Gorman, 1987; Mednick, Gabrielli, & Hutchings, 1984). These studies suggest that genetic influences are important in the development of antisocial behavior.

Despite this, heritability does not tell the whole story. While antisocial children are more likely to have an antisocial parent, the length of a father's presence in the home has been found to increase the association between father and child antisociality (Blazei et al., 2008). The level of antisociality of the father can moderate this association. It has been found that children who live with a father who engages in high levels of antisocial behavior are not only at genetic risk, but also at risk due to rearing practices of the father (i.e. environmental risk; Jaffee et al., 2003). The behavior of children who live with fathers engaging in antisocial behaviors at such a high level is significantly worse than for children whose antisocial fathers do not live in the home. In contrast, antisocial fathers who live in the home and engage in these behaviors at a lower level have children with fewer behavior problems. The results of such studies illustrate the difficulties in teasing out the causes of antisocial behavior. However, these studies support the idea that it is not one risk factor or type of risk factor that leads to antisocial behavior but a combination of genetic and environmental factors.

Family, twin and adoption studies have established that the development of antisocial behavior involves the interaction between genes and the environment. While both are clearly important, the magnitude of their influence varies across studies. Miles and Carey (1997) explored 24 genetically informative studies that measured aggression, antisocial behavior,

juvenile delinquency, or hostility and found a heritability estimate of around 50%. A subsequent meta-analysis by Rhee and Waldman (2002), involved the review of 52 independent twin and adoption samples. Studies were included if they examined CD or APD clinical diagnoses, general antisocial behavior, aggression, or official records of arrests and convictions. They concluded that there were additive genetic (a^2 = .32), nonadditive genetic (d^2 = .09), shared environmental (c^2 =.16), and nonshared environmental (e^2 = .43) influences on antisocial behavior (Rhee & Waldman, 2002). Although the heritability estimate was somewhat lower (41%), it is possible this difference is due to inclusion or exclusion of particular studies and only taking into account one estimate of heritability from each reviewed study even when multiple estimates were available (Moffitt, 2005).

More recently, Moffitt (2005) reviewed the more than 100 studies on genetic and environmental influences of antisocial behavior. Instead of using statistical techniques to estimate the genetic effect, Moffitt (2005) examined the distribution of heritability estimates across the studies and found that they ranged from 0% to 80% with the majority converging around 50%. Additionally, shared environmental factors clustered around 20% while nonshared environmental influences accounted for the final 30%.

These large scale studies provide some resolution to the discrepancies found across individual studies and suggest a more even distribution between genes and environment than previously thought. It appears that genetic influences account for approximately 50% of the variance in antisocial behavior, while shared and nonshared environmental factors comprise the remaining 50% (Moffitt, 2005).

Genetic and Environmental Influences on the Domains of Antisocial Behavior. Although these findings on genes and environment may provide some resolution with regards to

the unitary construct of antisocial behavior, they do not provide information on the etiology of the domains of antisocial behavior. Several studies focused on the etiology of these domains have concluded that the domains have shared genetic and environmental influences, but each domain also has unique factors (Button, Scourfield, Martin, and McGuffin, 2004; Eley et al., 2003; Gelhorn et al., 2005; Gelhorn et al., 2006; Tackett, Krueger, Iacono, & McGue, 2005). These same studies have concluded that aggressive behaviors are more highly heritable, while nonaggressive behaviors are more influenced by shared environmental factors.

In an effort to further explore etiological differences within the domains, Burt (2009) conducted a meta-analysis of 34 independent studies that measured aggressive and/or nonaggressive domains of antisocial behavior. Genetic influences were large for the aggressive domain (65%) and more moderate for the nonaggressive domain (48%), while shared environment proved to be a more significant influence on the nonaggressive domain (18%) than the aggressive domain (5%). Unique environmental influences were marginally more important to nonaggressive (34%) than aggressive (30%) behaviors. These results suggest meaningful differences in the etiology of aggressive and nonaggressive antisocial behaviors.

Reasons for Variability. As with the definitions of antisocial behavior, there are several factors that help to explain the reasons for variability in the magnitude of genetic and environmental influences. The meta-analyses previously described by Rhee and Waldman (2002) examined moderators of this magnitude of which several were significant; the most relevant to the current study is age. Gender differences in the heritability of antisocial behavior have not consistently been found (Burt, 2009; Moffitt, 2005; Rhee & Waldman, 2002).

Age. The magnitude of the influence of genes and the environment on antisocial behavior changes across the life span. As young children, genetic influences appear to be the primary

predictor of antisocial behavior, but moving into adolescence shared and unique environmental factors appear to play a more significant role (Jacobson, 2005; Rhee & Waldman, 2009). As individuals move into adulthood, antisocial behavior is again largely influenced by genetic factors, with the impact of shared environmental influences declining (Jacobson, 2005; Lyons et al., 1995; Miles & Carey, 1997). In a recent prospective study that examined antisocial behavior at four timepoints from 8 years old to 20 years old, researchers found that genetic and non-shared environmental effects were most influential at ages 8-9 years and 19-20 years, while genetic, shared, and non-shared environmental effects influenced antisocial behavior at ages 13-14 years and 16-17 years (Tuvblad, Narusyte, Grann, Sarnecki, & Lichtenstein, 2011). The Child Behavior Checklist (CBCL; Achenbach, 1991) and a self-report delinquency questionnaire were used to measure antisocial behavior in this study.

Similarly, the genetic and environmental influences on the domains of antisocial behavior change across the lifespan. Burt (2009) found that genetic and environmental influences on aggressive and nonaggressive behavior do not differ in early and middle childhood, but in adolescence shared environment was more influential on nonaggressive than aggressive behavior. Overall, a slight increase in genetic influence and a decrease in shared environmental influences were seen for aggressive behaviors from childhood to adolescence, while genetic influences decreased for nonaggressive behaviors and shared environmental influences remained stable during this transition. This could mean that it is during adolescence that the domains are the most etiologically distinct.

Despite the importance of this study by Burt (2009), estimates of changes from childhood to adolescence were based on cross-sectional, not longitudinal, data. Additionally, the transition to adulthood was not examined because the studies included in the meta-analysis only measured

these behaviors in childhood and adolescence. To get a better understanding of genetic and environmental factors influencing the domains across the lifespan, exploration is needed into adulthood. It is the studies that assess antisocial behavior in individuals at multiple developmental points that assist in determining whether genetic risk factors for antisocial behavior overlap or are distinct for juveniles and adults (Slutske, 2001).

The Current Study

The current study attempted to build on previous research by using a longitudinal twin sample to explore the domains of antisocial behavior, the stability of these behaviors from adolescence into adulthood, and the association between antisocial behavior and substance use. These analyses were examined by gender. The current study benefitted from the availability of a longitudinal sample that included concurrent and retrospective report of antisocial behaviors. The specific questions answered by the current study were:

1) Was there evidence for the nonaggressive and aggressive domains within the population?
2) What influenced the stability of antisocial behavior and its two domains from age 14 to 22?
3) To what degree did the genetic and environmental influences on conduct disorder and adult antisocial behavior overlap?
4) What was the magnitude of genetic and environmental influences on conduct disorder and the associated outcome of alcohol dependence?
5) What was the magnitude of genetic and environmental influences on the relationship between adult antisocial behavior and alcohol dependence?
6) Were there gender differences in the prevalence, stability, or associated outcomes?

Methods

Participants & Procedure

The current research is based on a longitudinal study that examines genetic and environmental sources of variability in health-related behaviors in Finnish twins. FinnTwin12 is a population-based twin study that consists of five consecutive birth cohorts (1983-1987; Kaprio, Pulkkinen, & Rose, 2002; Rose, Dick, Viken, Pulkkinen, & Kaprio, 2004). Twins were initially identified using the Central Population Registry. Families were excluded if one co-twin was deceased or living outside Finland, the co-twins lived apart from both biological parents, or if there was no residential address available for a twin. Of eligible families 87% (N=2724) returned the family questionnaire and 2567 twin pairs completed baseline questionnaires (Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005). At baseline, a family questionnaire, questionnaires by both parents, and questionnaires by both twins were completed. At age 14, many of the same measures were completed, but additional questions regarding alcohol use and intoxication, peer alcohol use, smoking, drugs, and self esteem were added. Additional assessments with similar questionnaires were completed at age 17, 22, and are currently on-going.

Within this epidemiological sample, a sub-sample of twins were selected and called the intensive subset. The data for the current study is derived from this intensive subset. The sub-sample is comprised of a sample of twins from the 1983 cohort who live in urban areas of Finland (13%), a random sample of participants from the remaining cohorts (59%; 1984-1987), and all twins at elevated familial risk for alcohol problems based on parental reports of alcohol use (28%; Rose et al., 2004). To be included in the sub-sample, at least one parent had to return the family questionnaire and provide permission for school contact, at least one biological parent was Finnish speaking, and there could be no new information provided that would prevent the

twins from being included in the study, such as a twin is studying abroad or is handicapped in a way that precludes participation (Kaprio et al., 2002; Rose et al., 2004). The parental risk for alcohol-related problems was assessed using the Malmö- modified Michigan Alcohol Screening (Mm-MAST; Seppä, Sillanaukee, & Koivula, 1990). The Mm-MAST is a 9-item diagnostic screen for lifetime alcohol problems (see Appendix A). To increase predictive association with DSM alcohol disorders, two additional items were added for FinnTwin12 study. The Mm-MAST was included as part of the baseline parental questionnaire. Twins were considered at risk for alcohol related problems when one or both parents exceeded a cutoff of six or more of the eleven items (Rose et al., 2004).

The sub-sample consisted of 1035 twin pairs (N = 2070). Face to face interviews were conducted primarily during school hours using a notebook computer to enter the responses online (Kaprio et al., 2002). Participants in this subsample completed a full psychiatric interview at ages 14 and 22. At age 14, a total of 1854 twins completed the assessment; 678 MZ twins (339 males, 339 females), 598 DZ twins (325 males, 273 females), 542 Opposite Sex DZ (OSDZ) twins (267 males, 275 females), and 36 of unknown zygosity (14 male, 20 female, 2 unknown gender). At age 22, 73% of twins were retained (N= 1345, MZ twins = 220 males, 275 females, DZ twins = 218 males, 214 females, OZDZ = 188 male, 200 female, unknown zygosity = 8 male, 16 female, 6 unknown gender).

Measures

Measures of Antisocial Behavior and Psychiatric Outcomes. Participants in the study completed age-appropriate versions of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) (Bucholz et al., 1994; Kuperman et al., 2001). At age 14, the Child Semi-Structured Assessment for the Genetics of Alcoholism, Adolescent version (C-SSAGA-A) was

administered. The C-SSAGA-A is a polydiagnostic instrument that was developed by the Collaborative Study on the Genetics of Alcoholism (COGA; Kuperman et al., 2001). The C-SSAGA-A was derived from the diagnostic Interview for Children and Adolescents (Reich, Herjanic, Welner, & Gandhy, 1982) and makes lifetime diagnoses on a variety of psychiatric disorders including ADHD, CD, ODD, and alcohol dependence (Kuperman et al., 2001). At age 22, the adult version of the SSAGA was completed. The SSAGA is a comprehensive, semi-structured interview used to assess alcohol dependence and related psychiatric disorders (Bucholz et al., 1994). Administration takes an average of two hours. A full list of disorders assessed in the SSAGA is located in Appendix B. Psychotic disorders are not covered in the SSAGA, although there is a screener for schizophrenia.

The versions of the C-SSAGA-A and SSAGA used in FinnTwin12 make DSM-III-R and DSM-IV diagnoses, respectively, by translating each of the symptoms that comprise diagnostic criteria into questions with a yes/no response format (Bucholz et al., 1994; Dick, et al., 2005). The C-SSAGA-A and SSAGA code symptoms to indicate that the individual did not engage in the behavior, engaged in the behavior while under the influence of alcohol or drugs, or engaged in the behavior not under the influence. For this project, if the participant endorsed engaging in the behavior at any point, the symptom was coded as present. All symptoms were recoded in this dichotomous manner.

Although both instruments were originally developed in English, they were translated for FinnTwin12 by professional translators and then reviewed by Finnish and American psychiatrists with knowledge of the instrument. The items for CD are located in Appendix C and Appendix D contains the APD questions. For the current study, age 22 symptoms only included those associated with adult antisocial behavior (AAB) and not the retrospective report of conduct

disorder. Appendix E provides the AD items from the SSAGA. The first questions of the AD section were developed to screen out individuals who had never consumed alcohol, never drank regularly, or had never been intoxicated. Participants who did not endorse any of the above were not asked the remaining alcohol dependence questions. The current analyses include all individuals who indicated they had ever consumed alcohol, even if they were not a regular drinker or were never intoxicated. Those who never consumed alcohol were excluded from analyses as their inclusion would not be genetically informative.

The SSAGA has demonstrated good test-retest reliability for alcohol dependence, κ=.84 for a sample including users and non-users, which was somewhat better than that for alcohol abuse, κ=.74. The reliabilities for APD were mixed from κ=.42 in one study to κ=.70 in another. The difference is believed to be due to unreliable reporting or improper dating of childhood symptoms before the age of 15 (Bucholz et al., 1994). Item reliability was acceptable for most disorders.

Analyses

The primary goal was to analyze CD, AAB, and AD symptoms. CD symptoms were based on the DSM-III-R criteria for CD, while AAB and AD symptoms were derived from DSM-IV symptom criteria. Analyses were conducted in a stepwise fashion, with many analyses dependent upon the initial factor analyses of age 14 CD symptoms and age 22 AAB symptoms. Analyses began with an exploration of the structure of antisocial behavior at age 14 and age 22. Subsequent analyses used twin modeling to explore genetic and environmental contributions to CD, AAB, and AD, and the relationship between disorders. Lastly, latent class analysis was conducted to explore subtypes of antisocial behavior.

Factor Analyses

We first ran exploratory factor analyses on CD and AAB symptoms to determine if each disorder was a unitary construct in this sample. Factor analyses were run separately for males and females to explore whether the structure of antisocial behavior differed by gender.

Bivariate Modeling

Based on the results of the factor analyses, genetic analyses were conducted. To explore the genetic structure across time, bivariate modeling was used. Bivariate modeling provides estimates of genetic, shared environmental, and nonshared environmental influences. Importantly, bivariate modeling allows one to estimate the extent to which covariation between two traits is due to shared genetic and/or environmental influences incorporating information in the cross-twin cross-trait correlations (Dick et al., 2007). These models were investigated for age 14 CD and age 22 AAB and age 14 CD and age 22 AD.

Twin models allow researchers to estimate the magnitude of additive genetic influences (heritability), environmental influences that cause siblings to be more similar (shared environment), and environmental influences that cause siblings to be more different (nonshared environment; Neale & Maes, 1993; Dick, et al., 2007). This is done by comparing the observed correlations between monozygotic (MZ) and dyzygotic (DZ) twin pairs. MZ twin pairs share all of their genetic variation while DZ pairs share just 50% of their genetic variation on average (Gelhorn et al., 2005; Dick et al., 2007). When only additive genetic effects are present, correlations among DZ twins should be half that of correlations among MZ twins based on the percentage of shared genetic variation. Shared environmental effects are suggested by DZ twin correlations that exceed half the MZ twin correlations. Under the usual assumptions of twin modeling, shared environmental influences correlate 1.0 between pairs of both MZ and DZ co-twins (Plomin, DeFries, McClearn, & McGuffin, 2008). In contrast, nonshared environmental

influences are uncorrelated between co-twins and decrease the covariance between siblings. The nonshared environmental factors also includes measurement error for the model.

For the twin models, symptom counts were treated as ordinal variables as in previous studies using this dataset (Rose et al. 2004). All models were run separately by gender because of evidence of sex specific genetic effects at age 14 for conduct disorder in this sample (Rose et al., 2004). Other researchers answering similar questions about the relationship between conduct disorder and adult antisocial behavior also found sex specific genetic effects (Meier, Slutske, Heath, & Martin, 2011). Since analyses were run separately by gender, opposite sex twin pairs were not included in genetic models. Model fit was assessed using the change in chi-square between models and Akaike's Information Criterion (AIC; Akaike, 1987). A non-significant *p* and negative AIC are indicators of improved fit and parsimony.

Person Centered Approach

Latent class analysis was incorporated into the current study to categorize the sample based on antisocial behavior and explore if there were subtypes of conduct disorder or AAB. It was anticipated the classes would resemble the nonaggressive and aggressive domains previously described. Factor analysis and twin modeling are variable centered approaches, while LCA focuses on the individual. A person centered approach can be useful in looking at unobserved heterogeneity (Muthén & Muthén, 2000). It can also be useful for determining individuals susceptible to certain types of behaviors and identifying what distinguishes them from those who are not susceptible. Identification of subtypes could be useful in gaining a better understanding of the variety of possible symptom profiles and their etiology, course, and response to treatment (Nock, Kazdin, Hiripi, & Kessler, 2006).

Latent class analysis (LCA) uses a categorical latent variable to classify individuals who are similar into groups (Muthén & Muthén, 2000). The goal of LCA is to determine the smallest number of latent classes that describe the pattern of responses among a set of observed categorical variables. Parameters for the model include the probabilities of fulfilling each criterion given class membership and the estimates of class probabilities for each individual. In the current study, LCA was used to determine what the class structure looks like for antisocial behavior. Separate analyses were conducted exploring age 14 CD symptoms and age 22 symptoms of AAB. LCA was also conducted with a model combining all symptoms from age 14 CD and age 22 AAB. Correlations for posterior probabilities of class membership for MZ and DZ twin pairs were calculated. These correlations were used to estimate the heritability of class membership.

Results

Descriptive Statistics

At age 14, 14.7% of males and 8.4% of females met diagnostic criteria for conduct disorder. The mean number of symptoms was 1.10 for males (SD = 1.49) and .68 (SD = 1.14) for females. As adults, 17.5% of males (M = 1.33, SD = 1.33) and 6.2% of females (M = 0.73, SD = 1.01) met criteria for adult antisocial behavior at age 22. For alcohol dependence symptoms at age 22, 18.4% of males and 13.3% of females met diagnostic criteria. The mean number of symptoms endorsed was 1.38 (SD = 1.49) for males and 0.99 (SD = 1.38) for females.

Twin correlations (e.g., cross-twin, within-trait and cross-twin, cross-trait) were calculated to provide preliminary information on sources of variance among the measures. Cross-twin within-trait polychoric correlations were computed using symptom counts for each disorder. Genetic influence on CD, AAB, and AD is suggested by MZ correlations that exceed

DZ correlations in both males and females (see Table 4). For conduct disorder, the DZ correlations are more than half of the MZ correlations indicating that environmental factors also influence the development of this behavior. This is also true for alcohol dependence in females.

Table 4

Cross-Twin Within-Trait Correlations for CD Symptoms, AAB Symptoms, and AD Symptoms

	CD Symptoms	AAB Symptoms	AD Symptoms
FMZ	0.82*	0.47*	0.52*
FDZ	0.55*	-0.12	0.33*
MMZ	0.67*	0.49*	0.62*
MDZ	0.46*	0.12	0.22*

Note. * indicates $p < 0.01$

Cross-twin cross-trait correlations indicate the influence of genetic factors on the stability of antisocial behavior from adolescence to adulthood and on the relationship between antisocial behavior and alcohol dependence (see Table 5).

Table 5

Cross-Twin Cross-Trait Correlations for CD Symptoms, AAB Symptoms, and AD Symptoms

	CD-AAB	CD-AD	AAB-AD
FMZ	0.40	0.37*	0.34*
FDZ	0.08	0.17	0.02
MMZ	0.36	0.29	0.25*
MDZ	0.06	0.10	0.04

Note. * indicates $p < 0.01$

Factor Analyses

Structure of Conduct Disorder. Factor analyses were used to determine if nonaggressive and aggressive subtypes of CD were present in the current population. Initially, a twin from each pair was selected at random for exploratory phenotypic factor analysis. The data was then divided by gender and frequencies for each symptom were calculated.

Table 6

Prevalence Rates of CD Symptoms by Gender

Symptom	Male (%)	Female (%)
Stolen without confrontation	**26.3**	**22.1**
Run away from home overnight	0.2	1.0
Often lies	**7.7**	**6.6**
Deliberately engaged in firesetting	**12.7**	3.6
Often truant from school	**19.9**	**15.7**
Broken into someone's car, building or home	3.1	1.1
Deliberately destroyed others' property	**11.4**	**6.5**
Has been physically cruel to animals	**8.1**	1.2
Forced someone into sexual activity	0.1	0
Has used a weapon in more than 1 fight	**7.2**	**6.5**
Often initiates fights	**9.3**	2.2
Stolen with confrontation	0.4	0.2
Physically cruel to people	4.2	0.9

Note. Percentages highlighted in bold indicate symptoms with rates above the 5% cutoff.

Prevalence rates for the symptoms are listed in Table 6. Items with a prevalence of less than 5% in the sample were eliminated. For males, eight symptoms had prevalence rates high

enough to be included in the factor analyses. For females, only five symptoms had prevalence rates higher than 5%. As a result of the low prevalence rates, factor analyses were not conducted with females. The results using only 5 of 13 symptoms would not be representative of the structure of conduct disorder in females and it is possible the results would yield one or two-item factors which are not meaningful.

Exploratory factor analyses were conducted with the males by splitting the sample into their respective halves. Results of the analyses revealed a two factor solution in both halves, however, symptoms loaded differently on the two factors (see Table 7). The discrepancies in loading could be the result of the low prevalence of symptoms or the result of employing factor analysis with dichotomous data. Confirmatory factor analyses could not be conducted due to the disagreement in loadings.

Table 7

Factor Loadings for Conduct Disorder for Males

	First Half		Second Half	
	Factor 1	Factor 2	Factor 1	Factor 2
Stolen without confrontation	.62	.17	.18	.49
Often lies	.13	.02	.02	.25
Deliberately engaged in firesetting	.25	.28	.07	.60
Often truant from school	.57	.05	1.75	.23
Deliberately destroyed others' property	.60	.32	.19	.51
Has been physically cruel to animals	.35	.25	.05	.43
Has used a weapon in more than 1 fight	.14	.52	.04	.44
Often initiates fights	.23	.12	.14	.33

Structure of Adult Antisocial Behavior. To examine the structure of adult antisocial behavior, the factor analyses previously described were conducted again, but using APD symptoms as reported on the SSAGA at age 22. Similar to the age 14 symptoms of CD, prevalence rates on several items were below 5%. Males had six items with greater than 5% prevalence and females had five (see Table 8). Factor analyses could not be conducted with the age 22 data for either gender because resulting factors would not have enough items to be meaningfully interpreted.

The initial data analysis plan proposed using factor scores produced from factor analyses in conducting the remaining analyses, but due to the low prevalence rates and inability to properly run the factor analyses, the remaining analyses were conducted using symptom counts for each disorder. The decision to use symptom counts was made based on previous research. The use of symptom counts in biometrical analyses provides more power than the use of dichotomous data, such as diagnoses (Malone, Taylor, Marmorstein, McGue, & Iacono, 2004).

Table 8

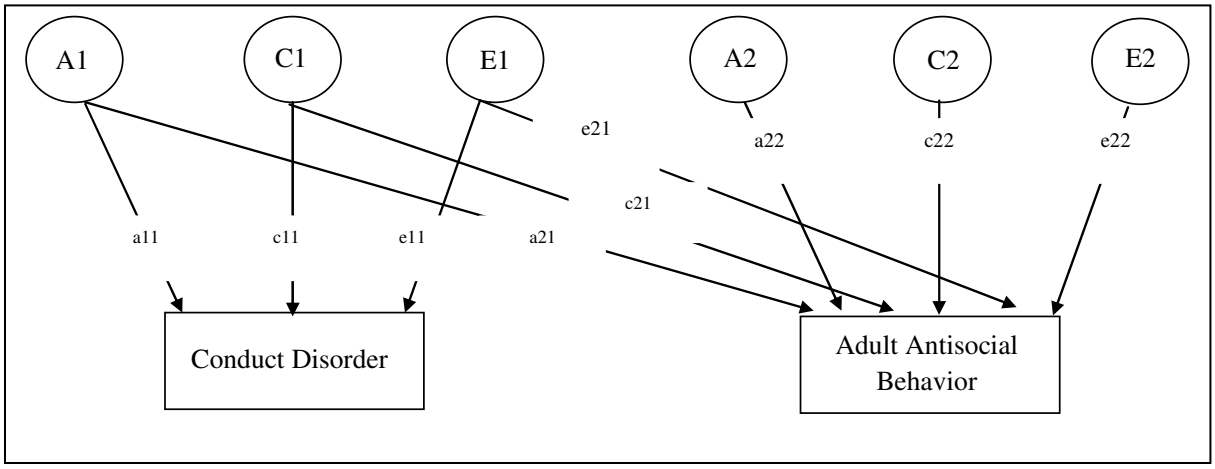
Prevalence Rates of APD Symptoms by Gender

Symptom	Male (%)	Female (%)
Fails to conform to social norms with respect to lawful behaviors	**14.6**	**7.4**
Deceitfulness, as indicated by repeated lying	**20.0**	**15.2**
Fails to plan ahead or is impulsive	3.0	2.6
Is irritable and aggressive as indicated by repeated physical fights or assaults	**44.0**	**28.5**
Is reckless regarding own safety or the safety of others	**30.4**	**7.2**
Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations	**10.7**	**7.0**
Lacks remorse	**9.9**	4.7

Note. Percentages highlighted in bold indicate symptoms with rates above the 5% cutoff.

Bivariate Analyses

Bivariate analyses were used to explore the genetic and environmental influences from adolescence into adulthood, and to explore the relationship between antisocial behavior and alcohol dependence. Figure 1 provides a path diagram with pathways labeled to correspond with the tables presenting the results of model fitting. The order in which pathways were dropped was determined based on the results of the full model. Pathways with the lowest estimated magnitude were dropped first in model fitting. The selection of additional pathways to remove from the models was based on the results of each model fit and will be described in more detail for each set of analyses.



Conduct Disorder and Antisocial Behavior. The genetic structure of the relationship between CD and AAB was examined using twin modeling. All models were run separately by gender. The model fits are shown in Table 9 and Figure 2 provides path estimates from the full model. For females, the first model tested was the full ACE model. The full model provided low estimates for shared environmental influences. Model #2 tested whether all shared environmental influences on AAB could be dropped and the results did not indicate a significant decrease in fit. Shared environmental influences on AAB were not included in subsequent model fitting. The next pathway removed was unique environmental influences common to the

two disorders. This decision was also based on pathway estimates from the full model. Dropping unique environmental influences that were common between the two disorders (#3) did not cause a significant decrease in fit. The unique environmental pathway between disorders was not included in the remaining models.

The next models explored genetic influences on AAB and the extent to which they overlapped with CD. These models were compared to a model without shared environmental influences on AAB and no common unique environmental pathway. First, all genetic influences on AAB (#4) were dropped resulting in a significant decrease in fit. This provided evidence of genetic influences on AAB, but it was unclear if they were shared with CD or specific to AAB. The genetic pathway specific to AAB was removed from the model (#5) and this did not cause a significant decrease in fit compared to model #3, but dropping shared genetic influences did cause a significant decrease in fit (#6). This indicates the genetic influences on AAB at age 22 overlap with those on CD at age 14. The final model tested was compared to a model with no shared unique environmental pathway, no common shared environmental influences, and no AAB specific genetic or shared environmental influences. Removing common environmental influences specific to conduct disorder (#7) did not cause a significant decrease in fit.

Results for males were similar to those of females and the order in which pathways were dropped was based on the same rationale. The initial model was the full ACE model, which like females, provided low estimates for shared environmental influences on AAB and between

Table 9

Fit Statistics from Bivariate Models of Conduct Disorder (CD) and Adult Antisocial Behavior (AAB)

		Females			
		Model Compared to	ΔX^2	Δdf	*P*
1	Full Model	N.A.	(2304.72)	(1088)	N.A.
2	Drop all C on AAB (c21, c22)	1	0.05	2	0.98
3	Drop shared E (e21)	2	0.51	1	0.48
4	Drop all A on AAB (a21, a22)	3	79.10	2	<.001
5	Drop AAB specific A (a22)	3	0.37	1	0.54
6	Drop shared A (a21)	3	58.24	1	<.001
7	Drop CD Specific C (c11)	5	2.30	1	0.13

		Males			
		Model Compared to	ΔX^2	Δdf	*p*
1	Full Model	N.A.	(2998.08)	(1076)	N.A.
2	Drop all C on AAB (c21, c22)	1	0.01	1	0.99
3	Drop shared E (e21)	2	0.12	1	0.73
4	Drop all A on AAB (a21, a22)	3	90.12	2	<.001
5	Drop AAB specific A (a22)	3	2.08	1	0.15
6	Drop shared A (a21)	3	29.37	1	<.001
7	Drop CD Specific C (c11)	5	2.18	1	0.14

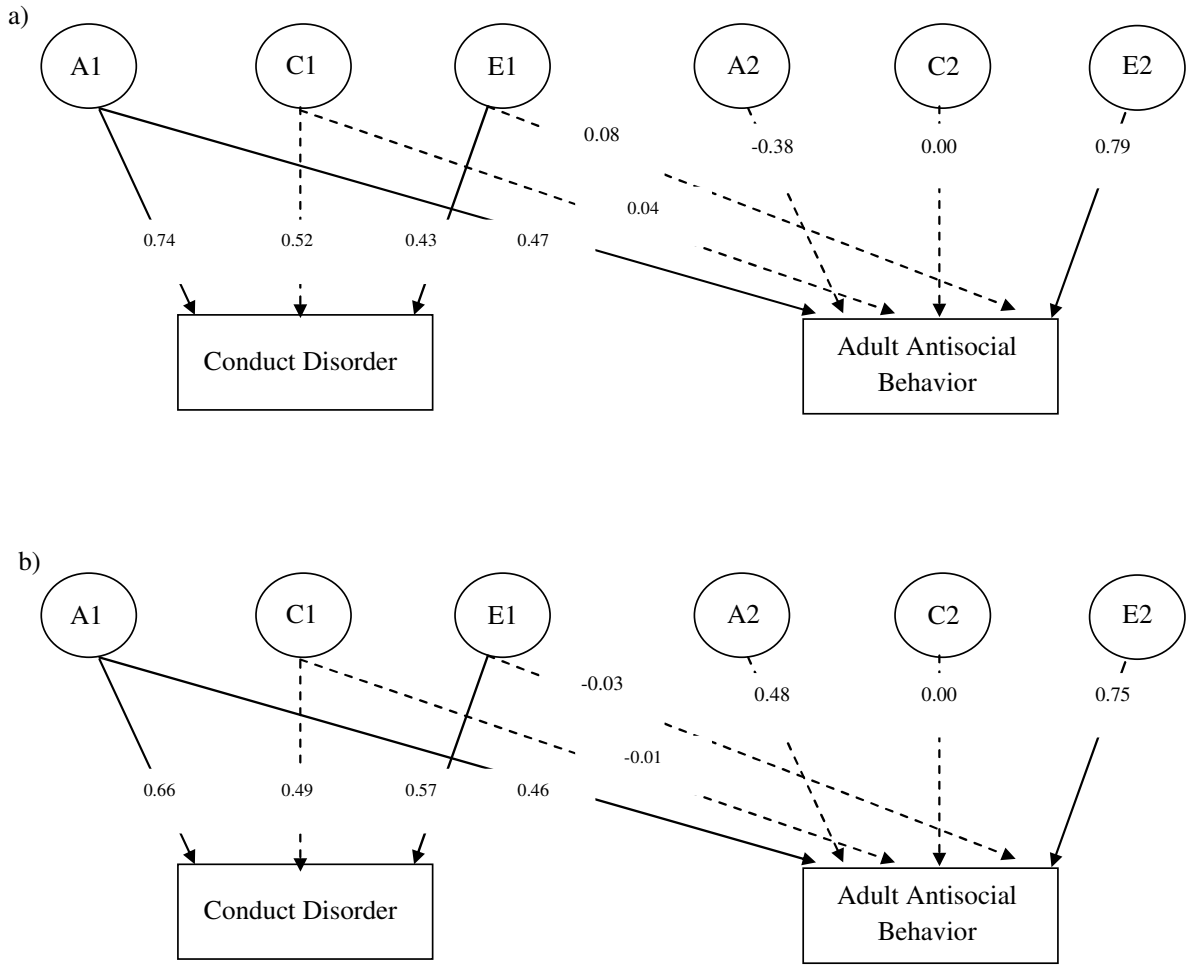


Figure 2. (a) Path estimates for additive genetic, common environmental, and unique environmental influences from the full model for females. (b) Path estimates for additive genetic, common environmental, and unique environmental influences from the full model for males. Dashed lines indicate pathways that could be dropped from the model without causing significant decrease in fit.

disorders. When all shared environmental influences on AAB were removed from the model (#2),there was not a significant decrease in fit. Likewise, when the shared unique environmental influence pathway was dropped (#3), model fit was not impacted. Subsequent models were compared to a model with no shared environmental influences on AAB and no common unique environmental pathway between disorders. Although removing all genetic influences on AAB

(#4) resulted in a decrease in fit, it was possible to remove genetic influences specific to AAB (#5) from the model. Dropping the genetic influences shared between disorders resulted in a decrease in fit (#6). This suggests overlap of genetic influences between age 14 CD and age 22 AAB. Therefore, like with females, the final model was compared to one with no AAB specific genetic influences, no shared environmental influences on AAB, and no unique environmental pathway shared between disorders. Shared environmental influences on CD (#7) could also be dropped without resulting in a significant decrease in fit.

Conduct Disorder and Alcohol Dependence. Bivariate modeling was used to determine the genetic structure of the relationship between conduct disorder and alcohol dependence, a commonly associated outcome in adulthood. All models were run separately by gender and the model fits are shown in Table 10. Figure 3 includes path estimates from the full model. For females, the first model tested was the full ACE model. Subsequent model fitting was based on the results of the full model. The unique environmental pathway common between disorders had low estimates in the full model and was dropped first. This did not cause a significant decrease in fit and this pathway was not included in the rest of the models. In the full model, estimates for the shared genetic pathway and the shared environmental pathway common between disorders were both low. Therefore, separate models explored whether these influences on AD could be removed from the model. Dropping all genetic influences (#3) on AD did not cause a significant decrease in fit and neither did dropping all shared environmental influences (#4) on AD. Although each of these could be dropped separately without resulting in a worse fitting model, it was not possible to drop all genetic and all shared environmental influences (#5). These results suggest that there may not be sufficient power in the sample to discriminate between A and C effects and neither path should be dropped.

The remaining models for females explored the structure between the two disorders. Model #6 assumes that there are no shared genetic influences between conduct disorder and alcohol dependence. This model did not cause a significant decrease in fit. The next model (#7) tested this same idea for common environmental influences, and it also did not result in a significant decrease in fit. However, when a model was fit that eliminated all shared genetic influences and common environmental influences, the model fit the data significantly worse. Similar to the analyses previously described, these results could be related to power in the sample. The remaining models tested whether the genetic and environmental factors of conduct disorder were shared with alcohol dependence. It was possible to drop genetic factors specific to alcohol dependence (#9), common environmental influences specific to alcohol dependence (#10), and both genetic and common environmental influences on alcohol dependence (#11) without causing a significant decrease in fit. These models suggest that the genetic influences and shared environmental factors overlap between disorders. Shared environmental influences on CD (#12) could also be dropped without a significant decrease in fit.

Like females, model fitting for males began by fitting the full ACE model. Based on estimates from the full model, all shared environmental influences on AD were dropped in the second model. This did not cause a significant decrease in fit. Previous studies have found minimal influence of shared environmental factors on alcohol dependence at this age, and accordingly, subsequent models assumed no shared environmental influences on alcohol dependence (Hicks et al., 2007). Shared unique environmental influences (#3) could also be dropped without decreasing the fit of the model. Model #4, which dropped all genetic influences on AD, resulted in a significant decrease in fit. Therefore, the individual genetic pathways

Table 10

Fit Statistics from Bivariate Models of Conduct Disorder (CD) and Alcohol Dependence (AD)

		Females			
		Model Compared to	ΔX^2	Δdf	*p*
1	Full Model	N.A.	(2402.22)	(1074)	N.A.
2	Drop shared E (e21)	1	1.33	1	0.25
3	Drop all A on AD (a21, a22)	2	2.45	2	0.29
4	Drop all C on AD (c21, c22)	2	1.18	2	0.56
5	Drop all A and C on AD (a21, a22, c21, c22)	2	105.04	4	<.001
6	Drop shared A (a21)	2	1.63	1	0.20
7	Drop shared C (c21)	2	0.85	1	0.36
8	Drop all shared A and C (a21, c21)	2	27.22	2	<.001
9	Drop AD specific A (a22)	2	0.70	1	0.40
10	Drop AD specific C (c22)	2	0.28	1	0.60
11	Drop AD specific A and C (a22, c22)	2	2.15	2	0.34
12	Drop CD specific C (a11)	11	0.75	1	0.39

		Males			
		Model Compared to	ΔX^2	Δdf	*p*
1	Full Model	N.A.	(3071.29)	(1071)	N.A.
2	Drop all C on AD (c21, c22)	1	0.10	1	0.95
3	Drop shared E (e21)	2	0.11	1	0.74
4	Drop all A on AD (a21, a22)	3	168.22	2	<.001
5	Drop shared A (a21)	3	19.25	1	<.001
6	Drop AD specific A (a22)	3	4.21	1	<.05
7	Drop CD Specific C (c11)	3	1.78	1	0.18

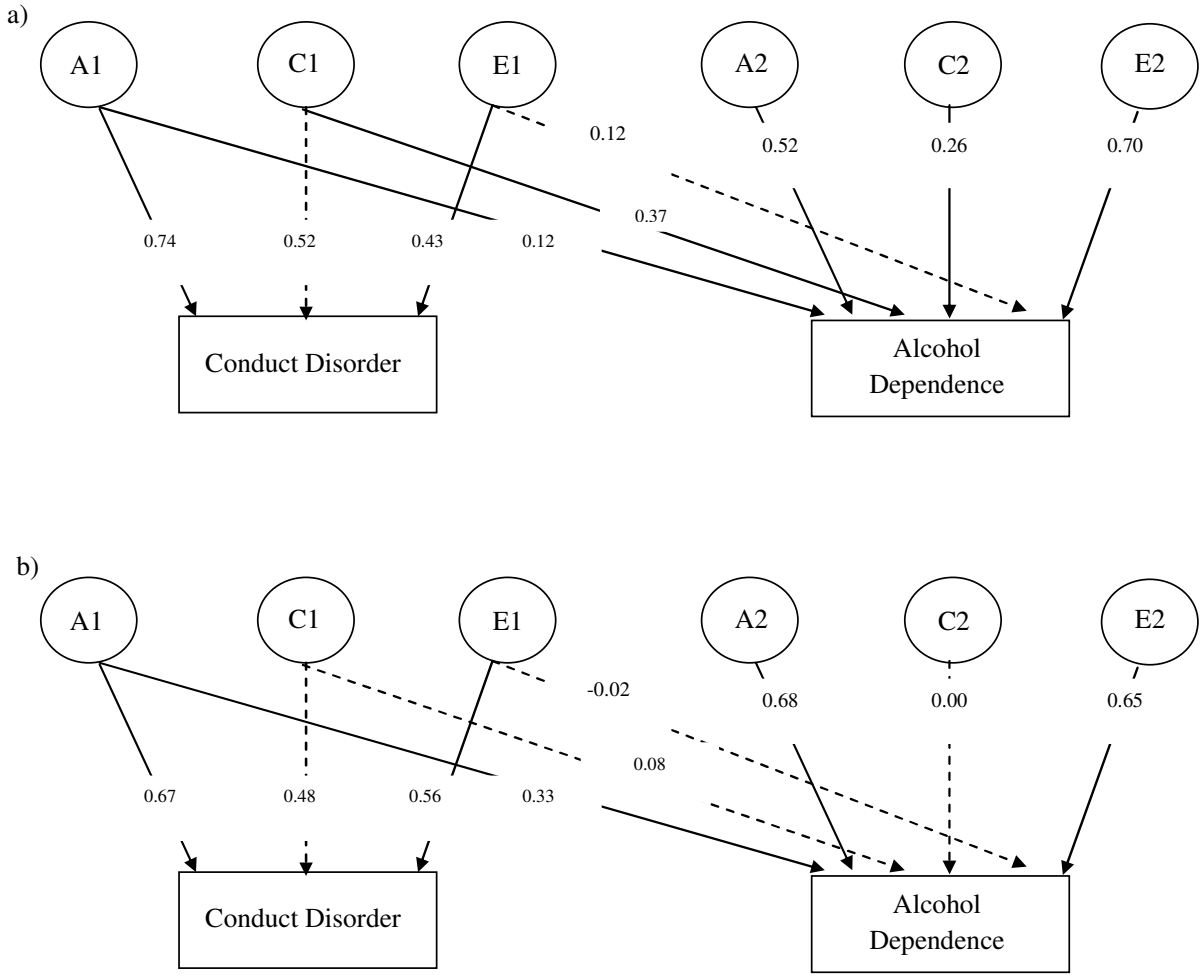


Figure 3. (a) Path estimates for additive genetic, common environmental, and unique environmental influences from the full model for females. (b) Path estimates for additive genetic, common environmental, and unique environmental influences from the full model for males. Dashed lines indicate pathways that could be dropped from the model without causing significant decrease in fit.

influencing AD were explored. The subsequent model (#5) dropped the genetic pathway between CD and AD, which tested whether genetic influences were distinct to each disorder. This resulted in a significant decrease in fit. Removing the AD specific genetic influences (#6) also resulted in a significant decrease in fit. These results suggest that although there is overlap between genetic influences for the disorders, each disorder also has genetic influences that are

unique. The final model tested whether shared environmental influences on CD (#7) could be dropped and this did not cause a significant decrease in fit.

Antisocial Behavior and Alcohol Dependence. The genetic structure of the relationship between AAB and AD in young adulthood was explored using bivariate analyses. Fit statistics from the full model are in Table 11 and Figure 4 has path estimates from the full model.

The order in which pathways were dropped was initially determined by the full model. Based on the full model, pathways with low magnitude were dropped first. In model #2, all shared environmental influences on AAB were removed and this did not cause a significant decrease in fit. Therefore, shared environmental influences on AAB were removed from the model and not included in subsequent model fitting. Model #3 dropped the common pathway for shared environmental influences, which did not cause a significant decrease in fit. . After determining that all genetic influences on AD could not be dropped (#4), the individual genetic paths were removed one at a time. The genetic influences shared between AAB and AD could not be dropped without a significant decrease in fit (#5), but genetic influences specific to AD could (#6). This indicates that the genetic factors influencing AAB and AD at age 22 overlap. Subsequent models were fitted without the genetic influences specific to AD. In the next model (#6), the common pathway for unique environmental influences was dropped and this resulted in a significant decrease in fit. For the final model (#7), the shared environmental influences on AD were removed and this resulted in a significant decrease in fit. Overall, it appears that there is overlap between genetic influences from AAB and AD and there are shared and specific unique environmental influences on the disorders.

Table 11

Fit Statistics from Bivariate Models of Adult Antisocial Behavior (AAB) and Alcohol Dependence (AD)

		Females			
		Model Compared to	ΔX^2	Δdf	*p*
1	Full Model	N.A.	(2233.70)	(956)	N.A.
2	Drop C on AAB (c11)	1	0.01	1	0.94
3	Drop shared C (c21)	2	0.00	1	1.00*
4	Drop all A on AD (a21, a22)	3	16.28	2	<.001
5	Drop shared A (a21)	3	16.12	1	<.001
6	Drop AD specific A (a22)	3	0.00	1	1.00*
7	Drop shared E (e21)	6	4.63	1	<.05
8	Drop AD specific C (c22)	6	6.10	1	<.05

		Males			
		Model Compared to	ΔX^2	Δdf	*P*
1	Full Model	N.A.	(2467.76)	(840)	N.A.
2	Drop all C (c11,c21, c22)	1	0.00	3	1.00*
3	Drop shared E (e21)	2	6.16	1	<.05
4	Drop all A on AD (a21, a22)	2	39.31	2	<.001
5	Drop shared A (a21)	2	8.23	1	<.001
6	Drop AD specific A (a22)	2	15.98	1	<.001

Note. *Change in AIC is lower than -4 due to difficulties optimizing model fit in the penultimate model.

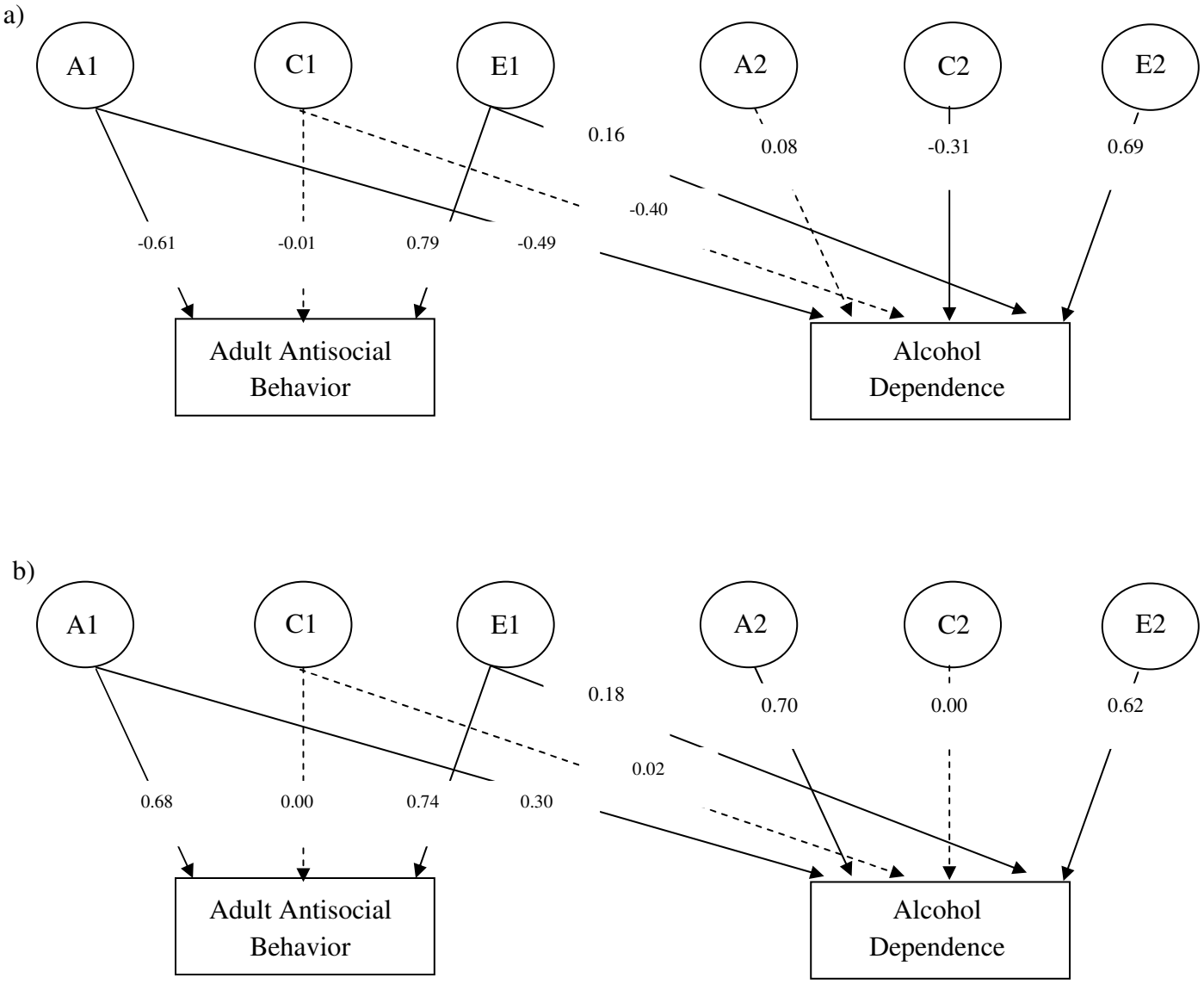


Figure 4. (a) Path estimates for additive genetic, common environmental, and unique environmental influences from the full model for females. (b) Path estimates for additive genetic, common environmental, and unique environmental influences from the full model for males. Dashed lines indicate pathways that could be dropped from the model without causing a significant decrease in fit.

The full model was run for males and the first pathways removed from the model were those with low magnitude. In the next model (#2) all shared environmental pathways were dropped. Results indicated that shared environmental influences could be dropped without a significant decrease in fit. The remaining models did not include any shared environmental

influences. Model #3, which removed shared unique environmental influences, resulted in a decrease in fit. All genetic influences on AD were dropped (#4), but this decreased the fit of the model. Models #5 and #6 explored whether the shared genetic pathway and AD specific pathway could be removed, but both caused a significant decrease in fit. These results indicate that for males there is overlap between the genetic and unique environmental influences for these two disorders, and also genetic and environmental influences unique to each disorder.

Model Statistics. The standardized estimates of variance attributed to genetic and environmental influences, along with 95% confidence intervals are presented in Table 12 for the full ACE models. For several of the estimates, the confidence intervals are large, which suggests insufficient power to discriminate between A and C effects.

Genetic influences are the largest contributor to CD in males and females, but unique environmental influences are the biggest contributor to AAB. The estimate of genetic influence for males remained the same at both timepoints, while it decreased for females. A decrease in shared environmental influences across time is also seen in both genders. In contrast, based on these estimates, the etiology of alcohol dependence differs by gender. For males, genetic and unique environmental influences account for the greatest amount of variance, with the estimate of shared environmental influences at almost zero. For females, unique environmental influences are the greatest contributor and the remainder of the variance is split between genetic and shared environmental influences.

Table 12

Standardized Estimates of Additive Genetic Influences (a^2), Common Environmental Influences (c^2), and Unique Environmental Influences (e^2) from full models

	Male			Female		
	A	C	E	A	C	E
Conduct Disorder	0.44 (0.11-0.76)	0.24 (0.00-0.52)	0.32 (0.23-0.43)	0.55 (0.23-0.86)	0.27 (0.00-0.56)	0.18 (0.12-0.27)
Adult Antisocial Behavior	0.44 (0.06-0.59)	0.00 (0.00-0.31)	0.56 (0.41-0.74)	0.37 (0.12-0.54)	0.00 (0.00-0.17)	0.63 (0.45-0.83)
Alcohol Dependence	0.58 (0.18-0.70)	0.01 (0.00-0.34)	0.42 (0.30-0.57)	0.29 (0.00-0.63)	0.21 (0.00-0.54)	0.50 (0.35-0.69)

Note. The estimates provided for Conduct Disorder and Adult Antisocial Behavior are from the full model for CD-AAB. These estimates were consistent with those in the full model for CD-AD and AAB-AD. The estimates for Alcohol Dependence are from the full model for CD-AD, but these estimates are consistent with those in the full model for AAB-AD.

Table 13 provides additive genetic, common environmental, and unique environmental correlations between disorders as provided by the full model. These correlations indicate how much the same genetic, shared environmental, and unique environmental factors contribute to the different disorders (Dick et al., 2005). For CD-AAB, genetic influences were highly correlated between disorders. The correlation for shared environmental influences was not meaningful in both males and females. Genetic influences accounted for the greatest proportion of the covariance between the disorders.

The correlations for CD-AD suggest differences in the genetic and environmental factors contributing to the relationship between the disorders. For males, genetic influences were correlated, but the correlations for shared and unique environmental influences were not meaningful. In females, it was shared environmental influences that were most highly correlated. Although genetic influences contributed to the greatest proportion of covariance for

males, shared environmental influences accounted for more than half of the covariance in females.

With AAB-AD, the pattern for males and females was again similar. The additive genetic correlation in females was much higher than that in males. However, the proportion of covariance accounted for by genetic influences was comparable in both genders. Covariance was primarily attributed to genetic influences, followed by unique environmental influences in both males and females.

Table 13

Additive Genetic Correlations, Common Environmental Correlations, and Unique Environmental Correlations between the disorders from full model

	Male			Female		
	rA	rC	rE	rA	rC	rE
CD-AAB	0.69	N/A	N/A	0.77	N/A	N/A
CD-AD	0.43	N/A	N/A	0.23	0.82	0.17
AAB-AD	0.40	N/A	0.28	0.98	N/A	0.23

Note. N/A indicates minimally significant or non-significant pathways in the full model.

Person-Centered Approach

LCA was conducted with age 14 CD symptoms, age 22 AAB symptoms, and a combined model. For each set of analyses, LCA models were fit for one through four classes using the 13 CD symptoms and 7 AAB symptoms for all twins (e.g., MZ, DZ, and OSDZ). A uniform approach for determining the number of classes does not exist, so several statistics, together with theoretical framework, were utilized to assess model fit (Nylund, Asparaouhov, & Muthén, 2007). The Lo-Mendel-Rubin likelihood ratio test (LMR-LRT) compares $k - 1$ and the k class

model and provides a *p* value indicating if the additional class significantly improves model fit (Lo, Mendell, & Rubin, 2001; Vuong, 1989). The LMR-LRT was first used as an indicator of fit improvement. Where the LMR-MRT indicated improved fit, the Bayesian Information Criteria (BIC; Schwarz, 1978) and entropy were also reviewed. The BIC, like the LMR-LRT, has been found to be a good indicator of class enumeration, where improvement in fit is designated by a decrease in value (Kass & Raftery, 1995; Keribin, 1997). Entropy coefficients designate how well a model predicts profile membership on a scale ranging from 0 to 1.

Table 14 provides the fit statistics for CD, AAB, and the combined model, with the best model indicated in bold. For age 14 CD, the LMR-LRT supported an increase from one to two classes ($p < .001$) and two to three classes ($p = .036$), but not from three to four classes ($p = .414$). Although the BIC increased slightly from two to three classes, the entropy coefficient remained relatively stable. Just over two-thirds (68.3%, $N = 503$) of the adolescents were members of the low risk group (CD1). These youth had a low likelihood for antisocial behaviors at age 14. Slightly more than a quarter of the adolescents (27.2%, $N = 503$) were probabilistically assigned to the moderate risk class (CD2). This class was at risk for less aggressive antisocial behaviors, like stealing without confrontation and truancy, but overall there was a lower likelihood for engagement in multiple behaviors than CD3. CD3, the adolescent class, was comprised of 4.5% ($N = 84$) of the sample. These youth were highly likely to engage in multiple antisocial behaviors, including those classified in other research as aggressive and nonaggressive (see Table 15).

For AAB, *p* values from the likelihood ratio test supported the increase from one to two classes ($p < .001$), but not the increase from two to three classes ($p = .224$). The BIC continued to increase with each class addition, while entropy remained fairly consistent. The majority

Table 14

Model Selection Criterion for LCA on Conduct Disorder and Adult Antisocial Behavior

	Number of Classes		
	2	*3*	*4*
Conduct Disorder			
BIC	9749.38	**9755.85**	9826.64
Entropy	.746	**.698**	.757
Likelihood Ratio *p* (*k* vs. *k*-1)	.000	**.036**	.414
Adult Antisocial Behavior			
BIC	**6209.13**	6245.46	6289.06
Entropy	**.66**	.77	.71
Likelihood Ratio *p* (*k* vs. *k*-1)	**.000**	.224	.109
Combined			
BIC	**13078.01**	13086.69	13127.89
Entropy	**.70**	.73	.73
Likelihood Ratio *p* (*k* vs. *k*-1)	**.000**	.079	.166

(80.3%, *N*= 1076) of age 22 participants were probabilistically assigned to AAB1, the low risk class. The high risk class, AAB2, was comprised of 19.7% (*N* = 264) of the sample and was marked by higher likelihood for engagement in multiple antisocial behaviors, especially consistent irresponsibility and failure to conform to social norms.

An LCA was conducted where all symptoms of age 14 CD and age 22 AAB were included in the model. Results were similar to those of the AAB. The increase from one to two classes was supported ($p < .001$), but the increase to two classes resulted in a non-significant *p*-value ($p = .079$). The BIC increased for each subsequent class, but entropy was roughly the same. Table 17 shows the break down for the low risk and high risk classes. For the low risk class (Comb1, 70.8%, *N* = 945), there was very little risk for childhood conduct behaviors, but as

adults these individuals failed to conform to social norms. The high risk class (Comb2) included 29.2% (N = 389) of the sample. These individuals were at higher risk of engaging in multiple antisocial behaviors in both adolescence and adulthood, such as being frequently truant from school, deceitfulness, and irritability and aggression.

Table 15

CD Symptom Endorsement Rates by Class

	CD1- Low Risk	*CD2- Moderate Risk*	*CD3- High Risk*
Stolen without confrontation	.072	.575	.845
Has run away from home overnight at least twice while living in parental or parental surrogate home	.000	.014	.041
Often lies	.020	.183	.190
Deliberately engages in fire setting	.023	.162	.501
Often Truant	.041	.456	.568
Broken into someone else's house, building, car	.003	.020	.291
Destruction of property	.012	.178	.738
Physically Cruel to Animals	.021	.051	.420
Has forced someone into sexual activity with him/her	.000	.002	.000
Used weapon in more than 1 fight	.020	.100	.445
Often initiates physical fights	.000	.100	.393
Stolen with confrontation	.000	.011	.000
Physically Cruel to People	.004	.042	.257

Table 16

AAB Symptom Endorsement Rates by Class

	AAB1- Low Risk	*AAB2- High Risk*
Fails to conform to social norms with respect to lawful behaviors	.324	.833
Deceitfulness, as indicated by repeated lying	.090	.502
Fails to plan ahead or is impulsive	.017	.074
Is irritable and aggressive as indicated by repeated physical fights or assaults	.257	.745
Is reckless regarding own safety or the safety of others	.114	.563
Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations	.067	.172
Lacks remorse	.001	.333

Table 17

Combined Symptom Endorsement Rates by Class

	Comb1- Low Risk	*Comb2- High Risk*
CD 1- Stolen without confrontation	.093	.552
CD 2- Has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning)	.000	.016
CD 3- Often lies	.023	.186
CD 4- Deliberately engages in fire setting	.025	.197
CD 5- Often Truant	.058	.406
CD 6- Broken into someone else's house, building, car	.002	.055
CD 7- Destruction of property	.014	.237
CD 8- Physically Cruel to Animals	.022	.105
CD 9- Has forced someone into sexual activity with him/her	.000	.002
CD 10- Used weapon in more than 1 fight	.023	.161
CD 11- Often Initiates physical fights	.014	.158
CD 12- Stolen with confrontation	.000	.010
CD 13- Physically Cruel to People	.001	.072
AAB1 Fails to conform to social norms with respect to lawful behaviors	.367	.646
AAB2 Deceitfulness, as indicated by repeated lying	.093	.371
AAB3 Fails to plan ahead or is impulsive	.012	.063
AAB4 Is irritable and aggressive as indicated by repeated physical fights or assaults	.247	.611
AAB5 Is reckless regarding own safety or the safety of others	.108	.451
AAB6 Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations	.061	.145
AAB7 Lacks remorse	.011	.207

For each model, intraclass correlations for probability of membership indicate that there is a genetic basis for class assignment. For each class, the MZ correlation is greater than the DZ correlation. Table 18 provides heritability estimates and twin correlations between classes. Heritability estimates were calculated by subtracting the DZ correlation from the MZ correlation and multiplying the result by two (Plomin et al., 2008). The LCA was run with both males and females in the analyses after determining that class structure did not differ when run separately by gender. MZ and DZ correlations are presented for the overall sample.

Table 18

Correlations between MZ and DZ Twins and Heritabilities for Latent Class Assignments

	MZ Correlation	DZ Correlation	h^2
Conduct Disorder			
Low Risk	0.62	0.31	0.63
Moderate Risk	0.56	0.24	0.64
High Risk	0.52	0.21	0.62
Antisocial Behavior	0.35	0.00	0.68
Combined	0.53	0.17	0.72

Note. Correlations for AAB and Combined are the same for both classes so only one correlation and heritability estimate are presented.

Discussion

This study used data from a longitudinal Finnish twin study to examine the genetic and environmental influences of antisocial behavior from adolescence to young adulthood. It also examined associations between antisocial behavior and alcohol dependence. Specifically, the current study explored: (1) whether there was evidence for nonaggressive and aggressive domains within the study population; (2) influences on the stability of antisocial behavior from age 14 to age 22; (3) the extent to which it is the same genetic and environmental influences impacting antisocial behavior at age 14 and age 22; (4) the magnitude of genetic and environmental influences on the relationship between childhood conduct disorder and alcohol dependence in young adulthood; (5) the magnitude of the genetic and environmental influences on the relationship between adult antisocial behavior and alcohol dependence in young adulthood; (6) whether the genetic and environmental influences on these relationships differ by gender.

Our primary findings were as follows: (1) We did not find evidence supporting the two-factor structure of conduct disorder in the study sample. (2) The stability of antisocial behavior from adolescence to adulthood was primarily attributed to genetic influences. (3) The genetic and environmental factors influencing adolescent CD largely overlapped with those influencing AAB at age 22 in both males and females. (4) In males, there were genetic influences shared between CD and AD, but also age specific genetic influences. Results of model fitting for females suggested both shared genetic influences and shared environmental influences on the relationship between the disorders. (5) Genetic influences largely overlap between AAB and AD for females, but in males there were both shared genetic risk and unique genetic influences on each disorder.

(6) Based on results of twin modeling, the structure of genetic and environmental influences of CD-AAB was similar in males and females, but it differed when looking at CD-AD and AAB-AD.

Results of the LCA found a three-class structure for age 14 CD, a two-class structure for age 22 AAB, and a two-class structure for the combined symptoms. These classes could all be characterized by level of risk for the behaviors (e.g. low and high, or low, moderate, and high), rather than by distinct patterns of symptom endorsement. Heritability estimates for the classes ranged from .62 to .72, suggesting class membership was genetically influenced.

Prevalence Rates

The prevalence rates for conduct disorder (14.7% for males, 8.4% for females) are similar to rates found in other studies. For example, previous estimates ranged from 4.3-16% for males and 2-9% for females (Jacobson, Prescott, & Kendler, 2002; Tackett et al., 2003; APA, 2004; Gelhorn et al., 2004; Compton, Conway, Stinson, Colliver, & Grant, 2005; Button et al., 2007; Gelhorn et al., 2007; Meier, Slutske, Heath, and Martin, 2011). The rates of adult antisocial behavior (17.5% of males and 6.2% of females) were also comparable to previous studies. Studies looking at AAB found rates ranging from 8.3-16.5% for males and 2.4-8.5% for females (Jacobson et al., 2002; APA, 2004; Compton et al., 2005; Meier et al., 2011). While the rate for alcohol dependence in males (18.4%) is consistent with previous studies (13-23.9%), the rate for females (13.3%) was higher than the lifetime prevalence in those same studies (5.5-8.2%; Kendler, Prescott, Myers, & Neale, 2003; Grant et al., 2004). The studies by Kendler et al. (2003) and Grant et al. (2004) are both United States based population studies, and the current study sample comes from the Finnish population. It is possible the slight elevation in female prevalence is a product of culture, since both male and female prevalence rates are consistent

with findings from a Finnish population-based study (16% males, 13% females; Aalto, Seppa, Kiianmaa, & Sillanaukee, 1999).

Factor Analyses

Within the current sample, the non-aggressive and aggressive domains of conduct disorder were not seen, possibly due to low prevalence of symptoms. Although a two-factor structure emerged for males, the factor loadings differed in the split-halves of the sample. Factor analysis could not be conducted with females due to low prevalence rates of symptoms. The number of symptoms with greater than 5% endorsement was not enough to create meaningful factors. Overall, the current results do not fit with results of previous research using factor analysis to explore the structure of conduct disorder.

Previous studies have found conduct disorder is not a unitary construct and confirmed the presence of aggressive and non-aggressive domains (Fergusson et al., 1994; Tackett et al., 2003). While some of these studies utilized clinical populations, these domains have also been confirmed in population-based studies. The current study followed the methodological approach of Tackett et al. (2003). Specifically, in the current study, each symptom was rated as either present or not present and these values were used for the exploratory factor analysis. However, there are several differences with the Tackett et al. (2003) study that could influence the outcome of the factor analysis. First, the current study utilized data from 945 males and this sample was divided in half to create the split-halves, while Tackett et al. (2003) analyzed a sample of 1,669 males, and there is no indication that a split-half approach was taken. It has been found that larger sample sizes tend to produce outcomes that are more accurate (Costello & Osborne, 2005). A second difference is that in the current study, symptoms with less than 5% prevalence were eliminated leaving only five symptoms for females and seven symptoms for males. Tackett et al.

(2003) eliminated two of the thirteen symptoms due to 0% prevalence. The subject to item ratio can impact the results of factor analysis (Costello & Osborne, 2005). The decision to use the more conservative cutoff in the current study was made based on a subsequent paper by Tackett et al. (2005). In that study, Tackett et al. (2005) used a sample of male twins to confirm the two-factor structure of conduct disorder and explore the genetic structure of the factors. Items with a prevalence rate less than 5% were not included in analyses (Tackett et al., 2005). It is important to note that similar to the current study, Tackett et al. (2003, 2005) had to eliminate females from their analyses due to low prevalence rates of symptoms.

Another study that yielded differing results used confirmatory, rather than exploratory, factor analysis (Fergusson et al., 1994). An index score was created that represented the extent to which a participant engaged in overt and covert antisocial behaviors after determining that the prevalence rates of symptoms were too low to produce valid results in a factor analysis. Results suggested that conduct disorder was not a unitary construct, and that overt/aggressive and covert/nonaggressive domains exist within the disorder. The methodology of this study was different than that of the current study, which may be the reason for the differing outcomes.

Although research does support the presence of these two domains, there has been studies where that was not the case. Gelhorn and colleagues (2008) used IRT to look at symptom criteria in a community sample of adolescents, assess range of severity indicated by the current symptoms, and to test for sex differences in the criteria. Initial analyses included an exploratory factor analysis and the outcome supported the current study in that researchers did not find conduct disorder to be a two-factor construct (Gelhorn et al., 2008). A single factor was the best representation of the data.

With the advent of DSM-V, there is debate over whether a categorical approach is as useful as a continuum approach to CD (Moffitt et al., 2007). A recent study comparing categorical and dimensional diagnostic approaches with CD found increasing risks of negative outcomes with increasing symptomology (Fergusson, Boden, & Horwood, 2010). Additionally, the dimensional approach had better predictive validity of outcomes (Fergusson et al., 2010). While the prevalence rates and dichotomous data in the current study are likely the reason factor analyses did not reveal a two-factor model, results of new research is suggesting the non-aggressive and aggressive classification may not be the subtypes most representative of conduct disorder.

Although an attempt was also made to explore the factor structure of adult antisocial behavior, the prevalence rates of symptoms were too low to complete these analyses. Previous research was not found that explored whether adult antisocial behavior had a factor structure similar to conduct disorder.

Conduct Disorder and Adult Antisocial Behavior

Results of the current study provide support for the significant influence of genetic factors on the development and persistence of antisocial behavior during the period from adolescence to young adulthood. The magnitude of genetic and shared environmental influence on antisocial behavior decreased from age 14 to age 22 for females, while the influence of unique environmental factors increased. A decrease in shared environmental influences and increase in unique environmental influences was also seen for males, while heritability remained relatively stable. Overall, for both males and females, at age 14 genetic influences had the greatest impact on antisocial behavior, but by age 22, unique environmental influences, which

includes influences that make siblings different and error in the model, accounted for the greatest proportion of the variance in antisocial behavior.

Results of twin modeling indicated that for both males and females the genetic influences on conduct disorder largely overlapped with adult antisocial behavior at age 22. Genetic influences accounted for the largest proportion of covariance in the relationship between CD and AAB.

In the current study, the genetic influences on CD largely overlapped with those on AAB in both males and females. In contrast, there are two studies supporting age specific genetic influences on antisocial behavior (Jacobson et al., 2002; Silberg, Rutter, Tracy, Maes, & Eaves, 2007). Researchers had a difficult time explaining these unique genetic influences and these age specific effects have not been found in other studies. More frequently, researchers have found that genetic factors influencing AAB overlap with those factors that influence CD (Lyons et al., 1995; Meier et al., 2011). The changes in the magnitude of genetic influences are consistent with a study by Jacobson and colleagues (2002). In that study researchers saw relative stability of heritability from adolescence to adulthood for males. For females, a slight decrease in genetic influences from adolescence to adulthood was noted (Jacobson et al., 2002).

The role of the shared environment has also varied across studies. In the current study, results of model fitting suggested that shared environmental influences did not play a meaningful role in the relationship between CD and APD. Large confidence intervals suggest these results should be interpreted cautiously, because there was not enough power to discriminate between models. Shared environmental influences have been found to be more influential than genetics in adolescence, while other studies have found shared environmental influences impacted adult

antisocial behavior (Lyons et al., 1995; Goldstein, Prescott, & Kendler, 2001). Overall, studies and meta-analyses have concluded that shared environmental influences play a less significant role in antisocial behavior as the individual ages (Jacobson et al., 2002; Miles & Carey, 1997; Moffitt, 2005; Silberg et al., 2007; Tuvblad, Narusyte, Grann, Sarnecki, & Lichenstein, 2011). This decrease was seen in the current study.

In the current study, genetic influences explained the largest portion of variance in CD, but in AAB it was unique environmental influences that had the greatest impact for both males and females. Like with genetic and shared environmental influences, research outcomes on the magnitude of the influence of unique environmental influences vary. Some studies have found unique environmental influences account for the largest portion of variance in adolescent CD and AAB (Lyons et al., 1995; Goldstein et al., 2001). Tuvblad and colleagues (2011) found that unique environmental influences were most important in young adulthood for males, but not for females. A review of 52 twin and adoption studies concluded that unique environmental effects accounted for the most variance in adulthood and similar to the current study, noticed the magnitude of the effect increased with age (Rhee & Waldman, 1997). Therefore, the current study confirmed previous findings where the magnitude of shared environmental effects decrease and that of unique environmental effect increase with age.

It is possible the results of the current study also lend support to Moffitt's (1993) concept of life-course persistent antisocial behavior, because a single set of genetic factors influenced the stability of antisocial behavior over time. The current study did not specifically address this question but there have been several studies confirming the life-course persistent model by finding a single latent factor that explained the developmental trajectory of antisocial behavior (Silberg et al., 2007; Tuvblad et al., 2011). The etiology of that latent factor remains up for

debate due to conflicting findings in the amount of influence attributed to genetics and the environment.

Conduct Disorder and Alcohol Dependence

Results of the current study suggest that there are overlapping genetic influences between CD in adolescence and AD in young adulthood in males, and in females the relationship was influenced by shared genetic and environmental influences. The ACE estimates for AD suggest differences between males and females with regards to the source of variance in alcohol dependence. For females, estimates indicate that unique environmental influences account for the greatest amount of variance, while it is genetic influences for males. In the current study, results of the twin models for female suggested limited power in the model, making it difficult to discriminate between A and C effects. Overall, model fitting suggests that shared genetic and environmental influences account for the covariance between conduct disorder and alcohol dependence. Looking only at the full model, nonshared environmental influences accounted for the most variance in alcohol dependence in young adulthood. The remaining variance was divided between genetics and shared environmental influences.

For males, when looking at the relationship between conduct disorder and alcohol symptoms in young adulthood, the final model indicated that the relationship between disorders is solely attributed to genetic factors. The final model suggested that there are also specific genetic influences for conduct disorder and alcohol dependence, in addition to genetic factors common to both disorders. Environmental influences (shared or unique) did not contribute in a meaningful way to the liability between the disorders.

There is substantial literature on the relationship between antisocial behavior and alcohol dependence, but few studies look at etiology of the relationship over time. Twin studies have found common genetic influences, like those seen in the current study (Slutske et al., 1998). Using retrospective report of conduct disorder in males and females, Slutske et al. (1998) found that genetic influences accounted for the largest amount of variance in the relationship between CD and AD. Also similar to the present results is the finding that each disorder had unique genetic factors (Slutske et al., 1998).

A more recent study looking at antisocial behavior and alcohol dependence in males also found that it was genetic and unique environmental influences impacting the relationship over time, with shared environmental influences playing a nonsignificant role (Malone, Taylor, Marmorstein, McGue, & Iacano, 2004). Similar to the current study, Malone et al. (2004) found continuity of genetic effects across the ages, but also new genetic influences that emerge as the adolescent moves into adulthood. Some have also posited that results of this nature suggest conduct disorder is an earlier manifestation of genetic factors that contribute to alcohol dependence, therefore making conduct disorder a genetically influenced risk factor of alcohol dependence (Slutske et al., 1998; Rose et al., 2004).

Adult Antisocial Behavior and Alcohol Dependence

Results of twin modeling for males and females differed. For females, results of twin modeling indicate genetic factors account for the largest amount of covariance between AAB and AD and that in young adulthood, genetic factors largely overlap between the disorders. It was also determined that there are unique environmental influences shared between the disorders. Although shared environmental effects were not found to be influencing the

relationship between the disorders or AAB, their influence on AD could not be removed from the model without causing a significant decrease in fit.

In males, genetic influences accounted for the majority of covariation between the disorders. There were genetic influences shared between the disorders and disorder specific genetic influences. In the model for males, all shared environmental influences could be removed without causing a significant decrease in fit. However, it was determined that there were unique environmental influences contributing to the covariance between the disorders.

Researchers have found a relationship between AD and AAB, at times suggesting they may be variations of the same disorder (Robins, 1998). However, the etiology of this relationship has not been fully examined. In the current study, there was overlap of genetic influences for females and some overlap for males, with unique environmental influences also contributing to the relationship. A study utilizing within-twin and cross-twin correlations for alcoholism and antisocial behavior found the relationship between disorders in males was attributed to genetic influences, while the relationship in females suggested no shared genetic influences (Pickens, Svikis, McGue, & LaBuda, 1995). In an all male study, covariance between disorders was attributed to genetic influences and unique environmental influences, like in the current study (Malone et al., 2004). Additionally, although the researchers saw overlap between genetic effects, they also found disorder specific genetic influences (Malone et al., 2004).

Although not part of the current analyses, it is interesting to note the way the etiology of the relationship between AD and AAB changes from adolescence to young adulthood. The current study examined the relationship between AAB and CD at age 22, but a previous study

explored the relationship between age 14 CD and age 14 AD using the same sample. While in the current study genetic influences were the primary contributor to the relationship between the disorders, Rose et al. (2004) found that the covariation between CD and AD at age 14 could be attributed entirely to shared environmental effects. This suggests that as individual ages, genetic influences become more influential on this relationship and shared environment has less significance.

Similar to the idea that there is a single latent factor influencing antisocial behavior across the lifespan, several studies have looked at an externalizing factor linking the disorders together. Although not a question in the current study, the results do support the idea that these disorders that fall in the externalizing domain may be manifestations of a common factor based on the overlap between AAB and AD in females. This factor is characterized by a lack of impulse control and behavioral restraint (Krueger et al., 2002). Several studies exploring this factor have not only confirmed its presence but also the influence of genetic and environmental effects on the factor (Hicks et al., 2007; Kendler, Prescott, Myers, & Neale, 2003; Krueger et al., 2002; Malone et al., 2004). Like other studies in this area, outcomes related to differences in gender vary by study. Some researchers have found the etiology of the externalizing factor to be the same in males and females, while others have found an increase in heritability for males and increase in environmental effects for women (Hicks et al., 2007; Kendler et al., 2003).

There are a number of reasons results of the current study may vary from other findings. For one, the present study utilizes prospective report of conduct disorder, AAB, and AD symptoms. Only a handful of studies have taken the prospective approach. Retrospective reports can lead to bias and studies comparing retrospective and prospective report have found weak correlations between the two (Henry et al., 1994; Jacobson et al., 2002). Additionally, the

age of the samples and how it is classified differs from study to study. In the current sample adolescence was operationalized as age 14, while other studies used age 17 and classified it as adolescence (Krueger et al., 2002; Malone et al., 2004). Finally, the operationalization of antisocial behavior varies between studies. The current study utilized diagnostic criteria from various versions of the DSM, but some studies utilize the CBCL or other measures of externalizing behaviors (Tuvblad et al, 2011).

Latent Class Analysis

Latent class analysis identified classes for CD and AAB that were best described by level of risk for engaging in antisocial behavior. A three-class structure was identified for CD, and AAB and the combined model yielded a two-class solution. It was anticipated that the results of the LCA would mirror those of the factor analysis and classes would emerge that were similar to aggressive and non-aggressive factors. However, the results of the factor analyses prevented this comparison. The current study did explore the class structure for age 14 conduct disorder, age 22 adult antisocial behavior, and a model combining the CD and AAB symptoms.

For conduct disorder, each successive class appeared at higher risk for conduct disorder than the previous. When looking at the prevalence of CD in the classes, 0% of individuals met criteria for conduct disorder in the low risk class, 41.6% met criteria for CD in the moderate risk class, and 100% of individuals met criteria in the high risk class. Individuals engaging in the most behaviors were in the most severe, highest risk class. It is interesting that the highest probability behavior across all three classes is stolen without confrontation, a behavior typically classified as non-aggressive in studies exploring domains. Previous studies have found that this particular symptom is not a good predictor of severe antisocial behavior and that it is a child-like

antisocial behavior (Gelhorn et al., 2007; Lambert, Wahler, Andrade, & Bickman, 2001). The high prevalence rate may be reflective of the young age of the sample.

Results for AAB revealed a two-class structure, low risk and high risk. In these analyses, .5% of the low risk class met criteria for AAB, and 57.6% met criteria in the high risk class. The highest endorsement probability within both classes was with respect "fails to conform to social norms with respect to lawful behaviors." This symptom included questions about destruction of property, stealing with confrontation, stealing from family, unauthorized use of a credit or debit card, and breaking into a building or car. It is possible that at age 22 these are age normative behaviors that many individuals engage in, even if at a low risk for continued antisocial behavior. The analyses combining all of the symptoms revealed a two-class solution that paralleled that of AAB. In the low risk class, 0% met criteria for CD and 1.1% met for AAB. For those in the high risk class, 37.8% had a CD diagnosis and 36.8% had one for AAB.

The correlations and heritability estimates indicate that class membership was largely influenced by genetic factors. This is consistent with the twin analyses which reflected the substantial influence of genetics on CD and AAB. Other studies looking at LCA in CD or AAB did not provide this information for comparison.

The current study did not find subtypes within CD or AAB, rather the classes represented increasing risk for and severity of antisocial behavior. Previous studies have successfully identified subtypes of antisocial behavior with classes that expand beyond the aggressive and non-aggressive subtypes (Muthén & Muthén, 2000; Nock et al., 2006). Nock et al. (2006) found five-subtypes in an epidemiological sample of males and females. These subtypes were categorized as Rule Violations, Deceit/Theft, Aggressive, Severe Covert, and Pervasive CD.

A study by Bucholz, Hesselbrock, Heath, Kramer, and Schuckit (2000) did not find subtypes, but did identify a class structure they interpreted as reflecting severity of antisocial behavior. Running analyses separately by gender, researchers identified a 4-class solution for women and a 5-class solution for men where each class represented an increase in severity of antisocial behaviors. Bucholz et al. (2000) included both CD and ASPD symptoms in their LCA. Interestingly, they did find that one class for males supported the "late bloomer" taxonomy; an individual who had minimal symptoms in childhood and adolescence but were more severe in adulthood (Bucholz et al., 2000). This type of distinction could not be made in the current study because only two classes resulted from the combined LCA. It is important to note that prevalence rates of symptoms and overall sample size was higher in the Bucholz et al. (2000) study. These factors can influence outcomes in LCA.

Strengths and Limitations

Although the current study addressed some of the limitations of other studies by its inclusion of females, using prospective report of symptoms, and exploring etiology in a longitudinal sample, it is not without its own limitations.

The first limitation is the prevalence rates of the symptoms in the sample. The rates are actually on the upper end of ranges found in other studies, but even with this, the low endorsement of symptoms made conducting factor analysis, particularly with females, difficult (APA, 2004; Button et al., 2007; Jacobson et al., 2002; Kendler, Prescott, Myers, & Neale, 2003; Tackett et al., 2003; Compton, Conway, Stinson, Colliver, & Grant, 2005; Gelhorn et al., 2004; Grant et al., 2004; Gelhorn et al., 2007; Meier et al., 2011). The low prevalence rates prevented the current study from examining whether the structure of antisocial behavior differed in males and females. Research suggests that the developmental pathways to antisocial behavior are

different, and it would valuable to know if antisocial behavior manifests in the same way in males and females (Fontaine, Carbonneau, Vitaro, Barker, & Tremblay, 2009; Silverthorn & Frick, 1999). Issues with prevalence rates for females are not limited to the current study and females have been excluded from similar studies due to low rates.

A second limitation is the only source of data is self-report. The use of multiple informants can provide additional information about a behavior. However, incorporating data from multiple informants can be difficult because there is often variation between informants. Differences in etiology of a disorder have been found that are dependent upon reporter (Eley et al., 1997; Miles & Carey, 1997; Rhee & Waldman, 2002). Also, with regards to antisocial behavior, the individual's report could include covert behaviors that other informants do not know about (Dick et al., 2004). Due to the longitudinal nature of the current study, using multiple informants could mean having different raters at age 14 and age 22, because the informant with reliable information on the individual's behavior, may not be the same person at both timepoints. Trying to analyze data across informants can confound the association between assessments (Tuvblad, 2011).

A final limitation is the composition of the sample, including the racial distribution and the method in which the sample was ascertained. The sample is entirely Caucasian and results may not generalize to other races or ethnicities. Also, the sampling procedure was not entirely random as a small proportion (13%) of the twins were identified from urban areas from one particular birth cohort (for convenience) and 28% were identified for presumed higher risk for alcohol problems based on familial risk for alcoholism, which could limit the comparison of current study results to those from more representative epidemiological samples. With that in

mind, the prevalence rates of disorders within the sample are consistent with those found in more general population-based studies.

Despite these limitations, there are a number of strengths in the current study. First, the study uses prospective report of symptoms. Frequently with studies involving AAB, report of CD is based on retrospective information, and rates can be biased by an individual's ability to recall. Secondly, the study seeks to fill a gap in the literature by including females. Although this did cause some difficulties during analyses, results contribute to the slowly growing body of research related to females and antisocial behavior. Finally, the longitudinal nature of the study allowed for the exploration of how the structure of genetic and environmental influences changes from adolescence to early adulthood.

Future Directions

Historically, research on antisocial behavior has focused on males and studies that have attempted to incorporate females have faced similar problems as the current study. However, it is important to understand more about antisocial behavior in females. Although both males and females face negative outcomes as a result of adolescent antisocial behavior, antisocial adolescent girls become women who have up to a 40 times higher rate of criminal behavior than other women, face a high risk of early death, complex psychiatric problems, higher rates of substance abuse and poor physical health (Pajer, 1998). Due to the problems with prevalence rates, other researchers have suggested using samples where the rates of antisocial behavior should be higher, such as adjudicated or clinical populations (Tackett et al., 2005). While this would limit generalizability, it would provide a better understanding of antisocial behavior in the populations it is most severely impacting. A second approach is to incorporate measures that assess a variety of antisocial behaviors (Tackett et al., 2005). If antisocial behavior does in fact

present differently in males and females, using measures that address a wider range of antisocial behavior would ascertain information on how it looks in females. A better understanding of the manifestation and etiology of antisocial behavior in females could help inform prevention and treatment efforts.

Future research should continue to explore the relationship between these disorders over time and how the genetic and environmental structure changes as individuals age. In the current study, the primary focus was adolescence to young adulthood. Previous research has demonstrated that the magnitude of genetic and environmental influences shift during different developmental periods (Tuvblad et al, 2011). The age 22 timepoint in the current study is around the time that Moffitt (1993) anticipated those adolescent-limited individuals desist from antisocial behavior, which means that results could include both life-course persistent and adolescent individuals. Looking at additional timepoints would provide a better understanding of the genetic and environmental structure of these two taxonomies. Additionally, it would allow for exploration of the role that alcohol dependence plays in maintaining antisocial behavior or vice-versa.

Based on results of the factor analysis and LCA in the current study, and other more recent research examining the factor structure and possibility of subtypes with antisocial behavior, it is possible a two-factor structure is not the most appropriate model. Further research is needed to explore conduct disorder and adult antisocial behavior and determine if there are more appropriate ways to classify these individuals. It is possible the use of Item Response Theory (IRT) would be a more effective method of exploring the construct of conduct disorder and adult antisocial behavior. IRT explores the severity of a disorder in each participant by examining the specific symptoms endorsed (Gelhorn et al., 2008). It also allows for the

identification of which symptom criteria significantly predict psychopathology. Interventions could then be tailored to the different subtypes.

Overall, these disorders are associated with negative outcomes throughout the lifespan and have significant implications for society as a whole. If researchers can better understand antisocial behavior and associated outcomes, like alcohol dependence, the next step would be to develop more effective prevention and intervention strategies.

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Appendix A

Malmö- modified Michigan Alcohol Screening (Mm-MAST)

1) Do/did you take a drink before going to party?

2) Do/did you usually drink a bottle of wine or corresponding amount of beer or other alcoholic beverages over the weekend?

3) Do/did you drink a couple of drinks (or beers) a day to relax?

4) Do/did you tolerate more alcohol now than before?

5) Have/had you difficulties not drinking more than your friends?

6) Do/did you fall asleep after moderate drinking without knowing how you got to bed?

7) Do/did you have a bad conscience after drinking?

8) Do/did you take a drink (the day after a party) for your hangover?

9) Do/did you try to avoid alcoholic beverages for a determined period of time, e.g., a week?

10) After you have/had taken a drink, do you find it hard to stop?

11) Have/had you ever felt that anyone close to you thinks that you should drink less?

Appendix B

SSAGA Diagnostic Areas

A DEMOGRAPHICS
B MEDICAL HISTORY
C SOMATIZATION
D TOBACCO
E ALCOHOL
F MARIJUANA
G DRUGS
H EATING DISORDERS
I DEPRESSION
J DYSTHYMIA
K MANIA
L PSYCHOSIS
M ANTISOCIAL PERSONALITY
N SUICIDAL BEHAVIOR
O PANIC
P AGORAPHOBIA/SOCIAL PHOBIA
Q OBSESSIVE/COMPULSIVE
R SUBJECT COMMENTS
S COMORBIDITY
T INTERVIEWER'S OBSERVATIONS

Appendix C

Conduct Disorder Questions on the C-SSAGA-A (Age 14)

Most people do things that get them in trouble with their parents or teachers. I am going to ask you about different ways of getting into trouble.

E1A. Have you ever been suspended from school?

B. How many times have you been suspended from school?

C. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

Can you tell me why you were suspended?

E2A. Have you ever been expelled from school (kicked out for the rest of the year)?

B. How many times have you been expelled from school?

C. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

Can you tell me why you were expelled?

E3A. Have you ever stolen anything, like money from someone's purse or did you shoplift something from a store?

B. Have you ever stolen anything else besides money from someone's purse or by shoplifting? For example, did you ever take anything from somebody at school?

C. How many times have you stolen things altogether?

D. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

E. Have you ever used a credit card without permission or signed someone else's name on a check?

F. How many times have you done anything like use a credit card without permission or sign someone else's name on a check?

G. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

H. How old were you the first time you (NAME WHAT CHILD DID, E.G., STOLE MONEY FROM MOM'S PURSE OR SIGNED SOMEONE ELSE'S NAME ON CHECK.)?

I. When was the last time you did anything like that? Was it...

WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

J. How old were you then?

E4A. Have you ever run away from home overnight or longer?

B. How many times have you run away like that?

C. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

D. Did you go back home after you ran away?

E. How old were you the first time you ran away?

F. When was the last time you ran away? Was it...

WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

G. How old were you then?

E5A. Of course everybody tells lies or makes up stories once in a while. I'd like to ask if you lie or make up stories a lot?

B. Do you get into trouble a lot because people say you're lying? (Do your teachers/friends/parents get upset with you because they think you're lying?)

C. What is the main reason you lie(people say you're lying)?

D. How old were you when you first started telling lies, or when people said you were lying?

E. When was the last time you told lies or when people said you were lying?

Was it...

WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS

WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

F. How old were you the last time?

E6A. Have you ever set any fires on purpose that you weren't supposed to set?

B. How did it happen and what happened because of the fire(s)?

C. **CODE ACCIDENTAL OR DELIBERATE, OR SOME OF BOTH.**

D. How many times have you set fires on purpose like that?

E. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

F. How old were you the first time you set a fire on purpose?

G. When was the last time you set a fire on purpose?

Was it...
WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

H. How old were you then?

E7A. Have you ever skipped school?

B. How many times have you skipped school?

IF DK ASK C.

C. Was it

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

D. How old were you the first time you skipped school?

E. When was the last time you skipped school?

Was it...
WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

F. How old were you the last time you skipped school?

E8A. How about cutting classes-have you cut classes?

B. How many days have you cut classes?

IF DK ASK C.

C. Was it...

1 DAY
2 DAYS
3 OR MORE DAYS

D. How old were you the first time you cut classes?

E. When was the last time you cut classes?

Was it...
WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

F. How old were you the last time?

E9A. Have you ever broken into somebody else's house, building, or car?

B. How many times have you done that?

IF DK ASK C.

C. Was it

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

D. How old were you the first time you broke into somebody else's house,building, or car?

E. When was the last time you did that?

Was it...
WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

F. How old were you the last time?

E10A. Have you ever wrecked or destroyed someone else's property on purpose?

B. What happened?

C. How many times have you wrecked someone else's property on purpose?

IF DK ASK D.

D. Was it

1 TIME
2 TIMES
3-5 TIMES

6-10 TIMES
11+ TIMES

E. How old were you the first time you wrecked someone else's property on purpose?

F. When was the last time you did that?

Was it...
WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

G. How old were you the last time?

E11A. Have you ever hurt or killed an animal like a cat, a dog, or a pet like a bird, a gerbil, or a hamster?

B. How did it happen?

C. **NOTE TO INTERVIEWER: WAS THE HARMING DELIBERATE AND CRUEL?**

D. How many times have you done that?

IF DK ASK E.

E. Was it

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

F. How old were you the first time it happened?

G. When was the last time that happened?

Was it...
WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

H. How old were you the last time?

E12. Have you ever forced anyone to do sexual things with you?

E13A. Have you ever gotten into physical fights with other people?

B. How many times have you started these fights with other people?

C. Was it

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES

11+ TIMES

D. How old were you the first time you started a fight?

E. When was the last time you started a fight?

Was it...

WITHIN THE PAST TWO WEEKS

WITHIN THE PAST MONTH

WITHIN THE PAST SIX MONTHS

WITHIN THE PAST YEAR

OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

F. How old were you the last time?

E14A. Regardless of who started the fight, have you ever used other things besides your hands like sticks, rocks, or sharp objects when you've been fighting? (Did you ever use a knife or a gun?)

B. How many times have you used other things besides your hands in a fight?

IF DK ASK C.

C. Was it...

1 TIME

2 TIMES

3-5 TIMES

6-10 TIMES

11+ TIMES

D. How old were you the first time you used something besides your hands in a fight?

E. When was the last time you got into a fight and used something besides your hands?

Was it...

WITHIN THE PAST TWO WEEKS

WITHIN THE PAST MONTH

WITHIN THE PAST SIX MONTHS

WITHIN THE PAST YEAR

OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

F. How old were you the last time?

E15A. Have you ever mugged someone (held them up with a gun or knife) or snatched their purse? Have you ever bullied another kid until he gave you

B. How many times have you done something like that?

C. Was it...

1 TIME

2 TIMES

3-5 TIMES

6-10 TIMES

11+ TIMES

D. How old were you when you first mugged someone?

E. When was the last time? Was it...

WITHIN THE PAST TWO WEEKS

WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

F. How old were you the last time?

E16A. Other than when fighting, have you done anything on purpose to hurt another person or to cause them physical pain?

B. What did you actually do?

C. **NOTE TO INTERVIEWER: THE HARMING HAS TO BE CRUEL WAS THE HARMING CRUEL?**

D. How many times have you hurt another person on purpose?

IF DK ASK E.

E. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

F. How old were you the first time you hurt another person on purpose?

G. When was the last time?

WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS
WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

H. How old were you the last time?

E17A. Have you ever been in trouble with the police? Can you tell me what happened?

B. Have you ever appeared in juvenile court? Can you tell me what happened?

C. How many times have you been in trouble with the police or appeared in juvenile court?

IF DK ASK D.

D. Was it...

1 TIME
2 TIMES
3-5 TIMES
6-10 TIMES
11+ TIMES

E. How old were you the first time you got in trouble with the police and/or appeared in juvenile court?

F. When was the last time?

WITHIN THE PAST TWO WEEKS
WITHIN THE PAST MONTH
WITHIN THE PAST SIX MONTHS

WITHIN THE PAST YEAR
OVER A YEAR AGO

IF OVER A YEAR AGO, ASK:

G. How old were you the last time?

Appendix D

Antisocial Personality Questions on the SSAGA (Age 22)

Note: This section covers antisocial behavior present prior to age 15 in order to make a diagnosis of childhood conduct disorder, which is Criterion C for a diagnosis of ASPD. However, the analyses delineated above will focus on adult antisocial behavior as reported subsequent to age 15, Criterion A of ASPD.

Now I'd like to ask you some questions about when you were younger.

M1 Did you ever play hooky from school for an entire day at least twice in 1 year?
- A. Was that only in your last year in school or before that?
- B. How old were you when you first played hooky?

M2 Were you ever suspended or expelled from school?
- A. How old were you when you were first suspended/expelled?

M3 Did you ever run away from home overnight?
- A. Why did you run away?
- B. Did this happen more than once?
- C. After you ran away, did you return home?
- D. How old were you when you first ran away from home overnight?

M4 Throughout your life have you told a lot of lies or have you ever used a false name or alias?
- A. Why did you tell a lot of lies or use an alias?
- B. How old were you when you first told a lot of lies or used an alias?
- C. How old were you the last time?

M5 Did you more than once steal money or things from your home or family?
- A. How old were you the first/last time?
- B. Did you more than once steal or shoplift from stores or from other people (without their knowing it)?
- C. How old were you the first/last time?
- D. Did you more than once forge anyone's signature on a check or credit card?
- E. How old were you the first/last time?

IF M5, M5B AND M5D ARE ALL CODED 1, SKIP TO M6. OTHERS ASK F.

- F. Since your 15th birthday, have you stolen things (or forged a signature) 3 or more times?

M6 Have you ever damaged someone's property on purpose?
- A. How old were you when you first did this?

IF A IS LESS THAN 15 ASK B. OTHERS SKIP TO D.

- B. Did you more than once damage someone's property before you

turned 15?
C. Since your 15th birthday, have you damaged someone else's property on purpose?
D. Have you done this 3 or more times since your 15th birthday?

M7 Did you start physical fights with persons other than your brothers or sisters 3 or more times?
A. At what age did you first start fights?
B. (Even though you didn't start fights,) Since your 15th birthday, did you get into physical fights (other than in combat or as part of your job)?
C. Did this happen 3 or more times since your 15th birthday?

M8 Did you more than once use a weapon like a stick, gun or a knife in a fight (other than in combat or as part of your job)?
A. How old were you the first time you used a weapon like that?
B. How old were you the last time you used a weapon like that?

M9 (Outside of fighting) have you ever physically injured anyone on purpose?
A. How old were you the first time?
B. How old were you the last time?

M10 Have you had any traffic tickets in your life for things like speeding, or running a red light, or causing an accident?
A. How many tickets have you received?
IF DK ASK:
1. Was it at least 4?
B. How old were you the first time?

M11 Have you ever been arrested for anything other than traffic violations?
WHAT WAS THE REASON?
A. How old were you the first/last time you were arrested?
B. How many times have you been arrested?
C. Have you ever been convicted of a felony?
D. Have you ever spent time in jail for something other than using drugs or alcohol?
E. Since you got out of jail have you ever been arrested for things other than drugs or alcohol?

M12 When you were younger did you often challenge your parents, teachers, or other adults by refusing to do things they asked you to do, just because you didn't want to? (things like not doing chores or running errands, not participating in class, or not behaving well at home or at school)
A. How old were you the first time?

M13 Did you often throw temper tantrums as a child?
A. How old were you the first time?

M14 Were you often a bully, deliberately hurting or being mean to other children?

M15 Were you ever mean to animals including pets?

A. How old were you the first/last time?

M16 Did you ever deliberately set any fires you were not supposed to?

A. How old were you the first/last time?

B. Since your 15th birthday, have you 3 or more times set fires you weren't supposed to?

M17 Was there ever a time when you really enjoyed outsmarting people in authority (like parents, your boss, or the police), to the point that you would often go out of your way to put something over on them?

A. How old were you the first/last time?

B. Since your 15th birthday, have you 3 or more times put something over on people?

M18 Did you ever break into someone's car or house or any place else (not because you were locked out)?

A. How old were you the first/last time you did that?

B. Has this happened 3 or more times since you were 15?

M19 Have you ever taken money or property from someone else by threatening them or using force, like snatching a purse or robbing them?

A. How old were you the first/last time?

B. Has this happened three or more times since you were 15?

M20 You mentioned that before your 15th birthday you (LIST 5*). Did you continue to do at least two of these things for a period of six months or longer?

M21 Since your 15th birthday, have you ever done anything that you could have been arrested for even if you weren't, such as:

1. Deliberately writing bad checks?
2. Receiving, selling or buying stolen goods (fencing), selling drugs, or running numbers?
3. Being paid for having sex with someone?
4. Were you paid with drugs?
5. Finding customers for male or female prostitutes or call girls?

IF NO 5 CODED IN M21.1-5 ASK A. OTHERS SKIP TO B.

A. Since your 15th birthday, have you ever done anything else that you could have been arrested for, even if you weren't?

B. Have you done these things 3 or more times?

Now I'm going to ask you a few more questions about your relationships and your sexual experiences.

M22 Since you were 18, have you ever had a friendship or love relationship that lasted continuously for more than one year?

M23 How old were you when you first had sexual intercourse?

A. How many sexual partners have you had in our life?
IF 10 OR MORE ASK B. IF ONLY 1 SKIP TO M26.
B. Have you ever had sex with as many as 10 different people within a single year?

M24 Have you ever been unfaithful to any person in a romantic or love relationship, that is, when you had an affair or one-night stand?
A. During any relationship did you ever have a period of more than one year when you did not have any other sexual relationships?

M25 During (any) marriage (or live-in relationship), did you have sexual relations outside of the relationship with 2 or more different people?

M26 Have you ever forced anyone into sexual activity, including intercourse?
A. How old were you the first time?

M27 Since you were 15, have you ever been accused of child abuse, or been the subject of a complaint on the child abuse hotline?

M28 Since you were 15, have you often hit, physically attacked, or thrown things at anyone (including your wife/husband/partner/children)?

M29 Since you were 15, have you quit 3 or more jobs before having another job lined up?
A. Since you were 15, have you enrolled in and dropped out of 3 or more academic programs?

M30 On any job you have had since you were 15, have you been late or absent an average of 3 day a month or more?

M31 In the last five years, have you been without a job for 6 months or more?
A. Other than when you were in school, or sick, on strike, laid off, a full-time homemaker, retired, (or in jail), were you ever without a job for 6 months or more within the past 5 years?

M32 Since your 15th birthday, have you ever traveled around without any arrangements or had no regular place to live for a month or more?
DON'T COUNT VACATIONS.

M33 Since your 15th birthday, have you often failed to pay debts that you owed, had things you bought taken back, or failed to take care of other financial responsibilities? (Examples: credit card charges, loans from family or friends, car or house loans.)

M34 Since you were 15, have you:
1. often not provided financial support to your children when you were supposed to?
2. often left young children under 6 at home alone while you were out shopping or doing anything else?

3. had a neighbor feed or take care of a child of yours (or one you were looking after) because no one was taking care of or feeding him/her at home?
4. had a nurse, social worker or teacher say that your child (or one you were caring for) wasn't getting enough to eat, wasn't being kept clean or wasn't getting needed medical attention?
5. more than once run out of money for food for your family because you had spent the food money on yourself or on going out?

IF ANY 3, 5, OR 6 CODED IN M34. 1-5 ASK A.

A. How old were you the first time this happened?

M35 Have you often ignored the feelings of others in order to do what you wanted?

M36 Have you frequently lost your temper, or has it been easy to annoy you or make you mad?

A. Have you often felt irritable, angry or resentful?

M37 Have you often felt that others were to blame for your troubles?

A. Have you often felt that others were to blame for your mistakes?

M38 Please review these items you told me about before. When you were in situations such as these where you harmed or took advantage of others in some way, did you more often than not feel bad or guilty afterwards?

A. Was that because you felt the person(s)involved deserved it more times than not?
B. How old were you the last time you were in any of these situations?

Appendix E

Alcohol Questions on the SSAGA (Age 22)

E1 Now I would like to ask you some questions about your use of alcoholic beverages, like beer, wine, wine coolers, champagne, or hard liquor like vodka, gin, or whiskey. Have you ever had a drink of alcohol?

A. So you have never had even one drink of alcohol?

E2 Let's begin with the last week. Did you have any drink containing alcohol in the last week?

A. We would like to know the number of alcoholic drinks you've had each day in the last week, and how long it took you to drink them. Today is _____. Let's begin with yesterday.
How many drinks of (beer, wine, liquor) did you have on **(NAME DAY OF WEEK)?**

B. How long did it take you to drink that? (IN MINUTES)

C. When was the last time you had a drink?

E3 Would you say that your (drinking/not drinking) in the past week was typical of your drinking habits within the past 6 months?

A. We would like to know the number of drinks containing alcohol you would have in a typical drinking week and how long it would take to drink them. On a typical (Monday, Tuesday...) how many drinks of (beer, wine, liquor...) would you have?

B. How long would it take you to drink that? (IN MINUTES)

E4 At what age did you begin to drink regularly--that is, drinking at least once a month for 6 months or more?

A. How old were you the first time you got drunk, that is, your speech was slurred or you were unsteady on your feet?

B. Was it before you were 15 years old?

C. Did you get drunk more than once before you were 15?

E5 What is the largest number of drinks you have ever had in a 24-hour period?

E6 Was there ever a time when you drank almost every day for a week or more?

A. Think about those periods when you drank almost every day for at least a week. What was the largest number of drinks you would drink almost every day for at least 1 week?

B. So almost every day during this period you drank at least (# FROM A) drinks?

C. How long did this period last?

D. When you were drinking this amount were you able to function normally?

E. Did you ever have a period of a month or more when you had at least one drink each week?

E7 While drinking, has one or two drinks of alcohol ever caused you to:

A. 1. flush or blush -- that is, your face and hands felt hot and your face turned red?
2. break out into hives?
3. feel very sleepy?
4. have nausea?
5. have headaches, or head pounding or throbbing?
6. have heart palpitations, where your heart beat so hard you could feel it?

B. Did (SX) ever keep you from drinking alcohol?

E8. (Since (AGE OF REGULAR DRINKING IN E4)), what is the longest period you have gone without drinking?

A. How many times have you gone without drinking for 3 months or more?
B. Can you tell me when these periods occurred?

E9 Have you 3 or more times wanted to stop or cut down on drinking?

A. How old were you the first time?
B. Have you ever tried to stop or cut down on drinking?
C. Were you always able to stop or cut down when you wanted to?
D. How old were you the first time?

E10 Did you ever need a drink just after you had gotten up (that is, before breakfast)?

A. Did you ever take a drink just after you had gotten up?
B. How old were you the first time?
C. Did this happen 3 or more times?

E11 In situations where you couldn't drink, did you ever have such a strong desire for it that you couldn't think of anything else?

A. How old were you the first time?

E12 Have you ever gone on binges or benders when you kept drinking for 2 days or more without sobering up, except for sleeping?

A. Did you neglect some of your usual responsibilities then?
B. How many binges like that have you had?
C. Did you go on binges 3 or more times?
D. How old were you the first time?

E13 Have you ever started drinking at times you promised yourself that you wouldn't, or have you ever drunk more than you intended to?

A. Have you ever continued drinking for more days in a row than you intended to?
B. How old were you the first time?
C. Did this happen 3 or more times?

E14 Have you ever started drinking and become drunk when you didn't want to?

A. How old were you the first time?

B. Did this happen 3 or more times?

E15 Has there ever been a period of several days or more when you spent so much time drinking or recovering from the effects of alcohol that you had little time for anything else?

A. Did this period last for a month or more?

B. How old were you the first time?

E16 Did your drinking ever become so regular that you would not change when or how much you drank no matter what you were doing or where you were?

A. How old were you when your drinking first became that regular?

E17 1. Were there ever objections about your drinking from family, friends, doctor or clergyman?

2. Did your drinking ever cause you to have problems with your family or friends?

3. Have you ever lost friends on account of your drinking?

4. Did your drinking ever cause you to have problems at work or school?

5. Did you ever get into arguments when you had been drinking?

6. Did you ever hit things or throw something when you had been drinking?

7. Did you ever hit anyone in your family when you had been drinking?

8. Did you ever hit anyone else when you had been drinking without getting into a fight?

9. Did you ever get into physical fights while drinking?

E18 Have you ever...

1. hidden alcohol from others so that you wouldn't run out in case you needed a drink?

2. bought liquor at several different places so no one would know how much you purchased?

3. tried to get someone to buy liquor for you because you were ashamed to buy it yourself?

4. hidden alcohol from others so that they wouldn't know if you were drinking or how much you were drinking?

5. hidden empty liquor bottles and got rid of them secretly?

A. Would you only do these because your family or friends were against drinking in general?

B. Would you only do these because you were drinking under the legal drinking age?

E19 Did you ever drink unusual things such as rubbing alcohol, mouthwash, vanilla extract, cough syrup, or any other non-beverage alcohol?

A. How old were you the first time?

E20 After you started drinking regularly did you ever become tolerant to alcohol, that is, you drank a great deal more in order to get an effect, or found you could no longer get high on the amount you used to drink?

A. Would you say the increase was 50% or more?
B. Did you ever find you could drink a lot more before you would get drunk?
C. Would you say the increase was 50% or more?
D. How old were you the first time?

E21 Some people try to control their drinking by making rules, like not drinking before 5 o'clock or never drinking alone. Have you ever made any rules to control your drinking?
A. How old were you the first time?

E22 Have you ever given up or greatly reduced important activities while drinking -- like sports, work, or associating with friends or relatives?
A. How old were you the first time?
B. Did this happen 3 or more times or for a month or more?

E23 Has your drinking or being drunk or hung over often interfered with your working or taking care of school or household responsibilities?
A. How old were you the first time?

E24 Did your drinking cause serious or repeated problems in any marriage/love relationship?
A. How old were you the first time?
B. Did you continue to drink knowing it caused these problems?

E25 Did you ever think that you were an excessive drinker?
A. How old were you the first time?

E26 Have you ever felt guilty about drinking?
A. How old were you the first time?

E27 Have you ever been arrested for drunk driving?
A. Has your drinking and driving ever resulted in your damaging your car or having an accident?
B. How old were you the first time?
C. How many times has this happened?
D. Did this happen 3 or more times?

E28 Have you ever been arrested or detained by the police even for a few hours, because of drunk behavior (other than drunk driving)?
A. How old were you the first time?
B. How many times has this happened?
C. Did this happen 3 or more times?

E29 Have you accidentally injured yourself when you were drinking, that is, had a bad fall or cut yourself badly, been hurt in a traffic accident, or anything like that?
A. How old were you the first/last time?
B. How many times has this happened?
C. Did this happen 3 or more times?

E30 When you were (very) drunk did you ever drive a car, motorcycle or boat, use a knife, power equipment or gun, cross against traffic, climb or swim, or put yourself in any other situation where you might have gotten hurt?

A. How old were you the first time?
B. How many times has this happened?
C. Did this happen 3 or more times?

E31 Have you ever had blackouts when you didn't pass out while drinking, that is, you drank enough so that the next day you couldn't remember things you had said or done?

A. How old were you the first time?
B. How many blackouts have you had from drinking?
C. Did you have 3 or more blackouts?

E32 People who cut down, stop or go without drinking after drinking steadily for some time may not feel well. These feelings are more intense than the usual hangover. When you stopped, cut down or went without drinking, did you ever experience any of the following problems? COL I

1. Did you have the shakes (hands trembling)?
2. Were you unable to sleep?
3. Did you feel anxious or depressed?
4. Did you sweat?
5. Did your heart beat fast?
6. Did you have nausea/vomiting?
7. Did you feel physically weak?
8. Did you have headaches?
9. Did you hear or see things that weren't there?

A. How old were you the first time you had the shakes (hands trembling)?
B. What was the longest time that this/any of these problem(s) lasted?
C. Was there ever a time when two or more of these problems occurred together?
D. Which ones?
E. How old were you the first time these problems occurred together?
F. How many times did you have problems like these (this)?
G. Did this occur 3 or more times?
H. On 3 or more different occasions have you taken a drink to keep from having any of these problems (or to make them go away)?
I. Did you ever take any medication/drug to avoid any of these problems (or to make them go away)?

E33 When you stopped, cut down, or went without drinking, did you have fits, seizures, or convulsions, where you lost consciousness, fell to the floor, and had difficulty remembering what happened?

A. How old were you the first time this happened?
B. How many times did this happen?
C. Did this occur 3 or more times?
D. On 3 or more different occasions have you taken a drink to keep from having these symptoms or to make them go away?

E. Did you ever take any medication/drug to avoid these symptoms or to make them go away?

E34 Did you have the DT's, where you were very confused, extremely shaky, felt very frightened or nervous, or saw things that weren't really there when you stopped, cut down or went without drinking?

A. How old were you the first time this happened?
B. How many times did this happen?
C. Did this occur 3 or more times?
D. On 3 or more different occasions have you taken a drink to keep from having these symptoms or to make them go away?
E. Did you ever take any medication/drug to avoid these symptoms or to make them go away?

E35 There are several other health problems that can result from long stretches of drinking.

Did drinking ever:
1. cause you to have liver disease or yellow Jaundice?
2. give you stomach disease or make you vomit Blood?
3. give you pancreatitis?
4. damage your heart (cardiomyopathy)?
5. cause your feet to tingle or feel numb for many hours?
6. give you memory problems even when you weren't drinking (not blackouts)?
7. other problem? Specify _____
A. How old were you when you first found out drinking had given you any of these health problems?
B. Did you continue to drink knowing that drinking caused you to have health problems?

E36 Have you ever continued to drink when you knew you had any (other) serious physical illness or condition that might be made worse by drinking?

A. What illness?
B. How old were you the first time?
C. Have you used alcohol 3 or more times while taking medication or drugs you knew were dangerous to mix with alcohol?
D. What medication/drugs?
E. How old were you the first time?

E37 Has drinking ever caused you emotional or psychological problems like: NO

1. Feeling depressed or uninterested in things for more than 24 hours to the point that it interfered with your functioning?
2. Feeling jumpy or easily startled or nervous to the point that it interfered with your functioning?
3. Having such trouble thinking clearly for more than 24 hours that it interfered with your functioning?
4. Feeling paranoid or suspicious of people for more than 24 hours to the point that it interfered with your relationships?

5. Hearing, seeing, or smelling things that weren't really there?
A. Did you continue to drink after you knew it caused you any of these problems?
B. How old were you the first time?

E38 Have you ever brought up any problem you might have had with drinking with any professional?

A. Did you talk with:
1. A psychiatrist?
2. Another medical doctor?
3. A psychologist?
4. Another mental health professional?
5. A clergyman?
6. Anyone else? Specify _____

B. How old were you the first time you brought up any problem you had with drinking?
C. With whom did you speak first?

E39 Have you ever been treated for a drinking problem?

A. Were you treated at:
1. AA or other self help
2. Outpatient alcohol program
3. Outpatient, other
4. Inpatient alcohol program
5. Inpatient for medical complications
6. Other, specify _____

B. How old were you the first time you were treated?
C. Where were you treated first?

E40 Please review these experiences. When was the most recent time you had this experience/any of these experiences?

A. Was there ever a period lasting a month or longer when you had 3 or more of these experiences occurring together? NOTE: MUST BE 3 FROM DIFFERENT GROUPS.
B. Was there ever a period lasting a month or longer when you had 2 or more of these experiences occurring together? FROM 2 DIFFERENT GROUPS.
C. How old were you the first/last time you had 3 or more of these experiences (FROM DIFFERENT GROUPS) occur within a period lasting a month or more?

E41 Please review these experiences. When was the most recent time you had this/any of these experiences?

A. Was there ever a period lasting a month or longer when you had 3 or more of these experiences occurring together? NOTE: MUST BE 3 FROM DIFFERENT GROUPS.
B. How old were you the first/last time you had 3 or more of these experiences (FROM 3 DIFFERENT GROUPS) occur within a period lasting a month or more?

C. Did 3 or more of these occur at some time during the last 12 months?

E42 **ARE THERE 2 OR MORE 5'S CODED IN QS. E9-E37?**

Vita

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