Risk Factors for Conduct Disorders and Antisocial Personality Disorders

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Introduction

Conduct disorder is one of the most common psychiatric disorders of children and adolescents between the ages of four and 16 (Shamsie 2001). Conduct disorders are prevalent among 5-10% of the children and adolescents ages 8-16, diagnosed in late childhood or early adolescent years (Hill 2000). Conduct disorder is more common among boys than girls, with studies indicating that the rate among boys in the general population ranges from 6% to 16% while the rate among girls ranges from 2% to 9%. Diagnoses of conduct disorder peak for boys at age 10, whereas diagnoses of conduct disorder among girls rise through age 16 (Cohen et al., 1993). Conduct disorder is not only associated with impaired functioning in childhood (Earls, 1994) but can lead to psychiatric and social difficulties in adult life (Robins 1978). Some studies demonstrate that up to 40% of those who had been diagnosed as having conduct disorder in childhood continue to have serious psychological disturbances in adulthood (Shamsie 2001). Conduct disorder is a devastating condition, not only because youth with the disorder repeatedly inflict physical harm and property loss on others, but also because the youth themselves are at risk for other forms of psychopathology (e.g., depression, suicide, substance abuse).

The high prevalence and severity of the problems, personal and societal, arising from aggressive and disruptive behaviors in children demand an understanding of the multitude of nested risk factors for conduct and antisocial personality disorders (Hill 2002). In this review we hope to present the current state of knowledge about non-genetic, potentially modifiable risk factors for conduct disorders (CD) and antisocial personality disorders (APD).

Definition

Conduct disorder is one of three disruptive behavior disorders, the others being oppositional defiant disorder and attention-deficit hyperactivity disorder. CD is a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, according to the current definition of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The child or adolescent usually exhibits these behavior patterns in a variety of settings—at home, at school, and in social situations—and they cause significant impairment in his or her social, academic, and family functioning. These behaviors fall into four major groupings: 1) aggressive conduct that causes or threatens physical harm to other people or animals; 2) Non-aggressive conduct that causes property loss or damage. 3) Deceitfulness or theft. 4) Serious violations of rules. The onset of conduct disorder may occur as early as age 5 or 6, but more usually occurs in late childhood or early adolescence; onset after the age of 16 years is rare (American Psychiatric Association, 1994). Conduct disorder may be diagnosed in individuals who are older than 18 years of age, but only if the criteria for Antisocial Personality Disorder (APD) are not met. Conduct disorder is classified as "mild" if there are few, if any, conduct problems in excess of those required for diagnosis, and if these cause only minor harm to others (e.g., lying, truancy and breaking parental rules). A classification of "moderate" is applied when the number of conduct problems and effect on others are intermediate between "mild" and "severe". The "severe" classification is justified when many conduct problems exist which are in excess of those required for diagnosis, or the conduct problems cause considerable harm to others or property (e.g., rape, assault, mugging, breaking and entering) (American Psychiatric Association, 1994).
Antisocial Personality Disorders (APD) may also constitute disruptive behaviors and psychopathology among adolescents and young adults, thus risk factors for APD are included in our review as well. Usually, APD cannot be diagnosed until age 18 but some symptoms of APD may be present in a subgroup of disruptive youths at a younger age. The early presence of such symptoms may identify those youths with CD who eventually qualify for APD (Frick, 1998).

The definition of APD in DSM-IV (American Psychiatric Association, 1994) is more of a combination of personality feature and behavior; for example “irritability and aggressiveness”, as indicated by repeated physical fights and assaults”. DSM-IV lays down the following criteria for APD diagnosis: a) pervasive pattern of disregard for and violation of rights of others, occurring since age 15 years, as indicated by three (or more) of the following: Failure to conform to social norms with respect to lawful behaviors, deceitfulness, impulsivity, irritability and aggressiveness, reckless disregard for safety or self or others, consistent irresponsibility, lack of remorse; b) current age at least 18 years; c) evidence of CD with onset before 15 years; d) Occurrence of antisocial behavior is not exclusively during the course of schizophrenia or a manic episode.

Criteria for Inclusion

The main goal of this review is to present the current state of knowledge about non-genetic, potentially modifiable risk factors for CD/APD in population-based samples using a longitudinal design. Therefore, strict inclusion criteria were used. First, only prospective, population-based studies published between 1990-2003 were reviewed. This study design ensures that the risk factor of interest was present prior to the diagnosis of CD/APD. Studies that used retrospective or cross-sectional reports of risk factors were excluded.

Second, only studies with an outcome of DSM-diagnosed CD/APD were included; however, we did not differentiate between the DSM-III, DSM-III-R, and DSM-IV. Studies that used symptom scales, such as the Child Behavior Checklist or other non-DSM scales (i.e. Rutter scale) to diagnose CD/APD were excluded.

Third, studies in which the subjects or probands were clinic-referred or were currently undergoing any form of psychiatric treatment were excluded. Hence, for instance, children of opiate-dependent parents from an urban hospital were not included, or births to substance-abusing mothers recruited from a drug-treatment clinic or WIC program were not included. Also, studies involving solely police reports of outcome were excluded. Studies where prior psychiatric disorders or conduct problems were assessed in the cohort over time from childhood to adulthood (in addition to other risk factors) were included.

Fourth, studies using Oppositional Defiant Disorder or “delinquency” or “violence” or “externalizing symptoms”, “conduct problems” as the outcomes to classify CD/APD were excluded.

Fifth, we included studies of comorbidity, where the outcomes of interest were CD in the presence of other disorders, such as substance abuse or ADHD.

Finally, studies involving subjects from adoption or twin studies were included.

For this review, a search of Current Contents, Medline, PubMed and PsychInfo, from 1990 till May 2003 of English language literature was conducted using the following keywords: conduct disorder, antisocial personality disorder, antisocial, conduct, risk factor, cause, causality, longitudinal, clinical trial, and prospective. Also, reference lists of key journal articles as well as author and study names were further researched to assure inclusion.
of related studies. After duplicates were removed, approximately 17,804 articles matching conduct disorder, antisocial personality disorder and/or one or more of the above keywords were identified. Of these articles, 17,661 were excluded for not meeting the study criteria. The remaining 143 studies were retrieved and further reviewed for inclusion criteria (See Flow Chart). Of these, 40 studies were excluded due to clinic-based population or proband, 33 excluded as the outcome was other than conduct disorder or APD, 8 were literature reviews, 24 used a non-DSM criteria or instrument (i.e. CBCL), 23 excluded due to study design (i.e. retrospective data collection, cross-sectional), and 5 for other reasons (i.e. genetic risk factors); resulting in a total of 15 studies.

**Evidence Tables**

The results of the studies are presented in evidence table 2 (organized by risk factor). Some of the studies using CD symptoms are not presented in the table but are mentioned in the text. Several other studies that may have used clinic-referred probands (i.e. births to mothers who abused substances during pregnancy from obstetric clinics, or from WIC) or symptom scales to approximate DSM criteria are also not included in the evidence tables but mentioned in the text.

The longitudinal birth cohort data from the Christchurch Health and Development study (1,265 births in 1977, New Zealand), and the Dunedin Multidisciplinary Health and Development Study (1,037 births in 1972-73, New Zealand) were repeatedly utilized and have been very effective in informing predictability of CD/APD prospectively. The Cambridge study in Delinquent Development, of 411 males from working-class inner-city area of South London who were followed from childhood into adulthood, also provided useful information towards development of APD. Most studies used multivariate logistic regression analyses. As much as possible, we opt to present, in the discussion section, prospective percentages (i.e. percentage of participants who develop CD or APD) and the odds or risk ratios, a measure of strength of effect; as unlike other measures (e.g. correlations), odds ratio is not affected by changes in the prevalence of the predictor or outcome variables (Fleiss 1981).

**Discussion**

While a huge body of longitudinal research exists that discusses risk factors for conduct disorders, there is however a great variability in methodology, sampling, measurements, and outcome variables. Nonetheless, several studies have consistently shown the presence of individual, family and environmental factors that may increase the risk of later conduct disorders. Risk factors of CD include the following: 1) family factors, such as psychiatric problems in parents, criminal behavior in fathers, family dysfunction, and inconsistent parenting; 2) child factors such as male gender, biological vulnerability, difficult temperament, early behavioral problems, low IQ and school failure; 3) community factors such as socioeconomic disadvantage, delinquent peers, and poor school environment. (Shamsie 2001)

Moffitt et al further emphasized the importance of differentiating risk factors for life-course persistent vs. adolescence-limited forms of antisocial behavior (i.e. CD or APD). They hypothesized that the prospective predictors of life-course persistent antisocial behavior may include gender, temperament, cognitive abilities, school achievement, personality traits, mental disorders (e.g. hyperactivity), family characteristics (child-rearing practices, parent
and sibling deviance), and socioeconomic status; whereas the knowledge of peer delinquency should be considered in adolescence-limited APD (Moffitt and Caspi 2001).

**Developmental Trajectories**

Over the past decades the use of a developmental perspective to study psychopathology has led researchers to look at the specific points of onset of specific disorders in the developmental pathway. Sylvana et al., 2001 examined developmental trajectories of disruptive behaviors among girls’ during the elementary school years and predicted CD symptoms and diagnosis in adolescence with trajectories of these behaviors (Cjte, Zoccolillo et al. 2001).

Four groups of girls, ages 6 to 12, following trajectories with distinct levels of disruptive behaviors were identified: a low, medium, medium-high and high trajectory. More than two-thirds of the girls with CD were in the medium-level or higher trajectories. The girls in the medium-high and high trajectories were at significant higher risk for CD, compared with girls in the low group (OR= 4.46). However, confounding by family environment, peer relationship, maternal smoking and social disadvantage and other important covariates was not adjusted for.

In 2002, the authors further observed that there were certainly sex differences in the prevalence of the childhood behavioral profiles representing risk for CD (Cote, Tremblay et al. 2002). Boys had a significant risk for CD if they were hyperactive (OR=4.27), hyperactive and unhelpful (OR= 2.83), or hyperactive, fearless and unhelpful (OR= 3.93). Girls had a significant risk for CD only if they were hyperactive and unhelpful (OR=4.61). Almost 8 times more boys than girls exhibited a sex-specific risk profile and almost 3 times more boys had a CD diagnosis during adolescence than girls. This study supports previous studies showing the importance of childhood hyperactivity as a risk for antisocial disorders in males.

Similarly, following a cohort of hyperactive children into adulthood, Fischer et al concluded that young adult with passive-aggressive personality were 4 times higher odds of having APD (Fischer, Barkley et al. 2002).

The Dunedin study, which uses the Rutter scale in conjunction with additional items to conform to the DSM-IIIR to diagnose APD, suggest that behavior styles observed in childhood (at age 3) are significantly related to mental-health problems at age 21 years, even with adjustments for social class characteristics. Under-controlled children, characterized by impulsive, restless, and distractible behavioral style, were at increased risk of psychiatric problems and criminal activities at age 21, and most severely impaired by their condition. Under-controlled children were 2.9 times more likely to be diagnosed with APD (P<.05, 95% CI, 1.1-8.1) compared to normal children. No significant sex-by-temperament interaction effect was observed (Caspi et al 1996).

Based on a longitudinal study of 742 boys from low socioeconomic areas of a large metropolitan city in Quebec, Canada, Vitaro et al. concluded that proactive aggression, not reactive aggression at age 12, significantly predicted conduct disorder symptoms (B=0.24, p<.05) and delinquency at ages 14 to 16. Being in the proactively aggressive category tripled the risk of having a disruptiveness diagnosis (CD and/or ODD) (B=1.12, Wald=11.00, OR=3.06). Possible explanations include that proactive boys tend to be more similar to their peers than classmates on proactive aggression but not on reactive aggression, or that they tend to be less isolate, anxious or withdrawn than reactive aggressive boys. Strengths of the study include: a large sample, multiple informants and dependent measures, and a longitudinal perspective. The study did not
however include data on peer relations or family dynamics or internalizing problems (Vitaro, Brendgen et al. 1999).

**Maternal smoking during pregnancy**

The association between maternal smoking and conduct disorders is moderate. There seems to be some evidence of a dose-response relationship between number of cigarettes smoked and percentage of offspring with CD. In the Christchurch study, Fergusson et al evaluated the effects of maternal smoking during pregnancy on the development of psychiatric disorders, including CD, (Fergusson, Woodward et al. 1998) among New Zealand birth cohort of 1,265 subjects aged 16 to 18 years. Even after controlling for potential confounders (i.e. maternal age, education level, planned pregnancy, childhood sexual abuse, parental use of physical punishment parental criminal behavior), there was a clear and significant relationship between CD symptoms and maternal smoking during pregnancy. Children whose mothers smoked at least one pack of cigarettes per day during pregnancy were twice as likely to develop CD by age 18 than children of non-smoker mothers. The mean rate of CD symptoms increased proportional to the number of cigarettes, and were more than twice as high among boys than girls. Also children of mothers who smoked during pregnancy were exposed to higher levels of social disadvantage, parental deviance and family dysfunction throughout childhood compared with the children of women who did not smoke during pregnancy; controlling for these explained almost all of the association between smoking and CD.

Similarly, Wakschlag et al found maternal smoking to be a robust independent risk factor for conduct disorder in male offspring, 177 clinic-referred 7 to 12 year old boys, followed for 6 years (Wakschlag, Lahey et al. 1997; Wakschlag and Hans 2002). They found that mothers who smoked more than half a pack of cigarettes daily during pregnancy were significantly more likely to have a child with conduct disorder (OR= 4.4; p=.001) than mothers who did not smoke during pregnancy. This association was statistically significant when controlling for socioeconomic status, maternal age, parental antisocial personality, substance abuse during pregnancy, and maladaptive parenting.

Note that findings from several excellent studies that utilized data on children born to substance abusing or drug-dependent mothers from clinics or children of opiate-dependent or alcoholic parents could not be included, as the results were not population-based or generalizable.

**Low birth weight**

Although low birth weight and perinatal complications have been associated with psychiatric disorders (Szatmari 1985), a study by Szatmari et al found that rates of CD at 5 years were not increased among 82 extremely low birth weight (ELBW) babies in Ontario, Canada compared to babies born normal weight (Szatmari, Saigal et al. 1990). Based on parental assessments, 2.4% of the ELBW children had CD compared to 0.5% of the controls and according to teacher assessment 1.6% vs. 3.2% of the cases and controls had CD. The authors note the age of the sample and availability of better assessments of psychiatric diagnoses tools as possible explanation of the limited findings.

Wakschlag et al also found, among a sample of 77 African American low-income children followed for 10 years since birth, some with a documented history of prenatal exposure to opiate drugs, that after controlling for maternal smoking during pregnancy, birth
weight was no longer significantly associated to boys’ CD symptoms (B=-0.16, p>.05) (Wakschlag and Hans 2002).

**Family Structure and Characteristics**

A longitudinal study of psychiatric disorder by Rowe et al looked at 4 annual waves of data from a rural community showed significant links between family/ environmental adversity and development of CD (Rowe, Maughan et al. 2002). Boys with atypical family structure (i.e. teenage parent at birth, moved often, stepparent) assessed at wave 1 were at 5 times higher odds of developing CD at subsequent waves. Biological parents characteristics and social disadvantage did not contribute significantly to prediction. Moreover, boys with ODD had 12.9 higher odds of developing CD compared to the boys without ODD at wave 1, adjusting for the presence of depression and anxiety. Despite the large overall sample size, some of the longitudinal analyses may have suffered from low power, as the number of cases was relatively low. The cross-sectional nature of the data collection, although conducted over time, may influence the direction of the effects observed. Also the study covered the core years from ages 9 to 13 when in fact many of the disruptive behaviors are common, and it did not include direct measures of early childhood. Finally, the study focused on a predominantly rural community perhaps limiting the generalizability of the findings to the general population.

Cadoret et al similarly found that adverse adoptive home environment, defined as adoptive parents who had marital problems, were divorced, separated, or had anxiety conditions, depression, substance abuse/ and or dependence, or legal problems, independently predicted increased adult antisocial behaviors (Cadoret et al 1995). Adverse home environment interacted with biological background of APD to result in significantly increased risk of conduct disorder in the presence of but not in the absence of a biological parent with APD.

Moreover, data from Christchurch study of 1,265 New Zealand children showed that exposure to interparental violence, particularly father initiated violence, may be associated with later increased risks of conduct disorder.(Fergusson and Horwood 1998)

Data from the Cambridge study in Delinquent Development in which 411 males living in a working-class inner-city area of South London were followed since age 8 (1961-92) into adulthood revealed similar findings. Farrington et al (2000) found that having a convicted parent before the 10th birthday was the most important explanatory predictor of antisocial personality at age 18 (and at age 32); prospectively, 48% of the boys with a convicted parent became antisocial, compared with 14% of the remainder (OR=5.6, CI=3.4, 9.4). Other strong family-related predictors included having a large family size, a delinquent sibling, a nervous or psychiatrically treated mother, poor parental child-rearing behavior (harsh or erratic discipline, parental conflict), the father being uninterested in his children. In terms of the interactions, ASP at age 18 was especially likely when there was both a nervous mother and a large family (62% of boys were antisocial). Similarly, 61% of the boys with poor child-rearing and low school attainment were antisocial (p=0.06).

The Christchurch and Development study by Fergusson et al suggested that exposure to parental separation during childhood was associated with small but detectable increases in risks of adolescent conduct disorder, even after controlling for social and contextual factors (Fergusson et al 1994).

Finally, as CD has both genetic and environmental components, several recent studies have explored the effects of shared and non-shared environmental (i.e. family) factors on
development of CD amongst twin samples. (Simonoff, Pickles et al. 1998; Meyer, Rutter et al. 2000; Loeber, Farrington et al. 2001). For instance, a study of 1,350 twin pairs from Virginia by Meyer et al. found that family discord and maladaptation were associated with a roughly two-fold increase in risk for conduct disorder symptomatology. Although parental conduct disorder was included in the model the environmental mediation effect for family maladaptation remained, but that for family discord was lost (Meyer, Rutter et al. 2000).

**Low Socioeconomic status**

Many studies have found correlations between poverty and crime, although this is highly contextual and not necessarily causal (Bassarath 2001). Low family socioeconomic status seems to be moderately predictive of development of antisocial behaviors among youth. A significant predictive relationship was found between low-level parental occupation (i.e. generally low skilled or requiring low mental complexity) (OR=2.5) and having foreign-born parents (OR=2.8) and conduct disorders among population-based sample of 5-6 year old children from the Netherlands (Kroes, Kalff et al. 2002). Although the findings are rather generalizable to other countries (i.e. U.S.) because the risk factors are “general” to all children, there was a low retention rate from the initial sample (57.5%) and the population of interest was from the south of the province of Limburg that has relatively fewer foreign-born families and more highly educated inhabitants compared to the rest of the country. These risk factors are consistent with those found in other studies focusing on older children (Costello et al. 1996; Velez et al. 1989; Williams et al. 1990).

In the Cambridge study, having low family income (OR=3.5), living in poor housing (OR=2.0), low social class (OR=2.1) or having a large family size (OR=3.6) were significant predictors of APD at age 18 (Farrington 2000). Poverty may be linked to APD either because poor children cannot achieve their aims by legitimate means and hence resort to illegitimate methods, or because crime involves a cost-benefit decision and poor children have less to lose (Farrington, 1996b).

**Deviant Peers**

By adolescence, delinquent peers contribute greatly to the overall spread of antisocial behavior, particularly in the presence of poor relationships with parents (Elliot et al. 1985). A study by Kasen et al showed that having a higher proportion of deviant friends in adolescence, even after adjusting for the effects of school academic performance, as well as age, gender, earlier conduct problems and SES, increased the risk of antisocial personality disorder among youth in upstate New York (Kasen, Cohen et al. 1998). One unit increase in proportion of deviant friendships in school from ages 12 to 18 increased the odds of having APD by 94% (OR=1.94, 95% CI 2.79, 1.36) in young adulthood from ages 19 to 25. Prior conduct problems and adolescent friendships with deviant peers both had independent effects on outcome, despite the association between them (r = .34, p < .001), supporting an additive model of risk prediction.

The Christchurch study by Fergusson et al found that children most at risk of forming deviant peer affiliations are those from socially disadvantaged backgrounds, dysfunctional families, who showed early onset conduct problems and other difficulties (Fergusson and Horwood 1999). Findings from a large birth cohort study that began about two decades ago (Fergusson & Horwood, 1996) support continuities between problem behavior in childhood and both delinquent behavior and deviant peer bonds in adolescence as well as reciprocity between adolescent delinquent behavior and deviant peer bonds. This study further extends
that friendships with deviant peers in adolescence are predictive of later deviancy independent of predisposing conduct problems. The study had several strengths: a randomly selected, representative sample, a lengthy interval between measurement of predictors and young adult deviant outcomes, adjustment of school effects for IQ and SES, and inclusion of potentially relevant predictors in addition to school experiences (in particular, a highly predictive measure of childhood conduct problems with adequate psychometric characteristics).

Tremblay and Vitaro, in several studies emphasized the importance of family factors such as parental monitoring, attachment to parents and boys’ personal factors such as disruptive profiles during childhood to influence effect of peer deviancy on development of delinquency during adolescence (Vitaro et al 2000, Tremblay 1994).

**Academic Achievement/ School Performance**

Do levels of academic achievement and academic aspirations, the emphasis placed on learning, or the degree of conflict in the school setting predict later deviant outcomes independent of disadvantaged SES, low intelligence, predisposing conduct problems, and having many friends who engage in deviant activities or who are not achievement oriented? A study of youth in upstate New York by Kasen et al found that the risk of APD declined significantly with higher academic achievement in school (Kasen, Cohen et al. 1998). For each unit increase in academic achievement, a 51% decrease in odds of APD (OR=0.49, 95%CI: 0.25, 0.94) was observed, controlling for age, gender, SES, earlier conduct problems, deviant and achieving peers. The authors found that academic achievement, academic aspirations, and learning-focused and conflictual school settings were predictive of deviant outcomes independent of age, gender, IQ, SES, childhood conduct problems, and proportion of deviance-oriented or achievement-oriented friends in adolescence. IQ (OR=1.19, 95%CI: 0.81, 1.74) and low SES (OR=1.14, 95%CI: 0.79,1.64) were moderately associated with APD in young adulthood.

The Cambridge study by Farrington also supported that attending a high delinquency rate school, having a low nonverbal IQ and low school attainment at ages 8-10 were also significant predictors of antisocial personality at age 18 (p< .05). Children with low attainment or low nonverbal IQ at ages 8-10 had 3 times higher odds of developing APD at age 18 (Farrington 2000).

**Reading disability**

Williams et al used data from a longitudinal study of child development (N = 698) to examine relationships between early reading attainment and antisocial behavior at ages 7 and 9 years and subsequent reading and delinquent behavior in adolescence. Reading disability at 9 years old, however, predicted conduct disorder at age 15 in boys (Williams and McGee 1994).

Similarly, samples of poor and normal readers were followed through adolescence and into early adulthood to assess continuities in the comorbidity between reading difficulties and disruptive behavior problems. Reading-disabled boys showed high rates of inattentiveness in middle childhood, but no excess of teacher-rated behavior problems at age 14 and no elevated rates of aggression, antisocial personality disorder or officially recorded offending in early adulthood. Reading problems were associated with some increases in disruptive behavior in their teens in girls. (Maughan, Pickles et al. 1996)
Mild Head Injury

A fully prospective longitudinal design tracked a large birth cohort of children with confirmed mild head injury before age 10 as outpatient, inpatient and reference non-injured group. After accounting for several demographic, family, and pre-injury characteristics, the inpatient but not the outpatient group displayed increased hyperactivity/inattention and conduct disorder between ages 10 to 13, as rated by both mothers and teachers. Most cases of mild head injury in young children do not produce any adverse effects, but long-term problems in psychosocial function are possible in more severe cases, perhaps especially when this event occurs during the preschool years (McKinlay A 2002).

Substance Use

Several researchers have found conduct disorders to be correlated with earlier tobacco, alcohol or drug use, particularly before age 12, and conclude that adolescent substance abuse precedes psychiatric disorders (Johnson and Kaplan 1990, Robins 1993, Kandel and Davies 1986). A longitudinal study by Brook et al of predominantly White children ages 1 to 10 from upstate New York followed for 9 years, found a significant relationship between earlier adolescent drug use and later depressive and disruptive disorders in young adulthood, controlling for earlier psychiatric disorder (Brook et al 1997). Alcohol use significantly predicted antisocial personality disorder (APD), controlling for net disorder, as the rate of APD doubled for heavy users compared to nonusers. Marijuana users were 30% more likely to develop APD than nonusers. Tobacco and illicit drug use resulted in 24% and 23% greater odds of APD compared to nonusers, respectively. The control for temporal sequence and baseline measures was highly critical in this study to confidently conclude that psychiatric disorders in fact represent a change resulting from drug use and not merely the earlier impact of disorders on later drug use, which has also been highly cited (Boyle and Offord 1991, Henry et al 1993).

A study by Johnson et al. found that early alcohol use without parental permission was associated with higher levels of conduct problem behaviors by the ages of 10-12 years and higher rates of growth in those behaviors during the transition from late childhood to early adolescence for both boys and girls. The authors suggested that perhaps unsanctioned early alcohol use puts youths on an accelerated pathway of conduct problem behaviors and development of conduct disorder in adolescence (Johnson E.O. et al 1995).

Childhood Psychiatric Disorders

Prior antisocial behavior

Prior antisocial behaviors is probably the strongest predictor of future offending for males (Bassarath 2001). The above study by Kasen et al also concurred that a one standard deviation increase in conduct problems increased the risk of APD in young adulthood, thus indicating a strong long-term association between earlier problematic behavior and later deviancy (Kasen, Cohen et al. 1998). Fischer et al also found that among 146 hyperactive children, with conduct problems in childhood significantly increased the odds of having APD in young adulthood (OR=2.72, 95%CI=1.06,6.96) compared to children without earlier conduct problems, controlling for childhood ADHD (Fischer et al 2002). There are many
studies showing that childhood conduct disorder symptoms predict adult antisocial personality disorder symptoms (e.g. Offort and Bennett 1994, Zoccolillo, Pickles et al 1992, Rutter 1994).

Moreover, the study by Farrington et al found that children classified as having antisocial personality at age 8-10 had 3.8 higher odds of developing APD by age 18 compared to the boys without earlier ASP (Farrington 2000). About 43% of antisocial males at age 8-10 were antisocial at age 18 (compared with 16% of the remainder). West and Farrington (1977) has previously documented that there is a syndrome of antisocial behavior (including aggression, stealing, substance abuse, sexual promiscuity and reckless driving) that arose in childhood and tended to persist in to adulthood. These strong relationships over time suggest that the stability may primarily lie in the individual rather than in the environment. Yet, recently several studies have shown that young adults with APD did not necessarily display symptoms of CD in adolescence or childhood.

**Social Phobia**

Amongst 776 young people in Upstate New York, Pine et al studied the persistence of conduct disorders into young adulthood and its predictability by social phobia. They found that low scores of social phobia scales at time 1 (mean age13.7), perhaps by means of cognitive or biological factors, were predictive of later risk for conduct disorder. There was a significant negative association between social phobia and diagnosis of conduct disorder at age 22, (B=-0.11 (SD=-0.22), p<.01). Gender, age, presence of ADHD and conduct disorder at time 1 were other significant predictors of later risk for conduct disorder (Pine et al 2000).

**Multiple Factors**

Finally, based on the perspective of developmental psychopathology and supported by notions of high comorbidity amongst CD and other disorders, it is highly likely that there is a dynamic interplay between several shared risk and protective factors i.e. individual, peer, family, neighborhood, contributing to developmental of CD. No single factor accurately predicts later conduct problems (Bassarath 2001). Hence amongst others, Fergusson et al. evaluated a host of psychosocial and child factors as predictors of CD using the Christchurch study (Fergusson, Lynskey et al. 1996). Over the course of 21 years among a birth cohort of New Zealand children, the authors assessed noted risk and protective factors for psychiatric disorders such as a measure of family life events, family conflict, family history of offending, early conduct problems, parental attachment, and affiliations with delinquent peers. Additional measures of family social background, maternal depression, family and childhood adversity, parental substance abuse behaviors and self-esteem were also included. In a study assessing shared variation between affective and conduct disorders, the authors noted the strong positive affiliations with early conduct problems (B=0.22) and association with delinquent peers (0.15) and negative “protective” affiliations with child’s cognitive ability (-0.12) and parental attachment (-0.14).

In the Cambridge study, Farrington et al combined multiple risk factors present at age 8-10 among a 411 South London males, to identify a very high risk group of whom two-thirds became antisocial (APD) at age 18 (Farrington 2000). Of these, having a convicted parent, large family size, low intelligence or attainment, and child-rearing factors including a young mother and a disrupted family seemed to be the most important psychosocial
predictors of APD in young adulthood. The youth classified as vulnerable i.e. having at least 3 or more of these risk factors were almost 10 times more likely to have APD at age 18 than non-vulnerable youth. (Farrington 2000).

**Conclusion**

Although, there are numerous studies assessing the role of various risk factors in development of conduct disorders amongst adolescents, our ability to include many excellent studies was limited due to the strict inclusion criteria noted above. Of the literature not included, major topics included studying mechanisms and development pathways to CD, treatment interventions for CD, prevalence of CD or disruptive behaviors, and long-term effects into adulthood of being diagnosed with CD in childhood. Further, of those studies that did evaluate risk factors for CD or APD, the majority were excluded due to a lack of DSM diagnosis (e.g. used CBCL, Rutter Scale), study design (i.e. cross-sectional surveys), or use of a clinic or treatment sample (i.e. substance abusing probands). Several studies utilized prospective population based study samples yet ascertained risk factors for CD/ APD either retrospectively or at the same time as the diagnosis of CD, compromising the temporal causality.

In terms of future research, prospective longitudinal study design are needed in which psychosocial risk factors are measured repeatedly from birth onwards, in a representative sample and in which different potential theoretical linkages between these risk and protective factors can be linked to CD or APD. Although a longitudinal study of a single cohort still cannot control for historical period or exposure effects, it is a necessary step to demonstrating age-related, i.e., developmental, changes. It has also been suggested that intervention experiments are needed in which these factors are targeted in an attempt to reduce early onset of APD or CD. In addition, Elkins et al 1997 suggest that in the future, increased attention may be warranted for individuals with unusually late onsets of severe antisocial behavior in late adolescence or early adulthood because these individuals appear to share many of the same negative characteristics in adulthood as individuals with early onsets prior to adolescence. Moreover, antisocial behavior in girls has been sorely understudied (Zoccolillo, 1993). However, conduct problems are the second most common diagnosis among girls during the teenage years and appear equally stable over time for boys and girls (Robins, 1986). Vitaro et al 1999 suggested that future research should try to distinguish between person or object-oriented subtypes or process mechanisms by which proactive aggression leads to maladjustment. Understanding gender differences in the course and severity of disorder may lead to important information about etiology. Finally, further research is needed to establish the precise causal mechanisms by which these factors affect psychopathological development of psychiatric disorders over time. For instance, Vitaro et al 1999 suggested that future research should try to distinguish between person or object-oriented subtypes or process mechanisms by which proactive aggression leads to maladjustment. Hence, principles of developmental psychopathology should inform the understanding of transactional interactions amongst person-environment leading to development of conduct disorders and antisocial behaviors.

In conclusion, conduct disorder may be the most important social and public health problem in children and adolescent. Literature in the last decade has indeed advanced our knowledge of the various modifiable risk factors. It is imperative that this knowledge of
individual, family and environmental factors is readily utilized for prevention and intervention early on to optimize healthy child and adolescent development.
Flow Chart: **Risk factors for Conduct Disorder/ Antisocial Personality Disorder**

17,804 studies matching the key words
11,974 (Conduct Disorder) and 5,830 (Antisocial Personality)

17,661 excluded for not meeting criteria

143 studies reviewed for inclusion criteria

40 studies excluded due to clinic-based population

33 studies excluded due to an outcome other than CD

8 studies excluded due to literature reviews

24 studies excluded due to non-DSM criteria or instruments (i.e. CBCL)

23 excluded due to study design (i.e. cross-sectional, retrospective data collection)

5 excluded due to other reasons (e.g. non-modifiable risk factors)

15 studies for inclusion
## Risk Factor: Developmental Trajectories

<table>
<thead>
<tr>
<th>Author</th>
<th>Pub. Date</th>
<th>Study Design</th>
<th>Sample Description</th>
<th>Sample Size</th>
<th>No. Of Cases</th>
<th>Outcome Criteria</th>
<th>Outcome Mode of Reporting</th>
<th>Risk Factor</th>
<th>Risk Factor Criteria/ Measure</th>
<th>Risk Factor Mode of Reporting</th>
<th>Adjusted OR/RR (95% CI)</th>
<th>OR/RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cote Sylvana, Zoccolillo M., Tremblay R., Nagin D., Vitaro F.</td>
<td>2001</td>
<td>Prospective Cohort</td>
<td>Kindergarteners in Quebec’s public schools (1) 946 girls, random representative (2) 444 girls with elevated scores on SBQ† Most White, French-speaking, living with biological parents</td>
<td>4,648</td>
<td>1,390</td>
<td>DSM III-R</td>
<td>DISC-2</td>
<td>Developmental Trajectories of Disruptive behavior</td>
<td></td>
<td>Teacher and parent</td>
<td></td>
<td>High Vs. Low 4.46* Medium Vs. Low 2.2</td>
</tr>
<tr>
<td>Cote Sylvana et al</td>
<td>2002</td>
<td>Prospective Cohort</td>
<td>Boys and girls from Quebec’s public schools followed for 7yrs Representative and oversampled disruptive</td>
<td>4,648</td>
<td>1,569</td>
<td>DSM III-R</td>
<td>DISC-2</td>
<td>Development trajectories of behavior over 7 years</td>
<td></td>
<td>Teacher ratings every year for 7 yrs</td>
<td></td>
<td>For boys: Hyperactive: 4.27* (1.8-10.16), Hyperactive, unhelpful: 2.83* (1.07-7.46), Hyper, unhelpful, fearless: 3.93* (1.27-12.17) For girls: Hyperactive, unhelpful: 4.61* (1.31-16.24)</td>
</tr>
</tbody>
</table>

* p<.05

† SBQ: Social Behavior Questionnaire
### Risk Factor: Childhood Behavior (Aggressive, Under-controlled)

<table>
<thead>
<tr>
<th>Author</th>
<th>Pub. Date</th>
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<th>OR/RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitaro F. et al</td>
<td>1998</td>
<td>Prospective Cohort</td>
<td>Kindergarten boys from low-SES areas in Quebec.</td>
<td>1,037</td>
<td>DSM III-R DISC-2 at age 14 to 16</td>
<td>Proactive and Reactive aggressiveness at age 12</td>
<td>Social Behavior Questionnaire at age 12</td>
<td>Teacher Mothers</td>
<td>Social family adversity</td>
<td>Proactive Aggression: 1.27* (1.20, 1.35)</td>
</tr>
<tr>
<td>Caspi et al</td>
<td>1996</td>
<td>Prospective Cohort</td>
<td>Birth cohort from Dunedin, New Zealand, followed at ages 3, 5, 7, 9, 11, 13, 15, 18, and 21 years. (Multidisciplinary Health and Development Study)</td>
<td>961</td>
<td>DSM-III-R DIS</td>
<td>Behavioral styles – inhibited, under-controlled, reserved, well-adjusted, confident</td>
<td>Scales derived from the Collaborative Study on Cerebral Palsy, Mental Retardation, and Other Neurological Disorders of Infancy and Childhood</td>
<td>90-minute testing session by blinded 1 to 10 examiners</td>
<td>Under-controlled: 2.9* (1.1, 8.1)</td>
<td>Social class, gender</td>
</tr>
<tr>
<td>Farrington et al</td>
<td>2000</td>
<td>Prospective Cohort</td>
<td>Males from working-class inner-city area of South London, followed from age 8 into adulthood</td>
<td>411</td>
<td>DSM-III-R APD scales developed according to DSM-III-R; Admin at ages 18 (shown) and 32.</td>
<td>Individual: High neuroticism Unpopular Troublesome Antisocial Vulnerable</td>
<td>Questionnaires, IQ test</td>
<td>Teacher, peer or parent ratings</td>
<td>(see r.f. categories) 2.2* 1.3 3.8* 3.8* 9.7*</td>
<td>Other covariates</td>
</tr>
</tbody>
</table>
### Risk Factor: Maternal Smoking

<table>
<thead>
<tr>
<th>Author</th>
<th>Pub Date</th>
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<th>Sample Description</th>
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<th>No. Of Cases</th>
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<th>Outcome Mode of Reporting</th>
<th>Risk Factor</th>
<th>Risk Factor Criteria/Measure</th>
<th>Risk Factor Mode of Reporting</th>
<th>Adjusted OR/RR (95% CI)</th>
<th>OR/RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fergusson D M, Woodward LJ, Horwood LJ.</td>
<td>1998</td>
<td>Prospective Cohort</td>
<td>Children born in Christchurch New Zealand in mid-1977 followed for 21 years.</td>
<td>1,265</td>
<td>1,022</td>
<td>DSM-IV criteria</td>
<td>At 18 years, a sample questioned of mental health problems using CIDI and self-report delinquency inventory in past 2 years CD symptoms</td>
<td>Maternal Smoking during pregnancy Categories: Non-smoking 1-9 cig/day 10-19 cig/day &gt;=20 cig/day</td>
<td>Mean number of cigarettes smoked per day during pregnancy High correlation between # cigarettes smoked per day across 3 trimesters (r&gt;0.85)</td>
<td>Mothers questioned at birth</td>
<td>&gt;=20 cig/non =0.72 (boys: 1.32, girls:0.28) 10-19/non =0.60 1-9/non =0.47 regr coeff=0.124 (.036) p&lt;.001 Linear increase in boys mean CD score as exposure increased</td>
<td>Maternal age, education, planned pregnancy, childhood sexual abuse, parental use of physical punishment parental criminal behavior</td>
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</tbody>
</table>

### Risk Factor: Low Birth Weight

<table>
<thead>
<tr>
<th>Author</th>
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<th>OR/RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Szatmari P et al.</td>
<td>1990</td>
<td>Ambispective</td>
<td>Low birth weight babies born in Central-Western Ontario matched to five-year-old children from the Ontario Child Health Study</td>
<td>290</td>
<td>82</td>
<td>DSM-III</td>
<td>SDI including items from CBCL and additional items – parent and teacher</td>
<td>Very low birth weight</td>
<td>&lt; 1000 g</td>
<td>Not given</td>
<td>No significant difference in %CD between cases and controls Parent: 2.4% vs. 0.5% Teacher: 1.6% vs. 3.2% (OR not given)</td>
<td>None</td>
</tr>
</tbody>
</table>

*p < .05*
## Risk Factor: Family Structure and Characteristics

<table>
<thead>
<tr>
<th>Author</th>
<th>Pub Date</th>
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<th>OR/RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rowe R et al.</td>
<td>2002</td>
<td>Longitudinal</td>
<td>9-12 yr olds from rural community in North Carolina, 4 waves of data from the Great Smoky Mountains Study</td>
<td>4,500</td>
<td>179</td>
<td>DSM-IV CAPA</td>
<td>Atypical family structure, gender</td>
<td>4.6 (1.5-14.1)</td>
<td>Comorbid depression and anxiety</td>
</tr>
<tr>
<td>Cadoret RJ et al.</td>
<td>1995</td>
<td>Prospective Cohort</td>
<td>Adoptee offspring separated from biological parents at birth with antisocial peers or alcohol abuse problems. Matched cases and controls based on adoption agency, age, sex and age of biological parent at birth.</td>
<td>95 males, 102 females</td>
<td>197</td>
<td>DIS Screening interview by computer</td>
<td>Adverse adoptive home environment (i.e. adoptive parents with marital problems, divorced, separated, had anxiety conditions, depression, substance abuse, legal problems) DIS Screening interview, 3 psychiatrists confirmed diagnoses by institutional records</td>
<td>Standardized model coefficients: Adverse adoptive home env: 0.44* (1.55) Bio parent with antisocial personality: 0.21* (1.23)</td>
<td>Genetic-environment interactions, controlling for biological parents (alcohol or APD)</td>
</tr>
<tr>
<td>Kroes M et al.</td>
<td>2002</td>
<td>Prospective Cohort</td>
<td>All parents of children in 2nd grade of normal kindergarten schools south of province Limburg (the Netherlands)</td>
<td>2,290</td>
<td>452 (93 cases)</td>
<td>DSM-IIR DICA (Dutch) at yr 2</td>
<td>Parental occupation, foreign-born</td>
<td>Yearly survey – self-report</td>
<td>Low-level parental occupation: 2.5 (1.2-5.4) Foreign-born: 2.8 (1.2-6.7)</td>
</tr>
<tr>
<td>Author</td>
<td>Pub Date</td>
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<tr>
<td>Vitaro F. et al</td>
<td>1998</td>
<td>Prospective Cohort</td>
<td>Kindergarten boys from low-SES areas in Quebec.</td>
<td>1,037</td>
<td>677</td>
<td>DSM III-R</td>
<td>DISC-2 at age 14 to 16</td>
<td>Sociofamily adversity score including socioeconomic disadvantage, education levels of parents, mother’s age at birth</td>
<td>Social Behavior Questionnaire at age 12</td>
</tr>
<tr>
<td>Farrington et al</td>
<td>2000</td>
<td>Prospective Cohort</td>
<td>Males from working-class inner-city area of South London, followed from age 8 into adulthood</td>
<td>-</td>
<td>411</td>
<td>DSM-IIIR</td>
<td>APD scales developed according to DSM-IIIR; Admin at ages 18 (shown) and 32.</td>
<td>Socioeconomic: low family income Poor housing Low social class Large family size</td>
<td>Socio-demographic, parental questionnaires, SBQ</td>
</tr>
</tbody>
</table>

*p<.05
### Risk Factor: Deviant Peers

<table>
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<tr>
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<th>Outcome Mode of Reporting</th>
<th>Risk Factor Criteria/ Measure</th>
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<th>OR/ RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kasen, Brook et al</td>
<td>Prospective Cohort</td>
<td>Children in the Community Project, initiated in 1975 with a random sample of families with a child aged 1-10 from two upstate NY; urban, suburban, and rural areas were represented.</td>
<td>976 452 DSM-III-R</td>
<td>APD at 19-25, derived from the self-rated Personality Diagnostic Questionnaire DISC-1- Conduct Problems at 10-16</td>
<td>Deviant and Achieving Friends</td>
<td>At ages 12-18, Deviant friends scale Achieving friends scale</td>
<td>Mothers and youth</td>
<td>APD with 1 unit increase in exposure: Deviant friends: 1.94* (2.79-1.36) Achieving friends: 0.67 (1.04, 0.43)</td>
<td>Age, gender, IQ, SES, conduct problems, and each school scale</td>
</tr>
</tbody>
</table>

### Risk Factor: Substance Use

<table>
<thead>
<tr>
<th>Author</th>
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<th>Risk Factor Criteria/ Measure</th>
<th>Risk Factor Mode of Reporting</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Brook J et al</td>
<td>Prospective Cohort</td>
<td>Randomly selected families from upstate NY- White male and female youths aged 1-10 followed for 9 years</td>
<td>NA 975 DSM-III-R</td>
<td>DISC-1 from mothers and youths (APD during young adulthood)</td>
<td>Drug use during adolescence</td>
<td>DISC-1 Paper and pencil assessment of drug use</td>
<td>Mothers and youth</td>
<td>Tobacco use= 1.24 (1.02-1.51) Alcohol use= 1.27 (1.01-1.63) Marijuana use= 1.30 (1.06-1.59) Illicit drug use= 1.23 (1.06-1.32)</td>
<td>Control for age and gender, earlier APD</td>
</tr>
</tbody>
</table>

*p<.05*
### Risk Factor: Mild Head Injury

<table>
<thead>
<tr>
<th>Author</th>
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<th>Risk Factor</th>
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<th>Risk Factor Mode of Reporting</th>
<th>Adjusted OR/RR (95% CI)</th>
<th>OR/RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>McKinlay A. et al</td>
<td>2002</td>
<td>Prospective Cohort</td>
<td>Children born in Christchurch New Zealand in mid-1977 followed for 21 years.</td>
<td>1,265</td>
<td>DSM III</td>
<td>Avg symptom score from ages 10-13; selected items from Rutter and Connors scale to conform to DSM criteria, Teachers and mothers.</td>
<td>Mild head injury from 0-10 years</td>
<td>Inpatient, outpatient or reference groups; Pre and post injury functioning</td>
<td>Parental interview, teacher report, standardized testing, and medical records</td>
<td>Adjusted Mean (SD) (combined teacher/mother): Inpatient: 28.79 (5.4)* Outpatient: 26.39 (5.0) Reference: 26.43 (4.2)</td>
<td>No ORs</td>
</tr>
</tbody>
</table>

### Risk Factor: Prior Psychiatric Disorders

<table>
<thead>
<tr>
<th>Author</th>
<th>Pub Date</th>
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<tbody>
<tr>
<td>Pine D.S. et al.</td>
<td>2000</td>
<td>Prospective Cohort</td>
<td>Children ages 1-10 in upstate New York households selected in 1975, followed in 1983, 1985, 1992</td>
<td>760</td>
<td>DSM-III</td>
<td>DISC (CD at age 16)</td>
<td>Family Sociopathy</td>
<td>DSM-III using DISC Index of parental sociopathy</td>
<td>Parent and child interviews</td>
<td>1.42 (0.73, 2.75)</td>
<td>Earlier CD symptoms, age, gender, social class, family sociopathy, ADHD, interactions</td>
</tr>
</tbody>
</table>

* p<.05
## Risk Factor: School Experience/ Academic Achievement

<table>
<thead>
<tr>
<th>Author</th>
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<tbody>
<tr>
<td>Kasen, Brook et al</td>
<td>1998</td>
<td>Prospective Cohort</td>
<td>Initiated in 1975 a random sample of families with a child aged 1-10 from two upstate NY counties; representative of urban, suburban, and rural areas.</td>
<td>976</td>
<td>452</td>
<td>DSM-IIIR</td>
<td>APD at 19-25, derived from the self-rated Personality Diagnostic Questionnaire DISC-1- Conduct Problems at 10-16</td>
<td>School Academic Performance, Aspirations IQ Gender</td>
<td>4 scales at ages 12-18: Academic Aspiration scale, Academic Achieve scale, learning focus scale (school structure) Quick test of intelligence (IQ)</td>
<td>Mothers and youth</td>
<td>APD with 1 unit increase in Exp: Boys: 4.41* (1.87-10.41) Acad Achievmnt: 0.49* (0.25-0.94) Acad Aspiration: 0.69 (0.42-1.14) IQ: 1.19 (0.81-1.74) SES: 1.14 (0.79-1.64)</td>
<td>Age, gender, IQ, SES, conduct problems, deviant friends, achieving friends, and each school scale simultaneously.</td>
</tr>
<tr>
<td>Farrington et al</td>
<td>2000</td>
<td>Prospective Cohort</td>
<td>Males from working-class inner-city area of South London, followed from age 8 into adulthood</td>
<td>411</td>
<td>--</td>
<td>DSM-IIIR</td>
<td>APD scales developed according to DSM-IIIR; Admin at ages 18 (shown) and 32. School: Delinquent school Low nonverbal IQ Low attainment Parent uninterested in education Individual: High neuroticism Unpopular Troublesome Antisocial Vulnerable Questionnaires, IQ test Teacher, peer or parent ratings (see r.f. categories)</td>
<td></td>
<td>2.6* 3.0* 2.1*</td>
<td>Other covariates</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p<.05
## Risk Factor: Multiple Factors

<table>
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<tr>
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<th>Outcome Mode of Reporting</th>
<th>Risk Factor Criteria/ Measure</th>
<th>Risk Factor Mode of Reporting</th>
<th>Adjusted OR/RR (95% CI)</th>
<th>OR/RR Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fergusson DM et al</td>
<td>1996</td>
<td>Prospective Cohort</td>
<td>Birth cohort of New Zealand children at age 15-16.</td>
<td>1,265</td>
<td>934</td>
<td>DSM III-R</td>
<td>Parental and self-reports using DIS and SREDS – dichotomized symptoms of CD at 14-15 and 15-16 yrs</td>
<td>Early Conduct Problems (age 8)</td>
<td>Rutter and Conners questions</td>
<td>Parental, teacher, self-reports</td>
<td>At 15-16 years: OR unadj=4.7 (2.8-7.8) OR adj=2.7 (1.5- 5)</td>
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<td></td>
<td></td>
<td>Intelligence (age 8)</td>
<td>WISC-R</td>
<td>Parents asked yearly when child age 11-14, 15</td>
<td>Delinq. peers: 0.15 (OR=1.16) Parental attachment - 0.14 (OR=0.87) Life events: 0.04 (OR=1.04) Early conduct probs: 0.22 (OR=1.25) Cognitive abil: -0.12 (OR=0.89) Family conflict: .05 (OR=1.05) Gender: -.04 (OR=0.96) Multiple: .48 (OR=1.62)</td>
</tr>
</tbody>
</table>

* p<.05
RISK FACTORS FOR CONDUCT DISORDER
FOREST PLOTS
1. Cote et al., 2001. DSM-IIIR criteria for 4,648 kindergarteners in Quebec’s public schools (946 randomly representative girls, and 444 girls with elevated scores on SBQ). Most children were White, French-speaking, and living with their biological parents.

2. Cote et al., 2002. DSM-IIIR criteria for boys and girls from Quebec’s public schools followed for 7 years (representative and oversampled disruptive).

3. Vitaro et al., 1998. DSM III-R criteria for a prospective cohort of 1,037 kindergarten boys from low-SES areas in Quebec.
Family Structure and Characteristics

Risk Factors by Study

Atypical family structure (Boys) 1
Family conflict 2
Foreign-born 3
Life events 2
Low-level parent occupation 3
Parental attachment 3
Sociofamily adversity (adjusted) 4
Sociofamily adversity (unadjusted) 4
Under-controlled 5

Odds Ratio/Relative Risk

1. Rowe et al., 2002. DSM-IV criteria for 1,420 9-12 year olds from a rural community in the Great Smokey Mountains Study (4 waves of data).
4. Vitaro et al., 1998. DSM III-R criteria for a prospective cohort of 1,037 kindergarten boys from low-SES areas in Quebec.
Maternal Smoking During Pregnancy

Prior Psychiatric Disorders

Risk Factors by Study

ADHD

Earlier CD symptoms

Early conduct problems

Family sociopathy

Social phobia

Odds Ratio/Relative Risk

Miscellaneous Factors

RISK FACTORS FOR
ANTISOCIAL PERSONALITY DISORDERS
FOREST PLOTS
Childhood Social Development

Farrington et al., 2000. DSM-IIIR criteria for a prospective cohort of 411 males from a working-class inner-city area of South London, followed from age 8 into adulthood.
Family Structure and Characteristics

Risk Factors by Study

- Behavior problem sibling
- Boys
- Convicted parent
- Delinquent sibling
- Disrupted family
- Large family size
- Low family income
- Low social class
- Nervous mother
- Parent uninterested in ed.
- Poor child rearing
- Poor housing
- Poor supervision
- SES
- Uninterested father
- Young mother

Odds Ratio/Relative Risk

1. Farrington et al., 2000. DSM-IIIR criteria for a prospective cohort of 411 males from a working-class inner-city area of South London, followed from age 8 into adulthood.

2. Kasen et al., 1998. DSM-IIIR criteria for 976 children ages 12-18 in the Community Project, initiated in 1975 with a randomly selected sample of 976 families with a child between 1 and 10 years of age living in two upstate New York counties.
1. Kasen et al., 1998. DSM-IIIR criteria for 976 children ages 12-18 in the Community Project, initiated in 1975 with a randomly selected sample of 976 families with a child between 1 and 10 years of age living in two upstate New York counties.

2. Farrington et al., 2000. DSM-IIIR criteria for a prospective cohort of 411 males from a working-class inner-city area of South London, followed from age 8 into adulthood.
Kasen et al., 1998. DSM-IIIR criteria for 976 children ages 12-18 in the Community Project, initiated in 1975 with a randomly selected sample of 976 families with a child between 1 and 10 years of age living in two upstate New York counties.
Brook et al., 1998. DSM-IIIR criteria for randomly-selected families from upstate New York, White male and female youths aged 1-10 followed for 9 years.
References


