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## Development of conduct problems in girls: Testing theoretical models and examining the role of puberty

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Development of conduct problems in girls: Testing theoretical models and examining the role of  
puberty

A Dissertation

Submitted to the Graduate Faculty of the  
University of New Orleans  
in partial fulfillment of the  
requirements for the degree of

Doctor of Philosophy  
in  
Applied Developmental Psychology

by

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## Abstract

In an attempt to understand girls' involvement conduct problems, this dissertation first reviews two existing theoretical approaches that provide an explanation for the development of conduct problems. Specifically, the available literature on the development and correlates of conduct problems in boys suggests the subtypes of conduct disorder represent two developmental trajectories. The adolescent-onset pathway is associated with deviant peers and few characterological problems, whereas the childhood-onset pathway is associated with emotion regulation deficits, negative parenting, callous and unemotional traits, and neurological deficits. Research also suggests a gender-specific model, the delayed-onset model, for the development of conduct problems in girls. Following this theoretical review, differential predictions made by the competing theoretical models are tested in a community sample of school-aged girls and boys. Participants were 202 children (87 males and 115 females) in grades 5-9. The students ranged in age from 10 to 17 years old ( $M = 13.16$ ). Similar to the total student body, the ethnic breakdown of the sample was as follows: African-American (60%), Caucasian (24%), Hispanic (6%), and Other (5%). Data was also collected from the students' parents and teachers. Results indicated that girls' conduct problems did not follow either model in a consistent manner. Specifically, adolescent-onset conduct problem girls, childhood-onset conduct problem boys, and adolescent-onset conduct problem boys differed from non-conduct problem children but did not differ significantly amongst themselves on study variables (e.g., deviant peer association, hyperactivity/impulsivity, emotional dysregulation, callous/unemotional traits). However, results suggest that gender-specific risk factors should be taken into account when developing theoretical models for girls' conduct problems. For example, early pubertal maturation is a particularly salient risk factor for conduct problems in girls, and pubertal development interacts

with emotion regulation problems to place girls at high risk for deviant behavior. Finally, implications for prevention and intervention as well as future research are discussed.

**KEYWORDS:** conduct problems, girls, development, puberty, pubertal timing, theory, delayed-onset, adolescent-onset, childhood-onset, age of onset, emotion regulation, callous-unemotional

## Introduction

Despite the universal acknowledgement that boys engage in more serious forms of violence and crime than girls, aggressive and antisocial behavior in girls has recently moved to the forefront of research, policy, and programming agendas (Odgers & Moretti, 2002). This is largely due to increasing rates of violent offending among girls. Between 1993 and 2004, arrests of girls have generally increased more (or decreased less) than arrests of boys in most offense categories. Of particular concern are violent crimes. While male juvenile arrest rates for aggravated assault and simple assault fell from the mid-1990s through 2002, rates for girls have remained near their highest levels (Snyder, 2004, 2006). Between 1993 and 2004, increases were seen in the areas of aggravated assault (a 7% increase in girls versus a 29% decrease in boys; Snyder, 2004) and simple assault (31% increase versus 1% decrease; Snyder, 2006). As a result of the recent rise in female aggression and delinquency, there has been a concurrent increase in interest in understanding the development, incidence, and prognosis of aggressive, violent, and antisocial behavior in girls.

The extensive body of research on antisocial behavior currently available has largely been conducted on male samples (Loeber, 1982; McCord, 1979; Moffitt, 2003). This literature is consistent in implicating multiple risk factors in the development of antisocial behavior in youth. These risk factors include cognitive and neuropsychological deficits, personality traits, dysfunctional parenting, low socio-economic status, history of abuse, substance use, and deviant peers to name a few (Farrington, 2002; Frick & Morris, 2004; Loeber, 1991; Moffitt & Caspi, 2001; Patterson, Reid, & Dishion, 1992a; Raine, Reynolds, Venables, & Mednick, 1997). However, research examining the development of antisocial behavior in girls is more limited and there is either an implicit or explicit assumption that the same risk factors operate in the same

manner for both boys and girls. The focus of this dissertation is to examine this assumption. This study will review the literature on the existing developmental models for conduct problems and aggression in boys and examine how well this research appears to generalize to girls. It will then review a gender-specific, competing theoretical model to explain the development of antisocial behavior in girls and outline a study to test predictions made from these competing models.

### *Conduct Problems and Age of Onset*

Research on the development of antisocial behavior in boys suggests that there are several distinct pathways through which boys develop behavior problems. Current research suggests that an early-onset of conduct problems is one of the strongest predictors of continued antisocial behavior into adulthood; these children are at greater risk for continued offending and have a poorer prognosis than those who begin engaging in conduct problems in adolescence (Frick & McCoy, 2001; Hinshaw, Lahey, & Hart, 1993; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). Thus, subtypes (childhood- or adolescent-onset) of conduct disorder have been adopted by the *Diagnostic and Statistical Manual of Mental Disorders 4<sup>th</sup> Edition – Text Revision*, where onset is determined by at least one symptom prior to age 10 (*DSM-IV-TR*; American Psychiatric Association, 2000). This distinction was based on a number of longitudinal studies suggesting coherence of etiology, symptoms, course, and prognosis. Specifically, a number of longitudinal studies have identified two developmental trajectories determined by onset of severe antisocial behavior either in childhood or adolescence (1993b; Moffitt & Caspi, 2001; Moffitt et al., 1996; Moffitt, Caspi, Harrington, & Milne, 2002).

#### *Adolescent-Onset Type*

In the adolescent-onset subtype, antisocial behavior emerges with the onset of puberty

and is thought to be a function of a “maturity gap” (Moffitt, 1993a; Moffitt et al., 2002). Moffitt (1993a) describes this maturity gap as the difference between physical maturity and societal maturity often seen in industrialized societies. Specifically, adolescents reach physical maturity as early as 13 years old, yet they are not considered adults until after 18 or 21 years of age. This differential in domains of maturity is believed to act as a risk factor for engaging in status offenses, which in turn engenders feelings of independence and “adulthood.” In fact, it can be noted that some level of delinquent behavior may be viewed as normative, as some studies suggest that only a small percentage of adolescents report engaging in no delinquent or illegal activities at all (Krueger, Caspi, Moffitt, White, & Stouthamer-Loeber, 1996). However, youths with adolescent-onset conduct disorder are believed to represent an exaggeration of a normal developmental process of identity formation, of which rebellion is an indicator, and affiliate with deviant peers (youth with childhood-onset conduct disorder). Antisocial behavior is first modeled by deviant peers then rewarded and reinforced by its consequences, which are viewed by youths as indicators of maturity: break-down of parental relationships, challenging authority, appearing older (by engaging in status offenses), and tempting fate (Moffitt, 1993a). However, youth with adolescent-onset conduct problems are more likely to engage in minor conduct problems (such as substance use, vandalism, and sexual activity) and are less likely to engage in felonies and violent behavior, compared to the childhood-onset group (Moffitt, 1993a; Moffitt et al., 2002). Moreover, because their pre-delinquent development was relatively normal and healthy (positive parental relationships and no family maladjustment or personality disturbances), the majority of young people who become adolescent-onset delinquents are able to desist from crime when they adopt conventional adult roles (careers, marriage, children, etc.; Moffitt, 1993a; Moffitt et al., 2002). However, Moffitt (2002) suggests that this recovery can be delayed by “snares,” or the

consequences of delinquency: incarceration, a criminal record, substance abuse, teen parenthood, injury, or lack of education.

### *Childhood-Onset Type*

In contrast, youth in the childhood-onset type, are characterized by more aggression, higher rates of cognitive and neuropsychological dysfunction, more disturbances in their autonomic nervous system functioning, and more severe problems of impulse control [often leading to high rates of Attention Deficit Hyperactivity Disorder (ADHD) diagnoses], than children in the adolescent-onset subtype (Kamphaus & Frick, 2002). Moffitt (1993b) suggests that inherited or acquired neuropsychological variation (initially manifested as difficult temperament, subtle cognitive deficits, or hyperactivity) acts as a risk for difficult behavior early in life. This difficult behavior is then exacerbated by a high-risk social environment such as, inadequate parenting, disrupted family bonds, or poverty (Moffitt & Caspi, 2001; Moffitt et al., 1996; Moffitt et al., 2002; Patterson et al., 1992a). The sequence of transactions between difficult child behavior and environmental risk accumulates across development gradually leading to a disturbance in character, with hallmark features of physical aggression and antisocial behavior. In contrast to the adolescent-onset of delinquent behaviors, which is considered relatively temporary and near normative, childhood-onset conduct problems are proposed to be persistent and pathological, reflective of a characterological disturbance (Moffitt & Caspi, 2001).

This distinction between early- and adolescent-onset pathways to conduct problems has been supported in a number of studies (for a review, see Moffitt, 2003). For example, Piquero and Brezina (2001) found that adolescent-onset delinquency is characterized by involvement in rebellious but not aggressive delinquency. Further, rebellious delinquency is accounted for by the interaction between early maturity and the autonomy aspects of peer activities. Piquero (2001)

also found that verbal performance on the WISC was negatively correlated with measures of early-onset offending (violent and non-violent criminal offending, and serious offending). In another study Simons, Wu, Conger, and Lorenz (1994) found differential predictors of delinquency between early- and late-starters. Specifically, for late-starters, quality of parenting predicted affiliation with deviant peers, which in turn was associated with criminal justice involvement. For early-starters, quality of parenting predicted oppositional/defiant behavior. This behavioral orientation, in turn, predicted affiliation with deviant peers and involvement with the criminal justice system. Further, possession of an oppositional/defiant orientation interacted with type of peer group; that is, criminal justice system involvement was highest among youths who were oppositional/defiant *and* who had deviant friends (Simons et al., 1994).

However, research indicates childhood-onset conduct problem youth are not a homogenous group and further distinctions can be made in terms of the types of dysfunctional processes that may be operating. That is, there may be multiple developmental pathways to childhood-onset conduct problems, with each trajectory involving interactions of different correlates. This developmental psychopathology approach to childhood-onset conduct problems arose from evidence that childhood-onset conduct disorder could be divided into two subgroups characterized by different temperament or personality profiles. One subgroup centers on problems in emotional regulation as a defining characteristic to identify one sub-type of conduct disordered youth; the other subgroup focuses on specific affective deficits (i.e., the presence of callous-unemotional traits) as the major defining characteristic of a severe childhood-onset subtype.

#### *Problems in Emotion Regulation and Conduct Problems*

Initial research has shown that a significant proportion (65-90%) of children with

childhood-onset conduct disorder also exhibit severe problems of hyperactivity, impulsivity, and attention (HIA) and can be diagnosed with ADHD (Frick, Barry, & Bodin, 2000; Hinshaw, 1987). While there is clear evidence that comorbid ADHD and conduct problems designates a severe group of conduct problem children at high risk for persistent antisocial behavior, recent research suggests that a primary problem within this group with both ADHD and conduct problems involve problems with emotion regulation. That is, children who have difficulties regulating their emotions are characterized high rates of negative emotional reactivity, low frustration tolerance, reactive aggression, and a propensity to react with high levels of negative emotion to aversive stimuli (Frick & Morris, 2004; Little, Jones, Henrich, & Hawley, 2003). These children also exhibit regulatory deficits in both inhibitory and attention control (as evidenced by high levels of HIA problems), and thus may be over-represented in children with conduct disorder as well as ADHD (Barkley, 1997; Dodge, Lochman, Harnish, Bates, & Pettit, 1997; Shields & Cicchetti, 1998; Waschbusch, 2002).

Children with problems in emotion regulation have a temperamental style characterized by high levels of negative reactivity, which is associated with and predictive of conduct problems (Morris et al., 2002; Rothbart & Ahadi, 1994). Furthermore, their aggressive and antisocial behavior is strongly associated with dysfunctional parenting practices and with deficits in verbal intelligence (Loney, Frick, Clements, Ellis, & Kerlin, 2003; Wootton, Frick, Shelton, & Silverthorn, 1997). They also tend to have a hostile attribution style, resulting in a propensity to be highly reactive to emotional and threatening stimuli and respond aggressively to provocation in social situations (Crick & Dodge, 1996). Furthermore, there is a wealth of literature indicating children with deficits in emotion regulation have: an early-onset of conduct problems, poor impulse control, high levels of anxiety, greater familial criminality, high likelihood of antisocial

persistence, poor adult adjustment, a higher rate of adult antisocial behavior, and are rejected by peers (Farrington, 1991; Farrington, Loeber, & Van Kammen, 1990; Frick, O'Brien, Wootton, & McBurnett, 1994; Loeber, Brinthaup, & Green, 1990; Lynam, 1996, 1998; McBurnett & Pfiffner, 1998; Walker, Lahey, Hynd, & Frame, 1987).

### *Callous-Unemotional Traits and Conduct Problems*

On the other hand, callous-unemotional (C/U) traits refer to a general lack of emotionality, as well as a lack of guilt over misdeeds and a lack of empathy toward others (Frick et al., 2000). Christian, Frick, Hill, Tyler, and Frazer (1997) have found that the presence of C/U traits identified a more severe group of conduct problem children than those with emotion regulation problems but no C/U traits. That is, those high on C/U traits showed higher rates of police contact, parental diagnoses of Antisocial Personality Disorder, and a greater variety of oppositional defiant and conduct disorder symptoms (Christian et al., 1997). Caputo, Frick, and Brodsky (1999) found that, not only were C/U traits associated with early-onset of antisocial behavior, high levels of C/U traits differentiated violent sex offenders from other violent offenders and non-violent offenders. Research has also demonstrated that children with C/U traits show several distinct characteristics from those without C/U traits. For example, the presence of C/U traits is associated with reduced electrodermal responses to distress cues and threatening stimuli (Blair, 1999), as well as deficits in moral reasoning (Blair, Jones, Clark, & Smith, 1997). Other research has suggested that C/U traits are associated with a reward-dominant response style (O'Brien & Frick, 1996) and with higher levels of fearlessness (Barry et al., 2000). C/U traits have also been found to be negatively associated with anxiety (Forth, Kosson, & Hare, 2003; Frick et al., 1994). Furthermore, conduct problems among children with C/U traits appear to be less strongly associated with dysfunctional parenting practices and deficits in verbal

intelligence than among those children with emotion regulation problems (Loney, Frick, Ellis, & McCoy, 1998; Wootton et al., 1997).

While Moffitt and others have shown clear distinctions between adolescent- and childhood-onset conduct problems, the above two bodies of research suggest further distinctions can be made within the childhood-onset trajectory. Two distinct developmental pathways to childhood-onset conduct problems have been proposed and each is associated with developmental deficits (Frick & Morris, 2004). First, problems in emotional regulation (including impulsivity, frustration, angry outbursts, and negative reactivity) can place a child at risk for developing severe conduct problems and reactive aggression. Specifically, emotional dysregulation can impair the development of executive functions, social cognitive skills, social adjustment and peer associations, and may result in coercive parental relationships (Crick & Dodge, 1996; Dodge & Frame, 1982; Frick & Morris, 2004; Kochanska, 1997; Patterson et al., 1992a). Second, callous-unemotional traits are associated with low emotional reactivity, which can place a child at risk for problems in conscience development, insensitivity to parental sanctions, and a reward-dominant response style that utilizes aggression instrumentally (Frick et al., 2003b; Frick & Morris, 2004; Kochanska, 1997). See Table 1 for a summary of the differential correlates and mechanisms under each pathway.

In summary, conduct problems in boys follow several distinct trajectories; adolescent-onset offending has been linked to processes of social mimicry and peer influence whereas childhood-onset offending patterns have been linked to pathological social, individual, or family conditions (Moffitt, 1993a). Furthermore, childhood-onset offending can be further differentiated into processes involving either problems in emotion regulation or deficits in conscience development (Frick & Morris, 2004). Although Moffitt and Caspi (2001) propose these

Table 1

*Pathways to Antisocial Behavior*

<b>Childhood-onset</b>	
<b>Callous-Unemotional Traits</b>	<b>Problems in Emotion Regulation</b>
<ul style="list-style-type: none"> <li>• Cold and callous interpersonal style</li> <li>• Both reactive and proactive aggression</li> <li>• Thrill and adventure seeking</li> <li>• Reward dominant response style</li> <li>• Low emotional reactivity</li> <li>• Negative associations with anxiety</li> </ul>	<ul style="list-style-type: none"> <li>• Negative emotional reactivity</li> <li>• Emotional dysregulation</li> <li>• Poor impulse control</li> <li>• High rates of family dysfunction</li> <li>• High rates of reactive aggression</li> <li>• Deficits in verbal intelligence</li> <li>• Hostile attribution style</li> <li>• Associated with anxiety</li> </ul>
<b>Developmental Mechanism</b>	<b>Developmental Mechanism</b>
<p>Low emotional reactivity leads to problems in conscience development, insensitivity to parental sanctions, and a reward-dominant response style that utilizes aggression instrumentally. The transactions between difficult child behavior and environmental risk accumulate across development gradually leading to a disturbance in character, with hallmark features of physical aggression and antisocial behavior.</p>	<p>Problems in emotional regulation are exacerbated by a high-risk social environment involving inadequate parenting, disrupted family bonds, and poverty. The transactions between difficult child behavior and environmental risk accumulate across development gradually leading to a disturbance in character, with hallmark features of physical aggression and antisocial behavior.</p>
<b>Adolescent-Onset</b>	<b>Delayed-onset</b>
<ul style="list-style-type: none"> <li>• Rebellious personality style</li> <li>• Low rates of aggression</li> <li>• Low rates of neuropsychological dysfunction</li> <li>• Low rates of family dysfunction</li> <li>• High rates of internalizing disorders</li> <li>• Crimes symbolizing adult privilege</li> <li>• Lack of violent offenses</li> </ul>	<p>Same as childhood-onset pathways</p>
<b>Developmental Mechanism</b>	<b>Developmental Mechanism</b>
<p>Changes in biological maturity and rebelliousness increase associations with deviant peers. Antisocial behavior is modeled by deviant peers, then rewarded and reinforced by its consequences.</p>	<p>Same as childhood-onset pathways</p>

trajectories apply equally well to the development of antisocial behavior in boys and girls, there is currently a lack of research on these causal pathways and their differential developmental correlates in girls. In fact, there is relative lack of research examining conduct problems in girls in general. The following section will review the available research on conduct problems in female samples before outlining an alternative, gender-specific developmental pathway for girls.

### *Conduct Problems in Girls*

The main reason usually cited for the scarcity of studies investigating antisocial behavior in girls is that there are fewer delinquent and antisocial girls than boys (Robins, Tipp, & Pryzbeck, 1991). Generally, conduct disorder is more prevalent in boys than girls, with a reported ratio of boys to girls of 4:1 (Silverthorn & Frick, 1999). However, it is important to note several significant developmental changes in girls regarding this sex ratio. Keenan and Shaw (1997) report that during the first five years of life, there are almost no sex differences between boys and girls in most types of behavioral dysfunction. However, after age four, the rate of girls' behavior problems decreases while the rate of behavioral problems for boys either increases or stays the same. This leads to the male predominance of behavioral problems seen throughout childhood. Numerous studies have indicated that the sex ratio between girls and boys narrows in adolescence, due to an increase in the number of girls engaging in antisocial behaviors (particularly non-aggressive and oppositional behaviors; Offord et al., 1987). Furthermore, the ratio of male to female crime drops from 4:1 to 2:1 when self-report measures of delinquency are used instead of official statistics (McCabe, Rodgers, Yeh, & Hough, 2004). When using self-report information, girls are as likely as boys to report drug use, school related offenses, property destruction, and violent crimes against family members (McCabe et al., 2004).

There have been numerous attempts to explain the gender differences in rates of

antisocial behavior between girls and boys during the school-age years. Some are based on the assumption that there are true gender differences in the prevalence of the causal factors that lead to antisocial behaviors. However, many of these explanations also suggest that, while it may be less common, when a girl has a predominately male disorder, she has a more severe manifestation (Eme, 1992). This is supported by research indicating that conduct disorder in girls is a very serious and impairing mental health problem. For example, girls with conduct disorder are at higher risk than boys for comorbid internalizing conditions, co-occurring substance abuse, and sexual promiscuity (Keenan, Stouthamer-Loeber, & Loeber, 2005). Research also suggests that girls with conduct problems come from very dysfunctional backgrounds and have serious problems in adjustment, both concurrently and later in development. For example, Fergusson and Woodward (2000) found that girls with conduct problems were significantly more likely a) to come from socially disadvantaged family backgrounds characterized by young motherhood, limited maternal education, and single parenthood; b) to be raised in families characterized by high levels of early punitive parenting, physical punishment, parental change, or parental conflict; c) to report higher rates of comorbid attention problems; d) to perform less well on tests of intelligence; and e) to be raised by parents with problems with offending, alcohol, and substance use (Fergusson & Woodward, 2000). Furthermore, girls with antisocial behavior problems are at increased risk for early mortality, educational under-achievement, criminal offending, psychiatric difficulties, social service involvement, marital dysfunction, teenage pregnancy, sexual victimization, and partner violence (Fergusson & Woodward, 2000). Furthermore, there are significantly higher rates of physical and sexual abuse among antisocial girls compared to both the general population and antisocial boys (Silverthorn & Frick, 1999).

However, other explanations have assumed that the causal factors for antisocial behavior are equally prevalent in boys and girls but that girls manifest gender specific forms of antisocial behavior or that they are subject to gender specific differences in socialization. Research on gender differences suggest that some risk factors may have gender-specific impacts, or may interact with other risk factors in gender-specific ways (Odgers & Moretti, 2002). For example, Keenan and Shaw (1997) suggest the different prevalence in conduct problems may be due to caregivers encouraging more prosocial and internalizing behaviors (e.g. shyness) in their school-aged daughters than in their sons. Other researchers have found that boys and girls may engage in different types of conduct problems and aggression. When compared with girls, boys show higher levels of overt behaviors: cruelty, bullying, destructiveness, weapon carrying and initiating fights (O'Keefe, Carr, & McQuaid, 1998). However, this gender difference decreases when more covert behaviors are examined, such as lying and running away (O'Keefe et al., 1998). Other studies have found that while boys report more overt aggression than do girls, girls and boys report similar levels of relational aggression (Crick, 1996). Overt aggression involves harm to others through physical means, where as relational aggression involves harming others through purposeful manipulation or damage to social relationships (Crick, 1996). Alternatively, antisocial tendencies may manifest in girls as other pathological behaviors, such as somatization symptoms (Lilienfeld, 1992). Still other authors have proposed that it may not be the type of behaviors that are different in boys and girls, but simply that the rate and severity of conduct problems is lower in girls and thus, same-sex norms are essential (Zoccolillo, 1993).

While these studies do explain gender differences in prevalence rates, they do not adequately explain why some girls do engage in similar antisocial behaviors as boys, nor do they explain the *changes* in girls' prevalence rates across development. These issues need to be

explained in any theoretical model of the development of conduct problems for girls.

A number of researchers have argued that the two trajectory model for boys outlined above applies equally well to the development of antisocial behavior in girls, with most girls following the adolescent-onset trajectory (Fergusson & Horwood, 2002; Moffitt & Caspi, 2001). However, there has not been compelling evidence that the two-trajectory model explains both the male predominance of severe antisocial behaviors throughout much of childhood *and* the changes in sex ratios of conduct problems across development (Silverthorn & Frick, 1999). Furthermore, there is evidence that girls with conduct problems have similar risk factors as childhood-onset boys, despite being more likely to have an adolescent-onset of antisocial behaviors. To address these issues, Silverthorn and Frick (1999) suggest a single developmental pathway to conduct problems for girls: a *delayed-onset* (see Table 1 for a summary of the correlates and developmental mechanism involved).

#### *The Delayed-onset Model*

Under the delayed-onset model girls with conduct problems share the same risk factors associated with childhood-onset conduct problem boys. That is, as noted previously, antisocial girls have been shown to come from dysfunctional family environments, have cognitive and neurological dysfunction, and show very poor adult outcomes. However, girls do not begin to exhibit conduct problems until the onset of puberty. Silverthorn and Frick (1999) propose a number of reasons for this delay: first, girls may be reinforced and socialized by parents to express their problems through internalizing behaviors rather than externalizing behaviors in childhood (Keenan & Shaw, 1997). Second, girls may be socialized to adhere to gender stereotypes, where they are discouraged from showing aggressive behaviors (Underwood, 2003). Finally, girls may experience more protective factors in childhood. For example, girls in

elementary school tend to receive more praise, less negative attention and higher grades (Silverthorn, Frick, & Reynolds, 2001). Alternatively, while dysfunctional parenting and deviant peers are risk factors for delinquent behavior, positive parental and social relationships may act as particularly salient protective factors for girls.

The delayed-onset model generates several predictions:

1. First, it maintains that while some girls may begin their offending in childhood, the majority of girls' conduct problems will not begin until they reach puberty.
2. The model further asserts that conduct problem girls (regardless of age-of-onset) will be more similar to childhood-onset, rather than adolescent-onset boys, in terms of familial dysfunction, cognitive and neuropsychological deficit, temperamental vulnerabilities, and interpersonal callousness.
3. Finally, it proposes that there will be no group of conduct problem girls who are analogous to adolescent-onset boys.

The delayed-onset model has generated a number of studies investigating these hypotheses with mixed results. In support of the delayed-onset model, Silverthorn, Frick, and Reynolds (2001) found that adjudicated girls almost always showed an adolescent onset to their severe antisocial behavior but were more similar to childhood-onset boys than adolescent-onset boys in predisposing factors (e.g., dysfunctional family environments, cognitive and/or neuropsychological dysfunction). Specifically, childhood-onset boys and delayed-onset girls had similar numbers of ODD symptoms, similar rates of C/U traits and poor impulse control, and similar variety of crime scores (Silverthorn et al., 2001).

McCabe, Rodgers, Yeh, and Hough (2004) also tested several predictions related to the delayed-onset model for girls with conduct disorder. Although they found that males were

significantly more likely to have childhood-onset conduct disorder than females, close to half of females with conduct disorder also met the criteria for the childhood-onset subtype. McCabe et al. (2004) also concluded that risk factors (such as parent antisocial behavior, parental monitoring, maltreatment history, and parent mental illness) appeared to differentiate between childhood-onset and adolescent-onset groups in a similar way among males and females. This is similar to findings by Tolan and Thomas (1995) who found that early onset (before age 12) relates to higher rates of more serious acts over a longer period of time for boys and girls, but that the contribution is small once psychosocial predictors are considered.

Fergusson and Horwood (2002) examined gender related variations in offending trajectories from adolescence to young adulthood. For all analyses, conducted (total offenses, property offenses, and violent offenses) there was evidence of early-onset chronic offending and adolescent-onset offending for both males and females. What distinguished males and females was not the presence or absence of an early-onset pathway, but rather the fact that far fewer females followed this pathway (with female chronic violent offending being particularly rare). Fergusson and Horwood (2002) found that the majority of female offending (21%) followed an adolescent-limited trajectory, characterized by low rates of conduct problems during middle childhood, a moderate risk of offending in early adolescence, rising to a peak at around the age of 13 years, and declining to a lower risk from age 17 years onward.

White and Piquero (2004) found that 23% of their African-American, female offender sample evidenced an early-onset of delinquent behavior. However, they did find that late-onset female offenders were similar to early-onset male offenders on 9 out of 10 risk factors. What is interesting is that early-onset female offenders were significantly higher than late-onset female offenders on 5 of the 10 risk factors including: SES, school retarded codes, and cognitive

abilities. Furthermore, with the exception of adult offending, female late-onset offenders showed similarities on all risk factors to male late-onset offenders. However, it should be noted that White and Piquero (2004) used an onset age of before or after 13-years to designate childhood- or adolescent-onset. Using this age could conceivably place a number of adolescents in the childhood-onset group.

While there is some evidence in the above studies supporting the delayed-onset model (Silverthorn & Frick, 1999; Silverthorn et al., 2001), there is considerable discrepancy in the literature regarding the average age of onset of conduct problems, as well as the severity, and the persistence of the problems for girls (Fergusson & Horwood, 2002; McCabe et al., 2004). Furthermore, the two-trajectory model that can be seen in boys has not been clearly demonstrated for girls. While childhood-, adolescent-, or delayed-onset may offer useful distinctions for conduct problems for girls, most of the studies investigating these trajectories have not incorporated key temperamental differences (such as C/U traits or deficits in emotion regulation) into their studies. More research is needed investigating whether these individual differences have utility for designating distinct trajectories and subtypes of conduct problem girls. It is also important to note that pubertal timing could explain the considerable confusion regarding the childhood-/adolescent-onset distinction for girls. That is, girls may be classified as “childhood-onset,” based on their chronological age, but developmentally may be more appropriately classified as “adolescent-onset” if they have an early-onset of puberty. Further, although the timing of puberty has been documented as a risk factor for conduct problems in girls, research on developmental pathways to conduct problems has not typically considered the potential role of pubertal timing.

### *Transitional Stress and Pubertal Timing*

There is a significant body of research indicating that the onset of puberty is a salient risk factor for conduct problems in girls (Buchanan, Eccles, & Becker, 1992; Caspi & Moffitt, 1991; Ge, Conger, & Elder, 1996; Simmons & Blyth, 1987). Puberty is a pivotal transition event in the life course; a biological event embedded within cultural, social, and personal significance. The observed increase in female conduct problems during adolescence has led to several theories to explain the role of puberty in the development of conduct problems in girls. The most common cause for the association is that puberty is a disruptive experience that may give rise to psychological disturbance. This *stressful change hypothesis* posits that girls experiencing the transition to puberty will manifest higher levels of distress and behavior problems than pre- or post-pubertal girls (Simmons & Blyth, 1987). This theory rests on the assumption that puberty is, in of itself, a stressful transition and girls will experience the greatest emotional disturbance at the onset of menarche, as it is the most discrete event associated with physical maturation and fertility (Ge et al., 1996).

However, it has been suggested that the psychological and behavioral implications associated with differences in the *timing* of puberty may be more consequential for girls' development than puberty itself (Caspi & Moffitt, 1991). Two alternative hypotheses (*off-time* and *early timing*) are based on research indicating that pubertal timing has a greater influence on emotional and behavioral problems than the simple occurrence of the transition to puberty (Buchanan et al., 1992). The *off-time hypothesis* predicts that both early and late maturing girls will experience more emotional and behavioral difficulties than their on-time maturing peers (Ge et al., 1996). This theory is based on the belief that being off-time can have negative consequences for a girl because she risks negotiating the demands of her new status without the

benefit of those social and institutional structures that support and smooth the way for girls who are on-time (Caspi & Moffitt, 1991). In contrast, the *early timing hypothesis* posits that girls who mature early would manifest the most emotional and behavioral problems because they have not had adequate time to acquire, integrate, and consolidate the adaptive coping skills necessary to traverse adolescence (Ge et al., 1996). Early maturation may also have negative consequences for girls because precocious puberty may trigger an insidious series of social comparisons by peers at a developmental period that is already characterized by heightened vulnerability (Caspi, Lynam, Moffitt, & Silva, 1993). In addition, early-maturing girls may be vulnerable to peer-pressure, because others attribute greater social maturity to them than is warranted by their chronological age (Eichorn, 1975). There have been several tests of the *early timing hypothesis*. For example, longitudinal studies have indicated that early maturing girls are at risk for more negative moods and behaviors, conduct problems in school, lower academic success, substance abuse, norm violations, sexually precocious behavior, interactions with deviant peers, and antisocial behavior than girls who mature on-time or late (Simmons & Blyth, 1987; Stattin & Magnusson, 1990; Susman, Dorn, & Schiefelbein, 2003).

Longitudinal studies that have tested these three competing theories have not found support for either the *stressful change hypothesis* or the *off-time hypothesis*. In two different studies using planned comparisons, late maturing girls manifested significantly fewer emotional and behavioral problems than their early- and on-time maturing counterparts, contrary to both the *stressful change* and *off-time* hypotheses (Caspi & Moffitt, 1991; Ge et al., 1996). However, among the three maturing groups, early maturing girls manifested the highest levels of emotional and behavioral distress across adolescence; supporting evidence for the *early timing hypothesis*. For example, early maturing girls demonstrated more global psychological distress (involving

depression anxiety, hostility, and somatization) than their on-time or late maturing peers (Ge et al., 1996). It is also important to note that, while girls who matured early experienced more adjustment problems than other girls, this was only true for early maturing girls who had a history of behavioral or emotional problems (particularly antisocial, anxious-withdrawn, and HIA problems; Caspi & Moffitt, 1991; Ge et al., 1996). Early-maturing girls with a history of childhood behavioral problems also experienced more adjustment difficulties than their early-maturing peers without a history of behavior problems. They also experienced more behavior problems than their on-time maturing peers with a history of childhood behavior problems. It is interesting to note that early-maturing girls with *no* history of childhood behavioral problems experienced fewer difficulties in adolescence than girls who matured on-time with a history of behavior problems. These results suggest an interaction between childhood behavioral problems and early-onset of puberty for predicting problems in adolescence, including conduct problems (Caspi & Moffitt, 1991).

Considering the above evidence suggesting that early pubertal timing may be an important risk factor for conduct problems in girls, any gender-specific developmental model of conduct problems should incorporate early pubertal timing. However, the delayed-onset model only incorporates the onset of puberty and seems to follow most closely with the *stressful change hypothesis*. That is, under the delayed-onset model, *only* those girls who have preexisting risk factors similar to childhood-onset boys are at risk for developing severe conduct problems. The onset of conduct problems is then associated with the onset of puberty, regardless of the timing of maturation.

Moffitt's adolescent-onset trajectory also would be most consistent the *stressful change hypothesis*; however, the difference from the delayed-onset model is that, for the majority of girls

with conduct problems, the effect of puberty is indirect; that is, the association between puberty and conduct problems is mediated by involvement with delinquent peers (Caspi et al., 1993). Thus, the onset of puberty is associated with involvement with deviant/older peers, which is then related to conduct problems.

The *early-onset hypothesis* seen in Ge, Conger, and Elder (1996), and Caspi and Moffitt (1991) would require a modification to both existing theoretical models. It suggests that while early-onset of puberty may be a stressful pubertal transition, it does not generate uniform behavioral reactions among girls; rather, it accentuates pre-transition differences between them. Thus, those girls who have preexisting vulnerabilities (such as C/U traits and/or emotion regulation problems) and who *also* have an early-onset of puberty are at particularly high risk for severe conduct problems.

More research is needed to investigate the role of puberty and pubertal timing in the development of conduct problems in girls, and how these factors contribute to the developmental models discussed above.

#### *Statement of Problem and Theoretical Model*

In summary, the most well-established developmental taxonomy in the study of conduct problems and aggression is the distinction between childhood- and adolescent-onset antisocial behaviors. While Moffitt and Caspi (2001) maintain that this classification applies equally well to males and females, Silverthorn and Frick (1999) proposed a modification for girls that involves a “delayed-onset” trajectory. However, a limited number of studies have investigated whether the childhood-onset and adolescent-onset trajectories exist for girls, or whether the development of antisocial behavior in girls follows a gender-specific pathway. Thus, in an attempt to understand girls’ involvement in delinquency, this dissertation will test differential predictions from these

two competing theoretical models.

According to Moffit (1993a) and others (such as Fergusson & Horwood, 2002), the processes involved in the development of conduct problems are similar for both boys and girls. That is, there are three developmental pathways in both boys and girls: childhood-onset with emotion regulation problems, childhood-onset with C/U traits, and an adolescent-onset. This model maintains that *very few* girls will have a childhood-onset of conduct problems and most girls will start their conduct problems in adolescence. Moreover, adolescent-onset girls will have fewer contextual and dispositional risk factors than childhood-onset girls, and their conduct problems will be primarily due to associations with deviant peers.

According to the “delayed-onset” model proposed by Silverthorn and Frick (1999), and similar to Moffit’s model, girls generally do not develop conduct problems until adolescence. However, unlike Moffit’s model, girls with conduct problems will have a similar profile of risk factors to that of childhood-onset boys, despite the adolescent-onset to their conduct problems. That is, they will have problems in emotion regulation, come from dysfunctional family environments, have lower verbal IQ, problems in impulse control, a hostile attribution bias, C/U traits, thrill seeking, and higher expectations of positive outcomes for aggression (associated with more instrumental, as well as reactive aggression). It is important to note that while both models predict the existence of two groups of conduct problem girls based on age-of-onset (childhood or adolescent) with few girls falling in the childhood-onset group, they have different predictions regarding the adolescent-onset group. According to the delayed-onset model, there will be no group of girls analogous to the adolescent-onset group of conduct disordered boys, who do not show significant levels of the above mentioned risk factors.

In the current study, predictions made by Moffit’s (1993a) model will be compared to

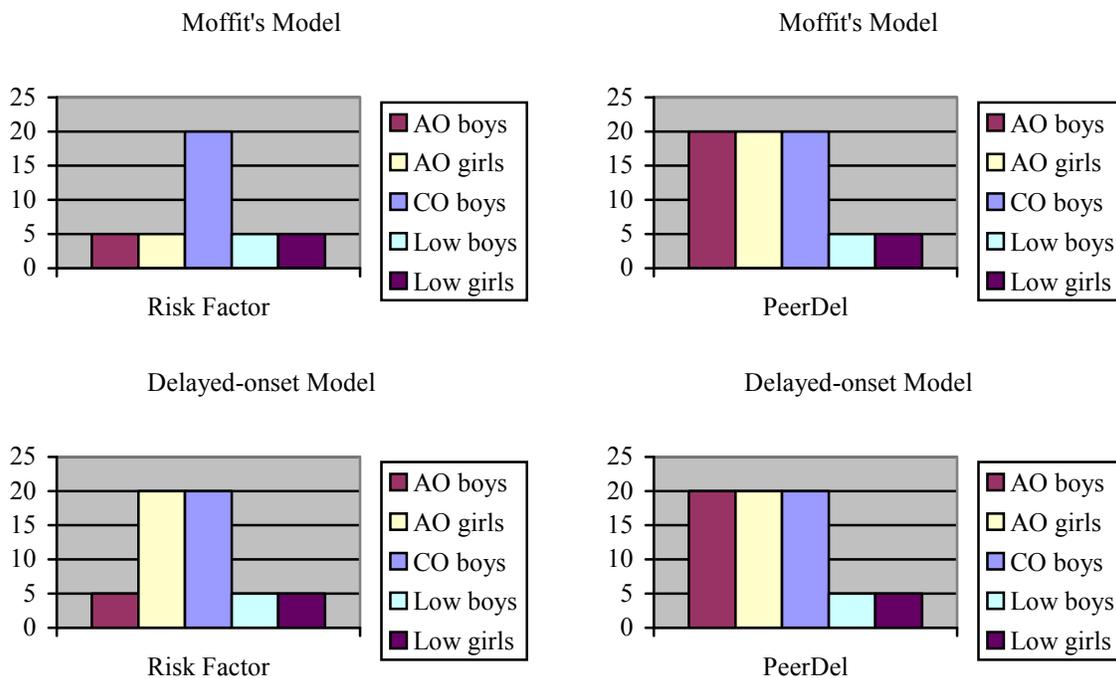
predictions made by the delayed-onset model, specifically:

*Hypothesis One: Model Differences* (see Figure 1)

Moffit's Model: Adolescent-onset girls will differ from youth without conduct problems on deviant peers, adolescent-onset girls will not differ from childhood-onset boys on this variable. Adolescent-onset girls with conduct problems will differ on other individual risk factors from childhood-onset boys with conduct problems. Adolescent-onset girls will not differ from adolescent-onset boys or youth without conduct problems on these factors.

Figure 1

*Differential Predictions of Gender- by-Group Interactions*



Note. AO = adolescent-onset; CO = childhood-onset

Delayed-onset Model: Adolescent-onset girls will differ from youth without conduct problems on deviant peers, adolescent-onset girls will not differ from childhood-onset boys on this variable. Adolescent-onset girls with conduct problems will differ on other individual risk factors from youth without conduct problems and from adolescent-onset boys with

conduct problems; they will not differ from childhood-onset boys with conduct problems.

The role of puberty will also be examined. Moffitt’s model maintains that the majority of girls with conduct problems follow the adolescent-onset trajectory. However, the association between puberty and conduct problems is indirect and the onset of puberty is associated with involvement with deviant/older peers, which is then related to conduct problems. On the other hand, the delayed-onset model suggests that *only* those girls who have preexisting risk factors similar to childhood-onset boys are at risk for developing severe conduct problems. The onset of conduct problems is then associated with the onset of puberty, regardless of the timing of maturation, for girls who possess key risk factors.

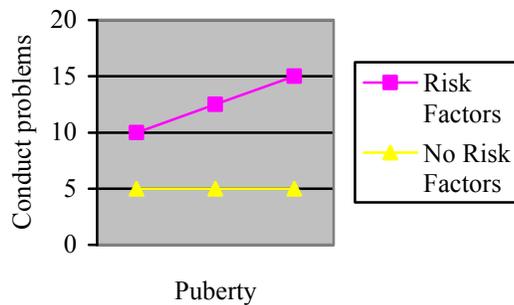
*Hypothesis Two: Role of Puberty*

Moffitt’s Model: For girls, the association between puberty and conduct problems will be mediated by involvement with delinquent peers.

Delayed-onset Model: For girls, C/U traits and/or emotion regulation problems will moderate the association between puberty and conduct problems (see Figure 2).

Figure 2

*Predicted Interaction of Pubertal Development by Risk Factor*



Finally, early pubertal maturation has been shown to put girls at risk for norm violations, sexually precocious behavior, substance abuse, associations with deviant peers, and increased

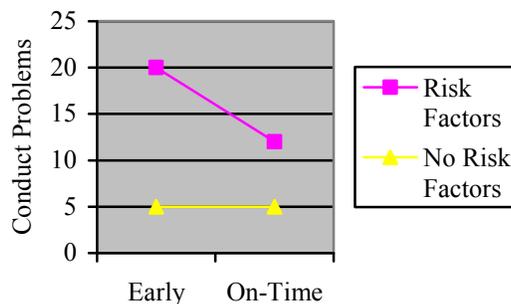
antisocial behavior. Thus, the role of early pubertal timing will also be examined. First, an “alternative hypothesis” will be offered for the role of puberty in Hypothesis Two; that is, pubertal *timing* (not just the onset of puberty) may interact with preexisting risk factors in the development of conduct problems. If the *early-timing hypothesis* is supported, it represents a modification of both existing theoretical models. That is, those girls who have preexisting vulnerabilities (such as C/U and/or emotion regulation problems), who *also* have an early onset of puberty, are at particularly high risk for severe conduct problems (see Figure 3) and thus:

*Hypothesis 3: Role of Early Pubertal Timing*

- a. There will be an interaction between early maturation and girls with preexisting vulnerabilities. That is, C/U traits and/or emotion regulation problems will moderate the association between early maturation and conduct problems.
- b. Early-maturing girls with preexisting vulnerabilities will have more behavior problems than early maturing peers without preexisting vulnerabilities, and on time maturing peers with preexisting vulnerabilities.
- c. Early-maturing girls with no preexisting vulnerabilities will have fewer conduct problems than on-time maturing girls with preexisting vulnerabilities.

Figure 3

*Predicted Interaction of Pubertal Timing by Risk Factor*



## Method

### *Participants*

Participants were 5<sup>th</sup> - 9<sup>th</sup> grade public school children in the New Orleans metropolitan area, their parents, and their teachers. The measures included in this study were collected as part of a larger data collection procedure. Two schools agreed to participate in a study of the timing of puberty and adjustment. Of the 670 eligible children at the two schools, 53% of parents responded to the invitation to participate and two-thirds of respondents consented to the study. Thirty-three students were absent the day of data collection; therefore, the final sample included 202 children (87 males and 115 females), which is approximately 35% of the eligible total student body. The students ranged in age from 10 to 17 years old ( $M = 13.16$ ). Similar to the total student body, the ethnic breakdown of the sample was as follows: African-American (60%), Caucasian (24%), Hispanic (6%), and Other (5%). Data on the 202 participants was also collected from 103 parents and 29 teachers (6 males and 23 females). The teachers' ethnicity was 59% Caucasian and 41% African-American. The parents' ethnicity was African-American (58%), Caucasian (26%), Hispanic (7%), and Other (6%).

### *Procedure*

This dissertation involved the secondary data analysis of data collected under the Louisiana Board of Regents Support Fund R&D, Research Competitiveness Subprogram (RCS) LEQSF (2000-03)-RD-A-45.

In this study, an invitation to participate in the study was sent home to the parents/guardians of all children in grades 5 through 9 at the target schools. Only students who received permission from their parents were allowed to participate. Data were collected from the students during class time after parental permission was obtained. All children had the

procedures explained to them, and were asked if they would like to participate. All participants were informed that they could withdraw from the study at any time. After child assent was obtained, questionnaires were handed out in packets. The instructions for each measure were read aloud and a time limit was set for the completion of each measure. After completion of the student packets, each child received a \$5.00 gift certificate to McDonald's Restaurant.

Individual teachers were contacted and asked to complete questionnaires on each participating student. The packets of questionnaires were left in the teachers' mailboxes at school and were collected within a three-week period. All teachers received a \$50.00 Wal-Mart gift certificate upon completion of the questionnaires. Additionally, all teachers were entered into a raffle to win a \$100.00 Wal-Mart gift certificate and, if they returned the forms early, they were also entered into an additional "early-bird" raffle to win a \$75.00 Wal-Mart gift certificate.

Parents were given questionnaire packets to fill out on their child and received a gift certificate upon completion of the questionnaires.

### *Measures*

#### *Background Information*

Background information was collected from children, parents and teachers. The questionnaires measured general information about the child. This included age, gender, grade, SES, ethnicity, household, and education history (skipped or repeated a grade, special classes, etc.). The parent questionnaire had items pertaining to the child's psychiatric history and the teacher questionnaire had items pertaining to how long the teacher had known the student and the student's current grade in that teacher's class. The questions were either open-ended (school, age, and teacher) or forced choice (grades, ethnicity, income, and household members).

#### *Self-Report of Delinquency (SRD; Elliott, Huizinga, & Ageton, 1985)*

The SRD is a 46-item structured interview that assesses the number and types of delinquent behavior in children. The SRD was developed from a list of all offenses reported in the Uniform Crime Report with a juvenile base rate of greater than 1% (Elliott et al., 1985). For each of 36 delinquent acts (e.g., destroying property, stealing, carrying weapons, selling drugs, hitchhiking, physical fighting, rape, alcohol and drug use, arrest) the youth is asked (a) whether or not he or she has ever engaged in the stated problem behavior, (b) the number of times the act has occurred, (c) the age at which he or she first engaged in the behavior, and (d) whether or not he or she has friends who have engaged in the behavior (see Peer Delinquency subscale). This measure assesses for the occurrence and frequency of specific types of delinquent acts, including Drug Offenses (9 items), Violent Offenses (8 items), Property Offenses (7 items), and Status Offenses (4 items). The General Delinquency scale, which totals the type of delinquent acts, was used in the Conduct Problems composite scale. The Peer Delinquency and General Delinquency scales were computed by taking the mean of the items multiplied by the total number of items (36 in this case), which prorated any missing items. Internal consistency for the General Delinquency scale in this sample was excellent (total scale  $\alpha = 0.88$ ; girls only  $\alpha = 0.85$ ; boys only  $\alpha = 0.87$ ).

*Youth's Inventory-4 (YI-4) and Adolescent Symptom Inventory-4 (ASI-4; Gadow & Sprafkin, 1997; Gadow & Sprafkin, 1999)*

The YI-4 and ASI-4 are youth-, parent-, and teacher-report scales designed to screen for the presence of common *DSM-IV-TR* diagnoses found in adolescents. The YI-4 consists of 128 items, the parent version has 120 items, and the teacher version has 81 items that screen for the presence of ADHD, Conduct Disorder (CD), Generalized Anxiety Disorder (GAD), Oppositional Defiant Disorder, Major Depressive Disorder, as well as other *DSM-IV-TR* symptoms. For the

purposes of this study, only those sections dealing with CD and the hyperactive/impulsive symptoms of ADHD were used. Each symptom on the YI-4 and the ASI-4 is rated on a 4-point scale based on the frequency of its occurrence, ranging from “Never” to “Very Often.” Attachments to the YI-4 and parent-report ASI-4 were also included in this study, where youth and parents were asked to indicate how old they were (or their child was) the first time they engaged in any of the CD symptoms.

Individual items on the YI-4 and ASI-4 were scored using the symptom severity method. Item scores were summed to generate a symptom severity score for each category (Gadow & Sprafkin, 1999). By using this method, it is possible for a youth to receive a score for CD indicating symptoms of high severity, but who may not have a clinically significant symptom cutoff score for that category. The symptom severity scores were used in the Conduct Problem composite scale. In order to account for any missing items, scales were computed by taking the mean of the items multiplied by the total number of items. Subscales were created using the higher score for each item from the three informants (parent, teacher, or child), as recommended by Piacentini, Cohen, and Cohen (1992). The correlations between the raters were not significant for the CD subscale ( $p > 0.05$ ). For the ADHD (Hyperactivity/Impulsivity) subscale, child and teacher ratings were correlated ( $r = 0.19, p < 0.01$ ), teacher and parent ratings were correlated ( $r = 0.41, p < .001$ ), and child and parent ratings were not significantly correlated ( $r = 0.05, ns$ ).

Test-retest reliability of the YI-4 symptom severity scores has been shown to be moderate to high for most symptom categories, with correlations ranging from 0.35 to 0.92 over periods of two to four months in clinic referred samples of youth, aged 11-18 (Gadow et al., 2002). Internal reliability of the YI-4 symptom categories is also sufficient, with alphas ranging from 0.66 to 0.87 (Gadow et al., 2002). Internal consistency for the severity scales (ADHD and CD)

used in this study was satisfactory. Chronbach's alpha coefficients on the YI-4 were as follows: ADHD scale = 0.87 (boys = 0.88 and girls = 0.87); and CD scale = 0.91 (boys = 0.93 and girls = 0.75). Chronbach's alpha coefficients on the teacher-report ASI-4 were as follows: ADHD scale = 0.95 (boys  $\alpha$  = 0.95 and girls  $\alpha$  = 0.95); and CD scale = 0.74 (boys  $\alpha$  = 0.76 and girls  $\alpha$  = 0.73). Chronbach's alpha coefficients on the parent-report ASI-4 were as follows: ADHD scale = 0.94 (boys  $\alpha$  = 0.94 and girls  $\alpha$  = 0.94); and CD scale = 0.74 (boys  $\alpha$  = 0.71 and girls  $\alpha$  = 0.77).

### *Conduct Problems*

Conduct problems were defined as delinquent behaviors (as measured by the General Delinquency scale of the SRD) and conduct disorder symptoms (as measured by YI-4 and ASI-4). An aggregate "Conduct Problem" variable was created. To form this composite, the General Delinquency and Conduct Disorder (highest rater) subscales were converted to z-scores and summed. Z-scores are used in order to account for differences in variance among the scales so that each scale contributes equally to the composites. The correlation between the General Delinquency and Conduct Disorder subscales was  $r = 0.44$ ,  $p < .001$ .

### *Age of Onset of Conduct Problems*

Research on rater reliability of age of onset has demonstrated that adolescents are accurate informants. For example, in a clinic-referred sample of adolescents, both parent report and adolescent self-report showed similar median age of onset for antisocial behaviors; however, adolescent self-report was better than parental report at predicting external criteria (e.g. severity of impairment; Lahey, Gordon, Loeber, Stouthamer-Loeber, & Farrington, 1999). Additionally, Farrington and colleagues (1991) report that self-report will access behaviors that may have not come to the attention of authorities. Thus, the age-of-onset of serious antisocial behavior that was utilized for data analysis in this study was the youngest age reported from two sources: the

parent-report ASI-4 and the self-report YI-4.

There has been little consistency in the literature concerning the operational definition of childhood- versus adolescent-onset. In various studies, childhood-onset has been defined as: the first CD symptom or first police contact occurring at age 9 or younger; behavior problems at age 11 or younger; or CD symptoms at 9 or younger (American Psychiatric Association, 2000; Moffitt et al., 1996; Silverthorn et al., 2001). On the other hand, adolescent-onset has been defined at various ages as well: the first CD symptom or police contact occurring at age 12 or older; behavior problems at age 15 or older; or CD symptoms at age 10 or older (American Psychiatric Association, 2000; Moffitt et al., 1996; Silverthorn et al., 2001). This study utilized the criteria for childhood and adolescent onset that is indicated in the *DSM-IV-TR* (American Psychiatric Association, 2000). Specifically, childhood-onset was defined as onset of at least one symptom of conduct disorder prior to age 10, as indicated on the ASI-4 or YI-4. Adolescent-onset was defined as the absence of any conduct disorder symptoms prior to age 10.

*Antisocial Process Screening Device (APSD; Frick & Hare, 2001)*

The APSD is a 20-item measure of antisocial behavior in children and includes child, parent, and teacher rating forms. Each item is rated on a 3-point scale as either 0 (not at all true), 1 (sometimes true), or 2 (definitely true). The APSD was modeled after the Psychopathy Checklist-Revised (PCL-R), which assesses psychopathic traits in adults (Hare, 1991). Frick, Bodin, and Barry (2000) conducted a factor analysis in a large community sample of children (n = 1136) and found that the APSD can be divided into three distinct factors: (1) poor impulse control (IMP), (2) narcissistic personality features (NAR), and (3) callous and unemotional traits (C/U). The IMP and C/U scales are relevant to the hypotheses in this study. The impulsivity component consists of 5 items (e.g., “acts without thinking,” “does not plan ahead”) measuring

an impulsive interpersonal style. The C/U scale consists of 6 items (e.g., “does not show emotions,” “is not concerned with the feelings of others”) measuring a callous and unemotional interpersonal style. The C/U scale has been shown to identify a distinct subgroup of children with conduct problems that are more severe than other children with conduct disorder (Christian et al., 1997). The C/U scale has also identified children with conduct problems who show characteristics consistent with the construct of psychopathy (Loney et al., 2003).

Child self-report, parent-report, and teacher-report ratings on the APSD were used to measure antisocial features of our sample. Internal consistency for the teacher-report APSD scales was modest (total sample C/U  $\alpha = 0.74$ ; boys C/U  $\alpha = 0.75$ ; girls C/U  $\alpha = 0.73$ ; total sample IMP  $\alpha = 0.71$ ; boys IMP  $\alpha = 0.66$ ; girls IMP  $\alpha = 0.73$ ). Furthermore, two items on the C/U scale (#19 and #20) had item-total correlations below  $r = 0.20$ . However, these items were retained in analyses. Research has indicated that while deleting these items may increase the reliability of the scale, it reduces its convergent validity with external criteria (Poythress et al., 2006). The internal consistency for the parent-report scales was also modest (total sample C/U  $\alpha = 0.67$ ; boys C/U  $\alpha = 0.68$ ; girls C/U  $\alpha = 0.66$ ; total sample IMP  $\alpha = 0.70$ ; boys IMP  $\alpha = 0.74$ ; girls IMP  $\alpha = 0.66$ ). All item-total correlations were above  $r = 0.20$ ; thus all items were retained. Internal consistency for the self-report was poor (C/U = 0.30; IMP = 0.50). Parent- and teacher-report correlations for the C/U and IMP scales were low to moderate ( $r = 0.23$  and  $r = 0.28$ ,  $p < 0.05$ , respectively). Ratings from parents and teachers were combined using the higher score for each item from either informant, as recommended in the APSD manual (Frick & Hare, 2001) and elsewhere (Piacentini et al., 1992).

*Sensation Seeking Scale for Children (SSS-C; Russo et al., 1993)*

The SSS-C is a 26-item self-report measure of sensation seeking behavior in children.

The scale consists of three subscales: (1) Thrill and Adventure Seeking (TAS), (2) Drug and Alcohol Attitudes (DAA), and (3) Social Disinhibition (SD). Children are asked to rate their preference for or against sensation seeking behaviors, choosing between items such as, “I’d never do anything that’s dangerous” and “I sometimes like to do things that are a little scary.” In this study only the 12-item TAS subscale was used. Analogous to the adult version, the TAS subscale is most appropriate for measuring the fearlessness component of callous and unemotional traits (Frick et al., 2003b). Internal consistency for the TAS subscale in this sample was sufficient (total sample  $\alpha = 0.79$ ; boys  $\alpha = 0.68$ ; girls  $\alpha = 0.82$ ).

*Behavioral Assessment System for Children (BASC; Reynolds & Kamphaus, 1993)*

The BASC is a multi-method, multidimensional behavior rating scale designed to evaluate a broad range of both adaptive and maladaptive behaviors in children ages 2 ½ to 18 years of age. The BASC includes a self-report scale (SRP), a teacher rating scale (TRS), and a parent rating scale (PRS). The TRS and PRS have 138 and 126 items, respectively, which are descriptions of observable positive and negative behaviors. The SRP has 186 items, which are descriptions of positive and negative personality traits, thoughts, attitudes, and feelings. Items are answered on a Likert-type scale, where 0 = Never, 1 = Sometimes, 2 = Often, and 3 = Almost always.

For this study, teacher- and self-report was used, as the parent-report BASC was available for only half of the sample. First, items on the BASC were coded to form subscales measuring problems in emotion regulation. To form the Emotional Dysregulation scale, items were identified that were similar to those used on other scales of emotional dysregulation, particularly items referring to susceptibility to emotional arousal, irritability, and negative affectivity (Mezzich, Tarter, Giancola, & Kirisci, 2001). These items were somewhat different

for the child and teacher versions of the scale and they are listed in Table 2. The items on each scale were summed and higher scores indicate increasing levels of problems in emotion regulation. The internal consistency coefficients for the constructed Emotional Dysregulation scales in this sample were as follows: child total sample  $\alpha = 0.70$ , boys  $\alpha = 0.67$ , girls  $\alpha = 0.70$ ; teacher total sample  $\alpha = 0.78$ , boys  $\alpha = 0.80$ ; girls  $\alpha = 0.77$ . Furthermore, all item-total correlations were 0.20 and above. However, the teacher- and self-reports were not significantly correlated ( $r = 0.05, p > 0.05$ ); thus the two scales were not combined to form a composite scale.

Table 2

*Items on the BASC Emotional Dysregulation Scale*

<b>Item</b>	<b>Emotional Dysregulation - Teacher</b>
15	Is a sore loser
35	Throws tantrums
37	Argues when denied own way
44	Works well under pressure (R)
69	Is easily upset
90	Cries easily

<b>Item</b>	<b>Emotional Dysregulation - Child</b>
6	I like to argue
29	Little things bother me a lot
36	My feelings get hurt easily
54	I get mad at my parents sometimes
84	I feel really “stressed out”
86	When I am angry I throw things
92	I go from happy to mad very fast
116	I sometimes get mad
150	I am sometimes jealous
179	I still have fits of temper

*Note.* (R) = reverse coded.

*Alabama Parenting Questionnaire (APQ; Frick, 1991)*

The APQ measures the five domains of parenting that have been most consistently related to conduct problems: poor monitoring/supervision, involvement, inconsistent discipline, corporal punishment, and positive reinforcement. The youth self-report of the APQ consists of

42 items that assess the frequency of different parenting practices and uses 3 to 10 items to assess each construct. On the global report forms, items are rated on a frequency scale of 1 to 5, where 1 = Never and 5 = Always. Item scores are used to construct five different parenting subscales: Parental Involvement, Positive Reinforcement, Poor monitoring/Supervision, Inconsistent Discipline, and Corporal Punishment. The sum of the responses of the items in a particular domain composes each subscale score. The child self-report scales of the APQ demonstrated adequate reliability and only minimal correlation with social desirability in a clinic-referred sample of youth ages 6-13 years (Shelton, Frick, & Wootton, 1996). In a clinic referred sample, Inconsistent Discipline, Corporal Punishment, Parental Involvement subscales were most strongly predictive of conduct problems in school-aged children (Frick, Christian, & Wootton, 1999).

The internal consistency of the APQ subscales was assessed. The Chronbach's alpha coefficients were as follows: Parental Involvement – Mom (total sample  $\alpha = 0.86$ ; boys  $\alpha = 0.82$ ; girls  $\alpha = 0.89$ ); Parental Involvement – Dad (total sample  $\alpha = 0.88$ ; boys  $\alpha = 0.83$ ; girls  $\alpha = 0.91$ ); Positive Parenting (total sample  $\alpha = 0.85$ ; boys  $\alpha = 0.80$ ; girls  $\alpha = 0.88$ ); Poor Monitoring and Supervision (total sample  $\alpha = 0.60$ ; boys  $\alpha = 0.58$ ; girls  $\alpha = 0.61$ ); Inconsistent Discipline (total sample  $\alpha = 0.55$ ; boys  $\alpha = 0.58$ ; girls  $\alpha = 0.52$ ); Corporal Punishment (total scale  $\alpha = 0.63$ ; boys  $\alpha = 0.55$ ; girls  $\alpha = 0.68$ ). The item-total correlations on the Poor Monitoring and Supervision, Inconsistent Discipline, and Corporal Punishment scales were examined. Two items on the Poor Monitoring and Supervision scale and one item on the Inconsistent Discipline scale were found to have item-total correlations below  $r = 0.20$ . However, removing those items did not increase the internal consistency of the scales, thus all items were retained.

Past research has indicated that the parenting constructs can be combined into two

composites: a Positive Parenting composite involving the Parental Involvement and Positive Parenting scales and a Negative Parenting composite involving the Poor Monitoring and Supervision, Inconsistent Discipline, and Corporal Punishment scales (Frick et al., 1999; Frick, Kimonis, Dandreaux, & Farrell, 2003c; Shelton et al., 1996). To form these composites, subscales were converted to z-scores and summed. Z-scores are used in order to account for differences in variance among the scales so that each scale contributes equally to the composites.

The correlation between Parental Involvement and Positive Parenting subscales was  $r = 0.68, p < .001$ . The correlations between the negative parenting scales were as follows: Inconsistent Discipline and Poor Monitoring,  $r = 0.49, p < .001$ ; Inconsistent Discipline and Corporal Punishment,  $r = 0.10, ns$ ; Corporal Punishment and Poor Monitoring,  $r = 0.26, p < .001$ .

*Physical Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988)*

The PDS is a 9-item questionnaire designed to assess pubertal developmental status and provide similar information to the Tanner Growth Ratings (Petersen et al., 1988). Questions pertain to general physical development, such as growth spurts, development of body hair, and skin changes. There are also gender specific questions such as breast development and menstruation for girls, and voice changes and facial hair for boys. Items are rated on a scale of 1 to 4, with 1 indicating that changes have not yet begun and 4 indicating that changes are completed. Youth are also asked to rate their development as compared to other children their age, where 1 = Much earlier; 2 = Somewhat earlier; 3 = About the same; 4 = Somewhat later; and 5 = Much later. Questions address specific information such as height, weight, and age when physical changes began. Because menarche was originally coded as 1 = No and 2 = Yes, it was recoded as 1 = No and 4 = Yes to create a comparable metric to the other items on the scale

(McBride, Paikoff, & Holmbeck, 2003). To determine pubertal development for girls, the items regarding breast development, body hair growth, and menarcheal status were averaged ( $M = 2.96$ ,  $SD = 0.85$ , Chronbach's  $\alpha = 0.66$ ; Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Davison, Susman, & Birch, 2003; Petersen et al., 1988). Following the method used in other studies, a PDS score  $\geq 2.5$  was used to identify those who had entered puberty (Davison et al., 2003; Petersen et al., 1988).

Pubertal timing is a relative concept indicating whether individual physical development occurs earlier than, at the same time as, or later than that of his or her same-gender and same-age peers (Ge, Kim, Brody, Conger, & Simons, 2003). That is, the appropriate comparison group for determining pubertal timing is one's age-mates within a particular social context rather than a distant regional or national population (Ge et al., 1996). Thus, girls' self-perception of their development, as "Somewhat earlier" and "Much earlier" than their peers, was used to classify early pubertal timing. Using these criteria, 37 girls were classified as having early pubertal timing, and of this 37 all but four had experienced menarche.

The reliability of the PDS was assessed and alpha coefficients ranged from 0.68 to 0.83, in a sample of community adolescents (Petersen et al., 1988). Concurrent validity indicates that the PDS is a good approximation of pubertal development, as high correlations were found between the PDS and physician ratings of puberty ( $r = 0.60 - 0.67$ ; Petersen et al., 1988). The self-report PDS was used in this study due to the fact that parental-report of puberty was available for only 54 of the girls. Adolescent girls have been found to be accurate reporters of their menarcheal status, and girls' self-ratings on the body hair growth and breast development questions have been found to correlate strongly with physician ratings (Brooks-Gunn et al., 1987). Internal consistency of the PDS for the current sample of girls was  $\alpha = 0.66$ . The item-

total correlations of the scale were examined; however, all item-total correlations were above  $r = 0.20$  and all items were retained.

### *Deviant Peers*

The Peer Delinquency sub-scale of the SRD was used to assess deviant peer association. On the SRD, participants report on their friends' engagement in a wide variety of disruptive behaviors (e.g. shoplifting, skipping school, selling drugs) in a "yes" or "no" format. The number of behaviors in which there is some level of peer involvement was summed and used to assess the level of deviant peer group affiliation (see Henry, Tolan, & Gorman-Smith, 2001; Lahey et al., 1999; Loeber, Farrington, Stouthamer-Loeber, Moffitt, & Caspi, 1998; Simons, Whitbeck, Conger, & Conger, 1991). Past research has concluded that the Peer Delinquency sub-scale has high internal consistency in a sample of community adolescents ( $\alpha = 0.80$ ; Ge et al., 1996). Internal consistency of the Peer Delinquency sub-scale for the current sample was 0.95 (boys  $\alpha = 0.94$ ; girls  $\alpha = 0.93$ ).

## Results

### *Data Cleaning and Descriptive Statistics*

Univariate graphical techniques and descriptive statistics (SPSS, 2002) were used to evaluate the assumptions underlying the analyses to be carried out (see Table 3). Most variables were normally distributed, where skew and kurtosis were within acceptable limits. A notable

Table 3

### *Descriptive Statistics of Main Study Variables by Gender*

	<b>N</b>	<b>Min</b>	<b>Max</b>	<b>Mean</b>	<b>(SD)</b>	<b>Skew</b>	<b>Kurtosis</b>
<b>Girls</b>							
Peer Delinquency	114	0	36	10.89	(8.52)	.60	-.33
Hyperactivity/Impulsivity	115	0	24	10.21	(5.53)	.49	-.44
APSD IMP (T)	115	0	8	1.93	(2.06)	1.00	.26
APSD C/U (T)	115	0	11	4.31	(2.71)	.40	-.32
APSD IMP (C)	114	0	8	3.84	(2.04)	-.19	-.78
APSD C/U (C)	114	0	8	3.84	(1.87)	.13	-.32
Thrill/Adventure Seeking	115	1	36	17.39	(7.84)	.09	-.58
Emotional Dysregulation (C)	115	0	10	5.45	(2.36)	-.12	-.37
Emotional Dysregulation (T)	115	0	13	3.68	(2.82)	1.35	1.77
Positive Parenting	115	-3.79	4.71	-.13	(2.03)	.23	-.60
Negative Parenting	115	-4.54	6.65	-.27	(2.08)	.37	.31
Conduct Problem	115	-1.87	4.75	-.43	(1.19)	1.36	2.59
Age of Onset	73	5	14	10.40	(2.56)	-.68	-.11
Pubertal Total	115	1	4	2.97	(.85)	-.50	-.94
<b>Boys</b>							
Peer Delinquency	87	0	36	14.13	(9.65)	.34	-.83
Hyperactivity/Impulsivity	87	1	27	10.63	(6.14)	1.03	.71
APSD C/U (T)	87	0	9	2.89	(2.20)	.46	-.20
APSD C/U (T)	87	0	11	5.00	(2.77)	.26	-.61
APSD C/U (C)	86	0	7	4.12	(1.69)	-.42	-.36
APSD C/U (C)	86	1	9	4.45	(1.81)	.25	-.42
Thrill/Adventure Seeking	86	8	35	21.06	(6.39)	.11	-.62
Emotional Dysregulation (C)	87	0	10	4.62	(2.30)	.05	-.66
Emotional Dysregulation (T)	87	1	16	4.17	(3.03)	1.48	2.48
Negative Parenting	87	-3.96	7.20	.36	(2.23)	.54	.57
Positive Parenting	87	-3.61	4.47	.18	(1.57)	.38	.38
Conduct Problem	87	-1.87	9.43	.53	(1.97)	1.92	4.79
Age of Onset	60	5	14	9.25	(2.90)	-.03	-1.22

*Note.* SD = standard deviation; APSD IMP = Antisocial Process Screening Device Impulsivity factor; APSD C/U – Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher-report; (C) = child-report.

exception was the teacher-report emotional dysregulation scale. For this measure, there was large positive skew and kurtosis, due to the low rate of behaviors reported. Using the guidelines suggested by Tabachnik and Fidell (2001), a logarithmic transformation was used on this scale. Since results of analyses using the transformed variable were identical to those using the original variable, results using the original variable are reported here. All variables were examined for outliers within each gender. For girls, one univariate outlier was found on each of the conduct problem composite, teacher-reported emotional dysregulation, and negative parenting composite scales ( $z > 3.29, p < .001$ , two-tailed test). One case was identified by Mahalanobis distance as a multivariate outlier [ $\chi^2(13) < 34.52, p < .001$ ] and was omitted from all subsequent analyses. For boys, one univariate outlier was found on each of the conduct problem composite and teacher-reported emotional dysregulation scales ( $z > 3.29, p < .001$ , two-tailed test). Two outliers were found on each of the conduct disorder symptom and GAD scales ( $z > 3.29, p < .001$ , two-tailed test). However, there were no multivariate outliers identified by Mahalanobis distance [ $\chi^2(13) < 34.52, p < .001$ ], thus all male univariate outliers were retained in analyses.

All variables were examined for significant associations with gender, age, ethnicity, social-economic status, and GPA (see Table 4). In general, the only study variable to be consistently associated with demographic variables was peer delinquency. Peer delinquency was moderately correlated with gender, age, SES, and GPA. Gender and GPA were the two demographic variables significantly correlated with study variables. Specifically, boys scored higher than girls on most study variables, with the exception of teacher-reported impulsivity and self-reported emotional dysregulation. GPA was moderately correlated with teacher-report measures of C/U traits and impulsivity. GPA was also significantly correlated with child-reported hyperactivity/impulsivity and emotional dysregulation.

Table 4

*Descriptive Data on Study Variables and Correlations with Demographic Variables*

	Mean	(SD)	Sex	Age	Race	SES	GPA	Sp.Ed
<b>Total Sample</b>								
Peer Delinquency	12.29	(9.15)	-.18*	.33**	-.02	.16*	-.16*	-.01
Hyperactivity/Impulsivity	10.39	(5.79)	-.04	-.04	.04	-.01	-.19*	.03
APSD IMP (T)	2.34	(2.17)	.22**	-.02	.12	-.04	-.46**	.13
APSD C/U (T)	4.61	(2.75)	-.13	-.02	.04	-.03	-.37**	.05
APSD IMP (C)	3.96	(1.90)	-.07	.11	-.05	.16*	.02	-.12
APSD C/U (C)	4.10	(1.86)	-.16*	.05	.09	.06	-.10	-.03
Thrill/Adventure Seeking	18.96	(7.46)	-.24**	.01	-.29**	-.04	.08	.00
Emotional Dysregulation (C)	5.09	(2.36)	.175*	.10	-.02	.16*	-.04	-.14
Emotional Dysregulation (T)	3.89	(2.92)	-.08	-.12	.06	-.01	-.26**	.18*
Positive Parenting	0	(1.85)	-.08	.24**	.02	.14*	-.07	-.06
Negative Parenting	0	(2.17)	-.15*	-.03	.14	-.07	-.10	-.11
Conduct Problems	0	(1.64)	-.29**	.25**	.03	.13	-.07	.10
Pubertal Total (Girls)	2.97	(.847)	---	.65**	.09	-.01	-.09	-.13
Menarche Age (in months)	145.5	(17.42)	---	.30*	-.02	-.14	.12	.07

*Note.* SD = standard deviation; SES = socio-economic status; GPA = grade point average; Sp.Ed = Special Education; APSD IMP = Antisocial Process Screening Device Impulsivity factor; APSD C/U – Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher-report; (C) = child-report; for Sex 1 = males and 2 = females; for Race 1 = Caucasian and 2 = Minority; for Sp.Ed. 0 = no and 1 = yes; \*  $p < 0.05$ ; \*\*  $p < 0.01$ .

Bivariate Pearson correlations were also conducted to assess the nature of associations among the study measures (see Tables 5 & 6). Many of the variables had significant low to moderate correlations with each other. Notable exceptions include age-of-onset of conduct problems, which was not associated with any other variable, and thrill and adventure seeking, which was only correlated with child-reported APSD impulsivity. Significant correlations varied by gender. Notably, knowing deviant peers was associated with many variables for girls (e.g. hyperactivity/impulsivity, C/U traits, emotional dysregulation, puberty, and negative parenting) but not for boys (where it was associated only with conduct problems and a lack of positive parenting). Girls' thrill and adventure seeking was also significantly associated with

Table 5

*Total Sample Correlations among Main Study Variables*

	Hyp/ Imp	IMP (T)	C/U (T)	IMP (C)	C/U (C)	Thrill Seek	ED (C)	ED (T)	Pos Par.	Neg Par.	Conduct Probs.	Age of Onset
<b>Peer Delinquency</b>	.19**	.13	.08	.31**	.23**	.12	.18*	.10	.21**	.15*	.56**	.03
<b>Hyperactivity/ Impulsivity</b>	1	.33**	.21**	.40**	.23**	.14	.35**	.38**	.11	.38**	.32**	.01
<b>APSD IMP (T)</b>		1	.53**	.04	.18*	.07	.01	.71**	.06	.13	.21**	-.07
<b>APSD C/U (T)</b>			1	.03	.17*	.08	-.07	.49**	.03	.09	.20*	.06
<b>APSD IMP (C)</b>				1	.23**	.18*	.55**	.09	.24**	.39**	.34**	-.01
<b>APSD C/U (C)</b>					1	-.02	.21**	.24**	.39**	.22**	.31**	-.01
<b>Thrill/Advent. Seek</b>						1	.06	-.01	.01	.04	.14	-.02
<b>Emotional Dysreg. (C)</b>							1	.05	.26**	.34**	.12	.01
<b>Emotional Dysreg. (T)</b>								1	.11	.17*	.22*	.14
<b>Positive Parenting</b>									1	.10	.21**	.06
<b>Negative Parenting</b>										1	.29**	-.02
<b>Conduct Probs.</b>											1	.00
<b>Age of Onset</b>												1

*Note.* APSD IMP = Antisocial Process Screening Device Impulsivity factor; APSD C/U – Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher report; (C) = child-report; \*\* correlation is significant at the 0.01 level (2-tailed); \* correlation is significant at the 0.05 level (2-tailed).

Table 6

*Correlations by Gender among Main Study Variables*

	Peer Del	Hyp/ Imp	IMP (T)	C/U (T)	IMP (C)	C/U (C)	Thrill Seek	ED (C)	ED (T)	Pos. Par.	Neg. Par.	Cond Probs	Age Onset	Pub. Girls	Early Mens
<b>Peer Delinquency</b>	----	.24*	.06	.06	.44**	.34**	.19*	.38**	.08	.18	.24*	.60**	.10	.28**	-.29**
<b>Hyper/ Impulsivity</b>	.14	----	.27**	.25**	.46**	.31**	.24**	.35**	.30**	.08	.39**	.43**	.09	.02	-.16
<b>APSD IMP (T)</b>	.12	.40**	----	.55**	.09	.16	.09	.00	.74**	.01	.18	.09	-.01	.01	-.03
<b>APSD C/U (T)</b>	.05	.15	.47**	----	.04	.07	.07	-.04	.54**	.02	.15	.12	-.03	-.02	-.11
<b>APSD IMP (C)</b>	.10	.33**	-.08	-.02	----	.38**	.19*	.60**	.12	.30**	.47**	.49**	.02	.19*	-.23*
<b>APSD C/U (C)</b>	.05	.14	.14	.26*	-.02	----	-.03	.41**	.19*	.42**	.32**	.40**	.00	.19*	-.27
<b>Thrill Adventure</b>	-.06	-.04	-.09	.03	.12	-.12	----	.09	.01	-.05	.07	.27**	.10	.12	-.30*
<b>Emotional Dysreg (C)</b>	.02	.38**	.10	-.06	.52**	.02	.12	----	.07	.32**	.36**	.40**	-.10	.18	-.17
<b>Emotional Dysreg (T)</b>	.10	.47**	.68**	.41**	.02	.28**	-.08	.06	----	.13	.23*	.09	-.14	-.02	-.11
<b>Positive Parenting</b>	.22*	.15	.08	.02	.10	.32**	.06	.21	.06	----	.17	.23*	.09	.09	-.03
<b>Negative Parenting</b>	.00	.37**	.00	-.02	.28**	.04	-.09	.40**	.08	-.02	----	.35**	.05	.21*	-.26*
<b>Conduct Probs.</b>	.51**	.26**	.21*	.23*	.23*	.20	-.11	.03	.30**	.20	.21	----	.16	.32**	-.34**
<b>Age Onset</b>	.01	-.05	-.02	.21	-.06	.05	-.09	.01	-.11	.04	-.01	.00	----	.08	.46**

*Note.* Girls are above diagonal and Boys are below diagonal (----); APSD IMP = Antisocial Process Screening Device Impulsivity factor; APSD C/U – Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher report; (C) = child-report; for Early Menstruation 0 = Early and 1 = On time/late; \*\* correlation is significant at the 0.01 level (2-tailed); \* correlation is significant at the 0.05 level (2-tailed).

hyperactivity/impulsivity and conduct problems but this was not true for boys. Child-reported emotional dysregulation was significantly associated with anxiety, child-reported C/U traits, lack of positive parenting, and conduct problems for girls but not for boys. Furthermore, girls' reported C/U traits were associated with GAD symptoms and not associated with conduct problems. However, this pattern was the opposite for boys; that is, child-reported C/U traits were associated with conduct problems and not anxiety. A lack of positive parenting was associated with impulsivity and conduct problems in girls but not boys, and negative parenting was associated with conduct problems for girls but not boys.

#### *Hypothesis One: Model Differences*

Based on Moffitt's model, adolescent-onset girls with conduct problems were predicted to differ on individual risk factors from childhood-onset boys with conduct problems but not from adolescent-onset boys. Based on the delayed-onset model, adolescent-onset girls with conduct problems were predicted to differ on individual risk factors from adolescent-onset boys with conduct problems, but not from childhood-onset boys. Both of the models predicted that adolescent-onset girls would not differ from either childhood- or adolescent-onset boys on deviant peers. This hypothesis was tested using a series of one-way analyses of variance (ANOVA) comparing conduct problem groups.

To create groups, the conduct problem (CP) composite was divided at the 75<sup>th</sup> percentile of the full sample into two groups (low/average and high CP). Both groups were then divided into boys and girls. Those youth in the high CP group were further divided according to their age-of-onset of first conduct problem, as describe previously. This resulted in 6 groups: low CP girls, low CP boys, early-onset high CP girls, late-onset high CP girls, early-onset high CP boys, and late-onset high CP boys. Because the number of childhood-onset girls was, as expected,

small ( $n = 4$ ; precluding and statistical analyses with this group), only five groups were used to test this hypothesis. The results of these analyses are provided in Table 7.

Table 7

*One-way Analyses of Variance*

<b>Source</b>	<b>Sum of Squares</b>	<b>Df (Num)</b>	<b>Df (Den)</b>	<b>Mean Square</b>	<b>F</b>	<b><i>p</i></b>	<b>Partial Eta<sup>2</sup></b>	<b>Power</b>
Peer Delinquency	2432.57	4	187	608.14	9.07	.000	.16	.99
Hyperactivity/Impulsivity	553.50	4	188	138.38	4.29	.002	.08	.93
APSD IMP (T)	65.46	4	188	16.37	3.73	.006	.07	.88
APSD C/U (T)	49.02	4	188	12.25	1.59	.178	.03	.49
APSD IMP (C)	76.73	4	186	19.18	5.83	.000	.11	.98
APSD C/U (C)	32.46	4	186	8.12	2.37	.054	.05	.68
Thrill & Adventure Seeking	1162.67	4	187	290.67	5.56	.000	.10	.98
Emotional Dysregulation (C)	65.79	4	188	16.45	3.08	.017	.06	.80
Emotional Dysregulation (T)	69.38	4	188	17.34	2.03	.092	.04	.60
Negative Parenting	57.68	4	188	14.42	3.23	.014	.06	.82

*Note.* APSD IMP = Antisocial Process Screening Device Impulsivity factor; APSD C/U – Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher report; (C) = child-report.

Examination of the univariate analyses revealed main effects of CP group on peer delinquency, ADHD hyperactivity/impulsivity symptoms, teacher- and child-reported APSD impulsivity, thrill and adventure seeking, child-reported emotional dysregulation, and negative parenting ( $p < 0.05$ ). Teacher- and child-reported APSD C/U and teacher-reported emotional dysregulation did not have significant main effects ( $p > 0.05$ ). Post-hoc analyses (see Table 8), using Tukey’s procedure largely revealed that the three conduct problem groups differed from non-conduct problem children but did not differ significantly amongst themselves.

Specifically, adolescent-onset CP girls, childhood-onset CP boys, and adolescent-onset CP boys had significantly more delinquent peer affiliates than low/average CP girls, but were not significantly different from each other. This result supports our hypothesis and is consistent with both models. However, adolescent-onset CP girls did not significantly differ from other high CP

Table 8

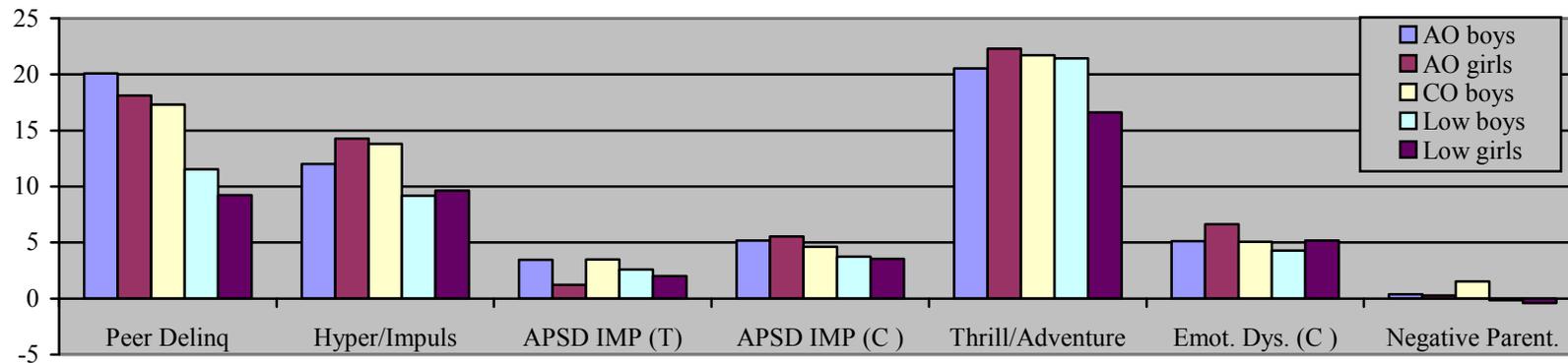
*Pair-wise Comparisons of Means and Standard Deviations across Groups.*

	LateOnset Girls n=13 Mean (SD)	LateOnset Boys n=11 Mean (SD)	EarlyOnset Boys n=17 Mean (SD)	LowCP Girls n = 97 Mean (SD)	LowCP Boys n= 55 Mean (SD)	F df =4	Eta <sup>2</sup>
Peer Delinquency	18.12 (5.82) <sup>ab</sup>	20.10 (8.75) <sup>a</sup>	17.30 (8.67) <sup>ab</sup>	9.22 (7.80) <sup>c</sup>	11.53 (9.01) <sup>bc</sup>	9.07	.16
Hyperactivity/Impulsivity	14.27 (5.49) <sup>ab</sup>	12.00 (7.03) <sup>abc</sup>	13.78 (6.91) <sup>a</sup>	9.65 (5.44) <sup>bc</sup>	9.17 (5.46) <sup>c</sup>	4.09	.08
APSD IMP (T)	1.23 (1.24) <sup>a</sup>	3.45 (1.69) <sup>ab</sup>	3.47 (1.97) <sup>b</sup>	2.00 (2.09) <sup>ab</sup>	2.58 (2.34) <sup>ab</sup>	3.73	.07
APSD IMP (C)	5.54 (1.80) <sup>a</sup>	5.18 (1.72) <sup>ab</sup>	4.63 (1.41) <sup>abc</sup>	3.52 (1.96) <sup>c</sup>	3.73 (1.66) <sup>bc</sup>	2.37	.05
Thrill/Adventure Seeking	22.29 (9.43) <sup>ab</sup>	20.55 (6.70) <sup>ab</sup>	21.72 (6.80) <sup>ab</sup>	16.62 (7.54) <sup>a</sup>	21.43 (6.24) <sup>b</sup>	5.56	.10
Emotional Dysregulation (C)	6.62 (2.72) <sup>a</sup>	5.12 (3.12) <sup>ab</sup>	5.07 (2.47) <sup>ab</sup>	5.18 (2.25) <sup>ab</sup>	4.28 (2.08) <sup>b</sup>	3.08	.06
Negative Parenting	.28 (2.014) <sup>ab</sup>	.39 (2.79) <sup>ab</sup>	1.52 (1.73) <sup>a</sup>	-.40 (2.10) <sup>b</sup>	-.16 (2.08) <sup>b</sup>	3.23	.06

*Note.* Means with the same superscript are not significantly different from each other ( $p < 0.05$ ); APSD IMP = Antisocial Process Screening Device Impulsivity factor; APSD C/U – Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher report; (C) = child-report; (SD) = standard deviation.

Figure 4

*Conduct problem group means on study variables.*



*Note.* APSD IMP = Antisocial Process Screening Device Impulsivity factor; (T) = teacher report; (C) = child-report; AO = adolescent-onset; CO = childhood-onset.

groups on ADHD hyperactivity/impulsivity symptoms or child-reported emotional dysregulation, although they did score significantly higher than low/average CP boys. For child-reported APSD impulsivity, adolescent-onset CP girls were significantly higher than low/average CP boys and girls, but not significantly different from high CP boys. Furthermore, adolescent-onset CP girls did not score significantly different from any of the groups on thrill and adventure seeking or negative parenting. These results are not consistent with either model. Group differences are graphically presented in Figure 4.

The above ANOVA's were then repeated controlling for demographic variables, using GPA, SES, ethnicity, and age as covariates for all analyses. Results indicated that only peer delinquency, hyperactivity/impulsivity, and thrill/adventure seeking maintained significant overall main effects for conduct problems groups after controlling for these demographic variables.

#### *Hypothesis Two: Role of Puberty*

The second hypothesis tested the differential predictions made by each of the models for girls and the role of puberty in the development of conduct problems. Under Moffit's model, the association between puberty and conduct problems was predicted to be mediated by involvement with delinquent peers. Under the delayed-onset model, C/U traits and/or emotion regulation problems were predicted to moderate the association between puberty and conduct problems. To test if deviant peers was a mediator between puberty and conduct problems, as suggested by Moffit's model, the methods outlined by Baron and Kenny (1986) were used. First, puberty was regressed onto the mediator variable, deviant peers. Second, puberty was regressed onto the outcome variable, conduct problems. Third, the mediator, deviant peers, was regressed onto conduct problems. Fourth, puberty was regressed onto conduct problems after controlling for deviant peers. These results are provided in Table 9.

Table 9

*Multiple Regression Analyses Testing the Mediation Role of Deviant Peers*

Conditions for Mediation	Unstandardized Coefficients		$\beta$	t	p	R <sup>2</sup>	R <sup>2</sup> $\Delta$	df1	df2	F $\Delta$
	b	se								
1. 1 Pubertal Total <sup>a</sup>	2.65	.90	.27	2.96	.004	.07	.07	1	112	8.75
2. 1 Pubertal Total <sup>b</sup>	.41	.12	.31	3.50	.001	.10	.10	1	112	12.26
3. 1 Peer Delinquency <sup>b</sup>	.08	.01	.58	7.48	.000	.33	.33	1	112	56.01
4. 1 Peer Delinquency <sup>b</sup>	.07	.01	.53	6.74	.000	.33	.33	1	112	56.01
2 Pubertal Total	.22	.10	.17	2.17	.032	.36	.03	1	111	4.71

Note. (a) Dependent variable is Peer Delinquency; (b) dependent variable is Conduct Problems

From the results provided in Table 9, the significant beta weights demonstrate that the first three conditions for mediation were met. In the fourth regression, pubertal total remained a significant predictor of conduct problems after controlling for deviant peers. However, it is still possible that significant mediation occurred, even when the statistical test continues to be significant after taking the mediator into account (Holmbeck, 2002). Thus, the strength of mediation was tested using the post-hoc probing method outlined in Holmbeck (2002). The standard error of the indirect effect was computed using the following formula:

$$se_{\text{indirect effect}} = [(b_{yx})^2(se_{zy.x})^2 + (b_{zy.x})^2(se_{yx})^2]^{1/2}$$

where  $b$  = the unstandardized beta,  $se$  = standard error,  $yx$  = the prediction of deviant peers from puberty, and  $zy.x$  = the prediction of conduct problems from deviant peers, with puberty in the model. Computations were as follows:

$$\begin{aligned} se_{\text{indirect effect}} &= [(2.649^2)(.01^2) + (.07^2)(.895^2)]^{1/2} \\ se_{\text{indirect effect}} &= [(7.017)(.0001) + (.0049)(.801)]^{1/2} \\ se_{\text{indirect effect}} &= (.0007017 + .003925)^{1/2} \\ se_{\text{indirect effect}} &= (.0046267)^{1/2} \\ se_{\text{indirect effect}} &= .06802 \end{aligned}$$

The standard error of the direct effect was computed using the following formula:

$$z = \frac{b_{\text{indirect effect}}}{se_{\text{indirect effect}}}$$

where  $b$  for the indirect effect is the product of  $b_{yx}$  and  $b_{zy.x}$ . Computations were as follows:

$$z = \frac{(2.649)(.07)}{.068} \quad z = 2.73, p = .006$$

The significant  $z$ -score suggests that there was a significant change in the relation between puberty and conduct problems, when the level of deviant peer involvement was controlled.

Thus, there was partial support for Moffitt's model, in that deviant peers partially mediated the association between girls' pubertal development and conduct problems.

Because pubertal status was highly correlated with age ( $r = 0.65$ ;  $p < .001$ ), the above analysis was repeated while controlling for age. Results can be found in Table 10.

Table 10

*Multiple Regression Analyses Testing the Mediating Role of Deviant Peers between Puberty and Conduct Problems, while Controlling for Age*

Conditions for Mediation	Unstandardized Coefficients		$\beta$	$t$	$p$	$R^2$	$R^2\Delta$	df1	df2	F $\Delta$
	$b$	$se$								
1. Age <sup>a</sup>	1.43	.62	.27	2.32	.022	.11	.11	1	112	13.87
Pubertal Total	.93	1.15	.09	.81	.421	.12	.01	1	111	.65
2. Age <sup>b</sup>	.11	.08	.16	1.36	.177	.09	.09	1	112	10.72
Pubertal Total	.27	.15	.21	1.81	.073	.11	.03	1	111	3.27
3. Age <sup>b</sup>	.08	.06	.12	1.43	.154	.09	.09	1	112	10.72
Peer Delinquency	.07	.01	.54	6.62	.000	.35	.26	1	111	43.78
4. Age <sup>b</sup>	.01	.07	.02	.15	.878	.09	.09	1	112	10.72
Peer Delinquency	.07	.01	.53	6.52	.000	.35	.26	1	111	43.78
Pubertal Total	.21	.13	.16	1.62	.109	.36	.02	1	110	2.61

Note. (a) Dependent variable is Peer Delinquency; (b) dependent variable is Conduct Problems.

Results indicated that the first criterion for mediation was not met. That is, pubertal total was not significantly associated with conduct problems after controlling for age. This suggests that older girls associated with more deviant peers, irrespective of pubertal status.

To test whether C/U traits and/or emotion regulation problems moderated the association between puberty and conduct problems, as suggested by the delayed-onset model, four (both teacher and child-report measures were tested) hierarchical regressions were used (Aiken, 1991).

To test for moderation, the predictor variables were first centered to control for multicollinearity. Second, an interaction variable was created from the cross-product of the centered C/U traits and puberty variables, and from the emotion regulation problems and puberty variables. In all analyses conduct problems was the dependent variable, the moderator was entered in the first block, puberty in the second block, and an interaction term of the moderator by puberty in the third block (Aiken, 1991).

Results (see Table 11) indicated that there were both additive and interactive effects.

Table 11

*Multiple Regression Analyses Testing the Moderating Role of C/U Traits and Emotional Dysregulation on Girls' Pubertal Status and Conduct Problems*

Analyses	Unstandardized Coefficients		B	T	p	R <sup>2</sup>	R <sup>2</sup> Δ	df1	df2	FΔ
	b	se								
1. APSD C/U (T)	.06	.04	.14	1.58	.117	.02	.02	1	112	2.10
Pubertal Total	.40	.12	.31	3.45	.001	.12	.10	1	111	12.61
C/U (T) x Puberty	-.02	.04	-.04	-.43	.665	.12	.00	1	110	.19
2. APSD C/U (C)	.20	.05	.34	3.94	.000	.15	.15	1	112	19.58
Pubertal Total	.33	.11	.25	2.93	.004	.21	.06	1	111	8.65
C/U (C) x Puberty	.01	.06	.01	.13	.894	.21	.00	1	110	.02
3. Emot. Dysreg. (C)	.16	.04	.33	4.01	.000	.14	.14	1	112	18.35
Pubertal Total	.32	.11	.25	2.99	.003	.21	.07	1	111	9.03
Emo. Dys. (C) x Pub.	.15	.05	.24	2.86	.005	.26	.06	1	110	8.20
4. Emot. Dysreg. (T)	.03	.04	.08	.84	.400	.01	.01	1	112	.747
Pubertal Total	.38	.12	.29	3.25	.002	.11	.10	1	111	12.42
Emo. Dys. (T) x Pub.	-.08	.05	-.15	-1.63	.106	.13	.02	1	110	2.66

*Note.* APSD C/U = Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher report; (C) = child-report; AO = adolescent-onset; CO = childhood-onset.

Specifically, girls' pubertal development, child-reported C/U traits, and child-reported emotional dysregulation were significant predictors of conduct problems. There was also a significant interaction between pubertal development and child-report emotional dysregulation. The significant interaction indicated that the relation between girls' pubertal status and conduct problems changed depending on their level of emotional dysregulation. To clarify the nature of

the interaction, one needs to compute two new conditional moderator variables (Aiken, 1991; Holmbeck, 2002). The slopes of the regression pubertal status on conduct problems were calculated at two levels of emotional dysregulation: one standard deviation above the mean (high) and one standard deviation below the mean (low; Aiken, 1991). Results (see Table 12) indicate that puberty remains a significant predictor of conduct problems at high levels of emotional dysregulation, but is not predictive of conduct problems at low levels of emotional dysregulation. The regression lines can then be plotted by substituting high and low values of puberty. These lines can be seen in Figure 5.

Table 12

*Multiple Regression Analyses Testing the Moderating Role of Emotional Dysregulation on Girls' Pubertal Status*

Analyses	Unstandardized Coefficients		B	T	p	R <sup>2</sup>	R <sup>2</sup> Δ	df1	df2	FΔ
	b	se								
1. high Emo. Dys. (C)	.16	.04	.33	4.01	.000	.14	.14	1	112	18.35
Pubertal Total	.68	.16	.52	4.20	.000	.21	.07	1	111	9.03
hiEmDys x Puberty	.15	.05	.35	2.86	.005	.26	.06	1	110	8.20
2. low Emo. Dys. (C)	.16	.04	.33	4.01	.000	.14	.14	1	112	18.35
Pubertal Total	-.03	.17	-.02	-.18	.861	.21	.07	1	111	9.03
loEmDys x Puberty	.15	.05	.37	2.86	.005	.26	.06	1	110	8.20

Note. (C) = child-report.

Figure 5

*Regression Lines for Pubertal Total and Conduct Problems, as Moderated by Emotional Dysregulation.*



Note. b = simple slope

Because pubertal status was highly correlated with age ( $r = 0.65$ ;  $p < .001$ ), the above analysis was repeated while controlling for age. Results can be found in Table 13. Results indicated that, when controlling for age, puberty was no longer predictive of conduct problems. However, child-reported C/U traits and emotional dysregulation remained significant predictors of conduct problems. The interaction between pubertal status and child-report emotional dysregulation also remained significant after controlling for age.

Table 13

*Multiple Regression Analyses Testing the Moderating Role of C/U Traits and Emotional Dysregulation on Girls' Pubertal Status and Conduct Problems, While Controlling for Age*

Analyses	Unstandardized Coefficients		B	T	p	R <sup>2</sup>	R <sup>2</sup> Δ	df1	df2	FΔ
	b	se								
1. Age	.11	.08	.16	1.37	.172	.09	.09	1	112	10.72
APSD C/U (T)	.06	.04	.14	1.59	.114	.11	.02	1	111	2.43
Pubertal Total	.27	.15	.21	1.77	.079	.13	.03	1	110	3.37
C/U (T) x Puberty	-.02	.04	-.04	-.44	.659	.14	.00	1	109	.20
2. Age	.105	.08	.14	1.24	.219	.09	.09	1	112	10.72
APSD C/U (C)	.20	.05	.33	3.87	.000	.21	.12	1	111	16.45
Pubertal Total	.22	.14	.17	1.49	.139	.22	.02	1	110	2.25
C/U (C) x Puberty	.02	.06	.02	.24	.814	.22	.00	1	109	.06
3. Age	.06	.08	.08	.75	.454	.09	.09	1	112	10.72
Emot. Dysreg. (C)	.15	.04	.32	3.79	.000	.18	.10	1	111	13.21
Pubertal Total	.26	.14	.20	1.84	.068	.21	.03	1	110	3.46
Emo. Dys. (C) x Pub.	.15	.05	.24	2.86	.005	.26	.06	1	109	8.16
4. Age	.12	.08	.17	1.45	.149	.09	.09	1	112	10.72
Emot. Dysreg. (T)	.03	.04	.08	.90	.370	.10	.01	1	111	1.11
Pubertal Total	.24	.15	.18	1.57	.120	.12	.03	1	110	3.25
Emo. Dys. (T) x Pub.	-.08	.05	-.15	-1.68	.097	.14	.02	1	109	2.81

Note. APSD C/U = Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher-report; (C) = child-report.

*Hypothesis Three: Role of Early Pubertal Timing*

a) Early maturation and conduct problems

To test whether C/U traits and/or emotion regulation problems moderated the association between early maturation and conduct problems, hierarchical regressions were used, according to

the method outlined by Aiken (1991). As above, both C/U traits and emotion regulation problems were centered to control for multicollinearity; however, pubertal timing was not centered because it was a categorical variable (with early-onset coded “0” and on-time or late-onset coded “1”). Girls’ self-perception of their development as “Somewhat Earlier” and “Much Earlier” than their peers was used to classify early pubertal timing. Thirty-seven girls had an early-onset of puberty and 76 had either on-time or late maturation. An interaction variable was created from the cross-product of the centered C/U traits and early puberty variables, and from centered emotion regulation problems and early puberty variables. In both analyses, conduct problems was the dependent variable. In all regressions, moderators were placed in the first block, early puberty in the second block and the interaction term was placed in the third block (Aiken, 1991). The hypothesis was not supported as, not only was early puberty not a significant predictor of conduct problems, but the addition of the interaction term in the third block did not lead to a significant increase in the amount of variance explained by either regression equation (as seen by a change in  $R^2$  for the analysis with CU traits and a change in  $R^2$  for the analysis with emotion regulation problems).

Because the hypothesis was not supported, several exploratory analyses were conducted. To determine if the operational definition of early puberty had influenced the results, the definition of early puberty was changed. Girls were assigned by their age (in months) at menarche to one of 2 groups: early menstruation and on-time/late menstruation. The early group constituted the first 20% of the distribution in age at menstruation of the current sample. The reported age at menarche (in months) for the whole distribution ranged from 99 to 204 ( $M = 145.45$ ;  $SD = 17.42$ ;  $Mdn = 144$ , or 12 years of age). This is similar to Caspi et. al. (1991), who also took the 20<sup>th</sup> percentile of their cohort ( $M = 155.28$ ;  $SD = 12.12$ ;  $Mdn = 156$  or 13 years of age). The average age at menarche for the early and on-time/late maturing girls in the current

study was 10.3 years old (123.8 months;  $SD = 9.7$ ) and 12.7 years old (151.8 months;  $SD = 13.6$ ), respectively. Girls in our sample were also divided into high and low C/U traits and high and low emotional dysregulation using a median split of the sample to delineate the groups. The groups were then tested for mean differences on conduct problems using 2 x 2 univariate ANOVAs. Table 14 provides the results of these analyses.

Table 14

*One-way Analyses of Variance*

Source	Sum of Squares	Df (Num)	Df (Den)	Mean Square	F	<i>p</i>	Partial Eta <sup>2</sup>	Power
1. Early Menstruation	14.40	1	67	14.40	13.10	.001	.16	.95
APSD C/U (T)	.59	1	67	.59	.53	.468	.01	.11
Early Mens. x C/U (T)	.01	1	67	.01	.01	.937	.00	.05
2. Early Menstruation	7.21	1	67	7.21	6.85	.011	.09	.73
APSD C/U (C)	4.06	1	67	4.06	3.86	.054	.05	.49
Early Mens. x C/U (C)	1.79	1	67	1.79	1.70	.197	.03	.25
3. Early Menstruation	4.73	1	67	4.73	5.34	.024	.07	.62
Emot. Dysreg. (C)	13.81	1	67	13.81	15.58	.000	.19	.97
Early Mens. x ED (C)	2.20	1	67	2.20	2.48	.120	.04	.34
4. Early Menstruation	16.35	1	67	16.35	15.20	.000	.19	.97
Emot. Dysreg. (T)	2.40	1	67	2.40	2.23	.140	.03	.31
Early Mens. x ED (T)	1.27	1	67	1.27	1.18	.282	.02	.19

*Note.* APSD C/U = Antisocial Process Screening Device Callous/Unemotional factor; (T) = teacher-report; (C) = child-report.

Results indicate that there were significant main effects for early menstruation, child-reported emotional dysregulation, and a trend for child-reported C/U traits ( $p = 0.054$ ). There were no significant interactive effects ( $p > 0.05$ ). Pair-wise comparisons indicated that girls who menstruated earlier than their peers had significantly more conduct problems. Girls who had high levels of emotional dysregulation also had significantly more conduct problems. Furthermore, girls who were high in C/U traits had more conduct problems than those girls who were low in C/U traits. Because there was no interaction, these results are more consistent with an additive influence of early puberty and dispositional risk factors in predicting conduct problems in girls.

These additive effects are illustrated in Table 15.

Table 15

*Means and (Standard Deviations) of Conduct Problems for Girls with and without Early Menstruation, Separated by C/U Traits and Emotional Dysregulation*

	<b>Low APSD C/U (C)</b>	<b>High APSD C/U (C)</b>	<b>Low Emot. Dys. (C)</b>	<b>High Emot. Dys. (C)</b>
Early Menstruation	-.14 ( 1.70)	.94 (.86)	-.58 (1.09)	1.08 (.88)
Ave/Late Menstruation	-.57 (.1.23)	-.36 (.85)	-.80 (.94)	-.09 (.95)

*Note.* APSD C/U = Antisocial Process Screening Device Callous/Unemotional factor; (C) = child-report.

## Discussion

The current study contributes to a growing body of research that investigates the development of conduct problems in girls. Moffitt (1993a) has proposed that the two typologies of conduct problems in boys share age of onset as a defining feature, with early-onset serving as the *best discriminator* between the two groups. In prior research, differentiation of childhood-onset and adolescent-onset conduct problems in boys has been important because the differential trajectories have different profiles of comorbidity, life adaptation, and may have different prognoses or respond differently to treatments. Moffitt and Caspi (2001) contend that these differences may be applied to girls in the same manner. However, contrary to this contention, results of the present study indicated that adolescent-onset girls were not significantly different from childhood-onset boys on any study variables: hyperactivity, impulsivity, callous/unemotional traits, thrill and adventure seeking, emotional dysregulation, and negative parenting, or deviant peers.

These results would appear to lend support to the “delayed-onset” model proposed by Silverthorn and Frick (1999), which suggests conduct problem girls will have a similar profile of risk factors to that of childhood-onset boys, despite the adolescent-onset to their conduct problems. Unfortunately, support for the delayed-onset model was not conclusive. Results demonstrated that while the conduct problem girls and boys differed from non-conduct problem children, they did not differ significantly amongst themselves in a consistent manner. That is, adolescent-onset girls were similar to both childhood- *and* adolescent-onset CP boys. Similar results were found by White and Piquero (2004) who reported that late-onset female offenders showed similarities on 9 of 10 risk factors to both male late-onset offenders and male early-onset offenders. Ferguson and Horwood (2002) also found that the relationship between risk factors and offender trajectory group membership were similar for boys and girls (boys and girls did not

differ in terms of the six risk factors investigated). They also found that girls were less predisposed than boys to following later-onset or chronic offending trajectories. In fact, while the majority of female offenders in their study fell into an early-onset group, the highest rates of offending were found in a later-onset adolescent limited group (Fergusson & Horwood, 2002).

Furthermore, while results suggested that the childhood- and adolescent-onset sub-types do not apply well to girls (as suggested by Silverthorn & Frick, 1999), our results did not support the early/late typology in boys either. Our sample of adolescent-onset boys scored similarly to childhood-onset boys on most study variables, which is inconsistent with the current developmental model of conduct problems in boys (Moffitt, 2003). That neither Moffitt's model nor the delayed-onset model was well supported in our sample suggests that further research is needed to refine developmental models of conduct disorder in both genders. However, it should be noted that results of research investigating whether or not risk factors for aggression differ for boys and girls often depends on the type of sample examined. That is, unique characteristics of the current sample or methodological issues may have contributed to the lack of support for the early/late-onset distinction. For example, our study is largely a minority sample, whereas other studies have utilized primarily Caucasian participants. Also, other studies have used varying criteria in the operational definitions of conduct problems and childhood/adolescent-onset. For example, Moffitt et al. (1996) used behavior problems beginning under 11 years to designate childhood-onset and at least 9 illegal acts at 15 years or older to designate adolescent-onset. Silverthorn, Frick, & Reynolds (2001) designated those with first CD symptom or police contact occurring at age 9 or younger as childhood-onset and first CD symptom or police contact at 12 or older as adolescent-onset. However, the current study utilized first delinquent behavior or CD symptom at age 9 or younger for childhood-onset and 10 years or older for adolescent-onset, (as outlined in the *DSM-IV-TR*, American Psychiatric Association, 2000). Current results may have

been more consistent with either model if our definition of early/late-onset mirrored other studies. In fact, Odgers and Moretti (2002) suggest that the theoretical differences suggested by the delayed-onset and Moffitt models may be due to such differences in sampling procedures. Specifically, Odgers and Moretti (2002) indicate that the findings from normative samples suggest that the risk factors for conduct problems do not differ between boys and girls (Fergusson & Horwood, 2002; Moffitt, Caspi, Rutter, & Silva, 2001). Such findings contrast with research from juvenile justice and conduct disorder samples, which have shown that high-risk boys and girls demonstrate the presence of similar types of risk factors, but that girls are more likely to exhibit co-occurring and elevated levels of risk across multiple domains (such as higher levels of psychiatric disorders, maltreatment, and physical/sexual abuse; Odgers & Moretti, 2002). Further research, with both normative and adjudicated/clinical samples, is required to determine whether girls who begin participating in antisocial behavior during adolescence are indeed comparable in risk factors to childhood-onset boys.

The current study also examined risk factors for conduct problems and how they may relate to girls' pubertal development. The hormonal changes that occur during puberty involve several endocrine systems that contribute to observable bodily changes (Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006). The observed increase in female conduct problems during adolescence has led to several theories to explain the role of puberty in the development of conduct problems in girls. For example, the *stressful change hypothesis* posits that girls experiencing the transition to puberty will manifest higher levels of distress and behavior problems than pre- or post-pubertal girls (Simmons & Blyth, 1987). However, despite the fact that gonadal steroids conspicuously rise at the time that sex differences emerge in a broad range of behaviors, hormone-behavior relationships have demonstrated consistently small effects on emotion regulation, mood, and psychopathology (Zahn-Waxler et al., 2006). In one of the

strongest tests of activation effects of gonadal steroids, Finkelstein and colleagues conducted a randomized double-blind, crossover placebo-controlled trial on adolescents with delayed puberty (Finkelstein, Susman, & Chinchilli, 1997). Males were injected with testosterone and females were injected with estrogen in increasing dosages meant to mimic natural puberty. The direct effects of testosterone and estrogen on behavior problems, aggression, mood, and cognition were found to be surprisingly sparse (Zahn-Waxler et al., 2006).

This was supported in our results, which demonstrated that pubertal development itself was not a significant risk factor for girls' conduct problems, once chronological age was accounted for. Furthermore, the association between puberty and conduct problems was not mediated by deviant peers, after controlling for age, which does not support the predictions made by Moffit's model. Rather, older girls had more deviant peers, irrespective of pubertal status. Thus, it is unlikely that the bio-physical processes of puberty are the cause for a dramatic increase in girls' conduct problems during adolescence. More likely, it is socio-environmental correlates of puberty that are associated with the onset of behavior problems in girls. Pubertal development co-occurs with social and psychological changes (Susman et al., 2003). However, our results did indicate that, even after controlling for age, girls' pubertal development can interact with existing emotion regulation problems to put girls at particularly high risk for conduct problems. This is somewhat supportive of the *stressful change hypothesis*. However, it indicates that pubertal development is not a universally stressful event for girls. Rather, it is only a risk factor for those girls with concurrent issues in emotional regulation. This is supported by research that indicates emotional dysregulation is a risk factor for disruptive behavior disorders (Frick, 2004; Shields & Cicchetti, 2001). This result also supports the predictions made by the delayed-onset model.

Furthermore, given the importance of social comparison in this period, pubertal timing is

generally regarded as having a greater influence on emotional and behavioral problems than the simple occurrence of the transition to puberty (Buchanan et al., 1992; Caspi & Moffitt, 1991). Pubertal timing may be conceived of as a measure of an individual's relative development in comparison with expected pubertal maturation at a given age. Usually, pubertal timing is estimated on the basis of comparisons of individual's maturational status within a reference group, usually between subjects in the same age range, same grade, or same class. While the *off-time hypothesis* predicts that both early and late maturing girls will experience more emotional and behavioral difficulties than their on-time maturing peers (Ge et al., 1996), research has not been shown to support this hypothesis in terms of girls' conduct problems. Rather, research supports the *early timing hypothesis*, which posits girls' early maturation leads to behavioral problems (Caspi & Moffitt, 1991; Ge et al., 1996; Simmons & Blyth, 1987). It is believed this association occurs because these young girls have not had adequate time to acquire, integrate, and consolidate the adaptive coping skills necessary to traverse adolescence (Ge et al., 1996).

Based on this research, our third hypothesis tested the role of early maturation in the development of girls' conduct problems. Appropriate comparison group for determining pubertal timing is one's age mates within a particular social context rather than a distant regional or national population (Ge et al., 1996). Thus, girls' perceptions of their own development, compared to their peers, were initially used to classify early pubertal timing. However, defined in such a manner, early puberty was not a significant predictor of conduct problems, contrary to many past studies (Caspi et al., 1993; Ge et al., 2003; Graber, Brooks-Gunn, & Warren, 2006). While an individual's own perception of timeliness in pubertal development is crucial, this perception is typically influenced by the particular peer group which serves as reference group. That is, two young people who are equally mature may perceive themselves as on-time or off-time, depending on the level of maturation of the adolescents with whom they associate (Alsaker,

1992). Such variations may have accounted for our non-significant findings.

Exploratory analyses were conducted using an alternative definition of pubertal timing: girls' age at menarche, relative to our sample. The average age at menarche, then, for the early and on-time/late maturers was 10.3 years old and 12.7 years, respectively. These results suggest that the average early maturing girl experienced menarche during the fifth grade and on-time/late girls during the seventh to ninth grades. Investigation of these groups indicated that an early onset of puberty had an additive influence with dispositional risk factors in predicting conduct problems in girls; that is, both early puberty and emotional regulation problems led to increased risk for conduct problems. This additive influence has been seen in other studies showing early pubertal maturation is related to the development of behavioral and emotional problems only when it occurs in the context of additional risk factors such as a history of behavior problems, stressful life events, or relatively immature cognitive or emotional functioning (Keenan, Loeber, & Green, 1999). These results support the *early-timing hypothesis* and studies indicating early maturing girls have more psychological distress and conduct problems than their on-time or late maturing peers (Caspi et al., 1993; Ge et al., 2003; Graber et al., 2006).

Explanations for these findings suggest that a rapid ascent toward adolescence leads early maturing girls to confront new stressors, environments, norms, and expectations before they are psychologically prepared for such challenges (Ge et al., 1996; Haynie, 2003). Early maturing girls, often isolated from their on-time maturing peers, tend to associate with older adolescents (some of whom may be deviant peers). This increases their emotional distress (Ge et al., 1996), because conforming to the older adolescents' behavioral standards leads early-maturing girls to engage in behaviors that are considered relatively deviant for their actual age (Stattin & Magnusson, 1990). The study results for girls suggest that once they become early maturers (relative to their peers), especially as soon as fifth grade, the social consequences become so

significant that the experience has a lasting effect on their emotional and behavioral functioning (Ge et al., 2003). Interestingly, early maturation has not been found to be a risk factor for boys (Ge et al., 2003). Unlike the pattern for girls, boys' adaptation is only temporarily disrupted when they were in the midst of peak pubertal change – supporting the stressful change hypothesis, rather than the early timing hypothesis. Once they pass this period of peak change, boys appear to “bounce back” (Ge et al., 2003).

Such results lend credence to the argument that there are fundamental differences in development of antisocial behavior among boys and girls (as suggested by Patterson, Reid, & Dishion, 1992b). Our results suggest that early maturation is a significant risk factor for conduct problems in girls. Examining pubertal timing in future studies of the development of girl's conduct problems is important, as early maturation could account for girls' “childhood-onset” of conduct problems, as seen in previous studies (Fergusson & Horwood, 2002; McCabe et al., 2004; White & Piquero, 2004).

#### *Limitations of Study*

Results from the current study need to be interpreted in light of several limitations. First, this study was conducted with a sample of community-based youth, without the inclusion of a clinical comparison group. As a result, the findings cannot be generalized to juvenile offenders or psychiatric populations. Further, although the ethnic breakdown of our sample was representative of youth in the city of New Orleans, it was primarily composed of African-American youth (60%), which may limit the generalizations that can be made to other ethnic groups. Our sample size was also small, which may have affected the power to detect significant associations among variables. That is, our small sample size may have limited our ability to differentiate a small group of severe conduct problem girls with callous/unemotional traits, due to the low base-rate of such individuals in a normative population (Jackson, Rogers, Neumann,

& Lambert, 2002). Under-representation of conduct problem youth might have attenuated the present statistical associations. Future research should incorporate a large enough sample in order to adequately investigate if conduct problem girls are homogenous, or if they, like childhood-onset conduct problem boys, can be broken down further into two sub-groups each with varying developmental correlates and risk trajectories.

A second limitation is the lack of observational measures in the study. For example, a noninvasive measure of pubertal development such as the Pubertal Development Scale was an effective tool for studying a sensitive topic (Ge et al., 2003). However, sole reliance on self-report measures of pubertal development, to a certain extent, may reduce the reliability of the assessment. Future studies should strive to supplement self-report measures with more objective measures such as physician- or nurse-assisted assessments of Tanner stages.

Another methodological limitation is that the Emotional Dysregulation scale was constructed from the BASC for the purpose of this study. Concordant and discriminant validity in school-aged children has not been established for the constructed scale. While significant differences were found on the self-report measure, study findings could have been due to deficits in the scale itself. The possibility of overestimation of statistical associations because of shared method variance must also be taken into account. As a result, these findings need to be replicated using an established measure of emotional dysregulation and multiple informants to control for shared method variance.

Given the cross-sectional nature of the study, causal interpretations regarding associations among conduct problems, individual differences, and pubertal timing cannot be made. For example, while it is certainly likely that early maturation may increase the likelihood that a young girl will develop conduct problems, it is also possible that a child with conduct problems will experience associated environmental stressors which could promote early

maturation. For example, children with conduct problems often grow up in families of lower socioeconomic status. It has been observed that high-fat diets accelerate the onset of puberty, which is particularly seen in Western low-SES populations because healthier low-fat food is much more expensive (Davison et al., 2003; Tremblay & Frigon, 2005). Future research might address these issues by employing longitudinal study designs to determine the temporal ordering of contextual factors, emotional processing and psychopathic traits, and comparing community and incarcerated youth with conduct problems to controls on a variety of individual risk factors.

### *Summary and Implications*

One challenge facing researchers, professionals, and parents is identifying risk and protective factors that contribute to whether or not a given youth with conduct problems will continue antisocial/criminal activities into adulthood, and to identify those individuals who are at risk for developing conduct problems. The current developmental model for severe conduct problems assumes that emotion regulation problems and callous/unemotional traits are primary risk factors for serious conduct problems. Boys with conduct problems, who have a profile of callous/unemotional traits, consistently show higher rates of sensation seeking, greater variety of conduct problems, are negatively associated with anxiety, and designate a subgroup of youth that are more instrumentally, as well as reactively aggressive, than conduct-disordered children without these traits (Frick, Cornell, Barry, Bodin, & Dane, 2003a). This subgroup is also characterized by different patterns of social-information processing; for example, a reward-dominant response bias with greater focus on positive aspects of aggression (Frick et al., 2003a; Pardini, Lochman, & Frick, 2003). Frick and Morris (2004) suggest that the antisocial and aggressive behavior of children with callous/unemotional traits seems to be related to deficits in conscience development rather than problems in emotion regulation. These boys also show characteristics suggestive of a temperament characterized by psychologically low autonomic

reactivity and behaviorally by low levels of fear, rather than a temperament characterized by high emotional reactivity and low effortful control of their emotions and behaviors.

While callous/unemotional traits have been demonstrated to be a significant risk factor for chronic and severe antisocial behavior in boys, the same has not been consistently demonstrated in girls. A notable absence in the current results is the lack of callous/unemotional traits to designate a more severe group of conduct problem girls. However, conduct problems are far from being a rare condition among girls; some studies indicate conduct disorder is the second most common psychiatric disorder in girls (McCabe et al., 2004). This study demonstrates that emotional dysregulation may be a more salient risk factor for conduct problems in girls.

Children with conduct problems, who have problems in emotion regulation and high levels of oppositional behavior, are associated with high levels of reactive aggression, dysfunctional parenting, and lower verbal IQ (Crick & Dodge, 1996; Frick & Morris, 2004; Patterson, 1996). This subgroup is characterized by a social-information processing style marked by a hostile attribution bias (Schultz & Shaw, 2003). Frick and Morris (2004) propose that a child with a temperamental style characterized by emotional dysregulation, negative reactivity, and deficits in effortful control is at high risk for disruptions in social development. Specifically, problems in emotion regulation can set the stage for dysfunctional parent-child interactions (Patterson et al., 1992a). Such poor emotional regulation can result from a number of interacting causal factors, such as inadequate socialization in their rearing environments (Morris, 2001); deficits in their executive functioning that make it difficult for them to delay gratification and anticipate consequences (Giancola, Mezzich, & Tarter, 1998); or temperamental problems in response inhibition (Derryberry & Rothbart, 1997). The problems in emotional regulation can lead to very impulsive and unplanned aggressive acts for which the child may be remorseful afterward, but that he or she still had difficulty controlling. However, there are limitations to an

*emotional dysregulation model*. That is, problems in emotion regulation seem to best explain conduct problems and aggression displayed in the context of high emotional arousal and the overt and angry confrontation of others, such as defiance, argumentativeness, fighting, and assault. However, emotional dysregulation is limited in explaining the development of covert forms of conduct problems, instrumental forms of aggression, and findings showing low levels of autonomic arousal are associated with antisocial and aggressive behavior (Frick & Morris, 2004).

Perhaps the most important implication of the current study is that the development of conduct problems in girls does not follow current models in a consistent manner. Thus, this dissertation suggests four developmental pathways to conduct problems and antisocial behavior in youth; adolescent-onset boys, childhood-onset boys with emotion regulation problems, childhood-onset boys with C/U traits, and a gender-specific trajectory for girls, which incorporates early pubertal timing and emotion regulation problems. While Moffitt and Caspi (2001) assume a linear and additive model of risk, other models should be considered. We emphasize the importance of understanding female aggression and related antisocial behaviors through a dynamic developmental framework that recognizes the cumulative and transactional impact of risk and protective factors over time (Odgers & Moretti, 2002). Whether or not the weight and interaction of risk factors operates similarly for girls and boys is unclear. Some risk factors (like early pubertal timing) may have gender-specific impacts, or may interact with other risk factors in gender specific ways (such as pubertal development and emotional dysregulation).

Such research is important because subtle difference in the relations between risk factors and offending across male and female samples may interfere with the ability of girls to benefit from the rehabilitative programs offered by the juvenile justice system. Since the majority of programs are male oriented (reflective of the fact that males comprise the majority of

incarcerated youth), treatment programs may not be addressing the needs of girls (Broidy, Cauffman, Espelage, Mazerolle, & Piquero, 2003). For example, there is some evidence to suggest that highly aggressive and antisocial girls are more likely to have been traumatized through sexual abuse and other forms of maltreatment than their male counterparts (Corrado, Odgers, & Cohen, 2000), and that girls are influenced differently by factors within close relationships (Doyle & Moretti, 2000). These findings suggest that a slightly different approach may be required for girls; one that addresses trauma related processes and emphasizes the nature of attachment in each girl's life (Odgers & Moretti, 2002). By improving our understanding of the trajectories that lead to delinquency among males and females, and the ways that risk factors influence these trajectories, this study lays the foundation for the development of more effective gender appropriate intervention strategies, and provides a scientific foundation upon which policy makers can base debates over the proper treatment of juvenile offenders. In addition, it will allow the generation of more specific hypotheses regarding the nature of both male and female offending to be explored in larger-scale longitudinal investigations (Broidy et al., 2003).

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## Vita

Cedar O'Donnell was born in Alert Bay, British Columbia and received her B.Sc. in May 1998 from the University of Victoria in Victoria, Canada. She graduated with honors in Psychology with a minor in English Literature. Following her undergraduate education, she worked at various emergency shelters and group homes with homeless adults and at-risk youth. Cedar completed her M.Sc. in Clinical Psychology at Acadia University in Wolfville, Nova Scotia, where she was nominated for the Governor General Award. Following her graduation she worked as a substance abuse counselor in Norwalk, CA before beginning her doctoral studies at the University of New Orleans in August, 2003. Cedar finished her doctoral studies with her clinical internship at Park Place Behavioral Healthcare in Kissimmee, FL. Cedar plans to continue her professional career in North Carolina.