The Origins of Life-Course Persistent Offending Revisited:

Does Self-Control Mediate the Effect of Neuropsychological Deficits on Early-Onset Offending?

by

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A Thesis Presented in Partial Fulfillment
of the Requirements for the Degree
Master of Science

Approved April 2014 by the
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May 2014

ABSTRACT

The link between childhood neuropsychological deficits and early-onset offending—the assumed precursor to life-course persistent offending—has been well established, yet the underlying mechanisms facilitating this relationship are less understood. Support is growing for the claim that self-control is a key mechanism that links neuropsychological deficits to early-onset offending. Despite this, findings are mixed with regard to the mediating effect of self-control in the relationship between neuropsychological deficits and antisocial behavior. These studies largely support the notion that self-control exerts a mediating effect on neuropsychological deficits when the offending being studied is less serious. Using data from the National Longitudinal Survey of Youth (NLSY), the present study seeks to build upon the existing literature by examining whether self-control mediates the relationship between neuropsychological deficits and two types of early-onset offending—low and high risk—as a means of testing core tenets of Gottfredson and Hirschi's (1990) and Moffitt's (1993) criminological theories. Findings show that while self-control and neuropsychological deficits independently predict general early-onset offending, these effects vary as a consequence of early-onset offender type. The results point to the need for future research to explore the possibility that the early-onset offender group that leads to persistent offending could be more precisely defined. Examining early-onset offending as a single construct limits our ability to make inferences about those offenders that are the most persistent in their offending patterns and, arguably, more likely to continue offending over the life-course.

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INTRODUCTION

In her widely acknowledged typology of offending, Moffitt (1993) introduces two types of offenders, adolescent-limited and life-course persistent, that are defined by their unique onset and persistence of antisocial behavior. According to her theory, life-course persistent offenders begin their life-long involvement in antisocial behavior during childhood and are thus referred to as early-onset offenders. Indeed, a common theme within life-course criminology concerns the importance of early-onset offending in predicting criminal persistence through adulthood (Moffitt, 1993; Patterson, Forgatch, Yoerger, and Stoolmiller, 1998; Piquero and Chung, 2001; Sampson and Laub, 1993). Moffitt (1993) attributes early-onset offending to underlying impairments in brain functioning, or neuropsychological deficits, more specifically deficits in verbal and executive functioning.

Verbal and executive functions are responsible for an array of abilities that are housed in the frontal lobes of the brain. According to Moffitt (1993), "individual variation in brain function may engender differences between children in activity level, emotional reactivity, or self-regulation (temperament); speech, motor coordination, or impulse control (behavioral development); and attention, language, learning, memory or reasoning (cognitive abilities)" (p.681). It is because of these deficits that childhood behavioral problems develop, translating to delinquency during adolescence and progressively more serious offending into adulthood.

The link between neuropsychological functioning and early-onset offending has been well established in criminological literature (Moffitt, Lynam, and Silva, 1994; Ogilvie, Stewart, Chan, and Shum, 2011). What is less clear are the causal mechanisms

through which neuropsychological deficits facilitate early-onset offending. Moffitt (1993) contends that neuropsychological deficits—often manifested as "poor scores on tests of language and self-control"—account for early-onset offending (p.681). This raises an important question: Is self-control a product of cognitive deficits and if so, does it mediate the effect of neuropsychological deficits on early-onset offending? Gottfredson and Hirschi (1990) posit that low self-control is largely a product of parental socialization and reject any biological underpinnings to its development. According to their theory, levels of self-control are established between the ages of 8 and 10 and manifest as late childhood behavioral problems that translate into habitual offending over the life-course.

There is a growing body of literature that evidences support for the notion that self-control is influenced by neuropsychological deficits **(**Beaver, Wright, and DeLisi, 2007; Beaver et al., 2010; Cauffman, Steinberg, and Piquero, 2005; Jackson and Beaver, 2013; Ratchford and Beaver, 2009). The existing literature on this topic, however, is limited in at least two ways. First, none examined how low self-control and neuropsychological deficits, when included in the same model, predict early-onset offending. Many of the studies focus on adolescent delinquency and offending (e.g. see Beaver et al. 2008, Cauffman et al. 2005) but not forms of delinquency that Moffitt would characterize as early-onset offending (i.e., offending that takes place prior to the age of 14). To address this limitation, the current study looks at forms of offending that Moffitt theorizes about by examining how low self-control and neuropsychological deficits predict early-onset offending.

Second, and more importantly, there have been inconsistent findings with regard to the mediating effects of self-control in the relationship between neuropsychological

functioning and antisocial behavior. Some findings support a fully mediating effect but only when examining less serious antisocial behavior (Beaver et al., 2010; Cauffman et al., 2005; Jackson and Beaver, 2013), whereas findings that point to a partially mediating or a null effect involve more serious delinquent behavior (Beaver et al., 2010; Cauffman et al., 2005). These findings suggest that neuropsychological deficits exert a direct effect on antisocial behavior but only for serious offending. This is consistent with Moffitt's (1993) contention that neuropsychological impairments are associated with delinquent behavior when the behavior is "extreme and persistent" (p. 680). This facet of the theory deserves more attention for the reasons discussed below.

Moffitt (1993) posits that the "stability of antisocial behavior is closely linked to its extremity," yet studies of early-onset offending often use a dichotomous measure, not capturing the extremity of the offending taking place (p.676). This study addresses this limitation by examining two types of early-onset offenders: low and high risk offenders prior to the age of 14. Disaggregating early-onset offending by levels of offending will facilitate our examination of the most extreme and persistent early-onset offenders—those argued to be most influenced by neuropsychological deficits and low self-control. Using data from the 1992 wave of the National Longitudinal Survey of Youth (NLSY), the mediating effect of self-control in the relationship between neuropsychological deficits and low/high risk early-onset offending is examined.

THEORETICAL FRAMEWORK

Early-Onset Offending

Moffitt's (1993) developmental taxonomy was first presented two decades ago, and has proven to be one of the most empirically tested theories in criminology and psychology. She introduces an offender typology that highlights the biological, psychological, and social factors that differentiate two offending trajectories: adolescent-limited (AL) and life-course persistent (LCP). According to Moffitt (1993), AL offending is a product of "social mimicry" which stems from the temporary inclination to engage in the delinquent activities as portrayed by the more serious offending population (LCP offenders; p. 686). These offenders engage in normative delinquent behaviors during adolescence as a means of rebelling against society's maturational status in order to achieve an autonomous status.

LCP offenders, on the other hand, are the more serious offenders, displaying antisocial tendencies in early childhood. Moffitt's (1993) theory posits that early-onset offending is an important precursor to LCP offending. Indeed, early-onset offending has been documented as one of the most salient predictors of continued offending in criminological literature dating as far back as the Gluecks' (1950) sample of 500 delinquents to Sampson and Laub's (1993) follow up of these same individuals in adulthood (Farrington et al., 1990; Moffitt, Lynam, and Silva, 1994; Nagin and Farrington, 1992a, 1992b; Patterson et al., 1998). Indeed, Robins' (1978) work was among the first to illustrate that childhood antisocial behaviors were among the best predictors of continued antisocial behavior in adulthood, above and beyond social class and family background. Therefore, it can be argued that early-onset offending is one of

the first known and consistently documented individual-level characteristics that predicted continuity of offending across various samples and methodologies.

Despite the advancements made in the way of understanding the relationship between early-onset and persistent offending, these studies have largely employed an official, dichotomous measure of early-onset offending that limits our ability to observe levels of offending masked within this measure. Many studies examining early-onset offending have operationalized it as the age in which first police contact occurs, typically limited to age 14 and 15 or younger (Bacon, Brame, and Paternoster, 2009; Ge, Donnellan, and Wenk, 2001; Gibson and Tibbetts, 2000; Patterson et al., 1998; Piquero and Chung, 2001; Tibbetts and Piquero, 1999). There are two main limitations to this measure. The first is that the measure does not tap into the frequency of antisocial behavior underlying the offending, only that these offenders align at the age in which they initiate offending. While the age of onset is important, the extent of the offending that is going on during this time frame can arguably serve as the factor that increases the risk for future offending. As Moffitt (1993) contends, stable antisocial behavior is a function of its extremity (p.671). Therefore, we argue that it is not just the onset of the offending that matters, but also the frequency of this offending that contributes to the propensity to offend over the life-course. Consequently, collapsing early-onset offending into a single construct limits our ability to make inferences about those early-onset offenders that are the most persistent within this group, and according to Moffitt, are the most likely to maintain high levels of offending across the life-course. Further, by combining a potentially heterogeneous group of offenders, we are not gaining a clear picture of the processes that link neuropsychological deficits to offending.

The second limitation of using official arrest data as an indicator of early-onset offending is that this measure does not encompass the full range of antisocial behaviors exhibited during preadolescence—especially those behaviors that do not warrant law enforcement intervention. Moffitt (1993) argues that this reliance on official measures of early delinquency essentially censors the left hand side of the age-crime curve, limiting our knowledge of offending that takes place prior to official police contact (p.675). She goes on to note "research on childhood conduct disorder has now documented that antisocial behavior begins long before the age when it is first encoded in police data banks… [with] the steep decline in antisocial behavior between the ages of 17 and 30 [being] mirrored by a steep incline in antisocial behavior between ages 7 and 17" (p.675).

Further, Moffitt (1993) discusses the issue of offender overlap in distinguishing normalized adolescent offending from offending that is unique to life-course persistent offenders. She claims that life-course and adolescent-limited offending trajectories are indistinguishable during adolescence, particularly between the ages of 15 and 17 due to the prevalence and normalcy surrounding delinquency during this age. Therefore, when conceptualizing and operationalizing early-onset offending, these factors must be taken into consideration. If offending between the ages of 15 and 17 is relatively normal among adolescent populations, early-onset offending should be limited to that which occurs prior to the age of 15. Given that, we can then infer that self-reported offending taking place between the ages of 7 and 14 can be a good indicator of early-onset offending and thus a marker for a greater propensity for future offending over the life-course.

In addition to the limitations to operationalizing early-onset offending, there is an ongoing debate surrounding the importance of early-onset offending and the processes through which it affects future offending over the life-course. Some theorists, mostly life-course theorists, argue that early-onset offending sets in motion a series of negative consequences (stigmatization, weakened social bonds, limited access to conventional activities) that accumulate and subsequently place these offenders at a higher risk for continuity of offending into adulthood (e.g. Sampson and Laub, 1993). Propensity theorists, like Gottfredson and Hirschi (1990), argue that early-onset offending is just a manifestation of inherent individual-level traits such as low self-control and underlying deficits in neuropsychological functioning. These theorists argue that once developed, these traits are relatively stable over time and account for the relationship between early-onset and persistent offending (Nagin and Farrington, 1992a, 1992b). The latter is a premise of Moffitt's (1993) theory that has been widely tested in previous research. More specifically, she argues in favor of the notion that individual differences in criminal propensity account for variations in offending over the life-course. Indeed, studies have found overwhelming empirical support for the relationship between neuropsychological deficits and early-onset offending, which is to be discussed in the section that follows.

To address these limitations, the current study disaggregates early-onset offending into two groups: low and high risk early-onset offending. Studies often collapse early-onset offending into one category, limiting our ability to make unique inferences about early-onset offenders that exhibit persistent levels of offending. Moffitt (1993) links the stability of antisocial behavior to its level of extremity (p.671); and it is among these "extreme and persistent" antisocial individuals that neuropsychological deficits are

associated (p.680). Therefore, research examining early-onset offending should recognize this distinction. The significance of early-onset offending in predicting future offending goes beyond the age of onset, and extends to the frequency with which the early-onset offending occurs. We argue that higher frequency early-onset offenders represent a distinct offender group that is linked to persistent offending over time, and is thus called a "high risk early-onset offending group" that is distinct from lower frequency early-onset offenders, that we call the "low risk early-onset offending group". In addition to providing clarity to a body of work with mixed findings, disaggregating early-onset offending by levels of offending will facilitate an empirical test of these tenets of Moffitt's (1993) theory and provide insight into the mechanisms that uniquely predict high versus low risk early-onset offending.

Neuropsychological Deficits

A key component of Moffitt's (1993) developmental taxonomy is concerned with the risk factors that condition the onset and persistence of offending. According to Moffitt (1993), LCP offenders suffer underlying deficits in neuropsychological functioning that are involved in the inhibition of criminal behavior. More specifically, deficits in verbal and executive functioning are vital to the development of a seriously antisocial individual. The origins of these deficits have been linked to abnormal functioning of the frontal lobes of the brain that are responsible for activities pertaining to "verbal functions' such as language and memory, and 'executive functions' such as abstract reasoning, anticipating and planning, sustaining attention and concentration, and inhibiting inappropriate behavior" (Piquero, 2001, p. 194). Deficits in verbal and executive functioning are said to manifest as difficult temperament during childhood,

delinquent behavior during adolescence, and serious, even violent offending later in life (Moffitt, 1993). Neuropsychological deficits are said to be further exacerbated by adverse home environments characterized by poor and/or abusive parenting, criminogenic environments, and the lack of maternal attachment, thereby making these at-risk children prone to developing and sustaining deviant behavior (e.g. see Raine, Loeber, Stouthhamer-Loeber, Moffitt, Caspi, and Lynam, 2005). Scholars have tested this notion finding support for Moffitt's (1993) "interactional hypothesis" (Tibbetts and Piquero, 1999).

An abundance of studies also support Moffitt's (1993) arguments that neuropsychological deficits have a significant effect on antisocial behavior (Moffitt and Caspi, 2001; Moffitt, Lynam, and Silva, 1994; Raine et al., 2005). Indeed, two recent meta-analyses concluded that poor executive functioning is significantly correlated with antisocial behavior, with these findings holding across various methodologies (i.e. varying definitions of antisocial behavior and measures of executive function; Morgan and Lilienfeld, 2000; Ogilvie et al., 2011). In their pioneering study testing these premises, Moffitt et al. (1994) examined whether neuropsychological deficits at age 13 were predictive of current and future delinquency reported during ages 15 and 18 among a sample of boys. Their findings reflect support for her theory in that the boys with the highest rates of delinquency and the poorest rates of neuropsychological functioning at age 13 had the highest rates of offending by the age of 18. Additionally, even though this group of offenders constituted only 12% of the overall sample population, they accounted for nearly 46% of offenses and 59% of convictions on record (Moffitt et al., 1994, p.293). Therefore, those offenders with the greatest deficits to neuropsychological functioning in

preadolescence were the most persistent offenders by the age of 18—largely supportive of tenets of Moffitt's (1993) developmental taxonomy.

Moffitt and Caspi (2001) took this a step further by disaggregating LCP and AL offending trajectories among a sample of children and adolescents as a means of determining whether risk factors for offending varied by pathway. Those on the LCP offending trajectory consisted of children with extreme antisocial behavior reported between the ages of 5 and 11 and continued extreme delinquent behavior by age 15 or 18. Adolescents in the AL trajectory were those that also reported extreme delinquency at ages 15 or 18, but did not report extreme antisocial behavior during childhood. Their findings illustrated that those on the LCP path demonstrated greater impairments to neuropsychological functioning and poorer parenting than those on the AL path. The authors further note that the rates of delinquency reported during adolescence were almost indistinguishable between LCP and AL offenders, lending credence to the notion that childhood antisocial behavior is a key precursor of persistent offending that differentiates serious offenders from those offenders whose offending is limited to adolescence (AL offenders). Thus, key tenets of Moffitt's (1993) theory are further supported by these studies.

Not all studies, however, support Moffitt's (1993) contentions. Ge et al.'s (2001) study of the development of persistent offending found that cognitive abilities measured as intelligence and scholastic achievement demonstrated no effect on the age of first arrest, or any arrests prior to the age of 17, but a significant effect on frequent offending after the age of 18. Similarly, Piquero (2001) found that the verbal and executive measures of neuropsychological deficits (as measured by the Wechsler Intelligence Scale

for Children) were unrelated to the early-onset of offending (measured as the acquisition of a police contact prior to 14). Perhaps these studies yielded these results because of how early-onset offending was operationalized—both with official measures. As previously mentioned, official measures of early-onset limit our ability to capture those individuals whose behavior does not warrant law enforcement involvement. Therefore, perhaps these samples of offenders did not capture the nature of the early offender to the extent that Moffitt (1993) hypothesizes as important and therefore failed to find a relationship between neuropsychological deficits and early offending.

Although much progress has been made in the way of establishing these relationships, the findings are mixed with regard to the mechanisms involved in the development of persistent offending. A sizeable gap remains in this body of literature that leaves us with questions surrounding the processes by which neuropsychological deficits affect early-onset offending. Existing literature examining the causal mechanisms that predict early-onset offending is somewhat scarce, thereby limiting our knowledge surrounding the individual traits that facilitate a criminal propensity across the life-course.

According to Moffitt (1993), "for persons whose adolescent delinquency is merely one inflection in a continuous life-long antisocial course, a theory of antisocial behavior must locate its causal factors early in their childhoods and must explain the continuity in their troubled lives" (p. 674). With emerging research evidencing support for a relationship between neuropsychological deficits and self-control (e.g. see Beaver et al., 2007; McGloin, Pratt, and Maahs, 2004), it is imperative that this relationship is explored further to better grasp the underlying processes at work in the development of

an early-onset offender. Both of these theories—Moffitt's (1993) taxonomy and Gottfredson and Hirschi's (1990) general theory of crime—emphasize the stability of an individual-level trait that conditions the propensity to offend over the life-course. Given that, how do these traits work in conjunction to predict early-onset offending? The following section provides an overview of literature that examines the interrelations between the theories in predicting antisocial behavior.

The Mediating Role of Self-Control

Gottfredson and Hirschi's (1990) general theory of crime has become one of the most empirically tested theories in criminology, with a number of studies showing support for their premises (e.g. see Arneklev, Grasmick, Tittle, and Bursik, 1993; Grasmick, Tittle, Bursik, and Arneklev, 1993; Pratt and Cullen, 2000). According to the theory, levels of self-control are largely a function of three effective child-rearing practices: supervision, recognition of deviant behavior, and discipline—a construct termed parental efficacy. Gottfredson and Hirschi (1990) describe individuals with low self-control as impulsive risk-takers that have an inability to orient their actions toward future goals. Levels of self-control are established between the ages of 8 and 10, remaining fairly stable thereafter, facilitating a propensity for criminal behavior over the life-course.

Since its introduction, scholars have tested the relationship between parental efficacy and self-control finding mixed support for their thesis (e.g. Hay 2001; Perrone, Sullivan, Pratt, and Margaryan, 2004). Several studies have revealed findings that suggest Gottfredson and Hirschi's (1990) parenting thesis is too narrow in the conceptualization of self-control (e.g. Burt, Simons, and Simons, 2006; Hay, 2001). For instance, research

has shown factors other than parenting to play a role in the development of self-control including biological factors such as prenatal exposure to cigarette smoke (Turner, Livecchi, Beaver, and Booth, 2011), genetic factors (Beaver et al., 2009; Wright and Beaver, 2005), as well as contextual factors including school (e.g. Teasdale and Silver 2009; Turner, Piquero, and Pratt, 2005) and neighborhood contexts (Pratt et al., 2004; Unnever et al., 2003). Neuropsychological deficits have also surfaced as a significant predictor of low self-control (Cauffman, Steinberg, and Piquero, 2005; Ratchford and Beaver, 2009). These findings suggest that neuropsychological functioning may influence self-control. If this is the case, neuropsychological deficits may be affecting early-onset offending through its influence on self-control.

Indeed, there is an emerging body of literature focusing on neuropsychological functioning and its relationship to self-control (Beaver et al., 2007; Cauffman, Steinberg, and Piquero, 2005; Ratchford and Beaver, 2009). This research largely supports the notion that self-control could be a product of deficits in neuropsychological functioning, more specifically deficits in executive functioning. Executive functions have been linked to the prefrontal cortex and are responsible for an array of abilities including "initiation, planning, cognitive flexibility, abstraction, and decision making that together allow the execution of contextually appropriate behavior" (Ishikawa and Raine, 2003, p.281). Thus, dysfunction in the prefrontal cortex has been linked to characteristics similar to those attributed to low self-control such as impulsive and immediately gratifying behavior.

It has been argued that these cognitive functions underlie the expression of self-control (Beaver et al., 2007). Using data from the Early Childhood Longitudinal Study, Kindergarten Class of 1998-1999, Beaver and his colleagues (2007) address this question

by examining whether executive dysfunction predicts levels of self-control. They measured executive dysfunction using the Early Screening Inventory-Revised test that included scales gauging fine and gross motor skills. Their findings revealed that both of these neuropsychological measures significantly predicted low self-control among kindergarten and first grade students. Beaver et al. (2007) acknowledge that their study is limited in that the relationships among neuropsychological deficits, self-control, and delinquency could not be examined, however, more recent studies have addressed this gap in literature. This study, in particular, will address this limitation by examining how these interrelationships among neuropsychological deficits and low self-control apply to early-onset offending.

A handful of studies have looked at the mediating effects of self-control on the relationship between neuropsychological deficits and antisocial behavior, yielding mixed results. As the pioneering study in this area, Cauffman et al. (2005) examined the psychological, physiological, and neuropsychological predictors of antisocial behavior between two groups of adolescents, offenders from the California Youth Authority (CYA) and high school students self-reporting delinquent behavior. Their findings show that only self-control, not neuropsychological deficits, predicts minor forms of deviant behavior. Among the more serious delinquent population, both neuropsychological deficits and self-control surfaced as predictors. Further, while self-control significantly predicted offender status, it did not fully mediate the effects of neuropsychological deficits on offender status (p.155). The authors argue that while self-control accounts for variations in minor forms of antisocial behavior, it seems that neuropsychological and biological correlates differentiate serious offenders from this population. The authors

conclude with the contention that, "variations in antisocial behavior that are within the normal range (among nonserious offenders) may be best explained by self-control" (p.160). Thus, when examining risk factors for antisocial behavior, disaggregating the variable by seriousness is essential for understanding the pathways to various types of antisocial behavior.

Following Cauffman et al.'s (2005) research, a handful of studies revealed similar findings with regard to the mediating effects of self-control. Ratchford and Beaver (2009) examined various social and biological predictors of self-control and delinquency finding that neuropsychological deficits significantly predicted levels of self-control. In predicting delinquency (measured as the number of times the individual stayed out past curfew, physically hurt someone, or had stolen something from the store), all six scales of low self-control and a measure of parenting remained significant. Neuropsychological deficits did not have an effect. The authors conclude with a notion similar to Cauffman and colleagues' (2005) in that "when biological factors[1] do have direct effects on behaviors, it is usually on serious, violent forms of delinquency, not the more common types of minor delinquency that are captured in the Delinquency Scale we used" (p. 160).

In their study examining the interrelationships among genetics, neuropsychological deficits, and self-control, Beaver et al.'s (2010) findings confirm this typology of delinquency documenting self-control as a mediating variable in the relationship between deficits in neuropsychological functioning and less serious delinquency, but not in relation to violent delinquency. For violent delinquency,

[1] Ratchford and Beaver (2009) use verbal IQ as a biological risk factor, along with low birth weight and birth complications. It should be noted that research supports the notion that social influences also play a role in the development of IQ; therefore, IQ is not merely biological in nature.

neuropsychological deficits maintained statistical significance across all models, regardless of inclusion of other interaction terms; further suggesting that these deficits maintain direct effects on delinquency for serious offending.

Recently, Jackson and Beaver (2013) examined the relationships among neuropsychological deficits, self-control, and adolescent misconduct finding that neuropsychological deficits emerged as the most salient predictor of low self-control; and that low self-control further mediated the relationship between neuropsychological deficits and misconduct (measured as parent reports of their child's inclination to fight, steal, cheat, lash out, etc.). These studies consistently demonstrate that the mediating effect of self-control in the relationship between neuropsychological deficits and antisocial behavior is largely dependent upon the seriousness of the measured behavior; supporting the notion that neuropsychological deficits potentially differentiate offending patterns among those delinquents whose offending is more serious—consistent with Moffitt's (1993) taxonomy.

This research presents an important limitation to current literature examining early-onset offending. Studies often collapse early-onset offending into one category, limiting our ability to make unique inferences about early-onset offenders that exhibit extreme levels of offending. In her theory, Moffitt (1993) makes the argument that "A substantial body of longitudinal research consistently points to a very small group of males who display high rates of antisocial behavior across time and in diverse situations" (p.678). As we have noted, it is among these antisocial individuals whose behavior is "extreme and persistent" that neuropsychological deficits are associated (p.680). Following this logic, if the persistence of antisocial behavior is dependent upon its

extremity, research examining early-onset offending should make this distinction more clear. The early-onset of offending is not significant merely because of the age in which the antisocial behavior begins, but because of the frequency and extremity with which the offending occurs that is associated with persistent offending across time. Consistent with this idea, we test whether there are two qualitatively different groups within the early-onset offender population that have yet to be examined: High and low risk early-onset offenders that differ both in rates of offending and, arguably, the risk factors influencing offending.

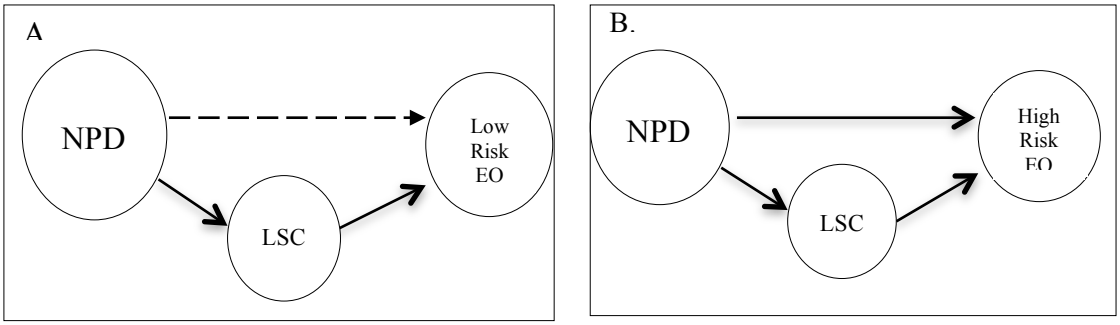
CURRENT FOCUS

Research shows that there are differences in the correlates predicting serious and less serious antisocial behavior, directing future research to confirm this notion using "more detailed analyses across distinct types of offending" (Cauffman et al., 2005, p.160). This study aims to address this call for future research by examining the relationships between neuropsychological deficits, self-control, and two types of early-onset offending. A handful of studies have examined this relationship but have only focused on less serious misconduct, delinquency, or more violent delinquent behavior between the ages of 12 and 19. Early-onset offending—a hallmark of Moffitt's (1993) taxonomy—has yet to be examined as an outcome.

Although studies have documented an association between neuropsychological functioning and self-control, there have been inconsistent findings with regard to the mediating effect of self-control in the relationship between neuropsychological functioning and antisocial behavior. Some findings support a fully mediating effect but only when examining less serious antisocial behavior (Beaver et al., 2010; Cauffman et al., 2005; Jackson and Beaver, 2013), whereas findings that support a partially mediating or null effect involve more serious delinquent behavior (Beaver et al., 2010; Cauffman et al., 2005); supportive of the tenets described in Moffitt's theory. She argues that the effect of neuropsychological deficits on early-onset offending is largely determined by the extremity and persistence of the antisocial behavior taking place. Therefore, we would expect for these deficits to exert a stronger effect on higher risk early-onset offenders, even with low self-control included in the analysis. This facet of Moffitt's theory has not been empirically tested.

Using cross-sectional data from the National Longitudinal Survey of Youth, the present study examines whether self-control has a mediating effect in the relationship between neuropsychological deficits and two types of early-onset offending: low risk and high risk offenders between the ages of 10 and 14. Given that extant research suggests that factors linked to antisocial behavior vary by the type of behavior being exhibited, it is argued that when early-onset offending is collapsed into a single construct, distinct offender groups are masked. Given this, two arguments are empirically tested. As illustrated in Figure 1, we argue that the effect of neuropsychological deficits on low risk early-onset offending will be fully explained (mediated) by low self-control (Model A; Hypothesis 1). Secondly, it is argued that both neuropsychological deficits and low self-control will exert independent and significant effects on high risk early-onset offending (Model B; Hypothesis 2); supporting the notion that neuropsychological deficits exert a more direct effect on antisocial behavior—even with self-control being held constant—when the behavior is more extreme. If these claims are supported, future research must consider an alternative approach to the study of early-onset offending in which the effects of low self-control and neuropsychological deficits are tested using two qualitatively distinct early-onset offender groups, high and low risk.

Figure 1. Models illustrating the hypotheses to be empirically evaluated. Model A demonstrates a mediating effect whereas Model B demonstrates independent effects.



RESEARCH STRATEGY

Data

The data used for this study were collected from children of the National Longitudinal Survey of Youth (NLSY79). The NLSY is an ongoing, nationally representative, longitudinal study that was initiated in 1979 and sponsored by the United States Department of Labor. Originally, the survey was intended to examine the circumstances surrounding education, employment, and family-life of men and women between the ages of 14 and 22. To date, this effort has yielded extensive information on over 12,600 individuals that are racially and ethnically diverse. Since then, the study has expanded its scope to include a child supplement that was added to the survey in 1986 and administered every two years to the children of the females in the original NLSY79 cohort. This effort has since collected information on over 11,000 children. These assessments provide extensive information on behavioral problems, cognitive functioning, and temperament exhibited by these youth, which facilitates the testing of the hypotheses in this study.

The current study uses the child supplement restricting the sample to respondents that were between the ages of 10 and 14 during the 1992 wave of surveys (N = 1419). Doing so facilitates the examination of delinquent behavior characteristic to early-onset offenders (i.e., antisocial or delinquent behavior exhibited prior to 14 years of age). Approximately 15 percent of these cases have missing values, which if deleted reduces the sample by 215 cases (N = 1204). Missing values were imputed using the similar response pattern imputation (i.e., hot deck imputation) in LISREL. In his comparison of four missing value imputation methods, Gmel (2001) found that hot deck imputation was

"superior" to the other methods because it resulted in the least biased and most consistent imputed values (p.2379). For our analysis, this method salvaged 17.7 percent (38 cases) of the missing cases, yielding a sample size of 1242. The study sample includes 49.8 percent females and 50.2 percent males between the ages of 10 and 14 (Mean = 12.04, SD = 1.36) that are 39.5 percent white and 60.5 percent nonwhite.

Measures

Early-Onset Offending. As a means to establishing a baseline measure of *early-onset offending*, this study uses self-reports of antisocial behavior despite the majority of research in this area employing an official dichotomous measure of early-onset. As Tibbetts (2009) concludes in his review of early-onset literature, "it is clear that early-onset is a consistent, strong predictor of future offending…and this finding holds across various sampling populations, using all forms of measure of criminality (official, self-reports, etc.), and across various geographic regions" (p.185). Thus, our use of self-report data is appropriate, especially given our interest in addressing certain limitations to official measures of early-onset in capturing levels of offending. Respondents were asked to report how often in the last year (never, once, twice, more than twice) they had hurt someone badly enough to need a doctor, damaged school property, taken something without paying for it, or gotten drunk. Respondents were also asked whether they had ever used marijuana or other drugs. A single report on any one of these items was coded to indicate a general early-onset offender status (1 = yes, 0 = no; Cronbach's $\alpha = .61$, mean inter-item correlation = .06).

The early-onset offender groups were created using the same six items that were used to create the general early-onset offending variable. The items were first

standardized into *z*-scores and combined into a composite measure of early-onset offending. To create the offender categories, this composite variable was disaggregated by z-scores to create two dummy variables: low and high risk early-onset offender. The *high risk early-onset offender* group was created by taking only those *z*-scores greater than or equal to 5.72 standard deviations above the mean and assigning this group a value of "1" and else as "0" (N = 79; Mean = 11.87). This classification is based on prior literature documenting that a small percentage (6%) of chronic offenders account for a majority of crimes (for a review see Farrington et al., 2003; Moffitt, Lynam, and Silva, 1994; Wolfgang, Figlio, and Sellin, 1972). These individuals account for 18% of the offender group, and 6% of the total sample (identical to Wolfgang et al.'s (1972) cohort of chronic offenders), but are responsible for 45 percent of the reported offenses. Although we are not longitudinally testing whether these early-onset offenders continue to constitute the "chronic" offender group as documented by prior research, we have reason to believe that high risk early-onset offenders are at the greatest risk of being persistent offenders over the life-course. After all, it is the stability of extreme antisocial behavior that Moffitt hypothesizes as being linked to persistent offending. The *low risk-early-onset offender* group was created by using the same standardized early-onset offender variable described above and recoding z-scores less than 5.72 standard deviations above the mean as "1" and else as "0" (N = 358; Mean = 1.00). This lower rate offender group captures the remaining 82 percent of the offender sample, and represents the lower-level offenders. Using these dummy variables, a multinomial variable was created for the analysis representing the two offender statuses and a non-

offender status (0 = non-offender, 1 = low risk early-onset offender, 2 = high risk early-onset offender).

Neuropsychological Deficits. Moffitt (1993) emphasizes the role of neuropsychological deficits in predicting serious antisocial behavior, especially deficits in verbal and executive functions. Verbal measures of IQ have shown to be the most reliable measure of neuropsychological deficits (e.g., Moffitt, Lynam, and Silva, 1994). Following this lead, the current study measures *neuropsychological deficits* using the Peabody Picture Vocabulary Test (PPVT).

The PPVT has shown to be one of the most reliable and valid measures of verbal intelligence (Baker, Keck, Mott, and Quinlan, 1993; D'Amato et al., 1988). This test assesses a child's ability to nonverbally identify pictures that best represent the meaning of a word. The children follow a "basal and ceiling" procedure in which the child identifies eight consecutive items correctly to establish a basal score, and misidentifies six of the eight consecutive items to establish a ceiling. The child's raw score is calculated by adding the number of correct responses between the basal and ceiling procedures to the overall basal score (Baker et al., 1993). In addition to raw scores, the NLSY also has standardized PPVT scores that were normed on a nationally representative sample of children and youth in 1979. This study uses the standardized PPVT score. The measure of neuropsychological deficits is coded so that higher scores are indicative of greater deficits.

Low Self-Control. Gottfredson and Hirschi (1990) claim that individuals with low self-control are impulsive, risk seeking, and simple-task oriented. The issue of how best to measure this construct, in all its complexities, is an ongoing debate in criminological

literature (for a review see Burt, 2014; Piquero, 2008). Gottfredson and Hirschi suggest that behavioral measures best capture the construct, while others opt for attitudinal measures as a means to avoiding the problem of tautology. In their meta-analysis of self-control, Pratt and Cullen (2000) conclude that the effect of self-control on crime and analogous behaviors is the same regardless of how self-control is measured. In this study, self-control is measured attitudinally using a 5-item measure, which has been used in previous work, tapping maternal reports of their child being restless, impulsive, easily confused, having trouble taking their mind off certain thoughts, and difficulty concentrating. (McGloin, Pratt, and Maahs, 2004). A factor analysis of these five items yielded a single factor solution (λ= 2.048) with factor loadings ranging from .564 to .789 indicating unidimensionality of this construct. The low self-control scale was reverse coded with higher scores indicating lower levels of self-control (Cronbach's α = .77, mean inter-item correlation = .40).

Control Variables. Given the literature documenting that minority males from higher risk neighborhoods are at an increased risk for offending than their white, socially advantaged counterparts, it is important to control for these variables to account for possible confounding effects. Several child demographic variables were controlled for including *age* (in years), *male* (0 = female, 1 = male), *minority* (0 = White, 1 = Minority), and *poverty status* (1 = yes, 0 = no). Poverty status was determined by evaluating whether a family fell below the poverty line in any given survey year weighted by the number of family members in each household as stipulated by the Center for Human Resource Research (CHHR).

A fifth control variable, *parental efficacy*, was also included. According to both Moffitt's taxonomy (1993) and Gottfredson and Hirschi's (1990) theories, parenting is a key component in the relationships among self-control, neuropsychological functioning, and antisocial behavior. Parental efficacy is conceptualized, per Gottfredson and Hirschi, as the usage of parental controls to inculcate the practice of considering future consequences before acting. For that reason, it is necessary to consider the effects that parenting may have on self-control, neuropsychological deficits, and early-onset offending, in order to disentangle the independent effects of self-control and neuropsychological deficits on early-onset offending.

The parental efficacy measure was drawn from existing research (McGloin, Pratt, and Maahs, 2004; Wright and Cullen, 2001). In this study, the measure of parental efficacy was constructed using 23 items from the NLSY that gauge direct/indirect controls and parental support (for a detailed description see Wright and Cullen, 2001). The parental control measures include: *maternal supervision*, a two-item measure asking mothers to report how often they know where their children is and how many of their child's friends they can identify by name or sight; *parental attachment*, a two-item measure asking a child how close they felt with their mother and father; and *household rules*, a four-item measure asking a child if there are rules in place surrounding TV usage, doing homework, dating, and reporting whereabouts when not home (Cronbach's $\alpha = .40$, mean inter-item correlation = .14). The measure for *parental support* was a 15-item scale measuring child and maternal reports of supportive behavior and activities (e.g., encouraging hobbies and extracurricular activities, spending time together at movies, church, doing homework, as well as showing affection) (Cronbach's $\alpha = .64$, mean inter-

item correlation = .11). These items were standardized and combined into their respective scales to reflect the various components of parental efficacy. These subscales were then factor analyzed yielding a single factor solution (λ= 1.50) with factor loadings ranging from .498 to .756. Therefore, these subscales are representative of a single construct, parental efficacy. Higher scores indicate higher levels of parental efficacy.

Analytic Strategy

The analyses proceed in three stages. We first conduct a preliminary test of the hypotheses using bivariate statistics. This analysis calculates the associations between the two theoretical variables of interest (low self-control and neuropsychological deficits), the three categories of early-onset offending, and controls. This test also allows us to rule out issues of multicollinearity, given that all of the correlations are below .38 (Mean VIF: 1.14). The second stage of the analysis allows for the assessment of our argument for testing the meditational hypothesis (hypothesis 1). According to Baron and Kenny (1986), three regression equations must be estimated to test for mediation including regressing the mediating variable onto the independent variable, regressing the dependent variable onto the independent variable, and regressing the dependent variable onto both the independent and mediating variable (p.1177). To satisfy the first condition, we regress low self-control (the mediator) on neuropsychological deficits, parental efficacy, and controls. If neuropsychological deficits surface as a significant predictor of low self-control, as we hypothesize, we have satisfied the first meditational condition and can proceed with the remainder of the analysis.

The third, and final stage of the analysis proceeds in two steps to test the remaining conditions of the meditational hypothesis first, among general early-onset

offenders and second, between low and high risk early-onset offenders. We hypothesize that low self-control will mediate the effect of neuropsychological deficits on low risk early-onset offending. Thus, if the measure of neuropsychological deficits is a significant predictor in Model 1 and this effect does not persist in Model 2, the hypothesis will be confirmed. We also hypothesize that both neuropsychological deficits and low self-control will exert significant, independent effects on high risk early-onset offenders; therefore, if in Model 2 both neuropsychological deficits and low self-control emerge as significant predictors of high risk early-onset offending, this hypothesis will also be confirmed.

Given the categorical nature of the dependent variables, the first step estimates two logistic regression models to establish the effects of neuropsychological deficits and low self-control on the general measure of early-onset offending (1 = yes, 0 = no). The second step employs multinomial logistic regression to assess the effects of these traits on the likelihood of membership in each of the offender groups (low and high risk). Both of the research hypotheses will be tested simultaneously across these models. Model 1 assesses the effects of neuropsychological deficits on low and high risk early-onset offending, net of controls. Model 2 includes low self-control in the model to observe any mediating effects.

RESULTS

To better understand the differences between the groups, we present the mean comparisons of variables across the early-onset offender and non-offender groups in table 1. Given the sample size, it is not surprising that the *t*-tests demonstrate that for all of the variables, the difference in means across these groups is statistically different from zero. As we can see, levels of self-control and parental efficacy are lower and neuropsychological deficits and poverty greater among the early-onset offender group. This shows initial support for Moffitt's (1993) argument that neuropsychological deficits are a precursor to early-onset offending. Keeping in mind that this is often the point in which most studies stop (comparing early-onset to non-offending), the next set of comparisons disaggregate early-onset offending into two groups to see whether there are

Table 1. Testing the Equality of Means across Variables for Early-Onset Offender and Non-Offender Groups Using *T*-tests (N=1242)

Variables	Early-Onset Offending		Non-Offending		
	M	*SD*	*M*	*SD*	*t*-ratio
Low Self-Control	2.57	2.35	2.03	2.01	-4.23***
Neuropsychological Deficits	92.51	17.14	87.09	17.35	-5.28***
Parental Efficacy	-.63	7.44	.34	7.46	2.21*
Poverty	.42	.49	.29	.45	-4.57***
Male	.62	.49	.44	.50	-5.96***
Minority	.66	.48	.58	.49	-2.85**
Age	12.25	1.37	11.95	1.34	-3.93***

Note: Entries *M* are means, *SD* are standard deviations, and *t*-ratios.
T-ratios tell us whether the difference in means between the two groups is statistically different from zero.
*** $p \leq .001$, ** $p \leq .01$, * $p \leq .05$, (two-tailed test)

differences in the means of these variables when looking at types of early-onset offending.

Table 2 provides the mean comparisons across low and high risk early-onset offenders. The findings provide initial support for our arguments that there are two distinct early-onset offender groups that have qualitative differences. Similar to the bivariate correlations (discussed below), levels of self-control were lower for high risk early-onset offenders, demonstrating a statistically significant difference in levels of self-control between these two groups. The same is seen for neuropsychological deficits, with high risk early-onset offenders suffering a significantly greater magnitude of deficits than their lower risk counterparts. These preliminary tests of hypothesis are largely supportive of our second hypothesis that both neuropsychological deficits and low self-control will

Table 2. Testing the Equality of Means across Variables for Low and High Risk Early-Onset Offender Groups Using *T*-tests (N=437)

Variables	Low Risk Early-Onset		High Risk Early-Onset		
	M	*SD*	*M*	*SD*	*t*-ratio
Low Self-Control	2.36	2.24	3.52	2.61	-4.05***
Neuropsychological Deficits	91.42	17.20	97.44	16.02	-2.85**
Parental Efficacy	-.33	7.50	-2.02	7.00	1.84
Poverty	.41	.49	.47	.50	-1.03
Male	.58	.50	.80	.41	-3.72***
Minority	.65	.48	.70	.46	-.77
Age	12.22	1.35	12.40	1.45	-1.04

Note: Entries *M* are means, *SD* are standard deviations, and *t*-ratios.
T-ratios tell us whether the difference in means between the two groups is statistically different from zero.
*** $p \leq .001$, ** $p \leq .01$, * $p \leq .05$, (two-tailed test)

exert independent effects on high risk early-onset offending. The same cannot be said about evidence supporting our first hypothesis. What these findings illustrate is that the mean of low self-control is higher for high risk rather than low risk early-onset offenders, and is unlikely to exert a mediating effect on neuropsychological deficits.

Table 3 presents the bivariate statistics and zero-order correlations between the theoretical variables of interest and the dependent variables. In looking at the theoretical relationships of interest, there is a weak but significant relationship between neuropsychological deficits and low self-control ($r = .19$, $p<.001$) suggesting that these variables represent independent constructs. Furthermore, the relationships between these variables and all three manifestations of early-onset offending are significant in the expected directions ($r< .15$, $p<.01$), with one exception. The relationship between low self-control and low-risk early-onset offending is not significant and near zero[2] ($r = .04$). This indicates that the effect of self-control in predicting early-onset offending matters only for higher risk offenders rather than lower risk. Indeed, for general and high risk early-onset offending, low self-control has a significant association (general: $r = .11$, $p<.001$; high risk: $r = .14$, $p<.001$) suggesting that the effect of low self-control on the high-risk sample is what is accounting for the relationship between self-control and general early-onset.

These bivariate statistics are largely unsupportive of our first hypothesis—that low self-control will mediate the effects of neuropsychological deficits on low risk early-onset offending—for two reasons. First, the correlation between neuropsychological deficits and low self-control is not strong. Second, low self-control was not associated

[2] The range and variance of low self-control were the same across all early-onset groups, allowing the author to rule out the issue of range restriction as a potential cause.

Table 3. Bivariate Associations Between Study Variables (N=1242)

	Y1	Y2	Y3	X1	X2	X3	X4	X5	X6	X7
Y1 Early-Onset Offending	-									
Y2 Low Risk Early-Onset Offending	.86***	-								
Y3 High Risk Early-Onset Offending	.35***	-.17***	-							
X1 Neuropsychological Deficits	.15***	.09**	.13***	-						
X2 Low Self-Control	.11***	.04	.14***	.19***	-					
X3 Parental Efficacy	-.06*	-.03	-.07**	-.02	-.01	-				
X4 Poverty	.13***	.10***	.07**	.37***	.10**	-.10**	-			
X5 Male	.17***	.09***	.15***	.03	.17***	.02	-.02	-		
X6 Minority	.08**	.06*	.05	.38***	.02	-.03	.32***	-.01	-	
X7 Age	.11***	.08**	.07*	.03	-.04	-.15***	.04	.02	.02	-
Mean	.35	.30	.06	88.99	1.21	.00	.33	.50	.61	12.05
SD	-	-	-	17.46	.87	1.00	-	-	-	1.36

Notes: Table reports bivariate associations (Pearson's *r*), means, and standard deviations for each variable.
$^{***}p \leq .001$, $^{**}p \leq .01$, $^{*}p \leq .05$, (two-tailed test)

with low risk early-onset offending, which means that it is unlikely to exert a mediating effect on neuropsychological deficits in this relationship. Despite these findings, it will be useful to see how these results fair in multivariate analyses, net of controls.

Table 4. OLS Regression Testing the Effects of Neuropsychological Deficits on Low Self-Control (N=1242)

Variables	Low Self-Control		
	b	(SE)	*t* -ratio
Neuropsychological Deficits	.01	(.002)	6.08***
	[.19]		
Parental Efficacy	-.00	(.003)	-.34
	[-.01]		
Poverty	.11	(.06)	2.04*
	[.06]		
Male	.28	(.05)	5.88***
	[.16]		
Minority	-.13	(.05)	-2.40
	[.07]		
Age	-.03	(.02)	-1.71
	[-.05]		
Constant	.64	(.25)	2.58**
F -Test		15.21***	
R^2		.07	

Note: Entries (*b*) are unstandardized regression coefficients, standardized coefficients are in brackets, and standard errors in parentheses.
*** $p \leq .001$, ** $p \leq .01$, * $p \leq .05$, (two-tailed test)

Because we are interested in assessing the effects of neuropsychological deficits on low self-control, table 4 presents the OLS regression analysis of these effects on low self-control net of controls. Consistent with prior research, neuropsychological deficits ($b = .01$, $\beta = .19$, $p<.001$)[3] is positively associated with low self-control such that children with greater deficits tend to have lower levels of self-control. This finding is central for our hypotheses testing in that if levels of self-control are influenced by deficits in

[3] The Breusch-Pagan test was used to determine whether the model had issues of heteroscedasticity. The *p*-value was .18 indicating that we can fail to reject the null of homoscedasticity. Therefore, the model is homoscedastic.

neuropsychological functioning, this could be the avenue through which neuropsychological deficits influence early-onset offending. Being male and in poverty also maintained significant associations with low self-control. Only 7% of the variance in self-control was explained by the model, therefore as noted previously, neuropsychological deficits does not explain much of the variance in self-control. This analysis satisfies the first condition of Baron and Kenny's (1986) criteria for testing mediating effects. Having established the relationship between neuropsychological deficits and low self-control, the next steps of the analysis work to understand how these variables, when included in the same model, predict early-onset offending.

Table 5. Logistic Regression Models of Low Self-Control and Neuropsychological Deficits Predicting Early-Onset Offending (N=1242)

Variables	Model 1			Model 2		
	Early-Onset Offending			Early-Onset Offending		
	b	(SE)	e^bStdX	*b*	(SE)	e^bStdX
Low Self-Control	-	-	-	.16*	(.07)	1.15
Neuropsychological Deficits	.01***	(.004)	1.27	.01**	(.004)	1.24
Parental Efficacy	-.01	(.01)	.91	-.01	(.01)	.91
Poverty	.37**	(.14)	1.19	.35**	(.14)	1.18
Male	.73***	(.12)	1.44	.69***	(.13)	1.41
Minority	.06	(.14)	1.03	.09	(.14)	1.04
Age	.16***	(.05)	1.24	.16***	(.05)	1.25
Constant	-4.29***	(.66)	-	-4.41***	(.66)	-
LR χ^2		86.64***			91.60***	
McFadden's R^2		.05			.06	

Notes: Entries are (*b*) unstandardized logistic regression coefficients, standard errors, and the (e^bStdX) change in odds for a one standard deviation increase in the independent variable, all other variables held constant. The models assess the probability of membership in the early-onset offender group relative to non-offending (i.e. non-offenders are the reference group).

***$p \leq .001$, **$p \leq .01$, *$p \leq .05$, (two-tailed test)

First, we want to assess the effects of the key theoretical variables on general early-onset offender status. Keep in mind that prior literature often tests correlates of early-onset offending using a dichotomous measure of offending that represents two groups (1 = at least one police contact, 0 = no police contacts). Therefore, this initial set of analyses presented in table 5 seeks to establish how these factors predict early-onset offending in the context in which it is typically analyzed. The first model includes only neuropsychological deficits and controls. Consistent with hypotheses, neuropsychological deficits (b = .01, p<.001) is a significant predictor of early-onset offending. More specifically, for a standard deviation increase in neuropsychological deficits, the odds of being an early-onset offender relative to a non-offender increase by 27%. Being male and in poverty also influenced the likelihood of membership in the early-onset offender group.

We add low self-control to the second model to observe its mediating effect on neuropsychological deficits in predicting early-onset offending. The results show that low self-control (b = .16, p<.05) only slightly mediates the effect of neuropsychological deficits on early-onset offending, with the standardized effects decreasing by 3 percentage points and remaining significant at the .01 level. More specifically, a one standard deviation increase in low self-control increases the odds of early-onset offending relative to non-offending by 15%, net of other factors in the model. Thus, when measuring early-onset offending as a dichotomous measure, both neuropsychological deficits and low self-control emerge as predictors. The following analyses disaggregate this variable into two offender groups to observe whether these effects vary by levels of offending among low and high risk early-onset offenders. We hypothesize that self-

control will mediate the effect of neuropsychological deficits on low but not high risk early-onset offending. For high risk early-onset offenders, we posit that both of these factors will exert independent effects.

Table 6 analyzes the effects of these variables in a multinomial logistic regression context across three groups: low and high risk early-onset offending with non-offenders as the reference group. Values (e^bStdX) should be interpreted as the odds of being a low or high risk early-onset offender relative to a non-offender for a one standard deviation change in the independent variable, all other variables held constant. According to the first set of models—in which only neuropsychological deficits and controls are included to predict both low and high risk early-onset offending relative to non-offending—neuropsychological deficits (low risk: $b = .01$, $p<.05$; high risk: $b = .03$, $p<.001$) emerges one of the strongest predictors across both groups. In other words, compared to non-offenders, low and high risk early-onset offenders are more likely to suffer deficits to neuropsychological functioning, which accounts at least in part for their early-onset offending—per Moffitt's (1993) theory. For a one standard deviation increase in neuropsychological deficits, the odds of high risk early-onset offending relative to non-offending increase by 77% compared to a 20% increase in the odds of low risk early-onset offending.

To test the mediating effect of low self-control on low and high risk early-onset offending, it was included as a predictor in the second set of models in table 6. Thinking back to the bivariate statistics, at this level low self-control was only associated with high, not low risk early-onset offending. In this multivariate context, the results remain the same. While low self-control exerted a slight mediating effect on neuropsychological

Table 6. Multinomial Logistic Regression of Neuropsychological Deficits and Low Self-Control in Predicting Low and High Risk Early-Onset Offender Status Relative to Non-Offender Status (N=1242)

Variables	Model 1						Model 2					
	Low Risk EO v. Non-offender			High Risk EO v. Non-offender			Low Risk EO v. Non-offender			High Risk EO v. Non-offender		
	b	(SE)	e^bStdX	*b*	(SE)	e^bStdX	*b*	(SE)	e^bStdX	*b*	(SE)	e^bStdX
Low Self-Control	-	-	-	-	-	-	.10	(.08)	1.09	.53***	(.16)	1.58
Neuropsychological Deficits	.01*	(.004)	1.20	.03***	(.01)	1.77	.01*	(.004)	1.18	.03***	(.01)	1.62
Parental Efficacy	-.01	(.01)	.95	-.04*	(.02)	.74	-.01	(.01)	.95	-.04*	(.02)	.74
Poverty	.36*	(.15)	1.19	.38	(.28)	1.20	.35*	(.15)	1.18	.34	(.28)	1.18
Male	.57***	(.13)	1.33	1.68***	(.29)	2.31	.54***	(.13)	1.31	1.54***	(.30)	2.16
Minority	.08	(.15)	1.04	-.04	(.30)	.98	.09	(.15)	1.05	.03	(.30)	1.01
Age	.14**	(.05)	1.22	.22*	(.09)	1.34	.15**	(.05)	1.22	.24**	(.09)	1.38
Constant	-3.93***	(.69)	-	-9.19***	(1.35)	-	-4.00***	(.69)	-	-9.65***	(1.36)	-
LR χ^2	114.31***						126.85***					
McFadden's R^2	.04						.05					

Notes: Entries are (b) unstandardized logistic regression coefficients, standard errors in parentheses, and the (e^bStdX) change in odds for a one standard deviation increase in the independent variable, all other variables held constant.

The models assess the probability of membership in low and high risk early-onset offender groups relative to nonoffending (i.e. non-offenders are the reference group).

These findings remained unchanged when tested with a more conservative measure of high risk offending (5% of total population and 15% of the offender population).

Hausman tests of IIA assumption finds evidence for the null hypothesis (Model 1 chi2= 1.63; Model 2 chi2=.401), indicating that the odds are independent of other alternative

$^{***}p \leq .001$, $^{**}p \leq .01$, $^{*}p \leq .05$, (two-tailed test)

deficits (decrease in 2 percentage points) in predicting low risk early-onset offending, we found that low self-control had no effect in predicting this type of offending. These results are unsupportive of our first hypothesis and suggests that perhaps low risk early-onset offenders do not have levels of self-control that are significantly different from their non-offender counterparts.

The second model also allows us to test the effects of low self-control and neuropsychological deficits in predicting high risk early-onset offending relative to non-offending. The results show that for high-risk early-onset offenders, both neuropsychological deficits (b = .03, p<.001) and low self-control (b = .53, p<.001) exert significant, independent effects, which is supportive of our second hypothesis. More specifically, this model shows that for a one standard deviation increase in neuropsychological deficits, the odds of being a high risk early-onset offender relative to a non-offender increase by 62%; and for a standard deviation increase in low self-control, the odds increase by 58%, net of each other and other factors in the model. Taken together, the results illustrated in table 6 both support and refute our hypotheses. The effects of neuropsychological deficits were not fully mediated by low self-control regardless of offender type, contrary to our first hypothesis. Further, both neuropsychological deficits and low self-control were significant in predicting high risk early-onset offending, consistent with the second hypothesis.

To supplement these findings, table 7 provides an analysis of these same constructs, but in comparing high risk early-onset offending relative to low risk. The first model shows that relative to low risk early-onset offending, the odds of high risk early-onset offending increase by 48% for a one standard deviation increase in

neuropsychological deficits (b = .02, p<.01). Being male (b = 1.11, p<.001) was the only other significant predictor in the model, being associated with a 74% increase in the odds of high risk relative to a low risk early-onset offending. Low self-control is included in

Table 7. Logistic Regression of Neuropsychological Deficits and Low Self-Control in Predicting High Risk Relative to Low Risk Early-Onset Offender Status (N=437)

Variables	Model 1			Model 2		
	High v. Low Risk Early-Onset			High v. Low Risk Early-Onset		
	b	(SE)	e^bStdX	*b*	(SE)	e^bStdX
Low Self-Control	-	-	-	.43**	(.16)	1.46
Neuropsychological Deficits	.02**	(.01)	1.48	.02*	(.01)	1.37
Parental Efficacy	-.03	(.02)	.79	-.03	(.02)	.78
Poverty	.02	(.28)	1.01	-.01	(.14)	1.00
Male	1.11***	(.30)	1.74	.99***	(.31)	1.64
Minority	-.12	(.31)	.95	-.07	(.31)	.97
Age	.07	(.09)	1.10	.09	(.09)	1.13
Constant	-5.26***	(1.39)	-	-5.64***	(1.40)	-
LR χ^2		114.31***			126.85***	
McFadden's R^2		.04			.05	

Notes: Entries are (b) unstandardized logistic regression coefficients, standard errors, and the (e^bStdX) change in odds for a one standard deviation increase in the independent variable, all other variables held constant. The models assess the probability of membership in the high risk early-onset offender group relative to low risk early-onset offending (i.e. low risk early-onset offenders are the reference group).
***p≤.001, **p≤.01, *p≤.05, (two-tailed test)

the second model, with findings illustrating that the effects of neuropsychological deficits are partially mediated by low self-control (reduction of 9 percentage points). For a one standard deviation increase in low self-control, the odds of being a high risk relative to low risk early-onset offender increase by 46%. Therefore, it appears that low self-control and neuropsychological deficits increase the likelihood of high risk, more so than low risk, early-onset offending. In other words, high risk early-onset offenders are more likely to suffer greater deficits in neuropsychological functioning and lower levels of self-

control than lower risk early-onset offenders, and as a result are at an increased risk of offending at higher rates, and perhaps continued offending over the life-course.

DISCUSSION

The current study builds on prior research in two important ways. First, this study adds to existing literature that examines the relationships among low self-control, neuropsychological deficits and antisocial behavior by extending the analysis to early-onset offending. Second, we propose an alternative conception of early-onset offending that encompasses two distinct offender groups, low and high risk early-onset offenders. Moffitt's (1993) theory argues that the extremity and persistence of early antisocial behavior is the factor most influential in predicting future offending, not necessarily the age of onset alone. Furthermore, she posits that the effect of neuropsychological deficits on antisocial behavior is most pronounced for the extremely antisocial individuals; which facilitates a high risk early-onset offender status. Therefore, disaggregating early-onset offending by levels of offending allows for the examination of these core tenets of Moffitt's theory.

Furthermore, understanding the mechanisms underlying high risk early-onset offending can provide insight into the 5-6% of chronic offenders that offend over the life-course. With literature suggesting low self-control is influenced by deficits in neuropsychological function, this study tests whether low self-control mediates the relationship between deficits and early-onset offending. The literature on this topic documents that these variables function differently for different levels of offending (serious versus less serious); therefore, we test these mechanisms in the context of predicting low and high risk early-onset offending. Toward that end, two important findings warrant further discussion.

While the analyses reveal that both low self-control and neuropsychological deficits were significant in predicting general early-onset offending, these findings changed once we account for levels of offending. More specifically, for low risk early-onset offenders, low self-control did not mediate the effect of neuropsychological deficits, which was contrary to our first hypothesis. This finding is not consistent with prior research that documents low self-control as a significant predictor of less serious antisocial behavior, exerting partially or fully mediating effects on neuropsychological deficits. In fact, low self-control did not emerge as a predictor of low risk early-onset offending, only deficits in neuropsychological functioning surfaced as a predictor.

By contrast, low self-control and neuropsychological deficits both surfaced as significant predictors of high risk early-onset offending compared to non-offending, which is consistent with our second hypothesis. In supplemental analyses, we found that high risk early-onset offenders averaged greater levels of deficits and lower self-control than low risk early-onset offenders. Furthermore, a standard deviation increase in both low self-control and neuropsychological deficits increases the odds of high risk relative to low risk early-onset offending by between 37 and 46%. In sum, our findings reveal that while low self-control was significant in predicting general early-onset offending, this finding was largely driven by the effect of this variable on the high risk early-onset offender group.

These findings have important implications for the study of early-onset offending. If we measure early-onset offending dichotomously (1 = yes, 0 = no), we may be making false inferences as to the mechanisms facilitating the onset of offending. The effect of low self-control on general early-onset offending observed in the present study is an

artifact of its influence on the higher rate offenders masked within this measure. Therefore, the results point to the need for future research examining the causes and correlates of early-onset offending to explore the possibility that the early-onset offender group that leads to persistent offending could be more precisely defined.

This study does not come without its limitations, which are threefold. We used a cross-sectional design, which could be viewed as a limitation in that we were not able to test whether the high risk early-onset offender group constitutes the chronic offender group in adulthood. Future research would benefit from looking at these offender groups longitudinally to examine levels of self-control, neuropsychological deficits, and offending across time. Second, our measure of early-onset offending is not perfect. Issues of underreporting and over reporting have been known to exist within self-report measures; but even with official measures, there is a possibility that some individuals are arrested because they are more likely to get caught, not because they are disproportionately involved. Thus, there are limitations to both official and self-report measures of antisocial behavior. Future research should examine if the findings of this study remain unchanged when using official measures of early-onset offending.

Lastly, we were not able to directly test Moffitt's (1993) contention that neuropsychological deficits are influenced prenatally. We used verbal IQ as a proxy for cognitive functioning, which is an indirect measure. With literature showing that the environment influences IQ, we cannot definitely say that our measure of neuropsychological deficits reflects solely poor prenatal care and/or perinatal exposure to toxins (Kamin, 1974). Despite this, many studies have used verbal IQ as a proxy for

neuropsychological deficits, and have documented it as one of the most reliable measures neuropsychological functioning (Moffitt, Lynam, and Silva, 1994).

Given the salience of early-onset offending as a hallmark for persistent offending across the life-course, understanding the mechanisms that work to influence the initiation into offending has important policy implications. Moffitt (1993) attributes the development of neuropsychological deficits to a child's exposure to toxins during critical stages of brain development as well as birth complications that affect the central nervous system, including exposure to cigarette smoke, drugs, and alcohol and low birth weight. These deficits are further exacerbated by a child's exposure to adverse home environments such as violent, neglectful, and/or criminogenic households. Because our findings illustrate that deficits are greater among those individuals with the highest risk to continue offending, policies should be geared toward the prevention of such deficits in cognitive development among communities that are most vulnerable to perpetuating the environments and behaviors most conducive to their development.

Pregnant women—especially within disadvantaged communities who have limited access to resources— should be educated on adequate prenatal care and provided access to resources that can facilitate such care (i.e., clinics that offer free gynecological examines and prenatal services). According to Moffitt (1994), "…among poor blacks, prenatal care is less available, infant nutrition is poorer, and the incidence of fetal exposure to toxic infectious agents is greater, placing infants at high risk for the nervous system problems that research has shown to interfere with prosocial child development" (p.38-39). In table 3 of the analysis, neuropsychological deficits were significantly, positively correlated with being in poverty ($r = .37$, $p < .001$) and being a minority

($r = .38$, $p < .001$). Therefore, providing assistance to these predominantly minority, lower income communities may aid in prevention efforts to reduce the number of youth being exposed to harmful toxins and increase chances of healthy brain development and prosocial life outcomes.

REFERENCES

Arneklev, B.J., Grasmick, H.G., Tittle, C.R., & Bursik Jr., R.J. (1993). Low Self-Control and Imprudent Behavior. *Journal of Quantitative Criminology*, 9, 225-247.

Bacon, S., Paternoster, R., & Brame, R. (2009). Understanding the Relationship Between Onset Age and Subsequent Offending During Adolescence. *Journal of Youth Adolescence*, 38, 301-311.

Baker, P. C., Keck, C. K., Mott, F. L., & Quinlan, S. V. (1993). *NLSY Child Handbook, Revised Edition: A Guide to the 1986-1990 NLSY Child Data.* Columbus, OH: Center for Human Resource Research.

Baron, Reuben M., & David A. Kenny (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *JOURNAL OF PERSONALITY & SOCIAL PSYCHOLOGY*, 51, 1173-1182.

Beaver, K.M., Schutt, J.E., Boutwell, B.B., Ratchford, M., Roberts, K., and J.C. Barnes. (2009). Genetic and Environmental Influences on Levels of Self-Control and Delinquent Peer Affiliation: Results from a Longitudinal Sample of Adolescent Twins. *Criminal Justice and Behavior*, 36, 41-60.

Beaver, K. M., Vaughn, M.G., DeLisi, M., & Higgins, G.E. (2010). The Biosocial Correlates of Neuropsychological Deficits: Results from the National Longitudinal Study of Adolescent Health. *International Journal of Offender Therapy and Comparative Criminology*, 54, 878−894.

Beaver, K., Wright J.P., & Matt DeLisi. (2007). Self-Control as an Executive Function: Reformulating Gottfredson and Hirschi's Parental Socialization Thesis. *Criminal Justice and Behavior,* 34, 1345-1361.

Burt, Callie H. (2014). *Self-Control and Crime: A Sociological Perspective*. In K.M. Beaver, J.C. Barnes, and B.B. Boutwell (eds.) *The Nurture versus Biosocial Debate in Criminology: On the Origins of Criminal Behavior and Criminality*. SAGE publishers.

Burt, Callie Harbin, Ronald L. Simons, & Leslie G. Simons. (2006). A Longitudinal Test of the Effects of Parenting and the Stability of Self-Control: Negative Evidence for the General Theory of Crime. *Criminology* 44, 353-396.

Cauffman, E., Steinberg, L., & Piquero, A. R. (2005). Psychological, neuropsychological, and physiological correlates of serious antisocial behavior in adolescence: The role of self-control. *Criminology*, 43, 133-175.

D'Amato, R. C., Gray, J. W., & Dean, R. S. (1988). Construct validity of the PPVT with neuropsychological, intellectual, and achievement measures. *Journal of Clinical Psychology,*44, 934-939.

Farrington, D.P., Loeber, R., Elliott, D.S., Hawkins, D.J., Kandel, D., Klein, M., McCord,J., Rowe, D., & Tremblay, R. (1990). Advancing knowledge about the onset of delinquency and crime. In B. Lahey and A. Kazdin (eds.), *Advances in Clinical and Child Psychology*. Vol. 13. New York: Plenum.

Ge, X., Donnellan, M.B., & Wenk E. (2001). The Development of Persistent Criminal Offending in Males. *Criminal Justice and Behavior*, 28, 731-755.

Gibson, Chris L., & Stephen G. Tibbetts. (2000). A Biosocial Interaction in Predicting Early Onset of Offending. *Psychological Reports*, 86, 509-518.

Gleuck, E.T., & Gleuck, S. (1950). *Unraveling Juvenile Delinquency*. New York: The Commonwealth Fund.

Gmel, Gerhard (2001). Imputation of missing values in the case of a multiple item instrument measuring alcohol consumption. Statistics in Medicine, 20(15), 2369-2381.

Gottfredson, Michael & Travis Hirschi. (1990). *A General Theory of Crime*. Stanford: Stanford University Press.

Grasmick, H., Tittle, C.R., Bursik, Jr., R.J., and Arneklev, B.J. (1993). Testing the core empirical implications of Gottfredson and Hirschi's general theory of crime. *Journal of Research in Crime and Delinquency*, 30, 5–29.

Hay, Carter. (2001). Parenting, Self-Control, and Delinquency: A Test of Self-Control Theory. *Criminology*, 39, 707-736.

Hope, T., Grasmick, H., & Pointon, L. (2003). The Family in Gottfredson and Hirschi's General Theory of Crime: Structure, Parenting, and Self-Control. *Sociological Focus*, 36, 291-311.

Ishikawa, S.S., & Raine, A.(2003). Prefrontal deficits and antisocial behavior: A causal model. InB. B.Lahey,T. E.Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 277-304). New York: Guilford.

Jackson, Dylan B. & Kevin Beaver. (2013). The influence of neuropsychological deficits in early childhood on low self-control and misconduct through early adolescence. *Journal of Criminal Justice,* 41: 243-251.

Kamin, L. (1974). *The science and politics of lQ*. Hillsdale, NJ: Edbaum.

McGloin, J.M., Pratt, T.C., & Maahs, Jeff. (2004). Rethinking the IQ-Delinquency Relationship: A Longitudinal Analysis of Multiple Theoretical Models. *Justice Quarterly* 21, 603-635.

McGloin, J.M., Pratt, T.C., & Piquero, A.R. (2006). Life-Course Analysis of the Criminogenic Effects of Maternal Cigarette Smoking During Pregnancy: A Research Note on the Mediating Impact of Neuropsychological Deficit. *Journal of Research in Crime and Delinquency*, 43: 412-426.

Moffitt, Terrie. (1993). Adolescence-limited and life-course-persistent anti-social behavior: A developmental taxonomy. *Psychological Review*, 100, 647–701.

Moffitt, Terrie. (1994). Natural histories of delinquency. In E. Weitekamp and H. Kerner (eds.), Cross-National Longitudinal Research on Human Development and Criminal Behavior. Amsterdam, The Netherlands: Kluwer Academic Publishers.

Moffitt, Terrie E. & Avshalom, Caspi. (2001). Childhood Predictors Differentiate Life-Course Persistent and Adolescent-Limited Antisocial Pathways among Males and Females. *Development and Psychopathology*, 13, 355-375.

Moffitt, T. E., Lynam, D. R., & Phil A. Silva. (1994). Neuropsychological Tests Predicting Persistent Male Delinquency. *Criminology*, 32, 277-300.

Morgan, A. B., & Scott Lilienfeld. (2000). A Meta-Analytic Review of the Relation Between Antisocial Behavior and Neuropsychological Measures of Executive Function. *Clinical Psychology Review*, 20, 113-136.

Nagin, Daniel S. & David P. Farrington. (1992a). The onset and persistence of offending. *Criminology* 30:501-523.

Nagin, Daniel S. & David P. Farrington. (1992b). The stability of criminal potential from childhood to adulthood. *Criminology* 30:235-260.

Ogilvie, J. M., Stewart, A. L., Chan, R. C. K., & David H. K. Shum. (2011). Neuropsychological Measures of Executive Function and Antisocial Behavior: A Meta-Analysis. *Criminology*, 49, 1063-1107.

Patterson, G.R., Forgatch, M. S., Yoerger, K.L., & Stoolmiller, M. (1998). Variables that initiate and maintain an early-onset trajectory for juvenile offending. *Development and Psychopathology,* 10, 531-547.

Perrone, D., Sullivan, C.J., Pratt, T.C., & Margaryan, S. (2004). Parental Efficacy, Self-Control, and Delinquency: A Test of a General Theory of Crime on a Nationally Representative Sample of Youth. *International Journal of Offender Therapy and Comparative Criminology,* 48, 298-312.

Piquero, Alex R. (2001). Testing Moffitt's neuropsychological variation hypothesis for the prediction of life-course persistent offending. *Psychology, Crime, and Law*, 7, 193-215.

Piquero, Alex R. (2008). Measuring Self-Control. In Erich Goode (Ed.), *Out of Control: Assessing the General Theory of Crime* (pp. 26-37). Stanford, CA: Stanford University Press.

Piquero, Alex & He Len Chung. (2001). On the relationships between gender, early onset, and the seriousness of offending. *Journal of Criminal Justice,* 29, 189-206.

Pratt, Travis C. & Francis T. Cullen. (2000). The Empirical Status of Gottfredson and Hirschi's General Theory of Crime: A Meta-Analysis. *Criminology,* 38, 931-964.

Pratt, T.C., Turner, M.G., & Piquero, A.R. (2004). Parental Socialization and Community Context: A Longitudinal Analysis of the Structural Sources of Low Self-Control. *Journal of Research in Crime and Delinquency*, 41, 219-243.

Raine, A., Loeber, R., Stouthhamer-Loeber, M., Moffitt, T. E., Caspi, A., and Don Lynam. (2005). Neurocognitive Impairments in Boys on the Life-Course Persistent Antisocial Path. *Journal of Abnormal Psychology,* 114, 38-49.

Ratchford, M. & Kevin M. Beaver. (2009). Neuropsychological Deficits, Low Self-Control, and Delinquent Involvement: Toward a Biosocial Explanation of Delinquency. *Criminal Justice and Behavior,* 36, 147-162.

Robins, Lee N. (1978). Sturdy childhood predictors of adult antisocial behaviour: replications from longitudinal studies. Psychological Medicine, 8, pp 611-622.

Sampson, R.J., & Laub, J. H. (1993). *Crime in the Making: Pathways and Turning Points Throughout Life.* Cambridge, MA: Harvard University Press.

Teasdale, B., & Silver, E. (2009). Neighborhoods and Self-Control: Toward an Expanded View of Socialization. Social Problems 56, 205-222.

Tibbetts, Stephen G. (2009). Perinatal Development and Determinants of Early Onset of Offending: A Biosocial Approach for Explaining the Two Peaks of Early Antisocial Behavior. In Joanne Savage (ed.), The Development of Persistent Criminality. Oxford University Press: New York, p. 179-201.

Tibbetts, Stephen G. & Alex R. Piquero (1999). The Influence of Gender, Low Birth Weight, and Disadvantaged Environments in Predicting Early Onset of Offending: A Test of Moffitt's Interactional Hypothesis. *Criminology* 37, 843-878.

Turner, M. G., Livecchi, C.M., Beaver, K. M., & Jeb Booth. (2011). Beyond the Socialization Hypothesis: The Effects of Maternal Smoking during Pregnancy on the Development of Self-Control. *Journal of Criminal Justice*, 39, 120-127.

Turner, M.G., Piquero, A., & Travis C. Pratt. (2005). The School Context as a Source of Self-Control. *Journal of Criminal Justice*, 33, 327-339.

Unnever, J.D., Cullen, F.T., & Travis C. Pratt. (2003). Parental Management, ADHD, and Delinquent Involvement: Reassessing Gottfredson and Hirschi's General Theory. *Justice Quarterly*, 20, 471-500.

Wolfgang, M.E., Figlio, R.M., & Sellin, T. (1972). *Delinquency in a birth cohort*. Chicago: University of Chicago Press.

Wright, John Paul & Kevin M. Beaver. (2005). Do Parents Matter in Creating Self-Control in their Children? A Genetically Informed test of Gottfredson and Hirschi's Theory of Low Self-Control. *Criminology*, 43,1169-1202.

Wright, J. P, & Cullen, F. T. (2001). Parental efficacy and delinquent behavior: Do control and support matter? *Criminology, 39,* 677-705.