Journal of Criminal Justice 47 (2016) 32–40

Contents lists available at ScienceDirect

Journal of Criminal Justice

The link between infant neuropsychological risk and childhood antisocial behavior among males: The moderating role of neonatal health risk

Dylan B. Jackson *, Jamie Newsome

The University of Texas at San Antonio, United States

ARTICLE INFO

Article history:
Received 17 May 2016
Received in revised form 29 June 2016
Accepted 30 June 2016
Available online xxxx

Keywords:
Antisocial behavior
Childhood
Infancy
Moderating effects
Neuropsychological risks
Perinatal risks
Neonatal health

ABSTRACT

Purpose: The current study examines the association between infant neuropsychological risk and childhood antisocial behavior, and whether neonatal health risk moderates this association.
Methods: Longitudinal data from the Early Childhood Longitudinal Study: Birth Cohort (ECLS-B) were analyzed using hierarchical regression.
Results: The findings suggest that the association between infant neuropsychological risk and childhood antisocial behavior is contingent on both biological sex and neonatal health risk. Males who experienced neonatal health risks exhibited significantly higher levels of childhood antisocial behavior as neuropsychological risk during infancy increased.
Conclusions: The relationship between neuropsychological risks and childhood antisocial behavior may be exacerbated by neonatal health risks among males. Implications for theory, prevention, and intervention are considered.

Developmental, life-course research in criminology has revealed a number of risk factors for antisocial behavior, many of which are present at the earliest stages of human development (Brennan, Grekin, & Mednick, 1999; Kandel & Mednick, 1991; Moffitt, 1993; Raine, Brennan, & Mednick, 1994, 1997; Raine, Brennan, Mednick, & Mednick, 1996; Tibbetts & Piquero, 1999; Vaske et al., 2015; Wright et al., 2008). Some scholars, however, have sought to uncover additional variables that may exacerbate or mitigate the relationships between early risk factors and antisocial behaviors (Baron & Kenny, 1986; Farrington & Welsh, 2007; Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997). In doing so, these scholars aim to refine explanations of antisocial behaviors, improve accurate identification of at-risks youths, and enhance early prevention and intervention strategies.

Research examining the moderators of the link between early biological risk factors and antisocial outcomes have primarily focused on social moderators (Raine et al., 1994, 1997; Raine et al., 1996; Tibbetts & Piquero, 1999; Vaske et al., 2015), revealing significant biosocial effects on antisocial outcomes. Another line of research, however, suggests that early biological risks can interact with each other to increase one's risk for antisocial behavior (Brennan et al., 1999; Gibson & Tibbetts, 1998). Among the most robust early biological predictors of antisocial behavior are deficits in neuropsychological functioning (and proxy measures of such deficits). Neuropsychological risk factors have been repeatedly identified as important predictors of behavioral problems emerging in early childhood and persisting into later developmental stages (Moffitt, 1990, 1993, 2006). The empirical evidence indicating neuropsychological risk contributes to antisocial development is now abundant (Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011), and additional research has been devoted to investigating potential social moderators of the relationship (Raine et al., 1996; Tibbetts & Piquero, 1999). Even so, scholars have only rarely explored whether the link between neuropsychological risk and antisocial behavior is moderated by other biological factors (except see Beaver, DeLisi, Vaughn, & Wright, 2010; Utendale et al., 2014). The current study seeks to address this limitation by investigating whether health risks during the neonatal period moderate the relationship between infant neuropsychological risks and antisocial behavior during childhood.

1. Neuropsychological risk and antisocial behavior

Moffitt's (1993) dual taxonomy proposed two developmental trajectories for antisocial behavior. According to the theory, the majority of offenders can be classified as adolescence-limited offenders who engage in wayward behaviors during adolescence. For this group, delinquency is a means of coping with the frustrations associated with reaching biological maturity prior to reaching social maturity. Once social maturity is reached, adolescence-limited offenders typically desist and resume involvement in prosocial lifestyles. A smaller group of offenders are characterized by an early onset of deviance, and a more

* Corresponding author.
E-mail addresses: dylanbakerjackson@gmail.com, Dylan.Jackson@utsa.edu (D.B. Jackson).

http://dx.doi.org/10.1016/j.jcrimjus.2016.06.003
0047-2352/

serious and persistent criminal career. These life-course-persistent (LCP) offenders are hypothesized to suffer from neuropsychological deficits present at birth or shortly thereafter, which may be exacerbated by an adverse rearing environment (Moffitt, 1993).

Moffitt (1993) also proposed that neuropsychological risk among LCP offenders will result in temperamental, cognitive, and behavioral challenges over the life course, making it exceedingly difficult for them to escape an antisocial lifestyle. Tests of this hypothesis would, ideally, incorporate direct measures of neurological functioning such as those garnered from neuroimaging techniques. Criminologists seeking to understand this relationship across the life course, however, have relied on a wide range of indirect measures to assess neuropsychological risk. For example, neuropsychological deficits in childhood have been operationalized using the Peabody Picture Vocabulary Test-Revised (PPVT-R; Rowland, Schwarts, Nedelec, & Beaver, 2012), the Wechsler Intelligence Scale for Children (WISC; McGloin, Pratt, & Piquero, 2006; Piquero, 2001; Piquero & White, 2003), and the Early Screening Inventory – Revised (Jackson & Beaver, 2013). During adolescence, neuropsychological deficits have been measured using the California Achievement Test (CAT; McGloin et al., 2006; Piquero & White, 2003), the Cambridge Neuropsychological Test Automated Batteries (CANTAB; Cauffman, Steinberg, & Piquero, 2005), the PPVT-R (Beaver, Vaughn, DeLisi & Higgins, 2010; Jackson & Beaver, 2016; Schwartz & Beaver, 2015), and the WISC-R (Moffitt, Lynam, & Silva, 1994). The PPVT-R has also been used to assess neuropsychological deficits in adulthood (Beaver, Vaughn, et al., 2010; Jackson & Beaver, 2016). This line of inquiry has consistently revealed that these indicators of neuropsychological deficits are related to traits and behavioral patterns that are associated with serious offending.

An important prediction in Moffitt's (1993) theory, however, is that neuropsychological deficits are present at or shortly after birth. In general, scholars have encountered various challenges in their attempts to measure neuropsychological deficits at very early developmental stages. As a result, a number of researchers have relied on proxy measures of neuropsychological deficits such as low birth weight (McGloin & Pratt, 2003; Tibbetts & Piquero, 1999; Vaske et al., 2015), and maternal cigarette use during pregnancy (Gibson, Piquero, & Tibbetts, 2000; Gibson & Tibbetts, 2000; Piquero, Gibson, Tibbetts, Turner, & Katz, 2002). The rationale for this strategy is that early development may be hindered by these risk factors in ways that result in neuropsychological deficits, which in turn lead to antisocial behavior (Liu, 2004). Only one study has tested this assumption empirically by investigating the nature of the relationship between maternal cigarette use during pregnancy, neuropsychological functioning during early adolescence, and LCP offending (McGloin et al., 2006). The results indicated that maternal cigarette use was associated with reductions in neuropsychological functioning ($B = -2.66$, $p < 0.05$), but that the effects remained significant in models predicting LCP offending while controlling for neuropsychological functioning ($B = 1.86$, $p < 0.10$). In other words, maternal cigarette use had independent effects on LCP offending that were not mediated through neuropsychological deficits. McGloin et al. (2006) concluded that the relationships between maternal cigarette smoking and LCP offending may operate through alternative mechanisms, which casts doubt on the validity of maternal cigarette smoking as a proxy for neuropsychological deficits.

Relatedly, a later study by Beaver, Vaughn, et al. (2010) explored the relationships between early risk factors and neuropsychological deficits. The results indicated that breastfeeding during infancy ($B = -0.13$, $p < 0.05$) and exposure to cigarette smoke in the household during adolescence ($B = 0.09$, $p < 0.05$) were significant predictors of neuropsychological deficits in adolescence and adulthood. However, being born low birth weight and having a criminal father were *not* significant predictors of neuropsychological deficits. Although there is evidence that prenatal and perinatal factors are related to later offending (Liu, 2011; Raine, 2002), some studies have suggested that low birth weight individuals offend at a similar or lower rate than normal birth weight individuals (Cooke, 2004; Hack et al., 2004). These mixed results, in addition to the findings from McGloin et al. (2006) and Beaver, Vaughn, et al. (2010), suggest that the relationships between pre- and perinatal risks, neuropsychological risks, and the development of antisocial behavior are not entirely clear and warrant further investigation.

2. The interplay of early biological risks in the development of antisocial outcomes

Several studies have linked prenatal and perinatal factors to antisocial behavior in childhood and later stages of development (Liu, 2011; Raine, 2002). Most of these studies have investigated whether social factors may exacerbate biological risks; however, research has also investigated whether interactions between multiple biological risk factors may increase one's risk for antisocial behavior. For example, a recent study of preschoolers (n = 109) in an urban, Southern United States community revealed that prenatal stress is most likely to predict disruptive behavioral disorders in male children who were also exposed to high levels of testosterone in the womb (operationalized using a proxy measure of 2D:4D finger length ratios; Martel & Roberts, 2014). Research using a birth cohort of males in Copenhagen, Denmark (n = 4169) has also revealed that individuals who encounter *both* maternal cigarette use and delivery complications incur a significantly higher risk of engaging in violent offending as adults than individuals who experience one of these early biological risks in isolation (Brennan et al., 1999).

Other studies have considered Apgar scores, which quantify the overall physical condition of the infant at birth, in combination with other biological risks. Apgar scores are derived by assigning a score of 0, 1, or 2 on five signs of health (i.e., heart rate, respiratory effort, reflex irritability, muscle tone, color) at one and five minutes after birth. Apgar scores of 7 or higher are considered normal. In a cohort study of 727 children in Norway, Moster, Lie, and Markestad (2002) found that individuals who had low five minute Apgar scores *and* neonatal health risks during the first week of life had a significantly higher risk of experiencing a variety of adverse outcomes in kindergarten, including attention deficit/hyperactivity diagnoses and behavioral problems. Importantly, the study buttressed the significance of the *interaction* between Apgar scores and neonatal health risks in conduct problems during childhood, as subjects who experienced either low Apgar scores or neonatal health risks incurred no additional risk of behavioral problems. Another study examined the interaction between Apgar scores one-minute after birth and maternal cigarette smoking during pregnancy on offending behaviors in adolescence and early adulthood in a sample of 832 inner-city African American youths in Philadelphia (Gibson & Tibbetts, 1998). The results revealed that neither of the risk factors independently predicted offending; however, the interaction between them was significantly predictive of offending ($b = -0.016$, $p < 0.01$).

While this body of literature remains somewhat limited, the findings from these studies provide some initial evidence that factors related to prenatal and neonatal health may exacerbate the effects of other biological risks. The interplay between these measures of early biological risk appears to have a number of adverse consequences—including problems with temperament, cognition, and behavior—across infancy (Laplante, Brunet, & King, 2016), childhood (Moster et al., 2002), adolescence (Gibson & Tibbetts, 1998), and adulthood (Brennan et al., 1999). Although some scholars have explored the possibility that biological risk factors might moderate the link between neuropsychological functions and conduct problems (Beaver, DeLisi, et al., 2010; Gower & Crick, 2011; Trampush, Jacobs, Hurd, Newcorn, & Halperin, 2014; Utendale et al., 2014), this body of research is also quite limited. Nevertheless, these studies provide some preliminary evidence that biological factors might moderate the relationship between neuropsychological deficits and antisocial behavior. For instance, Beaver, DeLisi et al. (2010) found that, among white males, neuropsychological deficits interact with MAOA genotype to predict both low self-control and