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Abstract

A body of empirical research has revealed that neuropsychological functioning is one of the most consistent predictors of antisocial behavior. It is somewhat surprising however that criminological research has been slow to examine the different factors that are implicated in the development of neuropsychological deficits. This study addresses this gap in the literature by examining the effects that a number of social and biological variables have on neuropsychological functioning. Analysis of the National Longitudinal Study of Adolescent Health (Add Health) indicates that postnatal exposure to cigarette smoke, duration of breastfeeding, maternal involvement, and household income predicts variation in adolescent and adulthood levels of neuropsychological functioning. Implications of the findings are noted and discussed.

Keywords

Add Health, biosocial, gender, neuropsychological deficits

The major thrust of criminological research explores the potential causes of diverse forms of antisocial behavior. Although this research has been relatively successful at identifying the major criminogenic risk factors, much less research has attempted to

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uncover the causes and the developmental pathways to many of these criminogenic risk factors (Farrington & Welsh, 2007). These knowledge gaps in understanding the causes of criminogenic risk factors are critically important on two main fronts. First, to develop rich theories of criminal behavior, it is essential to know why certain criminogenic risk factors emerge. Without knowing the precise causes of certain criminogenic risk factors, theorists are left with an incomplete knowledge base from which to construct theories. Second, the development of effective early intervention programs hinges on the ability to impede criminogenic risk factors from ever surfacing. For example, if research revealed that a particular risk factor was due primarily to neighborhood conditions but not to parental socialization, then intervention programs should be designed to target neighborhood conditions, not parental socialization.

Against this backdrop, it is imperative that criminological research begin to study the antecedent causes of the most powerful criminogenic risk factors. One potent criminogenic risk factor of antisocial behavior is neuropsychological deficits (Moffitt, 1990, 1993; Morgan & Lilienfeld, 2000; Raine et al., 2005). Just like many other criminogenic risk factors, relatively little research has been conducted to examine the different factors that might be responsible for creating neuropsychological deficits (McGloin, Pratt, & Piquero, 2006). This is a serious impediment to prevention efforts that aim to foster healthy human development during the earliest stages of the life course. The current study addresses this shortcoming by exploring the association between neuropsychological deficits and a range of different social and biological variables using data from the National Longitudinal Study of Adolescent Health (Add Health).

The Relationship Between Neuropsychological Deficits and Antisocial Behaviors

An impressive line of research indicates that neuropsychological functioning is inextricably linked to the development of offending behaviors, especially serious physical violence. Of particular relevance is the literature surrounding Moffitt's (1993) developmental taxonomy. In this theory, Moffitt proposed two groups of offenders: adolescence-limited (AL) offenders and life-course-persistent (LCP) offenders. AL offenders engage in acts of delinquency only during adolescence; they do not exhibit antisocial behavior during childhood nor do they commit criminal offenses as adults, although they may experience some psychosocial problems in adulthood (Moffitt, 2006). In many ways, AL offending is normative in that most adolescents are at least minimally involved in delinquency, but these behaviors pass as youths mature into young adults. LCP offenders, in contrast, begin to display antisocial behaviors as children, they are involved in delinquency as adolescents, and they continue to engage in criminal acts in adulthood. LCP offenders, in short, are career criminals who accrue multiple arrests and, as a result, often spend a considerable amount of time incarcerated (DeLisi, 2001, 2005).

According to Moffitt (1993), the etiological factors that cause the development of AL offenders are very different from the etiological factors that cause the development

of LCP offenders. Specifically, AL offenders are thought to emerge from what has been coined the "maturity gap." The maturity gap refers to the disjuncture between biological maturity and social maturity. In contemporary society, adolescents are biologically mature (i.e., they have the capacity to reproduce, they may physically resemble adults, etc.) but they have a series of social restrictions placed on them that limit their autonomy. For example, among many other restrictions, adolescents cannot legally consume alcohol and they cannot vote. The end result is that there is a mismatch between their biological maturity and their social maturity, where their biological maturity exceeds their social maturity, a phenomenon that Moffitt termed the *maturity gap*.

Adolescents caught in the maturity gap seek out ways to reduce the chasm between their biological maturity and their social maturity. In doing so, it is seen that LCP offenders engage in many adult-like behaviors, such as drinking alcohol, smoking cigarettes, engaging in promiscuous sex, skipping school, and staying out late. Adolescents begin to mimic these adult-like behaviors, many of which are illegal. As adolescence passes, and as youths are afforded more and more privileges, they begin to escape the maturity gap and, as a result, their delinquent involvement drops appreciably. By the time AL offenders reach adulthood, they are not trapped in the maturity gap any more and thus they do not engage in criminal offenses. Whether the maturity gap is indeed the contributing factor to AL offending remains unresolved because only one study has directly examined Moffitt's maturity gap thesis (Piquero & Brezina, 2001). It should be noted however that the results of the Piquero and Brezina (2001) study provided some evidence supporting the relationship between the maturity gap and AL offending.

The etiological factors that promote the emergence of LCP offending are very different from those tied to the development of AL offenders. Because LCP offenders begin to display antisocial behaviors during childhood, the factors causing LCP offending must also be present early in the life course. In explicating her theory, Moffitt identified two causal factors—neuropsychological deficits and an adverse home environment, both of which must be present for LCP offenders to develop. Specifically, she argued that children born with neuropsychological deficits often have difficult temperaments, cognitive disabilities, and hyperactivity, making them a challenge to rear. Children with neuropsychological deficits who are born into prosocial environments are typically provided with adequate nurturing and socialization, thereby offsetting the criminogenic effects of neuropsychological deficits. In contrast, children with neuropsychological deficits who are born into an adverse home environment are often provided with inadequate nurturing and socialization, thereby exacerbating the criminogenic effects of neuropsychological deficits. Over time, the transactional process that occurs between these children (with neuropsychological deficits) and their criminogenic environment molds them into LCP offenders.

A body of empirical research has examined Moffitt's explanation of LCP offenders, the results of which have been relatively supportive (Moffitt, 2006). Studies have reported a link between various measures of neuropsychological deficits and various

measures of LCP offending in a broad range of heterogeneous samples (Gibson, Piquero, & Tibbetts, 2000; McGloin et al., 2006; McGloin & Pratt, 2003; Moffitt, 2006; Moffitt, Lynam, & Silva, 1994; Piquero, 2001; Raine et al., 2005; Tibbetts & Piquero, 1999; Turner, Hartman, & Bishop, 2007). In addition, some studies have shown that the effect of neuropsychological deficits on LCP offending is stronger when paired with an adverse home environment. For example, Tibbetts and Piquero (1999) reported that low birth weight—a proxy measure for neuropsychological deficits—had an effect on delinquency but only among males who were from low–socioeconomic status (SES) families; there was no effect of low birth weight on delinquency for males from higher SES families. In light of these findings, it is probably not surprising that Moffitt (1993, p. 680) has argued that "the link between neuropsychological deficits and antisocial outcomes is one of the most robust effects in the study of antisocial behavior."

Other studies have also examined the association between neuropsychological deficits and antisocial behaviors without explicitly testing Moffitt's theory. The results of these studies, once again, have highlighted the close nexus between neuropsychological functioning and delinquent and criminal involvement. To illustrate, Morgan and Lilienfeld (2000) conducted a meta-analysis of 39 studies (N = 4,589 respondents) that had examined the effect of neuropsychological functioning on measures of antisocial behaviors, such as psychopathy and antisocial personality disorder. This meta-analysis demonstrated a statistically significant and relatively strong effect (effect size = .62) of neuropsychological functioning on antisocial behavior. Taken together, the results garnered from prior research point to the likelihood that neuropsychological deficits are a consistent predictor of various forms of misconduct.

If neuropsychological deficits are implicated in the etiology of antisocial behaviors, then the central question is, what causes variation in neuropsychological functioning? Stated differently, why are criminals more likely to have neuropsychological deficits than noncriminals? Moffitt (1993) tackled this issue when she stated:

> One possible source of neuropsychological variation that is linked to problem behavior is disruption in the ontogenesis of the fetal brain. . . . Neural development may be disrupted by maternal drug abuse, poor prenatal nutrition, or pre- or postnatal exposure to toxic agents. . . . In addition, some individual differences in neuropsychological health are heritable in origin. . . . After birth, neural development may be disrupted by neonatal deprivation of nutrition stimulation and even affection. Some studies have pointed to child abuse and neglect as possible sources of brain injury in the histories of delinquents with neuropsychological impairment. (p. 680, references omitted)

As Moffitt's statement makes clear, a host of factors—some of which are social and some of which are biological—are responsible for creating variation in neuropsychological functioning.

This Study

The purpose of this study is to examine the contributors to neuropsychological deficits using a broad range of factors that have been found to be involved in neuropsychological functioning. We focused on a range of variables that are implicated in the development of neuropsychological deficits. Below we describe each of these variables in detail and provide a rationale for why they were included in the statistical models. To examine whether males are more vulnerable to these risk factors than females (Moffitt, Caspi, Rutter, & Silva, 2001), we calculated the models separately by gender.

Method

Data

Data for this study came from the National Longitudinal Study of Adolescent Health (Add Health), which is a prospective, nationally representative sample of American youths who were enrolled in Grades 7 through 12 in 1994-1995 (Udry, 2003). Stratified random sampling techniques were used to identify a sample of 132 middle and high schools. During a regularly scheduled school day, students attending these schools were administered a self-report survey. Respondents were asked questions about their friends, their family, and their behaviors. More than 90,000 adolescents participated in the Wave 1 in-school component of the study. A subsample of all students was then selected to be reinterviewed in their home, along with their primary caregiver (preferably their mother). These in-home interviews were designed to gather more detailed information about the respondent, including information that was sensitive (e.g., involvement in sexual behaviors). Overall, 20,745 youths and 17,700 of their primary caregivers participated in the Wave 1 in-home interviews (Harris et al., 2003).

Approximately 1 to 2 years later, the second wave of questionnaires was administered to 14,738 of the respondents. Because relatively little time had lapsed between data collection waves, many of the same items asked at Wave 1 were also asked during Wave 2 interviews. Youths, for example, were asked about their use of drugs, alcohol, and tobacco, and they were also asked about their involvement in other delinquent activities. Information relating to their family and peer relationships was also collected. In 2001-2002, approximately 7 years after the inception of the study, the third wave of interviews was conducted. Because most of the respondents were young adults at Wave 3, many of the questions asked at the previous two waves were removed and newer, more age-appropriate questions were added to the survey instruments. For instance, respondents were asked about their employment history, their marital status, and their lifetime contact with the criminal justice system. Overall, 15,197 respondents were successfully interviewed at Wave 3 (Harris et al., 2003).

The Add Health sample includes more than 15,000 respondents. Using a sample this large necessarily translates into very small effect sizes reaching statistical significance. As a result, we extracted a random sample of about 2,000 respondents to be analyzed. This sample size was chosen because it is similar to the sample size of other large, nationally representative samples, which allows for a more direct comparison of findings. Moreover, all models were also estimated with sample sizes of 1,000, 1,500, 2,500, and 3,000, and the substantive results remained unchanged.

Measures

Neuropsychological Deficits

One of the main difficulties in studying issues related to neuropsychological deficits is using a measure that accurately assesses neuropsychological functioning. Most criminological data sets, for example, do not include direct measures of neuropsychological functioning and, as a result, indirect measures are used quite frequently. Perhaps the most widely used protocols to evaluate neuropsychological deficits are tests that index verbal skills (Lynam, Moffitt, & Stouthamer-Loeber, 1993; Moffitt, 1990; Moffitt, Lynam, & Silva, 1994; Piquero, 2001). In the Add Health study, verbal skills were measured with an abbreviated version of the Peabody Picture Vocabulary Test (PPVT).

Research investigating the psychometric properties of the PPVT has found it to be a valid and reliable measure of verbal skills and receptive vocabulary (D'Amato, Gray, & Dean, 1988; Dunn & Dunn, 1981). Evidence also exists revealing that the PPVT correlates with other measures of neurological skills (Quattrocchi & Golden, 1983). Add Health participants were administered the PPVT during Wave 1 interviews and again during Wave 3 interviews. Originally, the PPVT scores were coded such that higher values indicated more verbal skills. For ease of interpretation, however, the PPVT was reverse coded, where higher scores indicated more neuropsychological deficits. The PPVT variables used in the current study had been transformed into standardized scores by the Add Health research team. The correlation between the two neuropsychological deficits measures was $r = .60$ ($p < .05$, two-tailed test). Table 1 includes the descriptive statistics for the PPVT scores and the other variables and scales used in the analyses.

Biosocial Measures

Exposure to cigarette smoke. A line of research has revealed that maternal cigarette smoking is associated with reduced neurocognitive development (McGloin et al., 2006; Yolton, Dietrich, Auinger, Lanphear, & Hornung, 2005). At the same time, there is also some evidence indicating that postnatal exposure to cigarette smoke is related to neuropsychological deficits (Yolton et al., 2005). The Add Health data did not include any measures of whether the respondent was exposed to nicotine in utero.

Table 1. Descriptive Statistics for Add Health Sample Variables and Scales

	Mean	Standard deviation	Range
Dependent variables			
Neuropsychological deficits at Wave 1	101.12	15.47	10-136
Neuropsychological deficits at Wave 3	101.25	17.29	7-122
Biosocial measures			
Exposure to cigarette smoke	0.45	0.50	0-1
Length of breastfeeding	1.21	1.65	0-6
Birth weight	7.34	1.27	4-11.9
Criminal father	0.13	0.34	0-1
Socialization measures			
Household income	49.52	53.05	0-999
Maternal involvement	4.12	1.92	0-10
Maternal disengagement	8.87	3.38	5-25
Maternal attachment	9.44	1.05	2-10
Neighborhood disadvantage	0.28	3.86	–9.80-5.20
Control variables			
Age	15.94	1.67	12-21
Race	0.31	0.46	0-1
Gender	0.50	0.48	0-1

During Wave 1 interviews, however, mothers were asked whether anyone in their household currently smoked. This item was coded dichotomously where 0 = *no cigarette smokers in household*, and 1 = *at least one cigarette smoker in the household*. Presumably respondents who lived in households with cigarette smokers were exposed to more cigarette smoke than respondents who lived in households without any cigarette smokers.

Length of breastfeeding. A mounting body of literature has documented a link between breastfeeding and cognitive abilities (Anderson, Johnstone, & Remley, 1999; Caspi et al., 2007). Although far from conclusive, research findings tend to suggest that there is a positive correlation between the amount of time that babies are breastfed and their neurocognitive skills measured later in life, where neurocognitive skills tend to be higher among persons who were breastfed (Kramer et al., 2008; Mortensen, Michaelson, Sanders, & Reinisch, 2002). To take this finding into account, a one-item length of breastfeeding variable was included in the analyses. During Wave 1 interviews, mothers were asked to report whether they breastfed their child and, if so, the duration of breastfeeding. Responses to this question were coded as follows: 0 = *did not breastfeed*, 1 = *less than 3 months*, 2 = *3 months to less than 6 months*, 3 = *6 months to less than 9 months*, 4 = *9 months to less than 12 months*, 5 = *12 months to less than 24 months*, and 6 = *24 months or more*.

Birth weight. Low birth weight has been found to be a risk factor for an array of maladaptive outcomes (Conley, Strully, & Bennett, 2003), including antisocial behaviors (Raine et al., 2005; Tibbetts & Piquero, 1999). In addition, birth weight has also been found to have an inverse relationship with neurocognitive skills (Whitaker et al.,

2006). As a result, a birth weight variable was included in the analyses. During Wave 1 interviews, mothers were asked to indicate the respondent's birth weight. Birth weight is a continuous variable measured in pounds and ounces. In line with prior research (McGloin et al., 2006), the continuous birth weight variable was also recoded as a dichotomous dummy variable, where 0 = *greater than 5.5 pounds* and 1 = *less than or equal to 5 pounds 8 ounces*. All of the models were recalculated using this dichotomous birth weight variable. The pattern of results remained unchanged. Consequentially, the more specific continuous variable was retained in the analyses.

Criminal father. We included a one-item criminal father variable in the analyses for two main reasons. First, many criminogenic risk factors, including neuropsychological deficits, are genetically influenced (Moffitt, 1993). Studies that fail to control for genetic effects thus risk being misspecified (Wright & Beaver, 2005). Second, offspring of antisocial parents are at risk for being reared in disadvantaged environments (Farrington & Welsh, 2007). Including a measure of criminal father therefore helps to control for the potential genetic predispositions passed from parent to child, and it also helps to control for the deleterious social factors associated with having antisocial parents (Moffitt, 2005). During Wave 3 interviews, respondents were asked whether their biological father had ever served time in prison or jail. This variable was coded as a dichotomous dummy variable, where 0 = *no*, 1 = *yes*.

Socialization Measures

Maternal involvement. Adequate levels of stimulation are needed for neurocognitive skills to develop to their potential (Restak, 1986, 2001). The main sources of stimulation, particularly early in life, are found within the family. The Add Health data contain a 10-item maternal involvement index that taps into the amount of time the mother and her child spent together. During Wave 1 interviews, adolescents were presented with a list of 10 activities and were asked which ones they had participated in with their mother in the past month. For example, respondents were asked whether they had gone shopping with their mother, whether they had played a sport with their mother, and whether they had worked on a project for school with their mother. Following prior research (Beaver et al., 2007), each item was coded dichotomously (0 = *no*, 1 = *yes*) and responses to each item were then summed together to form the maternal involvement index ($\alpha = .55$).

Maternal disengagement. Neuropsychological deficits may be caused, at least in some instances, by parental neglect, maltreatment, and withdrawal (Tarter, Hegedus, Winsten, & Alterman, 1984). We examined this possibility by including a maternal disengagement scale. The Add Health data included five items that measure the extent to which the mother extends warmth and affection to her child. During Wave 1 interviews, for example, adolescents were asked whether their mother is warm and loving toward them and whether they are satisfied with their relationship with their mother. Responses to each of the questions were added to form the maternal disengagement scale, where higher scores indicated more maternal disengagement ($\alpha = .83$).

Maternal attachment. There is some evidence indicating that neuropsychological development is facilitated by parental bonding and attachment (Restak, 1986). Consequentially, a two-item maternal attachment scale that has been used previously (Beaver et al., 2007) was applied. During Wave 1 interviews, adolescents were asked how close they felt to their mother and how much they thought that their mother cared about them. Responses to these two items were summed and higher scores indicated more maternal attachment ($\alpha = .62$).

Neighborhood disadvantage. Children who live in disadvantaged neighborhoods are disproportionately exposed to various criminogenic risk factors, including subcultures that value aggression (Anderson, 1999), and research has also revealed that disadvantaged neighborhoods are associated with reduction in verbal IQ (Sampson, Sharkey, & Raudenbush, 2008). Moreover, various types of toxins, some of which interfere with normal brain development, are also much more common in the poorest communities (Chen, Matthews, & Boyce, 2002). As a result, a six-item neighborhood disadvantage scale was included in the analyses. During Wave 1 interviews, mothers were asked a series of questions about the conditions of their neighborhood. For example, they were asked whether crime, drug use, and other social problems were a serious concern in the neighborhood where they currently live. Responses to the items were summed together to form the neighborhood disadvantage scale ($\alpha = .73$). Higher scores on this scale indicated more neighborhood disadvantage.

Control Variables

Four control variables—household income, age, gender, and race—were included in the analyses to help avoid model misspecification. Household income was measured during Wave 1 interviews with the mother. Mothers were asked to report the total income of the household before taxes. Age was a continuous variable measured in years. Race (0 = *White*, 1 = *non-White*) and gender (0 = *female*, 1 = *male*) were included as dichotomous dummy variables.

Plan of Analysis

The analysis for this study proceeded by estimating a series of ordinary least squares (OLS) regression equations. In the first set of models, the Wave 1 neuropsychological deficits measure was used as the dependent variable and in the second set of models, the Wave 3 neuropsychological deficits measures was used as the dependent variable. These models were calculated for the full sample and separately by gender. None of the models were plagued by harmful levels of collinearity or multicollinearity.

Results

The analysis for this article began by examining the predictors of neuropsychological deficits measured at Wave 1. The results of the OLS models are presented in

Table 2. Multivariate Regression Equations Predicting Neuropsychological Deficits (at Wave 1) for the Full Sample and by Gender

	Full sample			Male sample			Female sample		
	b	β	*SE*	*b*	β	*SE*	*b*	β	*SE*
Biosocial measures									
Exposure to cigarette smoke	2.54	.09*	.61	2.11	.07*	.89	2.90	.10*	.69
Length of breastfeeding	–1.18	–.14*	.18	–0.95	–.11*	.26	–1.41	–.16*	.26
Birth weight	0.11	.01	.24	0.33	.03	.33	–0.08	–.01	.35
Criminal father	0.95	.02	.87	0.06	.00	1.27	1.58	.04	1.21
Socialization measures									
Maternal involvement	–0.61	–.08*	.16	–0.22	–.03	.23	–0.96	–.13*	.22
Maternal disengagement	–0.12	–.03	.11	–0.02	–.01	.16	–0.20	–.05	.15
Maternal attachment	0.08	.01	.36	–0.00	–.00	.57	0.12	.01	.47
Neighborhood disadvantage	0.01	.00	.08	–0.09	–.02	.11	0.08	.02	.11
Control variables									
Household income	–0.04	–.14*	.01	–0.05	–.16*	.01	–0.03	–.12*	.01
Age	0.20	.02	.18	0.26	.03	.26	0.14	.02	.25
Race	6.20	.21*	.65	6.70	.22*	.95	5.69	.19*	.89
Gender	–0.92	–.03	.61						

*$p < .05$.

Table 2. For the full sample, two biosocial measures—exposure to cigarette smoke and length of breastfeeding—had statistically significant effects on neuropsychological deficits. More specifically, respondents who were exposed to cigarette smoke incurred more neuropsychological deficits, whereas length of breastfeeding was inversely related to neuropsychological deficits. Birth weight and criminal father however failed to maintain a significant association with neuropsychological deficits. Of the four socialization variables, only maternal involvement was related to neuropsychological deficits, where it had a negative effect. Finally, household income was inversely related to neuropsychological deficits, and non-Whites on average received higher scores on the neuropsychological deficits scale than did Whites.

The second model in Table 2 contains the OLS model for male respondents, the results of which are strikingly similar to those reported for the full sample. Exposure to cigarette smoke, length of breastfeeding, household income, and race were once again significant predictors of neuropsychological deficits. Unlike the results generated from the full sample, the effect of maternal involvement failed to reach statistical significance. The last model in Table 2 presents the results for females, which are identical to those of the full sample. For example, exposure to cigarette smoke, length of breastfeeding, maternal involvement, household income, and race were all statistically significant predictors of neuropsychological deficits.

The results thus far tend to suggest that neuropsychological deficits in adolescence are associated with a number of different factors, including exposure to cigarette smoke, length of breastfeeding, and maternal involvement. The question

Table 3. Multivariate Regression Equations Predicting Neuropsychological Deficits (at Wave 3) for the Full Sample and by Gender

	Full sample			Male sample			Female sample		
	b	β	*SE*	*b*	β	*SE*	*b*	β	*SE*
Biosocial measures									
Exposure to cigarette smoke	2.72	.09*	.69	2.88	.09*	1.02	2.52	.08*	.94
Length of breastfeeding	−1.26	−.13*	.21	−0.95	−.10*	.30	−1.55	−.16*	.29
Birth weight	−0.10	−.01	.27	−0.08	−.01	.37	−0.11	−.01	.39
Criminal father	−0.14	−.00	.99	−1.06	−.02	1.46	0.52	.01	1.36
Socialization measures									
Maternal involvement	−0.60	−.07*	.18	−0.47	−.05	.27	−0.73	−.09*	.25
Maternal disengagement	0.04	.01	.12	0.10	.02	.18	−0.00	−.00	.17
Maternal attachment	0.26	.02	.40	0.41	.02	.65	0.15	.01	.52
Neighborhood disadvantage	−0.10	−.03	.09	−0.21	−.05	.13	−0.01	−.00	.12
Control variables									
Household income	−0.03	−.10*	.01	−0.04	−.12*	.01	−0.03	−.09*	.01
Age	−0.23	−.02	.20	−0.13	−.01	.29	−0.31	−.03	.28
Race	8.29	.24*	.73	9.58	.27*	1.09	7.04	.21*	.99
Gender	−1.04	−.03	.69						

*$p < .05$.

remains however whether the effects of these independent variables extend forward into adulthood. To examine this issue, the Wave 3 neuropsychological deficits variable was used as the dependent variable in a series of OLS regression models. It is to be kept in mind that the Wave 3 neuropsychological deficits variable was measured approximately 7 years after Wave 1. The results of these models are reported in Table 3. A close inspection of Table 3 reveals the exact same pattern of results as those reported in Table 2. In other words, the predictor variables of the Wave 1 neuropsychological deficits measure were identical to the predictor variables of the Wave 3 neuropsychological deficits measure.[1]

Discussion

An impressive amount of empirical research has demonstrated that various measures of neuropsychological deficits are robust predictors of antisocial behaviors (Moffitt, 1990, 1993, 2006; Morgan & Lilienfeld, 2000). To prevent the emergence of criminal and delinquent involvement, crime prevention programs should thus focus on reducing neuropsychological deficits from surfacing. Unfortunately, relatively little criminological research has explored the factors that are implicated in the development of neuropsychological deficits. The current study addressed this gap in the literature by examining the effects that a range of environmental and biological variables had on the neuropsychological functioning measured in adolescence and in young adulthood.

Analysis of the Add Health data revealed three main findings. First, respondents who were exposed to environmental tobacco smoke during adolescence had more neuropsychological deficits during adolescence and during adulthood than respondents who were not exposed to tobacco smoke. It is widely accepted that exposure to tobacco smoke in utero is associated with reduced cognitive skills and neuropsychological functioning (McGloin et al., 2006). The results of the current study suggest that exposure to cigarette smoke in adolescence—not just in utero—may also be a risk factor for neuropsychological deficits. What is particularly important is that the effect that exposure to tobacco smoke had on neuropsychological functioning did not erode over time, meaning that events that occur during adolescence have reverberations that are felt later in life. We should note that the exposure to tobacco smoke measure was relatively weak in that there is reason to believe that exposure to tobacco smoke has its strongest effects in utero (Willford, Day, & Cornelius, 2006). Unfortunately, the Add Health data did not include measures of prenatal exposure to cigarette smoke and thus we were forced to use a measure of postnatal exposure to cigarette smoke.

Second, the amount of time that the respondent was breastfed was a consistent predictor of neuropsychological functioning during adolescence and during adulthood for males and females alike. All else equal, respondents who were breastfed for a longer duration had fewer neuropsychological deficits. These findings draw attention to the importance of early intervention programs that focus on the prenatal and early postnatal time period. It is during these times that the human brain is developing at an astonishing rate. Whether the brain matures to its potential is guided in large part by environmental factors that are present early in life. Breastfeeding is just one of many different factors that have been shown to promote and foster normal brain development. Of course, it should be noted that the effects of exposure to smoke and breastfeeding on neuropsychological deficits cannot be concluded to be causal effects. It could be the case that important confounders, such as genetic factors, would render these effects nonsignificant. Replication studies need to be conducted to determine whether the results reported here would be observed in different samples.

Exactly how breastfeeding is associated with increased neuropsychological functioning is not completely understood. On the one hand, some scholars have argued that the nutrients found in breast milk are much better able to support brain growth when compared to the nutrients found in formula (Kramer et al., 2008). On the other hand, there is also the possibility that it is not breast milk per se that increases neuropsychological functioning but rather the bonding that occurs between mother and child throughout the breastfeeding process (Walsh & Yun, 2009). These two countervailing explanations remain a matter of debate and point to the need for future researchers to attempt to disentangle these two effects.

The third main finding was that maternal involvement was inversely related to levels of neuropsychological functioning, where greater levels of maternal involvement were associated with fewer neuropsychological deficits. This effect however was confined only to females. Interestingly, none of the other socialization measures,

including maternal disengagement, maternal attachment, and neighborhood disadvantage, were related to neuropsychological functioning.

The results of this study should be interpreted with caution in light of a number of different limitations. To begin with, the measure of neuropsychological functioning was assessed only with the PPVT. Although standardized verbal tests have been used before as a way to measure neuropsychological deficits (Lynam et al., 1993; Moffitt, 1990; Moffitt et al., 1994; Piquero, 2001; Ratchford & Beaver, 2009), future researchers should begin to measure neuropsychological functioning in different ways to examine the effectiveness of this measurement strategy. Unfortunately, the Add Health data did not include any other measures of neuropsychological deficits and these data also did not include measures of neuropsychological deficits early in life. It would have been ideal to examine the various predictors of neuropsychological deficits measures in infancy or early childhood.

Another limitation in this study was that the measures of length of breastfeeding and birth weight were retrospective items, which necessarily raise questions about the validity of these items. Future research should address this possibility by using longitudinal samples that include prospective measures of breastfeeding, birth weight, and other pre- and postnatal events. An additional drawback was that the measure of neighborhood disadvantage was based on maternal reports, which may have attenuated the effect of neighborhood conditions on neuropsychological deficits. Finally, we should note that neuropsychological functioning is the result of a multitude of factors (Moffitt, 1993). The current study was only able to examine the effects of a handful of these potential factors and so future researchers should begin to examine a much broader range of variables that span multiple levels of analysis.

Neuropsychological functioning is just one of myriad risk factors that are involved in the development of antisocial behaviors. Even so, reduced neuropsychological functioning is one of the most consistent predictors of antisocial behavior, making it an attractive risk factor for crime prevention programs to target for change. However, to change a risk factor, something must be known about its potential causes. This study shed some light on that issue and revealed that neuropsychological functioning can be affected via environmental exposure to cigarette smoke, duration of breastfeeding, and maternal involvement. Prevention and intervention efforts should cautiously evaluate these findings and then begin to map out the various ways to reduce neuropsychological deficits. Such an approach would likely reduce the number of serious violent offenders.

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Note

1. For Tables 2 and 3, we examined whether the coefficients were significantly different between males and females by using the *z*-score equation discussed in Paternoster, Brame, Mazerolle, and Piquero (1998). The results did not reveal any statistically significant differences between males and females.

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