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THE EFFECTS OF PRENATAL PROBLEMS, FAMILY FUNCTIONING, AND NEIGHBORHOOD DISADVANTAGE IN PREDICTING LIFE-COURSE-PERSISTENT OFFENDING

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Research examining Moffitt's dual taxonomy theory of offending has generally supported the idea that neuropsychological deficits interact with disadvantaged familial environments to predict life-course-persistent offending. Most research, however, has neglected to investigate the power of this interaction across different neighborhood and racial contexts. Using data extracted from the National Longitudinal Survey of Youth, Moffitt's biosocial hypothesis is tested across different neighborhood and racial contexts. The findings indicate that the biosocial interaction predicts life-course-persistent offending only among non-Whites in disadvantaged neighborhoods. Stated differently, macro-level structural factors appear to moderate the effects of individual and family risks. That poor non-Whites reside in neighborhoods that are ecologically distinct from those in which poor Whites reside exacerbates the criminogenic effects of individual-level deficits and family disadvantage.

Keywords: prenatal problems; family; neighborhood; life-course-persistent; developmental

Three traditions of theorizing and research characterize the literature on the causes of crime (Lynam et al., 2000; Piquero, Moffitt, & Lawton, 2005; R. J. Sampson, 1997). Macro-social theories ask what it is about the nature of ecological or geographical units that accounts for differential rates of crime. Beginning with the work of the social ecologists in the first half of the 20th century (Park, 1926; Park, Burgess, & McKenzie, 1925; Shaw & McKay, 1942), a rich tradition has emerged linking crime rates to the effects of community structural characteristics (e.g., poverty, population density, family disruption, and residential instability) and the organization of community social relations (e.g., social cohesion, informal social control) through which they may be mediated (Bursik, 1986; R. J. Sampson, 1997; R. J. Sampson & Groves, 1989).

The other two theoretical traditions share a common focus on identifying individual-level differences that differentiate delinquents and nondelinquents (i.e., micro-level theories). One tradition attends to the role of personal characteristics in the origins of criminal behavior. This approach, which traces its roots to the simplistic and long-since-discredited physiological theories that appeared around the turn of the 20th century (Goddard, 1914; Lombroso, 1876), has recently enjoyed a revival. Far from reductionist and bearing little resemblance to biological theories of the past, these new approaches draw on advances in

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neuroscience and attend to the ways that brain physiology, and the functioning of the central and autonomic nervous systems, influence learning, emotional reactivity, and psychological traits.

The second micro-level approach, and that which accounts for the bulk of the “individual difference” theories, focuses on features of the social environment. Twentieth-century criminologists have generated a number of explanations of crime (e.g., strain, social learning, social control) that draw attention to the nature of individual ties to social groups as well as the social processes in which individuals are involved, in accounting for the development of delinquent and criminal behavior. Researchers in this tradition have identified an impressive array of individual-level predictors that are consistently related to delinquency behavior (e.g., parental supervision, parental disciplinary practices, parental attachment, school attachment, school performance, and ties to deviant peers).

This fairly well-entrenched tripartite approach to understanding the etiology of delinquency and crime has not gone unchallenged. For at least three decades, there have been a number of attempts at theoretical integration (see, e.g., Messner, Krohn, & Liska, 1989). Although most of these integrations have involved attempts to marry various micro-level social theories (e.g., Cullen, 1994; Elliott, Ageton, & Canter, 1979; Thornberry, 1987), integrations that cross the traditional theoretical and disciplinary boundaries have also been proposed. Some of the most promising are the biosocial theories that focus on neuropsychological problems that place individuals at risk for maladaptive responses to adverse social environments (e.g., Eysenck, 1977; Mednick, 1977; Moffitt, 1993). In this framework, neuropsychological problems may contribute to delinquent and criminal behavior, depending on the nature of the social environment. In other words, it is the interaction of physiological features and social conditions that enhances the risk of delinquent behavior. Research has thus far produced some fairly compelling evidence of interactions between neuropsychological factors and family environments in the co-production of juvenile delinquency and adult crime (Brennan, Mednick, & Raine, 1997; Gibson & Tibbetts, 2000; Raine, Brennan, & Mednick, 1994; for a review of this literature, see Raine, 2002). The most robust effect involves the interaction of neuropsychological deficits (frequently stemming from oxygen deprivation in utero or at birth) and disadvantaged family environments (e.g., those characterized by low socioeconomic status [SES], parental criminality, harsh or erratic discipline) to increase the risk of aggressive, and especially violent, behavior. This research helps to explain why only some of the individuals exposed to aversive home environments, and only some of the individuals with neuropsychological impairments, engage in delinquency and crime.

Concomitant with this trend toward integration of biological and social theories at the individual level, a movement has also developed to integrate the micro and macro levels of analysis (R. J. Sampson, 1997; Wikstrom & Loeber, 2000). An integration of micro and macro theories recognizes the possibility that the relationships among individual traits, family characteristics, and offending may be conditioned by the community context in which individuals and families are embedded. It is increasingly recognized that multilevel multidisciplinary models that integrate biological-, family-, and community-level influences have the potential to provide a much more complete accounting of crime. For example, Tonry, Ohlin, and Farrington (1991) have observed that “most individual-level research is inadequate because it neglects variation in community characteristics, while community-level research fails to take account of individual differences” (p. 42). Despite this recognition, there has been limited theorizing and research on how the additive and joint effects of these influences work to produce crime.

This article contributes to the integration of individual-biological, individual-social, and macro-level theories by exploring (a) whether the confluence of certain individual-level physiological and family characteristics increases the explanatory power of these variables to explain delinquent behavior and, if so, (b) whether the effects of Person $\times$ Family interactions are exacerbated or tempered by conditions within the larger community environment. These goals are accomplished by testing Moffitt's (1993) biosocial theory of life-course-persistent offending both within and across different neighborhood contexts.

MOFFITT'S BIOSOCIAL THEORY

One of the most promising of the integrated biosocial approaches is that put forth by Terrie Moffitt (1993, 1994), who distinguishes between adolescent-limited and life-course-persistent offenders in explaining the aggregate age-crime curve. According to Moffitt, the vast majority of delinquents are normal adolescents who participate in offending for a relatively short period. Frustrated by the mismatch between their adult biological development and societal expectations that they refrain from adult behaviors, they commit illegal acts symbolic of claims to adult status (e.g., curfew violations, auto theft, underage drinking, retail theft, drug use), from which they desist once they gain access to adult roles. Moffitt refers to this group as the "adolescence-limiteds."

A much smaller group of offenders, referred to as the "life-course-persistents," begins to manifest conduct problems in very early childhood and continues to engage in antisocial behavior well into adulthood. Moffitt attributes life-course-persistent offending to the interface between neuropsychological deficits and disadvantaged social environments. More specifically, Moffitt suggests that neuropsychological impairments, which are most commonly generated in utero or in the early postnatal period, disrupt normal development of verbal and executive functions of the brain. These disruptions have negative effects on learning, reasoning, memory, problem solving, temperament, and impulse control, each of which increases the propensity toward antisocial, and especially aggressive and violent, behavior. This propensity may be ameliorated or exacerbated by the home environment. Raised in supportive family environments by parents who have both the emotional resources to cope with children whose temperament and learning problems render them difficult to manage and the financial resources to obtain remedial interventions, these children may develop fairly well. However, when children with neuropsychological vulnerabilities are reared in disadvantaged or adverse home environments (e.g., by parents who lack the emotional resources to cope constructively with a difficult child, who withdraw or react with hostility to children's oppositional behaviors, or who lack the financial means to afford remedial assistance), youth problems tend to be amplified over time.

Moffitt argues that a child with even mild neuropsychological dysfunction living in an adverse environment for childrearing is at risk for developing a constellation of negative traits—poor self-control, ill temper, low cognitive ability, and hyperactivity. These traits are likely to strain social relationships and provoke hostility and rejection from both adults and conventional peers. An invidious pattern of negative interaction between these youths and those around them prevents them from acquiring and practicing prosocial behaviors (see also Patterson, 1976, 1995; Patterson, Dishion, & Bank, 1984), eventuating in the gradual elaboration of a style of hostile attributions and responses that pervades all domains of

behavior. By the time these youths reach adolescence, frequently they have sabotaged the formation of prosocial skills and relationships and initiated a pattern of serious and violent offending that persists into adult life.

To date, several tests of Moffitt's theory have provided support for the notion that the interaction of child neuropsychological vulnerabilities and adverse family environments predicts precocious, persistent, and violent behavior (e.g., Moffitt, 1990; Moffitt, Lynam, & Silva, 1994; Piquero & Tibbetts, 1999; Tibbetts & Piquero, 1999). Moffitt (1993, 1997) has also suggested, however, that the vulnerability of children with neuropsychological risks to criminogenic environments may also extend beyond the home. More specifically, she argues that both neuropsychological risks and risk factors in the immediate family are likely to be exacerbated by poverty and by life in disorganized, economically disadvantaged neighborhoods.¹ Nevertheless, few studies of the relationship between individual or family characteristics and offending have explored the possibility that neighborhood environments may exacerbate or ameliorate these risks.

To be sure, research on the effects of neighborhood context on individual-level outcomes is in its infancy. There is some evidence that neighborhood features are at least modestly related to a variety of youth behavioral outcomes, including cognitive development (Brooks-Gunn, Duncan, Klebanov, & Sealander, 1993; Chase-Lansdale & Gordon, 1996), school performance (Garner & Raudenbush, 1991; Halpern-Felsher et al., 1997), and peer-reported aggression (Kupersmidt, Griesler, DeRosier, Patterson, & Davis, 1995). In addition, researchers have reported significant but weak correlations between neighborhood context and delinquency at the individual level of analysis (Elliott et al., 1996; Gottfredson, McNeill, & Gottfredson, 1991; Loeber & Wikstrom, 1993).

Although these studies are important, they do not address the question of whether the relationship between offending, individual vulnerabilities, and family disadvantage is affected by neighborhood conditions. If Moffitt is correct, the effect of neuropsychological risk on offending will be contingent on both family and neighborhood characteristics.

THE EFFECTS OF NEIGHBORHOOD ON INDIVIDUALS AND FAMILIES

There are many reasons to expect that the likelihood of offending for neuropsychologically vulnerable youths from adverse home environments may be different for those living in wealthy and well-organized neighborhoods than for those living in poor and socially disorganized ones (Wikstrom & Loeber, 2000). A number of mechanisms have been proposed through which neighborhoods might affect individual-level outcomes (Jencks & Mayer, 1990; Leventhal & Brooks-Gunn, 2000, 2003; R. J. Sampson, 1997). For example, R. J. Sampson (1997) proposed a social capital/collective efficacy model that is unique in linking neighborhood context, family characteristics, and individual-level outcomes. He suggests that social capital—defined as the extent to which adults are involved in stable social networks with the parents and relatives of their children's friends—is a critical feature of neighborhoods that affects parental socialization. These networks transmit norms regarding parenting and supervision and also share parental responsibility for the informal social control (monitoring and discipline) of children. Thus, family management practices—level of parental supervision, parental warmth, parental rejection, parent-child attachment—are mediated by the social organization of the community. Parenting styles and levels of child

supervision are not only a function solely of the characteristics and interactional patterns of parents and children but also are influenced by the larger community context in which families are embedded. Sampson hypothesizes that because high levels of poverty, family disruption, and residential instability tend to weaken social networks, the collective socialization of children in disadvantaged neighborhoods suffers.

Several studies have reported that parenting in disadvantaged families tends to be characterized by largely ineffective coercive and punitive strategies, including greater use of restriction and physical punishment, the issuance of commands without explanation, and lesser use of praise and encouragement (Earls, McGuire, & Shay, 1994; Halpern, 1990; Hanson, McLanahan, & Thomson, 1997; McLoyd, 1990). The critical issue is whether poor parental management is mediated by the ecological realities of the neighborhoods in which disadvantaged families reside. There is some evidence that it is. Coulton (1996) and Jarrett (1997) have indicated that parents adjust their parenting style to conform to neighborhood norms. In a qualitative study of two Philadelphia neighborhoods, Furstenberg (1993) reported that in a neighborhood where social networks among residents were strong, children were socialized not only by their own parents but also by friends, relatives, and neighbors. Furthermore, parents were assisted in "under the roof" management of their children by the mutual reinforcement of other neighborhood parents. In contrast, in less socially cohesive neighborhoods, families tended to isolate themselves from the surrounding community and to adopt individualistic styles of parental management. Lacking networks of social support, their children received less supervision and monitoring.

OFFENDING AND INDIVIDUAL VULNERABILITIES, FAMILY CHARACTERISTICS, AND NEIGHBORHOOD CONTEXT

Thus far, three studies have addressed the issue of whether neighborhood context conditions the effect of individual-level or family-level risk factors for delinquent or criminal involvement.² Using data from the Pittsburgh Youth Study, Lynam et al. (2000) examined the effect of impulsivity on boys' offending across neighborhoods in studies that employed both objective (i.e., SES) and self-reported measures of neighborhood quality. After controlling for family and sociodemographic factors, they found that nonimpulsive boys were at no greater risk for delinquency in poorer than in better-off neighborhoods; however, for impulsive boys, the risk of offending was exacerbated by residence in a poorer neighborhood (perhaps because of lower levels of informal social control).

Rankin and Quane (2002) explored the effects of neighborhood context and parental monitoring on offending among African American children in poor and mixed-income Chicago neighborhoods. Parental monitoring was significantly higher in neighborhoods with greater social cohesion, and youths whose parents monitored their activities more effectively had lower involvement in delinquency. Consistent with Sampson's predictions, the effects of neighborhood social capital on delinquency were mediated through parental management.

Finally, Piquero et al. (2005) tested Moffitt's thesis in a much more comprehensive way by examining the effects of early-life personal-level, family-level, and neighborhood-level variables on life-course-persistent offending (measured by frequency of arrests) among a group of Baltimore adults. They measured neuropsychological risk in terms of low birth

weight; family adversity in terms of maternal SES, age, and marital status at childbirth; and neighborhood disadvantage in terms of income, unemployment, education, female-headed households, poverty status, welfare status, and percentage Black. They found that, although neither individual risk nor neighborhood risk independently predicted life-course-persistent offending, individual and family risk combined to predict offending selectively among non-Whites. Furthermore, the effect of this combination was magnified for non-Whites in disadvantaged neighborhoods. The findings of this study sensitize us to the fact that neighborhood disadvantage is especially pronounced among non-Whites compared to Whites (Massey & Denton, 1993; R. J. Sampson & Wilson, 1995; Wilson, 1987) and counsels that research should continue to explore the nature of biosocial interactions across both neighborhood and race.

In sum, although there is good reason to believe that the effects of individual-level risk factors for delinquency may be conditioned by both family-level and neighborhood-level risks, there is a real paucity of research in this area. In an effort to contribute to the growing knowledge base of multilevel effects, in this study the separate and joint impacts of biological risk, family risk, and neighborhood disadvantage are explored in a national sample of youths.

CURRENT FOCUS

In the preceding discussion, it has been argued that the effects of the interaction between pre- and perinatal problems and family disadvantage may be exacerbated in disadvantaged neighborhoods. To be sure, this idea is not novel. In fact, in Moffitt's previous work (1997), she commented on this possibility and noted the absence of research on the subject, saying, "There is a great need for empirical data about how neuropsychological variables related to delinquency in different types of neighborhoods" (p. 155). Only recently have efforts been made to conduct research focused on how the neighborhood context conditions individual differences and influences life-course-persistent offending specifically (Piquero et al., 2005) and the development of children in general (Klebanov, Brooks-Gunn, Chase-Lansdale, & Gordon, 1997; Lynam et al., 2000). Accordingly, this study built on recent attempts to test Moffitt's biosocial hypothesis within and across different neighborhood contexts. Notably, these efforts advance the extant research in the following ways.

First, this study used a national sample of relatively high-risk adolescents. This sample is particularly noteworthy given that most previous research has relied on smaller samples that are less representative. In addition, a higher risk sample should yield a greater number of life-course-persistent offenders, supporting statistical analyses that might not be possible otherwise. Second, in categorizing offenders as either life-course-persistents or adolescence-limiteds, this study made use of a self-report measurement protocol. In doing so, two of Moffitt's (1994) criteria for identifying life-course-persistent offending were used: (a) involvement in violent offending and (b) involvement in offending over multiple measurement points. Third, following the advice of previous researchers who examined neighborhood contextual effects (Furstenburg, 1993), this study used perceptual measures of neighborhood disadvantage. Such measures are particularly advantageous in understanding youths' views of, and responses to, criminogenic influences within their neighborhoods. Finally, this study examined the preceding hypotheses across categories of race. This effort

is important in this regard given that no previous research has tested Moffitt's theory of life-course-persistent offending within and across different neighborhood contexts using perceptual measures of neighborhood disadvantage in a national sample of relatively high-risk youths.

METHOD

Data for this research were drawn from the National Longitudinal Survey of Youth, Child–Mother data (hereafter referred to as the NLSY). The NLSY is a prospective, longitudinal data-collection effort supported by the U.S. Department of Labor and administered by the National Opinion Research Center. First administered in 1979 to 12,686 individuals aged 14 to 21, the main purpose of the original NLSY was to assess youths as they completed high school and entered the workforce. A separate biennial data collection effort began in 1986 that included detailed assessments of each child born to the girls and women in the original NLSY data cohort (referred to as the NLSY Child–Mother data). As of January 1, 1998, a total of seven waves had been administered to individuals who range in age from birth to 27 years (Center for Human Resource Research, 2000).

PARTICIPANTS

To investigate the hypotheses, this study restricted the analyses to a subsample of 513 individuals who had reached the age of 15 by 1994 and who provided valid interviews during the years 1994, 1996, and 1998. These restrictions were placed on the sample for two reasons: First, individuals who reached the age of 15 by December 31, 1994—termed the “young adults”—were the only individuals in the main sample who received a self-administered questionnaire that contained detailed delinquency and crime measures, including violent behavior. It should also be noted that these items were not available in the survey prior to the 1994 wave. Second, although the rate of sample attrition in this study (never exceeding 6.7% in any one wave) is consistent with similar longitudinal research designs investigating adolescent problem behavior (see Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995; Smith, Lizotte, Thornberry, & Krohn, 1995), the restriction to respondents who completed all three interviews circumvented missing data problems (see Jessor et al., 1995).

We examined the effects of the attrition of the 467 participants who were interviewed in 1994 but not in one or more of the two subsequent waves. In particular, comparisons were made on the main variables of interest across the final sample and the attrition subsample. No significant differences emerged between the two samples, suggesting that those excluded experienced similar levels of prenatal problems, family disadvantage, and neighborhood disadvantage. In addition, we examined the differences across the two samples on the standard demographic measures of age, race, and sex. Although no race differences emerged, the attrition subsample possessed significantly more males and older individuals. Despite these differences, the intercorrelations among the measures were similar for the final sample and the attrition subsample. Therefore, although there are modest differences between the sample used in this research and the attrition subsample, relationships among the variables used in the subsequent analyses most likely would not have been significantly different had the attrition not occurred.

DEPENDENT VARIABLE

Moffitt argues that life-course-persistent offending can be manifested in a number of ways. The measure of life-course-persistent offending used in this study combines two of Moffitt's parameters of antisocial behavior, self-reported involvement in violent offending and chronicity in offending (see Moffitt, 1994). To measure violent offending, at each of the three waves, individuals were asked whether, in the preceding year, they had "hit or seriously threatened to hit someone," "attacked someone with the idea of seriously hurting or killing them," and "hurt someone badly enough to need bandages or a doctor." The responses for each year were summed and collapsed so that involvement in the incident equaled 1 and no involvement equaled 0. The chronicity dimension of the life-course-persistent offending measure was operationalized using individuals' involvement in any offending (i.e., violent or nonviolent) in at least two of the three waves (see the appendix for the list of these items). In sum, an individual is categorized as a life-course-persistent offender (1) if he or she self-reported a violent offense in at least one wave *and* self-reported some offending in at least two waves of data collection. Those not meeting this threshold but reporting at least some nonviolent delinquency were categorized as adolescence-limited offenders (0).

INDEPENDENT VARIABLES

Prenatal Problem Index. Research has shown that disruption to the neuropsychological development of the fetus can result from maternal involvement in risky behaviors during pregnancy (Kandel, Brennan, & Mednick, 1989; P. D. Sampson et al., 1997; Wakschlag & Hans, 2002). Among these, cigarette smoking and alcohol use are two of the most important. An abundance of research has documented that mothers who smoke during pregnancy place their children at an increased risk of crime in general (e.g., Brennan, Grekin, Mortensen, & Mednick, 2002; Rasanen et al., 1999) and life-course-persistent offending in particular (Brennan, Grekin, & Mednick, 1999; Gibson, Piquero, & Tibbetts, 2000; Piquero, Gibson, Tibbetts, Turner, & Katz, 2002). Moreover, the relationship between maternal smoking and childhood behavior survives statistical controls for a wide range of social factors, including SES, maternal age, maternal IQ, home environment, maternal rejection, parenting style, and parental criminality (for a review and discussion, see Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002).

Although the links are less strong and consistent, research suggests that maternal alcohol use during pregnancy is also associated with a host of negative outcomes, including lower IQ scores, learning problems, cognitive deficits, and behavioral problems (e.g., attention deficit hyperactivity disorder, conduct disorder, oppositional-defiant behaviors, and delinquency; see Hill, Lowers, Locke-Wellman, & Shen, 2000; Streissguth, Barr, Brookstein, Sampson, & Olson, 1999; West, Chen, & Pantazis, 1994).

Because the data lack direct measures of neuropsychological deficits, consistent with prior research (see Piquero & Tibbetts, 1999; Tibbetts & Piquero, 1999), measures of prenatal problems were used as a proxy for neuropsychological deficits. To measure prenatal problems, a summative index was constructed based on items measuring maternal smoking and drinking behaviors during pregnancy. Consistent with previous research examining prenatal risks (Raine, Brennan, & Mednick, 1997), this measure combines a dichotomous measure of maternal cigarette smoking with a dichotomous measure of maternal alcohol use during pregnancy to create the Prenatal Problems Index. Those reporting any involvement in

these behaviors during pregnancy received a score of 1 and those not reporting involvement received a score of 0. Therefore, the Prenatal Problems Index will range from 0 to 2, with higher values being indicative of greater prenatal risk.

Family Disadvantage Index. Individuals evidencing neuropsychological deficits are often born into families that are incapable of providing environments conducive to the healthy growth and development of their children. As Moffitt (1997) indicated, "Vulnerable infants are disproportionately found in environments that will not be ameliorative because many sources of neural maldevelopment co-occur with family disadvantage or parental deviance" (p. 126). Consistent with prior research, this study measures family disadvantage using the sum of four dichotomized items collected early in the youth's life (Kolvin, Miller, Fleeting, & Kolvin, 1988; Rutter, 1978; Stanton, McGee, & Silva, 1989). These items include (a) age of the mother at childbirth (1 = *younger than 18 years*, 0 = *18 years and older*), (b) maternal educational attainment at childbirth (1 = *less than high school graduate*, 0 = *at least high school graduate*), (c) maternal poverty status at childbirth (1 = *in poverty*, 0 = *not in poverty*), and (d) maternal marital status at childbirth (1 = *not married*, 0 = *married*). Higher scores on the family disadvantage measure are indicative of more disadvantaged familial environments.

Neighborhood Disadvantage. Measuring neighborhood disadvantage is a particularly daunting task considering the absence of a clear consensus in the literature regarding the relative benefits of objective measures (e.g., census data) and perceptual measures (e.g., reports from respondents regarding their immediate neighborhood surroundings). Each measurement strategy has advantages. Objective measures provide insight into the structural characteristics of a neighborhood (e.g., population density, ethnic heterogeneity, SES). Alternatively, perceptual measures provide insight into social ties that members with neighborhoods have with one another. Furthermore, to the extent that behavior is affected by individual perceptions of neighborhood conditions, rather than the objective conditions themselves, perceptual measures are preferable. Because Moffitt's theory is particularly concerned with the extent to which neighborhood socialization experiences exacerbate disadvantaged familial environments, this study measures neighborhood disadvantage in terms of respondents' perceptions. This falls comfortably in line with Furstenberg's (1995) research, which suggests that familial perceptions of the neighborhood context influenced youths' behavioral outcomes.

Similar to prior research (see Pratt, Turner, & Piquero, 2004; Turner, Piquero, & Pratt, 2005) that measured neighborhood disadvantage, this study uses maternal reports of seven items collected during Wave 4 (1992), when offspring were at a mean age of 13. Mothers responded to seven items assessing a variety of neighborhood problems. For example, they were asked to respond using a three-category response set to items such as "people in your neighborhood don't have enough respect for rules or laws," "too many parents in your neighborhood don't supervise their children," "there is not enough police protection in your neighborhood," and "people keep to themselves and don't care what goes on in the neighborhood." Responses were categorized as follows: 1 = *big problem*, 2 = *somewhat of a problem*, and 3 = *not a problem*. Items were recoded so that higher scores reflected a greater degree of neighborhood disadvantage. Analyses of the overall scale indicated that it was highly reliable ($\alpha = .88$), and factor analyses indicate that it formed a unitary factor ($KMO = .82$).

In many of the subsequent analyses, this study examined the biosocial interaction (between prenatal problems and family disadvantage) across different neighborhood contexts. That is,

this study investigated the hypothesis that the biosocial interaction is intensified in bad neighborhoods while having weaker, if not insignificant, effects in better neighborhoods. To capture unique neighborhoods, and consistent with past risk-related research (see Farrington & Loeber, 1999), this study dichotomized responses on the Neighborhood Disadvantage scale, categorizing those falling in the top quartile of the neighborhood measure as bad neighborhoods (1) and those falling below this threshold as good neighborhoods (0).³

Biosocial interaction. At the core of Moffitt's theory is the importance of a biosocial interaction between neuropsychological risks and disadvantaged familial environments. This study computed this interaction term by mean-centering the raw scores of the component factors and multiplying these factors. This method produces an interaction term that is not collinear with each of its component factors (Aiken & West, 1991; Jaccard, Turrisi, & Wan, 1990). Higher scores on the interaction term correspond with a higher degree of risk.

CONTROL VARIABLES

This study controlled for the age, sex, and race of the individual. Specifically, age was a continuous measure, capturing respondents' age in 1998. Sex was coded as females = 0 and males = 1. Race was also coded dichotomously: White = 0 and non-White = 1. Table 1 contains descriptive statistics for the full sample and race-specific samples (White/non-White). Presenting the data in such a format allows one to compare the distribution of each of the variables across categories of race and to illustrate the bivariate relationships in preparation for the race-specific analyses presented below.

ANALYTIC STRATEGY

The subsequent analysis proceeds in three stages. First, the bivariate differences on the key variables are examined across categories of offending. Second, Moffitt's biosocial hypothesis is tested in general and across different neighborhood contexts. Presenting the data in such a format allows for an examination of the effect of the biosocial interaction in the sample as a whole as well as examining its effects across types of neighborhoods. Third, Moffitt's biosocial hypothesis is tested across subgroups defined by both neighborhood disadvantage and race. It is important to note that consistent with prior research (see Tibbetts & Piquero, 1999), investigations of Moffitt's hypothesis are completed on the offender groupings only (adolescent-limiteds versus life-course-persistents).

RESULTS

Table 1 presents the means and standard deviations of each of the variables for the nonoffenders as well as for each category of offender. Offender groups differed significantly on sex, prenatal problems (and each of its components), and maternal marital status. That is, compared to the adolescence-limited group, the life-course-persistent group is comprised of a significantly greater proportion of males ($F = 9.818$; $p < .05$), who had higher levels of prenatal problems ($F = 8.012$; $p < .05$) and who were more likely to be born to a nonmarried mother ($F = 4.030$; $p < .05$). It is also noteworthy that, although not significant, differences in the predicted direction existed on each of the remaining independent variables of interest.

TABLE 1: Variables and Descriptive Statistics of Sample

Variable	Nonoffenders (n = 120)		Adolescent-Limited (n = 263)				Life-Course-Persistent (n = 130)				F
			White		Non-White		White		Non-White		
	M	SD	M	SD	M	SD	M	SD	M	SD	
Age (1998)	19.20	0.86	19.00	0.81	19.11	0.83	18.92	0.82	19.21	0.80	2.769
Sex (1 = male)	0.40	0.49	0.48	0.50	0.41	0.49	0.62	0.49	0.68	0.51	9.818*
Prenatal Problem Index	0.52	0.68	0.78	0.80	0.85	0.78	0.80	0.73	0.89	0.75	8.012*
Maternal alcohol use	0.23	0.42	0.39	0.47	0.38	0.51	0.40	0.50	0.42	0.49	6.044*
Maternal smoking	0.27	0.45	0.39	0.50	0.47	0.49	0.40	0.51	0.47	0.48	4.632*
Family Disadvantage	1.54	1.15	1.67	1.16	1.72	1.15	1.77	1.16	1.83	1.19	1.496
Maternal age at childbirth	0.43	0.50	0.45	0.49	0.47	0.51	0.46	0.50	0.50	0.49	0.283
Maternal education	0.37	0.48	0.36	0.47	0.36	0.48	0.34	0.47	0.36	0.48	0.121
Maternal poverty status	0.38	0.49	0.40	0.49	0.42	0.49	0.46	0.50	0.47	0.50	0.948
Maternal marital status	0.34	0.48	0.45	0.50	0.47	0.51	0.50	0.50	0.52	0.49	4.030*
Neighborhood Disadvantage	11.42	3.91	11.65	3.90	11.73	3.94	12.03	3.81	12.11	3.83	0.895
Bad neighborhood (1 = bad)	0.22	0.41	0.09	0.45	0.39	0.48	0.12	0.47	0.46	0.44	2.034
Biosocial interaction	0.03	0.89	−0.11	0.91	−0.15	0.90	0.00	0.85	0.02	0.91	1.765

Note. *F* values represent differences across the three groupings (i.e., nonoffenders, adolescent-limiteds, and life-course-persistents).

* $p < .05$.

As can be seen in Table 1, the life-course-persistent offending group is considerably larger than has typically been reported in prior research. Specifically, life-course-persistent offenders in the present sample represent approximately one third of the offending population; Moffitt (1993) has proffered that this group should constitute 5% to 8% of the offending population. Two explanations are offered to account for this finding. First, the NLSY is comprised of a relatively high-risk sample. The survey oversamples African Americans, Hispanics, and economically disadvantaged Whites—populations that Moffitt has pointed out should evidence the highest proportions of life-course-persistent offenders (Moffitt, 1997). Second, and perhaps more important, the measurement protocol identifying life-course-persistent offenders was based on self-reported offending. Self-reports typically uncover a great deal more delinquency than that reported in official data. Although self-reports of offending have been used in previous research testing Moffitt's theory (see Moffitt & Caspi, 2001; Piquero et al., 2002), they have generally been used in conjunction with official data. That is, no previous research has operationalized life-course-persistent offending relying solely on self-reported offending. It is likely that the use of self-reports, along with a high-risk sample, produced a higher proportion of life-course-persistent offenders than would be expected in the general population using official data.

As a first step in testing the biosocial hypothesis, the estimates of two separate models are presented in Table 2. Model 1 is a base model that examines the influence of the control variables, the Prenatal Problem Index, and the Family Disadvantage Index on life-course-persistent offending. This model indicates that only sex is a significant predictor of life-course-persistent offending ($B = 0.85$; $p < .01$). That is, males have a higher likelihood of engaging in a course of persistent and violent offending.

Model 2 in Table 2 adds the Prenatal Problem $\times$ Familial Disadvantage interaction term into the base equation. Similar to Model 1, this model suggests that, although the effect of the interaction term is in the predicted direction, only sex remains a significant predictor

TABLE 2: Logistic Regression Coefficients Predicting Self-Reported Violence: Models Examining Main and Interaction Effects ($n = 393$)

Variable	Model 1			Model 2		
	B	SE	Exp (b)	B	SE	Exp (b)
Age	-0.08	0.14	0.928	-0.08	0.14	0.928
Sex	0.85**	0.22	2.350	0.86**	0.22	2.353
Non-White	-0.05	0.26	0.955	-0.08	0.25	0.925
Prenatal Problem (PP) Index	0.06	0.15	1.062	0.02	0.14	1.018
Family Disadvantage (FD)	0.12	0.10	1.129	0.10	0.10	1.107
PP $\times$ FD Interaction	—	—	—	0.18	0.12	1.190
Constant	0.02	2.61		0.31	2.63	
Nagelkerke R^2	0.06			0.06		

* $p < .05$. ** $p < .01$.

of life-course-persistent offending ($B = 0.86$; $p < .01$). Thus, the biosocial interaction does not emerge as a significant predictor of life-course-persistent offending in the full offending sample.

THE BIOSOCIAL HYPOTHESIS WITHIN AND ACROSS DIFFERENT NEIGHBORHOOD CONTEXTS

The biosocial hypothesis was then tested within and across different neighborhood contexts. Table 3 presents the findings of the identical equations in Model 2 of Table 2 across different neighborhood contexts. Specifically, two separate equations were estimated to examine the effects of the biosocial interaction separately for those who scored in the top quartile of the Neighborhood Disadvantage scale (i.e., those in the worst neighborhoods) and those below this threshold. If the neighborhood context conditions antisocial behavior, and in light of the findings presented in Table 2, it should be expected that a significant biosocial interaction would emerge in the worst neighborhoods and insignificant effects in the less disadvantaged neighborhoods.

The first set of columns is identical to Model 2 in Table 2 and is only used for comparison purposes. Turning to the neighborhood-specific models, and as predicted, the findings suggest that the biosocial interaction significantly predicts life-course-persistent offending for those residing in the worst neighborhoods ($B = 0.54$; $p < .05$) while having a nonsignificant effect among those living in the less disadvantaged neighborhoods. Once again, being male ($B = 1.12$; $p < .01$) emerged as the only significant predictor of life-course-persistent offending for those residing in the lower three quartiles of the neighborhood disadvantage measures. In short, the effects of the biosocial interaction were exacerbated in the most disadvantaged neighborhoods but were insignificant in less disadvantaged neighborhoods.⁴

THE BIOSOCIAL HYPOTHESIS ACROSS DIFFERENT NEIGHBORHOOD CONTEXTS AND RACIAL BACKGROUNDS

Next, the equations examined above were estimated across groups defined by race and neighborhood context. That is, the biosocial hypothesis was estimated among Whites and non-Whites who resided in the more and less disadvantaged neighborhoods. If Moffitt's theory is correct, the biosocial interaction should significantly predict life-course-persistent offending for Whites and non-Whites in the most disadvantaged neighborhoods, with non-Whites potentially experiencing the strongest effects. The results displayed in Table 4 provide estimates for each of the four groupings.

TABLE 3: Logistic Regression Coefficients Predicting Self-Reported Violence: Full Model and Neighborhood-Specific Models

Variables	Full Model (n = 393)						Neighborhood-Specific Models					
	Top 25% (n = 116)			Lower 75% (n = 277)			Top 25% (n = 116)			Lower 75% (n = 277)		
	B	SE	Wald	Exp (b)	B	SE	B	SE	Wald	Exp (b)	B	SE
Age	0.05	0.13	0.12	1.047	-0.02	0.25	-0.12	0.17	0.01	0.977	-0.12	0.17
Sex	0.70	0.21	10.97*	2.005	0.41	0.40	1.12	0.28	1.02	1.500	1.12	0.28
Non-White	0.15	0.24	0.39	1.163	-0.40	0.68	-0.19	0.29	0.36	0.668	-0.19	0.29
Prenatal Problem (PP) Index	0.08	0.14	0.31	1.081	0.21	0.25	-0.06	0.17	0.71	1.238	-0.06	0.17
Family Disadvantage (FD)	0.14	0.09	2.07	1.147	0.04	0.19	0.16	0.13	0.05	1.041	0.16	0.13
PP × FD interaction	0.16	0.12	1.94	1.178	0.54	0.25	0.02	0.15	4.83*	1.717	0.02	0.15
Constant	-1.60	2.47	0.41		0.11	4.81	0.86	3.24	0.00		0.86	3.24
Nagelkerke R^2	.06				.09		.09				.09	

* $p < .05$. ** $p < .01$.

TABLE 4: Logistic Regression Coefficients Predicting Self-Reported Violence: Race/Neighborhood-Specific Models

Variable	Whites						Non-Whites					
	Good Neighborhood			Bad Neighborhood			Good Neighborhood			Bad Neighborhood		
	B	SE	Exp (b)	B	SE	Exp (b)	B	SE	Exp (b)	B	SE	Exp (b)
Age	-.31	0.29	0.73	2.39	1.75	10.91	0.05	0.22	1.05	-.16	0.26	0.85
Sex	1.78	0.51*	5.92	0.34	1.93	1.41	0.85	0.36*	2.34	0.47	0.43	1.59
Prenatal Problem (PP) Index	-.46	0.32	0.63	0.24	0.90	1.27	0.27	0.21	1.31	0.25	0.27	1.29
Family Disadvantage (FD)	0.34	0.25	1.41	0.66	0.73	1.93	-.06	0.17	0.94	-.04	0.20	0.96
PP × FD interaction	0.13	0.30	1.14	1.53	1.28	4.60	-.036	0.23	0.70	0.61	0.27*	1.84
Constant	4.36	5.55		-46.22	33.59		-2.23	4.28		2.29	4.98	
Nagelkerke R^2	.25			.45			.07			0.11		

* $p < .05$.

Focusing first on the good neighborhoods, the results in Table 4 suggest that being male corresponded with a higher propensity to manifest life-course-persistent offending for Whites and non-Whites ($B = 1.78$; $p < .05$ for Whites; $B = 0.85$; $p < .05$ for non-Whites). Notably, the biosocial interaction was insignificant for both racial categories with good neighborhoods. Turning to the models estimated for those residing in the most disadvantaged neighborhoods, the results indicate that the biosocial interaction significantly predicted life-course-persistent offending only for non-Whites ($B = 0.61$; $p < .05$), though having insignificant effects for Whites. That is, non-Whites living in the most disadvantaged neighborhoods who were at risk because of prenatal problems and familial disadvantage were significantly more likely to be life-course-persistent offenders. The null results of the biosocial interaction for Whites living in disadvantaged neighborhoods, however, should be tempered because of low sample size. More specifically, only 9.6% ($n = 12$) of the Whites resided in the most disadvantaged neighborhoods, compared to 38.8% ($n = 104$) of non-Whites. This latter finding is consistent with much prior research showing that non-Whites are greatly overrepresented in the most disadvantaged neighborhoods (R. J. Sampson & Wilson, 1995; Wilson, 1987).

DISCUSSION

During the past decade, Moffitt's biosocial hypothesis predicting life-course-persistent offending has received a fair amount of empirical scrutiny. Past research has rather consistently found that individuals suffering from early neuropsychological dysfunction and who are raised in disadvantaged familial environments have a higher propensity to manifest life-course-persistent offending (Moffitt, Lynam, & Silva, 1994; Raine et al., 1994, 1997). Although informative, the vast majority of this research has failed to examine the effects of this interaction within and across different neighborhood contexts (but see Moffitt, 1997; Piquero et al., 2005). Investigation of this hypothesis is important given that emerging research is beginning to suggest that individual differences are enhanced or exacerbated within disadvantaged environments (e.g., Lynam et al., 2000). Toward this end, this study made use of a national sample of adolescents to investigate Moffitt's biosocial hypothesis within and across different types of neighborhoods. In addition, guided by the extant literature on race and neighborhood disadvantage (see Massey, 1995; R. J. Sampson & Wilson, 1995; Wilson, 1987, 1991, 1996), this study also explored how Moffitt's biosocial hypothesis fared among Whites and non-Whites in different neighborhood contexts.

The study produced three important findings. First, the biosocial interaction failed to predict life-course-persistent offending within the full sample. Although generally inconsistent with previous research, this finding may be a function of two important considerations: (a) the measurement of life-course-persistent offending and (b) the fact that interaction effects are difficult to uncover with research designs that are not truly experimental in nature. As discussed previously, little research has relied solely on self-reports to operationalize life-course-persistent offending. Rather, efforts have tended to rely on official reports of formal contact with the criminal justice system (i.e., age of first arrest and/or conviction). It is therefore possible that the measurement strategy—based on self-reports—yielded a group of life-course-persistent offenders that included individuals who might not have been categorized as life-course-persistents if official measures of delinquency were used. In fact, prior research

has suggested that predicting life-course-persistent offending is at least somewhat dependent on the measurement strategy used (see Brennan et al., 1999; Piquero et al., 2002). Future research should attempt to replicate this study's use of self-reports to operationalize life-course-persistent offending before definitive conclusions can be drawn.

Second, the biosocial interaction was found to predict life-course-persistent offending for individuals living in the most disadvantaged neighborhoods. In other words, the neighborhood context conditioned the effects of the biosocial interaction. This finding is important because it is consistent with a growing body of research suggesting that individual risks, particularly those occurring early in the life course, are exacerbated in disadvantaged neighborhoods (Lynam et al., 2000). In addition, this study's findings are consistent with previous efforts examining similar hypotheses that relied solely on objective versus perceptual measures of neighborhood disadvantage (Piquero et al., 2005). As such, it appears that regardless of how it is measured (via objective or perceptual measures), the neighborhood context in which a youth is raised is an important determinant of behavioral outcomes (Klebanov et al., 1997).

Third, when the data were analyzed by race and neighborhood context, this study found that the biosocial interaction was a significant predictor of life-course-persistent offending for certain groups of individuals living within the most disadvantaged neighborhoods. Specifically, the biosocial interaction was only significant in predicting life-course-persistent offending for non-Whites living in disadvantaged neighborhoods; each of the remaining categorizations produced null results. Although this finding is somewhat consistent with Moffitt's (1993) predictions that the biosocial interaction would be stronger for non-Whites in disadvantaged neighborhoods, it is somewhat inconsistent given that the interaction should also emerge as being significant for the White population residing within disadvantaged neighborhoods.

This last finding is noteworthy because it suggests that disadvantaged neighborhoods in which non-Whites reside may be qualitatively distinct from disadvantaged neighborhoods in which Whites reside. In fact, non-Whites residing in the most disadvantaged neighborhoods scored almost a full standard deviation higher (i.e., a more disadvantaged neighborhood) on the neighborhood disadvantage measure than Whites residing in disadvantaged neighborhoods ($t = -3.29$; $p = .001$). Moreover, in these same neighborhoods, non-Whites differed significantly ($t = -2.46$; $p = .012$) from Whites on the Family Disadvantage Index, whereas no significant differences emerged on the Prenatal Problem Index ($t = .94$; $p = .35$). Thus, although Whites and non-Whites were not differentially predisposed to sources of prenatal problems, they did experience substantial differences in the social environments in which they were raised. These findings are consistent with prior research on the racial concentration of neighborhood and family disadvantage. In fact, it is reminiscent of R. J. Sampson and Wilson's (1995) search for U.S. cities with a population of 100,000 or more, where the proportion of Blacks living in poverty was equal to or less than Whites and where the proportion of Black single-parent families was equal to or less than Whites. In no city did they find Blacks living in ecological equality with Whites. Indeed, race differences in neighborhood and family disadvantage were so strong that the "worst" contexts in which Whites resided were considerably better off than the average context of Black communities (see also R. J. Sampson & Lauritsen, 1997). As evidenced, this weighty combination of neighborhood and family disadvantage compounds prenatal risk factors selectively among Blacks to increase the risk of violent and persistent offending.

In interpreting the findings, it is important to keep in mind a number of issues. First, because attrition naturally occurs with prospective longitudinal data collection efforts and because individuals falling in the attrition subsample are likely to be the most deviant (see Brame & Piquero, 2003; Cernkovich & Giordano, 1985), these findings might be viewed as a conservative test of the biosocial hypothesis. Although a large proportion of the attrition was based on financial constraints of the survey administration rather than participant self-selection, it must be kept in mind that the most deviant individuals were likely not interviewed in any given wave.⁵ In addition, recall that the sample for the present study was limited to individuals who completed each of the three waves of data collection. It is certainly within the realm of possibility that sampling constraints, attrition, and measures of life-course-persistent offending in combination produced a sample of offenders who are distinct from previous research. Indeed, future research efforts should investigate whether the measurement protocols used in this study provide similar findings.

Second, although the analysis explored the effects of the biosocial interaction among Whites and non-Whites, it did not explore how the pattern of relationships varied among Whites, Hispanics, and other ethnic minorities. Investigating whether the biosocial interaction varies across specific race or ethnic categories would be an appropriate direction for future research. Third, this study measured only two proxies for neuropsychological deficits—maternal drinking and smoking during pregnancy. Future research efforts might want to investigate neuropsychological deficits measured directly through neuropsychological testing in childhood. Fourth, although the measure of familial disadvantage is consistent with extant research, it focuses solely on family structure. In short, it does not capture family dynamics (e.g., parental conflict), including the parent–child interactions that Moffitt and others have identified as critical to child development (Loeber & Stouthamer-Loeber, 1986; Moffitt, 1993). Finally, although this study was longitudinal in nature, only three waves of data were employed covering a 6-year period. Future researchers will surely want to examine individuals for longer periods of time so as to investigate more fully the continuity of delinquent behavior that is typical of life-course-persistent offending.

Nevertheless, even taken from a conservative standpoint, these findings bear a resemblance to several related studies suggesting how individual differences are exacerbated within disadvantaged environments (see Peebles & Loeber, 1994; Piquero et al., 2005). This suggests that behaviors in general, and delinquent and criminal behavior in particular, are not the product of individual factors or the environment independently. Rather, these findings might suggest that individual differences may form the parameters of such behaviors, but it is the environment that increases or decreases the likelihood of manifestation of such behaviors. If so, policy makers should be mindful to consider the interplay of these interactions when determining the spending directions of resources affecting delinquent and criminal behavior.

In closing, the results of this study adds to a small but growing body of evidence that macro-level structural factors operating at the neighborhood level moderate the effects of individual- and family-level risks for chronic and violent juvenile offending. That poor non-Whites reside in neighborhoods that are ecologically distinct from those in which poor Whites reside appears to exacerbate the criminogenic effects of individual-level deficits and family disadvantage. Residence in such neighborhoods may also be implicated directly in the development of individual-level deficits and family dysfunction. It will be especially important for future research to begin to explore just how neighborhoods affect individual neuropsychological status and family life and help to shape the course of youth development.

APPENDIX

Items Used for Delinquency Scale

-
- Intentionally damaged or destroyed property that did not belong to you?
 - Gotten into a fight at school or work?
 - Taken something from a store without paying for it?
 - Other than from a store, taken something not belonging to you that was worth less than \$50?
 - Other than from a store, taken something not belonging to you that was worth \$50 or more?
 - Used force to get money or things from someone else?
 - Hit or seriously threatened to hit someone?
 - Attacked someone with the idea of seriously hurting or killing them?
 - Taken a vehicle without the owner's permission?
 - Broken into a building or vehicle to steal something or to just look around?
 - Knowingly sold or held stolen goods?
 - Helped in a gambling operation, like running numbers or books?
 - Hurt someone badly enough to need bandages or a doctor?
-

NOTES

1. In neighborhoods characterized by concentrated poverty, unemployment, and family disruption, pregnant women are less likely to receive adequate prenatal care, both mother and child are more likely to be malnourished, and there is greater risk of exposure to environmental toxins and infectious disease. Consequently, Moffitt contends that children born in these environments are at greater risk for neuropsychological deficits.

2. Wikstrom and Loeber (2000) explored the connection between risk and protective factors (including individual- and family-level ones), neighborhood context, and delinquency. Unfortunately, their analysis does not permit one to differentiate the effects of personal, familial, peer, and school risks.

3. We assessed the sensitivity of this measure by creating different cutoff points (i.e., mean splits, median splits, etc.). The results did not substantively change the importance of the main exogenous variables of interest.

4. A coefficient comparison test revealed that these differences were significant at the .10 level ($z = 1.78$).

5. In short, because of financial constraints, 263 of the individuals older than the age of 21 were not interviewed during 1998.

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