Journal of Criminal Justice 47 (2016) 84–99

Contents lists available at ScienceDirect

Journal of Criminal Justice

The effect of the maturity gap on delinquency and drug use over the life course: A genetically sensitive longitudinal design

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ARTICLE INFO

Article history:
Received 25 May 2016
Received in revised form 3 July 2016
Accepted 4 July 2016
Available online xxxx

Keywords:
Taxonomic theory
Moffitt
Adolescent-limited offending
MZ twin discordance
Genetic confounding
Biosocial criminology

ABSTRACT

Purpose: Relatively few studies have assessed Moffitt's claims regarding the etiology of the offending groups in her taxonomic theory. This gap is especially evident regarding adolescence-limited (AL) offending where empirical analyses of the maturity gap (the disjunction between biological and social maturity during adolescence) have produced mixed findings. Additionally, genetically sensitive analyses of the effect of the maturity gap on delinquency is entirely lacking from the literature. The current study provides such an analysis.
Methods: Using a sample of monozygotic (MZ) twins ($N_{\text{Indviduals}} = 524$; $N_{\text{Twin pairs}} = 262$) the current study addresses these gaps in the literature by assessing the influence of the maturity gap, parental conflict, and other theoretically relevant variables on delinquency and substance use in a sex-differentiated longitudinal analysis of MZ difference scores.
Results: Findings illustrated minimal influence of the maturity gap, parental conflict, and low self-control on delinquency and substance use in adolescence and adulthood. However, discordance in exposure to delinquent peers was associated with delinquency and substance use in adolescence but with little long-term effect.
Conclusion: Overall, the findings provide mixed support for Moffitt's ideas and illustrate the confounding effects of genetic factors in assessments of the etiology of antisocial behavior and tests of criminological theory.

1. Introduction

Since its publication in 1993, Moffitt's highly influential article outlining her developmental taxonomy of antisocial behavior has been cited over 7800 times in scholarly publications (Google Scholar, 2016). Although Moffitt's initial article and subsequent work (Moffitt, 1993; Moffitt, 1994) have guided scholarly efforts in a wide range of disciplines, her impact in criminology has been substantial. Moffitt proposed a theory that accounted for two of criminology's oldest and consistent empirical observations: the so-called age-crime curve (the moniker for which is a result of the spike in delinquency that associates adolescence and the desistance that accompanies entry into young adulthood) and the observation of variance in behavioral stability across individuals. Moffitt's theory not only advanced a two-group taxonomy of offending to account for these empirical observations (termed adolescence-limited offending and life-course persistent offending), she also provided different etiological processes underpinning the pattern of antisocial behavior of either group. It is on the etiological processes of the adolescence-limited offending group that the current paper is focused.

According to Moffitt, life-course persistent (LCP) offenders exhibit a pattern of antisocial behavior that begins early in life and continues through adolescence and adulthood. She proposed a biosocial explanation for LCP offending which focused primarily on the interactive effect of neuropsychological deficiencies and adverse developmental environments. In contrast to LCP offenders, adolescence-limited (AL) offenders display little antisocial behavior early in life but upon entry into adolescence engage in delinquency throughout their teen years. Unlike LCP offenders, however, AL offenders engage in relatively minor forms of delinquency and tend to desist such delinquency upon entering young adulthood. Recognizing the AL offending pattern as a normative aspect of development, Moffitt did not place the etiology of AL offending within neuropsychological impairments or deleterious developmental environments. Rather, she proposed that AL offending was a consequence of the normative process of psychosocial strain felt by adolescents resulting from the discrepancy or disjunction between their biological maturity and their social maturity or independence (Moffitt, 1993; Barnes & Beaver, 2010). Moffitt termed this discrepancy the maturity gap and claimed that AL offending was a manifestation of efforts to express social independence congruent with the typical behavioral maturity exhibited during the teen years in our evolutionary history (Moffitt, 1993). In specifying why youth engage in delinquency to address the strain of experiencing a maturity gap, Moffitt argued that AL offenders engage in a process of social mimicry of the adult-like and independent behaviors of LCP offenders.

Although Moffitt's theory is generally not considered a biosocial theory within criminology, the relevance of biological processes is quite

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http://dx.doi.org/10.1016/j.jcrimjus.2016.07.001
0047-2352/

clear. Indeed, the explicit focus of biological factors such as neuropsychological deficits, the evolutionary history and ontogenetic development of our species, along with an integration of social processes such as adverse developmental environments and economic conditions belies the theory's biosocial approach. Furthermore, recent research has shown that behavioral patterns illustrative of the developmental taxonomy (i.e., offending, desistence, and non-offending) are partially driven by genetic factors (Barnes & Beaver, 2010; Barnes, Beaver, & Boutwell, 2011; Schwartz & Beaver, 2015; Zheng & Cleveland, 2015). Moreover, developmental research has shown that pubertal timing, a key aspect of Moffitt's theory (i.e., biological maturity), appears to be substantially influenced by genetic factors (e.g., Eaves et al., 2004; Mustanski, Viken, Kaprio, Pulkkinen, & Rose, 2004). Finally, behavioral genetic and biosocial criminological research has shown that peer selection and the general influence of peers, a dominant factor in the process of social mimicry, is also influenced by genetic factors (Beaver et al., 2009; Kendler et al., 2007). Taken together, these lines of research demonstrate that analyses of Moffitt's ideas necessitate cognizance of the impact of biosocial processes in general and genetic factors in particular. Failing to account for the influence of biological and genetic factors risks an empirical assessment that could be substantially misspecified (Barnes et al., 2014a; Barnes, Boutwell, Beaver, Gibson, & Wright, 2014b). Nonetheless, few criminological assessments of Moffitt's ideas employ a genetically sensitive design (for exceptions see Barnes, 2013; Barnes et al., 2011; Boutwell, Nedelec, Lewis, Barnes, & Beaver, 2015; Jaffee et al., 2005; Schwartz & Beaver, 2015; Zheng & Cleveland, 2015).

Much of the criminological research investigating Moffitt's ideas has focused on two general aspects of her theory: (1) assessing the empirical validity of the behavioral taxonomy, and (2) assessing the empirical validity of the proposed etiology of LCP offending (e.g., Kjelsberg, 1999; Moffitt, Caspi, Harrington, & Milne, 2002). Despite its relevance to one of the most common observations in criminology (the age-crime curve) and the ubiquity of AL offending across cultures and time (Farrington, 1986), relatively few studies have assessed Moffitt's claims regarding the maturity gap and its influence on antisocial behavior (Dijkstra et al., 2015; Moffitt, 2008). Furthermore, while a small number of studies have shown that offending and non-offending behavioral patterns consistent with Moffitt's theory are influenced by genetic factors no study has examined the *effect* of the maturity on gap on delinquency using a genetically sensitive longitudinal design. Thus, the current study represents an effort to address these gaps in the literature by using a sample of monozygotic twins derived from a nationally representative longitudinal sample.

2. Criminological research on the maturity gap

The concept of a maturity gap appears to have strong empirical validity. As Moffitt (1993) illustrated, modernized societies have resulted in a detachment from the species-typical transitory process from childhood to adulthood. This time of transition is now referred to as adolescence and entails considerable maturational change in virtually every aspect of human biology (Tanner, 1981). Additionally, while adolescence is marked by continued social restrictions imposed by parental figures and other guardians and institutions, youth at this stage are afforded an increase in privileges and responsibilities relative to their childhood years (Agnew, 2003). Accompanying these social and biological changes is a psychological process wherein adolescents experience frustration at the limitations of their autonomy or more generally, their social maturity. Researchers have provided support for this psychological strain resulting from blocked expectations of maturity as well as an expressed desire for increased privileges and responsibilities congruent with an adult-like lifestyle (Galambos, Barker, & Tilton-Weaver, 2003). Thus, the impetus of the link between the maturity gap and AL offending appears to be an empirical reality.

Despite illustrations of psychological frustrations associated with adolescence fewer studies have provided direct empirical assessments of Moffitt's maturity gap hypothesis (Barnes & Beaver, 2010; Dijkstra et al., 2015). In a review of the relevant literature in 2008, Moffitt noted that the "most direct test of the adolescence-limited etiological hypothesis" (289) was completed by Piquero and Brezina (2001). Using longitudinal data, Piquero and Brezina (2001) examined the direct and interactive effects of physical maturity, behavioral autonomy with peers, and need for autonomy on delinquent behavior among adolescent boys. The authors operationalized the maturity gap in their study by creating two multiplicative interaction terms (physical maturity X behavioral autonomy with peers; physical maturity X need for autonomy). The authors concluded that their finding of an interactive effect between physical maturity and behavioral autonomy on rebellious delinquency, but not aggressive delinquency, was consistent with Moffitt's claims regarding the manifestation and etiology of AL offending. However, the authors also noted that given the lack of an observed effect of the theoretically expected interaction between physical maturity and desire for autonomy on delinquency their results only partially supported Moffitt's hypotheses.

More recently, Sentse, Dijkstra, Lindenberg, Ormel, and Veenstra (2010) examined the moderating effect of pubertal timing (*early* versus *normal*/*late*) on the relationship between protective parenting techniques and adolescent antisocial behavior in a longitudinal (2.5 years) sample of Dutch adolescents. The findings illustrated longitudinal positive main effects of both overprotective parenting and early biological maturation as well as an interactive effect on adolescent antisocial behavior. Importantly, these results applied only to the males in the sample. The authors concluded that the findings supported Moffitt's maturity gap hypothesis and indicated that overly protective parenting of early maturing adolescent boys is a potential mechanism underlying the maturity gap and its association with adolescent antisocial behaviors.

Dijkstra et al. (2015) also employed a Dutch sample of adolescent boys and girls and examined the extent to which biological maturation and social autonomy influenced parental conflict and whether this association affected adolescent delinquency and substance use. Employing a longitudinal (six-month timespan[1]) path model analysis, the findings indicated a lack of a direct effect of either social autonomy or biological maturity on either delinquency or substance use. Additionally, the authors failed to find an association between the interaction term for social autonomy and biological maturity and delinquency or substance use. However, the authors did observe a statistically significant association between these variables via the influence of parental conflict. Importantly, the authors noted that the findings applied equally to both the boys and the girls in their sample. As a result of their analyses, Dijkstra et al. (2015) concluded that the interplay between biological and social maturity is associated with delinquency and substance use in adolescence but only as it impacts parental conflict.

The literature outlined thus far has collectively indicated that biological maturity and social maturity have an influence on the likelihood of antisocial behavior during adolescence that is generally in line with Moffitt's maturity gap hypothesis. However, inconsistencies in the literature are also clearly present. Some authors have regarded these inconsistencies as reflecting the need to include specific mechanisms when examining the influence of a maturity gap (e.g., Dijkstra et al., 2015). Another potential reason for the inconsistencies observed in the extant literature is the reliance on a specific method of operationalization of the maturity gap concept. To reiterate, Moffitt conceptualized the maturity gap as a discrepancy between a youth's biological maturity and the youth's social maturity or social freedom. The discrepancy, or gap, is the *vital* component of the theoretical concept purported by Moffitt to account for AL offending patterns. As outlined in the above literature, the primary method by which researchers have operationalized this concept is to use multiplicative interaction terms including a measure of pubertal development and a measure of social autonomy. However,