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Biosocial Influences on Offending Across the Life Course

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Abstract

This chapter presents major biological and biosocial findings in relation to the development of offending. It reviews empirical findings on the association between two psychophysiological factors, heart rate and skin conductance, and offending. The chapter then discusses the heritability of antisocial behavior and the contribution of genetics to the understanding of developmental trajectories, stability, and change in offending. The structural and functional brain abnormalities in antisocial individuals across different age groups are then discussed, along with research on hormones and neurotransmitters. Next, the chapter highlights the applications of neuropsychology in the understanding of offending across the life span and reviews research on pre- and perinatal factors related to later offending. It concludes with potential areas for future research.

Keywords

biosocial influences, offending, heart rate, skin conductance, psychophysiological factors, antisocial behavior, genetics, brain abnormalities, neuropsychology

Disciplines

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Biosocial Influences on Offending Across the Life Course

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SINCE the late 20th century, the field of criminology has become increasingly aware of the contributions of biological sciences. Through longitudinal studies and research on different age groups, the role of biological factors in offending has been examined in a developmental context. Findings document that biological factors are associated with offending across the life span, although the strength of the associations may differ across development and between types of offenders. It is proposed that incorporating such factors into future developmental and life-course research and theories can lead to a better understanding of the etiology of offending.

In this chapter, major biological and biosocial findings in relation to the development of offending are presented. Offending is referred to as not only the violation of legal codes but also the broader spectrum of antisocial behavior. Section I reviews empirical findings on the association between two psychophysiological factors, heart rate and skin conduc-

tance, and offending. Section II discusses the heritability of antisocial behavior and the contribution of genetics to the understanding of developmental trajectories, stability, and change in offending. Section III describes structural and functional brain abnormalities in antisocial individuals across different age groups. Section IV covers research on hormones and neurotransmitters. It examines the role of cortisol, testosterone, serotonin, and dopamine on offending. Section V highlights the applications of neuropsychology in the understanding of offending across the life span, particularly in the domains of verbal and spatial intelligence and executive functioning. Section VI reviews research on pre- and perinatal factors related to later offending, including prenatal alcohol, nicotine, and lead exposure, minor physical anomalies, and birth (p. 326) complications. Each of the six sections aims to address issues important to the developmental and life-course criminological literature including whether the biological factor is consistently associated with offending throughout the life course and whether persistent offenders differ from other offenders in terms of biological influences. Section VII concludes with potential areas for future research.

I. Psychophysiology

Psychophysiology is the study of cognition, behavior, and emotions as revealed through bodily events (Hugdahl 2001). Heart rate and skin conductance are psychophysiological measures that have been frequently examined in relation to offending.

A. Heart Rate

Heart rate is controlled by both the parasympathetic and sympathetic branches of the autonomic nervous system. A meta-analysis concluded that low resting heart rate is likely the best-replicated correlate of antisocial behavior in children and adolescents (Ortiz and Raine 2004). Although more commonly examined in youths, low resting heart rate is also a risk factor for antisocial behavior in adults (Lorber 2004; Armstrong et al. 2009; Portnoy and Farrington 2015), making low resting heart rate a biological risk factor for offending across the life course.

Importantly, low resting heart rate has been found to predict future levels of antisocial behavior in prospective longitudinal research (Farrington 1997; Raine, Venables, and Mednick 1997; Sijtsema et al. 2010; Jennings, Piquero, and Farrington 2013). One study found that low resting heart rate as young as at age 3 years predicted aggression at age 11 years (Raine et al., 1997). Findings from the Cambridge Study in Delinquent Development showed that low resting heart rate at age 18 years predicted offending up to age 50 years independent of covariates including smoking, sports participation, impulsivity, binge drinking, body mass index, and early childhood individual and environment risk factors (Jennings et al. 2013). This study demonstrated for the first time that the predictive utility of low resting heart rate could extend into late adulthood.

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It has also been found that low resting heart rate is only important in explaining initial levels rather than change in antisocial behavior over time. Baker et al. (2009) found that children with low resting heart rate at age 9 years were significantly more antisocial overall, but the reduction in antisocial behavior with age as the children entered early adolescence was not associated with heart rate. This suggests that low heart rate is a fixed, static neurobiological risk factor for antisocial behavior that does not predict desistance from offending throughout early adolescence.

Several theoretical explanations have been proposed to explain the relationship between resting heart rate and antisocial behavior. According to stimulation-seeking (p. 327) theory, low autonomic nervous system arousal is an unpleasant physiological state, leading those with low resting heart rates to seek stimulating behaviors, including antisocial behaviors, in order to increase their level of physiological arousal to a more optimal level (Quay 1965; Raine 2002a). An alternative theory argues that low resting heart rate may reflect a relative lack of fear, which could predispose some individuals to commit antisocial acts that require a degree of fearlessness to complete. Low heart rate could also impede early childhood fear conditioning to socializing punishments (Raine 1993, 2002a). While support for these two theories has been broadly found (Latvala et al. 2015), recent studies documented that stimulation seeking, but not fearlessness, mediated the relationship between low heart rate and antisocial behavior (Sijtsema et al. 2010; Portnoy et al. 2014). Thus, a stimulation-seeking mechanism may be more likely to underlie this relationship.

B. Skin Conductance

Skin conductance is an index of sympathetic nervous system activity that can be measured at rest or during laboratory tasks. Reduced skin conductance reactivity during fear conditioning paradigms has been associated with psychopathy (Birbaumer et al. 2005) and antisocial behavior, particularly persistent proactive aggression (Gao et al. 2015). Conditioning during childhood is thought to be central to socialization and conscience development. It has been suggested that the failure to condition could be a factor that predisposes some individuals to offend later in life (Eysenck 1977). Findings for skin conductance measured at rest tend to be less consistent. A meta-analysis found that low resting skin conductance was significantly associated with higher levels of psychopathy in adults and conduct problems in children, but not aggression or conduct problems in adolescence (Lorber 2004).

Like heart rate, reduced skin conductance has been documented to predict future levels of antisocial behavior. One study found that reduced skin conductance arousal at age 15 years predicted criminal behavior at age 24 years (Raine, Venables, and Williams 1990). Reduced skin conductance fear conditioning as early as age 3 years has been found to predict offending at age 23 years (Gao et al. 2010). These findings suggest that childhood and adolescent skin conductance can help to explain future levels of criminal behavior.

C. Biosocial Interactions Involving Psychophysiology

Some studies have found that low resting heart rate combined with high social risk increases the likelihood of antisocial behavior (Raine et al. 2014). Similarly, skin conductance has been found to interact with social adversity to predict antisocial behavior, though patterns of interaction are not always consistent, with high skin conductance serving as a risk factor for antisocial behavior among children at high social risk in (p. 328) several studies (e.g., Cummings et al. 2007). In general, psychophysiological studies suggest a reduced pattern of autonomic arousal across the life course in antisocial individuals, although results may be partly dependent on the individual's social context.

II. Genetics

Compelling evidence from behavioral genetic research, which broadly includes twin, adoption, and family studies, shows that heritable influences also contribute to the development of offending. A review on 19 twin and adoption studies between ages 1 and 18 years found that heritability explained 65 percent and 48 percent of the variance in aggressive behavior and delinquent/rule-breaking behavior, respectively (Burt 2009). Additionally, summarizing results from 51 twin and adoption studies in children, adolescents, and adults, Rhee and Waldman (2002) found that genetic factors explained 41 percent of the variance in antisocial behavior. Given findings on the heritability of offending, molecular genetic research has identified candidate genes for offending. Lower monoamine oxidase A (MAOA) gene activity has been associated with violent behaviors and offending over the life course (e.g., Beaver et al. 2013). Other genes suggested to be associated with child and adult antisocial behavior include the catechol-O-methyltransferase (COMT) gene (Volavka, Bilder, and Nolan 2004; Hirata et al. 2013), the vasopressin receptor 1B (Zai et al. 2012) gene, the oxytocin receptor (OXTR) gene (Malik et al. 2012), the human dopamine transporter (DAT1) gene (Guo, Roettger, and Shih 2007), the D2 receptor polymorphism (DRD2) gene, and the D4 receptor polymorphism (DRD4) gene (Beaver et al. 2007; Boutwell et al. 2014).

Reviews have also found that aggressive and delinquent/rule-breaking behavior exhibit different etiological patterns across age (Burt 2009). Genetic influences on aggressive behavior increased across development, while shared environmental factors decreased. In contrast, delinquent/rule-breaking behavior showed a decrease in genetic influences across development, while shared environmental influences remained stable. The results show that aggressive behavior is primarily influenced by genetic factors, while delinquent/rule-breaking behavior is influenced by both genetic and shared environmental factors.

Since 2003, several studies have examined the genetic and environmental influences on psychopathic personality in children, adolescents, and adults (e.g., Viding et al. 2005; Viding, Frick, and Plomin 2007; Brook et al. 2010; Bezdjian et al. 2011; Hicks et al. 2012). According to the average twin correlations across these studies, the heritability of psy-

chopathic personality in males is approximately 64 percent. For females, the heritability of psychopathic personality is approximately 48 percent (Tuvblad 2013). A longitudinal study reported that 58 percent and 62 percent of the stable variance in two features of psychopathic personality, fearless dominance and impulsive antisociality, respectively, from ages 17 to 24 years were explained by genetic factors (Blonigen et al. 2006; Tellegen and Waller 2008).

(p. 329) With regards to different developmental trajectories for offending, twin studies have shown that genetic influences are more important for stable-high/childhood-onset than for increasing/transitory antisocial behavior (e.g., Taylor, Iacono, and McGue 2000; Tuvblad et al. 2011). For example, in one study, genetic influences contributed more strongly to early-onset rather than late-onset delinquent behavior in 11-year-old boys (Taylor et al. 2000).

Longitudinal twin studies have also examined the contribution of genetics to stability in antisocial behavior. One study that measured antisocial behavior from ages 8 to 20 years showed that the stability of antisocial behavior was explained by a common latent antisocial behavior factor, for which genetics accounted for 67 percent of the variance (Tuvblad et al. 2011).

Other twin studies have examined the genetic contribution to change in antisocial behavior. This line of research has generally reported that change or “new” variance in antisocial behavior is primarily due to non-shared environmental factors (e.g., Haberstick et al. 2006). Analysis of three waves of data from the Minnesota Twin and Family Study showed that while genetic influences were to a large extent responsible for the initial level of antisocial personality disorder symptoms, non-shared environmental influences were largely responsible for change (Burt et al. 2007).

A. Biosocial Interactions Involving Genetic Factors

As in molecular genetic studies, such as that by Caspi et al. (2002), which found that childhood maltreatment led to violence in adulthood among individuals with low levels of MAOA expression, behavioral genetic studies have generated a large number of gene-environment interaction (G×E) studies (for a review, see Dick 2011). Specifically, social factors such as family dysfunction, family warmth, high paternal punitive discipline, parental monitoring, religiosity, regional residency, and socioeconomic status have been found to moderate the genetic and environmental influences on antisocial behavior (Koopmans et al. 1999; Rowe, Almeida, and Jacobson 1999; Rose et al. 2001; Button et al. 2005; Tuvblad, Grann, and Lichtenstein 2006; Dick et al. 2007; Button et al. 2008; Middeldorp et al. 2014). Some studies have found higher heritability of antisocial behavior in individuals with low rather than high levels of social risk (Button et al. 2005), while others document that genetic influences contributed more to antisocial behavior when social risk was present (Dick et al. 2007).

III. Brain Imaging

Regarding structural abnormalities associated with offending, studies have largely focused on regions involved in decision-making (e.g., prefrontal cortex), emotion regulation (e.g., amygdala, hippocampus), and reward-processing (e.g., striatum). One of (p. 330) the first structural brain imaging studies of antisocial adults documented an 11 percent reduction in gray matter volume in the prefrontal cortex of men with antisocial personality disorder compared to normal controls and a psychiatric control group (Raine et al. 2000). Yang et al. (2005, 2009, 2010a) found reduced gray matter volume and thickness in the middle frontal and orbitofrontal cortex and reduced volume and surface deformations in the amygdala in psychopaths with prior convictions (i.e., unsuccessful psychopaths) compared to psychopaths without convictions (i.e., successful psychopaths) and non-psychopathic controls. More recently, violent offenders were shown to have abnormal hippocampal structure compared to non-violent controls (Boccardi et al. 2010; Yang et al. 2010b). These frontal and limbic deficits were similarly found in a large sample of nearly 300 incarcerated criminal offenders (Ermer et al. 2012). Using *vivo* diffusion tensor magnetic resonance imaging tractography, Craig et al. (2009) further showed impaired amygdala-orbitofrontal connections in psychopaths with convictions. However, findings for the striatum are not conclusive, as some studies have documented enlarged putamen, nucleus accumbens, and caudate (Schiffer et al. 2011), while others showed smaller nucleus accumbens in offenders (Boccardi et al. 2013).

In addition to structural imaging research, functional imaging studies have presented evidence for impaired brain functioning in criminal offenders, especially in the prefrontal and temporal cortex. A meta-analysis by Yang and Raine (2009) of 43 studies revealed that increased antisocial behavior was associated with reduced prefrontal structure and function, particularly in the right orbitofrontal, left dorsolateral prefrontal, and right anterior cingulate cortex. Employing a neurocognitive task, the non-verbal Stroop task, Schiffer et al. (2014) found reduced function in the anterior cingulate, dorsolateral prefrontal, superior temporal, putamen, and amygdala in violent offenders with antisocial personality disorder compared to non-offenders. One recent study using resting-state functional magnetic resonance imaging (fMRI) also revealed reduced activity, measured by the amplitude of low-frequency fluctuation, in the right orbitofrontal cortex as well as the left temporal pole, right inferior temporal gyrus, and left cerebellum in these offenders with antisocial personality disorder (Liu et al. 2014). In line with these findings, Ly et al. (2012) found thinner cortices in the right inferior frontal cortex, anterior temporal cortex, and anterior cingulate cortex, which also corresponded to reduced functional connectivity between the left insula and left dorsal anterior cingulate cortex in psychopathic compared to non-psychopathic criminal offenders.

Recently, in a longitudinal study, males with lower amygdala volumes at age 26 years were found to exhibit increased aggression, violence, and psychopathic traits at a 3-year follow-up (Pardini et al. 2014). Similarly, in a study on adult male offenders, lower anteri-

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or cingulate activity was associated with a greater likelihood of rearrest (Aharoni et al. 2013). These findings suggest that brain deficits can predict later offending.

Consistent with research on adult offenders, studies of delinquent children and adolescents have revealed abnormal brain structures and function. For example, in a sample of female adolescents with conduct disorders, aggressive symptoms were negatively correlated with right dorsolateral prefrontal cortex volume, while (p. 331) callous-unemotional traits correlated positively with bilateral orbitofrontal cortex volume (Fairchild et al. 2013). Delinquents with high psychopathy scores also showed higher activity in the anterior cingulate cortex, insula, and amygdala during fear conditioning compared to healthy controls (Cohn et al. 2013). Longitudinally, a thicker temporal cortex was linked to higher rates of change in psychopathy during childhood and adolescence (Yang et al. 2015). Taken together, meta-analyses document that youths with disruptive behavior disorder or conduct problems show consistent functional deficits in the dorsal and rostral anterior cingulate and the medial prefrontal cortex (Alegria, Radua, and Rubia 2016), as well as reduced gray matter volume in the insula, left amygdala, and frontal and temporal regions (Rogers and De Brito 2016). One study documented that limbic structural deficits such as in the amygdala were associated with not only early-onset but also adolescent-onset conduct disorder (Fairchild et al. 2011). However, more recently, evidence has been found for quantitative differences in structural brain organization between childhood-onset and adolescence-onset conduct disorder (Fairchild et al. 2016) and for different growth trajectories of cortical thickness for distinct conduct problem pathways (Oostermeijer et al. 2016).

A. Biosocial Interactions Involving Brain Imaging

Although few brain imaging studies to date have addressed the role of psychosocial risk and protective factors on offending, several studies have begun to address two related issues concerning whether home background moderates the relationship between violence and brain functioning and whether brain deficits combine with psychosocial deficits to predispose one to violence. Regarding the first issue, two studies using brain functioning as the outcome variable have demonstrated a moderating effect of home background, but in opposing directions. In one study, murderers from non-deprived home backgrounds showed a 14.2 percent reduction in functioning of the right orbitofrontal cortex relative to murderers from deprived home backgrounds characterized by abuse, neglect, and marital violence (Raine et al. 1998). It was argued that neurobiological deficits are more pronounced among violent individuals who lack the psychosocial deprivation that normally provides a “social push” toward violence. In contrast, a second fMRI study showed that violent offenders who had been severely abused as children were more likely to show poor temporal lobe functioning compared to violent offenders lacking abuse (Raine et al. 2001).

Turning to the second issue, using violence as an outcome variable, an anatomical magnetic resonance imaging study of individuals with antisocial personality disorder and high psychopathy scores showed that the combination of reduced prefrontal gray volume, low

autonomic responsivity, and a set of 10 psychosocial deficits correctly classified 88.5 percent of subjects into antisocial personality disorder or control groups (compared to 73 percent for psychosocial predictors only and 76.9 percent for biological predictors only; Raine et al. 2000). A second structural imaging study on the corpus callosum in psychopaths showed that the combination of psychosocial risk factors (p. 332) with callosal measures accounted for 81.5 percent of the variance (Raine et al. 2003). Structural brain measures accounted for a significant increase in the variance in psychopathic/antisocial behavior over and above psychosocial risk factors in both studies.

IV. Hormones and Neurotransmitters

Compared to brain imaging research, fewer studies have examined the relationship between hormones and offending. Two most frequently studied hormones in relation to antisocial behavior are cortisol and testosterone, regulated by the hypothalamus-pituitary-adrenal (HPA) axis and hypothalamus-pituitary-gonadal (HPG) axis, respectively.

A. Cortisol

Studies in children and adolescents have shown that cortisol may be related to antisocial behavior early in life. One meta-analysis found a mean effect size of $d = -.40$ for the relationship between basal cortisol levels and disruptive behavior or aggressive symptoms in children after study sample sizes were taken into account (van Goozen et al. 2007). The mean effect size for cortisol reactivity in response to a stressor across 4 studies after correcting for sample sizes was $d = .42$. A second meta-analysis on 72 study outcomes found that in preschoolers (aged 0 to 5 years), higher basal cortisol was associated with externalizing behavior ($d = .18$). Low basal cortisol was associated with externalizing behavior ($d = -.28$) in elementary school-aged children (aged 5 to 12 years; Alink et al. 2008). However, no significant association was found between basal cortisol and externalizing behavior in adolescents or between cortisol reactivity to stress and externalizing behaviors. Thus, despite smaller relations in the second meta-analysis, there is some evidence of a significant relationship between basal levels of cortisol and antisocial behavior.

Similar findings were obtained for adults as low cortisol levels were found in offenders with psychopathy compared to non-psychopathic offenders (Holi et al. 2006; Cima, Smeets, and Jelicic 2008). In a study on cortisol reactivity using a social stressor, a significant difference in cortisol levels from pre- to post-stressor was observed, but only in males with low rather than high levels of psychopathy (O'Leary, Loney, and Eckel 2007).

Several studies have examined cortisol levels in relation to different categories of offenders. Fairchild et al. (2008) found that basal cortisol level or cortisol reactivity to a stressor did not differ between male adolescents with early-onset and adolescence-onset conduct disorder. It has been proposed that structural abnormalities in the amygdala may underlie the finding that early- and adolescent-onset conduct disorder are associated with lower cortisol responses to stress as the amygdala is involved in initiating HPA responses to stress (Fairchild et al. 2011). However, another (p. 333) study on boys aged 7 to 12 years

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found that lower cortisol was more strongly related to persistently aggressive boys as well as those with childhood-onset conduct disorder compared to adolescence-onset conduct disorder (McBurnett et al. 2000). This is bolstered by findings that low basal cortisol levels predicted disruptive behavior in boys and girls only if conduct problems were already present at age 10 to 12 years (Sondeijker et al. 2008) and that persistently high-aggressive adolescents exhibited decreased cortisol levels consistently over time compared to low-aggressive adolescents (Platje et al. 2013). Such findings suggest that cortisol levels are related to the persistent trajectory of antisocial behavior rather than the prediction of the onset of behavior problems at later ages.

B. Testosterone

Experimental studies have shown that increased testosterone levels are associated with increased levels of aggression (Pope, Kouri, and Hudson 2000) and decreased levels of empathy (van Honk et al. 2011), which are associated with offending. Associations between higher levels of testosterone and antisocial behavior have been reported in children and adolescents. For example, adolescents with high levels of externalizing behaviors have been documented to have higher levels of testosterone than individuals with low levels of externalizing behaviors (Maras et al. 2003). Furthermore, testosterone levels were found to be higher in a disruptive behavior disorder group than in normal controls for an older subset of participants (aged 9 to 11 years) compared to those of younger ages (aged 5 to 8 years; Chance et al. 2000). However, some other studies have found mixed results on the testosterone–antisocial behavior relationship among children and adolescents (e.g., van Goozen et al. 1998; Dorn et al. 2009).

Additional studies have found a positive relationship between testosterone levels in adulthood and retrospectively reported severity of conduct disorder symptoms in childhood (e.g., Mazur 1995). Longitudinal studies have documented that testosterone levels in a community sample at ages 12 and 14 years predicted antisocial norm-violating behaviors at age 16 years (Tarter et al. 2009), and testosterone levels in conduct disordered boys at age 13 years predicted delinquency and criminal behavior at ages 16 and 21 years (Van Bokhoven et al. 2006). In adults, meta-analytic evidence suggests that testosterone is positively associated with aggression, with higher associations found for the age group of 22 to 35 years and in offender compared to non-offender populations (Archer et al. 2005). Higher testosterone levels also correlated positively with psychopathy scores in convicted criminals (Stålenheim et al. 1998). On the other hand, Glenn et al. (2011) found that instead of baseline testosterone, the ratio of baseline testosterone to cortisol reactivity after stress was significantly related to psychopathy. Generally, research suggests there is a small positive correlation between testosterone and antisocial behavior throughout the life span. Relationships seem to be weakest in young children and get stronger as individuals age (Yildirim and Derksen 2012).

(p. 334) C. Serotonin and Dopamine

Serotonin and dopamine are neurotransmitters that have been implicated in antisocial behavior and specifically in psychopathy. Most commonly, researchers have examined neurotransmitter metabolite levels in cerebrospinal fluid, such as HVA, a metabolite of dopamine, and 5-HIAA, a metabolite of serotonin (Freedman and Verdun-Jones 2010).

Studies have found that examining serotonin and dopamine levels together provides a better prediction of psychopathy scores. In a sample of violent offenders, the ratio of HVA to 5-HIAA was positively associated with psychopathy scores, particularly the Factor 2 Antisocial/Lifestyle score, which has been linked to life-course-persistent offending (Soderstrom et al. 2001; Yildirim and Derksen 2012). In a follow-up study, these results were replicated in a forensic sample. The HVA:5-HIAA ratio was positively related to childhood-onset disruptive disorders (Soderstrom et al. 2003).

Other research has suggested that serotonin levels are generally low in antisocial populations. One meta-analysis on 20 reports revealed reduced 5-HIAA in antisocial populations, particularly for individuals younger than 30 years, supporting the possibility that age-related increases in serotonin correlate with age-related declines in crime (Moore, Scarpa, and Raine 2002). Significantly lower serotonin levels were also found in boys with high levels of callous-unemotional traits (Moul et al. 2013). In children and adolescents with obsessive-compulsive disorder, participants with comorbid disruptive behavior disorders had significantly lower blood serotonin concentrations than participants with no comorbid behavior disorder (Hanna, Yuwiler, and Coates 1995). In the same study, a negative relationship was also found between serotonin concentration and externalizing and aggression scores. Additionally, reduced concentrations of somatostatin, a peptide which stimulates the release of serotonin, have been found in the cerebrospinal fluid of children with disruptive behavior disorders compared to children with obsessive-compulsive disorder (Kruesi et al. 1990). Although reduced serotonin levels have been found in children, findings suggest that the strongest serotonin effects on offending occur in young adulthood (Moore et al. 2002). All in all, despite some mixed findings (Hughes et al. 1996), there is reasonably strong evidence that serotonin and dopamine play a role in the development of offending.

D. Biosocial Interactions Involving Hormones and Neurotransmitters

Limited research has been conducted on biosocial interactions involving hormones and neurotransmitters. In one study, maltreatment was a significant moderator of the cortisol dysregulation-antisocial behavior relationship, such that low cortisol levels were more strongly associated with antisocial behavior in nonmaltreated children compared to maltreated peers (Hawes, Brennan, and Dadds 2009). In a similar vein, Hawes et al. (p. 335) (2009) suggested that early adversity plays a role in the development of antisocial behavior in children with low levels of callous-unemotional traits and higher basal cortisol levels, while high levels of callous-unemotional traits and low basal cortisol levels characterize a particularly severe subgroup for whom antisocial behavior develops somewhat inde-

pendently of social adversity. A study on Dutch adolescents documented that among individuals who experienced low levels of an environmental stressor, namely neighborhood density, lower cortisol activity significantly predicted higher levels of delinquency and aggression (Yu et al. 2016). Thus, HPA-axis dysfunction may play a more significant role in the development of chronic antisocial behavior for individuals who have not been exposed to adversity.

V. Neuropsychological Factors

Neuropsychology, the indirect, behavior-based assessment of brain dysfunction, has also been used to understand offending across the life span. Neuropsychological investigations of various forms of antisociality have largely targeted deficits in specific domains of cognitive functioning such as verbal and spatial intelligence and executive abilities.

A. Verbal and Spatial Intelligence

To date, the best-replicated cognitive correlate of antisocial, violent, and criminal behavior among non-mentally ill individuals is general intelligence (e.g., IQ or Full Scale IQ) deficits (Wilson and Herrnstein 1985). Reduced verbal relative to spatial/performance IQ—a possible marker for left hemispheric dysfunction—has generally been documented to characterize both males and females from different age groups across studies of antisocial individuals (Raine 1993; Isen 2010). However, some antisocial individuals, such as those with antisocial personality disorder and psychopathy, have not consistently shown intellectual performance or verbal intelligence deficits (Barkataki et al. 2006; Kosson et al. 2007), although relationships have been noted between some specific psychopathic traits (i.e., criminal versatility and violence) and verbal dysfunction (Rasmussen, Almvik, and Levander 2001). Thus, while global and/or verbal intellectual dysfunction may characterize adult antisocial individuals in general, they may not characterize specific constellations of criminogenic and antisocial traits.

Reduced verbal intelligence also appears largely characteristic of antisocial children and adolescents (e.g., Barker et al. 2007). Moffitt, Lynam, and Silva (1994) found that verbal deficits in early adolescence predicted delinquency in later adolescence for persistent, high-level offenders who began offending in pre-adolescence. However, mixed results have been found for juvenile psychopathy. Loney et al. (1998) found no verbal deficits in children with conduct problems and callous-unemotional traits, (p. 336) while Salekin et al. (2004) found that verbal intelligence was positively related to the superficial and deceitful interpersonal style traits and inversely related to the affective processing-disturbance traits of psychopathy in juvenile inmates. In summary, verbal deficits in populations of antisocial youth overall appear relatively consistent, though continued studies of psychopathic youth may assist in clarifying heterogeneous verbal IQ findings among antisocial juveniles as in adults.

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Longitudinal studies of community-based samples may call into question the classic view of verbal but not performance IQ deficits in antisocial individuals. In a Pittsburgh youth sample including childhood-limited, adolescent-limited, and life-course-persistent offenders, Raine et al. (2005) found both spatial and verbal impairments. In another sample from Mauritius, Raine et al. (2002c) found early spatial but not verbal deficits at age 3, and later spatial and verbal deficits at age 11 in persistently antisocial individuals. These results suggest that while early spatial deficits contribute to persistent antisocial behavior, verbal deficits may be developmentally acquired. Results support a proposed early starter spatial impairment model of life-course offending, in which early deficits in visuospatial functioning may interfere with mother-infant bonding, possibly reflecting right hemispheric dysfunction that disrupts emotional processing and regulation and, in turn, contributes to persistent offending.

B. Executive Functioning

Executive functioning deficits are thought to represent impairment in frontal lobe functioning and are indicated by performance errors on neuropsychological tests of strategy formation, cognitive flexibility, or impulsivity (i.e., category, maze-tracing, Stroop interference, card sorting, verbal fluency and tower tests, and go/no-go and gambling tasks). In Morgan and Lilienfeld's (2000) classic quantitative review of 39 studies, overall executive functioning deficits were observed in antisocial individuals compared to controls. Strongest effects were found for the Porteus Mazes test and antisociality defined by judicial status. More recently, executive dysfunction has been associated with aggressive, violent, and antisocial personality-disordered populations (e.g., Stanford et al. 2007; Hancock, Tapscott, and Hoaken 2010; Dolan 2012), property criminality (Barker et al. 2007), child molesters with and without pedophilia (Schiffer and Vonlaufen 2011), single as opposed to multiple homicide victims in indigent murder defendants and death row inmates (Hanlon et al. 2010), murderers with schizophrenia compared to non-violent men with schizophrenia (Hanlon et al. 2012), mentally challenged versus non-impaired forensic hospital patients (Bastert et al. 2012), and offenders characterized by reactive as opposed to instrumental violence (Broomhall 2005).

Psychopathy in adults has not been consistently associated with general executive functioning deficits (e.g., Kosson et al. 2007). Some neuropsychological studies have shown that psychopathy may be characterized more by orbitofrontal dysfunction, which is associated with processing rewards and punishments, and emotion (p. 337) (Rolls 2000; Blair et al. 2006). Additionally, successful, uncaught psychopaths have demonstrated significantly better dorsolateral prefrontal task performance relative to unsuccessful psychopaths and controls (Ishikawa et al. 2001), while white-collar criminals have been found to show increased executive functioning compared to offender controls (Raine et al. 2012). Furthermore, violent antisocial personality disordered offenders with and without psychopathy have demonstrated similar deficits in terms of "cool executive functioning," namely top-down processes subsumed by the dorsolateral and ventrolateral prefrontal cortex, that are distinctly cognitive in nature, such as working memory, response inhibition, planning, sustained attention, and attentional set-shifting, and "hot executive func-

tioning," namely processes with an affective, motivational, or incentive/reward component subsumed by ventromedial connections between the mesolimbic reward pathway and the ventromedial prefrontal cortex, such as appraisal of the motivational significance of a stimulus in emotional decision-making (De Brito et al. 2013).

Findings on children and adolescents have been more mixed, with executive functioning deficits characterizing some antisocial youths (e.g., Cauffman et al. 2005) and not others (Moffitt et al. 1994; Nigg et al. 2004). The development of executive functions along with the ongoing myelination of the frontal cortex into and beyond adolescence (Raine 2002b) may explain differential patterns of executive functioning deficits among children and adults. This is supported by findings of executive functioning impairments in older maximum security hospital patients (Nestor 1992) and more pronounced impairments on an orbitofrontal neuropsychological task in psychopathic adults than psychopathic children (Blair 2006).

C. Biosocial Interactions Involving Neuropsychology

In a study examining biosocial interactions, Gao et al. (2009) found that neurocognitive deficits indicated by more risky decision-making in the Iowa Gambling Task were associated with psychopathic tendencies only in children with higher socioeconomic status. Additionally, progressive cognitive dysfunction affected by adverse psychosocial experience may explain early-onset antisocial behavior (Aguilar et al. 2000) and lifetime, cumulative biosocial risk interactions may be stronger predictors of persistent aggression than risks only occurring in childhood or adolescence (Brennan et al. 2003). More specifically, Brennan et al. (2003), in a study of 370 Australian adolescents, identified that an interaction of biological risk factors including neuropsychological deficits and social risk factors predicted life-course-persistent aggression in boys and girls. Alternatively, the late-developing prefrontal cortex may be overloaded by the social and executive functioning demands of late adolescence, possibly leading to prefrontal dysfunction, behavioral inhibition failure, and significantly increased antisocial behavior (Raine 2002b). In sum, the neuropsychological literature demonstrates how the study of behavioral expressions of brain dysfunction has informed developmental neurobiological perspectives of offending across the life span.

(p. 338) VI. Early Health Risks

Evidence suggests that risk factors experienced early in life, such as during the prenatal and perinatal periods of development, are associated with longitudinal patterns of offending and may lead to the most detrimental effects over the life span (Day, Wanklyn, and Yessine 2014). Prenatal and perinatal factors that have been most closely linked to antisocial behavior include prenatal nicotine, alcohol, and lead exposure, minor physical anomalies, and birth complications.

A. Prenatal Nicotine, Alcohol, and Lead Exposure

Children who are exposed to maternal smoking during pregnancy have been documented to have an elevated risk of offending throughout the life course (Wakschlag et al. 2002). Numerous studies have found associations between prenatal maternal smoking and juvenile offending, delinquency, conduct disorder, and violent offending (e.g., Wakschlag et al. 1997; Brennan, Grekin, and Mednick 1999; Paradis et al. 2015). In particular, a dose-response relationship was observed between the degree of prenatal maternal smoking and the extent of offspring's nonviolent and violent offending assessed at age 34 years (Brennan et al. 1999). However, there is current debate regarding whether the nicotine exposure-offending association involves a genetic confound (Glenn and Raine 2014).

Prenatal exposure to alcohol results in cognitive, behavioral, social, and physical deficits and can lead to a diagnosis of fetal alcohol syndrome (FAS). Fetal alcohol exposure has been documented as a risk factor for antisocial behavior in children, adolescents, and adults (Olson et al. 1997; Fast, Conry, and Looock 1999). However, even without FAS, high rates of delinquency have been found in children and adolescents with heavy fetal alcohol exposure (Mattson and Riley 2000).

Besides nicotine and alcohol exposure, a prospective study found that prenatal maternal blood lead concentrations during the first and second trimesters of pregnancy were associated with higher rates of criminal arrests measured at ages 19 to 24 years (Wright et al. 2008). Another longitudinal study on 195 adolescents found that lead levels from the prenatal period to 6.5 years of age were associated with delinquent and antisocial behavior in middle adolescence (Dietrich et al. 2001). Although few longitudinal studies in this area exist, these studies demonstrate that prenatal lead exposure is associated with the development of offending.

B. Minor Physical Anomalies

Minor physical anomalies (MPAs) such as low-seated ears, single palmar crease, and furrowed tongue are considered indicators of fetal neural maldevelopment near the end (p. 339) of the first trimester or the beginning of the second trimester of pregnancy (Firestone and Peters 1983). Studies have found that MPAs are associated with greater antisocial behavior in children, adolescents, and adults, particularly for violent as opposed to non-violent offending (Glenn and Raine 2014). For example, MPAs measured at age 14 years predicted violent delinquency in 170 males at age 17 years, independent of childhood physical aggression or family adversity (Arseneault et al. 2000). Other studies have found that a larger number of MPAs is associated with recidivistic violent criminal behavior. One study documented that recidivistic violent offenders at ages 20 to 22 years had more MPAs between ages 11 to 13 years compared to individuals with one or fewer violent offenses (Kandel et al. 1989).

C. Birth Complications

Birth complications, such as pre-eclampsia, preterm birth, and breech fetal positioning, have also been found to predispose to later offending (e.g., Liu et al. 2009). For example, Kandel and Mednick (1991) found that high delivery complications were associated with adult violent offending. Additionally, findings from a longitudinal study, the Fragile Families and Child Well-Being Study, showed that low birthweight was linked to serious aggression and destructive behavior at age 5 years and the relationship was mediated by verbal skills (Vaske, Newsome, and Boisvert 2013). Other perinatal risk factors for later offending include being small for gestational age and a small head circumference (Babchishin et al. 2017).

D. Biosocial Interactions Involving Early Health Risks

Studies have documented that prenatal nicotine exposure, MPAs, and birth complications interact with social factors to predispose to later offending. For example, prenatal nicotine exposure was found to lead to an 11.9-fold and 14.2-fold increase in recidivistic violent offending in adulthood when combined with the individual social risk factor of being raised in a single-parent family and with a group of psychosocial risk factors, respectively (Räsänen et al. 1999). Increased risk has particularly been observed for persistent violent offending (Brennan et al. 1999; Gibson and Tibbetts 2000; Brennan et al. 2002). Moreover, MPAs in boys at age 12 years were related to violent, but not non-violent property offending at age 21 years, but only among individuals reared in unstable homes (Mednick and Kandel 1988). Similarly, Brennan, Mednick, and Raine (1997) and Pine et al. (1997) found higher rates of adult violent crime in males and greater risk for disruptive behavior and conduct disorder at age 17 years among individuals with both MPAs and social risk factors. A recent study documented that individuals born at low birthweight were at an increased risk of adult offending if they were born to adolescent mothers (Vaske et al. 2015). This is consistent with the finding that birth complications combined with early maternal rejection measured at (p. 340) age 1 year increased the likelihood of violent offending at ages 18 and 34 years (Raine, Brennan, and Mednick 1994, 1997). Birth complications have also been found to interact with other psychosocial factors such as poor parenting (Hodgins, Kratzer, and McNeil 2001), family adversity (Arseneault et al. 2002), and being an only child (Kemppainen et al. 2001) to lead to adult violent offending. These studies suggest that increased offending is observed when both early health risks and environmental risk factors are present.

VII. Conclusion

Through a review of extant research, this chapter sheds light on the development of anti-social behavior and risk factors for offending at different ages, issues central to developmental and life-course criminology. Despite some null findings, many biological risk factors such as autonomic underarousal, genetics, structural and functional brain abnormalities (particularly in the prefrontal and temporal cortex), low basal cortisol, high testos-

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terone, low serotonin, neuropsychological deficits, and early health risks are associated with antisocial behavior in children, adolescents, and adults. The strength of the risk factor–antisocial behavior associations may differ across development. For example, genetic influences increased across development for aggressive behavior and decreased across development for delinquent/rule-breaking behavior. Brain deficits predisposed individuals to more severe antisocial behavior if damage occurred earlier rather than later in life. In terms of hormones and neurotransmitters, cortisol levels were found to decrease across development for aggressive individuals. The relationship between high testosterone and antisocial behavior was weakest in young children and strongest in adults, while the effect of low serotonin on antisocial behavior was strongest in young adulthood. Furthermore, antisocial adults seem to suffer a greater degree of executive functioning deficits than younger antisocial populations. Studies examining biosocial interactions have also found that antisocial individuals exposed to fewer social stressors are more likely to exhibit biological risk factors compared to those with high social risk and that individuals are most likely to offend over the life course when both social and biological risk factors are present. Such interaction effects have been found in relation to child, adolescent, and adult offending.

The studies also revealed some differences in the associations between biological factors and offending for different types of offenders. For example, genetic influences were more important for childhood-onset compared to late-onset offending, brain deficits were associated with both early- and adolescent-onset offending, and a higher ratio of HVA to 5-HIAA was associated with life-course-persistent and child-onset offending. Additionally, it has been suggested that low heart rate, early spatial deficits, and perinatal complications may contribute particularly to life-course-persistent offending.

(p. 341) A. Future Directions

Despite these findings, greater research is needed to advance understanding of the role of biology in developmental and life-course criminology. One area of future research involves protective factors for offending. Although psychological and social factors such as attachment (Farrington 2005a) and life events (Wikström 2005) have been proposed in developmental and life-course theories as variables that inhibit offending, such theories do not consider the role of biological factors as possible protective factors. Studies such as that by Raine, Venables, and Williams (1995, 1996) have documented that high resting heart rate and skin conductance can serve as protective factors. For example, antisocial adolescents who desisted from adult crime had significantly better skin conductance conditioning at age 15 years than persisters who were criminal at age 29 years (Raine, Venables, and Williams 1996). Nonetheless, research on biological protective factors is very much more limited compared to that on risk factors. Additional research can provide much-needed insight on the topic of desistance.

Developmental and life-course criminology is also concerned with the effects of life events on the development of offending (Farrington 2005b). Recent findings have provided support for a social neurocriminology perspective, in which biological factors are influ-

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enced by social environmental processes to affect antisocial behavior, by documenting that low heart rate partly mediated the social adversity–antisocial behavior relationship (Choy et al. 2015; Fagan, Zhang, and Gao 2017). Given that marriage is associated with lower levels of testosterone in males (Gray et al. 2002) and the finding that adolescents who experience adversity in the form of maltreatment early in life exhibited lower gray matter volumes in corticostriatal-limbic regions such as the dorsolateral prefrontal cortex and amygdala (Edmiston et al. 2011), future efforts could be directed at understanding how social environmental factors can affect offending through changes in biology.

In addition, although several developmental models such as that of Moffitt (1993) and Lahey and Waldman (2005) recognize some biological influences, they do not necessarily emphasize the interactive effects between biological and psychosocial variables. Many theories still fail to incorporate biological factors such as genetics or hormones in understanding the etiology of offending (Barnes et al. 2014). More biological testing should be conducted particularly in prospective longitudinal studies to examine within-individual differences in offending and to investigate biosocial interaction effects. In light of the proposed notion that criminological variables are affected by genetic influences, longitudinal research can be beneficial in accounting for some genetic influences as respondents serve as their own control over many observation points (Barnes et al. 2014). Such efforts can greatly contribute to a better understanding of the role of biology in models of offending and pave the way for the development of early intervention and prevention strategies for crime reduction.

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