Why Developmental Criminology Is Still Coming of Age: The Influence of Biological Factors on Within-Individual Change

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With the founding in 2012 of the Division of Developmental and Life-Course Criminology, the study of the temporal dynamics of delinquent offending and its causes has come of age and a new impetus has been created to advance developmental studies of both offending and its causes. For the senior author of this chapter the origins of developmental study of offending started with the writing with Marc Le Blanc in 1988 of a first manifesto of developmental criminology inspired by many of our colleagues ("Toward a developmental criminology"), which was published two years later (Loeber & Le Blanc, 1990). Since that time, the chapter has been cited almost 500 times in the scholarly literature, which indicates how well other authors have, either positively or negatively, responded to the chapter. In a subsequent sequel, entitled "Developmental Criminology Updated," Marc and the senior author further expanded the theme, which was published in 1998 (Le Blanc & Loeber, 1998). Reflecting on these publications, the senior author recalls with fondness working with Marc, his clear conceptualization of difficult concepts concerning changes over time, and his vision for the future of developmental criminology. Marc certainly taught the senior author very much, helped him to grow into a developmental criminologist, and helped to develop the three longitudinal studies started by him and his colleagues, the Pittsburgh Youth Study, the Developmental Trends Study, and the Pittsburgh Girls Study.

Marc and the senior author's initial approach to developmental criminology focused on the specification of dynamic outcomes, such as the age of onset, the continuity in, and the desistance from offending. With an eye on causation of within-individual change over time, we reviewed developmental theories in criminology and particularly focused on social influences in young people's environment, including their parents, teachers, siblings, and peers. However, aside from establishing causation, insufficient focus was given to the role of biological factors on the development of individuals' offending. In the present chapter, we will briefly discuss how to best estimate causation in developmental and life-course criminology because this will set the stage for the discussion on the influence of biological factors on individuals' offending patterns, particularly the explanation of between-individual differences in offending compared to within-individual differences of offending with age. In the process of this review, we first seek to challenge biological studies to explain within-individual differences in offending as reflected in the age–crime curve. Second, we are interested in biological research that might explain changes in brain functioning as a result of systematic interventions. Third, we are interested to examine the role of biological

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factors to advance the screening of individuals at risk for serious delinquency, including violence.

Estimating Causes

Much has been written about the study of putative causes of offending (e.g., Farrington & Loeber, 2013; Murray, Farrington, & Eisner, 2009; Sampson, Winship, & Knight, 2013); in this chapter, we focus on some key biological elements. The major threats to the interpretation of causes come from the following:

- Reliance on correlates rather than predictors of offending.
- Not taking into account third factors that predict outcomes but which are not causal.
- For between-group comparisons of putative causes, the presence of inadequate comparison groups.
- Establishing causes between individuals instead of causes within individuals, with studies on between-individual differences suffering from more potential confounds than studies on within-individual differences. The advantages of the search of causes of within-individual differences lie in the fact that many factors associated with a given individual are controlled (or are the same) in these analyses, but this is not true for between-individual analyses.
- Along that line, examining by means of within-individual analyses whether events (such as marriage, having children, entering the work force) experienced by some individuals are followed by a change in their offending frequency.
- Choice of sample, with inferences about causation being very different when based on a normative sample (e.g., community sample) compared to a select sample (e.g., prisoners). Normative samples are best for the study of causes of the onset, continuity, and desistance from offending. In contrast, select samples, such as known delinquents, population samples, or individuals on parole, are best for the study of the causes of reoffending, or the causes of desistance among former offenders.

It is well known that key demonstrations of causality are difficult to achieve even with carefully executed longitudinal data. Superior tests of causality are quasi-experimental analyses or, even better, randomized trials in which some participants receive a particular intervention consisting of the modification of putative causal factors, while randomized controls receive no intervention. It should be understood, however, that most interventions attempt to change more than a single target, and that for that reason, inferences about causality in randomized intervention trials are not always conclusive.

Biological Factors

There is a flourishing literature on the biological bases of delinquency. Among the most recent overviews of biological factors are Adrian Raine's *The Anatomy of Violence* (New York, 2013) and Susman and Polak (2013), which extend to other major publications such as Hodgins, Viding, and Plodowski's *The Neurobiological Basis of Violence* (Oxford, 2009), and Raine, Brennan, Farrington, and Mednick's *Biological Bases of Violence* (New York, 1997). In this chapter biological factors include brain functioning (Séguin, Pinsonneault, & Parent 2015), neurotransmitters, physiological arousal, neurotoxins, genetic influences, and gene–environment interactions (Beaver, Schwartz, & Gajos, 2015). Raine (2013) succinctly summarized that genes influence brain structures and brain structures influence violence. More directly, genes influence neuroreceptors and neurotransporters (such as serotonin, MAOA, and 5-HT). The major brain structures relevant to violence are frontal and limbic/subcortical, with the latter operating through the amygdala and the hippocampus, and the anterior cingulate insula. Where we (Loeber & Pardini, 2008) differ from Raine (2013), pointed out that the relationship between biological factors and violence at the behavioral level is not always direct, but that the impact of biological factors on

violence often is mediated by underlying factors such as impulsivity or anger (e.g., Blair, 2012), and at early stages of development, less serious forms of aggression.

Critique of the Nonintervention Biological Studies

The promise of biological factors explaining delinquency and violence, although often touted, has only been partially realized (e.g., Burt & Simons, 2014). Loeber and Pardini (2008) found that the vast majority of biological studies of delinquency and violence show cross-sectional associations between the two rather than being based on biological factors predicting later delinquency outcomes (but see Jennings, Piquero, and Farrington (2013) showing that resting low heart rate predicted total and violent convictions up to age 50 and Aharoni et al.'s (2013) error-related brain activity predicting subsequent rearrest). Whereas Ortiz and Raine (2004, p. 154) found that low resting heart rate was the best-replicated biological correlate of antisocial and aggressive behavior, a recent meta-analysis of 115 effect sizes of resting heart rate versus antisocial behavior by Portnoy and Farrington (2014) found that there were only 15 effect sizes based on prospective studies while *none* of the studies reported effect sizes based on within-individual changes in offending.

It is not uncommon that postdiction happens in studies, with biological factors linked to past rather than future delinquency outcomes (e.g., Pardini, Raine, Erickson, & Loeber, 2014). Moreover, Loeber, and Pardini (2008) reported that "if longitudinal analyses were executed, they usually focused on comparison between deviant and nondeviant groups rather than on developmental types of offenders (e.g., early versus late-onset offenders) or developmental change in offending (e.g., persistence versus desistance from offending). Thus, neurobiological studies that treat the dependent variable of violence as a dynamic phenomenon have been relatively rare" (p. 2492). This applies to the explanation of the age–crime curve in offending, developmental pathways toward serious violence, desistance processes, and different developmental types of offenders (Loeber & Pardini, 2008, p. 2492).

Further, here is evidence for a GxE (gene–environment interaction), whereby exposure to early maltreatment was more likely to lead to antisocial behavior among those carrying a specific genotype (low activity MAOA) (e.g., review by Taylor & Kim-Cohen, 2007). In recent years the focus on identifying genes has shifted to the search for gene–environmental interactions. A recent meta-analysis by Byrd and Manuck (2014) focused on normative samples, and found no main effect for MAOA but that MAOA genotype moderated the association between early life adversities (maltreatment and other adversities) and later aggressive and antisocial outcomes ($p = 0.0044$). However, the authors failed to find replication for females. This may be linked to the fact that the gene encoding for MAOA is located on the X chromosome—since males only have one X chromosome and females have two X chromosomes. In addition, the results were much stronger for cross-sectional than for longitudinal studies ($p < 0.0045$ vs. $p = 0.019$). The available studies show that the interaction is specific to early maltreatment and does not hold for "environmental factors" or "early adversity" broadly speaking. It remains unclear why this would be the case. In addition, Duncan, Pollastri, and Smoller (2014) have pointed that replication has not always taken place in the largest samples, which would have the biggest statistical power to detect interaction effects.

In summary, studies have documented a single gene–environment interaction in several studies. These studies are based on comparisons between individuals and have not been studied yet on within-individual change in behavior over time. In addition, it is highly plausible that other genetic main or interaction effects remain to be discovered.

Most of the biological literature can be criticized for not being oriented to

developmental approaches to offending, and not explaining within-individual changes in delinquency. Instead, the focus of most biological studies of crime has been on between-individual differences and has assumed trait-like features of antisocial behavior or offending in those individuals who are thought to differ in their antisocial or delinquent propensity. Individual differences, also called the trait approach to the study of behavior (Morizot, 2015), is a key element in several theories, including Gottfredson and Hirschi (1990), Moffitt (1993), and Patterson, Reid, and Dishion (1992). Although understandable, there are major limitations to accepting the trait approach to offending. The key assumptions of the individual difference approach are that individual differences originate in childhood, that there is a relative high stability of behaviors over time, and that individual differences stay approximately the same over time. As we will elaborate below, the biological explanation of individual differences needs to be complemented by a biological explanation of within-individual changes over time.

Loeber and Pardini (2008) pointed out that scholars often have assumed that individual differences in neurobiological factors associated with violence are stable over time. The notion of stable individual differences is usually based on a rank ordering of individuals and whether such rank ordering is stable over time. Less often considered is whether the relative ranking between individuals remains constant with development. The relative proportional stability of individual differences is not supported by two types of data. First, the vast majority of trajectory models that distinguish between different categories of individuals, with each category following a distinct development outcome (Piquero, 2007; Piquero, Reingle Gonzalez, & Jennings, 2015), show distinct differences among categories of individuals who appear to be small at some ages and larger at other ages. Second, individuals tend to differ in terms of their age–crime curve, but these differences are not constant along the curve; instead, the differences are the largest at the peak of the curve, and much smaller in the upslope or downslope of the curve. Thus, the notion of stable, unchangeable individual differences in offending is probably wrong and individual differences may be larger at the peak of the age–crime curve than in the upslope or the downslope. This means that individual differences do exist but may be initially modest, then increase, and later diminish over time.

What is less clear however, is to what extent, the *downslope* age–crime curve is reflective of other forms of development, particularly the growth of internal controls and the decrease of impulsivity and sensation seeking and how these changes are linked to changes in brain functioning. Loeber and Farrington (2012) postulated that changes in internal controls with increasing age can be gauged by the following:

- More mature judgment.
- Better decision making in offending opportunities.
- Better executive functioning, reasoning, abstract thinking, planning.
- Less influenced by immediate undesirable consequences than longer term possible desirable consequences.
- Better impulse control, less likely to take risks and commit crimes for excitement and more likely to make rational prosocial choices.
- Better emotion regulation and self-regulation.
- Less susceptible to peer influences.
- Avoidance of self-harm.

Thus, the idea is that the peaking and falling in offending is directly correlated with the rise, peaking, and fall in impulsivity, sensation seeking, and several forms of cognitive and emotional self-control and under-control. Monahan and colleagues (2009) postulated that improvement in self-control during adolescence may explain desistance from delinquent behavior, presumably in the downslope of the age–crime curve when most desistance takes place (Loeber & Farrington, 2012; Moffitt, 1993). Although this seems plausible, there is no doubt that desistance from offending can take place prior to adolescence, during the earlier part of the age–crime curve. Thus, there can be desistance from offending for early-onset cases who subsequently desist in late childhood, while also

desistance is known to take place during early adolescence (Loeber & Farrington, 2008). Loeber, Pardini, Stouthamer-Loeber, and Raine (2007), using longitudinal data from the Pittsburgh Youth Study, found that *none* of the cognitive, physiological, parenting, or community factors, measured at age 16, predicted young men's desistance from offending between ages 17 and 20. In summary, there are currently no known physiological predictors of desistance from offending, and the biological underpinning of desistance, if any, remains unknown (see Kazemian, 2015).

Scientists have focused much more on the explanation of the downslope rather than the *upslope* of the age–crime curve. For example, Steinberg and colleagues (Steinberg et al., 2006) postulated a maturity gap that emerges in adolescence, with the body maturing (as evident from sexual development), accompanied by heightened sensation seeking and risky behaviors but also accompanied by a delay in the maturation of the brain in producing control mechanisms. Steinberg referred to this as "starting the engines without a skilled driver" (Steinberg et al., 2006). This mom-and-pop "mechanism" requires a better scientific explanation and formulation of a testable set of hypotheses of the mechanisms involved. A cross-sectional comparison between adolescents and emerging adults using proton magnetic resonance spectroscopy in a small number of subjects suggested frontal lobe GABA receptors maturation in the frontal brain (Silveri et al., 2013). Although a promising finding, only repeated measurements of brain functioning can demonstrate that GABA maturation and no other plausible mechanisms operate.

Genetic research may provide a clue for developmental-phase specific changes because genetic effects do not necessarily operate over the full life span, but may become active in certain age periods such as adolescence. Burt and Mikolajewski (2008) presented some evidence that specific candidate genes are associated with adolescent-onset antisocial behavior. As another example, early exposure to stress affects the HPA (hypothalamic–pituitary–adrenal) axis—(Van Goozen, 2005). Although there are many between-individual studies linking biological factors to delinquency and violence (see Raine, 2013), it remains unclear which biological factors can best explain the upslope and downslope of the age–crime curve for individuals. In addition, we do not know of biological factors that can explain escalation in the severity of offending that typically covaries with the shape of the age–crime curve.

The key question here is whether there are biological factors that are absolutely constant that can explain the age–crime curve or escalation patterns, or whether we need to think more about biological factors that change along the age–crime curve and influence its shape. Thus, most of the biological studies have not examined the extent to which biological factors emerge over time in individuals, change within individuals, and gradually exert their influence over delinquent behavior. Therefore, there is an urgent need for studies examining brain maturation in longitudinal follow-up samples. In addition, since interventions often focus on the reduction of impulsive behavior and the improvement of self-control, the impact of interventions on brain development is a very worthy topic of study (see below).

Although the age–crime curve appears universal, there are important variations of the curve which are associated with differences between individuals. Particularly, as we will see, the height of the curve is different for different populations, and the width of the curve is smaller in some populations and broader in other populations. The study of the variations in the shape of the age–crime curve is important because it tells us which possible causal factors are at play.

Almost all knowledge about the age–crime curve is based on cross-sectional data and rarely on the repeated measurements of delinquency of the same participants. Some important variations in the age–crime curve are: (a) the curves are higher for participants living in disadvantaged neighborhoods (Fabio, Tu, Loeber, & Cohen, 2011); (b) the curves tend to be higher for youth with cognitive impulsivity in early adolescence (Loeber et al., 2012); (c) the curve tends to be higher for youth of low intelligence (Loeber

et al., 2012); (d) the curves can be dramatically higher for some compared to other birth cohorts (Loeber & Farrington, 2008); and (e) the curves tend to peak earlier for females than males (Farrington, 1986).

Thus, there are important differences between individuals in the height and, possibly, the width of the age–crime curve. In that way, the age–crime curve represents both normative *and* deviant development. The normative development of the age–crime curve is that most youth appear to go through the age–crime curve. However, the deviant aspect of the age–crime curve is that some individuals experience a much higher and broader age–crime curve than others.

We want to conclude this section with one key other issue: the explanation of gender differences and within-gender explanations of antisocial behavior among girls. This neglected area needs to be advanced. For example, we need answers to the following questions: Why does the MAOA interaction with abuse not apply to girls? What is the genetic component of girls' antisocial behavior? What are the biological causes of the earlier peaking of the age–crime curve for females compared to males? Are there biological reasons for a larger proportion of adult-onset of antisocial behavior in females compared to males?

Interventions and Subsequent Brain Changes

To return to within-individual change over time, a key question in brain research is whether it is possible to demonstrate that specific functions of the brain change as a result of intervention to promote self-control and decrease offending. Thus, the key issue for interventions is whether there are possibilities to speed up decreased impulsivity and increased brain maturation, especially for vulnerable categories of youth. One of the advantages of the study of brain changes comparing pre- and posttreatment conditions is that they require data collection of within-individual differences over time and as such are important to the study of biological mechanisms underlying antisocial/behavior.

There are a few projects that have examined neural changes associated with treatment of children, of which we will highlight the study by Woltering, Granic, Lamm, and Lewis (2011). The authors studied children undergoing SNAP Treatment (Stop Now And Plan), a program to stimulate self-control in acting out children. The study found that, prior to treatment, the boys referred for externalizing behavior problems tended to process more from the ventral region of the brain (the "threat" center). The post-treatment examination of the children after 12 weeks showed marked improvements in their behavior using standardized behavioral rating measures and the brain measurement no longer showed hyper-firing in the ventral region. The children also reported that they were more relaxed and were less anxious (not feeling as threatened). This improvement was also reflected in the boys' performance on the Go/No-Go Points task, which was formulated as an anxiety-inducing game with the promise that "You get an amazing prize if you win." However, anxiety induction was used by programming the game so that the boys lost all points halfway through the session. The researchers examined differences in brain activation when the boys tried to inhibit their impulses and expected dorsal activation to increase as a function of treatment (they're going to exert more "top-down" control of their emotions).The Woltering et al. (2011) study is one of Pre–Post Intervention Studies showing pre–post changes in brain function following interventions aimed at impulse control. What is less clear, however, is the exact mechanism involved in the pre–post changes, and to what extent they concern anger or anxiety, or a combination of these. There is much need for replication of these pre–post intervention studies using within-individual brain measurements over time.

In addition, Brody and colleagues (2013) have pointed out the need for intervention researchers to undertake genetically informed randomized prevention trials, which focus on GxI (gene/intervention interactions) and their effects on mediators and intermediate processes related to

deviance (Brody et al., 2013). Although the authors focused on drug use and psychopathology as outcomes, their conceptualization could also be applied to delinquency and violence.

Screening Using Biological Factors

If basic biological factors are as important as is often claimed (e.g., Raine, 2013), to what extent is such information useful for practical purposes? One of these purposes is the screening of youth at risk for serious forms of delinquency. To the best of our knowledge, there are no screening instruments that have incorporated information on MAOA, heart rate, or any other biological measure other than gender (see reviews by Hoge, Vincent, & Guy, 2012; Le Blanc, 1998).

Summary

- This chapter emphasized the role of biological factors in intraindividual changes in criminal and antisocial behavior. We focused on gene–environment interactions and the limitations of the research to date.
- Most biological studies to explain delinquency and violence relied on between-individual differences, have been cross-sectional, and have not sufficiently dealt with the within-individual changes in offending during development, including the age–crime curve.
- Currently, biological factors are not included in the most used screening instruments to identify youth at risk for reoffending.
- We also reported on how intervention studies can approximate changes in brain functioning and that there is a future for genetically informed intervention randomized trials. According to Raine (2013, p. 59): "We stand on the threshold of unlocking many untold secrets of our genetic makeup. . ." of antisocial and violent behavior. However, this promise has yet to be fulfilled.

Future Research Needs

We advocate research in the following areas of the interaction between biological and environmental factors:

- Explanation of the upslope and the downslope of the age–crime curve, particularly in terms of biological factors explaining within-individual changes in offending and changes in putative underlying factors such as impulsivity and sensation seeking.
- Replication of findings in females of biological factors that apply to males and better explanations of why in certain instances biological factors explain in one gender but not the other.
- Better pinpointing which areas of the brain show changes in functioning as a result of successful interventions.
- The use of genetically informed interventions that can shed led light on crucial mediating processes.

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