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Neurobiology and the development of violence: common assumptions and controversies

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This paper addresses four common assumptions and related controversies regarding neurobiological factors explaining violence: (i) scholars often assume stability of individual differences in neurobiological factors pertaining to violence, yet much change occurs in aggression/violence during the life course, (ii) individual differences in aggression/violence reflect one or more underlying mechanisms that are believed to have neurobiological origins, yet there is little agreement about which underlying mechanisms apply best, (iii) the development of aggression/violence to some degree can be explained by social, individual, economic and environmental factors, yet it is unclear to what extent neurobiological factors can explain the escalation to, and desistance from, violence over and above social, individual, economic and environmental factors, and (iv) violence waxes and wanes in society over time, yet the explanation of secular differences in violence by means of neurobiological and other factors is not clear. Longitudinal analyses from the Pittsburgh Youth Study are used to illustrate several of these issues.

Keywords: neurobiology; aggression; violence; development; underlying factors; prediction

1. INTRODUCTION

Violence and serious property crime continue to lead to high levels of personal injury and financial damage for victims (Welsh *et al.* 2008), and stimulate concerns for safety and increased costs for security, police and preventive efforts. For example, for the year 2000 alone, the total costs resulting from non-fatal injuries and death due to violence were more than $70 billion in the USA (Corso *et al.* 2007). Research on the victim costs of crime shows that the victim costs of an average chronic juvenile offender committing crime between ages 7 and 17 amounts to about $1.25 million (based on self-reported delinquency; Welsh *et al.* 2008).

Despite a substantial decrease in violence perpetration and victimization since *ca* 1991–1993 in the USA (e.g. Blumstein & Wallman 2000; Baum 2005), the American prison population quadrupled between 1980 and 2000 (Rosenfeld 2004). Similarly, the British Crime Survey shows that in England and Wales violence had decreased since 1995 (Newburn 2007), but the prison population increased by two-thirds between 1993 and 2005. Thus, in both countries, legislation and the courts increased their emphasis on implementing punitive sanctions for crime rather than addressing the multiple causes—neurobiological, individual, social, economic and environmental processes—of criminal behaviour. Despite this practice, it is clear that furthering our understanding of the mechanisms through which neurobiological and other factors influence violence is the only way in which societies can develop and enact policies that will serve to maintain sustainable reductions in violent crime over time. In the following text in which we will explore these issues in greater detail, the term violence will refer to forcible robbery, attacking with intent to injure, sexual coercion or rape. Aggression on the other hand will refer to lesser injurious acts, while antisocial behaviour is a general term encompassing aggression, violence and non-violent forms of delinquency.

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2. MODELLING NEUROBIOLOGICAL AND SOCIAL, INDIVIDUAL, ECONOMIC AND ENVIRONMENTAL INFLUENCES ON VIOLENCE

To aid in the exploration of how neurobiological and social, individual, economic and environmental factors influence the developmental progression of violence, figure 1 shows a basic heuristic model based on existing theoretical and empirical work in the area. This model represents the interrelationship between several broad-based factors believed to be important in the development of violence, including genetics, social, economic and environmental influences, and the neurobiological factors of brain structure and function. In this model, social influences are posited to encompass interactions between the individual and parents, siblings, peers and eventually, partners and co-workers. Individual factors are factors such as academic achievement and school motivation. Economic factors comprise such factors as family socioeconomic status and welfare. Examples of environmental factors are neighbourhood disadvantage and school climate. It is hypothesized that genetic, social and other factors and their interaction contribute to changes in neurobiological structure and function, which in turn influence a developmental cascade of behaviours that can eventually lead to violence. Specifically, brain structure and function are believed to influence early emerging underlying factors that

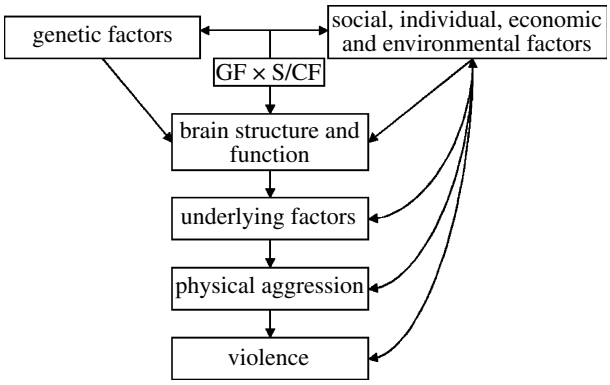

Figure 1. Relationships between elements of neurobiological and social/contextual factors that influence the development of violence.

increase one's propensity for violence (such as temperament), which in turn affect minor forms of aggression that then influence the probability of more serious violence. This cascade is also recursive, given that underlying factors, minor forms of aggression and violence can serve to influence social and environmental factors over time, as well as being influenced by them. While this heuristic model is thought to apply to both males and females, the nature of the factors contained within each broad-based category (e.g. genetic, social, individual, economic and environmental factors) that are important for understanding the development of violence may differ by gender.

Many neurobiological studies of violence often share a number of implicit or explicit assumptions about violence. These assumptions include:

— Individuals differ in their propensity to commit violence, and these differences are already present early in life.
— Aggression and violence have elements in common with personality traits that are presumed to be stable in individuals (e.g. Olweus 1979).
— The underlying causes of aggression and violence are attributed to factors present early in life, especially neurobiological factors.

While these assumptions are reasonable, the vast majority of neurobiological studies of violence have been cross sectional (or retrospective) rather than longitudinal and have concentrated on comparisons between offenders and non-offenders or between aggressive and non-aggressive youth (e.g. Raine 1993; Fishbein 2000; Rowe 2002). Even when studies are based on longitudinal data, the analyses are often focused on comparisons between deviant and non-deviant groups rather than those between developmental types of offenders (e.g. early versus late-onset offenders) or developmental change in offending (e.g. persistence in versus desistance from offending). Thus, neurobiological studies that treat the dependent variable of violence as a dynamic phenomenon have been relatively rare.

Over the years, the number of longitudinal studies has increased and findings on the stability and change in offending have clarified individual differences in developmental growth of violence and the factors influencing such growth. Thus, we know much more about individual differences pertaining to the age–crime curve, developmental pathways to violence, desistance and the types of individuals following different trajectories of violence. Also, we know more about underlying factors that are postulated to cause individual differences in the propensity for violent behaviour within populations. Some of these underlying factors, such as fearlessness and behavioural dysregulation, are thought to change with development. We pose that it is time to re-examine aggression and violence and underlying factors as dependent variables in neurobiological studies and ask ourselves which aspects of aggression, violence and underlying factors are best explained by neurobiological factors.

Yet, another aspect of neurobiological factors requires attention. Researchers agree that the impact of genetics, brain structure and functioning on behaviour is not immutable but can be changed through human interactions (e.g. Taylor & Kim-Cohen 2007). To what extent do social and other factors predict violence, and do they predict homicide as the most extreme expression of violence as well? How much is known about the degree to which neurobiological factors add to these predictors of violence, either as main effects or through interactions with social and environmental factors?

It is also important to recognize that the prevalence and incidence of violence in societies are rarely constant over decades. Instead, studies show large secular changes in violence in a matter of years. The causes of such secular changes have given rise to much speculation (e.g. Blumstein & Wallman 2000), and have included population structural factors (such as the size of crime-prone cohorts and immigration), poverty, family disruption, violence in the media, gang membership, teenage childbearing and many other factors. The question again is to what extent neurobiological factors explain secular changes in violence for populations of individuals as either main effects or in interaction with different population structural factors.

In summary, the present paper addresses the following four controversies regarding neurobiological factors explaining violence:

— Scholars often assume stability of individual differences in neurobiological factors pertaining to violence, yet much change occurs in aggression/violence during the life course.
— Individual differences in aggression/violence reflect one or more underlying mechanisms that are believed to have neurobiological origins, yet there is little agreement about which underlying mechanisms apply best.
— The development of aggression/violence to some degree can be explained by social and other factors, yet it is unclear to what extent neurobiological factors explain violence over and above the explanatory power of social and other factors. Also, the role of neurobiological factors in the escalation to, and desistence from, violence is poorly understood.
— Violence waxes and wanes in society over time, yet the explanation of secular differences in violence by means of neurobiological and other factors is not clear.

The four topics are interrelated: knowledge of stability and change in violence is the dependent variable that neurobiological factors attempt to explain. Underlying mechanisms, neurobiological, social and other factors constitute elements of models to explain violence. Finally, these explanations when applied to successive age cohorts are relevant to the explanation of secular changes in violence. In addressing these issues, part of this paper is based on longitudinal analyses from the Pittsburgh Youth Study[1] (PYS; Loeber *et al.* 1998, 2008). Owing to space limitations, this paper is not a review but serves to illustrate key points.

3. DEVELOPMENTAL CHANGE IN VIOLENCE

Neurobiological, social and other explanations of violence should take into account that there are at least four key sets of differences among individuals: the age–crime curve; desistance; developmental pathways; and developmental trajectories.

(a) *Age–aggression and age–violence curves*

There are at least two age-related normative curves relevant to the study of neurobiology of aggression and violence. The first curve concerns aggression and indicates that aggression is high in childhood and decreases afterwards. For example, Nagin & Tremblay (2005) have provided evidence that physical aggression in childhood peaks around age 2 and then decreases (see also Kingston & Prior (1995) and NICHD Early Child Care Research Network (2004)). There are individual differences, however, in terms of the timing and rate of outgrowing aggression during the preschool years. We are not aware of neurobiological studies that explain why this is earlier for some children than for others.

The second normative curve is usually called the age–crime curve (Farrington 1986). The age–crime curve for property crime and violence is a universally observed curve showing that the *prevalence* of offenders is low in late childhood and early adolescence, peaks in middle to late adolescence and decreases subsequently (Farrington 1986; Laub & Sampson 2003; Tremblay & Nagin 2005). The curve for violence is similar but tends to peak somewhat later (Loeber *et al.* 2008). The curve is slightly earlier for girls than boys (Farrington 1986; Elliott *et al.* 2005), which is indicative of a higher proportion of late-onset cases during adolescence in boys than girls.

The two age–antisocial behaviour curves pose a major challenge for the explanation of violence on the basis of neurobiological factors. Studies on neurobiological factors to date have not addressed why there is an increase, peaking and a decrease in offending in the same individuals over many years and why there are individual differences in the upslope, peaking and downslope of that curve. Somewhat of an exception is a study by Loeber *et al.* (2007) using data from the PYS, which in addition to social and other factors examined heart rate and galvanic skin response. Predictive analyses discriminating between desisters and persisters in delinquency between ages 17 and 20 showed that all of the significant predictors were either child or peer risk factors. None of the cognitive, physiological, parenting or community factors significantly predicted desistance from delinquency. The results leave open the possibilities that other neurobiological factors can explain desistance in the downslope of the age–crime curve, a point that we will return to when discussing underlying factors.

(b) *Desistance*

Desistance refers to individuals' cessation of delinquent acts. The notion that desistance primarily takes place in the downslope of the age–crime curve (e.g. Moffitt 1993) is mistaken because the age–crime curve is a prevalence curve and not a curve on the relative persistence of offending. Instead, desistance from antisocial behaviour and delinquency, including violence, takes place throughout childhood and adolescence (Tremblay *et al.* 2004; Prinzie *et al.* 2005; Loeber *et al.* 2008). Longitudinal research also shows that there is discontinuity in violence for a proportion of violent offenders. For example, in the PYS, desistance processes relating to violence operated from at least late childhood onwards and were documented throughout adolescence and early adulthood (Loeber *et al.* 2008). The probability of desistance from serious offending (which includes violence) is inversely related to age of onset (particularly, onset in late childhood is negatively associated with later desistance). Only one quarter of the early onset offenders desisted in serious offending later.

(c) *Developmental pathways*

Scientists are interested in very high-risk individuals likely to express violent behaviour years later. However, because violence as a rule emerges in middle to late adolescence, it is also necessary to focus on less serious forms of aggression that are developmental precursors to violence. Individuals differ in their development of different severity levels of aggression, with some developing minor aggression only while others progress to serious and repeated violence. Conceptually, this is often referred to as a developmental pathway to violence, with most individuals progressing very little on that pathway and a minority progressing to the most extreme forms of violence. Loeber and colleagues identified three empirically charted pathways in the PYS that youngsters typically follow in a remarkably orderly progression from less to more serious problem behaviours and delinquency from childhood to adolescence (Loeber *et al.* 1993, 1997, 2005). The development from minor antisocial behaviours to serious delinquency best fits a model of three incremental pathways (figure 2): (i) an overt pathway, which starts with minor aggression, has physical fighting as a second stage and severe violence as a third stage, (ii) a covert pathway prior to age 15, which starts with minor covert acts, has property damage as a second stage and moderate to serious delinquency as a third stage, and (iii) an authority conflict pathway prior to age 12, which starts with stubborn behaviour, has defiance as a second stage and authority avoidance (e.g. truancy) as a third stage. The pathways model has been documented in four longitudinal datasets (Tolan & Gorman-Smith 1998; Loeber *et al.* 1999; Tolan *et al.* 2000) and largely applies to girls as well (Gorman-Smith & Loeber 2005).

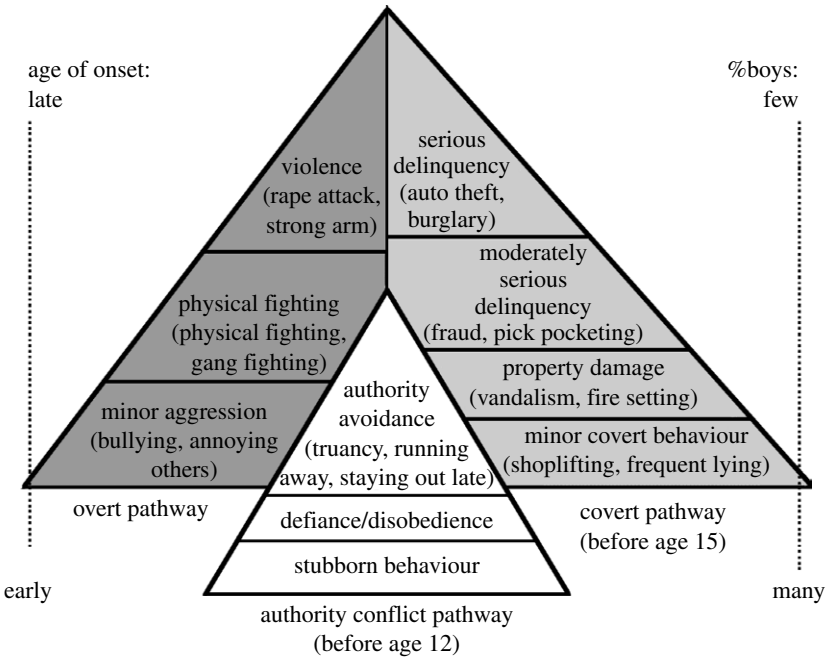

Figure 2. Developmental pathways to violence, property crime and authority conflict problems.

Some individuals were on a single pathway (e.g. some are on the authority conflict pathway but not on the other two pathways). The most affected individuals escalated on all three pathways. Escalation in either the overt or covert pathway was often preceded by boys' escalation in the authority conflict pathway (Loeber *et al.* 1993). In other words, conflict with authority figures was either a precursor or a concomitant of boys' escalation in overt or covert acts. Also, an early age of onset of problem behaviour or delinquency, compared with a later onset, was more closely associated with boys' escalation to more serious behaviours in the overt and covert pathways (Tolan *et al.* 2000). The pathway model accounted for the majority of the most seriously affected boys, that is, the self-reported high-rate offenders and court-reported delinquents (Loeber *et al.* 1993, 1997). In summary, developmental pathways in antisocial behaviour/delinquency and developmental transitions between different disruptive diagnoses share a conceptualization of escalation in the severity of antisocial behaviours with development in certain individuals but not in others. The pathway model also represents selection processes, in that increasingly smaller groups of youth become at risk for the more serious behaviours, comparable with a successive sieving process.

(d) *Developmental trajectories*

The specification of developmental pathways can be contrasted with the identification of developmental trajectories (sometimes called developmental types), which are defined as the classification of individuals according to their pattern of deviant behaviour over time. The assumption is that a population of individuals 'is composed of a mixture of groups with distinct developmental trajectories' (Nagin & Tremblay 2001, p. 21). Typically, trajectory analyses have been based on repeated measurements of a *single* indicator of problem behaviour. Usually, the results of trajectory analyses identify young males whose problem behaviour remains high over time, those who remain low, those whose problem behaviour increases and those whose problem behaviour decreases between childhood and early adulthood (e.g. Broidy *et al.* 2003; Bushway *et al.* 2003; Lacourse *et al.* 2003; Piquero in press). For example, figure 3 shows the developmental trajectories for violence in the oldest sample of the PYS (Loeber *et al.* 2008). The results show four violence trajectories: *no/low* (51.76%), *moderate declining* (28.4%), *high declining* (5.6%) and *late onset* (5.6%). Thus, not all trajectories of violence started at earliest measurement for this cohort (age 13); some violence emerged during adolescence. This late-onset trajectory has also been documented in other studies (e.g. Brame *et al.* 2001) and presents a new view on the development of violence. We have not found studies in which neurobiological factors predicted late-onset violence.

(e) *Controversies about developmental change*

We briefly reviewed developmental change relevant to violence from four angles: the age–crime curve, desistance, developmental pathways and categories of individuals with different developmental trajectories. Each of the four approaches indicates that stability in aggression and violence does occur. However, each of these approaches also elucidates the fact that there are major individual differences in aggression/violence that emerge over time, with some never escalating from minor forms of aggression to violence, some starting violence rather late and others desisting from aggression and violence. These findings pose a considerable challenge to neurobiological studies with the assumption that stable individual differences are trait-like for all aggressive or violent individuals and are already present early in life for *all* of those who eventually become violent. This is an oversimplification that runs counter to the developmental data. The next generation of neurobiological studies can benefit from addressing stability and developmental change in all its different expressions and, by doing so, become more

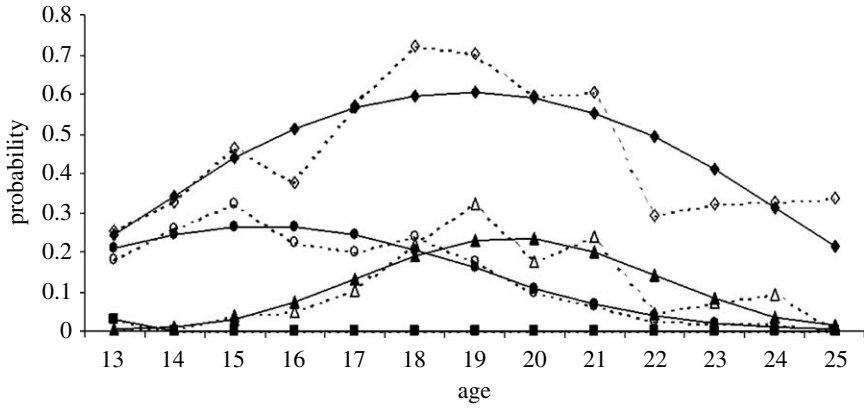

Figure 3. Developmental trajectories of violence in the oldest sample of the Pittsburgh Youth Study. Open squares, no/low Obs; filled squares, no/low Pred; open triangles, late-onset Obs; filled triangles, late-onset Pred; open circles, moderate declining Obs; filled circles, moderate declining Pred; open diamonds, high declining Obs; filled diamonds, high declining Pred.

representative of the variety of developmental expressions of aggression and violence that actually occur in the early life course of males and females.

4. IDENTIFYING UNDERLYING FACTORS

Research on the neurobiology of violence is based on the long-standing belief that certain individual difference characteristics play an important role in the development of violence. We will refer to these characteristics as *underlying factors* because they are postulated to underlie individual differences in the propensity for violent behaviour within the population. Several of the assumptions made about underlying factors, either implicitly or explicitly within the research literature, make them relevant to the study of neurobiological factors. These assumptions are similar to those articulated in Introduction of this paper and mainly pertain to the developmental origins and course of underlying factors. Perhaps the most common assumption is that underlying factors represent the behavioural manifestations of genetically driven differences in neurobiological functioning that subsequently lead to a predisposition for violent behaviour. Along these lines, underlying factors are often presumed to be stable and observable in early development (often referred to as temperament) well before the onset of violent behaviours. Additionally, these factors are frequently described as being useful for identifying a homogenous subgroup of individuals who have a common neurobiological risk factor for violence. Despite their inferred genetic and neurobiological underpinnings, a vast majority of studies continue to assess underlying factors using indirect methods such as rating scales, behavioural observations or performance-based tasks. As a result, there is often a fundamental disconnect between the theoretical conceptualization of underlying factors and the operationalization of these constructs.

While it is commonly accepted that no single underlying factor accounts for individual differences in the propensity towards violence, there is considerable disagreement among scholars on which underlying factors are most important for understanding the development of violence. Most contemporary models posit that there are two or more causal pathways to violence that are at least partially driven by underlying factors (Frick & Morris 2004; Raine 2002; Lahey & Waldman 2003; Blair *et al.* 2006). However, the relative importance placed on specific underlying factors varies greatly across developmental models, and there is continued debate about the relative use of specific underlying factors for understanding the development of violence. Despite these complexities, three broad categories of underlying factors that have received considerable attention in the theoretical and empirical literature are factors related to emotional/behavioural dysregulation, cognitive impairments and deficient processing of aversive stimuli. The empirical evidence linking these broad-based categories to early conduct problems and violence will be briefly reviewed here to provide a foundation from which to discuss ongoing controversies and implications for neurobiological research. Readers interested in more extensive discussions of the putative neurobiological mechanisms associated with these underlying factors are referred to subsequent articles in this issue, as well as previously published reviews (Frick & Morris 2004; Raine 2002; Lahey & Waldman 2003; Blair *et al.* 2006).

(a) *Emotional and behavioural dysregulation*

General problems related to emotional and behavioural dysregulation have long been implicated as underlying factors in the development of violence. Many studies characterize problems with emotional dysregulation as frequent experiences of negative affect (particularly irritability and anger), sudden mood swings and intense negative emotional reactivity with very little provocation (Lahey & Waldman 2003). On the other hand, behavioural dysregulation has been characterized by overactivity, poor inhibitory control, impulsivity, restlessness and inattention (Lynam 1996; Waschbusch 2002). While developmental studies on antisocial behaviour have clustered aspects of both emotional and behavioural dysregulation into a single construct referred to as a difficult (Giancola *et al.* 2006) or undercontrolled temperament (Henry *et al.* 1996), this practice has largely been abandoned as evidence suggests that these features are distinct constructs that may uniquely contribute to the development of psychopathology (for discussion, see Rothbart 2004).

Studies have consistently linked emotional dysregulation (particularly irritability and anger) to conduct problems and violence in children and adolescents. Features of emotional dysregulation have been associated with childhood aggressive behaviour across several different cultures (Rothbart *et al.* 1994; Chang *et al.* 2003; Oldehinkel *et al.* 2004). Moreover, this association has been found using several different assessment methods, including measuring emotional dysregulation using parent-report scales (Olson *et al.* 2000), behavioural observations (Owens & Shaw 2003), social cognitive measures (Orobio de Castro *et al.* 2002; Vitale *et al.* 2005), physiological measures of vagal reactivity (Beauchaine *et al.* 2007) and functional magnetic resonance imaging (fMRI) measures of neural reactivity to provocation (Coccaro *et al.* 2007; Eisenberger *et al.* 2007). In addition, there is evidence that the covariation between emotional dysregulation and aggression is influenced by shared genetic factors (Gjone & Stevenson 1997), and that emotional dysregulation predicts the development of aggression in children even after controlling for features of behavioural dysregulation (Rothbart *et al.* 1994).

Several studies have also found a consistent association between early behavioural dysregulation and childhood conduct problems (Rothbart *et al.* 1994; Gjone & Stevenson 1997; Lemery *et al.* 2002). It is well documented that conduct problems co-occur with difficulties related to behavioural dysregulation (Angold *et al.* 1999; Waschbusch 2002), with changes in attention deficit hyperactivity disorder symptoms paralleling fluctuations in conduct disorder symptoms over time (Lahey *et al.* 2002). However, studies exploring the prospective relation between problems with behavioural dysregulation and later antisocial and violent behaviour have provided somewhat mixed results (Lahey *et al.* 1995, 2002; Lynam 1996; Waschbusch 2002; Broidy *et al.* 2003). Specifically, after appropriately controlling for initial levels of antisocial behaviour, a majority of recent longitudinal investigations have found non-significant associations between features of childhood behavioural dysregulation and later antisocial behaviour (Lahey *et al.* 1995, 2002; Broidy *et al.* 2003). As a result, the ability of measures of behavioural dysregulation to provide unique information about the developmental course of antisocial and violent behaviour is questionable.

(b) *Cognitive impairments*

Another broad set of underlying factors implicated in the development of violence includes performance-based measures of cognitive abilities, particularly those related to intelligence and executive functioning (Lahey & Waldman 2003). In terms of the former, studies have consistently found that children and adolescents who exhibit antisocial and violent behaviour exhibit lower intellectual abilities than healthy controls (for reviews, see Henry & Moffitt 1997; Lahey & Waldman 2003; Nigg & Huang-Pollack 2003). Longitudinal evidence suggests that lower intellectual abilities are associated with antisocial and aggressive behaviour even after controlling for co-occurring problems with behavioural dysregulation (Séguin *et al.* 2004; Raine *et al.* 2005). However, recent studies have found that deficits in intellectual abilities may not distinguish children who exhibit chronic delinquent behaviour from those who desist from delinquent behaviour in late adolescence and early adulthood (Raine *et al.* 2005; Loeber *et al.* 2007).

Cognitive impairments related to the executive functions of working memory and response modulation have also been implicated in the development and maintenance of violence (for review see Morgan & Lilienfeld 2000). Longitudinal evidence suggests that difficulties with working memory may be particularly pronounced in childhood-onset physical aggression, and that this association may not be accounted for by low intellectual abilities or problems with behavioural dysregulation (Séguin *et al.* 1999, 2004). Moreover, fMRI evidence suggests that antisocial men with a history of violent behaviour exhibit functional neurobiological differences in the prefrontal regions subserving working memory, in comparison with healthy controls of normal intelligence (Kumari *et al.* 2006). In terms of response modulation, several studies have found that children and adults exhibiting antisocial behaviour tend to perseverate in responding to previously rewarded cues, even after the contingencies change and the response results in a punishment (for review, see Wallace *et al.* 2000). However, recent longitudinal evidence suggests that problems with response modulation may be characteristic of children who exhibit transient aggression, not those who exhibit persistent aggression across time (Séguin *et al.* 2002).

(c) *Deficient responding to aversive stimuli*

Deficits in affectively responding to aversive stimuli have also been implicated as an underlying factor in the development of violence. One influential model in this area suggests that low levels of fearfulness to threatening stimuli may underlie serious and chronic forms of violent behaviour. Along these lines, low levels of fearfulness have been related to chronic childhood behaviour problems (Shaw *et al.* 2003), childhood aggression (Rothbart *et al.* 1994), the onset of delinquent behaviour (Tremblay *et al.* 1994) and the commission of serious violence (Pardini 2006). In addition, physiological (Levenston *et al.* 2000; Raine 2002) and neurological (Birbaumer *et al.* 2005) markers of fearlessness have been associated with severe and persistent forms of antisocial and violent behaviour.

There is some evidence suggesting that fearlessness leads to the development of serious violence by inhibiting the development of guilt and empathy (Pardini 2006). Along these lines, Blair and colleagues have found that evidence indicating persistent forms of psychopathic violence are associated with an inability to effectively identify social distress cues in others, particularly fearful and sad faces (for reviews see Blair 2001 and Blair *et al.* 2006). However, it is important to note that neuroimaging studies have found that severe antisocial behaviour in children and adults is associated with an abnormal neural responsiveness to a wide variety of negatively valenced stimuli (Kiehl *et al.* 2001; Flor *et al.* 2002; Sterzer *et al.* 2005), not just features of distress in others. As a result, the nature of the association between low affective arousal to aversive stimuli and violence is still unclear.

(d) *Controversies regarding underlying factors*

While a rapidly growing body of research has implicated several underlying factors in the development of violent behaviour, several controversies regarding the nature of these associations and the potential implications for understanding the neurobiological underpinnings of violence remain. Some of the key issues that are in need of further study are as follows:

- The specificity of the proposed underlying factors for understanding the development of violent behaviour is unclear. While theoretical models often propose an underlying factor as a driver of violent behaviour, rather than a driver of antisocial behaviour in general, this hypothesis is rarely empirically tested. More importantly, several of the underlying factors described above have been empirically linked to psychopathology other than antisocial behaviour, such as internalizing problems (Guerin *et al.* 1997), substance use disorders (Cloninger *et al.* 1996) and schizophrenia (Morgan & Lilienfeld 2000). However, it is not clear why individuals with the same underlying factor would develop divergent forms of psychopathology.
- Underlying factors are often assumed to index specific aspects of neurobiological functioning, but they are frequently measured using behavioural rating scales. This practice is problematic given that the correlations between behavioural rating scales of underlying factors and aspects of neurobiological functioning are often counterintuitive and differ depending on the rating scale used (e.g. Horn *et al.* 2003).
- Underlying factors are often conceptualized as relatively immutable characteristics, even though emerging evidence suggests that aspects of temperament can be influenced by social factors such as parenting (Rapee 2002). Moreover, it is now apparent that the brain structures believed to subserve several underlying factors continue to mature into early adulthood (Gogtay *et al.* 2004). As a result, greater attention needs to be paid to the dynamic nature of underlying factors as well as the neurobiological factors believed to subserve them.
- Studies need to examine more critically the ability of underlying factors to predict the developmental course of violent and antisocial behaviours, including escalation and desistence. Longitudinal studies frequently do not control for prior levels of antisocial behaviour or environmental factors when looking at the predictive utility of underlying factors. In addition, many studies have not examined which underlying factors are most important for predicting violence after controlling for their co-occurrence.
- Measures of underlying factors sometimes include behaviours consistent with early forms of aggression and conduct problems, especially measures of emotional and behavioural dysregulation. Future studies should make clear empirical and theoretical distinctions between early forms of violent behaviour (e.g. hitting and threatening) and the underlying factors placing youth at risk for developing these behaviours (e.g. irritable mood).
- Researchers should begin breaking complex underlying factors into component pieces in order to better understand their relation with violence (for discussion, see Rothbart 2004). For example, as researchers have dismantled the construct of emotional dysregulation, it has become clear that anger is associated with increased levels of aggression (Rothbart *et al.* 1994), while increased fear seems to be negatively related to violence (Pardini 2006).
- The seemingly contradictory finding that violent behaviour is associated with both emotional dysregulation and low arousal to aversive stimuli needs to be more thoroughly examined. While models have suggested that this apparent paradox indicates the presence of different subgroups of violent individuals (e.g. Frick & Morris 2004; Blair *et al.* 2006; Pardini 2006), developmental research in this area is still limited.
- The possibility that different underlying factors may interact to produce an increased risk for violent behaviour needs to be more thoroughly examined. In support of this practice, Colder *et al.* (2002) found that lower levels of infant fearfulness were associated with increases in externalizing problems across early childhood only for those children with high levels of behavioural dysregulation. Similarly, more studies are needed examining which environmental factors may protect children who exhibit underlying factors from developing violent behaviour over time.

5. CAUSES OF VIOLENCE AND HOMICIDE

As shown in figure 1, we conceptualize that the underlying factors *and* behavioural manifestations of violence are the result of neurobiological, social, individual, economic and environmental causes. There is a voluminous literature on the neurobiological, social and other causes of violence (e.g. Hawkins *et al.* 1998; Lipsey & Derzon 1998). For the following text, we draw from several major reviews on predictors of violence, delinquency and conduct disorder (Lipsey & Derzon 1998; Loeber & Farrington 1998, 2001; Burke *et al.* 2002) and from the PYS. In that study, we found that 51 factors significantly predicted violence in young men (Loeber *et al.* 2005). Figure 4 shows a prediction index for violence constructed on the basis of the 11 strongest predictors (Loeber *et al.* 2005): truancy; low school motivation; onset of delinquency before age 10; cruelty to people; depressed mood; physical aggression; callous–unemotional behaviour; low family socioeconomic status; family on welfare; high parental stress; and bad (i.e. disadvantaged) neighbourhood (parent reported). Figure 4 shows that the higher individuals score on the index the more likely it is that they will commit violence later (odds ratio (OR) = 6.0 for four or more risk factors). Remarkably, the range of probabilities for future violence in the Pittsburgh data is from 3% at 0 risk factors to 100% at 11 or more risk factors.

Another important issue to be determined is whether homicide, as the most extreme form of violence, can be predicted among violent offenders, and whether, in this case as well, there is a dose–response association between the number of risk factors and later homicide. Loeber *et al.* (2005) found the following predictors of homicide among the violent offenders: high-risk score

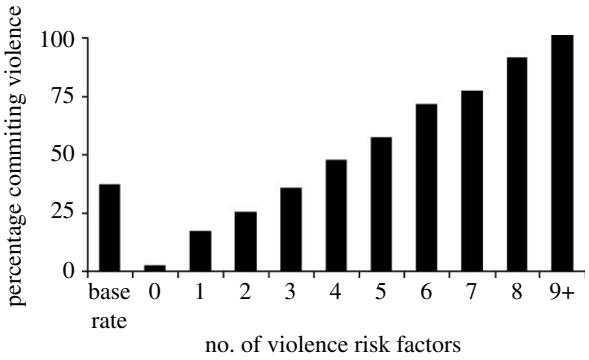

Figure 4. Proportion of boys committing violent offences for different levels of risk in the Pittsburgh Youth Study.

(of disruptive behaviour) at screening; positive attitude to substance use; conduct disorder by age 13; carrying a weapon; gang fighting; selling hard drugs; peer delinquency; repeating grade(s); and family on welfare. The results showed that the higher the number of risk factors, the higher the probability of homicide. The probability of homicide is low for 0 to three risk factors, but after that it almost linearly increases to approximately 15% at six or more risk factors. The OR, based on four or more risk factors, amounted to 14.5. Since genetic factors (and other neurobiological factors measured at a young age) were not available in this study, it remains to be seen to what extent neurobiological factors can contribute to the prediction of violence and homicide (genetic information is likely to be collected for these cohorts in the near future).

(a) *Controversies regarding causation*

The prediction results, and those of several other studies (e.g. Farrington 1997; Hawkins *et al.* 1998; Lipsey & Derzon 1998), indicate a robust association between the number of social and other risk factors and the probability of later violence. However, we want to emphasize the following controversies:

— There is only a single study of the prediction of homicide in a population sample (Loeber *et al.* 2005). Studies still have to demonstrate that neurobiological factors uniquely predict violence if a large range of known social and other factors are taken into account.
— The effect of neurobiological factors could be mediated through various individual difference characteristics represented by one or more underlying factors. For example, neurobiological problems associated with processing of fear stimuli could lead to callous–unemotional behaviour, which in turn is related to violence (Pardini 2006). Thus, future studies need to explain both underlying factors and different manifestations of aggression and violence as these phenomena develop over the life course.
— Further, although there is increasing evidence that gene–environment interactions are important (Taylor & Kim-Cohen 2007), the range of social and other factors that are documented in these interactions is large. For that reason, there is a need to better understand the mechanism(s) by which gene–environment interactions operate.

6. CAUSES OF SECULAR CHANGES IN VIOLENCE AND CAUSES OF COHORT DIFFERENCES IN VIOLENCE

Over the past century, the USA has seen a much higher rate of violence than most European countries, but it is far less known that this does not apply to all forms of violence, and that Europe at one time had a higher homicide rate than the USA. Data from the USA and a few European countries for the years 1980–1999 (Farrington & Jolliffe 2005) clearly show that the rate of homicide and rape is substantially higher in the USA compared with England and Wales, Switzerland and The Netherlands. However, the rate of robbery and burglary in the USA, based on victim surveys, was similar compared with several European countries, especially since the 1990s.

The crime rates between 1981 and 1999 have varied a great deal. Major increases and decreases in crime have taken place, such as decreases in homicide, rape, robbery and burglary in the USA, increases in burglary and rape in England and Wales, and increase in robbery in The Netherlands. Over a longer period of observation, the rate of homicide in Europe after 1200 was much higher than that in the USA in the last century, but the European rate has decreased dramatically since the early middle ages. The current rate of homicide in the USA is similar to the rate that was common in Europe around the year 1700 (Eisner 2004). The bottom line is that there are secular changes in violence rates of countries and cities, and that some of these secular changes have been large over time. The fact, however, is that secular changes are not necessarily taking place across countries at the same time. For these and other reasons, it is important to address the causes of secular changes in violence.

There is a voluminous literature with numerous hypotheses about the causes of secular changes in crime and violence (e.g. Blumstein & Wallman 2000). We propose that secular changes in violence pose a unique challenge to studies on the neurobiology of violence. Are there neurobiological factors that either solely or in conjunction with social/individual/economic/environmental factors influence secular changes in violence, and if so, by what mechanism(s)? As far as we know, there are no studies that have proven that one or more neurobiological factors can sufficiently explain secular changes in violence. Part of the problem of identifying any cause, neurobiological or otherwise, of secular changes in violence is the ecological fallacy (the assumption that all members of a group exhibit characteristics seen in aggregate statistics collected on the group at large). Another problem is that the causes of changes in the prevalence of violence in populations are not necessarily directly linked to changes in individuals' propensity to violence. We argue that the latter is an essential step, especially because neurobiological factors are thought to reside in individuals rather than in populations.

The yearly community rate of violence in a population over time actually is the sum of the violent offending rates of individuals of different ages, represented by an aggregation of successive age–crime curves of different age cohorts. Thus, over a period of, say, 1990–2000, the violence of some individuals may be

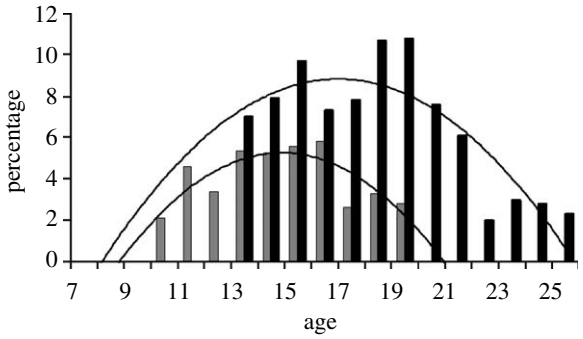

Figure 5. Different age–crime curves for violence in the youngest and oldest samples of the Pittsburgh Youth Study.

represented in 1995 and 1996, while a younger generation's violence is represented in 1997 and 1998. The years when their violence is most represented in the graphs are their peak years of violence (in the age–crime curve, about ages 15–22), which then show up markedly in population graphs of violence. Our approach to better address the origins of secular changes is to decompose secular changes in violence rate of populations into a series of age–crime curves for successive age cohorts of individuals whose violence contributes to secular levels of crime in the whole population.

Opening this avenue of inquiry has the advantage of making use of longitudinal studies with multiple cohorts. We argue that such studies allow us to identify the causes of violence for one cohort and compare these with the causes of violence for another cohort. This approach is particularly valuable when there are large cohort differences in violence. Figure 5 illustrates this point with the youngest and oldest cohorts in the PYS (who were on average 6 years apart). The oldest cohort compared with the youngest one had a substantially different age–crime curve: the curve was higher and appeared to have a larger base for the oldest cohort compared with the youngest one, suggesting that desistance processes in the downslope of the age–crime curve in the oldest cohort took place approximately 5 years later than in the youngest cohort. Even during the period in which the two cohorts overlapped (ages 13–19), there were major differences in violence-related outcomes, including gang membership and gun carrying. For example, gang membership decreased from a peak of 6% at age 15 to less than 2% at age 19 in the youngest cohort, but increased from 6% at age 13 to 9% at age 19 in the oldest cohort. In addition, gun carrying decreased from a peak of 8% at age 16 to 5% at age 19 in the youngest cohort, while gun carrying increased in the oldest cohort from 2% at age 13 to 17% at age 19. Two lessons can be learned from these results: (i) even with age cohorts just six years apart, there can be major changes in the age–crime curve and (ii) differences between cohorts in violence were accompanied by activities, gang membership and gun carrying, which are directly fuelling violence. Thus, if successive age cohorts display unusually high levels of violence, this will create a sequence of age–crime curves that are higher and broader than typical age–crime curves. In aggregate, these successive high age–crime curves will translate in higher rates of violence in communities where the cohorts reside and/or are active, thus contributing to secular changes in violence over time.

We translate this line of thinking in a shift from the question what the causes are of secular changes in violence to the question what the causes are of cohort differences in violence (see also Jacobson *et al.* 2000). The big advantage of this approach is that it brings us closer to the investigation of causes for individuals that in aggregate can represent the causes of violence in populations. For example, Fabio *et al.* (2006) examined predictors of the differences between the two cohorts in the PYS for self-reported violence and found that cohort differences no longer contributed to the regression equation once individual factors (gun carrying, gang membership, drug dealing and hard drug use), race, family socioeconomic status and period effects had been taken into account (period effects concern factors that are manifest during a specific period that affect all cohorts).

(a) *Controversies regarding secular changes in violence*

— It remains to be seen which, if any, of the individual factors that predict secular differences in violence are related to one or more neurobiological factors.
— It is also unclear to what extent mixtures of neurobiological and non-biological factors differ for boys and girls in the explanation of cohort differences and, down the line, secular differences in violence.

7. CONCLUSIONS

We started out by specifying several common assumptions in neurobiological studies and we pointed to four controversial areas that are relevant to the investigation of neurobiological factors and the development of violence. The majority of investigations of neurobiological factors have been cross sectional (or retrospective). As a consequence, conceptualizations about the dependent variable of aggression/violence in neurobiological studies have been mostly static rather than dynamic and have not reflected individuals' developmental changes in aggression and violence during the life course as evident from longitudinal studies. Although we agree that it is useful to search for neurobiological, social and other factors to explain stable individual differences in aggression and violence, longitudinal investigations on the neurobiology of violence need to address key questions about the change as well. Examples of such questions are: why do most children outgrow aggression in the first years of life? Why do some violent youth desist in violence? Why do some youth but not others progress along a pathway from minor forms of aggression to serious violence and homicide? Why do some individuals have a late onset of violence? Why are there categories of individuals with different trajectories of violence during childhood and adolescence? Answers to all of these questions can enrich the search for neurobiological underpinnings of violence, deepen theoretical explanations and, eventually, improve prevention and intervention problems targeting violence.

We also made a case that most conceptualizations of neurobiological, social and other causes interposed underlying factors as necessary links between neurobiological factors and aggression/violence. We illustrated the large variety of underlying factors that have been proposed, and the comparative lack of information about which of the underlying factors is most valid and can be measured most reliably. Although many scholars agree about the need to link neurobiological factors to underlying factors, they tend to disagree about which underlying factors are most valid and reliable and do not pay sufficient attention to possible relationships between underlying factors. Neurobiological studies often do not recognize that underlying factors may change and are possibly key elements of the explanation of the aggression–age and the crime–age curves. Finding out which neurobiological factors (in conjunction with social and other factors) influence changes in underlying factors appears to be a task of the highest priority.

We illustrated that although the predictive power of social, individual, economic and environmental factors in the prediction of violence and homicide is considerable, we do not sufficiently know what the additional explanatory power is of neurobiological factors. Even though progress has been made in the study of gene–environment interactions (Moffitt 2005; Taylor & Kim-Cohen 2007), these findings should be interpreted with caution. Environmental risk variables tend to occur in concert rather than singly. For instance, Koot *et al.* (in press) stressed that most findings on gene–environment interactions for a wide range of antisocial outcomes have been based on childhood maltreatment as an environmental risk factor. However, maltreatment may serve as an index of adverse familial conditions. Taylor & Kim-Cohen's (2007) meta-analysis showed that the *interaction* between genetic and environmental factors results in a risk that is far higher than the sum of the individual risk factors. However, a close examination of the results shows little agreement among the studies about the observed interaction mechanisms.

Finally, we showed that the explanation of secular changes in violence poses yet another series of challenges to neurobiological studies. If secular changes occur within years, it will be difficult to test which neurobiological factors are elements in the explanation of secular change. We argue that longitudinal studies with multiple cohorts are essential in this quest, because cohort differences in violence are a building block for secular changes in population violence.

Our experience lies in the developmental aspects of aggression and violence and to some extent in the study of neurobiological causes (e.g. see the work of our collaborators, such as Eaves *et al.* (1997), McBurnett *et al.* (1997) and Raine *et al.* (2005)). We hope, however, that researchers in the neurobiology of violence can see the merits of the avenues for investigation that we proposed and that this eventually will lead to improved knowledge about the causes of violence.

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ENDNOTE

[1]The Pittsburgh Youth Study began in 1986 comprising boys who were enrolled in the public schools in Pittsburgh. The sample is about evenly distributed between African American and Caucasian boys. The young males have been regularly followed up over a period of 13 years. The study consists of three age cohorts of boys who were in first, fourth or seventh grades of public schools at the time of the first assessment in 1987–1988 (called the youngest, middle and oldest cohorts). The participation rate of boys and their parents was approximately 85% of the eligible boys. On the basis of screening at the first assessment, antisocial boys were oversampled, but the final sample consisted of an additional sample of randomly selected non-deviant boys. The youngest cohort has been assessed 18 times between ages 7 and 19, while the oldest cohort has been assessed 16 times between ages 13 and 25. By contrast, the middle cohort was discontinued after seven assessments and had a single follow-up assessment at about age 24. One of the strengths of the PYS is the availability of multiple informants (including parents and teachers) to enhance the validity of measurements; in addition, official records of delinquency were collected. Further details about the study are available elsewhere (Loeber *et al.* 1998, 2008).

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