8 The Neuropsychology of Violence

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Introduction

Neuropsychology has long sought to assess the often-subtle, yet dramatic, effects of brain lesions on information processing and behavior. Following certain brain lesions, a previously well-adapted individual can become irritable, impulsive, incapable of sustaining concentration, and neglectful of social rules. In such cases, the individual's ability to process information in a socially adaptive way becomes severely impaired. The observation of these profound changes prompted the development of neuropsychological accounts for the deficits, seen in a broad range of behavior problems, including violent behavior.

Here, we update our original review of the neuropsychological approach to violent behavior published in an earlier edition of the handbook (Séguin, Sylvers, & Lilienfeld, 2007). In Section I we examine issues pertaining to the assessment of neuropsychological function and the assessment of clinical syndromes, including delinquency and criminality, associated with violence. In Section II, we review developmental issues affecting brain maturation and behavioral regulation. We integrate the aforementioned issues in Section III, where we review studies that help us understand violence from a neuropsychological perspective. As we note, because a key method in neuropsychology has been the use of lesion analyses, we examine the extent to which brain lesions contribute to violence. We then turn to the few neuropsychological studies of violence, and examine the larger body of literature on clinical syndromes associated with violence. There, we examine the extent to which neuropsychological problems have been identified in violence-prone individuals. Finally, we integrate the key observations derived from this review, address limitations in the extant research, and offer suggestions for further research on this important and still-growing area.

Section I: Assessment

Classic neuropsychological testing involves the administration of a battery of tests. These tests are designed to assess a variety of brain functions, ranging from basic perception to more complex neocortical problem-solving, and require either verbal or motor responses. The stimuli may be visual or auditory. Visual stimuli include pictures, abstract designs,

1 We would like to thank Patrick Sylvers, who co-authored the previous installment of this chapter with J. R. S and S. O. L and whose input at the time served to flesh out the current structure of the chapter.

and combinations of these stimuli, such as those found in various puzzles, mazes, assortments of objects, pictorial depictions of story lines, printed colors, words, and numbers. Auditory stimuli may involve spoken words, numbers, problems, or stories. Computerized batteries are usually limited to motor responses performed through the click of a mouse or by means of a touch-screen interface, although voice onset recording, eye tracking devices, electrophysiology, and functional brain imaging are increasingly used. Many of these tests qualify as neuropsychological because they were developed to test theories of brain function and were typically validated with lesion analysis studies, brain electrophysiological studies, or functional brain imaging studies. In other words, individuals with relatively well-circumscribed brain lesions performed poorly on such tests, or these tests were found to engage specific brain regions. Thus, these batteries provide a profile of strengths and weaknesses that presumably vary as a function of location and extent of lesions. Findings from these assessments can further help tailor intervention or prevention (Séguin & Pilon, 2013).

Neuropsychological lesions can result from pregnancy or birth complications, various illnesses, aging, head injury, intracranial tumors, cerebrovascular disorders, exposure to toxic substances, or corrective surgical procedures. The extent to which these events lead to neuropsychological lesions may in turn depend on a variety of moderators, such as the developmental timing of lesions, genetic makeup, and socioeconomic factors. They can also be temporary and reversible, such as those observed under the acute effects of drugs and alcohol or of certain illnesses. Finally, the results of neuropsychological tests are often assumed to reflect the competence of the individual. Nevertheless, there may be a sizable gap between competence and performance. Thus, interpretation of test results should take into account the individual's test motivation, affect (e.g., mood, anxiety), attention, capacity to remember the sometimes complex rules required for optimal performance, language of administration, and cultural background. We refer the reader to more specialized sources for additional information (Goldstein & McNeil, 2012; Lezak, Howieson, Bigler, & Tranel, 2012).

Finally, and to build on the important role of motivation, the more explicit integration of cognitive and affective neuroscience in the past two decades has contributed to more systematic consideration of both "cool" and rational versus "hot" and affective neuropsychological assessment (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006). Whereas "cool" tasks are designed to be more emotionally neutral, tasks can also vary in the degree to which they are administered in an emotionally arousing and "hot" context (Séguin, Arseneault, & Tremblay, 2007). Whereas the former tasks involve more "top-down" integrative regulation (e.g., dorsolateral frontal cortex), the latter tasks involve more "bottom-up" processing (e.g., ventromedial/orbital frontal cortex), such as those more generally described by Stuss (2011). Although the "cool – hot" distinction has considerable face validity and seems to be taken for granted by many, it is much harder to define operationally as it may not be as categorical as the language we use to describe it implies (Welsh & Peterson, 2014).

Clinical Syndromes Associated with Violence

Although physical violence is relatively easy to identify because of its overt nature, there is a paucity of studies examining the neuropsychology of violence. Much of what we know in this domain derives from research on conditions that are associated with violence rather than violence *per se*. Thus, to appreciate the strengths and limitations of this body of literature, we first discuss clinical syndromes most commonly associated with violence.

Violence research is conducted within two broad and overlapping nomenclatures: legal/judicial and clinical. In the legal/judicial areas, researchers have studied delinquency and criminal behavior. In the clinical arena, physical violence or aggression as a feature, and sometimes as a diagnostic criterion, can be found in a variety of disorders of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) (American Psychiatric Association, 2013) and *the International Classification of Diseases – Mental and Behavioral Disorders* (ICD-10CM) (World Health Organization [WHO], 1992). Accordingly, an approach linking neuropsychological function to violence as manifested in clinical disorders fits well within the objectives of the Research Domain Criteria project recently launched by the National Institute of Mental Health (www.nimh.nih.gov/research-priorities/rdoc/index.shtml). Specifically, physical aggression or violence may be found among disorders that are listed under disruptive, impulse-control, neurodevelopmental, and neurocognitive disorders, including Intermittent Explosive Disorder, other Specified Disruptive Impulse-Control and Conduct Disorder, Unspecified Disruptive Impulse-Control and Conduct Disorder, and Antisocial Personality Disorder (ASPD). Physical violence can also occur as a symptom of the Personality Change due to a General Medical Condition, Aggressive Type; Trauma or Stressor-Related Disorders, and in the newly added DSM-5 Disruptive Mood Dysregulation disorder. It is not a formal feature of, but is a potential consequence of, Delusional Disorder – persecutory type, Schizophrenia, Sleep-Wake Disorders, Bipolar Disorder, several Substance-Related and Addictive Disorders, and relational problems including Spouse or Partner Violence. Nevertheless, for none of these disorders is violence a necessary or sufficient criterion.

Psychopathy has not been listed as a separate disease category in DSM-5 or its predecessors. Nevertheless, although DSM-IV-TR regarded ASPD as essentially synonymous with psychopathy, in DSM-5, psychopathy has been added as a specifier to the ASPD category in Section III (but not to the main text) – the section of the manual devoted to newly emerging models and measures. Psychopathic traits are also listed in the Conduct Disorder (CD) category using the specifier "with limited prosocial emotions," which captures children and adolescents with pronounced callous-unemotional (CU) traits. Indeed, research has shown that children with CD and marked CU traits are distinct from children with CD without these traits in etiology, risk for ASPD later in life (Frick, Ray, Thornton, & Kahn, 2014), and neurocognitive functioning (Blair, Leibenluft, & Pine, 2014).

Physical violence has also been studied developmentally. However, in most developmental studies, aggression scales often neglect to distinguish physical from other forms of aggression. For example, the Child Behavior Checklist (CBCL,

Achenbach, Edelbrock, & Howell, 1987) yields an aggression scale that comprises 23 items, only three of which refer explicitly to physical aggression. Physical aggression in these scales can be defined as hitting, kicking, biting, use of a weapon, and getting into fights. In some cases, this definition is broadened to include bullying and threats of violence.

Given these limitations, it is possible for neuropsychological studies in the antisocial behavior literature to include nonphysically violent forms of CD, ASPD, psychopathy, or aggression. Further, the clinical syndromes in which physical violence is present are often comorbid with other conditions characterized by impulsivity, drug and alcohol abuse, Attention Deficit Hyperactivity Disorder (ADHD), and Oppositional Defiant Disorder (ODD). Although there is merit to examining how components shared across such externalizing problems (if not across all disorders) relate to neuropsychological function (Castellanos-Ryan et al., 2016), such an approach is also complemented by studies of specificity.

Section II: Developmental Issues

A developmental approach allows the identification of children for whom problems may be chronic as opposed to acute or transient. Accordingly, longitudinal studies in community samples have provided insight into trajectories of antisocial behaviors throughout development. A recent review of this literature indicates that antisocial behaviors tend to display different developmental trajectories, that they have shared but also unique risk factors, that the trajectories associated with violence generally emerge in the preschool years, and, although some trajectories remain high for a small proportion of children as they enter adulthood, antisocial behaviors tend to decline across adulthood – but are nonetheless accompanied by adaptational problems later in life (Séguin & Tremblay, 2013). The bulk of that research has been on males; so much more needs to be done to study females.

On the basis of early prospective longitudinal studies, Moffitt (1993) noted that adolescents with a form of early-onset/persistent antisocial behavior, which includes aggression and hyperactivity, showed poorer neuropsychological test performance in early adolescence than did those with a later onset. This finding has been supported by several studies (e.g., Johnson, Kemp, Heard, Lennings, & Hickie, 2015; Fairchild et al., 2009), although brain-imaging studies have not fully supported this distinction (e.g., Fairchild et al., 2011; Jiang et al., 2015). These findings underscore a key methodological point, namely, that comparing groups of adolescents or adults without knowledge of their natural developmental history can be fraught with problems. Nevertheless, the developmental sequence is often assumed to be from neuropsychological function to behavior but little research has been conducted to verify that assumption. Hence, developmental studies need to use a more fully longitudinal approach that permits the examination of reciprocal transactions between neuropsychological function and behavior (Pinsonneault, Parent, Castellanos-Ryan, & Séguin, 2015).

Risk Factors that May Affect Brain Development

As developmental patterns have been studied with longitudinal designs, there

has been increasing interest in conditions that can contribute both to those patterns *and* to poor neuropsychological function, such as a history of exposure during or after pregnancy to brain-altering psychopharmacological agents (e.g., cigarette smoke, alcohol, drugs), perinatal or birth complications, nutrition, traumatic experiences (e.g., abuse), chronic stress, or behavior problems that heighten the risk of head trauma through accidents or fights (e.g., ADHD, ODD). For example, cigarettes may exert early effects in the intrauterine environment and later in the home environment. For example, a review of 20 published articles on the association between tobacco smoke exposure in utero and cognition found robust associations for lower academic achievement and intellectual functioning after controlling for other variables (Clifford, Lang, & Chen, 2012). Nevertheless, genetically informed designs raise the possibility that at least some of this linkage may be genetic (e.g., D'Onofrio, Van Hulle, Goodnight, Rathouz, & Lahey, 2012).

The mechanisms through which in-utero or early-life adverse exposures affect brain development and violence risk are unclear, but an emerging overarching hypothesis is that early-life adversity produces long-lasting epigenetic alterations that influence gene expression profiles in key biological systems, such as the hypothalamic–pituitary–adrenal axis, the immune system, as well as neurotransmitters, such as serotonin (Booij et al., 2010; Wang et al., 2012), which, in turn, alter the ability to learn to inhibit physical aggression. Genetic factors probably moderate these effects of early-life stress on the epigenome and influence the risk for, or resilience to, developing violent behaviors (Provençal, Booij, & Tremblay, 2015). Furthermore, there could be an assumption that neurocognitive problems lead to behavior problems. This would not be surprising given that this literature developed out of lesion research and that remedial interventions often target cognitive abilities to change behavior. However, this assumption is rarely tested as it is equally possible for transactions to change direction across developmental stages. Consequently, children who show behavior problems could eventually get more or less support and exposure to alternative problem-solving approaches through social moderators, which would enhance or diminish their repertoire of cognitive and self-regulatory skills (see Pinsonneault et al., 2015). Finally, substance use during adolescence, such as smoking (Lydon, Wilson, Child, & Geier, 2014) or cannabis use (Castellanos-Ryan et al., 2017), probably also affects the developmental course of brain cognitive and motivational systems.

Section III: The Effects of Brain Lesions on the Risk for Violence

Given the background considerations reviewed thus far, we now address two complementary questions: (1) "Do some brain lesions dependably increase the risk for violent behavior?" and (2) "Do violence-prone individuals exhibit specific neuropsychological deficits?"

Interest in the cognitive and emotional regulation aspects of the brain-violence relation has centered largely on the role of the frontal lobe because of its centrality to the regulation of social behavior. Frontal patients have difficulty organizing and regulating their behavior in response to external stimuli. The more cognitive functions of the frontal lobe, often

referred to as the executive functions, include working memory, which involves the online maintenance of information and the active processing of that information as an individual engages in action, interference control (attention and inhibition), and cognitive flexibility (Diamond, 2013). The emotional regulation function was supported by a study of individuals with lesions comprising the orbitofrontal cortex, who tend to be disinhibited, socially inappropriate, susceptible to misinterpreting others' moods, impulsive, unconcerned with the consequences of their actions, irresponsible in everyday life, lacking in insight into the seriousness of their condition, and prone to weak initiative (Fazel, Philipson, Gardiner, Merritt, & Grann, 2009). The main consequence of such lesions has been linked with impaired performance on a wide variety of neuropsychological tests measuring reward and punishment sensitivity, decision making, and processing of social cues (Jonker, Jonker, Scheltens, & Scherder, 2015). In many imaging studies, differences in brain function or structure were independent of performance deficits, and therefore require additional neuropsychological testing to be evaluated. Many of these neuropsychological deficits reflect malfunction of limbic brain regions, such as the amygdala, cingulate, and/or striatum.

The ensuing syndrome from orbitofrontal lesions was historically labeled acquired sociopathy (Damasio, 2000), acquired ASPD (Meyers, Berman, Scheibel, & Hayman, 1992), or pseudopsychopathy. Because these lesions appear to lead to certain psychopathic-like behaviors, they were often thought to underlie physical violence. Nevertheless, there is limited support for this hypothesis. Studies of war veterans, for example, revealed that although veterans with prefrontal lesions exhibit more positive implicit attitudes towards violence compared with veterans without lesions, they do not differ on explicit measures of aggression. Thus, although prefrontal lesions may impact inhibition over automatic (implicit) aggressive reactions to perceived provocation (Cristofori et al., 2016), physical violence directly resulting from lesions is rare in acquired forms of antisocial behavior. This finding may in part be due to the fact that individuals with acquired brain lesions do not necessarily possess the many pre-morbid risk factors associated with physical violence. In fact, aggression (including physical aggression) appears to be more likely to be associated with acquired frontal lesions only when there is a premorbid history of psychopathology (Fazel et al., 2009). Otherwise, normative behavior may be the result of interactions among these dynamically related frontal systems (Verbruggen, 2016).

Other cortical abnormalities associated with aggressive outbursts include acute episodes of temporal lobe (temporolimbic) epilepsy (also known as interictal violence), which is similar in manifestation to IED. Nevertheless, violence in epilepsy seems to be lower than in the general population (Fazel et al., 2009). Taken together, these findings suggest that brain lesions are rarely sufficient causes of violence. At the same time, they may lead to violence in the context of other risk factors.

Neuropsychological Studies of Physically Violent Behavior

The other main approach to the neuropsychology of violence is to study physically violent individuals. We begin with the most severe form of violence, murder, and

move on to less severe forms of physical aggression. We then follow with disorders and antisocial behavior problems in which physical violence is sometimes present.

Murderers

Several studies of murderers have shown reduced activity in frontal brain regions, which is consistent with the dysfunctional frontal lobe hypothesis, a theory postulated for the first time more than two decades ago during the early years of brain imaging research. In one of the first studies, "frontal dysfunctions" were found in 65% of murderers (Blake, Pincus, & Buckner, 1995). In a more recent study, among incarcerated youth, homicide offenders (80% self-reported) showed structural differences in the temporal lobes, hippocampus, and posterior insula (Cope et al., 2014). Further, when these homicide offenders were compared to typically developing youth in a recent meta-analysis, they appeared to show consistently larger effect sizes across several structures (Rogers & De Brito, 2016).

Few studies have examined neuropsychological performance in murderers. The neuropsychological performance in murderers seems to depend on subtype; affective/impulsive murderers tend to display poorer neuropsychological performance than predatory/instrumental murderers, especially on measures of intelligence, memory, attention, and executive functions (Hanlon, Brook, Stratton, Jensen, & Rubin, 2013). These differences appear to be consistent with differences in brain activation (Raine et al., 1998).

Although the frontal lobe has been implicated in these and other studies, they almost surely account for only one portion of the explanation. Indeed, in addition to frontal hypoactivation, many imaging studies found increases in the activity of subcortical areas, which is consistent with neural models of aggression, postulating that aggression results from deficits in cortical top-down control and/or facilitation of bottom-up signaling triggered from subcortical limbic circuits (Davidson, Putnam, & Larson, 2000). Importantly, frontal lobe hypofunction is not specific to murderers (e.g., Siever, 2008). The lack of specificity indicates that other factors like genetic make-up, early development, and other factors reviewed in this book may play an important role in the risk for committing homicide.

Physical Aggression

Although murder is the most severe form of violence, investigators have also examined whether neuropsychological problems are evident in milder forms of violence, such as in physical aggression as observed in community samples. There are surprisingly few neuropsychological studies of physical aggression *per se*.

Séguin et al. (1995) sought to develop a neuropsychological test battery on the basis of (a) the frontal lobe and memory work of Petrides and Milner (1985) and (b) reviews of the neuropsychology of delinquency and conduct disorder, such as Moffitt's (1990), which identified deficits in language abilities, executive function, and cerebral dominance. Using that approach, we first found that working memory, a basic ability involved in executive function, was poorest in boys from a community sample with a history of physical aggression even after controlling for nonexecutive abilities (Séguin, Pihl, Harden, Tremblay, & Boulerice, 1995). In follow-up, we controlled statistically

for ADHD or teacher-rated hyperactivity and still found working memory impairments even after controlling for IQ (Séguin, Boulerice, Harden, Tremblay, & Pihl, 1999). In a third study, we selected young adult males not only on the basis of a physically aggressive history (teacher-rated from kindergarten to age 15), but also of hyperactivity (without a focus on inattention or impulsivity) (Séguin, Nagin, Assaad, & Tremblay, 2004). We found no statistical interaction, but we did find clear additive effects, even after controlling for test motivation. In other words, both physical aggression and hyperactivity exhibited significant independent and additive associations with neuropsychological function.

In sum, poor neuropsychological function is often found in adolescents and young adults from the community with a history of physical aggression. These deficits appear to be independent from other externalizing behavior problems. Developmentally, these associations and specificity to physical aggression or hyperactivity can be detected as early as the preschool years (Séguin, Parent, Tremblay, & Zelazo, 2009).

Neuropsychological Studies of Antisocial Disorders in Which Physical Violence May Be Present

The bulk of research on the neuropsychology of violence derives from clinically oriented studies of disorders and antisocial behavior problems that may include physical violence.

Antisocial Behaviors

Several of the behavioral problems listed here have been grouped under the global label of "antisocial behaviors." These behaviors, among others, were examined in meta-analyses of studies of executive function by Morgan and Lilienfeld (2000) and more recently updated and expanded by Ogilvie et al. (2011). The first meta-analysis by Morgan and Lilienfeld (2000) comprised 39 studies, yielding 4,589 participants total. To be included in the meta-analysis, tests of executive functioning must have attempted to measure volition, planning, purposive action, or effective performance, and either differentiated patients with frontal lesions from other patients or preferentially activated the frontal cortex in previous studies. To investigate whether antisocial behavior was related to executive functioning deficits *per se* rather than neuropsychological deficits in general, three neuropsychological tests that do not rely heavily on executive functioning were analyzed as "control" measures. The antisocial behavior groups used in the meta-analysis included individuals meeting criteria for one or more of the following antisocial behavior problems: ASPD, CD, psychopathic personality disorder, criminality, or delinquency.

The results indicated that the antisocial behavior groups performed significantly worse than comparison groups, with a combined and weighted effect size (Cohen's *d* [Cohen, 1992] of 0.62 standard deviations). Two of the three non-executive functioning neuropsychological tests also produced significant, albeit weaker, differences between groups, with the antisocial behavior groups performing significantly worse with effect sizes of $d = 0.34$ and $d = 0.39$ standard deviations. However, tests revealed significant heterogeneity of effect sizes, pointing to the possibility of moderators. When considering

the type of antisocial grouping used in the studies, the heterogeneity of effect sizes was reduced within each group and all group effect sizes remained significantly different than zero. Moreover, criminality (d = 1.09, weighted d = 0.94) and delinquency (d = 0.86, weighted d = 0.78) were most strongly associated with executive functioning deficits. Potential moderators including age, sex, ethnicity, and IQ were not associated with the magnitude of the observed effect sizes, although scores on some of these moderators (e.g., sex, ethnicity) were not reported in all studies.

The second meta-analysis by Ogilvie, Stewart, Chan, and Shum (2011), built on the Morgan and Lilienfeld (2000) meta-analysis, brought up the total number of studies to 126, involving 14,786 participants. This meta-analysis added more recent studies published since the original meta-analysis as well as a wider range of EF and non-EF tasks. It corroborated the results of the initial meta-analysis by showing an overall association between executive functioning and antisocial behaviors (d = 0.44), but also heterogeneity of effect sizes across definitions of antisocial behavior. This heterogeneity may in part have stemmed from the fact that studies used to estimate effect size could be considered under more than one antisocial behavior category. Nonetheless, and as in the initial meta-analysis, the largest effect size was found for criminality (d = 0.61). Other reported effect sizes were ODD/CD (d = 0.54), psychopathy (d = 0.42), delinquency (d = 0.41), physical aggression (d = 0.41), and ASPD (d = 0.19).

Although the effect sizes across meta-analyses were mostly similar, Ogilvie et al. (2011) suggested that some of the differences may have been due to the inclusion of a wider range of EF and non-EF tasks as well as measures and operationalization of antisocial behaviors that have become more specific and sensitive over the years. Some of our work can serve to illustrate this issue. For example, when forming groups on the basis of a history of physical aggression, the effect sizes of neuropsychological tests in a study using developmental trajectory methodology (empirically based) as was used in a later study (Séguin et al., 2004) were much larger than those found when using arbitrary thresholds (theoretically based) in an earlier study (Séguin et al., 1995).

By the time of the later meta-analysis (Ogilvie et al., 2011), there was then also a sufficient number of studies to examine the potential additive role of ADHD. The studies that included participants with comorbid ADHD diagnoses showed the largest effect sizes. The mechanisms through which ADHD adds these other antisocial behavior problems in their association with neurocognitive functioning remain to be better studied (see also hypotheses proposed by Sonuga-Barke, Cortese, Fairchild, & Stringaris, 2016). This finding supports the need for careful selection of study participants in future studies when studying specific relationships between executive functioning and antisocial behaviors.

Both meta-analyses identified several limitations in this body of research, including small sample sizes, poor comparison group selection, and minimal control over potentially confounding factors such as psychiatric comorbidity. Furthermore, Morgan and Lilienfeld (2000) pointed out that the finding that criminality and delinquency were associated with more pronounced executive functioning deficits than the other groups

is difficult to interpret given the differences in comparison groups used across studies. For example, executive functioning studies frequently compared criminals or delinquents with normal or unselected samples, whereas psychopaths were frequently compared with nonpsychopathic criminals. These concerns were supported empirically (Ogilvie et al., 2011).

Psychopathy

Psychopaths are generally more violent than other criminals (Hare, 1999) and consequently have received considerable research attention in the past three decades. Reports of neuropsychological performance among psychopathic participants, especially executive functioning, have been mixed. The two meta-analyses examining PCL-R and non-PCL-R-defined psychopathy indicate that the average effect sizes of neuropsychological (executive functioning) deficits were small to medium (d = 0.29 in Morgan and Lilienfeld [2000] and d = 0.42 in Ogilvie et al. [2011]).

Possible explanations for the high levels of variability across studies are differences in the operationalization of psychopathy as well as the existence of potential psychopathy subtypes. For instance, a number of studies have shown that low-anxious psychopaths show impaired functioning on neuropsychological tasks that rely primarily on frontal lobe function, such as executive functioning (Smith, Arnett, & Newman, 1992), cued attention (Zeier, Maxwell, & Newman, 2009), as well as economic decision making (Koenigs, Kruepke, & Newman, 2010). In addition, successful psychopaths (defined by being nonconvicted) showed better performance on an executive functioning task than unsuccessful (convicted) psychopaths (Ishikawa, Raine, Lencz, Bihrle, & LaCasse, 2001). It is unclear, however, whether this lattermost difference is independent of possible between-group differences in general intelligence.

Beyond studies using more classic neuropsychological tests, psychopaths appear to experience greater difficulty in shifting a dominant behavior when contingencies are changed and reversed. Newman and colleagues have proposed two pathways to account for that impairment: (1) a difficulty in regulation of affect and (2) a more fundamental information-processing deficit related to attention, i.e., a difficulty in shifting attention to peripheral but potentially meaningful information from the environment (Newman & Lorenz, 2003). This so-called response modulation theory is one of the most widely studied cognitive models of psychopathy. After its initial formulation more than three decades ago (Gorenstein & Newman, 1980), the theory has been refined over the years, taking into account new research. A recent meta-analysis (Smith & Lilienfeld, 2015), including 94 studies involving 7340 participants, found a small to medium effect size (d = 0.41) for the association between response modulation deficits and psychopathy, which is within the same range as those found with other psychological theories of psychopathy (Lilienfeld, Smith, & Watts, 2016). Nevertheless, the several clinical, demographic and methodological variables (e.g., anxiety levels, ethnicity, measures used) that were found to moderate the strength of the relationship, as well as the several methodological shortcomings of studies (e.g. publication bias), raise questions regarding the comprehensiveness of the theory in explaining psychopathy.

A number of other cognitive theories preceded and followed the response modulation theory. Recently, the impaired integration theory has been proposed to integrate cognitive and affective models of psychopathy with neurobiological data (Hamilton, Racer, & Newman, 2015). This theory postulates that psychopathy results from an impaired ability to rapidly integrate sensory information into a unified percept. This deficit in turn leads to unelaborated mental representations and an underdevelopment of associative neural networks. Underdeveloped connectivity in specific brain networks ostensibly underlies the psychopathy profile. For instance, decreased connectivity in networks involved in affective processing underlie the callous emotional traits present in psychopathy, whereas decreased connectivity in attentional networks leads to poor integration of different types of information (e.g., cognitive-affective, perceptual-motor). Although more research is needed to support this promising theory, such an integrative approach dovetails with developments in cognitive neuroscience, modeling behavioral and cognitive processes as dynamic large-scale neural networks, rather than as isolated, static brain regions.

Criminality and Delinquency

Some of the important theoretical work of Moffitt (1993) initially centered on delinquency. In one study, early-onset/persistent delinquents performed more poorly than did "late-onset" delinquents on IQ and other neuropsychological tests (Taylor, Iacono, & McGue, 2000), as predicted by Moffitt's (1993) developmental theory of antisocial behavior. In this study, however, IQ was not used as a covariate, rendering conclusions regarding the specificity of cognitive deficits unclear. Furthermore, the use of global scales of delinquency or criminality may obscure key relations between specific behaviors and neuropsychological function. For example, one study revealed that, among juvenile delinquents, IQ was positively related to theft, but negatively related to violence (Walsh, 1987). Presumably, this finding reflects the requirement for planning for theft, but an impulsive problem-solving style for violence. Intrigued by this isolated report, we analyzed data from the Rutgers longitudinal study (White, Bates, & Buyske, 2001). In that study, theft and violence were initially combined within a global index of delinquency. Neuropsychological function had failed to separate persistent from adolescence-limited delinquents. Following our own work with physical aggression and hyperactivity (Séguin et al., 2004), we identified trajectories for theft and for physical violence. Using the same tests as used by White et al. (2001), we replicated Walsh's (1987) finding and found the poorest neuropsychological function in highly violent individuals who were low on theft (Barker et al., 2007). In another follow-up to the Séguin et al. (2004) study, we found that verbal abilities were negatively related to trajectories of physical aggression but positively associated with theft (Barker et al., 2011). In that study, lower levels of inductive reasoning were associated with increases in theft across adolescence. Interestingly, symptoms of ADHD accounted for part of the neurocognitive test links with physical aggression, but did not account for the associations with theft. Together, these studies highlight the need for a finer parsing of the relation

between neuropsychological function and global indices of antisocial behavior.

One category of delinquency that is receiving increasing attention is sexual offending. A meta-analysis, involving 23 studies and a total of 1,756 participants, on neuropsychological performance in sex offenders confirmed that adult sex offenders performed worse on neuropsychological tasks than did members of the general population (d = 0.59), but also showed that sex-offending is highly heterogeneous (Joyal, Beaulieu-Plante, & de Chantérac, 2014). Subgroup analyses showed that sex offenders against children tended to perform lower on so-called higher-order executive functioning tasks compared to sex offenders against adults (albeit with small effect size, d = 0.23), whereas sex offenders against adults were similar to nonsex offenders, with lower scores in verbal fluency and inhibition. The meta-analysis highlights the need for studies in specific subgroups. Few studies have focused on adolescent sex offenders. One study examined a sample of 127 adolescent sex offenders and 56 adolescents convicted of nonsexual offenses (Morais, Joyal, Alexander, Fix, & Burkhart, 2016). In contrast to studies conducted in adults, adolescent sex offenders who had victimized children obtained significantly higher scores on measures of complex executive functioning tasks than both adolescent offenders with peer-aged or older victims and adolescent nonsex offenders. Taken together, these findings suggest possible complex interactions involving developmental age of the offender and victim. Longitudinal studies following adolescent offenders over time are needed to understand the relevance of neuropsychological performance for recidivism and recovery.

Many of the findings related to the neuropsychology of violence may bear implications for the justice system. Although neuroscientific evidence on altered frontal-limbic brain function in relation to violence in adults appears to be increasingly being used in the courtroom (see Klaming & Koops (2012) and Steinberg (2013) for reviews of the literature and case examples), decisions for trying adolescents who committed violent crimes in adult courts hinge on research on brain development and maturation. From a brain maturation perspective, some claim that adolescent brains are not sufficiently mature to justify trying violent adolescents in adult courts. Nevertheless, the overwhelming majority of adolescents are not violent, raising the question of why legal decision making should use age *per se* as a criterion (Satel & Lilienfeld, 2013). Moreover, neuropsychological function and brain maturation may account only for a relatively small amount of variance in physical violence. Thus, the sensitive issue of trying violent adolescents in adult courts should probably rest on broader grounds and evidence reviewed herein to inform the legal decision making system should be used with caution.

Intermittent Explosive Disorder

DSM-5 (American Psychiatric Association, 2013) places intermittent explosive disorder (IED) under the category of Disruptive Impulse-Control and Conduct Disorders. The primary feature of IED is the frequent experience of short and discrete episodes of aggressive behavior of rapid onset resulting in personal injuries or property damage. The course, onset, and prevalence of IED are poorly understood, although this condition appears to be

more common in males than females. Not surprisingly, there is a dearth of studies on the neuropsychological correlates of IED, although its neurobiology is better understood (Coccaro, 2012). In one controlled study, Best, Williams, and Coccaro (2002) showed that IED participants performed poorly on the Iowa Gambling Task compared with healthy controls. Nevertheless, there were no group differences on the Self-Ordered Pointing test, a working memory test most sensitive to physical aggression (Séguin et al., 2004). This finding is consistent with the idea that IED may be neurocognitively distinctive from other, more common forms of violence, suggesting an IED profile with predominantly affective and social rather than cognitive deficits (Lee et al., 2016). More research is needed to further understand whether impaired neuropsychological functioning in IED is distinct from other impulse-control disorders. Such research should be embedded in a developmental framework capturing the age of onset (Coccaro, 2012).

Conduct Disorder

When Morgan and Lilienfeld (2000) reviewed the CD literature they found a medium effect size of $d = 0.4$ (weighted $d = 0.36$) for poor performance on executive functioning tasks. The meta-analysis by Ogilvie et al. (2011), which mixed both ODD and CD but separated ADHD, reported a slightly higher effect size ($d = 0.54$).

Combining CD and ODD in analyses may be justified when addressing certain research questions, especially those aimed at understanding what is common among externalizing problems, if not across psychopathologies (Castellanos-Ryan et al., 2016). At the same time, this approach is problematic when trying to examine specificity to violence, because neither ODD nor CD necessarily encompass physical aggression. We first provide an example to illustrate the potential importance of accounting for the CD symptom of physical aggression in studies of the neuropsychology of antisocial behavior. In one study that focused on physical aggression and hyperactivity, 67% of boys classified as CD and 72% of boys classified as ADHD between the ages of 14 and 16 were in the high physical aggression and high hyperactivity trajectories, respectively, on the basis of teacher ratings from age 6 to 15 (Séguin et al., 2004). Nevertheless, across the entire sample only 5% and 6.7% of boys met criteria for CD or ADHD, respectively. This finding suggests that if one is high in physical aggression (or hyperactivity), one is more likely to meet criteria for CD (or ADHD). But that study was different from the typical ones selecting for CD and ADHD in that it selected children on the basis of physical aggression and hyperactivity instead. A selection based on CD and ADHD may not necessarily have included either physical aggression or hyperactivity, or these specific behaviors may have contributed only a low weight to the diagnoses. Thus, a focus on physical aggression and hyperactivity yielded a greater number of study participants, most of whom did not meet criteria for CD or ADHD, and those more specific behaviors were sensitive to neuropsychological function (Séguin et al., 2004).

Second, in addition to the comorbidity of other externalizing problems with CD, we have already highlighted the problem of heterogeneity of mechanisms correlated with two of the four key symptoms of CD – physical aggression and theft. There is now a growing number of studies

examining subtypes of CD symptoms that supports the replicability of the negative association between physical aggression and neurocognitive dysfunction, but that finds links between such dysfunction and theft or other nonaggressive conduct problems to be either essentially nonexistent (Barker et al., 2007; Hancock, Tapscott, & Hoaken, 2010) or positive (Barker et al., 2011; Walsh, 1987). Thus, from a neuropsychological perspective, the conflation of symptoms within CD may mask otherwise-important heterogeneity (Burt, 2009, 2013).

Antisocial Personality Disorder

In DSM-5, ASPD is diagnosed only in individuals over the age of 18 with evidence of CD. As we mentioned earlier, psychopathy is often found among individuals with ASPD. We also note that PCL-R scores > 20 but < 30 (the standard PCL-R cut-off for psychopathy) may also reflect ASPD, and that many ASPD individuals will have a criminal record. Compared with other categories in which antisocial behavior plays a role, there are relatively few neuropsychological studies of ASPD. The most recent meta-analysis included 11 studies and found that ASPD's associations with executive dysfunction are weak (effect size $d = 0.19$), (Ogilvie et al., 2011). One study found poorer executive function (working memory, cognitive flexibility, and inhibitory control) in ASPD relative to nonoffenders, regardless of the presence or absence of co-occurring psychopathy (De Brito, Viding, Kumari, Blackwood, & Hodgins, 2013).

Other Disorders Where Violence May Be Present

Although violence is likely to be found in the more explicitly antisocial disorders already reviewed, violence is often associated with alcohol- and substance-use disorders, schizophrenia, and bipolar disorder. Hence, we briefly review their relation to neuropsychological function.

Alcohol and Substance Use

A considerable limitation to the literature investigating neuropsychological function in violent problem behaviors and associated disorders is a potential confound with alcohol and substance abuse. In fact, most violent acts may occur under the influence of substances (Murdoch, Pihl, & Ross, 1990; Room, Babor, & Rehm, 2005). Although the culture of illegal drugs is also associated with violence, this is not dispositive evidence for the violence potential of the drugs themselves. The other key issue to consider is developmental transactions between substance use and neuropsychological function. Although several studies note cross-sectional correlations between substance use and neuropsychological function in acute states, few have examined whether the association found before the onset of substance use was changed afterwards or if use was related to developmental change in neuropsychological function. Building on a series of longitudinal studies on the neuropsychology of physical aggression (Séguin et al., 1995; Séguin, Arseneault, Boulerice, Harden, & Tremblay, 2002; Séguin et al., 2004), we have recently reported that early onset of cannabis use was predicted by pre-use IQ and poor short-term and working memory (Castellanos-Ryan et al., 2017). Nevertheless, we also showed that, by age 20, changes only in verbal IQ (accounted for by poor high-school graduation), trial-and-error learning, and reward-processing were linked to onset and frequency of use

in adolescence, even after controlling for CD, ADHD, and concurrent cannabis or alcohol use. Although such studies do not demonstrate causality, the developmental transactions observed need to be taken into account in studying the neuropsychology of violence.

Our central question concerns the role of neuropsychological factors in this equation. Most research on this topic has focused on executive function. The role of executive function in alcohol-associated aggression has been illustrated well by the work of Giancola (2004), who showed that aggression in reaction to provocation (using a shock paradigm) was possibly more a function of executive function than of alcohol. Nevertheless, synergistic mechanisms may be at play, as alcohol preferentially increased aggression in men with lower levels of executive function. Alcohol may also moderate the quality of executive function on a state (temporary) basis, mostly on the descending limb of the blood-alcohol curve (Pihl, Paylan, Gentes-Hawn, & Hoaken, 2004). Finally, recent models have attempted to integrate cognitive theories of alcohol-induced aggression with neurobiological theories of aggression. For example, Heinz, Beck, Meyer-Lindenberg, Sterzer, and Heinz (2011) proposed that the presence of certain risk genotypes/alleles in combination with adverse environmental factors (e.g., early-life stress) affect the functioning of key neurotransmitter systems (e.g., serotonin, GABA). This outcome in turn would lead to an increased activation in subcortical (bottom-up) limbic circuits and impaired (top-down) prefrontal function, which may place individuals at risk for both increased alcohol intake and impulsive aggression. Acute or chronic alcohol intake, in turn, can further inhibit executive control and facilitate aggressive behavior.

Schizophrenia

Although the prevalence of violence in schizophrenia and other disorders where psychosis is present is about 10% across several studies, its relative prevalence as opposed to the general population is highly variable, with odds ratios varying from 1 to 7 for men, to 4–29 for women (Fazel, Gulati, Linsell, Geddes, & Grann, 2009). In that meta-analytic study, the risk of violent behavior seemed to be highest when there was comorbid substance use, but the rate of violence did not differ between individuals with schizophrenia and with non-schizophrenia-related psychosis. One meta-analysis, investigating risk factors for violence in (primarily) schizophrenia, found that, among several clinical and demographic factors, substance misuse and particularly factors related to violent and nonviolent criminal history (e.g., history of assault, history of imprisonment for any offense, history of recent arrest for any offense, history of conviction for a violent offense) were the strongest predictors of risk of violent behavior (Witt, van Dorn, & Fazel, 2013).

Another meta-analysis (Schug & Raine, 2009) compared neuropsychological performance in individuals with (a) schizophrenia and antisocial behavior, (b) schizophrenia without antisocial behaviors, and (c) antisocial behaviors without schizophrenia. The authors found that individuals with schizophrenia and antisocial behaviors showed widespread cognitive impairments (IQ, attention, executive function, and memory) relative to individuals with antisocial behaviors without schizophrenia, and exhibited

reduced general intellectual functioning and memory dysfunction, relative to individuals with schizophrenia without antisocial behaviors. These associations were characterized by small effect sizes (d = 0.2–0.3 range), but may suggest that schizophrenia with antisocial behaviors may be a subcategory of schizophrenia, a finding supported by electrophysiological and imaging studies (Schug & Raine, 2009). The authors explicitly chose liberal, though arguably valid, approaches to classifying antisociality and schizophrenia. Accordingly, they noted considerable heterogeneity of effect sizes. Because of this they also highlight the need to further clarify neuropsychological differences between violent versus nonviolent forms of antisocial problems within studies of schizophrenia. This is also important because the stigma against schizophrenia stems mostly from the minority of patients that show violence. Thus, interventions could be tailored to a better understanding of these subtypes.

Mood Disorders

Bipolar disorder is associated with several reckless behaviors characterized by impulsivity, as well as poor judgment and planning (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). Bipolar disorder may be associated with violence when it co-exists with substance abuse (OR: 6.4), but markedly less so when substance is absent (OR: 1.3) (Fazel, Lichtenstein, Grann, Goodwin, & Langstrom, 2010). Bipolar disorder has been characterized by overall impairments in neuropsychological functioning, including impairments in intelligence, attention, verbal learning and memory, executive functioning, response inhibition, working memory, set shifting, and processing speed. Some of these impairments, such as those in attention, processing speed, verbal learning/memory, and verbal fluency, have, albeit to a lesser extent, also been observed in healthy first-degree relatives, suggesting a potential trait marker for bipolar disorder (Cardenas, Kassem, Brotman, Leibenluft, & McMahon, 2016). Whether violent bipolar patients are cognitively distinct from nonviolent patients is unknown.

The two core symptoms of the new DSM-5 Disruptive Mood Dysregulation Disorder are (a) recurrent severe temper outbursts that are out of proportion to the situation and inconsistent with the developmental stage and (b) high levels of irritability and anger for most of the day, nearly every day. Despite a conceptual and face-value relevance of this disorder to the study of violence (Mayes, Waxmonsky, Calhoun, & Bixler, 2016) there have yet to be studies of its cognitive neuropsychology.

Conclusion

The neuropsychology of antisocial behavior has a rich history, dating back at least to the mid-nineteenth century. As our review shows, however, not as much can be said about the neuropsychology of physical violence. The main problem is that the specificity of neuropsychological deficits to physically violent behavior has been difficult to establish. The bulk of our knowledge derives from studies of clinical syndromes in which the presence of violence is plausible, but rarely confirmed. Global measures of antisocial, disruptive, externalizing, delinquent, or criminal behavior are also often used. This state of

affairs reflects the heterogeneity of processes underlying those conditions.

Nonetheless, as this book shows, violence is a common outcome of a wide variety of heterogeneous conditions. Violence is present in many disorders for which there may be a partial neuropsychological basis, although that neuropsychological basis may not necessarily be for violence per se. To advance a research agenda in the study of the neuropsychology of violence, we recommend testing for the specificity of behavior problems, such as identifying physical aggression and isolating it from other co-occurring behavior problems. More explicit assessment of violence or physical aggression is needed with key contrasts to such disorders as ADHD, substance use, and to nonviolent forms of antisocial behavior. Although this approach should reduce the heterogeneity in the behavior of interest, it may not reduce the heterogeneity of underlying processes as much as one hopes; subtypes of physical violence will also need to be addressed. A well-documented history of behavior problems should also help to reduce heterogeneity. The study of the process of desistance from violence would be informative in this regard.

Our review shows that neuropsychological impairments, even in executive function, are not necessarily specific to physical aggression. We first observed that lesions among frontal lobe patients, despite their poor executive function, rarely lead to physical violence, though these lesions may lead to explicit forms of violence in the context of other risk factors. Further, there is considerable literature on other problems, such as ADHD, and their relation to executive function. Nevertheless, we noted that studies of ADHD have rarely controlled for co-occurring physical aggression. Further, neuropsychological variables tend to explain at most 8–10% of the variance in measures of violence. Therefore, studies need to examine potential moderators that may increase our ability to predict the risk of violence from neuropsychological dysfunction. Such factors could include a history of abuse or neglect, malnutrition, abilities to process and regulate emotions (including autonomic arousal), capacities to cope with stress and perceived provocation, perinatal factors, and genetic and epigenetic factors (e.g., Provençal et al., 2015). Although many of these variables have been widely studied, few have been examined in conjunction with neuropsychological function.

Furthermore, whereas neuroscience research up to the first decade of this century focused primarily on how cognitive deficits are linked to alterations in the function or structure of one or more single brain areas, cognitive theories have increasingly attempted to explain antisocial behavior-associated cognitive deficits as an alteration within a larger neural dynamic network. Greater application of advanced brain-imaging methodologies such as dynamic functional connectivity and multimodal imaging would help us in understanding the complex dynamic interplay between brain regions; for example, in how an alteration in one region could impact the function of others, and its possible consequences for violence. Neuropsychological function almost certainly comprises one element of an exceedingly complex model of violent behavior. As this chapter illustrates, however, it may provide one essential piece of a still-unsolved puzzle.

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