Examination of the Neurobiological Underpinnings and Cognitive Correlates of Psychopathy

by

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A dissertation submitted in partial fulfillment of
the requirements for the degree of

Doctor of Philosophy

(Psychology)

at the

UNIVERSITY OF WISCONSIN-MADISON

2018

Date of final oral examination: 8/31/2018

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ACKNOWLEDGEMENTS

I would like to express my gratitude to the following people, without whom this project would not be possible:

To my advisor, Joe Newman, for his wisdom, patience, and wine. It has been a privilege to work with such an esteemed scholar and gifted mentor, and I am sincerely grateful for his encouragement and guidance.

To my dissertation committee, for their continual support, insightful comments, and hard questions that encourage critical thinking and intellectual growth.

To my former lab mates, who were instrumental in the data collection process.

To the staff and inmates at Fox Lake Correctional Institution and Oshkosh Correctional Institution, for their participation and cooperation that made this research possible.

To my colleagues Phil Deming, Greg Kirk, and Jaryd Hiser, for their invaluable assistance helping me with imaging methodology.

To Brian and Annie, for their friendship and support from the beginning of my graduate career to the end.

To my family (especially Rhone, Joel, Linda, Michael, Julie, and Eric), for the sacrifices they have made on my behalf, as well as their unending love and reassurance despite still not knowing what I study.

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ABSTRACT

In the domain of psychopathy, prevailing models reflect an artificial dichotomy between affective and attention components of psychopathic dysfunction, contributing to a gap in the literature regarding the mechanistic underpinnings of the disorder. The purpose of this project is to examine a novel theoretical perspective of psychopathy that bridges dominant affective and attentional models. Specific objectives are: (1) To reconceptualize psychopathic dysfunction through the lens of a parsimonious theoretical and neurobiological mechanism; (2) To characterize information processing constraints in psychopathy; (3) To examine the association between information processing style and the formation and use of mental representations; and (4) To characterize the time course of attentional abnormalities in psychopathy and to assess for psychopathy-related differences in brain-behavior coordination during a selective attention task. This dissertation consists of five chapters that will introduce and examine evidence for a novel theoretical framework of psychopathic dysfunction. Specifically, **Chapter 1** reviews dominant models of psychopathy and offers a novel reconceptualization of psychopathy based on a neural network perspective. **Chapters 2 through 4** present three experimental studies assessing predictions of the II theory. These chapters first review related literature and then detail research methodology and data collection. They conclude with the results of analyses and a discussion of the study findings. The project will end with an integrative summary (**Chapter 5**) of the conclusions drawn from the findings, a discussion, and recommendations for further study.

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CHAPTER 1
RECONCEPTUALIZING THE PSYCHOPATHIC DEFICIT: THE IMPAIRED INTEGRATION (II) FRAMEWORK

Content from this chapter was published as: Hamilton, R. K., Hiatt Racer, K., & Newman, J. P. (2015). Impaired integration in psychopathy: A unified theory of psychopathic dysfunction. *Psychological Review, 122*(4), 770-791.

In his seminal work The Mask of Sanity, Hervey Cleckley popularized the notion that psychopathy is characterized by "some subtle and profound defect" (p. 403, Cleckley, 1988) that underlies the distinguishing features of the disorder, namely glibness, impulsivity, irresponsibility, and egocentricity. Since its first publication in 1941, research in the field of psychopathy has burgeoned, resulting in better understanding of psychopathy and antisocial behavior (see Hare & Neumann, 2008). Nonetheless, to date Cleckley's speculated core deficit remains elusive.

Two theoretical camps dominate the field of psychopathy. One camp conceptualizes psychopathy as a syndrome caused by deficient emotion processing; prevailing theories of psychopathy attribute psychopathic individuals' lack of guilt, superficiality, impulsivity, and antisocial tendencies to deviant affective processing (Blair, Mitchell, & Blair, 2005; Lykken, 1995). Specifically, emotion-focused models propose that psychopathic dysfunction stems from a fundamental deficiency in the ability to experience and learn from fear and to develop typical moral emotions such as guilt and empathy. This emotional depravity is thought to permit disinhibited behavior due to a lack of fear or remorse. Moreover, neuroimaging data support claims of dysfunctional emotion circuitry in the brain. Dominant neurobiological models suggest that limbic system abnormalities underlie emotional and behavioral dysregulation related to psychopathy (Kiehl, 2004; Patrick, 1994). In particular, these models posit that psychopathic individuals' emotional and behavioral dysfunction results from temporo-limbic system

hypoactivity and that abnormalities in the amygdala complex, the orbitofrontal cortex (OFC), and associated circuitry underlie psychopathic traits (Blair, 2003; Kiehl, 2006).

The other dominant conceptualization of the psychopathic syndrome centers on more general information processing deficits. Specifically, this perspective views psychopathy as a disorder of attention and suggests that psychopathic traits are not derived from a fundamental emotion deficit; rather, they are manifestations of a broader cognitive deficit. There are two lines of evidence that support this theory. First, psychopathic dysregulation is not specific to affective stimuli (Newman, Schmitt, & Voss, 1997). Indeed, psychopathic individuals fail to process neutral contextual information if this information is outside their attentional focus (see Baskin-Sommers, Wolf, Buckholtz, Warren, & Newman, 2012; Hiatt & Newman, 2006; Hiatt, Schmitt, & Newman, 2004; Zeier, Maxwell, & Newman, 2009). Gorenstein and Newman (1980) proposed that when psychopathic individuals are engaged in goal-directed behavior, they are unable to shift their attention from their current focus to accommodate information that is not directly relevant to the goal. This impairment hampers psychopathic individuals' ability to consider alternative, adaptive responses to situations and effectively regulate their behavior (MacCoon, Wallace, & Newman, 2004).

Second, psychopathic individuals show normal affective reactions when told to focus attention directly on threat-relevant cues. Whereas deficits in passive avoidance learning, electrodermal responses to threat cues, and fear-potentiated startle are commonly cited in support of emotion-deficit models, these well-replicated emotion deficits have been found to disappear under experimental conditions that establish emotion stimuli as the primary focus of attention (Arnett, Smith, & Newman, 1997; Baskin-Sommers, Curtin, & Newman, 2011; Newman, Curtin, Bertsch, & Baskin-Sommers, 2010; Newman & Kosson, 1986). It is only when these cues are

peripheral to a pre-established focus of attention that psychopathic individuals show deficits (e.g., Baskin-Sommers, Curtin, & Newman, 2011; Larson et al., 2013).

Although each prevailing model of psychopathy has its strengths, to date there is little integration across these theories. Current emotion-focused models fail to address the situational specificity of psychopathic dysfunction and non-affective information processing deficits. Current attention-based models attempt to account for affective as well as non-affective information processing deficits but have yet to integrate the rapidly growing evidence documenting brain-related abnormalities associated with psychopathy. This disconnect hinders the scientific understanding of the complete psychopathy construct. A final shortcoming of current theories of psychopathy is their simplification of the syndrome. Theories of emergence challenge the notion that complex psychological processes are the direct sum of underlying components; rather, they suggest that these phenomena arise from reciprocal relationships between lower level component parts (Sawyer, 2002). By underestimating the importance of dynamic neural processes in psychopathy, prevailing models oversimplify the nature of the disorder. These shortcomings call for the delineation of a new model of psychopathy that provides an integrative account of cognitive and affective deficits within the context of a plausible neurobiological framework.

The current state of the field of psychopathy is such that emotion and attention are treated as diametric underlying processes. A shared weakness of existing models is their polarization of emotion and attention. While "many behaviors may be reasonably well characterized in terms of cognitive-emotional interactions such that emotion and cognition are partly separable, in many situations, true integration of emotion and cognition may also take place" (Pessoa, 2009). Indeed, while the influences of emotion and attention are to a certain degree additive, these influences

frequently act in reciprocal manner (see Dolan, 2002; Pessoa, McKenna, Gutierrez, & Ungerleider, 2002; Pessoa & Ungerleider, 2004; Phelps, Ling, & Carrasco, 2006; Taylor & Fragopanagos, 2005; Vuilleumier, 2005). Moreover, they involve anatomically distinct yet overlapping neural circuitry (Okon-Singer, Hendler, Pessoa, & Shackman, 2015; Pessoa, 2008; Pessoa & Pereira, 2013; Storbeck & Clore, 2007; Touroutoglou, Hollenbeck, Dickerson, & Barrett, 2012; Vuilleumier, Armony, & Dolan, 2003; Yamasaki, LaBar, & McCarthy, 2002). Both emotion (Scherer, 2009) and attention (Desimone & Duncan, 1995; Postle, 2006) can be conceptualized as dynamic emergent properties of interactions among distributed brain networks (Pessoa, 2010). The term '*emergent properties'* implies that these constructs are not separate entities that are specifically implemented by the brain; rather, the characteristics of emotion and attention are constantly changing as new information enters each system and each system is continuously modulating the representation of information in the other (Courtney, 2004). Critically, the modulation of one system based on input from the other and the coordination of activity between different neural systems appear to rely on functional connectivity between brain areas (Courtney, 2004; Fingelkurts, Fingelkurts, & Kähkönen, 2005). Impaired integration of affective stimuli with focused attention would inevitably influence the emergent nature of cognition and vice versa. The emergent nature of emotion and cognition makes the understanding of the integrative nature of these constructs critical to their conceptualization. An integrative perspective of psychopathy would enable the preservation of the strengths of existing models while providing a more parsimonious and complete account of identified neural abnormalities and the full range of symptoms.

In recent years, there has been increased recognition of the utility of network models for understanding neural organization and functioning. The vast array of cognitive, affective, and

social functions that underpin human experience requires specifically choreographed patterns of interaction between neural networks (Buckholtz & Meyer-Lindenberg, 2012). The topological organization of neural networks is critical for their overall functioning. Brain network organization is designed such that it is optimized for functional specialization and global integration. Dysfunction of the connections between and within neural systems would hence disrupt the local or global functioning of a given circuit; such disruption can manifest as psychopathology (Buckholtz & Meyer-Lindenberg, 2012). In this way, deficient connectivity in systems-level circuits underpinning cognition and emotion relates to transdiagnostic symptoms displayed in myriad mental disorders. Thus, modern network theory can serve as a useful foundation from which psychopathological symptoms can be understood.

The goal of the current paper is to introduce a unified theoretical framework that provides a new way to conceptualize psychopathy. This theoretical framework is unified in the sense that it incorporates the core findings of each dominant model and assimilates their underlying assumptions into a perspective that integrates and expands these premises. Our Impaired Integration (II) framework borrows from the mechanistic infrastructure of neural network models and proposes that psychopathy is characterized by difficulty rapidly integrating multicomponent perceptual information, which in turn influences the quality of mental representations and shapes the development of associative neural networks. Central to the II theory is the use of systems-level analyses to advance scientific understanding of psychopathy. This preliminary brain-based perspective parsimoniously explains psychopathic dysfunction while bridging the gap between affective and cognitive models of psychopathy.

In the sections that follow, we review current models of psychopathy. We then outline the limitations of these models, as well as the specious nature of the emotion-attention dichotomy in

the field. Next, we propose a novel framework for psychopathy in which poor perceptual binding creates a snowball effect, disrupting associative processing and the development of integrative networks. We further outline how this framework can be used to conceptualize and account for the full range of psychopathic traits and deficits. On the whole, the current theory represents a neurobiological perspective that will break down the emotion-attention dichotomy to provide an integrated view of psychopathy with substantial implications for future research. Additionally, it aims to update working assumptions regarding neural substrates from a modular framework to a network perspective.

Emotion-Based Models of Psychopathy

Prevailing theories of psychopathy typically emphasize affective deficiencies. Lykken's low-fear hypothesis (1995) represents one of the best-formulated accounts of the psychopathic syndrome. According to this theory, psychopathic individuals have a "below average endowment of innate fearfulness" (Lykken, 1995, p. 154), which leads them to be insufficiently motivated to avoid punishment, especially in the face of reward. Support for this model is evident in Lykken's (1957) seminal investigation of psychopathy and anxiety. This study used a classical conditioning paradigm in which a buzzer served as the conditioned stimulus, electric shock served as the unconditioned stimulus, and skin conductance response served as the conditioned response. Results showed that, in general, psychopathic individuals displayed electrodermal hyporeactivity in anticipation of shock. More recent studies have further demonstrated that psychopathic individuals show poor fear conditioning (Birbaumer et al., 2005), poor passive avoidance learning (Newman & Kosson, 1986; Newman & Schmitt, 1998), and a general reduction of defensive reactivity to frank aversive stimuli (Patrick, 2001). These findings support Lykken's notion that psychopathy is characterized by a diminished fear response.

The low-fear model predicts that low fear contributes to symptoms of psychopathy via poor fear conditioning and poor passive avoidance learning (i.e., learning to inhibit behavior to avoid punishment). Lykken suggested that this alleged deficit makes psychopathic individuals more difficult to socialize, since many parenting methods rely on learning from responses to punishment. This model revolutionized the field of psychopathy by linking psychopathic dysfunction to a single underlying emotional process. Although it provides a compelling account of the fearlessness seen in psychopathy, it does not address the fact that this deficit disappears when fear-related cues are the direct focus of attention. Moreover, it does not specify the source or underpinning of the fear-conditioning deficit. Lastly, it does not sufficiently explain performance deficits on laboratory tasks that involve non-fear related emotions and affectively neutral stimuli (see Newman & Brinkley, 1997).

Building on the low-fear model, Blair (2001) proposed that disturbances in the processing of affective cues impair the development of associations between unconditioned emotional stimuli (e.g., distress cues) and conditioned responses (e.g., the inhibition of violence). According to Blair's (2006) Integrated Emotion System (IES) model, dysfunction of the amygdala prompts a cascade of deficient affective responding that contributes to inadequate moral socialization (see also Birbaumer et al., 2005; Blair, 2003). On a basic level, the amygdala aids in the detection of threat and enables appetitive and aversive conditioning (Hariri & Whalen, 2011). It also mediates an organism's bottom-up response to biologically relevant stimuli (Kim et al., 2011). The amygdala is critically involved in the formation of stimulus-reinforcement associations, making it integral for acting appropriately to the distress of others (Blair, 1995, 2007). Amygdala dysfunction is thought to impair the ability to experience and recognize negative affect in others, preventing the development of empathy and increasing the likelihood of

violence and general antisociality (Blair, Budhani, Colledge, & Scott, 2005; Marsh & Blair, 2008; Reidy, Zeichner, & Foster, 2009). Research has found evidence for reduced amygdala volume in psychopathic individuals, as well as attenuated amygdala activation (Blair, 2006; Blair, Jones, Clark, & Smith, 1997; Ermer et al., 2012; Gordon, 2004; Harenski, Harenski, Shane, & Kiehl, 2010; Kiehl et al., 2001; Yang, Raine, Narr, Colletti, & Toga, 2009) and electrodermal responses to distress cues (Blair, Morris, Frith, Perrett, & Dolan, 1999). Blair's IES model represents a significant step in linking interpersonal and affective traits in psychopathy to brain structure and function. Despite its explanatory power, the IES model fails to account for attentional modulation of psychopathic individuals' emotion-processing deficits. Additionally, the IES model does not explain psychopathic individuals' abnormal performance on non-affective tasks.

Kiehl (2004) developed an alternative theory of psychopathy that attempts to capture broader cognitive deficits and brain-based abnormalities not addressed by Blair's model. Termed the "Paralimbic Dysfunction Hypothesis of psychopathy," this theory posits that attentional and affective abnormalities seen in psychopathy result from hypofunctioning of neural circuitry comprised of regions of the frontal lobe, limbic system, and temporal lobe. This model is based on neuroimaging data that show widespread structural abnormalities in psychopathic individuals (see Anderson & Kiehl, 2012), in addition to lesion studies. The paralimbic system consists of the OFC, the amygdala, the parahippocampal gyrus, the anterior superior temporal gyrus, and parts of the cingulate gyrus. Past studies indicate that damage to areas in the paralimbic system is associated with deficits characteristic of psychopathy. For instance, lesions to the orbitofrontal cortex impair response reversal and inhibition (Iverson & Mishkin, 1970; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004), and result in deficient emotion processing (Bechara,

Damasio, & Damasio, 2000; Goodkind et al., 2012). Damage to the anterior cingulate leads to defective error monitoring (Botvinick, Cohen, & Carter, 2004), perseveration (di Pellegrino, Ciaramelli, & Ladavas, 2007), and difficulties processing emotional stimuli (Etkin, Egner, & Kalisch, 2011). Lesions to the medial temporal lobe, including the amygdala, result in emotional and behavioral impairments commonly seen in psychopathic individuals (see Kiehl, 2006).

In contrast to Blair's (2008) model, Kiehl focuses less directly on the amygdala and more on the broader network of the paralimbic system, a network critically involved in linking cognition, visceral states, and emotion (Eslinger, 2011). The power of the Paralimbic Dysfunction Hypothesis relates to its ability to accommodate the broad neurobiological abnormalities and non-affective (e.g., linguistic) deficits seen in psychopathic individuals. However, its prediction of generalized paralimbic dysfunction would suggest that psychopathic individuals should show global impairment on tasks involving the orbital frontal cortex, insula, cingulate cortices, amygdala, parahippocampal gyrus, and anterior superior temporal gyrus. Thus, it lacks specificity and implicates deficient performance on nearly all laboratory tasks. As a result, the Paralimbic Dysfunction Hypothesis does not account for the situational specificity of psychopathic dysfunction (e.g., Larson et al., 2013; Zeier et al., 2009).

Attention-Based Models of Psychopathy

While the predominant emotion models in psychopathy attribute psychopathic traits to a fundamental deficit in affective systems, attention-focused models argue that the syndrome reflects broader information processing deficiencies that are not specific to affective information. Perhaps the most delineated attention-based model of psychopathy is the Response Modulation Hypothesis (RMH; Gorenstein & Newman, 1980; Newman et al., 1997; Patterson & Newman, 1993). Response modulation, or "the temporary suspension of a dominant response set and a

brief concurrent shift of attention from the organization and implementation of goal-directed responding to [stimulus] evaluation" (Newman & Lorenz, 2003, p. 905), involves shifting attention from a dominant response set (i.e., primary focus of attention) to accommodate unanticipated nondominant cues. Deficient response modulation limits a person's ability to use contextual information that contraindicates goal-related behavior because this information is not integrated with the current attentional focus (MacCoon et al., 2004; Newman, 1998). Beyond affective and inhibitory cues that might contraindicate behavior, the response modulation model holds that the processing of future consequences and other peripheral or delayed considerations "could be disrupted or 'eclipsed' by the presence of more immediate, prominent, motivationally significant cues" (Newman, Gorenstein, & Kelsey, 1983, p. 147).

Newman and colleagues have used this framework to account for psychopathic deficits that undermine self-regulation, such as failure to learn from experience (e.g., Patterson & Newman, 1993) and decreased responsivity to cues that contraindicate current goal-directed behavior (e.g., Zeier et al., 2009). According to the RMH, disinhibition characteristic of psychopathy results from a failure to stop and reflect on the potentially maladaptive nature of a given behavior. Critically, stimuli that normally initiate response evaluation and self-regulation are typically peripheral to a goal-directed focus of attention. For instance, these stimuli can include moral conventions, legal requirements, long-term motivations, and past experiences. To the extent that poor RM curtails attention to these cues, psychopathic individuals are unlikely to moderate goal-directed behavior. Failure to integrate and reflect upon information likely contributes to a superficial (i.e., less well-elaborated) level of processing. This shallow processing in turn would disrupt the building of associtive networks between actions and their consequences. Failure to form these causal links would prevent an individual from considering

the potentially maladaptive effects of an action and cause him or her to act in a disinhibited manner by perseverating their dominant response set (Patterson & Newman, 1993).

The RMH further accounts for the situation-specific nature of psychopathic deficits in emotion processing (e.g., Newman & Schmitt, 1998). Specifically, it predicts that when cues that initiate response evaluation and self-regulation are the focus of attention, psychopathic individuals will not show characteristic deficits. Thus, this theory addresses the context specificity of psychopathic dysfunction. The RMH (1993) was the first model of psychopathy to account for the situation-specific nature of psychopathic dysfunction as well as more general deficits relating to affective processing. However, the neurobiological basis of this model has yet to be defined. Moreover, it traditionally has difficulty explaining evidence of affective dysfunction when complex emotional information is focal.

Most recently, Baskin-Sommers and colleagues (2011) have proposed the Attention Bottleneck Model of psychopathy. The central premise of this model is that an early attention bottleneck underlies poor response modulation in psychopathic individuals. Specifically, they suggest that abnormalities in early selective attention obstruct the processing of information unrelated to a dominant response set. Psychopathic individuals' information processing abnormalities can be characterized as a disorder of early selective attention such that selective attention abnormalities reduce the scope of attention to the point that anything other than the pre-potent focus of attention remains unelaborated (see MacCoon et al., 2004; Newman & Baskin-Sommers, 2011; Newman et al., 2010). When information is congruent and under the attentional spotlight, psychopathic individuals generally do not show clear expression of characteristic deficits. That is, when experimental manipulations encourage them to incorporate affective or inhibitory cues as part of their dominant response set, psychopathic individuals successfully

process the targeted information (Arnett et al., 1997; Baskin-Sommers, Curtin, & Newman, 2011; Hiatt & Newman, 2006; Meffert, Gazzola, den Boer, Bartels, & Keysers, 2013; Newman & Kosson, 1986).

Evidence for the Attention Bottleneck Model comes from studies utilizing attentional manipulations. Zeier et al. (2009) used a flanker-type task to test the effects of the bottleneck on the processing of peripheral information in psychopathic individuals. According to the attention bottleneck theory, psychopathic participants would display significantly less interference to response incongruent information than non-psychopathic participants when attention was cued to the target location (i.e., the response incongruent information was peripheral to the predefined target location) but display normal interference when there was no pre-potent focus of attention. The results supported this hypothesis and were consistent with the contention that attention moderates psychopathic individuals' responsivity to cues that conflict with the dominant response set (see Zeier & Newman, 2013). Baskin-Sommers and colleagues (2011) posit that psychopathic individuals fail to integrate unexpected or incongruent information with an ongoing attentional set because an attentional bottleneck prohibits processing of these cues. Moreover, this bottleneck may encourage the sequential processing that limits the ability to rapidly process perceptually complex stimuli even if these stimuli are task-relevant, thus contributing to an inefficient information processing style (Hamilton, Baskin-Sommers, & Newman, 2014). Consequently, psychopathic individuals remain oblivious of these cues and do not use them to regulate behavioral and affective responses (Newman & Baskin-Sommers, 2011). Baskin-Sommers and colleagues' Attention Bottleneck Model (2011) adds to the strengths of the RMH by providing a mechanism by which attentional dysfunction occurs. Additionally, it accounts for psychopathic individuals' deficient processing of complex focal information, predicting that an

early attention bottleneck filters information and reduces the ability to attend to multiple ongoing streams of information. Accordingly, it enables clearer predictions on cognitive and affective tasks. However, as with the RMH, it does not adequately explain neurobiological abnormalities seen in psychopathy.

Another attention-based model of psychopathy is Kosson's left-hemisphere activation (LHA) hypothesis (1996). This model proposes that psychopathic individuals' dysregulated behavior results from deficient processing of information under conditions that place substantial demands on the left hemisphere. For instance, psychopathic individuals display generally inefficient processing in attention, motor, and linguistic tasks that preferentially activate the left hemisphere (e.g., Kosson, 1998; Llanes & Kosson, 2006). In divided visual field paradigms, psychopathic participants display deficits specific to the LHA condition. While the authors of the LHA hypothesis have not specified the underlying mechanism, researchers have proposed that deficits consistent with this model could reflect deficiencies in interhemispheric integration (see Hiatt & Newman, 2007) as well as limited left hemisphere resources. The LHA Hypothesis (1996) similarly accounts for the situational nature of psychopathic dysfunction. However, it predicts global dysfunction during all tasks that tax left hemisphere resources and there is little support for global deficits in neuropsychological tasks that tap left hemisphere functioning (see Smith, Arnett, & Newman, 1992).

Moul and colleagues (2012) proposed the differential amygdala activation model (DAAM), a perspective that attributes emotional and cognitive dysfunction in psychopathy to hypoactivation of the basolateral amygdala. DAAM posits that this reduced activation causes a deficit in the reflexive shift of attention to salient stimuli. The authors emphasize that reflexive shifts of this sort are preconscious and not driven by top-down processes. In addition to

explaining deficits in fear recognition, the model explains passive avoidance and response-reversal deficits in psychopathy as an imbalance of activation between the basolateral amygdala and the central amygdala. The DAAM explains many of the same issues highlighted by Blair and Kiehl but is unique in that it frames these deficits as a problem with attentional orienting and salience detection. It represents the first model to assimilate emotional and attentional perspectives of psychopathy into a single framework. As a result, it represents a significant advancement in the field of psychopathy.

Despite the DAAM's integration of cognitive and affective aspects of psychopathy, it has yet to be applied to the range of psychopathy-related dysfunction. Moreover, its silence on the role of structures outside of the amygdala renders it incomplete. Recent neurobiological data demonstrate the widespread nature of brain abnormalities in psychopathy. Data show that psychopathy is characterized by a range of neural irregularities including morphological and functional abnormalities in frontal and temporal areas, cortical and subcortical gray matter structures, and white-matter pathways (Blair, 2012; Craig et al., 2009; Glenn & Raine, 2008; Gregory et al., 2012; McCloskey, Phan, & Coccaro, 2005; Meffert et al., 2013; Motzkin, Newman, Kiehl, & Koenigs, 2011). In addition to structural abnormalities and connectivity deficits within the temporal cortex, the brains of psychopathic individuals show widespread deficits in neural connectivity (Ly et al., 2012; Motzkin et al., 2011; Philippi et al., 2015). Yang and colleagues (2012) conducted a study in which they applied graph theory-based methods to examine information flow and connectivity in psychopathic and nonpsychopathic individuals. They found irregular interregional connectivity in the psychopathic individuals in areas throughout the brain. Taken together, these results indicate that information processing deficiencies in psychopathy may not solely reflect isolated structural abnormalities or deficient

function of a single brain region, but instead might relate to dysfunctional connectivity between and among neural systems.

A Call for Integration

While the delineated models have greatly advanced the field, each has its limitations and no one model addresses the psychopathic syndrome in its entirety. A shared weakness of all of these models is their modularity: they fail to address the interdependent, bidirectional nature of cognition and affect. As aforementioned, artificially separating emotion and cognition misrepresents the integrated nature of these constructs. Failure to acknowledge the reciprocal developmental association between them will fail to produce an integrated model that captures the complexity of the psychopathic syndrome. Overall, in isolation each model falls short in explaining some aspect of the disorder (see Table 1). This failure calls for a new neurobiological framework that integrates the full range of emotion and non-affective deficits while addressing the widely distributed brain irregularities. The current proposal outlines a novel theoretical framework that integrates and explicates the affective and attentional correlates of psychopathy while tying these deficits to a neurobiological substrate. The proposed framework interprets psychopathy through the lens of modern network theory. In the sections that follow, we will present an overview of neural systems involved in information processing and outline the importance of neural networks for integrative cognition. We will subsequently demonstrate how psychopathy can be conceptualized as a disorder of information integration that is associated with abnormal topographical patterns of neural connectivity.

Table 1. *Comparison of Predictions, Strengths, and Weakness of Dominant Models of Psychopathy and the II Framework*

Model	Predictions	Strengths	Weaknesses
Lykken's Low Fear Hypothesis	• Diminished fear responses • Failure to learn from punishment	• Compelling account of psychopathic fearlessness • Accounts for characteristic fear conditioning and passive avoidance deficits	• Does not address situational specificity of deficits • Does not specify mechanism • Does not explain deficits unrelated to fear
Blair's Integrated Emotion Systems Model	• Impaired affective processing • Reduced amygdala functioning	• Links brain and behavior • Explains key affective deficits	• Does not address situational specificity of deficits • Does not explain deficits unrelated to fear and distress
Kiehl's Paralimbic Hypothesis	• Paralimbic system hypoactivation • Poor performance on tasks involving paralimbic structures	• Accommodates broad neurobiological abnormalities • Addresses non-affective, as well as affective, deficits in psychopathy	• Makes vague, non-specific predictions regarding dysfunction • Does not address situational specificity of deficits
Newman's Response Modulation Hypothesis	• Attentional moderation of information processing deficits (normal emotion processing if emotion is focal) • Shallow processing of associations due to lack of reflection	• Addresses situational specificity of psychopathic dysfunction • Addresses non-affective, as well as affective, deficits in psychopathy	• Does not explain neurobiological abnormalities • Lacks defined neural substrate • Does not explain affective deficits when emotion is focal
Baskin-Sommers et al.'s Attention Bottleneck Model	• Attentional moderation of information processing deficits (normal emotion processing if emotion is focal) • Sequential processing of multi-component information	• Addresses situational specificity of psychopathic dysfunction • Addresses non-affective, as well as affective, deficits in psychopathy • Explains affective deficits when emotion is focal	• Does not explain neurobiological abnormalities • Lacks defined neural substrate
Kosson's Left Hemisphere Activation Model	• Dysfunction when substantial demand is placed on left hemisphere	• Addresses situational specificity of psychopathic dysfunction • Addresses non-affective, as well as affective, deficits in psychopathy	• Predicts global dysfunction during all tasks that tax left hemisphere resources, yet there is little support for global deficits in neuropsychological tasks that tap LH functioning • Lack of specificity regarding with LH activation impairs performance

Moul et al.'s Differential Amygdala Activation Model	• Impaired reorienting and salience detection • Context specific fear recognition, passive avoidance, and response reversal deficits • Hypofunctioning of the basolateral amygdala	• Integrates affective and cognitive components of psychopathy • Parsimonious	• Does not address widespread neurobiological abnormalities • Does not address psychological correlates of psychopathy (e.g., information processing style) • Has not been extended to address other well-replicated performance deficits associated with psychopathy (e.g., language, memory)
Impaired Integration Theory	• Abnormal connectivity undermines ability to integrate functioning of different neural systems • Aberrant brain topography characterized by reduced global efficiency, fewer long-range connections between networks and more local connections, lower synchronicity, and increased modularity • Deficits in complex information integration impair simultaneous processing	• Addresses situational specificity of psychopathic dysfunction • Addresses non-affective (e.g., attention, language, memory), as well as affective, deficits in psychopathy • Accounts for widespread neurobiological abnormalities • Integrates affective and cognitive components of psychopathy in unified framework • Provides common mechanism for attention bottleneck and affective deficits • Elucidates the nature of psychopathic individuals' information processing style • Makes specific, testable predictions • Parsimonious	• Fundamental tenets have yet to be substantiated • Current lack of empirical evidence on psychopathy-related abnormalities in the functioning of intrinsic connectivity networks • Difficulty defining nodes in graph theoretical approaches

The Integrative Basis of Cognition

Information processing involves the transformation of sensory information through a complex cascade of interactions between local and distributed neuronal groups (Buzsáki & Draguhn, 2004). These interactions allow for the dynamic integration of information at each step of the hierarchical sequence. Cognitive processes begin with the encoding of sensory information in primary sensory and motor cortices. These early sensory areas have a modular structure with

predominantly local connections (Sepulcre et al., 2010) and represent elementary perceptual features of stimuli (Fuster, 2003). Sensorimotor cortices produce output directed to unimodal association areas. Each of these regions is modality-specific, responding to output from a particular primary sensory area. Unimodal association areas represent multi-dimensional sensory information and bind elementary stimulus features into a more complex percept (Fuster, 2003). Functional streams of sensorimotor information converge within a multimodal integration network comprised of prefrontal, lateral temporal, limbic and paralimbic areas. These association areas, which are comprised of associative neuronal assemblies, serve as cortical epicenters within large-scale networks (Singer, 2013; Wright, 2015). They critically bind the output of unimodal and other transmodal areas into integrated cross-modal perceptual representations (Mesulam, 1998). Moreover, the widespread reciprocal connections between these regions enable top-down influence of unimodal areas. Among the heteromodal areas of the association cortex, the lateral parietal, lateral temporal, posterior cingulate, and medial/lateral prefrontal cortices act as terminal hubs that underlie higher-order cognitive functions such as internal representation, memory, learning, and decision-making (Fiddick & Clark Barrett, 2001; Geary, 2005; Gofrey-Smith, 2001; Sepulcre, 2014; Yeo et al., 2014).

In contrast to sensorimotor regions, the connectivity of association areas additionally includes long-range ‘short-cuts’ that link neurons in different cortical regions (see Mueller et al., 2013). The direct interconnections between spatially remote brain regions increase the efficiency of information processing since the transmission of information between any two nodes in a network requires few connections (Bassett & Bullmore, 2012). The greater proportion of connections within clustered nodes (i.e., brain regions) relative to between nodes gives the brain a modular structure that conserves wiring-costs while enabling the communication between

distinct neuronal populations. The properties of high clustering, high efficiency of information transfer, and modularity give the brain a 'small-world' architecture. This topography is characterized by dense local clustering of connections between neighboring brain regions yet a short path length between distance regions due to the presence of relatively few, direct long-range connections. Importantly, these topographical properties support specialized information processing, as well as distributed integrative processing of these specialized outputs (Bassett & Bullmore, 2006). Neural connectivity creates an integrated workspace characterized by rapid information exchange between distinct modules within a globally distributed network (Bullmore & Sporns, 2012).

Taken together, cognition is an emergent process that arises from the interaction and integration of bottom-up and top-down influences. Failure in any step in the processing stream would thus have critical consequences for the later elaboration and integration of information.

Associative Neural Networks

As noted, the association cortices comprise a series of interlocking large-scale networks (Buckner & Krienen, 2013). These systems critically underlie associative processing and, as a result, behavior and cognition (Buckholtz & Meyer-Lindenberg, 2012; Laird et al., 2011). The following sections outline five core interconnected functional networks and their roles in supporting higher-order cognition (see Menon, 2011). It is important to note that these circuits are not universally defined. Indeed, some researchers consider certain networks to be functionally analogous whereas others believe that they serve diverse functions despite their anatomical overlap (Wig, Schlaggar, & Petersen, 2011). Despite the debate, the presented networks are widely cited in the literature and underlie important domains of psychological

functioning, including executive functioning, attentional control, introspection, and salience processing.

Cognitive Control Networks

Frontoparietal Control Network (FpCN). The FpCN, also known as the executive control network or the central executive network, is comprised of frontal-parietal heteromodal association cortices (Seeley et al., 2007). More specifically, it consists of rostrolateral prefrontal cortex (rlPFC), middle frontal gyrus (MFG), anterior insula/frontal operculum (aIfO), dorsal anterior cingulate cortex (dACC), precuneus, and anterior inferior parietal lobule (aIPL; Spreng, Sepulcre, Turner, Stevens, & Schacter, 2013). Additionally, this network plays a critical role in goal-directed cognition by mediating the adaptable allocation of selective attention (Cocchi, Zalesky, Fornito, & Mattingley, 2013; Dosenbach, Fair, Cohen, Schlaggar, & Petersen, 2008).

Cingulo-opercular Network (CoN). Frequently co-activated with the FpCN, the CoN is involved in the extended implementation and maintenance of task-set. It is comprised of regions in the dACC, dorsal anterior prefrontal cortex (daPFC), aIfO, thalamus, and right aIPL. The CoN is suspected to underlie tonic alertness, or effortful, internally driven sustained attention (Sadaghiani & D'Esposito, 2014). There is evidence that the CoN mediates the dynamic switching between the default mode network (see below) and the FpCN, enabling the switching from an interoceptive state to a goal-directed state. Additionally, this system is thought to moderate activity of the FpCN following errors in task performance (Cocchi et al., 2013; Dosenbach et al., 2008). The IPL is part of the parietal association area, a region involved in multimodal sensory integration (Lynch, 1980). Moreover, the insular cortex has reciprocal connections with sensory, motor, limbic, and association areas of the brain, making it an

important integrative hub (Gu, Liu, Van Dam, Hof, & Fan, 2013; Sridharan, Levitin, & Menon, 2008).

Attentional Control.

Dorsal Attention Network. The dorsal attention system overlaps with the FpCN. This network subserves externally directed cognition and orienting attention towards the environment by generating top-down signals that bias sensory processing according to preexisting goals and expectations (Corbetta, Patel, & Shulman, 2008; Spreng et al., 2013; Vossel, Geng, & Fink, 2014).

Ventral Attention Network. The ventral attention network is activated by the presence of salient, task-relevant stimuli (Corbetta et al., 2008; Frank & Sabatinelli, 2012; Thiel, Zilles, & Fink, 2004; Vossel, Weidner, Driver, Friston, & Fink, 2012). It is a crucial mechanism mediating attentional disengagement, prompting stimulus-driven shifts in attention. 'Circuit breaker' signals from the ventral attention network interrupt ongoing, goal-directed activity in the dorsal stream and trigger reorienting toward salient stimuli (Corbetta et al., 2008; Kim, 2014). As aforementioned, the TPJ is the central hub of the ventral system; this region constitutes higher-order association cortices in the temporal and parietal lobes (Bukowski & Lamm, 2017).

Default Mode Network (DMN)

The DMN is a collection of brain regions whose neural activity is temporally synchronous and is deactivated during goal-oriented or attention-demanding tasks, thus having greater activation during a baseline state (Greicius, Krasnow, Reiss, & Menon, 2003). This network consists of a distributed set of regions that includes the parietal association area (Buckner, Andrews-Hanna, & Schacter, 2008). Specifically, it includes the posterior cingulate/retrosplenial cortex (PCC/Rsp), medial prefrontal cortex (mPFC) and inferior parietal

lobules (IPLs), and may also include the medial temporal lobe (MTL). The DMN refers to a mode of stimulus-independent thought that is characterized by introspection, self-referential thinking, and activities related to internally directed attention, such as thinking about the future, recollecting autobiographical events, or engaging in perspective taking (Buckner et al., 2008; Whitfield-Gabrieli & Ford, 2012).

Salience Network (SN)

The SN anatomically overlaps with the CoN and is also closely related to the ventral attention network (Menon, 2011); indeed, other than the fact that the coordinates in the insula are ventral to those in CoN, these two networks are highly comparable (Power et al., 2011). Accordingly, the SN comprises the bilateral insula, dACC, and ventrolateral prefrontal cortex (vlPFC; Seeley et al., 2007). As noted, the insula is a significant association area in the brain. The SN is activated in response to cognitive, biological, or emotional salience. This network is important for switching between other networks (i.e., altering which of the other networks is most active) to facilitate access to attentional resources and working memory upon detection of salient information (Sridharan et al., 2008). In this way the SN modulates the activity of other large-scale functional networks and flexibly enables behavioral adaptation (Goulden et al., 2014; Menon & Uddin, 2010; Uddin, Supekar, Ryali, & Menon, 2011).

Reconceptualizing the Psychopathic Deficit: The Impaired Integration (II) Framework

We propose that at the core of psychopathy lies a fundamental deficit in perceptual integration. Specifically, our II framework states that failure to rapidly bind components of multi-dimensional stimuli in psychopathy creates a perceptual bottleneck resulting in unelaborated mental representations and the development of abnormal topography in associative neural networks (see Figure 1). In the following sections we present the premises of our theory

as four conceptually separable, but interdependent processes. In each section we present evidence supporting the II perspective.

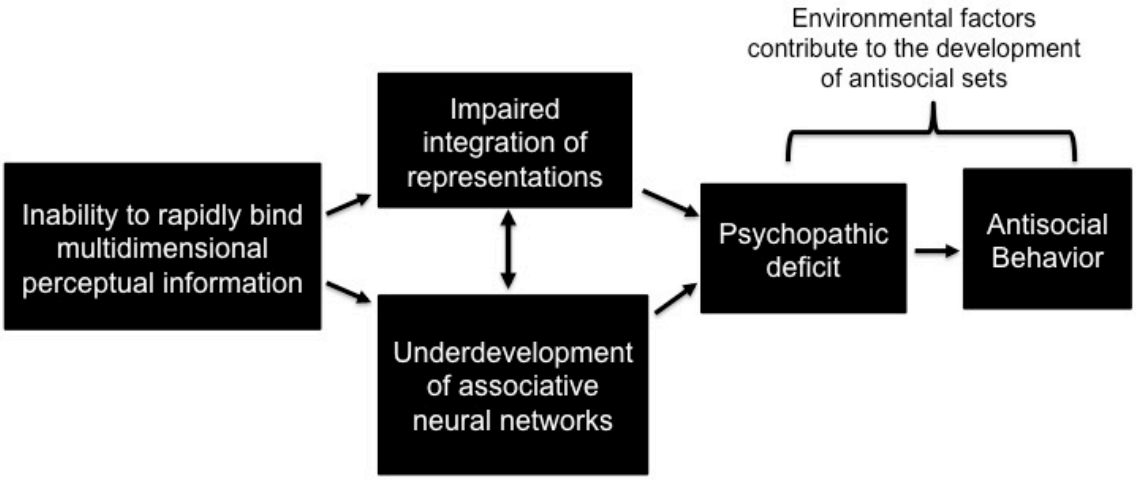

Figure 1. The Impaired Integration (II) theoretical framework. The II theory proposes that difficulty binding sensory features into a unified percept results in a perceptual bottleneck that fosters a sequential information processing style in psychopathic individuals. As a result, there are reciprocal developmental effects between information processing, learning, and neural connectivity such that white matter tracts within and between associative cortices display a unique topographical organization, resulting in reduced coordination of neural networks in the absence of effort. The topographical profile of psychopathic individuals enables reduced distractibility and shallow processing of extraneous information, which includes peripheral socioemotional cues. Combined with environmental influences that contribute to the development of antisocial sets, the integrative deficit results in the development of antisocial behavior characteristic of psychopathy.

Impaired Integration and Perceptual Binding

The II theory proposes that psychopathy is characterized by difficulty rapidly binding components of multidimensional sensory stimuli. In general, perceptual processing involves the prioritization of stimuli based on the salience and relevance of the stimuli. Specifically, bottom-up and top-down factors interact to produce a combined representation of 'priority' that guides selective attention (Fecteau & Munoz, 2006; Ptak, 2012). The allocation of attentional resources to multidimensional objects depends on perceptual load: when perceptual load is low, available processing resources 'spill-over' to lower priority stimuli. When perceptual load is high, attentional capture and processing of lower priority stimuli is attenuated (Cosman & Vecera, 2009; Lavie, 2005). The proposed integrative impairment in psychopathy is suspected to tax

perceptual processing resources, thereby mimicking conditions of high perceptual load. As a result, attention would not extend to the processing of distractor stimuli (Xu, Monterosso, Kober, Balodis, & Potenza, 2011) and overall processing of complex stimuli would be superficial and limited.

Evidence. Several lines of evidence support the proposal that psychopathic individuals fail to bind stimulus components under time pressure. Sadeh and Verona (2008) had psychopathic and nonpsychopathic offenders complete the Perceptual Load Task to test whether higher levels of psychopathy would be associated with reduced distractor processing at lower levels of perceptual load. Results were consistent with this prediction; individuals high in primary psychopathy displayed reduced distractor processing at a lower level of perceptual load than nonpsychopathic individuals. They concluded that psychopathic individuals may have reduced perceptual processing capabilities. Glass and Newman (2009) conducted a study in which criminal offenders completed an emotional memory task that assessed the effects of emotion on memory for focal and contextual information. Specifically, participants were instructed to remember a series of words, some of which were emotional. After the task, participants completed a free recall task, as well as a surprise test of the associated contextual features (location, box color, and word color). Although there was no psychopathy-related difference in memory bias for emotional over neutral words in the primary conditions, higher levels of psychopathy were associated with reduced memory bias in the contextual conditions. This finding suggests that, in the absence of explicit instructions to attend to contextual information, psychopathic individuals fail to bind this information into a unified percept. In other words, this information is not prioritized and thus does not make it through the perceptual bottleneck.

Further support for the importance of perceptual load comes from Baskin-Sommers and colleagues' (2013) picture-viewing study. This experiment assessed psychopathy-related differences in emotion-modulated startle. Critically, researchers manipulated processing demands by incorporating novel images and familiar images. Since familiarity reduces perceptual load and subsequently allows for more processing resources to be allocated to perceptual integration, researchers hypothesized that psychopathic individuals would show normal emotion-modulated startle when viewing familiar pictures. However, they expected psychopathic participants to display classic emotion-modulated deficits when viewing novel images due to increased perceptual demands and the concomitant inhibition of peripheral (in this case, affective) processing. Results were consistent with these predictions, suggesting that psychopathic individuals have difficulty rapidly processing multicomponent perceptual stimuli and that this deficit may undermine the processing of peripheral emotion cues.

Impaired Integration and Learning

In addition to influencing the encoding of perceptual features and the formation of mental representations, the purported integrative deficit would have a cumulative effect by interfering with associative processing. Comprehensive information processing depends on elaboration. Reflective attention is posited to be the mechanism by which mental representations become activated and maintained for prolonged processing and evaluation (Koenig & Mecklinger, 2008; Shipstead, Harrison, & Engle, 2012). In cognitively demanding situations, psychopathic individuals may engage in shallow information processing even if this information is focal because of limited attentional resources and reduced automatic integration of multiple components. Thus, although information may be perceived, shallow processing may preclude this information from being integrated with existing representations. In short, impaired

integration would both reduce the elaboration of currently held mental representations and impair associative linking of present and past knowledge.

Evidence. Psychopathic individuals' failure to link past memories and associations with current events when performing goal-directed activity might inhibit their ability to use this information to make memory-based predictions that guide future behavior (Newman, Patterson, & Kosson, 1987; Patterson & Newman, 1993). A striking example of this failure is psychopathic individuals' poor passive avoidance learning. Passive avoidance learning involves learning to inhibit a response that would otherwise result in punishment. It requires integration of an aversive event with a specific environmental context and the subsequent use of that association to inform future actions. Psychopathic individuals are characteristically unsuccessful at integrating and making use of punishment-related information while engaged in goal-directed behavior (Blair, 2001; Hare, 1965; Lykken, 1957; Newman et al., 1987). Patterson and Newman (1993) propose that, owing to their difficulty integrating peripheral associations, psychopathic individuals form relatively few inhibitory associations while engaged in goal-related activity. As a result, they are less prone to consider the potentially maladaptive consequences of their behavior. In short, failure to integrate past and present mental representations may make it difficult for psychopathic individuals to evaluate their behavior and learn from experience, therefore producing persistent self-regulatory deficits that typify the psychopathic syndrome.

Impaired Integration and Brian Topography

A perceptual bottleneck that undermines that ability to rapidly integrate multi-component sensory information would shape the development connections, resulting in a unique topographical profile characterized by disrupted coordination. Throughout the course of development, experience-evoked neural activity and spontaneous neural synchrony encourage

the formation and maintenance of neural networks. Specifically, these mechanisms support tighter coupling of some regions over time, as well as greater segregation of other regions and the weakening of interregional relationships.

Early in development, brain topography is locally organized, with sensorimotor connectivity well established and connector hubs located in language-related areas (Khundrakpam et al., 2013). During this time, resting-state connectivity networks are in an immature state with weak long-distance connections and a primarily modular structure (de Bie et al., 2012; Fransson, Åden, Blennow, & Lagercrantz, 2010). Neonates display strong connectivity between parietal and frontal regions, regions that comprise the orienting attention network. Over the next two years, functional connections between frontal and parietal areas strengthen with the anterior cingulate, a region implicated in executive attention (Posner, Rothbart, Sheese, & Voelker, 2012). During childhood, functional connections between the dlPFC and posterior parietal regions offer adaptive control during developmental immaturity. This frontoparietal system enables flexible learning of novel stimuli, an executive process that is critical for survival (Fair et al., 2007). Moreover, the functional connections within this system continue to strengthen with age. Research suggests that FpCN development is driven by activity in the insular cortex, a key node in the system that influences cross-network communication (Supekar & Menon, 2012). Overall, the brain displays a hierarchical sequence of maturation: primary sensorimotor connectivity develops early in life, with an increasingly distributed organization throughout adolescence. With age comes greater spontaneous correlated activity within developing brain networks such as the ventral and dorsal attention networks and the DMN (Fair et al., 2009). This restructuring increases neural efficiency to better handle the cognitive demand associated with high-order processing and social cognition (Khundrakpam et al., 2013).

From a developmental perspective, the II perspective theorizes that an underlying vulnerability (e.g., Hecht, 2011; Miskovich et al., 2018) or biochemical process (e.g., Braver & Cohen, 1999; Rodriguez, Kallenbach, Singer, & Munk, 2004; Schnitzler & Gross, 2005) results in an impaired ability to rapidly integrate multi-component information. This deficit, in turn, is suspected to cause the underdevelopment of connections to 'supporting' networks (i.e., those that develop later in childhood), reducing use-induced plasticity (see Elbert & Rockstroh, 2004). Conversely, 'leading' networks (i.e., those important for early survival and the initiation of behavior) may develop normally. Given the aforementioned primacy of the development of the FpCN and the overlapping dorsal attention network, the II theory supposes that the functional connections within these networks are intact. Importantly, the FpCN has particularly extensive brain-wide connections, suggesting that it can regulate other associative networks in a goal-directed manner (Cole, Repovš, & Anticevic, 2014; Pessoa, 2008). While disturbance in the ability to rapidly coordinate neural systems would obstruct rapid integrative process, it would not prohibit the effortful activation of supporting associative networks through the use of leading goal-directed executive networks.

Additionally, a perceptual bottleneck would influence the workings of neural assemblies, groups of neurons that are synaptically bound and frequently coactivated. Assemblies are constantly reincarnated through the interaction of bottom-up and top-down regions. Critically, they serve as the building blocks of mental representations and enable the conscious perception of unified constructs. Thus, irregularities in the processing stream would influence not only the workings of neural systems, but also the content of brain modules (Reser, 2013).

Evidence. To date, few studies have used graph theoretical approaches to evaluate functional connectivity in psychopathy. Indeed, there is relatively sparse data linking the

psychopathic syndrome to the neural networks described above. Nevertheless, there is some preliminary evidence that psychopathy is characterized by neural network abnormalities. Graph theoretical analyses by Philippi and colleagues (2015) on resting state functional connectivity data suggest that psychopathy is characterized by decreased functional connections between neural networks. While this work is preliminary, it provides strong support for the premise of the II theory. Philippi et al. found that psychopathy is characterized by a reduction in small-world network properties; in other words, the networks do not display the characteristics of high clustering, high efficiency of information transfer, and modularity (see Figure 2). Importantly, the networks seem less coordinated due to a lack of connections between distant hubs. Psychopathic individuals show lower functional connectivity between areas within the FpCN (the dACC and right IPS) as well as decreased connections between FpCN and CoN. Furthermore, psychopathic individuals show negative correlation between FpCN and DMN, an association not seen in nonpsychopaths (see also Sheng, Gheytanchi, & Aziz-Zadeh, 2010). These abnormalities may underlie the reduced ability to switch flexibly attention between internal and external foci. Although these findings require further testing and replication, the results support the proposition that psychopathy can be conceptualized as a disorder of neural network organization.

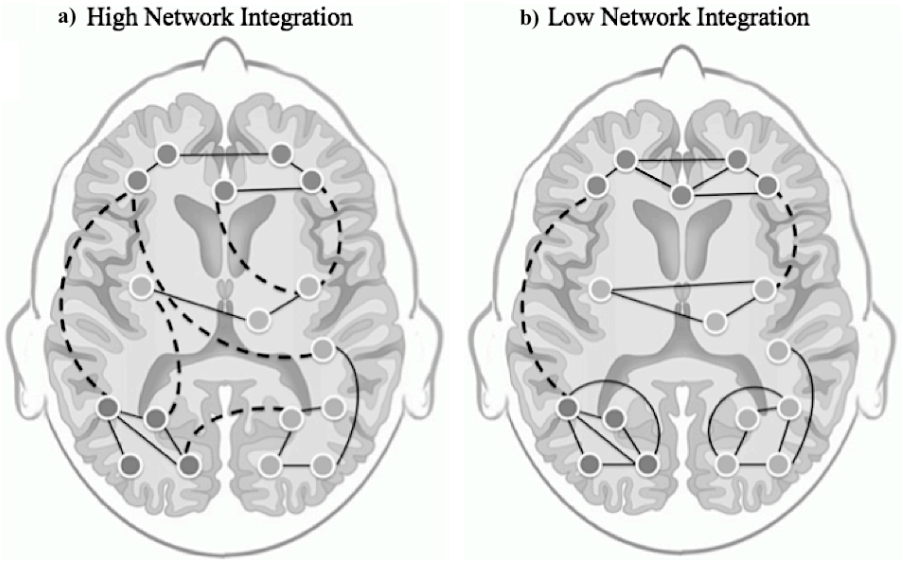

Figure 2. Graphic representation of brain topography supporting low and high integration. a) Typical brain topography is characterized by a small-world architecture that enables efficient integration within and between neural networks. Normal human brains have abundant intra-cortical connections that link local neurons (solid lines) as well as longer-range 'short-cuts' that link neurons in different cortical regions (dashed lines). This structure supports both specialized information processing and distributed integrative processing of these specialized outputs. b) According to the II theory, abnormal connectivity patterns in psychopathic individuals may contribute to low network integration. Networks in psychopathic individuals may have fewer long-range connections between networks and more local connections, contributing to increased modularity and low efficiency of information transfer. These topographical properties would encourage segregated rather than integrative processing.

Impaired Integration, Emotion, and Cognition

Abnormalities in integrated functioning of neural systems have prominent implications for general affective and cognitive functioning (Gläscher et al., 2010). Cognition subsequently influences experience, and experience shapes the structures of neural systems throughout the lifespan (Sporns, Chialvo, Kaiser, & Hilgetag, 2004). This process results in a feedback loop between alterations in brain circuitry and information processing. Impaired integration at both neural and psychological levels would have cumulative effects over the course of development, setting the stage for abnormal patterns of information processing later in life.

Affective Deficits. The II framework conceptualizes the callousness characteristic of the psychopathic syndrome as a result of underdeveloped connectivity within emotion-related circuitry. With regard to emotion and intrinsic connectivity networks, the SN and DMN are most

critically involved in affective processing. The insula is a prominent hub in the SN and plays a role in mapping visceral states associated with emotional experience (Bechara, 2001). Decreased connectivity between the anterior and posterior regions of the insular cortex is associated with deficits in emotional and interoceptive awareness (Ebisch et al., 2011). In psychopathy, disconnections within this node may impair the integration of afferent homeostatic signal and emotional experience, obstructing the ability to develop and utilize 'somatic markers' (van Honk, Hermans, Putman, Montagne, & Schutter, 2002).

The II theory also proposes that coordination between neural systems impairs affective processing in psychopathy. Empathy is a general concept referring to the ability to mentally simulate others' mental states through cognitive or vicarious affective responses (Preston & De Waal, 2002). Empathy is not a unitary process but rather consists of bottom-up affective–perceptual and top-down cognitive evaluative components (see Cox et al., 2012; Fan, Duncan, de Greck, & Northoff, 2011; McDonald & Messinger, in press). Emotional empathy involves somatic, sensory, and motor representation of other peoples' mental states. In contrast, cognitive empathy results in less robust mirroring of other persons' mental and bodily states (Atique, Erb, Gharabaghi, Grodd, & Anders, 2011; Hillebrandt, Dumontheil, Blakemore, & Roiser, 2013; Nummenmaa, Hirvonen, Parkkola, & Hietanen, 2008). It requires higher cognitive functions such as metacognition and mentalizing (Bernhardt & Singer, 2012; Shamay-Tsoory, 2011). Cox and colleagues (2012) found that higher levels of affective empathy relative to cognitive empathy are associated with increased connectivity among social–emotional processing regions in the CoN and DMN, whereas higher relative dominance of cognitive aspects are associated with increased connectivity among social-cognitive and interoceptive regions associated with a frontotemporal network and the CoN. Abnormal connectivity within the CoN, coupled with

stronger frontotemporal activity relative to DMN activity, may disrupt affective responding and the ability to integrate emotional and cognitive processes in psychopathic individuals. To the extent that children with psychopathic traits have impaired integration, they would be less likely to engage in perspective taking than their nonpsychopathic peers. A lack of engagement in this process may inhibit the development of emotional and/or cognitive empathy (e.g., Lohmann & Tomasello, 2003). Furthermore, decreased perspective taking is associated with diminished empathy and prosocial behavior later in life (Farrant, Devine, Maybery, & Fletcher, 2012). In sum, impaired integration may make certain developmental processes and the integration of multi-component information particularly challenging for psychopathic individuals, resulting in a failure to engage in these processes. This in turn would impede the development of empathy-related systems for use in adulthood.

Impaired connectivity and associated deficits in the flexible recruitment of neural networks can also explain psychopathic individuals' performance on moral-reasoning tasks. In healthy individuals, the DMN is activated during moral decision-making tasks. Reduced activity in this system is associated with utilitarian responding. Moreover, this effect seems to be mediated by SN activity. In general, the SN modulates the activity of other large-scale networks and is responsible for switching between the DMN and executive control network (Chiong et al., 2013). Psychopathic individuals' tendency to respond in a utilitarian manner when engaged in moral dilemmas (see Koenigs, Kruepke, Zeier, & Newman, 2012) supports the idea that psychopathy involves impaired coordination of neural networks.

Difficulty integrating cognitive and affective components of information processing may prompt psychopathic individuals to use alternative cognitive strategies to process emotional information (e.g., Decety, Skelly, & Kiehl, 2013). Psychopathic individuals may engage in

cognitive processes at the expense of emotional processing due to difficulty switching between cognitive and emotional neural systems (see Chiong et al., 2013). One example of a behavior that might relate to this process is instrumental aggression. Proactive aggression is committed for a goal-directed purpose, and thus during engagement in instrumental aggression affective processing falls secondary to goal-directed behavior (Berkowitz, 1993; Dodge, 1991).

Importantly, the II model predicts that psychopathic individuals would show normal functioning when engaged in a task that deliberately activates select neural regions due to intact functioning of the FpCN. According to Meffert and colleagues (2013), psychopathy may be conceptualized as a reduced propensity, rather than an inability, for certain spontaneous brain activation. In terms of the II model, connectivity abnormalities may limit the breadth of spontaneous associative activation. In other words, processing not integral for a given task may be particularly effortful and thus psychopathic individuals would not expend the resources to engage in such processing. In fact, when the cost of processing secondary information outweighs the benefits and expected utility of integration, the processing of stimuli that are not goal-related and therefore not primary is suppressed (Kurzban, Duckworth, Kable, & Myers, 2013). In psychopathy, habitually suppressing the processing of distracting stimuli may become an automatic process (see Mauss, Bunge, & Gross, 2007), thereby further influencing the development of emotion-related systems. However, top-down mobilization of resources would alter connectivity (see Gordon, Stollstorff, Devaney, Bean, & Vaidya, 2011; Hwang, Shine, & D'Esposito, 2017) such that deliberate attempts to activate this circuitry would be successful (e.g., Meffert et al., 2013).

Attentional Dysfunction. The II framework suggests that psychopathic individuals' attentional dysfunction and failure to process peripheral information during goal-directed activity

is due to disrupted communication between attentional systems. One prominent model that highlights the importance of neural connectivity for normal attention is Corbetta and Shulman's (2002) concept of distinct attention systems in the human brain. Their work highlights the existence of anatomically and functionally distinct attention systems that require interhemispheric communication to interact and enable flexible attentional control (Corbetta et al., 2008; Vossel et al., 2012). A critical consequence of psychopathic individuals' proposed connectivity deficit would be to disrupt the integration of information from lateralized attention networks and influence the dynamics of top-down and bottom-up attentional processes (Carter et al., 2010). For this reason, we focus on the importance of lateralized attention systems to illustrate the importance of neural connectivity for attention-related deficits. Nevertheless, it is important to note that attention-related processes are broadly distributed and not purely shaped by lateralized circuits (see Shipp, 2004). Accordingly, problems in connectivity need not be lateralized to undermine the integrative processes associated with attention circuits operating throughout the brain.

According to Corbetta and Shulman (2002), the dorsal and ventral attention systems described above act together to balance goal-directed and stimulus-driven attention. The two attentional systems interact dynamically, such that dorsal frontoparietal regions suppress unnecessary reorienting by restricting ventral system activation, while the ventral attention network sends signals to the dorsal system alerting it to potentially important stimuli (Corbetta & Shulman, 2002; Meehan et al., 2017; Vossel et al., 2014). Signals from right TPJ, the hub of the ventral attention network, act as a "circuit breaker" for ongoing, goal-directed activity in the dorsal stream. These signals disrupt goal-directed activity and prompt a shift in attention toward salient stimuli (Corbetta et al., 2008).

Deficient interhemispheric connectivity in psychopathy may account for disrupted coordination of the dorsal and ventral attention networks[1] (He, Shulman, Snyder, & Corbetta, 2007). Specifically, abnormalities in the corpus callosum, a white matter tract that connects the two hemispheres of the brain (Doron & Gazzaniga, 2008; Funnell, Corballis, & Gazzaniga, 2000; Gazzaniga, 2000), might affect the coordinated functioning of the right and left hemispheres. Reciprocal callosal connections allow for the dynamic coordination of widespread brain processes and support synchronization of neural activity. The corpus callosum acts both as a channel for the transmission of information between the hemispheres as well as a means through which one hemisphere can modulate the activity of the other (Putnam, Wig, Grafton, Kelley, & Gazzaniga, 2008; Westerhausen & Hugdahl, 2008). Furthermore, it is crucial for unifying attentional focus and coordinating the attentional resources of the cerebral hemispheres (Banich, 1995a, 1995b; Posner & Dehaene, 1994), as well as facilitating conscious perception and the processing of sensory stimuli (Müller-Oehring et al., 2009). Structural and functional deficiencies may undermine the development of normal cognition and contribute to abnormal lateralization. Raine and colleagues (2003) found that psychopathic individuals have increased callosal length and reduced callosal thickness. Motzkin, Newman, Kiehl, and Koenigs (2011, unpublished data) also found reduced fractional anisotropy (a measure of white matter integrity) of the splenium of the corpus callosum in psychopathic individuals. Furthermore, there is evidence of abnormalities in right to left functional connectivity and of increased intracortical inhibition in the right hemisphere in psychopathic offenders (Hiatt & Newman, 2007; Hoppenbrouwers et al., 2014).

[1] As highlighted throughout the manuscript, other neurobiological systems also play a role in broadening attention (e.g., amygdala subnuclei: Moul, Killcross, & Dadds, 2012; septo-hippocampal system: Gorenstein & Newman, 1980) and would be undermined with abnormal connectivity.

When psychopathic individuals' attention is engaged in goal-directed (left hemisphere mediated) behavior, interhemispheric connectivity irregularities may preclude the conscious registration of signals from and stimuli processed by the right hemisphere. Thus, poor connectivity could result in a failure to integrate circuit-breaking signals from the ventral attention network, inhibitory signals, and emotion cues with goal-directed activity. Since those cues are not attended, they do not get access to working memory for further processing, which consequently affects internal attentional processes (Awh, Vogel, & Oh, 2006; Knudsen, 2007). Moreover, insufficient connectivity between these networks may result in inefficiency of the ventral attention system's "circuit-breaker" function. In other words, abnormal connectivity may lead to a reduction of automatic elaboration of stimulus significance, contributing to impaired detection of salient stimuli and deficient reorienting towards cues that are non-dominant. Consequently, the dominant response set and ongoing goal-directed behavior mediated by the bilateral dorsal attentional system are unlikely to be modified.

As with emotion processing, the II framework posits that psychopathic individuals' attentional abnormalities will become apparent in situations that involve the integrative processes. Consistent with this proposition, psychopathic individuals demonstrate performance deficits on neuropsychological testing that involve perceptual-motor integration, such as the Porteus Maze task and Trails B, but not those that involve more focal processing, such as Trails A and Visual Retention tasks (Hiatt & Newman, 2006). The II theory also predicts psychopathic dysfunction in situations necessitating the integration of bottom-up and top-down processes. During goal-directed (top-down) behavior, psychopathic individuals would be expected to show deficiencies in integrating contextual information into a cognitive set. In other words, the II model predicts that psychopathic individuals would have difficulty attending to salient or

unexpected stimuli when they are inconsistent with their top-down attentional set (see Corbetta & Shulman, 2002). Notably, this failure to integrate incongruent contextual information would confer an advantage on tasks that require focused selective attention, such as Flanker-type tasks. In these situations, psychopathic individuals would be expected to have superior performance relative to nonpsychopathic individuals. Additionally, failure to integrate contextual information would undermine the development of associative connections that support depth of processing.

The II theory suggests that psychopathic individuals' deficits in cognitive processing will be context-specific rather than general. With regard to emotion, it suggests that psychopathic individuals will show deficiencies in affective processing if emotion stimuli are multidimensional, secondary to the current attentional focus, or require linkage with memories. Conversely, to the extent that minimal integration is required, it follows that psychopathic individuals would exhibit normal performance. Thus, to the extent that there is a limited amount of information, the affective information is focal, and the information does not need to be integrated with peripheral information, psychopathic individuals should not show deficient affective processing. The fact that the II theory does not predict global impairment in a given process distinguishes it from other models.

Beyond Emotion and Attention.

***Language Anomalies*.** Research has shown that in addition to anomalous emotion and attentional processing, psychopathy may be characterized by aberrant language processing. In general, psychopathic individuals speak in a poorly integrated, contradictory, and unintelligible manner relative to nonpsychopathic individuals (Hare, 1998; Williamson, Harpur, & Hare, 1991). Moreover, psychopathic individuals demonstrate difficulty identifying abstract words compared to concrete words (Kiehl, Hare, McDonald, & Brink, 1999). Whereas nonpsychopathic

individuals tend to group words by connotations, psychopathic individuals group words by denotation and literal meaning (Hare, Williamson, & Harpur, 1988). The nonverbal gestures and word patterns of individuals high in psychopathy are distinctive (Gillstrom & Hare, 1988; Hancock, Woodworth, & Porter, 2013), and they show reduced cerebral asymmetry in linguistic tasks (e.g., Kiehl et al., 1999).

Psychopathic individuals' lack of conceptual integration while speaking and difficulty with abstraction may reflect a lack of coordination between neural systems involved in integrative linguistic processing. In general, language comprehension involves dynamic integration of perceptual and specialized linguistic information. Processing of linguistically complex words requires the synchronization and functional coupling of sensory and language-related networks (Fonteneau, Bozic, & Marslen-Wilson, 2015). Accordingly, a lack of synchronization would impair comprehension of complex words and sentences and increase processing demands. Similarly, abstract conceptual processing involves functional coordination of regions in the temporal parietal cortex. Deficient connectivity in this system would selectively impair abstract word processing (Skipper-Kallal, Mirman, & Olson, 2015). A lack of coordinated activity between these systems would result in the linguistic deficits seen in psychopathy.

Overall, the II perspective calls for a broadening of the conceptualization of psychopathy beyond an emotion- or attention-based disorder. In the words of Cleckley (1988), the difference between an individual with psychopathy and one with a "normal or integrated personality consists of an unawareness and a persistent lack of ability to become aware of what the most important experiences of life mean to others" (p. 371). The II model suggests that impaired neural integration impairs the automatic formation of associative context and proper orientation to others. In other words, it proposes that psychopathic individuals do not "[mean] to do wrong"

(Cleckley, 1988, p. 47); rather, a cumulative consequence of overlooking context and consequences while engaged in the process of living may contribute to an antisocial lifestyle.[2]

Important Considerations

Explaining the Psychopathy Factors

According to the II perspective, the specific symptoms of the psychopathic syndrome can be understood as inadequate integration of multi-component information. This framework can be used to understand the common and differentiating properties of the widely replicated factor structure of psychopathy. The II theory proposes that while the behavioral correlates of the two factors are different, they may reflect a shared integrative deficit and neural network (i.e., small-world) abnormalities. Factor 1 (the Interpersonal/Affective factor) may be characterized by a habitual response style of not actively integrating information due to its effortful nature; this proposition is supported by Philippi and colleagues' (2015) finding of a positive association between Factor 1 and reduced resting state activity in the FpCN. When effort is engaged, however, top-down control is intact (Krusemark, Kiehl, & Newman, 2016; Larson et al., 2013). Moreover, Factor 1 may be more strongly associated with failure to integrate signals between the DMN and SN, which may contribute to decreased introspection, perspective taking, and attention to affective cues (e.g., Chiong et al., 2013; Sevinc & Spreng, 2014). On the other hand, Factor 2 Impulsive/Lifestyle) may be uniquely associated cortico-striatal disconnection within the cingulo-opercular network as well as decreased functioning of cognitive control networks, contributing to impulsivity and irresponsibility (see Cohn et al., 2015).

Differentiating Neural Topography

[2] As noted by Hecht (2011), abnormal interhemispheric integration has significant implications beyond attentional processes. Indeed, it can account for central psychopathic deficits in emotion and inhibitory processing.

In recent years, ample evidence suggests that abnormal functional or structural connectivity between neural regions is associated with the pathophysiology of various forms of psychopathology (e.g., Liu et al., 2015; Müller et al., 2003; Rich et al., 2008; Stein, Simmons, Feinstein, & Paulus, 2007). Specifically, there is ample evidence that brain network organization is disrupted in psychological and neurological disorders and that psychopathology can be understood as variations in aberrant neural network dynamics (Buckholtz & Meyer-Lindenberg, 2012; Menon, 2011; Stam & Van Straaten, 2012). With this paradigm shift from modular to network-based conceptualizations of psychopathology, it is important to distinguish the defining neuropathology of each disorder.

Two mental disorders that have been reconceptualized in terms of network function have been autism and schizophrenia (Menon, 2011). Network models of autism propose that the brains of autistic individuals are overall less functionally connected, with globally reduced long-range connections between brain regions (Wass, 2011). Specific findings include reduced long-range synchronization in the FpCN during executive function tasks (Just, Keller, Malave, Kana, & Varma, 2012; Velazquez et al., 2009), excessive local connectivity in the FpCN (Courchesne & Pierce, 2005), and SN and DMN hypoactivity (Monk et al., 2009). Symptoms of schizophrenia are associated with reduced neural clustering, modularity, and corticocortical connectivity, structural and functional deficits in the SN, DMN, and FpCN (Hoffman & McGlashan, 2001; Mamah, Barch, & Repovš, 2013; van den Heuvel & Fornito, 2014). The combination of structural and functional abnormalities within and between different neural networks contributes to the unique symptoms of these disorders (Menon, 2011).

In contrast to autism and schizophrenia, brain topography in psychopathy appears more functionally preserved. According to the II theory, the FpCN in psychopathic individuals

develops normally due to the primacy of this network relative to others. Since the FpCN functions normally when engaged, psychopathic individuals do not show executive function deficits seen in the more severe forms of psychopathology. Moreover, goal-directed behavior is intact and not globally impaired due to the ability of top-down signals to engage other networks that may not come online automatically.

Another important factor distinguishing psychopathy from other forms of psychopathology is the development of antisocial sets. Contextual factors shape delinquency. Social variables such as family supervision and community violence have critical effects on the child's sociomoral development. Indeed, growing up in a disorganized or disadvantaged household or neighborhood is linked with engagement in illicit activities (Neumann, Barker, Koot, & Maughan, 2010; Patchin, Huebner, McCluskey, Varano, & Bynum, 2006). Internal factors contributing to engagement in antisocial activities include the desire to maintain a certain identity, to increase stimulation, to obtain material goods, and to achieve status (López-Romero & Romero, 2010). In contrast to individuals with other forms of psychopathology, contextual factors likely shape psychopathic individuals' appraisal processes in a unique way and foster the development of antisocial goals.

Overall, the II theory posits that the social environment interacts with a fundamental deficit in information integration in psychopathic individuals, contributing to the development of antisocial behavior. It specifically suggests that the integrative deficit that impedes elaboration and encourages sequential processing is unique to psychopathy, and this deficit combined with contextual factors will lead to the development of antisocial behavior (see Figure 1). Moreover, the II perspective predicts a reciprocal and cumulative relationship between brain structure and

function. In short, the II theory posits that it is the combination of brain topography and environmental influences that shape cognition, motivation, and behavior.

Areas for Future Research

Future research is necessary to unveil the underlying cause of impaired integration. For instance, studies should investigate whether early attention-related deficits in accommodating multi-channel information precede evidence of widespread deficits in neural connectivity. It could be that connectivity abnormalities are consequences rather than the cause of psychopathic traits and that they represent an acquired adaptation that aids in the ability to ignore distracting and effortful processing. Additional research should also explore the role of biochemical factors in mediating communication across the brain and how hormonal abnormalities may exacerbate connectivity abnormalities (see van Honk & Schutter, 2006). This work will be crucial in delineating the causal relationship between brain and behavioral abnormalities.

Furthermore, future work should examine the extent to which deliberate effort ameliorates functional abnormalities in brain systems. Research suggests that cognitive remediation can facilitate sustained changes in connectivity patterns (e.g., Keller & Just, 2009; Penadés et al., 2013). Indeed, Baskin-Sommers and colleagues (2015) provided preliminary evidence that cognitive remediation training can also mitigate cognitive dysfunction in psychopathic offenders. Evidence that psychopathic dysfunction can be attenuated by engagement in effortful processing has significant implications for therapeutic interventions.

Additionally, studies should utilize event-related fMRI during tasks in which peripheral information is dependent upon different circuitry than primary information. This technique allows for the assessment of the interactions between anatomically distinct brain regions during cognitive tasks (Rissman, Gazzaley, & D'Esposito, 2004). Previous work has suggested that a

lack of neural connectivity may be reflected in a lack of EEG synchrony in the gamma band (30-80 Hz). In tandem with abnormal brain data, reduced and/or delayed gamma activation would provide further evidence for abnormal connectivity patterns (see Belmonte et al., 2004).

It is important to highlight that the II theory has implications for specifying bio-behavioral mechanisms of psychopathic behavior. Consistent with Hare, Williamson, and Harpur (1988), the II framework proposes, "psychopathic individuals may be 'wired up' differently without being neurologically damaged or impaired" (p. 87). If this proposition were true, then it would be erroneous to assume that psychopathic and nonpsychopathic individuals use the same neural circuitry to complete all tasks. To assume that the same observed behavior between two groups is a consequence of the same underlying process in psychopathic and nonpsychopathic individuals is a logical fallacy, specifically a fallacy of the converse. Individuals with psychopathy might process information differently than nonpsychopathic individuals but not show impaired performance. Indeed, numerous studies have shown that psychopathic individuals can perform similarly to control participants yet show different patterns of neural activation (e.g., Harenski, Harenski, Shane, & Kiehl, 2010; Kiehl, 2006). The II theory predicts that psychopathic individuals rely on local activation within regions necessary for task performance rather than the coordinated and integrated functioning of widespread neural systems.

Limitations

There are several restrictions to using the proposed methods to delineate network properties in psychopathy. One drawback is that the validity of identified networks depends on valid node selection; arbitrarily defined sampling grids do not provide theoretically acceptable estimates (Bullmore & Sporns, 2009; Power et al., 2011; Rubinov & Sporns, 2010; Spreng et al., 2013). Additionally, the nature of nodes and their connections largely determines the

interpretation of network organization (Rubinov & Sporns, 2010). Thus, caution is necessary in interpreting network properties since functional connectivity (or lack thereof) does not imply structural connectivity. An additional limitation of graph theory is that there are numerous measures of graph topology, but it is unknown which measures are most appropriate for neural network (Bullmore & Sporns, 2009).

A limitation of using correlations to derive functional neural networks from fMRI data is that transitivity of correlations (e.g., when there is a tie from a to b, and also from b to c, then there is also a tie from a to c) could contribute to an artifactual increase in the clustering coefficient. However, this problem can be remedied by using stricter correlation measures such as partial directed coherence (Sporns et al., 2004). Moreover, graph analysis requires large graph size for valid results (e.g., $N > 200$), and comparison of empirical networks requires precision so as to not over-rate or under-rate functional connections (Van Wijk, Stam, & Daffertshofer, 2010).

Despite these limitations, advances in neuroscience and modern network theory offer promising framework through which psychopathology can be understood by considering neuroimaging data (Bullmore & Sporns, 2009). Future research linking the parameters of brain topography to cognition, affective, and behavior could allow for identification of a psychopathy endophenotype based on brain network properties.

Conclusion

In the domain of psychopathy, prevailing models describe an artificial dichotomy between affective and attention components of psychopathic dysfunction, contributing to a gap in the literature regarding the mechanistic underpinnings of the disorder. Although dominant models have spawned great progress in the field, taken in isolation each theory has weaknesses:

dominant emotion-centric models of psychopathy are simultaneously too broad in their failure to account for the context-dependent nature of psychopathic individuals' emotion-processing impairments and too narrow in their failure to acknowledge psychopathic individuals' non-affective information processing deficits. Attention-based models account for the situation-specific nature of psychopathic dysfunction and accommodate deficits relating to affective processing, yet these models are not well linked to a neurobiological mechanism. The goal of the current proposal was to outline a model of psychopathy that explains the syndrome while bridging the gap between affective and cognitive models by providing a common underlying mechanism.

Unlike existing models, the II theory can account for both affective and attention deficits in psychopathy, as well as the situational nature of these deficits. The II framework uniquely contributes to the scientific literature by providing an integrative account of the psychopathic syndrome. This framework provides a mechanism for affective and cognitive dysfunction in psychopathy that accommodates neurobiological data regarding the diffuse nature of brain abnormalities. Moreover, it outlines the implications of impaired integration for information processing on a psychological level. The II perspective makes specific, testable predictions based on an analytic approach that provides a global account of neural functional architecture. Accordingly, it makes a significant contribution to the understanding of the psychopathic syndrome. In doing so, we have shown that we can integrate previously divergent literature on psychopathy into a unified framework. Importantly, this work generates novel questions for future research on psychopathic dysfunction.

CHAPTER 2
INTEGRATION CONSTRAINTS & INFORMATION PROCESSING STYLE IN PSYCHOPATHY: EVIDENCE FROM THE SIMULTANEOUS-SEQUENTIAL PARADIGM

Content from this chapter was published as: Hamilton, R. K. B., & Newman, J. P. (2018). Information processing capacity in psychopathy: Effects of anomalous attention. *Personality Disorders: Theory, Research, and Treatment, 9*(2), 182-187.

Information processing involves a cascade of neurobiological events that transforms sensory input into neural representations and subsequent behavior. Failure at any step in the processing sequence would therefore influence the content and quality of perceived information and thus the quality of one's behavioral response (Goldstein, 2010). Psychopathy is a clinical syndrome typified by socially and legally significant antisocial behavior. High levels of psychopathic traits are predictive of engagement in criminal behavior, violent offending, and high rates of recidivism (Hare & McPherson, 1984). Accordingly, specifying the information processing abnormalities of psychopathic individuals may provide crucial insight into their behavioral dysfunction.

Even before its formalization as a diagnostic entity, the psychopathy construct has long been recognized in historical literature and lore (Buzina, 2012). A commonality across both early and modern conceptions is the central role of moral degeneracy and impulsive, aggressive behavior. Deficient affective experience is traditionally considered the hallmark of the psychopathic syndrome (e.g., Blair, Mitchell, & Blair, 2005; Lykken, 1995).

Despite the centrality of affective dysfunction in psychopathy, research suggests that the construct is associated with broader cognitive deficiencies, suggesting that information processing abnormalities may subsume emotion deficits. These findings have spawned numerous theories positing diverse information processing deficits in psychopathy (e.g., Baskin-Sommers, Curtin, & Newman, 2011; Kosson, 1996; Patterson & Newman, 1993). Nevertheless, elucidation

of the dysfunction that accurately predicts the full range of information processing strengths and weaknesses in psychopathy has proven elusive (see **Chapter 1** for a review of prevailing emotion and cognitive models and their shortcomings).

Hamilton and colleagues (2015; see **Chapter 1**) recently proposed the Impaired Integration (II) theory of psychopathy, a framework that makes more explicit the neurological underpinnings and dysfunctional neurocognitive processes in psychopathy. The II theory leads to a variety of novel, testable predictions that concurrently build on and deviate from those from previous etiological theories. The II theory proposes that the fundamental deficit in psychopathy involves difficulty rapidly integrating multicomponent information. As a result, it is more difficult for psychopathic individuals to encode multiple stimuli or multiple aspects of stimuli simultaneously, bind spatially and temporally discontiguous information, and interpret perceptual input based on associative connections in long-term memory. Moreover, stemming from these limitations, psychopathic individuals have reduced attentional capacity for active encoding, maintenance of relevant stimuli, and integrating events and their contexts of occurrence with existing schemas. Thus, a central tenet of the II framework is that simultaneous processing capacity will be relatively impaired in psychopathy; accordingly, it predicts that psychopathic individuals will benefit from sequentially-presented multicomponent information.

One paradigm that is especially pertinent for examining differences in information processing capacity is the Simultaneous-Sequential paradigm. In this paradigm, participants are instructed to make judgments about briefly presented stimuli that are either presented simultaneously (i.e., all at once) or sequentially (i.e., subsets are presented one-at-a-time). Accordingly, the critical manipulation in this task is the number of simultaneously presented stimuli. Processing capacity limits are task-dependent and reflect limitations of perceptual

encoding and/or sematic categorization (Scharff, Palmer, & Moore, 2011b). These limits influence whether humans process sensory input simultaneously or sequentially on a given task. Typically, there is a selective advantage for sequential processing on tasks with low target-distracter discriminability (i.e., greater difficulty; Scharff, Palmer, & Moore, 2011a). The Simultaneous-Sequential paradigm was originally developed to study the rate of information processing in visual perception (Eriksen & Spencer, 1969) and since its conception has been used primarily to answer questions regarding visual capacity limitations (see Scharff et al., 2011a).

The current study sought to examine psychopathy-related differences in processing capacity for simple visual stimuli; more specifically, it aimed to elucidate the extent to which psychopathy is preferentially associated with sequential relative to simultaneous processing. The use of stimuli with intermediate target-distracter discriminability enabled us to accommodate the abilities of our sample while maximizing task sensitivity to individual differences in processing style. Based on the assumption that psychopathy is characterized by information processing restrictions that foster the sequential processing of information, we hypothesize that visual processing accuracy and response time in psychopathy will be moderated by information presentation style. Specifically, we predict that psychopathic individuals will have lower accuracy and slower response times on simultaneous trials and higher accuracy and faster response times on sequential trials.

Methods

Participants

Participants consisted of 87 Caucasian male inmates ages 20 to 55 (M = 31.03, SD = 8.22) from a medium-security prison in central Wisconsin. To be included in the study,

participants had to be between 18 and 55 years old, free of a history of psychosis or bipolar disorder, not currently taking psychotropic medication, and have an IQ score of 70 or greater.[3] All participants provided written informed consent according to procedures approved by the University of Wisconsin – Madison Human Subjects Committee. On the first day of the study, interviewers conducted semi-structured life history interviews with the inmates. This interview included questions on childhood, education, and occupational, interpersonal, and legal histories. Interviewers then reviewed institutional files to corroborate information provided during the interviews. The combination of interview and file information was used to rate psychopathy using to Hare's (2003) Psychopathy Checklist-Revised (PCL-R). One participant was excluded from analyses due to low accuracy on the experimental task (less than 60% correct across all trial types).

Psychopathy Checklist-Revised (PCL-R; Hare, 2003). The PCL-R consists of 20 items that are rated according to the degree to which a characteristic is present (significantly = 2, moderately = 1, not at all = 0). In the present sample, scores on this measure ranged from 10 to 34, with a mean of 22.51 (SD = 6.03). Interrater reliability (intraclass correlation) for PCL-R total score, based on six dual ratings, was .96.

Materials

Stimuli. Stimuli were 360 sets of four letters written in white font embedded in Gaussian noise patches presented on a gray background. Each set of letters consisted of three distractor stimuli (A, C, F, L, M, N, P, Q, T, V, W, X, or Z) and one target stimulus (B, G, H, R, or Y) that varied by block.

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[3] IQ was estimated using the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981). Psychiatric history was assessed using the *Structured Clinical Interview for Diagnostic and Statistical Manual Disorders* (First, Gibbon, Spitzer, & Williams, 1997). These measures were administered when participants first enrolled in the study.

Experimental Task. The simultaneous-sequential paradigm consists of a visual search task in which four stimuli, one of which is the predefined target, are presented either simultaneously or sequentially. In the current task, participants were presented with a target letter at the beginning of each block. Following the target display, participants saw a fixation cross that was followed by the presentation of four letters presented at once or divided equally in two separate frames. Participants were instructed to indicate the location of the target letter as quickly and accurately as possible.

The task consisted of 75 practice trials and 720 experimental trials. The first 45 practice trials consisted of 15 simultaneous, 15 sequential, and 15 repeated trials presented in isolated blocks so that participants could become familiar with each condition. On simultaneous trials, all letter stimuli were displayed in one 100 ms frame. On sequential trials, the stimuli were divided equally between two 100 ms frames that were separated by a 1 s fixation frame. On repeated trials, all four stimuli were presented simultaneously twice in two 100 ms frames that were separated by a 1 s fixation frame (see Figure 3). The repeated trials were included based on Scharff et al.'s (2011b) extended paradigm to address capacity processing models. Since this condition is not related to the present hypotheses, results pertaining to this condition are discussed in supplemental analyses.[4] Once all stimuli were presented, participants saw a cue screen that read 'Location?', at which time they were indicate the location of the target letter by pressing one of four designated buttons on a keyboard. The 'Location?' screen remained until participants responded or 1 s had elapsed. For the remaining 30 practice trials, all trial types were intermixed. To ensure a lack of practice effects on experimental trials, a unique target letter (U)

[4] Supplemental analyses testing predictions derived from perceptual capacity models will be publicly available on Rachel K. B. Hamilton's ResearchGate profile. Although space limitations preclude presentation of the full results, analyses demonstrated that psychopathy was uniquely associated with a serial processing style.

was used in practice sessions. The 720 experimental trials were divided into ten blocks (72 trials each) of randomized trial types; each block was separated by a 10-s break. The duration of the practice and experimental trials was approximately 40 minutes.

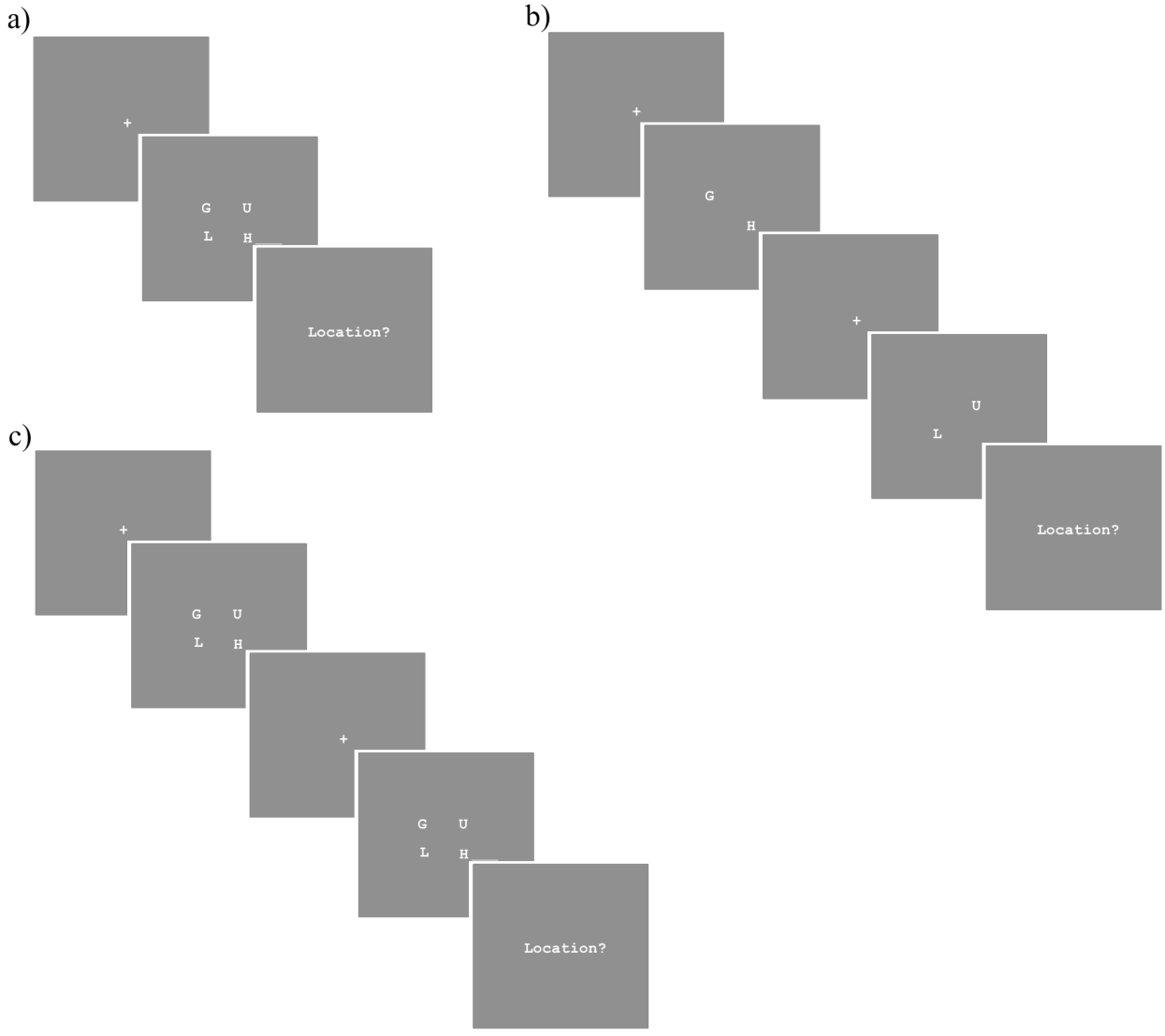

Figure 3. Trial structure and experimental conditions of Simultaneous-Sequential task. On each trial, participants saw a series of four letters, one of which was a predefined target. Letter presentation varied by condition. (a) Simultaneous trials: all four stimuli were displayed in one 100 ms frame. (b) Sequential trials: stimuli were divided equally between two 100 ms frames separated by a 1 s fixation. (c) Repeated trials: all four stimuli were displayed in two 100 ms frames separated by a 1 s fixation.

Data Analysis

The goal of the current study was to examine psychopathy-related differences in perceptual processing capacity. To test for psychopathy-related differences in performance we used two general linear models (GLMs) with trial type (sequential vs. simultaneous) and block (early vs. late) as repeated measures, *z*-scored PCL-R total score[5] as a continuously distributed between-subject factor, and accuracy and response time as respective dependent variables. To control for differences in overall performance, we included *z*-scored averaged simultaneous-sequential accuracy and response time as continuously distributed covariates in the respective analyses.[6, 7]

To characterize significant interaction effects, we used point estimates generated from the GLMs to estimate the conditional effect (i.e., simple slope) of trial type on mean accuracy for individuals high and low in psychopathy (1.25 SD above and below the sample mean PCL-R total score, respectively[8]). We repeated these analyses using response time as the dependent variable. Partial eta squared values are included as measures of effect size.

[5] Although alternative models postulate the existence of unique etiological contributions for diverse symptom groups (i.e., factors) in psychopathy (see Hare & Neumann, 2008), the II theory under investigation does not. Accordingly, we report results for PCL-R total scores only.

[6] Given our interest in quantifying psychopathy-related effects rather than effects related to general externalizing psychopathology, we re-ran all analyses substituting Externalizing Spectrum Inventory (ESI) total score for PCL-R total score and with ESI score as a covariate. These results revealed no significant associations between externalizing and the variables of interest, suggesting that the reported results represent psychopathy-related differences in processing capacity rather than differences due to the externalizing dimension.

[7] To ensure that the reported effects were not confounded by IQ, we re-ran analyses with this variable as a covariate. The inclusion of this variable did not significantly influence results.

[8] A SD of 1.25 was used to compute accuracy and response time point estimates so that high psychopathy predicted values would correspond to values for an individual with a PCL-R score over 30.

Results

Covariate-adjusted mean accuracy by trial type and estimated mean values for individuals high and low in psychopathy are presented in Table 2. Table 3 provides the inter-correlations among the study variables.

Table 2. *Covariate-Adjusted Mean Accuracy and Response Time by Trial Type*

	Condition mean		**High psychopathy**		**Low psychopathy**	
	ACC	**RT**	**ACC**	**RT**	**ACC**	**RT**
SEQ	.86 (.01)	584.72 (5.10)	.88 (.02)	567.05 (8.21)	.83 (.02)	602.39 (8.21)
SIM	.81 (.01)	792.23 (5.10)	.78 (.02)	809.90 (8.21)	.83 (.02)	774.55 (8.21)

Note: Psychopathy scores were obtained using the Psychopathy Checklist-Revised (PCL-R; Hare, 2003). Since we analyzed psychopathy continuously rather than using an extreme-group design, the values presented are point estimates (i.e., estimated using regression analyses) for low and high psychopathy points (1.25 SD below and above the sample mean PCL-R total score, respectively) on the distribution. Numbers in parentheses are standard errors. SEQ = performance on sequential trials; SIM = performance on simultaneous trials.

Table 3. *Bivariate Correlations (r values) between PCL-R Total Score and Performance Variables by Trial Type*

	PCL-R	SEQ ACC	SIM ACC	SEQ RT	SIM RT	Diff ACC	Diff RT
PCL-R	-						
SEQ ACC	.16	-					
SIM ACC	-.12	.27*	-				
SEQ RT	-.04	.17	-.38**	-			
SIM RT	.18	.31**	-.48**	.71**	-		
Diff ACC	.22*	.57**	-.63**	.45**	.66**	-	
Diff RT	-.29**	-.20	.14	.38**	-.39**	.28**	-

Note. The included difference scores represent the difference in accuracy and response time on sequential trials compared to simultaneous trials. These variables allow for the characterization of the magnitude of the association between study variables and the differential performance on simultaneous vs. sequential trials. PCL-R = psychopathy total score; SEQ ACC = accuracy on sequential trials; SIM ACC = accuracy on simultaneous trials; SEQ RT = response time on sequential trials; SIM RT = response time on simultaneous trials; Diff ACC = the difference in sequential trial accuracy relative to simultaneous trial accuracy, computed by subtracting accuracy on simultaneous trials from accuracy on sequential trials; Diff RT = the difference in response time to sequential trials relative to that on simultaneous trials, computed by subtracting response time on simultaneous trials from response time on sequential trials; * = $p < .05$; ** = $p < .01$

Psychopathy - Accuracy Analyses

There was a significant main effect of trial type ($F(1, 84) = 6.62$, $p = .01$, $\eta_p^2 = .07$), such that accuracy on sequential trials was significantly higher than accuracy on simultaneous trials. Moreover, there was a significant block effect ($F(1, 84) = 74.23$, $p < .01$, $\eta_p^2 = .47$), with participant accuracy increasing over the course of the task. The main effect of PCL-R score on task accuracy was not significant ($F(1, 85) = .16$, $p = .69$, $\eta_p^2 < .01$). The main effect of trial type was moderated by psychopathy level, $F(1, 84) = 4.35$, $p = .04$, $\eta_p^2 = .05$. Follow-up analyses estimating the effect of trial type for individuals high and low in psychopathy indicated that high psychopathy scores were associated with significantly greater accuracy on sequential trials relative to simultaneous trials ($F(1, 84) = 10.45$, $p < .01$, $\eta_p^2 = .11$). In contrast, accuracy across trial types did not differ for individuals with low psychopathy scores, $F(1, 84) < .01$, $p = .98$, η_p^2

< .01 (see Figure 4). There was no interactive effect of block and PCL-R score on accuracy ($F(1, 84) = 1.60$, $p = .21$, $\eta_p^2 = .02$), suggesting that changes in accuracy across block was comparable across psychopathy level. The three-way interaction between psychopathy score, block, and trial type was also not significant, $F(1, 84) = 2.12$, $p = .15$, $\eta_p^2 = .03$.

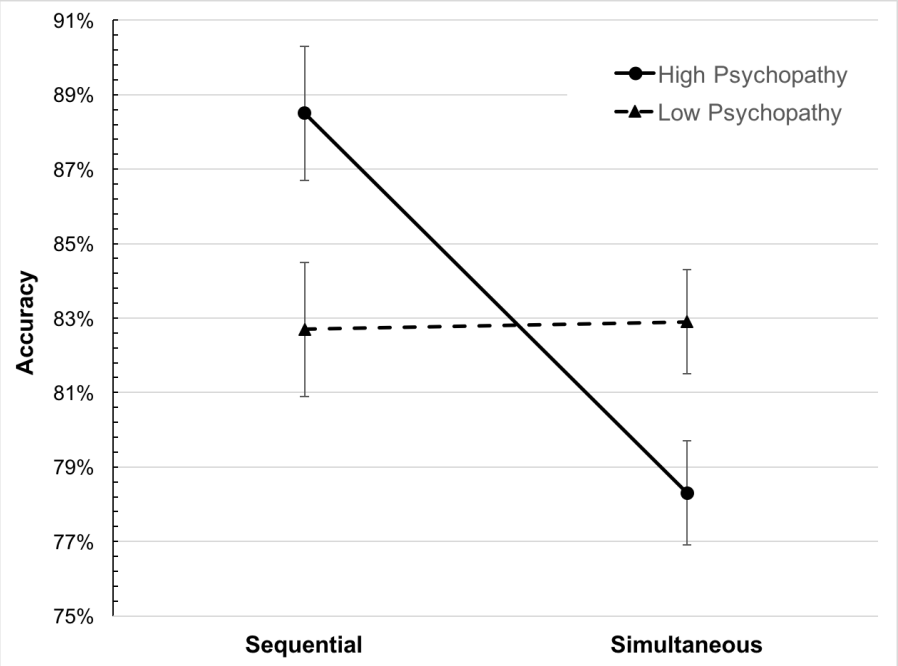

Figure 4. Accuracy by trial type for individuals high and low in psychopathy. Predicted values (point estimates) for task accuracy are depicted as a function of psychopathy level and trial type. High and low psychopathy levels are defined as the sample mean PCL-R score plus or minus 1.25 SD, respectively. Error bars represent standard error.

Psychopathy - Response Time Analyses

There was a significant main effect of trial type on response time ($F(1, 84)$ 413.78, $p <$ .01, $\eta_p^2 = .83$), such that responses on sequential trials were significantly faster than on simultaneous trials. There was also a significant block effect ($F(1, 84) = 50.57$, $p < .01$, $\eta_p^2 =$ .38), with mean response time decreasing over the course of the task. There was no significant main effect of PCL-R score on response time ($F(1, 85) =$ .65, $p = .42$, $\eta_p^2 < .01$). Consistent with hypotheses, the main effect of trial type was qualified by psychopathy, $F(1, 84) = 7.54$, $p = .01$,

η_p^2 = .08. Subsequent analyses demonstrated that although the effect of trial type on response time was significant among individuals with low psychopathy scores (F(1, 84) = 109.90, p < .01, η_p^2 = .57), this effect was larger among high psychopathy participants, F(1, 84) = 218.74, p < .01, η_p^2 = .72 (see Figure 5). Neither the block by psychopathy interaction (F(1, 84) = .05, p = .83, η_p^2 < .01) nor the three-way interaction between psychopathy score, block, and trial type was significant, F(1, 84) = 1.18, p = .28, η_p^2 = .01.

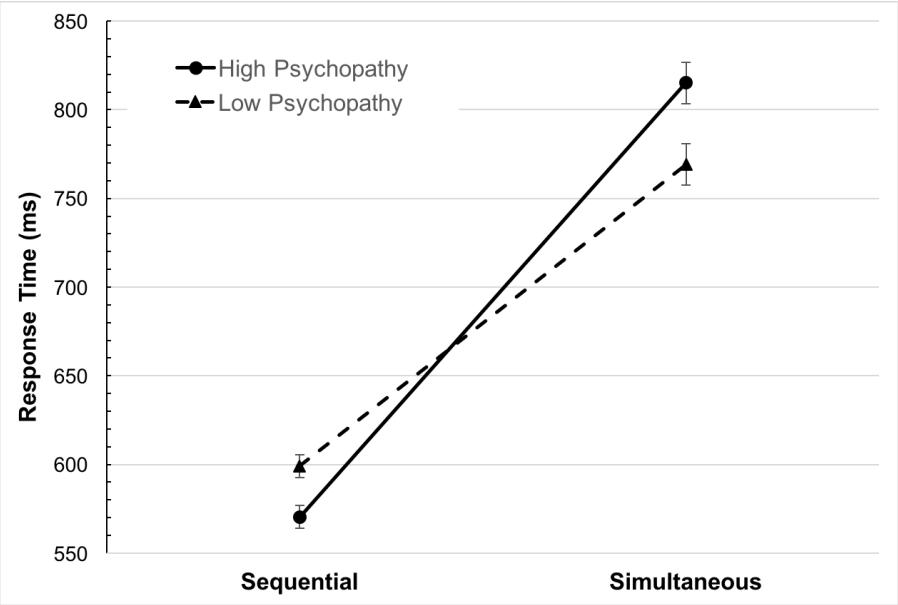

Figure 5. Response time by trial type for individuals high and low in psychopathy. Predicted values (point estimates) for response time are depicted as a function of psychopathy level and trial type. High and low psychopathy levels are defined as the sample mean PCL-R score plus or minus 1.25 SD, respectively. Error bars represent standard error.

Discussion

The primary goal of the current study was to test the hypothesis that psychopathic individuals process multicomponent visual information in a serial versus parallel fashion. The II theory predicts that psychopathy is characterized by an information-processing deficit that constrains the amount of information that can be processed in parallel. As a result, psychopathic individuals are expected to be relatively impaired in processing simultaneously presented stimuli and to benefit preferentially from the serial presentation of multicomponent information. As

predicted, psychopathy was associated with poorer performance on simultaneous trials relative to sequential trials.

The presented results are consistent with a growing body of evidence indicating that psychopathy is characterized by information processing anomalies that extend beyond emotion. According to the II perspective, the affective and disinhibitory symptoms seen in psychopathy are a downstream consequence of difficulty rapidly integrating components of multidimensional sensory stimuli. That is, inefficient processing undermines psychopathic individuals' use of affective and inhibitory cues, especially under time limitations.

The II theory is a useful framework for understanding psychopathic abnormalities within the context of the Research Domain Criteria (RDoC; see Cuthbert, 2014) initiative at The National Institute of Mental Health. It makes predictions at the neurobiological, cognitive, and behavioral levels, with the current study focusing on the cognitive domain. Other investigations are specifying cognitive correlates in psychopathy by examining how attention influences perception and memory, in addition to testing predictions of neural modularity.

Overall, the current study offers explicit support for the theoretical premise of the II framework (i.e., relatively impaired simultaneous processing), and outlines a promising avenue for future research. Although further replication is needed to verify the reliability of the current findings, the proposal that psychopathy is characterized by a deficit in the ability to rapidly process simultaneously-presented information has potentially important implications for therapeutic interventions. Specifically, if future evidence associates this deficit with symptoms of psychopathy, treatments focusing on general information processing constraints may be effective in reducing psychopathic disinhibition.

Limitations

Before concluding, it is important to note that the current study was not designed to differentiate the II theory and attention bottleneck (AB) model, and the findings are consistent with both models. The AB model holds that psychopathy is characterized by an attentional bottleneck that impedes effective response modulation, precluding the processing of secondary information that is incongruent with a primary focus of attention (see Newman & Baskin-Sommers, 2011). In the current task, all stimuli are perceptually similar and related to an explicit, primary (i.e., top-down) focus of attention. Although the perspectives are overlapping, the II theory is more specific and direct in generating the *a priori* prediction addressed in this report.

Another potential concern is that the correlations between psychopathy and performance within conditions are small and generally non-significant (see Table 3). However, the sensitivity and validity of these measures are more readily apparent through examination of the differential effects trial type on varying psychopathy levels. The differences in accuracy and response time to sequential relative to simultaneous trials are captured through difference scores in which simultaneous trial performance is subtracted from sequential trial performance. For this reason, we have reported these difference scores in Table 3.

Another potential limitation relates to the specificity of the studied population. The present sample consisted exclusively of European American male prisoners. The homogeneity of the sample limits the generalizability of the presented findings. While prior research suggests that the presence of specific laboratory correlates is not consistent across sexes (Vitale, Maccoon, & Newman, 2011) or racial groups (see Smith & Lilienfeld, 2015) studying psychopathy in diverse populations is important for complete understanding of the construct.

Thus, future research should explore perceptual processing capacity in psychopathic individuals of different sexes and from different racial backgrounds.

Conclusion

Overall, the current study provides novel support for the II theory. The finding that psychopathic individuals appear to benefit from the sequential presentation of information suggests that strategies for reducing psychopathic behavior should accommodate a serial processing style. Further research is needed to characterize the neurobiological correlates of impaired processing of simultaneously presented information. Neuroimaging studies will be useful in specifying the mechanism underlying this deficit. Moreover, future research should focus on the developmental trajectory of information processing deficits in psychopathy. Evidence of this deficit in children with psychopathic traits could enable early identification and remediation of potentially maladaptive information processing styles. Overall, research on the neurobiological underpinnings of cognitive abnormalities in psychopathy is necessary for developing a more precise understanding of and more effective treatments for psychopathic individuals.

CHAPTER 3
COGNITIVE CONSEQUENCES OF IMPAIRED INTEGRATION: STIMULUS ENCODING AND REPRESENTATIONAL CAPACITY IN PSYCHOPATHY

Content from this chapter is currently under review: Hamilton, R. K. B., & Newman, J. P. (2018). *Without a Trace: Impaired Stimulus Encoding and Representational Capacity in Psychopathy*. Manuscript submitted for publication.

Psychopathy is a clinical construct characterized by a collection of emotional, interpersonal, and behavioral features, including shallow affect, egocentrism, and antisociality (Hare, 2003; Hare & Neumann, 2005). Moreover, psychopathic criminals consume a disproportionality large number of legal resources and are at a higher risk of criminal and violent reoffending than non-psychopathic offenders (Hemphill, Templeman, Wong, & Hare, 1998; Kiehl & Hoffman, 2011). While psychopathy affects approximately one percent of Americans, roughly twenty percent of incarcerated individuals receive this classification (Hare, 2011; Ogloff, 2006). Given the interpersonal and social costs associated with this disorder, understanding of the mechanisms and processes underlying psychopathy is critical. Proper characterization of the underpinnings of the psychopathic syndrome will enable more accurate diagnosis and the development of effective interventions for psychopathic disinhibition.

Poor inhibitory control is commonly regarded as the principle problem of the psychopathic syndrome (Lykken, 1995). Psychopathic individuals consistently demonstrate poor passive avoidance, whereby they fail to inhibit behavior that is maladaptive or was previously punished (Masui & Nomura, 2011; Newman, Patterson, & Kosson, 1987; Newman, Widom, & Nathan, 1985). Nevertheless, they inhibit as well as non-psychopathic individuals when this requirement is made explicit or particularly salient. Similarly, psychopathic individuals demonstrate deficits in perspective-taking and empathic responding when not explicitly instructed to engage in these tasks (Drayton, Santos, & Baskin-Sommers, 2018; Keysers &

Gazzola, 2014; Meffert et al., 2013). Such findings suggest that psychopathic disinhibition may reflect a problem using the cognitive-affective states that automatically modulate the behavior of others. More specifically, psychopathy may reflect a reduced capacity to engage in typically automatic cognitive processes that moderate behavior (Patterson & Newman, 1993).

The generation and use of cognitive representations are critical for the automatic regulation of behavior (Bechara, Damasio, Tranel, & Damasio, 1997). Cognitive (or mental) representations refer to abstract, internal data structures encoded in the brain that mediate the perceptual experience of the external world (Crane, 2016; Palmer, 1977; Pitt, 2017; Smith, 1998; see also Zhang & Norman, 1994). They include unimodal sensory representations (percepts and body states) as well as multisensory, associative internal representations formed from the integration of individual sensory traces and previously generated representations stored in memory (Carlston & Smith, 1996; Jones, Ross, Lynam, Perez, & Leitch, 2011; Shaw, 2015; Talsma, 2015). The formation and maintenance of cognitive representations are critical neurocognitive abilities, as these representations regulate behavior in a relatively automatic fashion (Bargh, Gollwitzer, Lee-Chai, Barndollar, & Trötschel, 2001; Pearson, & Kosslyn, 2015; Pearson, Naselaris, Holmes, & Kosslyn, 2015; Salzman & Fusi, 2010). They guide thought, behavior, and emotion and are essential for learning, memory, reasoning, and the manipulation of information that enables successful interaction with the world (Pearson et al., 2015).

One theory that has linked cognitive representations to psychopathic dysfunction is the Somatic Marker Hypothesis (Damasio, Everitt, & Bishop, 1996). Somatic markers are signals from emotional representations of past experiences with reward and punishment that imbue behavioral options with affective significance. By ‘emotionally marking’ stimuli, somatic markers create stimulus-response associations, thereby promoting approach or avoidance

behavior in a relatively automatic manner. As associative mental representations, emotional representations involve the activation of both perceptual (somatosensory) processes as well as top-down knowledge (Barsalou, 1999; Barsalou, Simmons, Barbey, & Wilson, 2003; Damasio et al., 1996; Kragel & LaBar, 2016; Prinz, 2006). Without emotional representations, psychopathic individuals would be highly vulnerable to maladaptive dysregulated behavior (Damasio et al., 1996). Despite speculation regarding the relationship between psychopathy and representational abilities, this relationship has yet to be investigated outside the context of emotion (Hiatt et al., 2004; Newman et al., 1997; Sadeh & Verona, 2008; Wallace, Vitale, & Newman, 1999; Zeier et al., 2009). That is, it is important to determine whether such deficits reflect an emotion-specific problem or a more general problem using mental representations.

More recently, Hamilton and colleagues (2015) proposed the Impaired Integration (II) theory, a framework that offers a parsimonious account of cognitive and affective deficits in psychopathy and yields new predictions regarding the psychological mechanism underlying psychopathic dysfunction. According to this theory, psychopathy is characterized by difficulty rapidly processing and integrating multidimensional information. Due to its purported negative effects on simultaneous processing, this deficit is thought to undermine the formation and use of relevant associations that normally guide behavior. In other words, it might contribute to less efficient associative processing. In short, the proposed deficit is thought to impair parallel processing and thus the integration of information (e.g., multiple perceptual components, previously encoded information) that is vital for the formation and use of mental representations, including multicomponent perceptual information and previously coded information.

To examine the II theory's fundamental prediction that psychopathy is preferentially associated with sequential relative to simultaneous processing, Hamilton and Newman (2016; see

Chapter 2) used the Simultaneous Sequential paradigm with incarcerated offenders with varying levels of psychopathy. Based on the expectation that psychopathy is characterized by information processing limitations that hamper simultaneous processing and foster sequential processing, the researchers hypothesized that the speed and accuracy of visual processing in psychopathy would be moderated by information presentation style. Results were consistent with this prediction, demonstrating that psychopathy was uniquely associated with better performance on trials involving the serial (versus simultaneous) presentation of multicomponent information. This study was the first to demonstrate that psychopathy is distinguished by a unique information processing style involving superior sequential relative to simultaneous processing.

To date, research has not linked this proposed mechanism to information processing abnormalities in psychopathy. In other words, it is currently unclear how relatively poor simultaneous processing of multicomponent information affects basic information processing, including the ability to encode and make use of mental representations. A reduced capacity to acquire, store, and utilize new or previously obtained information would preclude the use of that information for decision-making. Indeed, such processes are critical for the inhibition of maladaptive responses (Aron, 2011; Pearson et al., 2015).

The goal of the current investigation is to explore psychopathy-related differences in the ability to form and utilize mental representations at various points in the information processing stream. In this study, we used a non-affective, change detection task that presented participants with multiple channels of information (i.e., different perceptual stimuli comprised of easily identifiable images of varying shape and color located at distinct spatial locations) to assess the formation and maintenance of mental representations. By using a non-affective task, the current study can explore information processing independent of emotion. Moreover, by using a task

designed to probe perceptual representational capacity, this study can probe for psychopathy-related differences in processes required for the formation and maintenance of mental representations. Thus, the current investigation can provide a stronger mechanistic understanding of cognitive limitations associated with psychopathy. Based on the II theory's prediction that an integrative deficit restricts rapid simultaneous processing of multicomponent information and leaves fewer attention resources available to encode a scene, we predict that high psychopathy individuals will show reduced representational capacity throughout the information-processing stream. More specifically, we predict that there will be a psychopathy-related reduction in representational capacity during early, sensory-perceptual encoding[9] (Stage 1) as well as later information processing stages (Stages 2 and 3), which necessarily depend upon early perceptual encoding of the information. This hypothesis contrasts with the prediction that psychopathy is associated with a selective late stage, working memory deficit (Stage 3; Gorenstein, 1991).

In addition to testing our primary hypothesis, we administered an embedded identification task following change trials to probe the visual detail (i.e., resolution) of mental representations (Sligte, Vandenbroucke, Scholte, & Lamme, 2010) to further clarify psychopathy related effects. Furthermore, we conducted supplementary analyses to evaluate the specificity of the deficit and examine the unique influences of interpersonal/ affective and impulsive/antisocial components of the psychopathy construct on representational capacity.

[9] The suspected encoding deficit is presumed to reflect attentional problems and to manifest when attention is taxed.

Methods

Participants

Participants consisted of 77 Caucasian male inmates ages 20 to 54 (M = 31.87, SD = 7.94) from a medium-security prison in central Wisconsin. To be included in the study, participants had to be between 18 and 55 years old, free of history of psychosis and bipolar disorder, not currently taking psychotropic medication, and have an IQ score of 70 or greater. Individuals meeting the inclusion criteria were invited to participate in an ongoing study. All participants provided written informed consent according to procedures approved by the University of Wisconsin-Madison Human Subjects Committee. On the first day of the study, inmates were called to a private office and completed a semi-structured life history interview. This interview included questions on childhood, education, and occupational, interpersonal, and legal histories. Following the interview, the interviewer reviewed the institutional file to corroborate information provided during the interview. The combination of interview and file information was used to rate psychopathy according to Hare's (2003) Psychopathy Checklist-Revised (PCL-R). After excluding two participants whose representational capacity scores were more than 2.5 standard deviations below the mean, the number of participants included in analyses was reduced to 75. For supplemental analyses involving the psychopathy factors, two participants were excluded since factor scores could not be computed due to omitted PCL-R items. As a result, the final sample size for these analyses was 73.

Psychopathy Checklist-Revised (PCL-R). We assessed psychopathy using Hare's Psychopathy Checklist-Revised (PCL-R; 2003). The PCL-R consists of 20 items that are rated according to the degree to which the characteristic is present (significantly = 2, moderately = 1, not at all = 0). In the current sample, scores on this measure ranged from 9 to 34, with a mean of

22.30 (SD = 6.37). Interrater reliability was assessed using intraclass correlation (ICC). Although dual, independent PCL-R ratings were available for only five of the participants in this study, ICC was .99.

Materials

Stimuli. Stimuli consisted of 50 colored line drawn objects used by Sligte et al. (2010). All experimental stimuli are displayed in Figure 6. Participants were shown memory and test displays containing eight randomly selected objects that were radially projected equidistant from a centrally presented red fixation point. Each object was presented equally often within each block. All stimuli were presented on a white background. Figure 7 depicts a sample memory display.

Figure 6. Stimulus set for change detection and identification tasks. Adapted from Sligte et al. (2010).

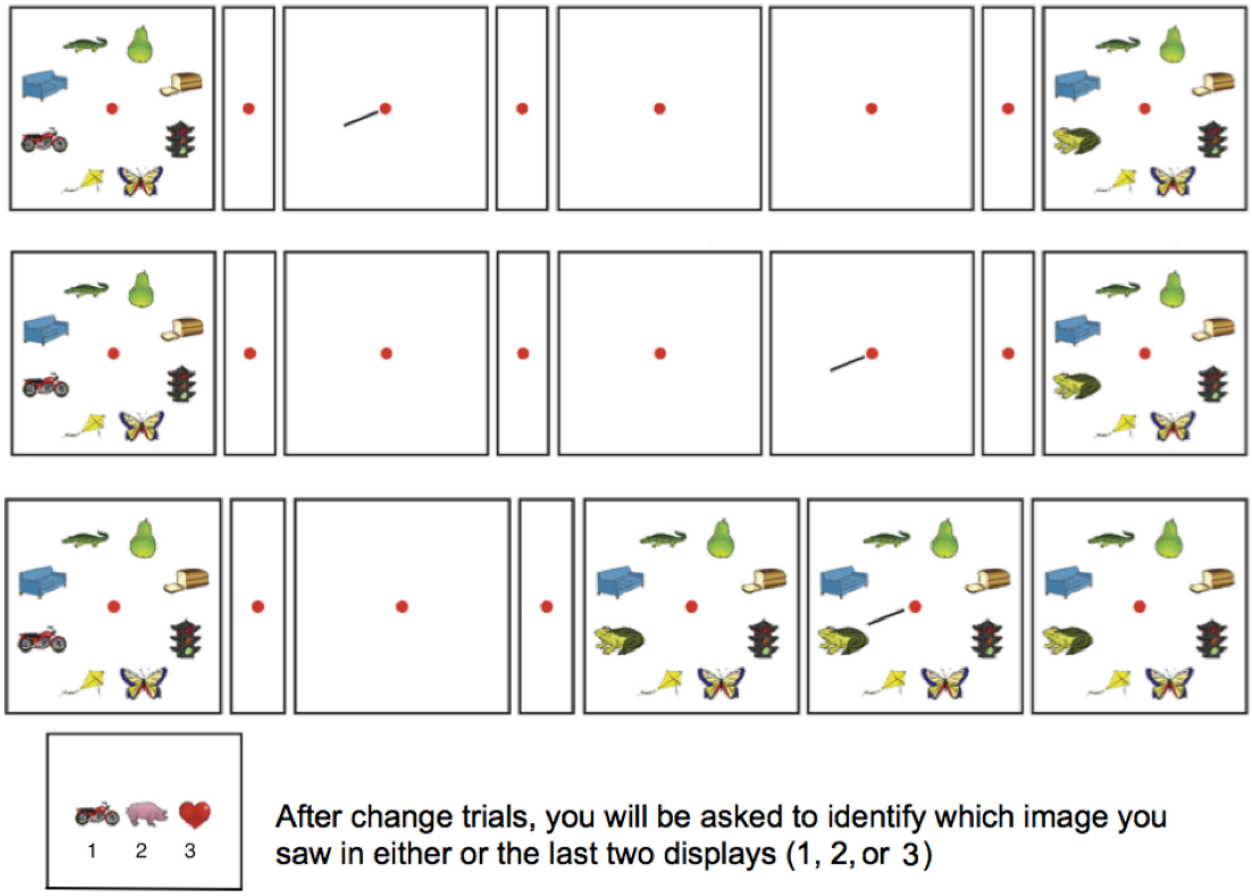

Figure 7. Trial structure of change detection task. At the onset of each trial, participants were presented with a red fixation dot in the middle of the screen that turned green at the start of the trial. They then saw a memory display containing eight objects. On each trial, one object was cued; the timing of the cue depended on the experimental condition (Stage 1: sensory-perceptual encoding trials; Stage 2: fragile, transitional state trials; Stage 3: attentional encoding trials). Following a retention interval, participants were shown a test display and indicated if the cued object changed between the memory and test displays. After each change trial, participants were shown an identification display that contained a line-up of three objects, one of which had been presented in the memory and/or test displays and two of which had not been featured on that trial. Participants were asked to detect the identification object from the array. Adapted from Sligte et al. (2010).

Experimental Task. The task consisted of five blocks of 80 trials (80 practice trials and 320 experimental trials). Each block was separated by a 10 sec break. On each trial, participants were presented with a red fixation dot in the middle of the screen that turned green for 1,000 ms to indicate the start of the trial. They then saw a 250 ms memory display containing eight objects. Participants were instructed to remember as many objects of this memory display as possible. On each trial, one object was cued (see below). Following a retention interval, participants were shown a test display that either mirrored the memory display (no-change trial,

50% of trials) or displayed seven of the original objects with one object substitution (change trial, 50% of trials). They were asked to indicate whether the cued object changed between the memory and test displays via button press. Test displays were present for two seconds or until the participant made a response.

After each change trial, participants were shown an identification display that contained a line-up of three objects, one of which had been presented in either the memory or test display (the identification object) and two which had not been featured on that trial. The identification object was either the pre-change object, the post-change object, or one of the seven distractor objects in the memory display.[10] Participants were asked to detect the identification object from the array. This display was shown until the subject made a response or three seconds elapsed.

The timing at which a cue was presented determined the experimental condition. To assess sensory-perceptual encoding (Stage 1), spatial cues were presented 10 ms after memory display offset. To assess representations during the fragile, transitional state (Stage 2), cues were presented 1,000 ms after memory display offset during the retention interval. Thus, change detection performance at Stages 1 and 2 depends on resolution and persistence of the whole array for 10 ms and 1,000 ms, respectively. On attentional encoding (Stage 3) trials, cues were presented 1,000 ms after memory display offset and 100 ms after the test display onset (i.e., after the possible change had already occurred). In this condition, the presentation of the test display (i.e., a nearly identical image to the memory display) overwrites the fragile representation. Therefore, performance at Stage 3 depends on both persistence over a delay and resistance to

[10] Each identification type consisted of the following number of experimental trials: 55 pre-change object identification trials, 55 post-change object identification trials, and 51 distractor object identification trials. These trials were equally distributed across blocks. No predictions were made for identification accuracy for distractor objects since we could not verify whether participants attended to this information.

being overwritten by the pre-cue presentation of the test display. The items that do persist were transferred to this more stable state because they were under the focus of attention. The interval between memory and test display was 2,000 ms for Stages 1 and 2, and 900 ms for Stage 3 (which served to equate the time between cue presentation and memory display offset in the Stage 2 and 3 assessments). Each of the three trial types were randomly and equally distributed throughout the task. The duration of the practice and experimental trials was approximately fifty-five minutes.

Computation of Independent Variables

Representational Capacity (*K*). Information is represented in the brain through patterns of neural activation, and representational capacity refers to the amount of information that can be stored in memory. Following Sligte et al. (2010), representational capacity was computed using a formula developed by Cowan (Cowan, 2001b) that transforms change detection accuracies to capacity estimates as a function of display set size: K = (hit rate - false positive rate) × number of objects presented. This formula is widely-used to quantify the maximum amount of visual information that can be stored in short-term memory (Cowan, 2001a). *K* provides an estimate of a person's representational capacity that corrects for guessing.[11]

Data Analysis

The chief aim of the current study was to test the putative effects of attentional abnormalities and associated perceptual constraints on the formation and maintenance of mental representations in psychopathic individuals. Accordingly, we conducted a general linear model

[11] Note that any negative capacity estimates do not mean that representational capacity is negative. Theoretically, it is impossible to have a representational capacity that is below zero, but the current statistical formula and design allow for these values. Negative K values are interpretable as random noise associated with participant guessing due to a failure to encode necessary information (Shipstead, Redick, Hicks, & Engle, 2012).

(GLM) with trial type (Stage 1, 2, or 3) as a repeated measure, standardized PCL-R score as a continuously distributed between-subject factor, and *K* as a dependent variable. To control for the effects of overall intelligence, estimated IQ was standardized and included as a continuously distributed between-subject covariate in all analyses.

Greenhouse–Geisser corrected *p*-values are reported to protect against violations of the assumption of sphericity. Partial eta squared values are included as measures of effect size.

Results

Preliminary Analyses

Preliminary analyses were conducted to explore the effects of the task variables, independent of personality measures. The omnibus test assessing condition-related differences in mean *K* was significant ($F(1.14, 84.11) = 18.83$, $p < .01$, $\eta_p^2 = .20$, 90% CI [.09, .32]. A subsequent Helmert contrast demonstrated that representational capacity on Stage 1 trials was significantly greater than capacity across the other two conditions, $F(1, 74) = 17.90$, $p < .01$, $\eta_p^2 = .20$, 90% CI [.07, .32]. Stage 2 representational capacity was also significantly greater than Stage 3 representational capacity, $F(1, 74) = 20.46$, $p < .01$, $\eta_p^2 = .22$, 90% CI [.09, .34]. This finding is consistent with the notion that mental storage capacity significantly decreases over the course of information processing (i.e., from early sensory processing to stimulus retention) due to increasingly strict limits on processing capacity (Bradley & Pearson, 2012; Sligte, 2015; Sligte et al., 2010).

Covariate-adjusted mean *K* values by trial type are presented in Table 4. These tables also include estimated values for individuals with low and high levels of psychopathy that were

derived from respective GLMs.[12] Table 5 provides the covariate-adjusted (i.e., partial) inter-correlations among the study variables.

Table 4. *Covariate-Adjusted Mean Representational Capacity by Trial Type*

	Condition Mean K	High psychopathy K	Low psychopathy K
Stage 1	2.24 (.47)	.83 (.74)	3.64 (.74)
Stage 2	1.58 (.32)	.61 (.50)	2.55 (.50)
Stage 3	.46 (.12)	.15 (.18)	1.35 (.18)

Note: Means and standard errors (presented in parentheses) reported here are from covariate-adjusted variables. Psychopathy scores were obtained using the Psychopathy Checklist-Revised (PCL-R; Hare, 2003). Since we analyzed psychopathy continuously rather than using an extreme-group design, the values presented are point estimates (i.e., estimated using regression analyses) for low and high psychopathy points (1.25 SD below and above the sample mean PCL-R total score, respectively) on the distribution. Stage 1 = estimates from Stage 1 trials; Stage 2 = estimates from Stage 2 trials; Stage 3 = estimates from Stage 3 trials; K = Cowan's (2001b) representational capacity estimate.

[12] The SD point of 1.25 was chosen to compute the point estimates for accuracy and response time so that the predicted values for high levels of psychopathy would correspond to values for an individual with a PCL-R score over 30.

Table 5. *Partial Correlations (r values) between PCL-R Score, Psychopathy Factor Scores, and Performance Variables by Trial Type*

	PCL-R	Factor 1	Factor 2	Stage 1 K	Stage 2 K	Stage 3 K	Pre CD:ID ACC	Post CD:ID ACC
PCL-R	-							
Factor 1	.78**	-						
Factor 2	.88**	.45**	-					
Stage 1 K	-.28*	-.30**	-.16	-				
Stage 2 K	-.28*	-.34**	-.14	.96**	-			
Stage 3 K	-.25*	-.20	-.18	.70**	.71**	-		
Pre CD:ID ACC	-.11	-.15	-.03	.50**	.51**	.35**	-	
Post CD:ID ACC	.09	.03	.14	.02	0.08	.15	.43**	-

Note. Values represent the controlling for the inter-correlations between PCL-R and performance variables controlling effect of IQ. PCL-R = PCL-R total score; Factor 1 = PCL-R Factor 1 score; Factor 2 = PCL-R Factor 2 score; Stage 1 K = Stage 1 representational capacity; Stage 2 K = Stage 2 representational capacity; Stage 3 K = Stage 3 representational capacity; Pre CD:ID ACC = identification accuracy for pre-change objects on correctly detected change trials; Post CD:ID ACC = identification accuracy for post-change objects on correctly detected change trials; * = $p < .05$; ** = $p < .01$

Testing Psychopathy-Related Differences in Representational Capacity

There was a significant main effect of psychopathy on *K*, F(1, 72) = 6.35, p = .01, η_p^2 = .08, 90% CI [.01, .19] (see Figure 8). Specifically, higher psychopathy scores were associated with reduced representational capacity across all processing stages. There was also an interactive effect of psychopathy and trial type on *K* ($F(1.15, 82.41) = 4.60$, $p = .03$, $\eta_p^2 = .06$, 90% CI [.003, .16]). Follow-up analyses revealed that psychopathy-related differences in representational capacity were most prominent at early relative to later information processing stages (Stage 1 vs. later stages: $F(1, 72) = 4.60$, $p = .04$, $\eta_p^2 = .06$, 90% CI [.004, .17]; Stage 2 vs. Stage 3: $F(1, 72) = 4.59$, $p = .04$, $\eta_p^2 = .06$, 90% CI [.003, .17]).

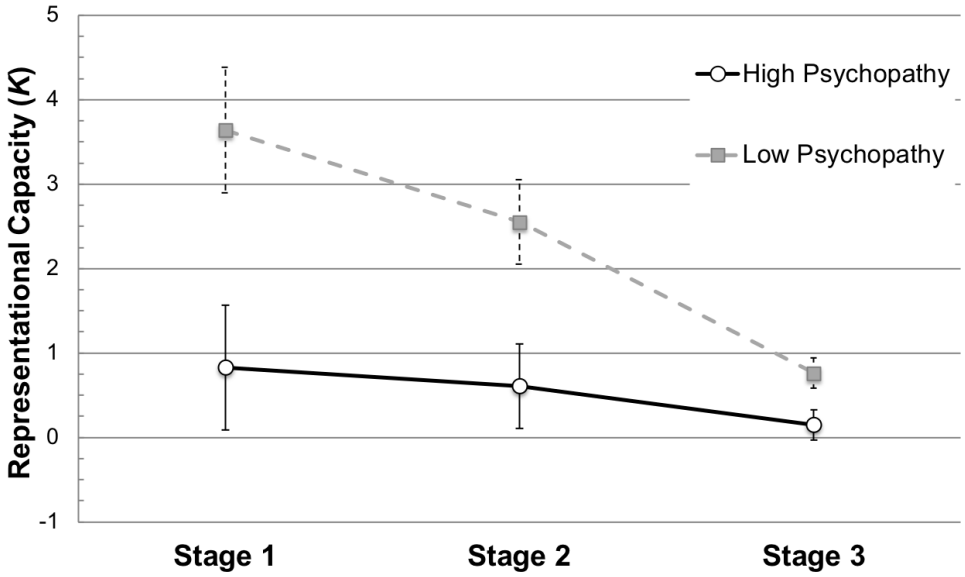

Figure 8. Representational capacity (*K*) as a function of experimental condition and psychopathy (±1.25 SD from the mean). Point estimates representing the average PCL-R score plus or minus 1.25 standard deviation were used to compute the effects of each condition by psychopathy level.

Supplemental Analyses

Representational Quality. In their paradigm, Sligte and colleagues (2010) included a post-change trial identification task in which participants were required to identify a pre- or post-change object from the memory or test display. Accurate change detection *plus* accurate identification of a previously displayed object requires participants to have a high-resolution (i.e., visually-detailed) representation of the object. For instance, a high-resolution representation would enable a participant to not only recognize that an object has changed from, say, a green pear to a red balloon; it would also enable the participant to distinguish between a red balloon and a red flower in the subsequent identification task. In contrast, a coarse representation of the pre-change object may allow a participant to notice that a change occurred but would not help him distinguish between the two red identification objects. Thus, greater identification accuracy of pre- and post-change objects following correctly-detected change trials (CD:ID accuracy) suggests higher representational resolution.

The II theory assumes that the fundamental effect of the proposed integrative deficit is not on the *quality* (i.e., level of detail) of encoded information, but on the *time-course* of mental representations construction. As a result, we did not predict psychopathy-related differences in CD:ID accuracy, since the critical information on these trials presumably fell within the attentional spotlight. Nevertheless, we conducted analyses of CD:ID variable for the sake of completeness.

As expected, there was neither a main effect of psychopathy on CD:ID accuracy ($F(1, 72) = .03$, $p = .87$, $\eta_p^2 < .01$, 90% CI [.00, .02]) nor an interactive effect of psychopathy and identification type (pre- vs. post-change identification objects; $F(1, 72) = 1.82$, $p = .18$, $\eta_p^2 = .03$, 90% CI [.00, .11]), suggesting high psychopathy individuals were as accurate at identifying objects as low psychopathy individuals when they correctly detected change (see Table 6). In short, the level of detail of the mental representations informing identification performance appeared comparable across psychopathy scores.

Table 6. *Covariate-Adjusted Mean Identification Accuracy on Correctly-Detected Change Trials by Object Type*

	Mean	**High psychopathy**	**Low psychopathy**
Pre-change CD:ID ACC	43.50% (.02)	40.90% (.03)	46.10% (.03)
Post-change CD:ID ACC	52.50% (.03)	54.30% (.04)	50.60% (.04)

Note: Means and standard errors (presented in parentheses) reported here are from covariate-adjusted variables. Psychopathy scores were obtained using the Psychopathy Checklist-Revised (PCL-R; Hare, 2003). Since we analyzed psychopathy continuously rather than using an extreme-group design, the values presented are point estimates (i.e., estimated using regression analyses) for low and high psychopathy points (1.25 SD below and above the sample mean PCL-R total score, respectively) on the distribution. Pre-change CD:ID ACC = identification accuracy for pre-change objects on correctly detected change trials; Post-change CD:ID ACC = identification accuracy for post-change objects on correctly detected change trials.

Characterizing Capacity Differences. One potential concern regarding the findings is that individuals high in psychopathy may be less motivated to perform well on the task relative

to participants with low PCL-R scores.[13] Differences due to low motivation may reflect a more general, non-specific deficit rather than one specific to representational capacity. Accordingly, we examined two additional indices of performance that are sensitive to motivation (Pachella, 1974; see also Sternberg, 2001) to address the plausibility of a general motivational deficit.

First, we conducted a GLM with standardized PCL-R score as a continuously distributed between-subject factor, standardized estimated IQ as a continuously distributed between-subject covariate, and average change detection response time as a dependent variable. Results indicated that the mean response time to indicate a detected change did not differ based on psychopathy score, $F(1, 72) = .06$, $p = .81$, $\eta_p^2 < .01$, 90% CI [.00, .03].

Next, we sought to compare patterns of change detection accuracy over time (i.e., during the first half of the task relative to the second). We conducted a GLM with standardized PCL-R score as a continuously distributed between-subject factor, time (first half vs. second half) as a repeated measure, and change detection accuracy as a dependent variable. Standardized estimated IQ was included as a continuously distributed between-subject covariate. There was a significant effect of time on change detection accuracy; participants were more accurate at detecting change in the second half of the experiment relative to the first half, $F(1, 72) = 9.81$, $p < .01$, $\eta_p^2 = .12$, 90% CI [.03, .26]. Furthermore, there was a marginal main effect of psychopathy ($F(1, 72) = 3.90$, $p = .05$, η_p^2 .05, 90% CI [.001, .16]), with low psychopathy individuals displaying moderately higher accuracy than high psychopathy participants ($M = .58$ and .49, respectively). Critically, there was no interaction between psychopathy score and time, $F(1, 72) = 1.68$, $p = .20$, $\eta_p^2 = .02$, 90% CI [.00, .11]. This finding suggests that all participants

[13] The inclusion of estimated IQ as a covariate in all analyses served to eliminate potentially confounding effects of a general aptitude deficit.

learned equivalently across trials (Mean Improvement High Psychopathy: 3%; Low Psychopathy: 1%).

Two-factor model of psychopathy. The primary goal of this study was to explore representational capacity in psychopathy across various processing stages. Some researchers advocate decomposing psychopathy into two factors (Factor 1: Interpersonal/Affective and Factor 2: Impulsive/Antisocial) in order to examine the unique effects of these dimensions (Harpur, Hare, & Hakstian, 1989). Accordingly, supplemental analyses were conducted to examine the effects of PCL-R Factors 1 and 2 on representational capacity. These factors were *z*-scored and entered simultaneously into a GLM with trial type (Stage 1, 2, or 3) as a repeated measure, *K* as a dependent variable, and standardized estimated IQ as a continuously distributed between-subject covariate to quantify the unique effects of these components.

There was a significant main effect of trial type ($F(1.15, 79.23) = 22.03$, $p < .01$, $\eta_p^2 = .24$, 90% CI [.11, .36]); a Helmert contrast demonstrated that representational capacity was largest at Stage 1 and smallest at Stage 3 (Stage 1 vs. Later: $F(1, 69) = 20.90$, $p < .01$, $\eta_p^2 = .23$, 90% CI [.10, .36]; Stage 2 vs. Stage 3: $F(1.15, 79.23) = 24.13$, $p < .01$, $\eta_p^2 = .26$, 90% CI [.12, .39]). Analysis revealed a main effect of Factor 1 ($F(1, 69) = 5.65$, $p = .02$, $\eta_p^2 = .08$, 90% CI [.01, .19]), such that higher Factor 1 scores were associated with reduced representational capacity (see Figure 9). This main effect was qualified by a significant interaction; specifically, tests revealed that Factor 1 was significantly negatively associated with representational capacity at Stages 1 and 2 ($B = -1.16$, $p = .03$, $r = -.28$; and $B = -.94$, $p = .01$, $r = -.33$), but not at Stage 3, $B = -.15$, $p = .28$. In other words, high Factor 1 scores were uniquely associated with reduced representational capacity at early information processing stages. There was neither a significant main effect of Factor 2 on *K* nor an interactive effect of trial type and Factor 2 on *K*, $F(1, 69) =$

.04, $p = .85$, $\eta_p^2 < .01$, 90% CI [.00, .02] and $F(1.15, 79.23) = .20$, $p = .69$, $\eta_p^2 < .01$, 90% CI [.00, .05], respectively.

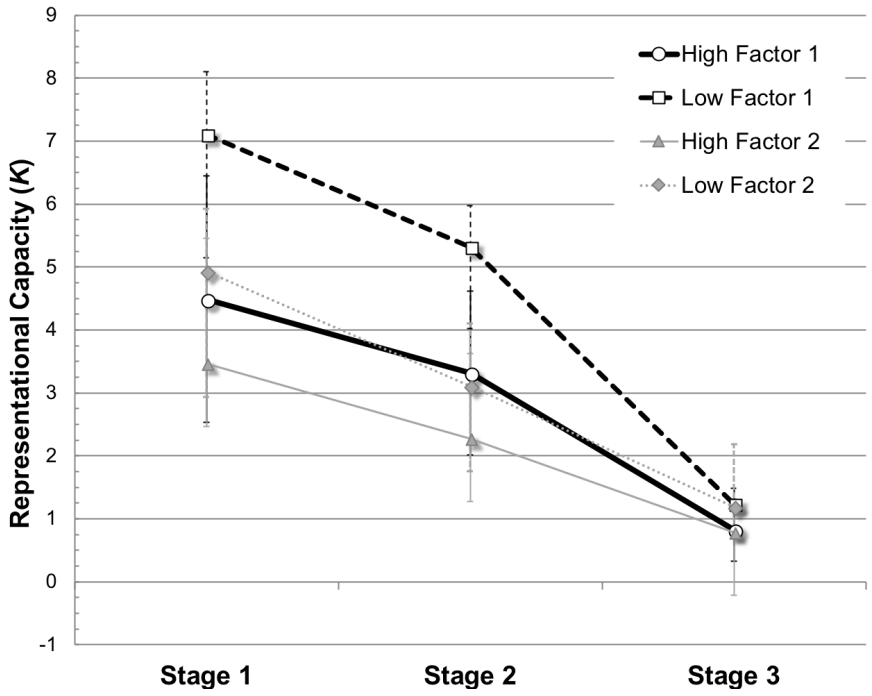

Figure 9. Representational capacity (*K*) as a function of experimental condition and psychopathy Factors 1 and 2 (±1.25 SD from the mean). Point estimates representing the average residualized Factor 1 and Factor 2 scores plus or minus 1.25 standard deviation were used to compute the effects of each condition by factor level.

Discussion

The main finding of the current study was that psychopathy is associated with reduced representational capacity across the information processing stream. Notably, this effect emerged at the earliest processing stage (Stage 1), indicating that psychopathy is associated with deficient perceptual encoding. Reduced representational capacity at later stages (Stages 2 and 3) suggests that this early encoding deficit adversely impacts later use of mental representations. However, there was no evidence that the greater demand for stimulus retention or need to resist interference associated with the later stages (see Sligte, 2015) contributed to psychopathy-related performance deficits. Supplemental analyses exploring the effect of psychopathy on

representational quality failed to reveal any differences, indicating that the quality (i.e., amount of visual detail) of constructed mental representations did not vary based on psychopathy level. In other words, although high psychopathy individuals displayed a deficit in the ability to (perceptually) encode the entirety of the complex visual array, the resolution of the information that fell within their focus of attention and 'got in' (i.e., was encoded) was not deficient.

The presented findings provide novel support for Hamilton and colleagues' (2015) II theory, which predicts that an integrative deficit in psychopathy creates a bottleneck that reduces information encoding and perceptual processing capacity under time constraints by limiting the amount of information that can be processed concurrently. The II theory predicts that high psychopathy individuals encode less information (in this case, from the stimulus array) and form fewer mental representations relative to their lower psychopathy counterparts under time constraints. The fact that there were no psychopathy-related differences in representational quality is also consistent with the II framework's notion that psychopathy is associated with slowed (i.e., in a sequential rather than simultaneous manner) rather than deficient multicomponent perceptual integration. Such findings are also consistent with the attention bottleneck model of psychopathy (Baskin-Sommers et al., 2013), which highlights similar deficiencies.

As aforementioned, the negative relationship between psychopathy and representational capacity emerged at the earliest information processing stage and persisted in downstream processing. Accordingly, the current study further lends support to the growing literature indicating that, in contrast to other syndromes of disinhibition, psychopathy is not distinguished by a selective deficit at later stages of processing involved in higher-order executive functioning (Baskin-Sommers, Brazil, Kohlenberg, Neumann, & Newman, 2015; Baskin-Sommers &

Newman, 2013; Finn, Justus, Mazas, & Steinmetz, 1999; Fitzgerald & Demakis, 2007). The fact that psychopathy was associated with reduced representational capacity across all stages of the information processing stream (i.e., from the perceptual encoding stage to the working memory stage) indicates that the fundamental deficit in psychopathy begins early and influences higher-order cognitive processes. This characterization is consistent with several recent publications (Anderson et al., 2017; Baskin-Sommers, Curtin, Li, & Newman, 2011; Krusemark et al., 2016; Sutton, Vitale, & Newman, 2002) and serves to differentiate psychopathy from other forms of externalizing psychopathology (Baskin-Sommers & Newman, 2013; Young, 2009).

Intriguingly, the early psychopathy-related deficit in perceptual encoding was specific to the interpersonal-affective component of psychopathy (Factor 1). Typically, Factor 1-specific deficits emerge in affective tasks (Dawel, O'Kearney, McKone, & Palermo, 2012; Justus & Finn, 2007; van Honk & Schutter, 2006; Vanman, Mejia, Dawson, Schell, & Raine, 2003). Accordingly, Factor 1 deficits may in part reflect information encoding difficulties rather than emotional dysfunction per se. In other words, Factor 1 traits might be understood as a deficit in the ability to rapidly 'take in' and utilize multicomponent information, which negatively impacts the amount of information available for use in emotion processing and decision-making.

It is interesting to consider that there may be two separate mechanisms through which psychopathy is associated with weak mental representations: Factor 1 may be more strongly related to an early encoding deficit, whereas Factor 2 may be more strongly be related to deficiencies in later cognitive processes responsible for the maintenance and manipulation of mental representations (i.e., working memory). Indeed, Gorenstein (1991) and Patrick et al. (1994) have linked PCL-R Factor 2 to difficulty maintaining representations other than those to which attention is directed (e.g., related to immediate needs or desires). Thus, there could be two

interacting processes underlying reduced representational capacity in individuals high in both Factor 1 and Factor 2. These processes may work synergistically to undermine information processing and contribute to the range of psychopathic symptoms.

There is increasing evidence that psychopathic individuals show a deficit in the ability to engage in typically automatic cognitive processes critical for self-regulation (Drayton, Santos, & Baskin-Sommers, 2018; Keysers & Gazzola, 2014; Meffert et al., 2013; Nentjes, Bernstein, Arntz, van Breukelen, & Slaats, 2015; Wallace et al., 1999); it is plausible that the current findings provide a basis for understanding this deficit. The presented findings suggest that deficient perceptual encoding interferes with the formation of mental representations in psychopathy in the presence of time constraints. In other words, psychopathic individuals appear to 'take in' less sensory information, perhaps due to difficulty processing multicomponent information (e.g., different perceptual components of a visual scene). Even though the current study employed simple, non-affective, visual stimuli to probe representational capacity for theoretical reasons (to limit alternative interpretations of the predicted deficit), we propose that the observed deficit may also apply to the processing of social and emotional representations.

As with any finding, the ultimate implications of the present study depend on replicability. However, the current investigation provides novel evidence regarding the psychological implications of attentional abnormalities for the formation of mental representations in high psychopathy offenders. Moreover, it opens up new areas of investigation relating to representational capacity in psychopathy. It suggests that the disinhibitory behavior characteristic of psychopathy may relate to difficulty forming, maintaining, and integrating multicomponent mental representations without focused attention. This integration difficulty may further hamper the assimilation of formed representations with contextual information. As

Sprague (1941) noted, “in the complexities of modern living it is more necessary than ever before to keep forming incessantly new estimates and forecasts of the probabilities to come. . . . This however the psychopath often does not do. . . . [The psychopath’s] difficulty in learning the lesson of experience comes from his propensity for dealing with part pictures rather than with the total picture of his situation” (p. 913-914). Moreover, even if mental representations are well-formed, the integrative deficit may undermine the automatic use of secondary representations because attention is devoted to other processing (see Shapiro, 1965).

One unresolved question relates to the nature of the causal relationship between the attention bottleneck as described by Newman and Baskin-Sommers (2011) and the proposed integration deficit. Hamilton and colleagues (2015, 2016) suggest that impaired integrative processing could create a bottleneck that obstructs simultaneous processing of perceptual information. However, it is also possible that an attentional bottleneck could contribute to difficulties rapidly integrating multicomponent information. Despite this uncertainty, the current experiment provides further support for the notion that cognitive abnormalities in psychopathy influence the ability to rapidly encode complex perceptual input and hamper the formation of mental representations.

Limitations

Before concluding, it is important to outline potential limitations of the current study. As noted, one possible concern relates to the effects of potentially confounding factors such as motivation on outcome measures. As demonstrated in the supplemental analyses, neither identification accuracy on correctly detected change trials nor mean response time were related to psychopathy level. Additionally, there were no psychopathy-related differences in learning across trials. This pattern of performance across various metrics supports the notion that the

presented findings on psychopathy-related differences in representational capacity are not driven by differences in motivation.

Another potential criticism of the presented study relates to the fact that the sample consisted exclusively of European American male offenders. This lack of racial diversity limits the generalizability of the presented findings. Previous research suggests that the presence of specific laboratory deficits is not consistent across sex (Verona & Vitale, 2006; Vitale et al., 2011) or racial groups (Anderson, 2017; Lorenz & Newman, 2002; Sullivan, Abramowitz, Lopez, & Kosson, 2006). However, studying psychopathy in diverse populations is important for comprehensive understanding of the construct. Accordingly, future research should assess whether the current findings generalize across race and gender.

Conclusion

Taken together, the current findings provide novel support for psychopathy-related differences in perceptual encoding capacity. It appears that information processing in psychopathy is constrained by difficulty processing complex information simultaneously under time constraints. Mental representations of information that fall outside the attentional scope (i.e. undetected changes) are too weak to meaningfully influence behavior, thus effectively fading without a trace. However, the amount of visual detail in the representations of information that falls in the attentional spotlight is largely intact. Further research is required to specify the psychological repercussions of attentional abnormalities for long-term memory processes in psychopathy (see Hamilton et al., 2015 for predictions based on the II theory). Additionally, future studies are needed to characterize mechanisms underlying representational capacity deficits in psychopathy. These studies could explore whether impaired integration and simultaneous processing deficits mediate the outlined association between psychopathy and

representational capacity (see Hamilton et al., 2015), or whether there is a link between encoding deficits and performance on somatic marker tests (see Damasio et al., 1996). Accordingly, they can evaluate the relationship between the neurobiological mechanisms thought to underlie those processes to integrate research across scientific domains.

CHAPTER 4
BEHAVIORAL & PSYCHOPHYSIOLOGICAL COORDINATION IN PSYCHOPATHY: RELATION OF FRONTAL N100 TO PSYCHOPATHY-RELATED DIFFERENCES IN SELECTIVE ATTENTION

Content from this chapter was published as: Hamilton, R. K. B., Baskin-Sommers, A. R., & Newman, J. P. (2014). Relation of frontal N100 to psychopathy-related differences in selective attention. *Biological psychology, 103*, 107-116.

Psychopathy is a personality disorder characterized by a collection of emotional, interpersonal, and behavioral features that include shallow affect, egocentricity, exploitation, lack of remorse, and impulsivity, as well as antisocial conduct (American Psychological Association, 2000; Hare, 1996). Although they represent only 15–20% of criminal offenders (Glenn & Raine, 2008), psychopathic offenders commit a disproportionate percentage of crimes (Harris, Skilling, & Rice, 2001) and have high rates of violent recidivism (Viding, 2004). Given the social and financial costs of their crime (Hare, 2006; Hare & Neumann, 2009; Reid, 1998), it is imperative to clarify the psychobiological processes responsible for psychopathic offenders' failures to manage their behavior.

One perspective suggests that psychobiological abnormalities in information processing underlie psychopathy's association with chronic antisociality and self-regulatory deficits. More specifically, research indicates that psychopathy is characterized by abnormalities in selective attention, such that psychopaths fail to allocate attention to potentially important peripheral stimuli while engaged in goal-directed activity. Baskin-Sommers, Curtin, and Newman (2011) propose that these abnormalities reflect an early attention bottleneck that limits the processing of information unrelated to their mental set (Baskin-Sommers, Curtin, Li, & Newman, 2011; Leber & Egeth, 2006).

The early stages of information processing involve the simultaneous processing of sensory elements; these sensory representations are only available for retrieval for a short length

of time. When an organism is engaged in goal-directed behavior, the first stage of processing is influenced by the behavioral relevance of stimuli. Zylberberg and colleagues (2009) postulate, "the 'memory' of a stimulus resides in the decaying trace of a stimulus transient response" (p. 13). Memory representations that are not selected for higher-level processing in working memory quickly fade. For psychopathic individuals, the establishment of an information-processing bottleneck may guide attention to stimuli consistent with the mental set and consequently preclude the elaborated processing of information that is inconsistent with or peripheral to goal-related focus (Baskin-Sommers et al., 2011; Newman et al., 2010). Consequently, this peripheral information may remain 'pre-conscious', or perceived but not consciously processed due to inattention (Dehaene & Changeux, 2011). As a result, representations of peripheral information may not be strong enough to modulate ongoing goal-directed behavior.

In fact, across experimental contexts psychopathic offenders display a pattern of selective attention, such that they fail to process peripheral information when their attention is already engaged in a goal-directed task. For instance, when non-psychopathic control participants engage in tasks involving low perceptual load (i.e., there are few distracter stimuli), they are more likely to experience distracter interference (Lavie & Tsal, 1994). However, the same dis-tractors elicit less interference in psychopathic individuals (Sadeh & Verona, 2008). Moreover, psychopathic individuals display significantly less behavioral interference than controls when engaged in tasks containing incongruent contextual cues (see Newman, Brinkley, Lorenz, Hiatt, & MacCoon, 2007; Newman et al., 1997; Zeier et al., 2009). A handful of electrophysiological studies support the proposal that psychopathic individuals show reduced responses to contextual cues when these cues are not directly related to their goal-directed focus of attention. Moreover, they provide

evidence for the early nature of these attentional abnormalities. Event-related potentials (ERPs) provide a high-resolution, temporally precise look at the earliest changes in visual processing associated with visual-spatial selective attention (Herrmann & Knight, 2001; Hillyard & Anllo-Vento, 1998). Jutai and Hare (1983) found that psychopathy was associated with reduced N100 amplitudes to task irrelevant tone pips while engaged in a selective attention task. Baskin-Sommers et al. (2012) demonstrated that psychopathic individuals were able to effectively ignore threat-related distractors (as indexed by larger P140) when they were peripheral versus central to their goal-directed behavior. The temporal nature of these waveforms is consistent with the notion that psychopathy is characterized by abnormalities early in the processing stream. The fact that they were modulated by attentional focus further supports the proposition that psychopathy is associated with anomalous early selective attention.

One paradigm that is well suited to examine abnormal processing of contextual cues is the "Box Stroop" task (Hiatt et al., 2004). During this task, color names (green, red, blue, or yellow) are displayed in black ink and are surrounded by a green, red, blue, or yellow box. Participants are told to say the color of the box. Like the standard Stroop effect, congruent word name and box color typically facilitate color naming, while incongruent stimuli generally cause interference. The Box Stroop, however, provides a clearer test of psychopaths' early selective attention compared to the standard paradigm because incongruent information (i.e., the word names) is spatially separated from, and thus peripheral to, the predominant focus of attention (i.e., the box color). Specifically, the spatial division enables the sensory amplification and early selection of attended to-be-processed features (Hillyard, Vogel, & Luck, 1998). Accordingly, individuals high in psychopathy display normal interference in the traditional Stroop task, yet they exhibit significantly less interference on the Box Stroop than controls (Hiatt et al., 2004).

This finding is consistent with the proposition that psychopathy is associated with abnormal attention processes. Thus, the Box Stroop represents a validated paradigm that yields psychopathy-related effects with conflict-laden stimuli. However, to date, there is no direct substantiation linking performance on this task to early attentional processes.

The goal of the current study is threefold. On the behavioral level, the current research seeks to replicate and extend Hiatt et al.'s (2004) findings of attentional abnormalities in psychopathic individuals. Specifically, we hypothesize that there will be a significant effect for psychopathy such that individuals high in psychopathy will show less interference than individuals with lower psychopathy scores (i.e., the difference between the time it takes for individuals high in psychopathy to respond to an incongruent trial compared to a neutral trial will be significantly less than for nonpsychopathic individuals).

The second goal of the study is to clarify the temporal profile of the hypothesized information processing abnormality. Specifically, we were interested in locating an ERP window that might be interpreted as a feed-forward processes related to early selective attention (i.e., within 200 ms of stimulus onset; Lamme & Roelfsema, 2000). Although past research is consistent with our proposal that the spatial separation of box and word stimuli enables early selection in psychopathic individuals, the behavioral evidence revealing reduced interference alone cannot specify the early versus late onset of psychopathy-related differences in performance. In this study, we use ERPs to explore the temporal dynamics of psychopathy-related effects in the Box Stroop. We predict that the ERPs to incongruent versus neutral stimuli in psychopathic individuals will differ from those in non-psychopathic individuals, and that these differences will be evident early in the information-processing stream.

The final goal of the study is to explore psychopathy-related differences in the association between ERP and interference data to determine whether the relationship between ERP amplitude and interference varies as a function of psychopathy. In light of postulated abnormalities in early selective attention, we predict that the association between behavior and ERP data will differ for high versus low psychopathic participants. This finding would support the idea that psychopathy is typified by abnormalities in early attention responses to goal-incongruent information.

Methods

Participants

Participants consisted of 117 Caucasian male inmates ages 18–45 (M = 30.44, SD = 6.70) from a medium-security prison in central Wisconsin. To be included in the study, participants had to be between 18 and 45 years old, free of history of psychosis or bipolar disorder, not currently taking psychotropic medication, and have an IQ score of 70 or greater. Individuals meeting the inclusion criteria were invited to participate in an ongoing study. All participants provided written informed consent according to procedures approved by the University of Wisconsin – Madison Human Subjects Committee. On the first day of the study, inmates were called to a private office and completed a semi-structured life history interview with an experienced interviewer. This interview included questions on childhood, education, and occupational, interpersonal, and legal histories. Following the interview, the interviewer reviewed the institutional file in order to corroborate information provided during the interview. The combination of interview and file information was used to rate psychopathy according to Hare's (2003) Psychopathy Checklist-Revised (PCL-R). Four participants were excluded from analyses due to less than 90% accuracy on the experimental task.

Psychopathy Checklist-Revised (PCL-R)

We assessed psychopathy using Hare's (2003) Psychopathy Checklist-Revised (PCL-R). The PCL-R consists of 20 items that are rated according to the degree to which a characteristic is present (significantly = 2, moderately = 1, not at all = 0). In the present sample, scores on this measure ranged from 5 to 36, with a mean of 23.14 (SD = 6.86). Interrater reliability (intraclass correlation) for PCL-R total score, based on six dual ratings, was .98.

Materials

Stimuli. Stimuli were 120 color words (red, blue, green, or yellow, written in black font) or neutral stimuli (string of the letter i) presented on a white background surrounded by a colored rectangular frame (red, blue, green, or yellow). The frames measured 2.3 by 3.2 cm. Word and neutral stimuli were presented centrally in the frames.

Experimental Task

The task consisted of 40 practice trials and 120 experimental trials. For the first 20 practice trials, color words (red, blue, green, or yellow) written in black font were presented centrally on the computer screen and participants were instructed to read the words. For the remaining 20 practice trials, colored rectangular frames were presented on the screen and participants were instructed to name the color of the frame (red, blue, green, or yellow). Each experimental trial consisted of a simultaneously presented color word or neutral stimulus enclosed by a colored rectangular frame. Participants were told to name the color of the frame while ignoring all other information. There was a total of 40 congruent trials (color word matched the frame color), 40 incongruent trials (color word differed from the frame color), and 40 neutral trials (iiiii surrounded by a frame color) (see Figure 10). Trials were ordered such that no words or colors appeared twice in a row. Each stimulus appeared for a minimum of .5

seconds until the participant responded or 1.5 seconds had elapsed. The inter-trial interval ranged from 1.5 to 2.5 seconds. The 120 experimental trials were divided into two blocks (60 trials each) separated by a 30 second break.

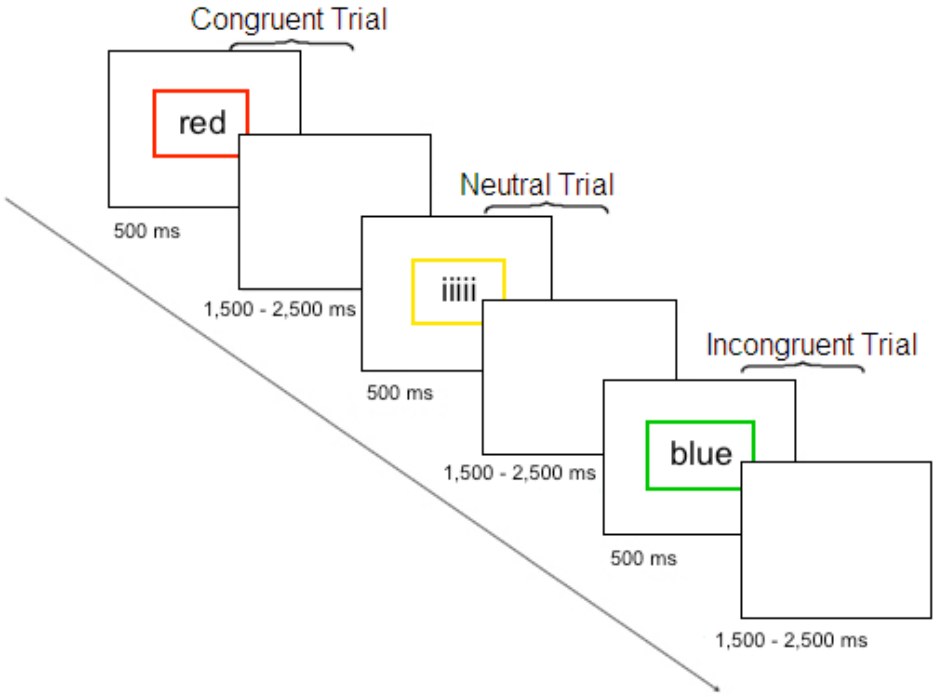

Figure 10. Trial structure of Box Stroop task. At the onset of each trial, participants saw two stimuli: text (red, blue, yellow, green, or iiiii) and a colored rectangular frame (red, blue, yellow, or green). These stimuli were presented on the screen until the participant responded or three seconds had elapsed. During these trials, participants were told to name the color of the frame while ignoring all other information. Following the offset of the stimuli, a blank screen appeared.

Participants were instructed to state their responses as quickly and accurately as possible into a headset-mounted microphone. Reaction time (RT) for the onset of the verbal response was recorded automatically by a voice-activated relay device. The accuracy of the responses and the reaction times were assessed offline. Consistent with Hiatt et al. (2004), behavioral interference and facilitation scores were computed for each participant by subtracting RT on neutral trials from RT on incongruent trials and subtracting RT on congruent trials from RT on neutral trials, respectively.

Physiological Recording and Data Reduction

Stimulus presentation and response collection were controlled by a PC-based MATLAB (The Mathworks) script and Neuroscan Synamps amplifiers and acquisition software (Compumedics, North Carolina). EEG was recorded at a 2500-Hz sampling rate from Ag–AgCl electrodes mounted in an elastic cap (Electro Cap International) along the midline (Fz, FCz, Cz, and Pz) and referenced to the left mastoid. EEG data were corrected for ocular artifacts from electrodes positioned to detect vertical electrooculogram (VEOG). The electrode impedance for all channels was kept below 10 kΩ. Data were further processed offline using the PhysBox plugin (Curtin, 2011) within the EEGLab toolbox (Delorme & Makeig, 2004) in MATLAB. Offline processing included low-pass filtering (4th-order, 20-Hz Butterworth low-pass filter), epoching (−500 to 1200 ms epochs), baseline correction, and artifact rejection (rejection of trials with voltages exceeding ±75 μV).

Given our prediction that the reduced interference response of psychopathic individuals is associated with abnormalities in early selective attention, a primary goal of this investigation was to find the earliest reliable component that tracked the condition manipulation (i.e., trial type) to assess whether there were psychopathy-related differences in the magnitude of this component. Visual inspection of the grand-averaged ERP data revealed that the earliest component on this task was a negative potential that peak around 100 ms post-stimulus onset (Figure 11). The magnitude of N100 was measured as the peak amplitude value in 90 ms to 185 ms post-stimulus-presentation window.

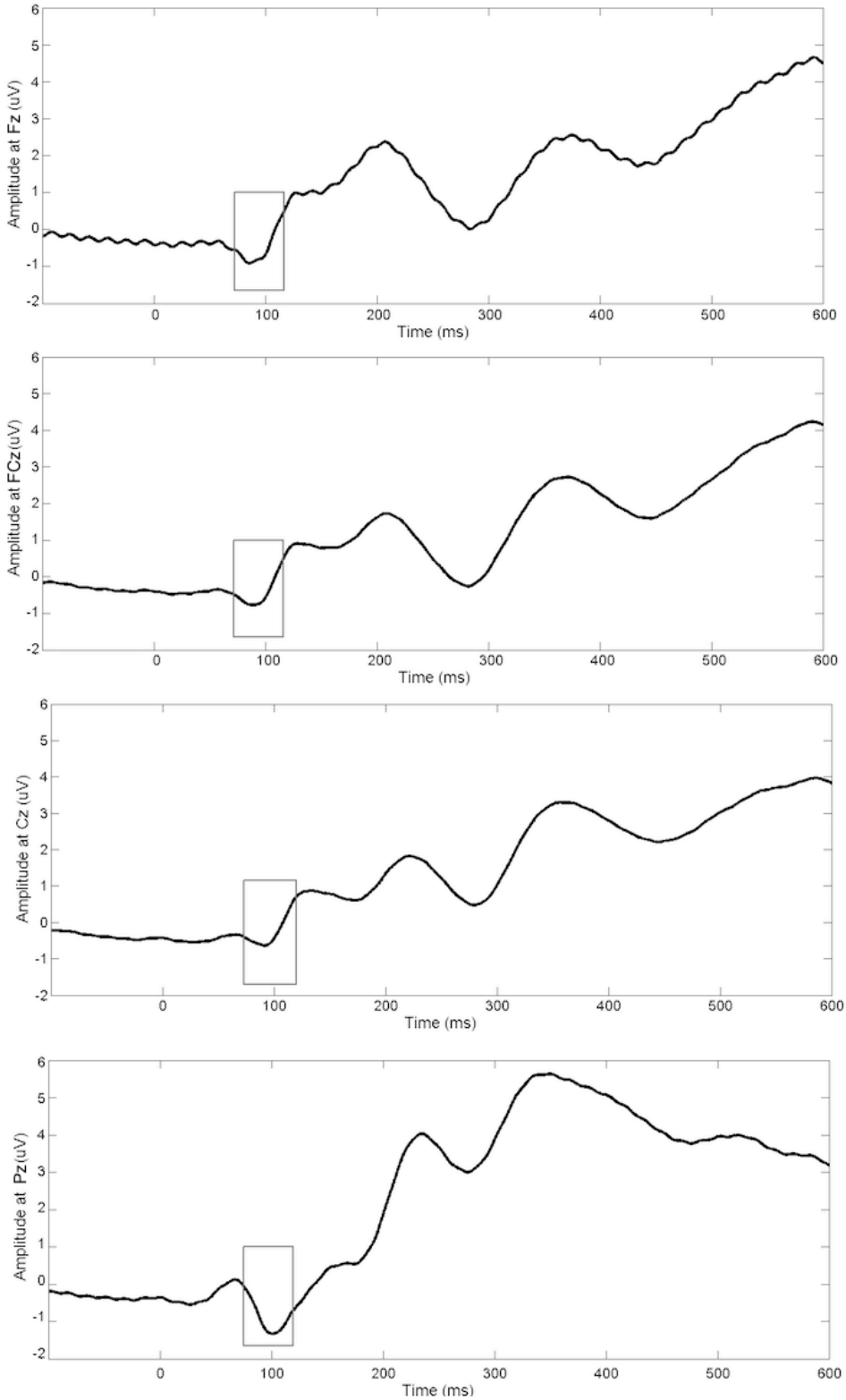

Figure 11. Grand average waveforms across scalp sites (Fz, FCz, Cz, and Pz). Stimulus-locked average ERP waveforms for all participants at midline electrode sites. A digital low-pass filter was applied offline before plotting the waveforms shown here. The boxes represent the peak identified as N100.

Waveforms were averaged separately for correct trials within each trial type (congruent, incongruent, neutral). Participants were excluded from analyses if they had fewer than 25 out of 40 valid trials in each condition. After data processing (e.g., controlling for electrical noise and excluding trials with significant artifact [±75 μV]), we eliminated 26 participants with fewer than 80% valid trials remaining from the ERP analyses. The final sample for the ERP analyses was 91.

Data Analysis

The goal of the analyses was threefold. To address the first hypothesis, we used regression to examine behavioral interference as a function of psychopathy using mean-centered PCL-R total scores. To address our second hypothesis that psychopathy is associated with early ERP differences, we computed difference scores for the N100 components at each of the four electrode sites by subtracting ERP amplitudes on neutral trials from those on incongruent trials. This variable represents a physiological response to incongruent versus neutral stimulus presentations. Given that N100 is a waveform of negative polarity, the larger (i.e., more positive) the difference score the smaller the ERP magnitude to incongruent versus neutral trials. These difference scores were analyzed using a general linear model (GLM), with scalp site (Fz, FCz, Cz, Pz) as a repeated measure and mean-centered PCL-R scores as a continuously distributed between-subject factor. The final set of analyses explored the relationship among behavioral interference, psychopathy total score, and the psychophysiological indices. To protect against violations of the assumption of sphericity, Greenhouse–Geisser corrected p-values are reported. Additionally, partial eta squared values are included as measures of effect size.

Results

Behavioral Analyses

Consistent with Hiatt et al.'s (2004) findings, the analysis revealed a significant main effect of psychopathy, such that higher PCL-R ratings were associated with less behavioral interference, $F(1, 115) = 6.97$, $p = .01$, $\eta_p^2 = .06$ ($r = -.24$). Also consistent with the findings of Hiatt and colleagues, there was no significant relationship between psychopathy level and behavioral facilitation, $F(1, 115) = .14$, $p = .71$, $\eta_p^2 < .01$. Mean reaction times and ERP amplitudes, as well as estimated mean values for individuals high and low in psychopathy, for incongruent and neutral trials are presented in Table 7, while Table 8 provides the inter-correlations among the study variables.[14, 15]

Table 7. *Mean RT and ERP Amplitude Values to Incongruent and Neutral Trials by Scalp Site*

	Condition Mean		High psychopathy		Low psychopathy	
	Incongruent	**Neutral**	**Incongruent**	**Neutral**	**Incongruent**	**Neutral**
RT	668 (9.76)	585 (6.72)	639 (14)	573 (10)	681 (14)	587 (10)
N100 Fz	-1.27 (.20)	-1.62 (.22)	-.84 (.28)	-1.64 (.31)	-1.70 (.28)	-1.60 (.31)
N100 FCz	-1.14 (.20)	-1.50 (.22)	-.80 (.29)	-1.43 (.32)	-1.49 (.29)	-1.57 (.32)
N100 Cz	-1.05 (.20)	-1.41 (.21)	-.78 (.28)	-1.24 (.30)	-1.33 (.29)	-1.58 (.30)
N100 Pz	-1.85 (.28)	-2.16 (.29)	-2.08 (.39)	-2.29 (.42)	-1.62 (.40)	-2.02 (.42)

Note: Psychopathy scores were obtained using the Psychopathy Checklist-Revised (PCL-R; Hare, 2003). Since we analyzed psychopathy continuously rather than using an extreme-group design, the values presented are point estimates (i.e., estimated using regression analyses) for low and high psychopathy points (1 *SD* below and above the sample mean PCL-R total score, respectively) on the distribution. Numbers in parentheses are standard errors. N100 Fz: N100 amplitude at Fz; N100 FCz: N100 amplitude at FCz; N100 Cz: N100 amplitude at Cz; N100 Pz: N100 amplitude at Pz.

[14] One of the primary research questions in the study conducted by Hiatt et al. (2004) involved the interactive effect of psychopathy and anxiety on Box Stroop task performance. They found both a main effect of psychopathy on interference levels as well as a psychopathy by anxiety interaction on interference such that low-anxious psychopathic participants demonstrated significantly less interference than their high-anxious counterparts. Given the specificity of Hiatt et al.'s (2004) anxiety-related finding, we tested the interactive effect of psychopathy score and Welsh anxiety score on behavioral interference. Results were non-significant, supporting the a priori prediction of a simple main effect of psychopathy score on interference.

[15] Although a priori hypotheses focused on behavioral interference, we also examined accuracy. First, there were relatively few errors and relatively little variability in accuracy. Second, we observed no psychopathy-related differences in accuracy. Third, controlling for accuracy did not alter the relationship between psychopathy (the independent variable) and behavioral interference (the dependent variable). Thus, we focused the remainder of our analysis on reaction time.

Table 8. *Bivariate Correlations (r values) between PCL-R Score, Behavioral Interference, and ERP Amplitudes at Fz and Pz*

	PCL-R	Int	Acc	N100i Fz	N100n Fz	N100i Pz	N100n Pz
PCL-R	-						
Int	-.24*	-					
Acc	.16	-.05	-				
N100i Fz	.23*	-.07	.14	-			
N100n Fz	-.01	-.14	.13	.42**	-		
N100i Pz	-.09	.09	-.03	.42**	.19	-	
N100n Pz	-.05	-.04	-.10	.21	.31**	.78**	-

Note. PCL-R = PCL-R total score; Int = behavioral interference (incongruent RT minus neutral RT); Acc = accuracy; N100i Fz = N100 to incongruent trials at Fz; N100n Fz = N100 to neutral trials at Fz; N100i Pz = N100 to incongruent trials at Pz; N100n Pz = N100 to neutral trials at Pz; * = p < .05; ** = p < .01

Psychophysiological Analyses

For the following sets of analyses involving the scalp site measure, Mauchly's test indicated that sphericity had been violated ($\chi^2 = 131.01$, $p < .001$ and $\chi^2 = 107.38$, $p < .001$). Accordingly, degrees of freedom are adjusted using the Greenhouse-Geisser correction in all analyses involving this repeated variable.

There was no main effect of scalp site on N100, $F(1.61, 137.03) = .05$, $p = .92$, $\eta_p^2 < .01$. There was also no significant main effect of psychopathy on N100 difference score when collapsing across scalp-site, $F(1, 85) = .95$, $p = .33$, $\eta_p^2 = .01$. There was, however, a significant two-way interaction between scalp site and PCL-R score, $F(1.61, 137.03) = 4.56$, $p < .01$, $\eta_p^2 = .05$. Due to the lack of a priori hypotheses regarding scalp site, we unpacked this omnibus interaction by examining the simple effects of psychopathy on the N100 difference score at each scalp site. These tests revealed that psychopathy was positively associated with the N100 difference score at Fz (B = .07, $p = .05$, $r = .22$), but not at the remaining scalp sites, FCz: B = .04, $p = .22$; Cz: B = .02, $p = .61$; Pz: B = -.01, $p = .62$. More specifically, as scores on

psychopathy increased the magnitude of frontal N100 amplitude to incongruent versus neutral stimuli decreased (see Figure 12 for the grand-averaged N100 waveforms by trial type for individuals with low and high PCL-R scores).[16]

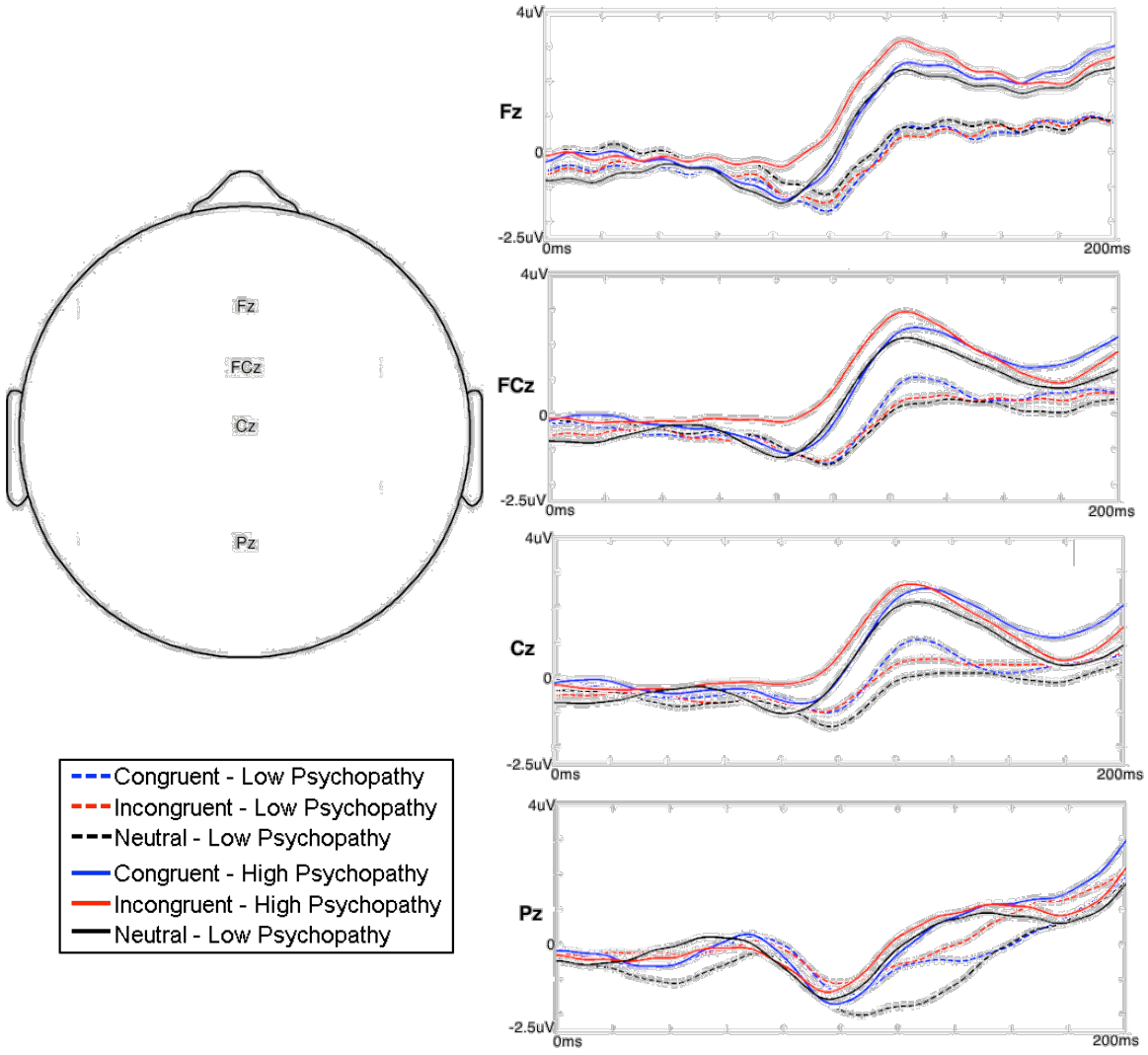

Figure 12. N100 waveforms by trial type for individuals with low and high psychopathy scores. The grand averages represent waveforms averaged across participants with PCL-R scores 1 standard deviation or more below the mean (low psychopathy group) and 1 standard deviation or more above the mean (high psychopathy group).

[16] To ensure that the effects of interest were not confounded by intelligence (computed based on scores on the vocabulary and matrix reasoning subtests of the Wechsler Adult Intelligence Scale (WAIS)), age, or education, we re-ran analyses with these variables as covariates. None of these variables had a significant effect on the behavioral or psychophysiological results.

Relationship between Psychophysiology and Behavior

An underlying premise of this study is that individuals high in psychopathy process peripheral information differently than those low in psychopathy. Accordingly, psychophysiological processes may operate differently for individuals high and low in psychopathy and the association between frontal N100 difference score and behavioral interference may vary by level of psychopathy. To clarify the relationship among these variables, we used GLM to analyze the effects of frontal N100 difference score (standardized), PCL-R score (mean-centered), and their interaction, on the dependent variable of interest, interference. The effect of frontal N100 difference score on interference was non-significant ($F(1, 85) = .43$, $p = .49$, $\eta_p^2 = .01$), suggesting that across the entire sample frontal N100 difference score was not predictive of behavioral interference. As noted above, there was a main effect of psychopathy ($F(1, 83) = 5.08$, $p = .03$, $\eta_p^2 = .06$, B = -.03), such that higher psychopathy scores were associated with less interference. However, this effect was qualified by a significant frontal N100 difference score by psychopathy interaction, $F(1, 83) = 4.84$, $p = .03$, $\eta_p^2 = .06$. Results revealed that the relationship between frontal N100 difference score and interference was moderated by level of psychopathy (see Figure 13). Whereas the magnitude of frontal N100 difference was significantly and positively related to interference among participants with low psychopathy scores (B = 21.57, $p = .02$), this frontal N100 difference was unrelated to interference in high scorers (B = -6.64, $p = .45$).

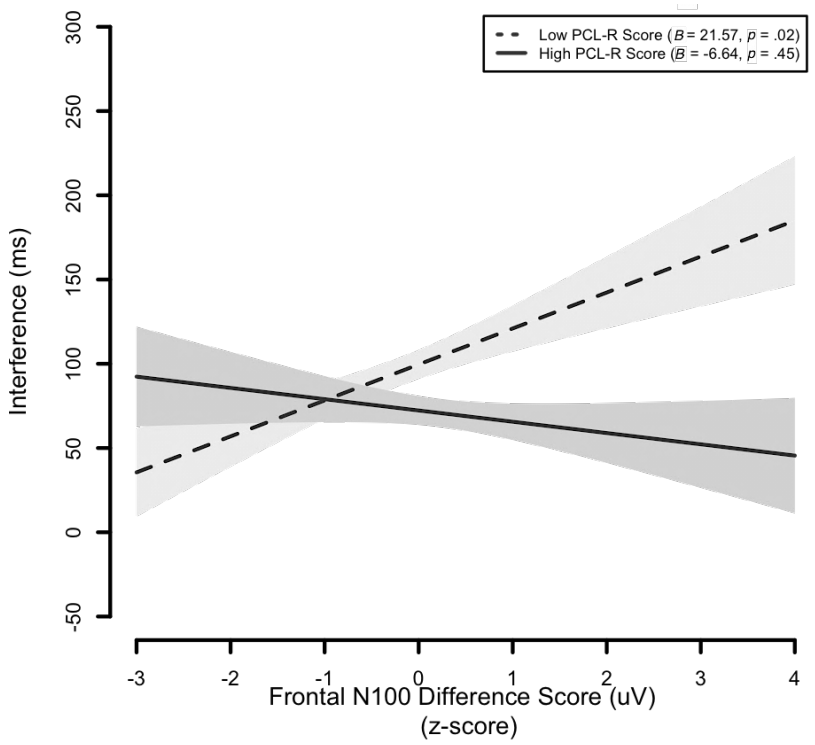

Figure 13. Interference as a function of frontal N100 difference score (Incongruent Fz N100 amplitude – Neutral Fz N100 amplitude) and psychopathy (±1 SD from the mean). Point estimates representing the average PCL-R score plus or minus 1 standard deviation were used to compute the effect of N100 difference score on interference by psychopathy level. This figure was generated using the R statistics and graphics program.

Supplemental Analyses

Total Stroop Effect. The primary aim of the investigation was to replicate the findings of Hiatt et al. (2004), who focused their analyses on the interference difference score (incongruent minus neutral reaction times). However, we also computed the Total Stroop Effect (TSE; Brown, Gore, & Pearson, 1998). This variable is defined as the difference in color-naming performance between incongruent and congruent stimuli. Better performance with congruent than with incongruent stimuli indicates that participants were less able to focus on naming the color than reading the word in the context of a task-incongruent word relative to a congruent word (see Donohue, Appelbaum, Park, Roberts, & Woldorff, 2013).

Behavior. Using regression, we examined TSE as a function of psychopathy (i.e., mean-centered PCL-R total scores). The analysis revealed a significant main effect of psychopathy on

TSE such that higher PCL-R ratings were negatively associated with the magnitude of the TSE, $F(1, 115) = 5.45$, $p = .02$, $\eta_p^2 = .05$ ($r = -.21$).

Psychophysiology. To assess the effect of psychopathy on psychophysiological correlates of the TSE, we computed N100 difference scores analogous to the TSE variable (abbreviated N100 TSE). Specifically, we subtracted the N100 amplitude on congruent trials from the N100 amplitude on incongruent trials for each of the four scalp sites. We entered these variables in a GLM as repeated measures with scalp site as a within-subjects categorical factor and PCL-R total score (mean-centered) as a between-subjects continuous factor. There was no main effect of scalp site on N100 TSE ($F(1.70, 146.05) = .40$, $p = .64$, $\eta_p^2 < .01$) nor main effect of psychopathy on N100 TSE, $F(1, 85) = .65$, $p = .42$, $\eta_p^2 = .01$. There was also no significant interaction between scalp site and psychopathy score on N100 TSE, $F(1.70, 144.19) = .14$, $p = .84$, $\eta_p^2 < .01$.

Two-Factor Model of Psychopathy. The primary goals of this study were to replicate and extend Hiatt et al.'s (2004) findings regarding PCL-R total scores. However, some researchers advocate decomposing psychopathy into two factors (Factor 1: Interpersonal/Affective; Factor 2: Impulsive/Antisocial) in order to examine the unique effects of these dimensions (Patrick, 2007). Accordingly, supplemental analyses were conducted to examine the effects of PCL-R Factors 1 and 2, rather than PCL-R total score, on behavioral interference and N100 difference score at Fz. These factors were mean-centered and entered simultaneously into regression models to quantify the unique effects of these constituents.

Interference. Analyzing behavioral interference within the two-factor framework demonstrated that the unique variance associated with Factor 1 was predictive of interference ($F(1, 114) = 4.81$, $p = .03$, $\eta_p^2 = .04$), while the unique effect of Factor 2 was non-significant, $F(1,$

114) = .14, p = .71, η_p^2< .01. Individuals with high scores on Factor 1 displayed significantly less interference than individuals with low Factor 1 scores.

Psychophysiology. Paralleling the effect for PCL-R total scores, analysis of the frontal (Fz) N100 difference scores revealed a trend-level association of Factor 1, $F(1,84) = 3.36$, p = .07, η_p^2= .04, B = .07. The relationship between Factor 2 and N100 difference score was non-significant, $F(1,84) = .00$, p = .99, η_p^2< .01).

Discussion

Psychopathic individuals fail to process all relevant information during goal-directed behavior, purportedly due to irregularities in early selective attention (Newman & Baskin-Sommers, 2011). The current study provides three key lines of evidence for the hypothesis that psychopathy is characterized by attention-related abnormalities that emerge early in the information processing stream. First, it demonstrates that higher levels of psychopathy are associated with reduced behavioral interference in the Box Stroop paradigm, a result that replicates Hiatt et al.'s (2004) finding. This finding supports the notion that psychopathy is characterized by abnormal information processing of goal-incongruent information.

Second, the results provide psychophysiological evidence that psychopathy-related differences on the Box Stroop task are apparent during the early stages of information processing. Specifically, our results for N100 recorded from the Fz electrode site demonstrated that individuals with higher PCL-R scores displayed smaller N100 responses to incongruent versus neutral stimuli. The fact that individuals high and low in psychopathy displayed differential cascades of information processing within approximately 100 ms of stimulus presentation is consistent with previous findings of early, psychopathy-related differences in information processing differences reported by Sadeh and Verona (2012), Baskin-Sommers et al.

(2012), and Veit et al. (2013). While these earlier studies found psychopathy-related differences in early ERPs in the context of affective stimulus processing, the present study complements these findings by demonstrating similar results in the context of a non-affective task containing incongruent information. The similarities between the results suggest that a comparable underlying process, such as reflexive orienting to salient stimuli, may account for these psychophysiological abnormalities (see Moul et al., 2012; Yantis & Jonides, 1990).

The third line of evidence for psychopathy-related differences in the processing of peripheral information concerns the relationship between the early psychophysiological differences and behavioral interference. As hypothesized, analysis of the association between frontal N100 difference score and interference across psychopathy levels revealed that highly psychopathic individuals do not exhibit the same relationship between N100 difference score and interference as individuals low in psychopathy. Specifically, for individuals with low PCL-R scores, there was a positive relationship between frontal N100 difference score and interference, such that larger N100 amplitudes to incongruent relative to neutral trials were associated with more behavioral interference; this relationship was virtually absent in high psychopathy individuals.

Given the pattern of this finding and previous research, we presume the relationships between frontal N100 difference score and interference in individuals low in psychopathy reflects normal information processing. In past work, researchers have linked the N100 component to early attentional engagement and orienting (see Esposito, Mulert, & Goebel, 2009; Mangun & Hillyard, 1991; Vogel & Luck, 2000; Zani & Proverbio, 2006). Moreover, smaller N100 amplitude may represent a failure to “gate in” sensory information, whereas larger N100 responses reflect attentional allocation to this information (Brenner et al., 2009; Lijffijt et al.,

2009). The positive relationship between interference and relatively larger incongruent N100 amplitude seen in low PCL-R scorers suggests that, among these individuals, larger N100 amplitudes reflect increased attentional allocation to incongruent information.

For individuals with high PCL-R scores, there was no relationship between the N100 difference score and interference. Although the absence of a direct relationship between the ERP data and interference among psychopathic individuals prohibits any definitive statement regarding the connection between brain and behavior, the significant interaction demonstrating that psychopathy moderates the association between frontal N100 and interference provides evidence that highly psychopathic individuals do not process conflict in the same manner as non-psychopathic individuals. Moreover, this difference is apparent within 100 ms of display onset. As such, it may be that the lack of a relationship between psychophysiological and behavioral measures is quite meaningful and indicates a unique pattern of processing among psychopathic individuals.

More specifically, the reduced frontal N100 response to incongruent versus neutral trials may reflect a failure to allocate attention to salient information. According to the Differential Amygdala Activation Model (DAAM; Moul et al., 2012), psychopathy may be characterized by basolateral amygdala (BLA) under-activation and central amygdala (CeA) over-activation. The BLA integrally mediates endogenous gaze shifting, and a relative imbalance of amygdala-based processes may disrupt the ability to reflexively shift attention to process peripheral bottom-up stimuli. Within the context of the current study, a failure to shift attention to the salient peripheral information (i.e., the color word) would enable psychopathic individuals to experience less conflict, thus resulting in decreased behavioral interference. It would not, however,

necessarily eliminate the facilitation effect, since semantic facilitation is a highly automatized process (see Balota, Yap, & Cortese, 2006).

One potential issue with the aforementioned interpretation is that it suggests a direct relationship between the N100 difference score and behavioral interference; however, no such relationship was found. An alternative, but not mutually exclusive, interpretation is that psychopathy may be characterized by a deficiency in integrative processes. There is increasing evidence of psychopathy-related variation in neural connectivity between regions such as the amygdala and prefrontal cortex (Craig et al., 2009; Glenn & Raine, 2008; Koenigs, Baskin-Sommers, Zeier, & Newman, 2011; Motzkin et al., 2011) and the insula and anterior cingulate cortex (Ly et al., 2012). Recently, researchers have speculated that reduced neural connectivity may be associated with less integrative processing of multi-component information (Hamilton et al., 2015). In psychopathy, abnormal patterns of connectivity may create a context whereby early brain responses are dissociated from later behavioral reactions. With regard to the current study, lack of behavioral interference and smaller frontal N100 are both indicative of weaker conflict registration in psychopathic individuals, yet their lack of association may reflect a more general problem integrating the products of multi-component processing. Thus, abnormalities in neural connectivity may account for the psychopathy-related differences present in the presented results.

Supplemental Analyses

Supplemental analyses revealed a negative association between psychopathy score and Total Stroop Effect (TSE). Within the context of the current task, a low TSE reflects similar reaction times to incongruent and congruent stimuli and is the sum of facilitation and interference effects. Typically, interference effects are greater than facilitation effects (MacLeod,

1991). Within the context of the current task, better (i.e., fast) performance in the incongruent condition manifests as a lower TSE. A low TSE suggests that a participant is able to pay attention to the box color despite the presence of conflicting information (see Donohue et al., 2013). The negative relationship between PCL-R total score and TSE, therefore, is consistent with the supposition that psychopathic individuals have anomalous selective attention.

While there was a significant relationship between psychopathy and the behavioral TSE score, there were no significant associations between PCL-R score and N100 TSE. Although on the surface this result may seem contradictory, it likely can be understood as a consequence of stimulus content. Specifically, one key difference between the congruent and neutral conditions (i.e., the control conditions used in the two different interference measures) is the presence of semantic content; congruent trials merit linguistic processing, whereas neutral trials do not. Given that both congruent and incongruent trials have meaningful semantic content, these trial types likely activate brain processes that are uninvolved in the neutral trials. The N100 amplitude differences in the incongruent versus neutral trials may thus relate to differences in the demand for semantic processing, with higher demand in the incongruent trials relative to the neutral trials. Accordingly, the psychopathy-related differences seen in the N100 difference scores (reflecting the discrepancy between incongruent and neutral trials) might relate to depth of semantic processing. Since the N100 TSE difference score relates to two trial types that involve semantic processing (i.e., incongruent and congruent), this variable would not show this difference in depth of processing. Consequently, at the brain level, psychopathy-related differences in semantic processing would manifest in the N100 difference score but not the N100 TSE score. The presence of the behavioral TSE effect may relate to the relative automaticity associated with semantic facilitation versus interference (see Balota et al., 2006).

In addition to analyzing the effects of psychopathy on ERPs and Box Stroop performance, we also examined the effects of the major PCL-R factors in order to clarify the psychopathy effects. Like the PCL-R total score, high Factor 1 scores were associated low levels of interference. Moreover, there was a positive trend-level association between Factor 1 and small frontal N100 difference scores. No brain or behavior differences were found for Factor 2. This link between Factor 1 scores and reduced interference is consistent with recent findings linking Factor 1 to superior selective attention (e.g., Baskin-Sommers, Zeier, & Newman, 2009; Dvorak-Bertsch, Curtin, Rubinstein, & Newman, 2009; Ishikawa, Raine, Lencz, Bihrle, & Lacasse, 2001; Racer et al., 2011). Moreover, the fact that the interference and N100 difference score effects found in this study are more closely related to the unique variance of Factor 1 versus Factor 2 suggests that they may be relevant for understanding the interpersonal and affective components of psychopathy. It additionally suggests that although Factor 1 is traditionally conceptualized as an affective-interpersonal deficit, it might be associated with broader information processing abnormalities that affect fear conditioning and associative learning (see Moul et al., 2012). The fact that the affective-interpersonal dimension of psychopathy was related to a lack of association between behavioral interference and psychophysiological correlates of attention alternatively suggests that a deficit in integrative processing may contribute to the expression of the affective and interpersonal symptoms of psychopathy as well as to their impulsive and antisocial symptoms (see Baskin-Sommers et al., 2012, 2011).

Limitations

A notable strength of this investigation was the inclusion of psychophysiological measures to clarify the timing of attention effects associated with psychopathy-related

differences on the processing of peripheral conflict information. However, the novelty of this application of psychophysiological techniques to the Box Stroop task creates problems of interpretation. In the absence of a substantial literature clarifying the meaning of the ERP components during performance on the Box Stroop task, interpretation of such results is necessarily ambiguous. Furthermore, it is not possible to ascertain whether participants were focusing on the color word or the rectangular frame. Nevertheless, one of the primary goals of this investigation was to examine the earliest observable waveform regardless of specific theoretical connotations. The fact that nonpsychopathic participants showed an association between their ERP responses and task performance suggests that the frontal N100 waveform is sensitive to peripheral conflict processing in these individuals. Future research is needed to address these concerns and allow for more confident interpretation of the abnormal selective attention displayed by psychopathic individuals on Box Stroop and other conflict-related tasks (see Wolf et al., 2012; Zeier et al., 2009; Zeier & Newman, 2013).

Potential concerns about the current investigation relate to minor analytical differences from Hiatt et al.'s (2004) study. Whereas Hiatt and colleagues evaluated this hypothesis using an extreme group approach, the current study employs dimensional analyses. Additionally, one of Hiatt et al.'s research questions centered on the interaction between psychopathy and anxiety, whereas the current study did not emphasize this variable. With regard to the analytic strategy, continuous analyses increase statistical power and preclude variability from being subsumed by categorization (Altman & Royston, 2006). The presented analyses do not focus on anxiety because Hiatt et al. observed less interference in psychopathic participants regardless of their level of anxiety (i.e., not just among primary psychopaths). Our different focus emphasizes our efforts to conceptually replicate Hiatt et al.'s finding. In other words, we aimed to show the

generality of the replication to demonstrate the robustness of the effect. Lastly, other than employing psychophysiological procedures (e.g., having participants wear an EEG cap), the methods between the two studies remained identical.

A final limitation of the current study relates to the exclusion of non-Caucasian inmates. Although this exclusion limits the generalizability of the findings, previous research suggests that non-Caucasian participants generally do not demonstrate similar laboratory correlates of psychopathy as Caucasian psychopathic individuals. In other words, the information processing deficiencies found in Caucasian psychopathic individuals do not appear to generalize to all racial groups (Baskin-Sommers, Newman, Sathasivam, & Curtin, 2011; Kosson, Smith, & Newman, 1990; Lorenz & Newman, 2002; Sullivan & Kosson, 2006). Future research should explore difference and similarities between psychophysiological correlates of selective attention in psychopathic individuals as a function of race.

Conclusion

On the whole, the present experiment contributes to the growing body of research demonstrating that psychopathy is characterized by abnormalities in the early stages of information processing as indexed by ERPs. The lack of relationship between early neural responses and behavioral responses seen in psychopaths suggests that individuals with and without psychopathy cannot be equated on a single metric for task performance or neuro-biological responses. More specifically, it suggests that individuals with psychopathy process information differently and their cognitive processes cannot be understood under the same framework as healthy individuals (e.g., Glenn, Raine, Schug, Young, & Hauser, 2009; Kiehl, 2006).

Modern models of self-regulation emphasize the crucial role of attention in determining what information is passed on for further processing or gated out of awareness (see Baumeister & Vohs, 2011; Kaplan & Berman, 2010). With regard to psychopathology, many individuals have difficulty suppressing stimulus-driven attention to salient cues, a bias that interferes with goal-directed behavior and social adjustment more generally (see MacCoon et al., 2004). Psychopathy appears to be characterized by an attentional bottleneck that, once established, hinders the processing of information that is peripheral to the current attentional focus. In other words, once a goal-related cue captures the attention of individuals with psychopathy, these individuals have difficulty reorienting to subsequent information that is inconsistent with or unrelated to their attentional set. Failure to rapidly integrate multiple streams of information, in turn, may undermine processing of motivationally salient cues that contraindicate goal-directed behavior and trigger the suspension of ongoing approach behavior (Patterson & Newman, 1993; Vuilleumier & Driver, 2007); these processes are the cornerstones of adaptive self-regulation. Ultimately, a better understanding of psychopaths' information processing deficits can aid in the formation of interventions, enhance self-regulation, and reduce recidivism in psychopathic offenders.

CHAPTER 5
GENERAL DISCUSSION

The central goals of this project were to (1) outline a novel theoretical perspective of psychopathic dysfunction that accounts for the shortcomings of existing models and accommodates burgeoning evidence of widespread brain abnormalities in psychopathy and (2) to test specific predictions based on that theoretical framework. As detailed in **Chapter 1**, the uniqueness of the II theory lies in its ability to simultaneously accommodate the emergent nature of cognitive and affective deficits in psychopathy, the widespread neurobiological abnormalities seen in psychopathic individuals, the broad range of traits that characterize psychopathy, the contextual variation of performance deficits, and the nature of psychopathic individuals' information processing style. Moreover, it makes specific, testable predictions and has strong implications for future research. The findings from the series of experiments presented in the previous chapters provide support for key predictions of the II theory using various methodologies. The study in **Chapter 2** demonstrated that psychopathy is associated with a unique information processing style characterized by a reduced capacity to process multicomponent perceptual information concurrently. The study in **Chapter 3** explored the impact of this information processing style on stimulus encoding and representational capacity; results suggest that difficulty processing multicomponent information in a simultaneous manner impairs early information encoding and adversely affects the formation and use of mental representations. The findings in **Chapter 4** confirm that psychopathy is distinguished by abnormal information processing of goal-incongruent information, provide psychophysiological evidence of cognitive abnormalities during the early stages of information processing, and suggest that psychopathy is associated with unique psychophysiological correlates. As a whole, the presented work provides a comprehensive context for understanding psychopathic

dysfunction that accounts for shortcomings of existing models and explains existing and discrepant behavioral and neurobiological findings within a parsimonious framework. It further provides empirical support for fundamental predictions of the II theory.

In the ensuing sections, I outline the clinical and developmental implications of the findings. I will then consider limitations of the presented work and outline areas of future research.

Clinical Significance

The findings of the study discussed in **Chapter 2** demonstrate that psychopathic individuals are relatively deficient at processing simultaneously-presented multicomponent information. The study in **Chapter 3** establishes how difficulty simultaneous processing complex information impairs information encoding and the formation of mental representations. Impairment in the formation of early perceptual representations precludes accurate and relevant predictive and comparative abilities, the appropriate attribution of salience to incoming data, and the ability to respond in an adaptive manner. These information processing limitations are important to consider in the development of treatment interventions to reduce psychopathic dysfunction because "if lower-level perceptual and/or attentional processes are degraded or abnormally biased, the brain will have difficulty adaptively performing other more complex multimodal operations, predictions, or decisions on the data" (Vinogradov, Fisher, & de Villers-Sidani, 2012, p. 53). Failure to address these factors will ultimately undermine the efficacy of any intervention.

Cognitive remediation offers a promising avenue through which various neurocognitive abilities can be improved. Cognitive remediation involves behavior-based training aimed at improving cognitive functioning through tasks exercising attention, working memory, social

cognition, and/or executive functioning (Medalia & Bowie, 2016). Cognitive remediation interventions can be delivered via computerized programs, through individual or group activities, or through neurofeedback. Cognitive remediation is effective at enhancing cognitive functioning in a range of psychiatric conditions, including schizophrenia (Bowie, Grossman, Gupta, Oyewumi, & Harvey, 2014; Chan, Hirai, & Tsoi, 2015), attention deficit-hyperactivity disorder (Chacko et al., 2014; Chevalier et al., 2017), anorexia nervosa (Tchanturia, Doris, Mountford, & Fleming, 2015; Tchanturia, Lounes, & Holttum, 2014), as well as others (e.g., Dickstein, Cushman, Kim, Weissman, & Wegbreit, 2015; Keshavan, Vinogradov, Rumsey, Sherrill, & Wagner, 2014; van Passel et al., 2016).

The aim of cognitive remediation techniques is to facilitate the re-organization of neural networks (Rabipour & Raz, 2012). Indeed, there is evidence that cognitive remediation alters neural network architecture. For instance, in addition to leading to improvements in concentration and cognitive control, attention training has been shown to enhance communication efficiency in executive attention networks in both preschool-aged children and children with attention deficit-hyperactivity disorder (Posner & Rothbart, 2007). There is further evidence that mindfulness-based cognitive remediation can increase functional connectivity within the dorsal and ventral attention networks (Taren et al., 2017). In individuals with schizophrenia, computerized cognitive training has been shown to increase interhemispheric information transfer between prefrontal cortices and to reduce aberrant activation in networks involved in the control of attention and working memory (central executive network) and in self-referential processing (default mode network; Penadés et al., 2013). Importantly, activity-dependent myelination and task-induced functional connectivity changes may take weeks or longer to occur (Taubert, Lohmann, Margulies, Villringer, & Ragert, 2011). In order for

cognitive remediation to induce robust changes in neural plasticity, neurocognitive training must be sufficiently extensive (Vinogradov et al., 2012). Given that cognitive remediation can ameliorate encoding and information processing deficits (Morimoto et al., 2014), it represents a potentially promising avenue for treatment of psychopathic dysfunction associated with inefficient information processing.

With regard to psychopathy, there is preliminary evidence that cognitive remediation training can mitigate psychopathy-related deficits in integrating contextual information. Baskin-Sommers and colleagues (2015) administered a cognitive remediation training program to a group of incarcerated criminal offenders who were high in psychopathic traits or high in externalizing traits without psychopathy. They randomly assigned participants to one of two conditions, which were designed to target specific cognitive-affective deficits thought to underlie each syndrome. Accordingly, half of the participants received an intervention focused on their deficit, while the other half engaged in an unmatched intervention. Based on the assumption that a core deficit in psychopathy is failure to attend to contextual information that is critical for self-regulation, the Attention to Context (ATC) training involved computerized tasks explicitly meant to encourage balancing attention to both primary and contextual information. In contrast, the Affective Cognitive Control (ACC) training involved tasks meant to improve cognitive control in the face of affective and motivationally salient information, a deficit seen in individuals with externalizing traits. Results indicated that participants enrolled in deficit-matched training improved on training tasks, and that their improved performance generalized to distinct post-training laboratory measures. These findings demonstrate preliminary success in ameliorating psychopathic dysfunction through the use of targeted cognitive remediation training. Cognitive

remediation aimed at rewiring aberrant neural network architecture in psychopathic individuals may therefore facilitate better integrative processing, thereby reducing behavioral deficits.

Developmental Implications

The II theory takes an inherently developmental perspective in considering the pathogenesis of psychopathic traits. Brain organization changes over the course of development (Hwang, Hallquist, & Luna, 2012; Menon, 2013). During the first two decades of life, synaptic pruning and strengthening of long-range white matter tracts cause widespread, coordinated change in brain connectivity. In contrast to the adult brain, the child brain is characterized by lower levels of hierarchical organization, a lack of multimodal functional hubs, and fewer long-range connections supporting integrative processing. These features enable increased network flexibility based on learning. Developmental processes result in changes in the structural and functional characteristics of the brain, which influence within- and between-network activity (Menon, 2013).

Neural network architecture undergoes experience-dependent alterations. The process of synaptic pruning is itself driven by the environment: connections that are most effectively entrained to the environment are strengthened, while connections that are not reinforced are eliminated (Balbernie, 2001). Importantly, the interplay between brain development and the environment is reciprocal: “the brain’s outputs influence its inputs, and these inputs in turn shape subsequent outputs” (Byrge, Sporns, & Smith, 2014, p. 4). In other words, experiences over the course of development influence what information is encoded, which over time alters brain network organization, which in turn influences behavior. This interaction between brain and behavior represents a developmental cascade that ultimately shapes adult neurobiology and behavioral tendencies.

The II theory suggests that psychopathic dysfunction is the result of interaction between an underlying biological deficit (i.e., impaired integration) and environmental factors that influence the development of antisocial tendencies (see **Chapter 1**). It is important to point out that it is currently unclear as to whether the proposed integrative deficit causes aberrant brain topography or if network abnormalities are independent of this deficit. While this is an important avenue of future research, the bidirectional nature of brain network formation and experiences mean that these factors interact irrespective of the causal relationship between them. Given the malleable nature of brain architecture in early childhood and the critical role early experiences play in neural development, early intervention with high-risk youth or children with callous-unemotional traits would likely facilitate optimal behavioral outcomes.

Limitations and Future Directions

The II theory's utility lies in its ability to guide research design, to integrate biological and behavioral data, and to potentially inform targeted interventions aimed at altering dysfunctional underlying processes. As a whole, the project provides direct empirical evidence supporting central premises of the II perspective. Despite the strengths of the studies, including using stringently classified psychopathic offenders as participants (PCL-R $\geq$ 30), targeted experimental paradigms meant to test specific cognitive processes, and multiple methodologies, it is important to consider potential limitations of the presented work.

First, while the proposed integrative deficit provides a parsimonious mechanism for the psychopathic syndrome, it is neither a necessary nor sufficient cause of psychopathy. A necessary cause is a process or state that must be present for a condition or event of interest to occur. In this case, the condition of interest is not solely dependent on the process or state. One salient example from the medical field is human papillomavirus (HPV). Infection with oncogenic

HPV is necessary for cervical cancer to occur. While cervical cancer is always preceded by this infection, the infection does not always lead to the development of cervical cancer (Bosch, Lorincz, Muñoz, Meijer, & Shah, 2002). In contrast, a sufficient cause is the minimum set of factors and circumstances that will produce the condition or event of interest. If these factors occur, the condition will inevitably develop. For instance, abnormally phosphorylated tau pathology is sufficient to cause dementia. In other words, dementia will inevitably occur in an individual with phosphorylated-tau neuropathology (see Ashraf et al., 2014).

Impaired integration and its associated neurobiological abnormalities are not necessary for psychopathy to occur because there are diverse causes of the syndrome. As a syndrome, 'psychopathy' is a descriptive term for a cluster of characteristic symptoms. Consistent with the concept of equifinality, numerous causal pathways can result in psychopathic symptoms, including lesions to the orbitofrontal and adjacent ventral mesial cortex (Koenigs & Tranel, 2006). Cases of acquired psychopathy are unlikely to share the same cognitive and neurobiological profile as developmental cases. Additionally, there are two recognized developmental pathways to psychopathy. Although the resulting variants are phenotypically similar, they are theorized to have distinct etiologies and are distinguished by differences in affective reactivity. Primary psychopathy is thought to reflect a constitutional deficit that hampers self-regulation and undermines affective processing (Lykken, 1995). It is associated with higher levels of interpersonal, affective, and behavioral traits and lower levels of anxiety (Lee & Salekin, 2010; Newman, MacCoon, Vaughn, & Sadeh, 2005). On the other hand, the hostile, callous behavior in secondary psychopathy is posited to reflect an environmentally-driven adaptation to factors such as parental rejection and abuse/neglect, particularly physical abuse (Dargis, Newman, & Koenigs, 2016; Poythress, Skeem, & Lilienfeld, 2006). Secondary

psychopathy is characterized by higher levels of neuroticism, autonomic arousal, impulsivity, and comorbid psychopathology (Kimonis, Frick, Cauffman, Goldweber, & Skeem, 2012; Skeem, Johansson, Andershed, Kerr, & Louden, 2007; Yildirim & Derksen, 2015). The II theory is thought to uniquely relate to the primary psychopathy subtype; impaired integration is conceptualized as an intrinsic vulnerability rather than an adaptive response to abuse and neglect. The heterogeneity within psychopathy speaks against a shared underlying deficit that is necessary for the development of psychopathic traits.

Impaired integration and its associated neurobiological abnormalities are not sufficient for psychopathy to occur because, as noted in **Chapter 1**, the theoretical deficit is posited to interact with environmental factors to determine behavioral manifestations. These factors include association with antisocial peers or community violence, which foster the development of antisocial goals (Fontaine, 2007; Visconti, Ladd, & Kochenderfer-Ladd, 2015). If the environment does not promote antisocial tendencies and drives, the impaired integration deficit is not expected to cause psychopathic traits.

The aforementioned points challenge the contribution of the II theory to the field of psychopathy and raise the question of why it is important to pursue. In response this argument, it is critical to separate the concepts of causality and utility. Just because a mechanism is not sufficient or necessary for the development of a condition does not mean that it does not contribute to the disorder or is not a level at which interventions can focus (Stehbens, 1985). For instance, smoking is neither a necessary or sufficient cause of lung cancer (Rothman & Greenland, 2005). Nevertheless, interventions aimed at decreasing smoking can lead to fewer cases of lung cancer, even if other factors remain the same. As in the case of lung cancer, it is likely that psychopathy is attributable to a confluence of causal factors, most of which are neither

necessary nor sufficient by themselves to cause the syndrome. By providing a specific cognitive mechanism through which psychopathic dysfunction can be understood, the II theory outlines a potential target for intervention.

A possible shortcoming of this project is that, while it outlines a promising mechanism for the psychopathic syndrome (i.e., impaired simultaneous processing), it does not clarify the cause of that mechanism. Future work should examine genetic (see Levine & Davidson, 2005; Posner & Rothbart, 2007) and epigenetic (e.g., Diwadkar, 2016; Muehlhan, Kirschbaum, Wittchen, & Alexander, 2015) factors that confer vulnerability to specific information processing limitations and/or neural network disruption. They should further explore the causal relationship between the proposed integrative deficit and attentional abnormalities in psychopathy to elucidate whether an inability to process information in parallel may contribute to an attention bottleneck that impacts downstream processing (see Newman & Baskin-Sommers, 2011) or vice versa. Further exploration of these distal influences can help more accurately characterize the confluence of factors involved in the development of psychopathy.

As aforementioned, the relationship between impaired integration and brain topography is unknown. Future studies could recruit children with callous-unemotional traits to see if they show simultaneous processing deficits. Studies could alternatively employ a longitudinal design to elucidate the temporal relationship between the purported integration deficit and aberrant neural network organization. These types of studies will be critical in specifying the development of psychopathic correlates and identifying sensitive periods for intervention.

While the studies in **Chapters 2 through 4** help clarify the nature of information processing abnormalities in psychopathy, they do not test integrative processing directly. Accordingly, future work could employ well-validated paradigms that explicitly vary the degree

to which integrative processing is necessary. One approach would be the use of a perceptual integration task (e.g., Liu, Wang, Zhou, Ding, & Luo, 2017) that varies the amount of information that is presented simultaneously or needs to be integrated. In this case, it would be expected that higher psychopathy scores would be associated with less accurate performance, particularly as integrative demands increase. Recent work shows that cross-frequency phase synchronization between the participating neuronal group underlies the coordination of distributed brain activity (Palva & Palva, 2017; Varela, Lachaux, Rodriguez, & Martinerie, 2001; Wang, Saalmann, Pinsk, Arcaro, & Kastner, 2012). The use of electroencephalogram (EEG), specifically EEG coherence during integrative tasks, therefore represents an additional metric for testing integrative processing. The use of multiple methodologies could further clarify the interactions between behavioral and neurophysiological measures.

Further investigation is necessary to determine if the proposed integrative deficit is specific to psychopathy or if it represents a more general vulnerability to psychopathology. MacCoon and colleagues (2004) proposed that the underlying deficit in psychopathy may relate to disruption of the context-appropriate balanced attention to dominant versus non-dominant cues. The authors acknowledged that this imbalance is not specific to psychopathy; rather, there are a variety of mechanisms underlying this deficit (e.g., decreased bottom-up activation in response to non-dominant cues versus over-allocation of top-down attentional resources to salient, dominant cues), which is apparent in several disorders such as anxiety and substance dependence (see also MacCoon, Abramson, Mezulis, Hankin, & Alloy, 2006; Maccoon & Newman, 2006). The impaired integration deficit may likewise be a transdiagnostic deficit, or it may represent a general diathesis for a range of psychopathology. Alternatively, it may be a

variant of normal human information processing. Accordingly, future research should explore this deficit cross-diagnostically.

An emerging area of research relates to the identification of network-based biomarkers to be applied clinically in diagnosing psychiatric conditions with neuroimaging (Bian, Xie, Topaloglu, & Cisler, 2013; Fried et al., 2017). Although the II theory offers specific predictions regarding network organization in psychopathy (see also **Appendix A**), application of network science to psychopathy is still in its infancy. Future studies could analyze weighted and/or directed functional or structural networks to better characterize psychopathy-related differences in information flow throughout the brain. Additionally, investigations into network architecture in psychopathy could employ a fine-grained brain-wide parcellation scheme to characterize connectivity abnormalities beyond the structures commonly implicated in psychopathic dysfunction. Lastly, prospective studies should explore integrative processing in diverse samples of individuals with psychopathic traits (e.g., women, individuals from different racial backgrounds, community versus forensic samples) so as to enhance the generalizability of the presented findings. Future research aiming to clarify and specify network topography and dynamics in psychopathy is critical for the identification of network-based biomarkers that will be of clinical and/or forensic utility.

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APPENDIX A
FUNCTIONAL NETWORK STRUCTURE IN PSYCHOPATHY: AN EXPLORATORY INVESTIGATION

The advent of modern neuroimaging techniques has spawned new insights into brain functioning, challenging localizationist perspectives of cognitive abilities with evidence that cognition and complex behavior rely on distributed neural networks (Biswal et al., 2010; Hallquist & Hillary, in press; Meehan & Bressler, 2012). The application of network science to neuroscience and the ensuing conceptualization of the brain as a complex network have generated new insights into the mechanistic understanding of the mind, brain, and behavior (Swanson & Lichtman, 2016). Recent research efforts are focused on generating a network map of the human brain to delineate normal and pathological configurations of structural and functional networks. Within the domain of neuropsychiatry, there is emerging evidence that a range of psychopathological disorders are characterized by perturbations in network functioning. These findings suggest that many psychiatric disorders can be understood as brain network pathologies (Bullmore & Bassett, 2011; He & Evans, 2010; Sepulcre, Sabuncu, & Johnson, 2012).

One disorder that is associated with a broad array of neurobiological abnormalities is psychopathy. Psychopathy is a clinical construct characterized by interpersonal, emotional, and behavioral traits such as egotism, superficial charm, shallow affect, deceitfulness, reckless impulsivity, and unmotivated antisociality (Cleckley, 1988; Hare, 2011; Hemphill et al., 1998). Although psychopathy is commonly conflated with the DSM-5 diagnosis antisocial personality disorder (ASPD) due to their overlapping associations with criminal behavior, psychopathy is unique from other forms of externalizing psychopathology through its profound deficits in social emotions, including guilt, empathy, and interpersonal attachment (Berg et al., 2013).

Importantly, psychopathic offenders have a disproportionate impact on the criminal justice system: they are two to three times more likely to be incarcerated, four times more likely to be convicted for criminal violence, four times more likely to violently reoffend, and perceivably less amenable to treatment compared to individuals without psychopathic traits (Hare, 1996; Harris, Rice, & Cormier, 1991; Hart & Hare, 1997; Radulović, 2012; Reidy, Kearns, & DeGue, 2013). Given the legal, emotional, and social costs of psychopathy, understanding the mechanisms underlying this syndrome is imperative (Kiehl & Hoffman, 2011; Reidy et al., 2015).

There is accumulating evidence that psychopathy is characterized by abnormalities in brain structure and function (Glenn & Raine, 2008). Perhaps some of the most cited findings regarding connectivity in psychopathy relate to disrupted connectivity within the paralimbic system and between this system and other structures (Craig et al., 2009; Stratton, Kiehl, & Hanlon, 2015; Volman et al., 2016; Wolf et al., 2015; Yoder, Porges, & Decety, 2015). Contreras-Rodríguez et al. (2015) demonstrated a frontal connectivity bias in psychopathy; specifically, they found that higher psychopathy scores were associated with increased functional activity within the dorsal prefrontal cortex. They also found reduced functional connectivity of prefrontal areas with limbic-paralimbic structures, as well as decreased connectivity between the insula and cingulate cortex. Psychopathy has also been shown to be associated with abnormal connectivity within reward circuitry. Specifically, psychopathic individuals show increased reward-related connectivity between the ventral striatum and dorsomedial prefrontal cortex during reward expectation (Geurts et al., 2016). Recently, Poeppl and colleagues (2018) conducted a meta-analysis synthesizing key findings of brain activity in psychopathy. They found that psychopathy was associated with decreased functional activity in regions implicated

in semantic processing and action execution (left and right lateral prefrontal cortices, respectively), social cognition (dorsomedial prefrontal cortex), and affective processing (right basolateral amygdala). Conversely, they found a positive relationship between psychopathy and activity in frontoinsular cortices, which the authors identified as regions functionally involved in cognitive reward processing and language processing.

From a network perspective, psychopathy is associated with alterations of intrinsic connectivity networks (Freeman et al., 2015; Juárez, Kiehl, & Calhoun, 2013; Motzkin et al., 2011; Seara-Cardoso & Viding, 2015; Sethi et al., 2015; Sheng et al., 2010). These networks are comprised of synchronous fluctuations in brain activity between distant cortical regions during task engagement or at rest (Fox et al., 2005; Laird et al., 2011; Rosenberg, Finn, Scheinost, Constable, & Chun, 2017; Van Dijk et al., 2009; Wisner, Atluri, Lim, & MacDonald III, 2013) and provide insight into the brain's inherent functional organization. Philippi et al. (2015) explored psychopathy-related differences in the resting-state functional connectivity of cortical networks. They found a negative correlation between total psychopathy score and connectivity between the lateral parietal cortex and dorsal anterior cingulate cortex, both of which are key components of a cingulo-frontal-parietal cognitive-attention network that supports attentional and motor control processes. Furthermore, they found that interpersonal-affective psychopathic traits were uniquely negatively correlated with connectivity in the default mode network (involved in internally-directed attention), the frontoparietal control (involved in externally-directed attention), and the salience/cingulo-opercular network (involved in salience detection and maintaining tonic alertness), whereas the impulsive-antisocial traits were positively correlated with connectivity in these networks. Yoder and colleagues (2015) examined functional connectivity in intrinsic connectivity networks during implicit and explicit moral evaluation

tasks. They found additional evidence for psychopathy-related reductions in functional connections to the salience network (see also Decety, Chen, Harenski, & Kiehl, 2013). They further showed that psychopathy was associated with increased recruitment of the dorsal attention network, a network involved in the top-down allocation of spatial attention, during explicit moral evaluations. A growing number of studies document abnormal network patterns in youth with psychopathic traits as well. Cohn and colleagues (2015) found that different juvenile psychopathic traits are associated with aberrant connectivity patterns in a variety of resting-state networks, including those involved in self-referential and moral processing, salience detection, and cognitive control (Finger et al., 2012; Thijssen & Kiehl, 2017; Yoder, Lahey, & Decety, 2016). Taken together, the presented findings suggest that psychopathy is characterized by wide-spread abnormalities in structural and functional networks.

Hamilton and colleagues' (2015) Impaired Integration (II) theory is a model that contextualizes neural networks abnormalities in psychopathy in a parsimonious account of psychopathic behavioral and neurobiological dysfunction. This theory proposes that the fundamental deficit in psychopathy is one of integration; it predicts that psychopathy is associated with reduced ability to rapidly process and synthesize multicomponent information simultaneously (Hamilton & Newman, 2016). A central assertion of the II theory is that psychopathy is characterized by aberrant network topology, which may contribute to and/or be the result of the purported integrative deficit. The II theory specifically predicts that psychopathy is associated with fewer long-range connections between networks and more local connections within networks critically involved in early survival and behavioral initiation (i.e., the dorsal attentional network). It further predicts reduced connectivity in and coordination with networks whose connection mature later in life, including the default mode (Fair et al., 2008), ventral

attention (Farrant & Uddin, 2015), and salience networks (Gao, Alcauter, Smith, Gilmore, & Lin, 2015; Menon, 2013). Widespread abnormalities in neural connectivity are thought to disrupt the coordination of information from divergent networks and hamper general information processing efficiency in psychopathy (Uddin et al., 2011).

Graph theory is a branch of mathematics that offers a targeted way to explore network structure of the brain, thereby providing a powerful tool to test key predictions of the II theory. To date, only a few studies have adopted graph theoretic approaches to analyzing network connectivity in psychopathy. However, there is emerging evidence that psychopathy is associated with reduced long-range functional links between multiple intrinsic connectivity networks and diminished network coordination (Philippi et al., 2015), in addition to alterations in neural communication as measured in different frequency bands (Tillem, van Dongen, Brazil, & Baskin-Sommers, 2018). On a local level, psychopathy has been associated with regional topology abnormalities in regions of the default mode network (Lindner, 2017) and reduced within-network connectivity and abnormal information flow in the frontal cortices (Yang et al., 2012). Overall, the presented results are consistent with the premise of the II theory that psychopathy is characterized by alterations in network architecture.

The current study seeks to expand upon these studies by investigating psychopathy-related differences in brain network architecture. To this end, we conducted a series of exploratory analyses using a data-driven functional network parcellation. Most existing studies use standardized regions of interest based on the structures or networks most commonly implicated in psychopathy. The shortcoming to this approach is that it yields limited information about connectivity since it focuses on several predefined regions and therefore precludes computation of global network measures. The selected functional parcellation is a brain-wide

scheme that partitions the cortex into seven large-scale brain systems that are homogeneous in their resting-state functional connectivity. Heuristically, these networks include visual, sensorimotor, limbic, dorsal attention, ventral attention, frontoparietal control, and default mode networks. An additional strength of the presented study is that it is guided by a theoretical framework that makes specific predictions regarding brain-wide connectivity patterns.

Based on the predictions of the II theory, we hypothesized that psychopathy would be associated with reduced long-range connectivity (as indexed by lower correlations between spatially-distinct networks, specifically cross-hemispheric networks), altered network structure (as indexed by graph metrics; see Table A-1 for a description of the measures of interest), and less integrated network functioning (as indexed by global graph metrics).

Methods

Participants

Participants were a subset of a larger sample recruited from a medium-security prison in central Wisconsin that have been reported on in previous work (Philippi et al., 2015). Participants consisted of 123 European American male inmates ages 20 to 54 (M = 30.75, SD = 7.10) whose functional imaging data met a conservative threshold for motion displacement (see Signal Preprocessing section below). To be included in the study, participants had to be between 18 and 55 years old, free of history of psychosis and bipolar disorder, not currently taking psychotropic medication, and have an IQ score of 70 or greater. Individuals meeting the inclusion criteria were invited to participate in an ongoing study. All participants provided written informed consent according to procedures approved by the University of Wisconsin – Madison Human Subjects Committee. On the first day of the study, inmates were called to a

private office and completed a semi-structured life history interview. This interview included questions on childhood, education, and occupational, interpersonal, and legal histories. Following the interview, the interviewer reviewed the institutional file to corroborate information provided during the interview. The combination of interview and file information was used to rate psychopathy according to Hare's Psychopathy Checklist-Revised (PCL-R; Hare, 2003). Seven participants were excluded for missing data, resulting in a final sample size of 116.

Psychopathy Checklist-Revised (PCL-R). We assessed psychopathy using Hare's Psychopathy Checklist-Revised (PCL-R; Hare, 2003). The PCL-R consists of 20 items that are rated according to the degree to which the characteristic is present (significantly = 2, moderately = 1, not at all = 0). In the current sample, scores on this measure ranged from 5 to 36, with a mean of 22.59 (SD = 8.05). Interrater reliability was assessed using intraclass correlation (ICC). Although dual, independent PCL-R ratings were available for only six of the participants in this study, ICC was .99.

BOLD / Imaging Data Acquisition

Resting-state fMRI images were acquired using a Siemens 1.5T Avanto Mobile MRI System with advanced SQ gradients (max slew rate = 200 T/m/s; 346 T/m/s vector summation; rise time = 200 μs) equipped with a 12-element head coil, property of the Mind Research Network. Head motion was restricted using padding and restraint. Participants were scanned on correctional facility grounds.

Resting-state functional images were collected while participants lay still and awake, passively viewing a fixation cross. T2*-weighted gradient-echo functional echo planar images (EPIs) were acquired with the following parameters: repetition time (TR) = 2000 ms; echo time (TE) = 39 ms; flip angle = 75°; field of view (FOV) = 24 × 24 cm; image matrix size = 64 × 64;

slice thickness = 4 mm; slice gap = 1 mm; voxel size = 3.75 × 3.75 × 5 mm^3; 27 sequential axial oblique slices; 158 volumes for a total of 5.5 minutes. Structural scans consisted of a T1-weighted anatomical image was acquired using a four-echo MP-RAGE sequence (TR/TE = 2530/1.64, 3.5, 5.36 and 7.22 ms; flip angle = 7°; FOV = 256 × 256 mm; image matrix = 128 × 128; slice thickness = 1.33 mm; voxel size = 1 × 1 × 1.33 mm; 128 interleaved sagittal slices). All four echoes were averaged into a single high-resolution image that was used for spatial normalization of EPI volumes and visualization of group statistics.

Signal Preprocessing

All fMRI data processing was performed using AFNI (Cox, 1996), FSL (FMRIB Software Library; Jenkinson, Beckmann, Behrens, Woolrich, & Smith, 2012), and FreeSurfer (Fischl et al., 2002). Preprocessing steps of EPI data consisted of: (1) slice-timing correction of EPI volumes using the first slice as a reference; (2) motion correction by rigid body alignment to the first EPI acquisition; (3) deobliquing of images; (4) omission of the first three volumes; (5) motion correction (3dvolreg function in AFNI) and (6) despiking to remove extreme, high frequency signal; and (7) bandpass filtering ($0.009 < f < 0.08$) and spatially smoothing with a 6 mm full-width at half-maximum Gaussian kernel (Fox et al., 2005).

Cortical reconstruction based on each subject's T1 anatomical image was performed using FreeSurfer's surface-based recon-all processing pipeline (http://surfer.nmr.mgh.harvard.edu/fswiki/recon-all). A population-based functional brain atlas (surfer.nmr.mgh.harvard.edu/fswiki/CorticalParcellation_Yeo2011) was resampled onto each individual subject’s cortical surface using FreeSurfer. Next, the unprocessed functional data were coregistered to the T1-weighted anatomical scans using FreeSurfer’s boundary-based registration (bbregister). The resultant transformation matrix would be used to align the EPI scans for each

participant to conformed FreeSurfer anatomical space (fsaverage) when projecting the volumetric data to the cortical surface. Subsequently, the data were denoised (i.e., WM, CSF, and 6 motion parameters were removed using fsl_glm). The residual data were then resampled onto the cortical surface (using FreeSurfer's mri_vol2surf). Finally, the timeseries of all vertices with each functional network node (see Defining Network Nodes section below) were averaged and organized into a 14 x 14 matrix.

Since individual differences in motion can contribute to resting-state correlations (Power, Schlaggar, & Petersen, 2015; Satterthwaite et al., 2017; Van Dijk, Sabuncu, & Buckner, 2012), motion was examined for each subject. We excluded subjects with: (1) mean framewise motion displacement (i.e., volume to volume movement across the time series) >2 mm, and/or (2) a total scan time of <4 min after censoring all time points with framewise motion displacement and extreme time series displacement ((i.e., time points in which >10% of voxels were outliers; Power, Barnes, Snyder, Schlaggar, & Petersen, 2012; Yan et al., 2013). These thresholds were selected to provide conservative criteria for motion correction.

Normalized T1 anatomical images were segmented into gray matter, white matter, and CSF segments using FAST (FMRIB Automated Segmentation Tool) in FSL. White matter and CSF segments were used as masks to extract a representative time series from each tissue type for use in nuisance regression.

Network Construction

Defining Network Nodes. To define the brain nodes, we used a pre-defined functional atlas of the human cerebral cortex (http://surfer.nmr.mgh.harvard.edu/fswiki/CorticalParcellation_Yeo2011; Yeo et al., 2011). This cortical parcellation was derived from a large-scale resting-state study that clustered whole-brain

connectivity networks according to the similarity of regions' functional connectivity profiles. This procedure resulted in seven clusters, whose boundaries correspond with the known topographic boundaries of the following functional networks: visual, somatomotor, limbic, dorsal attention (DAN), ventral attention (VAN), frontoparietal control (FpCN), and default mode (DMN).

Approximate components of each of these networks include regions within the following areas: visual cortices for the visual network; primary motor and primary somatosensory cortices for the somatomotor network; posterior parietal cortex (near the intraparietal sulcus), frontal eye fields, and precentral ventral frontal region for the DAN; frontal opercula, inferior parietal lobules, dorsal anterior prefrontal cortices, medial frontal cortices, anterior cingulate cortices, left parietal operculum, left temporo-occipital cortex, right precentral ventral frontal region, and right temporo-parietal-occipital region for the VAN; temporal poles and orbitofrontal cortices for the limbic network; lateral prefrontal cortices, posterior medial prefrontal cortices, precunei, cingulate cortices, intraparietal lobules, and posterior temporal association regions for the FpCN; and medial prefrontal cortices, lateral frontal cortices, posterior cingulate cortices, lateral parietal cortices, right ventral prefrontal cortex, and left parahippocampal complex for the DMN.

Defining Network Edges. Network edges were defined as the unweighted, undirected, Fisher-z transformed Pearson correlation of the timeseries signals between each node. Given that negative edge values are neurobiologically meaningful in functional neuroimaging (Sporns & Betzel, 2016) and that transformation of negative edge weights (e.g., by setting them to zero or substituting their absolute magnitude) can distort network properties (Fornito, Zalesky, & Breakspear, 2013), negative edges were retained.

For each participant, we compiled all pairwise associations between nodes into a 14 x 14 connectivity matrix. In general, if the value of an element of this matrix exceeds a predefined threshold based on either edge sparsity, significance, or absolute correlation strength, an edge is assumed to exist; otherwise, no existence would be assumed. The constructed binary graph, consisting of 14 nodes and undirected edges between nodes, was binarized such that any matrix entry was equal to 1 if an edge value exceeded a given threshold; otherwise, entries were set to zero. In this case, a value of 1 is indicative of a functional connection between the two nodes, while a value of zero designates an absence of a functional connection between network nodes.

Given that thresholding methods differentially influence computed network topology (Fallani, Richiardi, Chavez, & Achard, 2014; Langer, Pedroni, & Jäncke, 2013; Van Wijk et al., 2010), we ran analyses using two different thresholding methods. First, a relative sparsity threshold was applied, such the association matrix was thresholded in a proportional way (e.g., a threshold of 0.2 means that 20% of the strongest connections are maintained as edges). This approach is commonly used to threshold functional connectomes (see Bielczyk et al., 2018). In contrast to other thresholding methods, this approach prevents the biasing of network measures by individual differences in edge density and ensures equal numbers of edges across participants (Garrison, Scheinost, Finn, Shen, & Constable, 2015; Ginestet, Nichols, Bullmore, & Simmons, 2011). Since the selection of thresholds influences connection density and therefore network parameters (Bullmore & Sporns, 2009; Langer et al., 2013; Van Wijk et al., 2010), we used a range of thresholds (from .20 to .30, in increments of .01). This range was chosen based on research demonstrating that this range optimizes graph metric stability across time-series lengths and is biologically plausible (Hale, Mayhew, Przezdzik, Arvanitis, & Bagshaw, 2014; Sporns, 2011).

A separate set of graph analyses were run on the correlation matrix thresholded at significance levels of $p < 0.05$ and $p < 0.01$. Significance-based thresholding fixes a statistical threshold above which the percentile if functional connectivity correlations can be considered significant (i.e. distant from the null hypothesis of no-connectivity). Unlike proportional thresholding, significance-based thresholding does not fix the same edge density in the connectivity graphs across participants. Given that psychopathy, like some forms of psychopathology, may be characterized by intrinsic differences in functional connectivity, imposing equal densities for graphs describing connectivity in both psychopathic and nonpsychopathic participants may lead to the inclusion of a greater number of weak, potentially spurious links in the group with weaker connectivity (Bordier, Nicolini, & Bifone, 2017). As a result, significance-based thresholding can avoid erroneous evaluations of network topology (Toppi et al., 2012). It is important to note that global, significance-based thresholding can result in a difference in average degree when using the same fixed threshold for all networks under study (Van Wijk et al., 2010). At sparse densities, it can result in disconnected graphs, which can impact the quantitative values of many network metrics and bias comparisons of network metrics between participants with connected versus disconnected networks (see Alexander-Bloch et al., 2010).

Network Metrics

Normal brain function relies on the balance between segregation and integration of divergent brain regions; these processes allow for effective information processing and rapid information transfer within and between the networks (Sporns, 2011). Various topological properties of functional brain networks subserve segregated and integrated processing. Functional segregation relates to a network's ability to process specialized information in

spatially distributed node clusters, while functional integration refers to both the ability of a network to combine information from distributed nodes and to the efficiency of global communication (Deco & Boly, 2015; Ren, Li, Taya, Thakor, & Bezerianos, 2017; Sporns, 2013a). Measures of influence provide an index of node or edge importance: they quantify the extent to which individual nodes or edges contribute to information flow within the network based on interconnectedness of those nodes or edges (Sporns, 2013b). Segregation (-), integration (+), and influence (i) can be measured through various local and global graph metrics.

Local Nodal Measures.

Degree (i). Degree is the number of edges incident upon a given node. A node with a high degree has a large number of edges (i.e., functional connections); therefore, there is there is more interaction between that node and other nodes. Degree indexes how well the node is locally embedded in the network and therefore the importance of that node for network functioning (Rubinov & Sporns, 2010).

Betweenness Centrality (i). Betweenness centrality is the fraction of all of the shortest paths in the network that pass through a given node. Bridging nodes that connect disparate parts of the network often have a high betweenness centrality. As with degree, betweenness centrality indexes a node's involvement in the network.

Edge Betweenness Centrality (i). Edge betweenness centrality is the number of the shortest paths that go through an edge in a network. An edge with a high edge betweenness centrality value represents a bridge-like connector between two parts of a network. Edge betweenness centrality therefore identifies functional connections in the network that are crucial for information flow (Borgatti, 2005).

Global Network Measures.

Average Clustering Coefficient (-). Average clustering coefficient quantifies the tendency of a network to be divided into clusters, or cliques. It represents the mean of the fraction of each node's neighbors that are neighbors of each other. Higher average clustering values suggest greater functional connectedness across the brain network (Boccaletti, Latora, Moreno, Chavez, & Hwang, 2006).

Characteristic Path Length (+). In network science, the shortest path between two nodes is the path that includes the fewest number of edges. Characteristic path length, or average shortest path length, is the average of all of the minimum number of edges necessary to travel from one node to another in the network. It is one of the three most robust measures of network topology. A short average path length (i.e., lower path length) facilitates the quick transfer of information and therefore more efficient global information processing (Fornito, Zalesky, & Bullmore, 2016).

Global Efficiency (+). Global efficiency provides a measure of how effectively nodes exchange information within a network. It quantifies the extent to which nodes communicate with distant nodes and indicates the efficacy of information exchange throughout the entire network. High global efficiency in functional brain networks reflects increased overall capacity for parallel information transfer and integrated processing among distributed components of the system (Bullmore & Sporns, 2012; van Den Heuvel & Pol, 2010)

Analyses

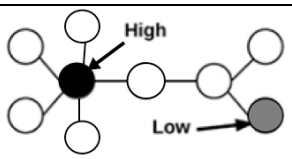
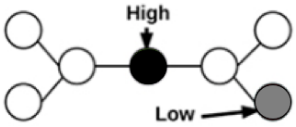
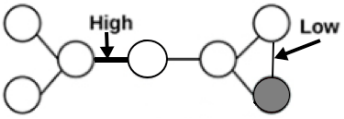
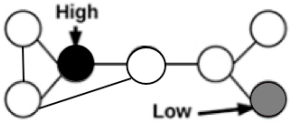
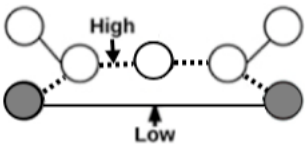
Analyses were conducted using the MATLAB GraphVar toolbox (version 2.01b; Kruschwitz, List, Waller, Rubinov, & Walter, 2015). First, we examined the correlation between

total psychopathy score and interregional connectivity (i.e., the raw connectivity matrix). Total psychopathy score was entered into a general linear model (GLM) as a continuously distributed between-subjects' factor, with Fisher-z transformed Pearson correlations between each node pair as the dependent variables.

The second set of analyses sought to explore the effect of psychopathy on the topological properties of the functional networks. To this end, we computed a series of local and global network graph metrics (see Table A-1) based on each participant's unweighted, undirected adjacency matrix and examined the association of each of these variables with total psychopathy score, controlling for the effect of IQ.

Benjamini–Hochberg false discovery rate (FDR) correction (Genovese, Lazar, & Nichols, 2002; McDonald, 2014) was applied to relevant analyses at a q value of 0.20.

Table A-1. Graph Theory Measures of Network Topology

Local Measures	**Definition**	**Illustration**
Degree	The number of edges incident upon a given node	
Betweenness Centrality	The number of times a node acts as a bridge along the shortest path between two other nodes	
Edge Betweenness Centrality	The number of shortest paths that pass through an edge	
Global Measures		
Average Clustering Coefficient	The proportion of neighbors of a given node that are connected to each other averaged across nodes	
Characteristic Path Length	The average of the shortest path lengths between all pairs of nodes in a network	
Global Efficiency	Inverse characteristic path length	

Preliminary Results

Association between Interregional Connectivity and Psychopathy

Figure A-1 depicts the association between total psychopathy score and Fisher-z transformed Pearson correlations between all possible two-way combinations of the 14 nodes.

Psychopathy score was associated with decreased functional connectivity between the left DAN and the right DMN (B = -.21, $t(112) = -2.24$, $p = .03$), the left DAN and the right FpCN (B = -.30, $t(112) = -2.84$, $p = .01$), and the left somatomotor network and the right FpCN, B = -.25, $t(112) = -2.68$, $p = .01$. There was a positive association between psychopathy and functional

connectivity between the left somatomotor and limbic networks, B = .22, $t(112) = 2.36$, $p = .02$, uncorrected. However, none of these psychopathy-related effects survived FDR correction.

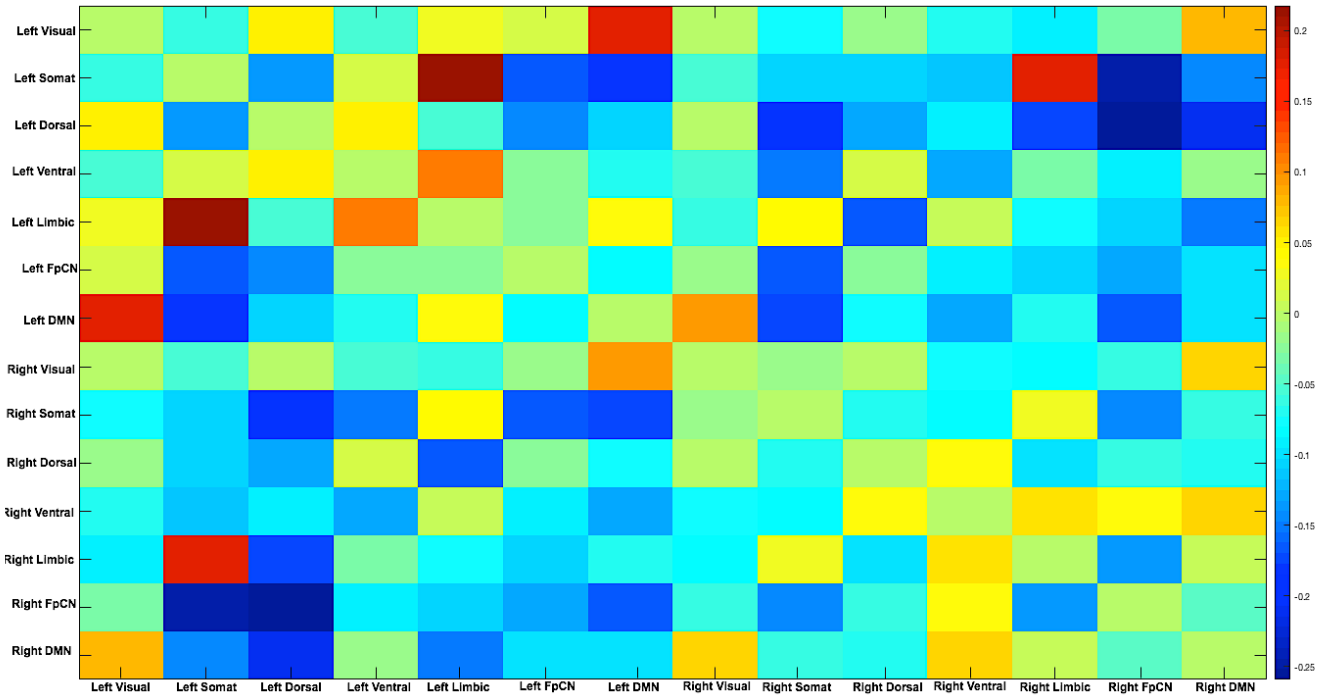


Figure A-1. Correlations between psychopathy score and regional correlation values

Association Between Measures of Network Organization and Psychopathy

Sparsity-Based Thresholded Graph. None of the associations between psychopathy and degree, psychopathy and betweenness centrality, or psychopathy and edge betweenness centrality survived FDR correction.

With regard to global network metrics, the association between psychopathy score and global efficiency was nonsignificant (B = .09, t(112) = .98, p = .33). The correlations between psychopathy and average clustering coefficient and psychopathy and characteristic path length were also nonsignificant, B = -.15, t(112) = -1.59, p = .11 and B = .12, $t(112) = 1.24$, $p = .22$.

Significance-Based Thresholded Graph. Psychopathy level was negatively associated with degree of the left and right somatomotor networks (B = -.26, *t*(112) = -2.84, *p* = .005 and B = -.25, *t*(112) = -2.74, *p* = .007) and with degree of the left FpCN (B = -.27, *t*(112) = -3.00, *p* = .003). There was a significant positive association between psychopathy and edge betweenness centrality in the right and left DAN (B = .31, t(112) = 3.50, p = .001), the left DAN and left somatomotor network (B = .25, t(112) = 2.79, p = .01), the left VAN and left DAN (B = .26, t(112) = 2.81, p = .01), the right VAN and left somatomotor (B = .12, t(112) = 2.53, p = .01), the left DMN and left visual network (B = .27, t(112) = 3.02, p = .003), and the left VAN and left visual network (B = .25, t(112) = 2.70, p = .01). Conversely, there was a negative association between psychopathy and edge betweenness centrality in the right DMN and left FpCN (B = -.28, t(112) = -3.13, p = .002). None of the associations between psychopathy and betweenness centrality survived FDR correction.

Psychopathy was significantly negatively correlated with average clustering coefficient, B = -.24, *t*(112) = -2.57, *p* = .01. There was a trend-level association between psychopathy and global efficiency, such that higher psychopathy scores were associated with lower global efficiency, B = -.18, *t*(112) = -1.92, *p* = .06. Additionally, there was a positive trend-level association between psychopathy and characteristic path length, B = .17, *t*(112) = 1.85, *p* = .07.

Discussion

The current study was an investigation into neural network organization in psychopathy. A graph theoretical approach was used for the analysis of the network data. We found preliminary support for the hypotheses that higher levels of psychopathy would be associated with reduced functional connectedness between far-range regions, aberrant network

organization, and reduced integrative properties. Specifically, analysis of interregional functional connectivity demonstrated that higher psychopathy scores tended to be negatively correlated with networks in contralateral hemispheres, with higher functional connectedness between networks in the same hemisphere. We found a similar pattern in centrality measures, with stronger correlations between psychopathy and the edge betweenness centrality in intra-hemispheric networks and weaker correlations between psychopathy and networks in contralateral hemispheres (e.g., the right DMN and left FpCN).

Generally, the psychopathy-related differences in edge betweenness centrality indicate atypical organization of network topology. Edge betweenness centrality indexes the relative importance of an edge within the overall architecture of a network. Psychopathy-related differences in interhemispheric edge betweenness centrality values indicates that interhemispheric functional connections may be less important for network functioning in high psychopathy participants. Moreover, the positive associations between psychopathy score and edge betweenness centrality in the left DAN, VAN, visual network, and somatomotor network suggests that functional connections between these networks play a critical role in efficient information transfer in high psychopathy individuals. The specificity of these findings to the left hemisphere may indicate atypical lateralization in psychopathy. The fact that the edge betweenness centrality in the DAN emerged as significant is consistent with the II theory's prediction of this network being intact due to its role in top-down attention (Hamilton et al., 2015).

Psychopathy-related variation in nodal degree further supports the relationship between psychopathy and aberrant neural network structure. This metric quantifies relative nodal importance in brain functioning, although high degree may not reflect nodal importance to

network function in functional connectivity networks because this metric can be strongly modulated by network size and because it fails to adequately capture a given node's unique connections (Power, Schlaggar, Lessov-Schlaggar, & Petersen, 2013). The negative association between psychopathy score and degree in left and right somatomotor networks and the left FpCN indicates that these networks are relatively functionally disconnected with neighboring networks. The low degree in the somatomotor networks appears counterintuitive, as externalizing symptomatology has been linked to increased degree in the sensory-motor network (dos Santos Siqueira et al., 2014). It is important to note that the somatomotor node used in this study is nonspecific and includes not only the somatosensory cortex and motor cortex, but also parts of the auditory cortex (Yeo et al., 2011). This lack of specificity precludes definitive interpretation of the presented findings. The negative association between psychopathy and degree in the left FpCN is largely consistent with research documenting reduced connectivity between the FpCN and other functional networks (Philippi et al., 2015). Wang and colleagues (2016) recognize the FpCN as a distributed hub that is critically involved in functional integration among systems. The negative correlation between psychopathy and degree in the left FpCN could reflect the fact that the left FpCN is less of a hub node in high psychopathy individuals. Overall, more research is necessary to clarify the importance of the somatomotor networks and FpCN for functioning in psychopathy.

The fact that higher psychopathy levels were significantly associated with reduced average clustering coefficient and showed a trend-level association with decreased global efficiency and increased characteristic path length suggests that psychopathy may be characterized by reduced small-world properties. Small-world organization is characterized by high levels of local clustering among nodes and low path length due to the presence of long-

range connections that globally link all nodes of the network (Bassett & Bullmore, 2006; Humphries & Gurney, 2008). Small-world topology can support both segregated and distributed information processing (Bassett & Bullmore, 2006). The network profile found for high psychopathy scores is consistent with more lattice-like network organization. Lattice network organization is a topology that supports more segregated processing due to high local clustering and high path length. This topology is associated with weaker computational power and synchronizability across distant regions (Watts & Strogatz, 1998). These findings have significant implications for how efficiently the brain integrates information between various functional networks and are consistent with the prediction of both altered global network structure and reduced integrative network features.

It is important to acknowledge the exploratory nature of this study, as it employs a course brain-wide parcellation scheme that identifies only 14 surface-based nodes. Yeo and colleagues (2011) advise that potential artifacts must be considered due to limited data resolution and MR susceptibility. The parcellation method does not distinguish between contributions of subregions within nodes, which is important considering that some networks contain correlated yet functionally distinct regions within some networks (e.g., the clustering of the somatomotor and auditory cortices). As a result, the presented findings are preliminary and therefore the offered interpretation is highly speculative. Overall, the presented study is offered as an illustration of how graph analyses may be used to examine the predictions of the II theory.

While the results are generally consistent with the predictions of the II theory, it is important to note that no relationships survived FDR correction for the graph that was thresholded based on sparsity level. There are a number of factors that could have reduced the strength of the findings. As aforementioned, sparsity-based thresholding equates edge density

across participants. The II theory predicts brain-wide alterations in network structure in psychopathic individuals, which may include the density of functional connections. Accordingly, it is may be that edge density is not equivalent across psychopathy level, and this method may have misrepresented real network topology. Moreover, the use of FDR correction to reduce false positives may be too stringent, and may result in false negatives (see Noble, 2009; Sanz-Arigita et al., 2010). A less conservative method, such as resampling-based tests, may be more appropriate for controlling for Type I error (Fallani et al., 2014).

One novel aspect of this investigation was its use of a data-driven parcellation scheme of the cortical surface. However, as aforementioned, the size of the graph is relatively small with only 14 nodes. Courser-grained divisions of the brain have greater potential to lead to false negatives as they may have not provided enough sensitivity to detect network alterations, particularly within-node differences. An associated consideration relates to the fact that the employed parcellation was based on the cortical surface, and therefore did not take into consideration subcortical structures that are frequently implicated in psychopathic dysfunction. Research suggests that structures such as the amygdala (Blair, 2003) and striatum (Glenn & Yang, 2012) are involved in the pathogenesis of psychopathy. Failure to isolate these regions may result in weaker findings.

Another potential limitation relates to the process of thresholding the connectivity matrix and its influence on inferred network properties. As noted, most graph theoretic measures depend on the number of nodes in the graph and connection density, and "a poor thresholding strategy can yield misleading results that artificially introduce significant differences or mask true differences in network topology" (Telesford, Simpson, Burdette, Hayasaka, & Laurienti, 2011, p. 297). To date, there is no universally accepted method for graph thresholding, as most

thresholding methods are associated with some degree of bias (Bullmore & Bassett, 2011). Given the lack of consensus regarding the optimal thresholding method, future studies should examine the effects of different thresholding techniques on psychopathy-related differences in brain topology to test the stability of the findings. Future research could also employ alternative analyze such as minimum spanning tree (van Dellen et al., 2018) or principal component analysis-based thresholding (Hanson, Westlye, & Lundervold, 2014) to overcome the thresholding problem. Biases introduced by thresholding must be taken into consideration when interpreting network parameters (Fornito et al., 2013; Langer et al., 2013; Van Wijk et al., 2010).

Although further research is necessary to replicate and expand upon the presented findings, the results provide direct preliminary evidence of aberrant functional network organization in psychopathy. As the first graph theoretical investigation of network properties in psychopathy that uses a data-driven parcellation scheme and is guided by the II theory, it represents a novel empirical contribution and stands as a pilot test of brain-wide network dynamics in psychopathy. Possible avenues of future research include the use of a more detailed parcellation scheme to better characterize within-network node functioning, exploration of structural network architecture in psychopathy, and the use of continuous weighted interregional correlations in the construction of brain networks (Achard, Salvador, Whitcher, Suckling, & Bullmore, 2006; Schwarz & McGonigle, 2011). Additionally, future studies could employ network science in psychopathy factor-level analyses (see Harpur et al., 1989) to characterize the association between psychopathy factor scores, their interaction, and network topology. By elucidating mechanisms underlying pathological cognition and behavior in psychopathy, the network perspective of brain functioning may be a useful tool in informing diagnostic

classification and the development of effective interventions targeting underlying dysfunctional processes.