

Impaired Integration in Psychopathy: A Unified Theory of Psychopathic Dysfunction

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This article introduces a novel theoretical framework for psychopathy that bridges dominant affective and cognitive models. According to the proposed impaired integration (II) framework of psychopathic dysfunction, topographical irregularities and abnormalities in neural connectivity in psychopathy hinder the complex process of information integration. Central to the II theory is the notion that psychopathic individuals are “wired up” differently” (Hare, Williamson, & Harpur, 1988, p. 87). Specific theoretical assumptions include decreased functioning of the Salience and Default Mode Networks, normal functioning in executive control networks, and less coordination and flexible switching between networks. Following a review of dominant models of psychopathy, we introduce our II theory as a parsimonious account of behavioral and brain irregularities in psychopathy. The II theory provides a unified theoretical framework for understanding psychopathic dysfunction and integrates principle tenets of affective and cognitive perspectives. Moreover, it accommodates evidence regarding connectivity abnormalities in psychopathy through its network theoretical perspective.

Keywords: psychopathy, integration, network theory, attention, emotion

In his seminal work *The Mask of Sanity*, Hervey Cleckley popularized the notion that psychopathy is characterized by “some subtle and profound defect” (Cleckley, 1941, p. 403) that underlies the distinguishing features of the disorder, namely glibness, impulsivity, irresponsibility, and egocentricity. Since the book’s publication, 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 and 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 seen in psychopathy (Kiehl, *in press*; 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 perspective. 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 re-

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sponses 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 preestablished focus of attention that psychopathic individuals show deficits (e.g., Baskin-Sommers et al., 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 nonaffective information processing deficits. Current attention-based models attempt to account for affective as well as nonaffective information processing deficits but have yet to integrate the rapidly growing evidence documenting brain 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 (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). The modulation of one system based on input from the other and the coordination of activity between different neural systems 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. Thus, acknowledgment of the emergent nature of these constructs is critical to their understanding. An integrative conceptualization 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 (Buckholz & 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 (Buckholz & 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 article 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 seeks to 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 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 nonfear related emotions and affectively neutral stimuli (see Newman & Brinkley, 1997).

Building on the low-fear model, Blair 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 integrated emotion system (IES) model (2006) 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; Blair, 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, Baird, & End, 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 nonaffective tasks.

Kiehl (in press) 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 OFC 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 nonaffective (e.g., linguistic) deficits seen in psychopathic individuals. However, its prediction of generalized paralimbic dysfunction suggests that psychopathic individuals should show global impairment on tasks involving the OFC, 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 emotion-focused models of 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, Schmitt, & Voss, 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 and decreased responsivity to cues that contraindicate current goal-directed behavior (e.g., Patterson & Newman, 1993). 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. Stimuli that normally initiate response evaluation and self-regulation are typically peripheral to a goal-directed focus of attention. These stimuli can include moral conventions, legal requirements, long-term motivations, and past experiences. To the extent that poor response modulation 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 would in turn disrupt the building of associative 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 enable 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 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, Curtin, and Newman (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. Baskin-Sommers and colleagues suggest that abnormalities in early selective attention obstruct the processing of information unrelated to a dominant response set. They propose that 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 prepotent focus of attention remains unelaborated (see MacCoon et al., 2004; Newman & Baskin-Sommers, 2011; Newman, Curtin, Bertsch, & Baskin-Sommers, 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 et al., 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 with psychopathic participants. Zeier, Maxwell, and Newman (2009) used a flanker-type task to test the effects of the bottleneck on the processing of peripheral information. According to the attention bottleneck theory, psychopathic participants would display significantly less interference to response incongruent information than nonpsychopathic 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 prepotent 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 also Zeier & Newman, 2013). Baskin-Sommers et al. (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 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 & Newman, 2014). Consequently, psychopathic individuals remain oblivious to these cues and do not use them to regulate behavioral and affective responses (Newman & Baskin-Sommers, 2011). The attention bottleneck model 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 (1996) left-hemisphere activation (LHA) hypothesis. This model proposes that psychopathic individuals’ dysregulated behavior re-

sults 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 as well as limited left hemisphere resources (see Hiatt & Newman, 2007). The LHA hypothesis similarly accounts for the situational nature of psychopathic dysfunction. However, it predicts global dysfunction during all tasks that tax left hemisphere resources; currently there is little support for global deficits in neuropsychological tasks that tap left hemisphere functioning (see Smith, Arnett, & Newman, 1992).

Moul, Killcross, and Dadds (2012) proposed the differential amygdala activation model (DAAM), a perspective that attributes emotional and cognitive dysfunction in psychopathy abnormalities in amygdala activation. The DAAM posits that reduced activation of the basolateral amygdala causes a deficit in reflexive shifts 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 scope 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; Koenigs et al., 2012; McCloskey, Phan, & Coccaro, 2005; Meffert et al., 2013). 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, Newman, Kiehl, & Koenigs, 2011; Philippi et al., 2015). Yang et al. (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 prevent the successful production of 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 nonaffective 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 present an overview of neural systems involved in information processing and outline the importance of neural networks for integrative cognition. We subsequently demonstrate how psychopathy can be conceptualized as a disorder of information integration that is associated with abnormal topographical patterns of neural connectivity.

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 multidimensional 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 temporoparietal, and limbic and paralimbic regions. 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 &

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	<ul style="list-style-type: none"> <input type="checkbox"/> Diminished fear responses <input type="checkbox"/> Failure to learn from punishment 	<ul style="list-style-type: none"> <input type="checkbox"/> Compelling account of psychopathic fearlessness <input type="checkbox"/> Accounts for characteristic fear conditioning and passive avoidance deficits 	<ul style="list-style-type: none"> <input type="checkbox"/> Does not address situational specificity of deficits <input type="checkbox"/> Does not specify mechanism <input type="checkbox"/> Does not explain deficits unrelated to fear
Blair's integrated emotion systems model	<ul style="list-style-type: none"> <input type="checkbox"/> Impaired affective processing <input type="checkbox"/> Reduced amygdala functioning 	<ul style="list-style-type: none"> <input type="checkbox"/> Links brain and behavior <input type="checkbox"/> Explains key affective deficits 	<ul style="list-style-type: none"> <input type="checkbox"/> Does not address situational specificity of deficits <input type="checkbox"/> Does not explain deficits unrelated to fear and distress
Kiehl's - hypothesis	<ul style="list-style-type: none"> <input type="checkbox"/> Paralimbic system hypoactivation <input type="checkbox"/> Poor performance on tasks involving paralimbic structures 	<ul style="list-style-type: none"> <input type="checkbox"/> Accommodates broad neurobiological abnormalities <input type="checkbox"/> Addresses nonaffective, as well as affective, deficits in psychopathy 	<ul style="list-style-type: none"> <input type="checkbox"/> Makes vague, nonspecific predictions regarding dysfunction <input type="checkbox"/> Does not address situational specificity of deficits
Newman's response modulation hypothesis	<ul style="list-style-type: none"> <input type="checkbox"/> Attentional moderation of information processing deficits (normal emotion processing if emotion is focal) <input type="checkbox"/> Shallow processing of associations due to lack of reflection 	<ul style="list-style-type: none"> <input type="checkbox"/> Addresses situational specificity of psychopathic dysfunction <input type="checkbox"/> Addresses nonaffective, as well as affective, deficits in psychopathy 	<ul style="list-style-type: none"> <input type="checkbox"/> Does not explain neurobiological abnormalities <input type="checkbox"/> Lacks defined neural substrate
Baskin-Sommers et al.'s attention bottleneck model	<ul style="list-style-type: none"> <input type="checkbox"/> Attentional moderation of information processing deficits (normal emotion processing if emotion is focal) <input type="checkbox"/> Sequential processing of multicomponent information 	<ul style="list-style-type: none"> <input type="checkbox"/> Addresses situational specificity of psychopathic dysfunction <input type="checkbox"/> Addresses nonaffective, as well as affective, deficits in psychopathy 	<ul style="list-style-type: none"> <input type="checkbox"/> Does not explain affective deficits when emotion is focal <input type="checkbox"/> Does not explain neurobiological abnormalities
Kosson's left hemisphere activation model	<ul style="list-style-type: none"> <input type="checkbox"/> Inefficient simultaneous processing <input type="checkbox"/> Dysfunction when substantial demand is placed on left hemisphere 	<ul style="list-style-type: none"> <input type="checkbox"/> Explains affective deficits when emotion is focal <input type="checkbox"/> Addresses situational specificity of psychopathic dysfunction 	<ul style="list-style-type: none"> <input type="checkbox"/> Lacks defined neural substrate <input type="checkbox"/> 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
Moul et al.'s differential amygdala activation model	<ul style="list-style-type: none"> <input type="checkbox"/> Impaired reorienting and salience detection <input type="checkbox"/> Context specific fear recognition, passive avoidance, and response reversal deficits <input type="checkbox"/> Hypofunctioning of the basolateral amygdala 	<ul style="list-style-type: none"> <input type="checkbox"/> Addresses nonaffective, as well as affective, deficits in psychopathy <input type="checkbox"/> Integrates affective and cognitive components of psychopathy <input type="checkbox"/> Parsimonious 	<ul style="list-style-type: none"> <input type="checkbox"/> Lack of specificity regarding with LH activation impairs performance <input type="checkbox"/> Does not address widespread neurobiological abnormalities <input type="checkbox"/> Does not address psychological correlates of psychopathy (e.g., information processing style) <input type="checkbox"/> Has not been extended to address other well-replicated performance deficits associated with psychopathy (e.g., language, memory)

(table continues)

Table 1 (continued)

Model	Predictions	Strengths	Weaknesses
Impaired integration theory	<ul style="list-style-type: none"> □ Abnormal connectivity undermines ability to integrate functioning of different neural systems, results in deficits in complex information integration □ Aberrant brain topography characterized by reduced global efficiency, fewer long-range connections between networks and more local connections, lower synchronicity, and increased modularity 	<ul style="list-style-type: none"> □ Addresses situational specificity of psychopathic dysfunction □ Addresses nonaffective (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 	<ul style="list-style-type: none"> □ 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

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). Direct interconnections between spatially remote brain regions increase the efficiency of information processing because 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 distant 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 has 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 (Buckholz & 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 et al., 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 rostralateral prefrontal cortex (rIPFC), middle frontal gyrus (MFG), anterior insula/frontal operculum (aIfO), dorsal anterior cingulate cortex (dACC), precuneus, and anterior inferior parietal lobule (aIPL; Spreng, Stevens, Chamberlain, Gilmore, & Schacter, 2010). 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 coactivated with the FpCN, the CoN is involved in the extended implementation and maintenance of task sets. It is comprised of regions in the dACC, dorsal anterior prefrontal cortex (daPFC), alFO, 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 toward the environment by generating top-down signals that bias sensory processing according to preexisting goals and expectations (Corbetta et al., 2008; Spreng et al., 2010, 2013).

Ventral attention network. The ventral attention network is activated by the presence of salient, task-relevant stimuli (Corbetta et al., 2008; Thiel et al., 2004; Vossel, Weidner, Driver, Friston, & Fink, 2012). It is a crucial mechanism of attentional disengagement, that prompts 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, Patel, & Shulman, 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 (Arnsten, 2009).

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 et al., 2003). This network consists of a distributed set of regions that includes the parietal association area (Buckner, Andrews-Hanna, & Schacter, 2008). It specifically includes the posterior cingulate/retrosplenial cortex (PCC/Rsp), medial prefrontal cortex (mPFC) and 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 (see 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 multidimensional 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.

Impaired Integration and Perceptual Binding

The II theory proposes that psychopathy is characterized by difficulty rapidly binding components of multidimensional sensory

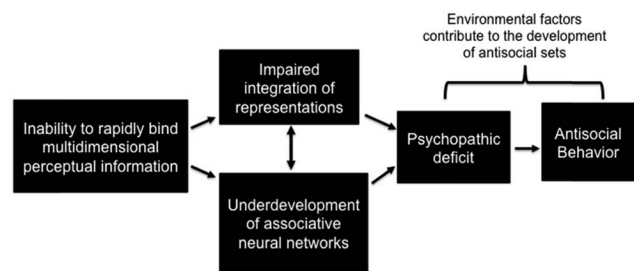


Figure 1. The impaired integration (II) theoretical framework. The II theory proposes that difficulty binding sensory features into a unified percept results in a perceptual bottleneck that fosters a sequential information processing style in psychopathic individuals. As a result, there are reciprocal developmental effects between information processing, learning, and neural connectivity such that white matter tracts within and between associative cortices display a unique topographical organization that contributes to 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.

stimuli. In general, perceptual processing involves the prioritization of stimuli based on stimulus salience and relevance. 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.

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 emotional and neutral words. 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. Because 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.

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 & Kosson, 1986). 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 Brain Topography

A perceptual bottleneck that undermines that ability to rapidly integrate multicomponent sensory information would shape 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 (see Figure 2).

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 primar-

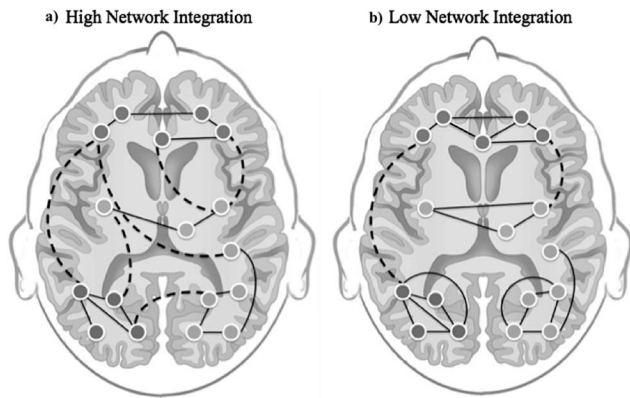


Figure 2. Graphic representation of brain topography supporting low and high integration. (a) Typical brain topography is characterized by a small-world architecture that enables efficient integration within and between neural networks. Normal human brains have abundant intracortical connections that 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. This topographical profile would encourage segregated rather than integrative processing.

ily modular structure (de Bie et al., 2012). Neonates display strong connectivity between parietal and frontal regions; these regions comprise the orienting attention network. Over the next 2 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 dPFC and posterior parietal regions offer adaptive control during developmental immaturity. This frontoparietal system enables flexible learning of novel stimuli, an executive process 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 CoN and 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., 2015) or biochemical process (e.g., Braver & Cohen, 1999; Rodriguez, Kallenbach, Singer, & Munk, 2004; Schnitzler & Gross, 2005) results in an impaired ability to rapidly integrate multicomponent information. This deficit, in turn, is suspected to cause the underdevelopment of connections within “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 (i.e., 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).

To date, few studies have used graph theoretical approaches to evaluate functional connectivity in psychopathy. Indeed, there are 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 et al. (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. (2015) 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 non-psychopaths (see also Sheng et al., 2010). These abnormalities may underlie the reduced ability to flexibly switch 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.

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 influences experience, and experience shapes the structures of neural systems throughout the life span (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” (see 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 involves less robust mirroring of others’ observed mental and bodily states (Atique, Erb, Gharabaghi, Grodd, & Anders, 2011; Hillebrandt, Dumontheil, Blakemore, & Roiser, 2013; Nummenmaa, Hirvonen, Parkkola, & Hietanen, 2008) and requires higher cognitive functions such as metacognition and mentalizing (Bernhardt & Singer, 2012; Shamay-Tsoory, 2011). Cox et al. (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 relatively higher levels 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 multicomponent information particularly challenging for psychopathic individuals, resulting in a failure to engage in these processes. This difficulty would subsequently 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, moral decision-making tasks activate the DMN. Reduced activity in this system is associated with utilitarian responding. 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; accordingly, affective processing falls secondary to aggressive 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, Gazzola, den Boer, Bartels, and Keysers (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, 2012) such that deliberate attempts to activate this circuitry would be successful (see 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, Geng, & Fink, 2014). A critical consequence of psychopathic individuals’ proposed connectivity deficit would be the disruption of the integration of information from lateralized attention networks and the alteration of 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 (Carter, Shulman, & Corbetta, 2010; Corbetta & Shulman, 2002; Montoya, 2009; Shulman et al., 2009). 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, Patel, & Shulman, 2008).

Deficient interhemispheric connectivity in psychopathy may account for disrupted coordination of the dorsal and ventral attention networks¹ (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; Banich, 1995b; Posner & Dehaene, 1994) and for 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 et al. (2003) found that psychopathic individuals have increased callosal length and reduced callosal thickness. Motzkin, Newman, Kiehl, and Koenigs (2011) further 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).

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. Because those cues are not attended, they do not get access to working memory for further processing, which consequently affects inter-

nal 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 toward cues that are nondominant. 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 tests 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, 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 and contradic-

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.

tory 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, 2014). 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, 2013). 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 (1941), 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 deficient 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, 1941, p. 47); rather, a cumulative consequences 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 multicomponent 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 et al. (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 & Newman, 2014;

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 (the impulsivity/lifestyle factor) may be uniquely associated with corticostriatal 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

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 (Muller et al., 2003; Öhman & Mineka, 2001; 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 (Buckholz & 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; Perez 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. Because 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.

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.

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 widespread deficits in neural connectivity. It could be that connectivity abnormalities are consequences rather than a 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, Curtin, and Newman (2015) provided preliminary evidence that cognitive remediation training can 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 connec-

tivity 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 biobehavioral 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 et al., 2010; Kiehl et al., 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, Sepulcre, Turner, Stevens, & Schacter, 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 analysis (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 overrate or underrate 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 taking into account neuroimaging data (Bullmore & Sporns, 2009). Future research linking the parameters of brain topography to cognition, affective, and behav-

ior 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 attentional 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. Specifically, 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' nonaffective 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 emotion 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.

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