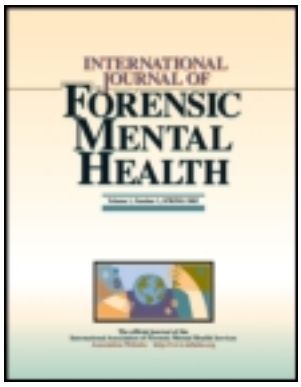

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Conceptualizing Psychopathy in Triarchic Terms: Implications for Treatment

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Clarifying the nature and origins of psychopathy is crucial to establishing effective methods for treating this severe form of pathology. The Triarchic Model of Psychopathy (Patrick, Fowles, & Krueger, 2009) is presented as a framework for resolving historic debates regarding the nature of psychopathy, and for guiding research on neurobiological influences contributing to its characteristic symptom picture. Evidence is reviewed for two distinct processes underlying "boldness" and "disinhibition" components of psychopathy in particular: underreactivity of the brain's defensive motivational system, and impairment in fronto-cortical regulatory circuitry. A third symptomatic facet, "meanness" (or callous-unemotionality) is theorized to reflect dysfunction in brain systems important for emotional empathy, and in endogenous neuromodulators such as oxytocin and vasopressin. We discuss how this variegated perspective on the nature and etiology of psychopathy can inform approaches to treatment. Specifically, focusing on feedback-based response modification and attentional retraining approaches as examples, we describe how specific neurobiologically-informed interventions might be developed to address distinct symptomatic components of psychopathy.

Keywords: psychopathy, triarchic model, boldness, meanness, disinhibition, biofeedback, attentional retraining

For many years, the dominant theoretical perspective on psychopathy has been that it is a unitary syndrome that arises from a core underlying pathology or deficit. Some etiologic theories of psychopathy have proposed that a basic deficit in emotional reactivity accounts for the characteristic affective, interpersonal, and behavioral features of the disorder; others have focused on disturbances in cognitive and attentional processing as the primary underlying cause. Here, we argue that progress in understanding this somewhat paradoxical condition—and, in particular, progress toward understanding influences that operate to spawn and maintain it—can be advanced by conceptualizing psychopathy in terms of separable phenotypic constructs that reflect differing sources of neurobiological influence.

A Triarchic model is described that characterizes psychopathy in terms of three distinguishable phenotypic facets, termed *disinhibition*, *boldness*, and *meanness*. Conceptualizing psychopathy in this differentiated manner can help to resolve historic debates about what psychopathy entails, and facilitate research on neurobiological processes associated with psychopathy and development of new methods for treating psychopathy that incorporate neurobiological insights. In particular, we review evidence for the role of two distinct processes in differing symptomatic features of psychopathy: underreactivity of the brain's defensive motivational system, which appears more relevant to affective-interpersonal features, and impairment in fronto-cortical regulatory circuitry, which appears more relevant to impulsive-antisocial features (Patrick & Bernat, 2009; Fowles & Dindo, 2009). Extrapolating from available data on behavioral and neurobiological correlates of the best-established measures of psychopathy currently in use, we consider the relevance of these two neurobiological processes to the constructs of the Triarchic model, and posit a role for separate influences in the emergence of meanness (or 'callous-unemotionality'; Frick & Marsee, 2006) specifically. We also consider how the Triarchic model, and available findings concerning the neurobiology of psychopathy as viewed through the lens of this model, can inform approaches to treatment of alternative variants of this high-impact clinical condition.

The paper begins with an overview of historic conceptions of psychopathy and contemporary assessment methods

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derived from these conceptions. The next major section describes the Triarchic Model (Patrick et al., 2009) as a framework for reconciling debates about what psychopathy is and clarifying how it should be measured and studied with respect to etiology. The third section discusses what is known about neurobiological influences contributing to psychopathy, and makes the case for differing brain mechanisms underlying observed deviations in emotional reactivity and cognitive-attentional processing. Distinct sources of neurobiological influence are considered in relation to constructs of the Triarchic model. The final major section discusses implications of material in preceding sections for the treatment of psychopathy, and considers how neurobiologically-informed interventions might be developed for targeting distinct symptomatic facets of psychopathy (cf. Seto & Quinsey, 2006), with particular emphasis on feedback-based response modification and attentional retraining approaches.

CONCEPTUALIZATION AND ASSESSMENT OF PSYCHOPATHY

Historic and Contemporary Conceptions

Alternative historic accounts have placed differing emphasis on distinguishable facets of psychopathy labeled disinhibition, boldness, and meanness in the Triarchic model (Patrick, 2010; Patrick et al., 2009). These accounts have generally portrayed psychopathy as a severe characterological condition that is highly resistant to treatment. The historic perspective that has influenced contemporary conceptions and assessment instruments the most is Hervey Cleckley's (1976/1941). His criteria for the disorder included indicators of behavioral deviancy and affective-interpersonal detachment, along with indicators of psychological stability (e.g., 'good intelligence,' lack of anxiety/neuroticism, high social efficacy). Notably, Cleckley did not describe psychopathic individuals as inherently violent or cruel, although he did view them as essentially untreatable. Cleckley's conception served as a referent for subsequent experimental studies of psychopathy that revealed evidence of deficits in physiological response to stressful or aversive events—beginning with David Lykken's (1957) classic study demonstrating reduced fear reactivity in youthful offenders high in psychopathy as defined by Cleckley, and continuing with work by Robert Hare and others in the 1960s and 1970s (for a review, see Arnett, 1997).

Whereas Cleckley's account of psychopathy was based on observations of psychiatric inpatients, other writers of his time who focused instead on criminal samples placed greater emphasis on callousness, cruelty, and exploitativeness. For example, McCord and McCord (1964) considered aggressive tendencies to be central to psychopathy—with rage substituting for fear in situations of provocation or threat, as a function of social disconnectedness more so than emotional incapacity. Along similar lines, Robins (1966) emphasized early and persistent aggressive antisocial deviance in her empirically-based account of youth who develop into adult "sociopaths," which served as the foundation for the diagnosis of antisocial personality disorder (ASPD) in the third and fourth editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III, DSM-IV; American Psychiatric Association, 1980, 2000). While the ASPD conception has been widely criticized (e.g., Hare & Hart, 1995) for deemphasizing core affective-interpersonal features, the proposed system for personality disorders in the upcoming fifth edition of the DSM (APA, 2012) includes provisions to address, at least in part, this shortcoming. Specifically, the DSM-5 conception of ASPD incorporates trait constructs from two distinct domains, labeled "antagonism" and "disinhibition." In parallel with this, a specifier has been proposed for child conduct disorder in DSM-5 to distinguish between psychopathic ("callous-unemotional") and nonpsychopathic (not "callous-unemotional") variants.

Methods for Assessing Psychopathy in Criminal and Non-Criminal Samples

Hare (1980) developed the Psychopathy Checklist-Revised (PCL) to address criticisms of the global rating approach to diagnosis used in his research up through the late 1970s. The current revised version (PCL-R) comprises 20 items scored on the basis of interview and collateral file information. The items of the PCL-R refer extensively to criminal acts/attitudes and capture the maladaptive features identified by Cleckley, but provide limited coverage of the positive adjustment features. The PCL-R contains distinct item subsets, which index affective, interpersonal, impulsive-irresponsible, and antisocial behavior features of psychopathy (Hare, 2003). There are a number of competing models regarding the structure of the PCL-R, which differ in the number of factors or facets identified, as well as the manner in which item subsets are interlinked, either in a higher order or in a correlated manner (for a review, see Cooke, Michie, & Skeem, 2007). Higher-order models specify an overarching latent "psychopathy" factor that accounts in part for differing symptomatic expressions, reflected in lower-order factors. Correlated models, on the other hand, characterize psychopathy as a multifaceted entity, encompassing distinguishable, albeit correlated, facets. The correlated 2-Factor model has been researched most extensively to date (Skeem et al., 2011). External correlates of Factor 1 include high narcissism, low empathy, and instrumental aggression (Hare, 2003). Controlling for overlap with Factor 2, PCL-R Factor 1 also shows positive and negative relations, respectively, with social dominance (e.g., Verona et al., 2001) and negative affective dispositions (Hicks & Patrick, 2006). Factor 2 is associated more exclusively with maladaptive tendencies, including early and persistent antisocial deviance, impulsivity, aggression (reactive, in particular), alcohol and drug problems, and general sensation seeking.

Of inventories that exist for assessing psychopathy in children and adolescents (Salekin, 2006), the Antisocial Process Screening Device (APSD; Frick & Hare, 2001) has been the most widely used and studied. Designed for use with clinic-referred children (ages 6–13 years) exhibiting behavioral problems, the APSD comprises 20 items rated either by parents or teachers that index an antisocial-aggressive variant of psychopathy, with limited coverage of adaptive features specified by Cleckley. Its items demarcate two distinct factors: Callous-Unemotionality (CU), reflecting emotional insensitivity and disregard for others, and Impulsive/Conduct Problems (I/CP), reflecting behavioral deviance, impulsivity, and inflated self-importance (Frick & Marsee, 2006). High I/CP children who lack CU tendencies show reduced intellectual ability along with high anxiety and emotional reactivity, and are prone to reactive (but not proactive) aggression. By contrast, children high on both APSD factors appear intellectually normal, are low in anxiety and neuroticism and less reactive to distressing stimuli, learn less readily from punishment and gravitate toward activities entailing novelty and risk, and show high levels of both proactive and reactive aggression with greater persistence over time.

The most extensively-validated measure for indexing psychopathy in non-forensic (community) samples is the self-report based Psychopathic Personality Inventory (PPI; original 187-item version, Lilienfeld & Andrews, 1996; revised 154-item version, Lilienfeld & Widows, 2005). The PPI was developed to comprehensively assess trait dispositions inherent in Cleckley's description, and includes eight subscales tapping lack of anxiousness (Stress Immunity), deficient fear (Fearlessness), dominance (Social Potency), impulsivity (Carefree Nonplanfulness), oppositionality (Rebellious Nonconformity), alienation (Blame Externalization), aggressive exploitativeness (Machiavellian Egocentricity), and lack of empathic concern (Coldheartedness). Factor analyses of the PPI subscales (e.g., Benning et al., 2003; Ross et al., 2009) have revealed two broad factors, one (Fearless Dominance, or PPI-FD; Benning et al., 2005a) defined by the Social Potency, Stress Immunity, and Fearlessness[1] subscales, and the other (Impulsive Antisociality, or PPI-IA; Benning et al., 2005a) by the Carefree Nonplanfulness, Rebellious Nonconformity, Blame Externalization, and Machiavellian Egocentrity scales. Scores on PPI-FD show positive associations with measures of agency and emotional stability along with narcissism and thrill seeking, and negative relations with measures of empathy, anxiousness, and depression (Benning et al., 2005a; Blonigen et al., 2005; Patrick et al., 2006b; Ross et al., 2009), and (as discussed in the next section) can be viewed as tapping the construct of boldness. In contrast, scores on PPI-IA are more uniformly indicative of deviancy—including child and adult antisocial behavior, high negative affect, impulsivity and aggressiveness, substance problems, and suicidal ideation—and can be viewed as predominantly indexing disinhibition (externalizing proneness). The eighth PPI subscale, Coldheartedness, does not load on either of these factors, indicating that it taps something distinct (i.e., callous-unemotionality or 'meanness'; see below).

In contrast with the PCL-R's correlated factors, the two factors of the PPI are uncorrelated (Benning et al., 2003). This difference can be traced to the differing strategies used to develop the two inventories: Whereas the PCL-R items were chosen to index psychopathy as a collective-unitary entity, the items of the PPI were chosen to index Cleckley's conception in terms of lower-order traits, without requiring trait scales to cohere in a unitary fashion. That the broad factors of the PPI emerge as uncorrelated suggests that psychopathy as described by Cleckley reflects a configuration or blending of disparate dispositions (i.e., a multifaceted construct), possibly arising from separate underlying etiologies (cf. Fowles & Dindo, 2009; Patrick & Bernat, 2009; Patrick et al., 2009)—as opposed to a unitary dispositional entity undergirded by a single, coherent etiology (i.e., a unitary, higher-order construct). These possibilities, which have important implications for treatment, are discussed further in the two major sections that follow.

Reconciling Contrasting Conceptions and Operationalizations: The Triarchic Model of Psychopathy

The Triarchic model of psychopathy was formulated as a framework for addressing lingering controversies and unresolved conceptual issues in the field (Patrick, 2010; Patrick et al., 2009). It can be viewed as a meta-conceptualization (i.e., an organizing descriptive framework for reconciling alternative existing conceptions and integrating findings across them) rather than a new theory of psychopathy or a replacement for existing conceptualizations. As such, the model can be useful for clarifying the nature of constructs embodied in differing measures of psychopathy, through delineation of their relations with constructs identified in the Triarchic model.

The Triarchic model views psychopathy as encompassing three distinguishable but intersecting symptomatic (phenotypic) components: disinhibition, boldness, and meanness. (*Note*: Labels for these component constructs were chosen to serve as readily recognizable descriptors; alternative descriptive labels for each construct are discussed below.) In contrast with other contemporary factor- or facet-oriented models of psychopathy (e.g., Cooke & Michie, 2001; Hare & Neumann, 2006; Frick & Marsee, 2006), the Triarchic model is a construct-based conceptualization not tied to any

[1]The Fearlessness subscale of the PPI loads on PPI-IA as well as PPI-FD (Benning et al., 2003; Ross et al., 2009). The reason appears to be that the items of this subscale reflect not just tendencies toward venturesomeness and tolerance of danger, but also boredom susceptibility and weak restraint; the venturesomeness component of PPI Fearlessness in particular accounts for its relationship with PPI-FD, whereas the boredom/weak restraint component accounts for its association with PPI-IA (Benning et al., 2005a).

particular assessment instrument. Existing psychopathy inventories can be viewed as indexing the Triarchic constructs to varying degrees and in differing ways. Additionally, in contrast with factor- or facet-oriented models embodied in specific inventories, the three components of the Triarchic Model are not considered elements or indicators of a unitary higher-order psychopathy construct. Rather, the Triarchic constructs are viewed as building blocks for alternative conceptions or variants of psychopathy described by historic and contemporary writers (i.e., psychiatric and criminologic, primary and secondary, successful and unsuccessful, aggressive and non-aggressive, anxious and non-anxious, etc.).

The following subsections discuss each of the Triarchic constructs in turn.

Disinhibition

"Disinhibition" refers to general proneness toward impulse control problems entailing a lack of planfulness, a focus on short-term goals and incentives, impaired regulation of affect and urges, and deficient behavioral restraint. Problems associated with this behavioral propensity—also termed externalizing proneness (Achenbach & Edelbrock, 1978; Krueger et al., 2002) or weak inhibitory ('effortful') control (Kochanska, 1997; Rothbart et al., 2007)—include child conduct disorder, adult antisocial behavior, angry (reactive) aggression, and substance use disorders (Krueger et al., 2002, 2007; Young et al., 2000). Historic accounts of psychopathy have emphasized this disinhibitory-externalizing component to varying degrees. Certain early writers defined psychopathy in terms that appear characteristic of externalizing individuals (e.g., Arieti, 1963; Partridge, 1928), and the concept of secondary psychopathy as described by Karpman (1941; see also Lykken, 1957) appears consistent with the clinical presentation of high externalizing individuals. Further, research over the past decade has shown that variance in PCL-R Factor 2 that is separate from Factor 1 largely reflects externalizing proneness (Patrick et al., 2005), as does the impulsive antisociality component of the PPI (PPI-IA; Blonigen et al., 2005). Based on its known correlates, the I/CP component of Frick and Hare's (2001) APSD also appears to index externalizing proneness.

However, researchers in the field would generally not consider disinhibition or externalizing per se to constitute psychopathy. Externalizing proneness is associated with high negative affect rather than deficient anxiety or fear (Krueger, 1999a), and with an elevated incidence of internalizing (anxiety, depressive) problems in childhood and adulthood (Achenbach & Edelbrock, 1978; Krueger, 1999b). Individuals of this sort have been viewed as amenable to treatment in part because of the presence of unwanted distress and dysphoria. By contrast, full ("primary") psychopathy is marked by salient "emotional detachment"—a lack of normal emotional sensitivity, along with impaired social relatedness (Cleckley, 1976; Lykken, 1995; McCord & McCord, 1964; Patrick & Bernat, 2009), that poses particular challenges for treatment. The Triarchic model proposes that this distinctive component of psychopathy reflects the presence of boldness or meanness, or a combination of the two. That is, it is when disinhibitory tendencies are coupled with dispositional boldness and/or meanness that a diagnosis of psychopathy is warranted.

Boldness

The term "boldness" encompasses social effectiveness, self-confidence, a high tolerance for risk and uncertainty, and the ability to remain composed in and recover quickly from stressful or threatening situations. Related concepts in the literature include fearless temperament (Kochanska, 1997; Lykken, 1995), fearless dominance (Benning et al., 2005a), and hardiness (Kobasa, 1979). In the Triarchic model boldness is viewed as related to, but not synonymous with, "fearlessness." The model conceives of fearlessness as an underlying constitutionally-based (genotypic) disposition, entailing reduced sensitivity of the brain's defensive motivational system to cues signaling threat or punishment (Fowles & Dindo, 2009; Kramer et al., 2012; Patrick et al., 2012). This genotypic disposition can be expressed in differing ways, with boldness representing one phenotypic expression. As discussed below, dispositional fearlessness may also contribute to phenotypic meanness.

Cleckley's characterization of psychopathy highlighted boldness—reflected in social charm, absence of anxiety or neurotic symptoms, failure to learn from punishment, lack of emotional sensitivity, and lack of proneness to suicide—in conjunction with disinhibitory tendencies. Characteristics of low fear and unresponsiveness to punishment were also emphasized in Hare's early psychophysiological research (cf. Hare, 1978) and in influential theories of psychopathy by Fowles (1980) and Lykken (1995). Contemporary inventories for assessing psychopathy emphasize boldness to differing degrees. Factor 1 of the PCL-R appears to index boldness to some extent (Benning et al., 2005a; Poythress et al., 2010), in particular through Interpersonal items of charm/glibness and grandiose sense of self-worth (Patrick et al., 2007)—features notably absent from the DSM conception of ASPD (Patrick, Venables, & Drislane, in press). The FD factor of the PPI indexes boldness as a more adaptive expression of dispositional fearlessness, dissociable from impulsive-antisociality, that may be of particular relevance to psychopathy in nonviolent, non-offender samples (cf. Lykken, 1995).

Meanness

"Meanness," which is more central to criminal-delinquent conceptions of psychopathy (McCord & McCord, 1964; Quay, 1964), encompasses deficient empathy, lack of close attachments, abrasivenness, exploitativeness, and empowerment through cruelty or destructiveness. In contrast with social withdrawal, meanness entails active resource-seeking

without concern for and at the expense of others ('agentic disaffiliation'; Patrick et al., 2009). Related terms include callous-unemotionality (Frick & Marsee, 2006), antagonism (Lynam & Derefinko, 2006), and cold-heartedness (Lilienfeld & Widows, 2005). Elements of meanness are represented in the PCL-R by items comprising its Affective facet (callousness, shallow affect, lack of remorse, failure to accept responsibility for actions). Elements of meanness are also evident to some extent in the PCL-R's Interpersonal items, which reflect tendencies toward toughness, superiority, and exploitativeness. Youth psychopathy inventories modeled after the PCL-R (APSD, PCL:Youth Version [PCL:YV; Forth, Kosson, & Hare, 2003], Child Psychopathy Scale [CPS; Lynam, 1997]) also emphasize meanness in their affective-interpersonal items.

An important conceptual and applied issue is whether meanness can be operationalized separately from criminal or antisocial behavior. In the PCL-R and measures modeled after it (including the PCL:YV, APSD, and CPS) meanness tends to be embedded in items that focus on antisocial acts or attitudes, or that blend meanness with boldness or disinhibition. One exception appears to be the Coldheartedness scale of the PPI, which indexes absence versus presence of emotional sensitivity and empathic concern. Recent work (Drislane et al., 2012) indicates that items from the Coldheartedness scale can be combined with selected items from other PPI subscales (e.g., Machiavellian Egocentricity, Fearlessness) into an index of meanness that correlates only modestly with indices of disinhibition and boldness. Another exception may be the Inventory for Callous-Unemotional Traits (Kimonis et al., 2008), developed to index affective-interpersonal features of psychopathy as represented in the APSD via self-report.

Further evidence that meanness can be separated from disinihibitory tendencies comes from recent quantitative modeling work on the structure of externalizing psychopathology and related traits (Krueger et al., 2007; Venables & Patrick, 2012). Findings of this work indicate that a disposition entailing low empathy and excitement-seeking contributes to aggressive behavior—particularly aggression involving predatory coercion and abuse of others—separately from general disinhibition proneness. Indicators of the "callous-aggression" factor in this modeling research closely parallel the item content of the CU factor of the APSD and its behavioral correlates, which include proactive aggression and thrill-seeking tendencies (Frick & Marsee, 2006).

Notably, other external correlates of the APSD CU factor (which include low levels of anxiety and neuroticism, weak responsiveness to threatening stimuli, and tolerance for unfamiliarity; Blair, 2006; Frick & Marsee, 2006; Marsh et al., 2008) suggest that low dispositional fear plays a role in phenotypic meanness. As discussed in the next section, it seems likely that other etiological factors combine with dispositional fearlessness to produce a harmful, maladaptive expression of this underlying disposition in the form of meanness, as compared to a more benign, adaptive expression in the form of boldness.

NEUROBIOLOGICAL INFLUENCES IN PSYCHOPATHY

In discussing neurobiological studies of psychopathy, it is important to consider some limitations in research of this kind. One is the assumption in much existing neurobiological research that psychopathy comprises a unitary diagnostic entity, analogous to a discrete physical disease, whose observable symptoms can be traced to a coherent underlying biological disturbance. As discussed in earlier sections, multiple lines of evidence indicate that psychopathy is not a unitary condition. It encompasses distinguishable symptomatic facets with differing (in some cases opposing) external correlates, and even individuals who score high on all facets appear heterogeneous in terms of trait dispositions (e.g., Hicks et al., 2004; Skeem et al., 2007) and physiological or behavioral response patterns (e.g., Newman et al., 1997; Sutton et al., 2002). Another limitation is the measurement gap that exists between indices of brain processes and diagnostic entities like psychopathy. Basic psychometric principles dictate that even indicators of the same construct will correlate only moderately when assessed in differing measurement domains. As indices of only somewhat related constructs assessed in differing domains, self-report or interview based measures of psychopathy and brain-based indices of affective or cognitive processes can be expected to correlate only *modestly*, at best. Yet another point is that neurobiological differences associated with psychopathy may reflect symptomatic correlates or sequelae of the disorder rather than underlying causal processes. For these reasons and others, it is important to bear in mind that while brain systems and processes provide valuable points of reference for advancing understanding of psychopathy and refining approaches to treatment, psychopathy and its facets will not ultimately be reducible to neural circuits or firing patterns.

Alternative Neuro-Etiological Perspectives

Various neurobiologically-oriented models have been advanced to account for the diagnostic condition of psychopathy (Blackburn, 2006; Patrick & Bernat, 2009). Broadly speaking, these can be grouped into two categories. One consists of theories that propose some underlying *emotional* deficit or deviation. For example, Cleckley (1976) postulated that psychopathic individuals are generally deficient in the capacity for affective experience, such that they learn to mimic the emotional reactions of others in order to interact effectively and achieve basic goals, without awareness of the artificiality their reactions. While most subsequent affective models of psychopathy have posited a more specific weakness in *negative* emotional reactivity, entailing

deficient anxiety or fear response (Fowles, 1980; Lykken, 1957; Hare, 1978), Blair (2006) proposed that psychopathy reflects a broader deficit arising from dysfunction in the amygdala, affecting both positive and negative emotions as postulated by Cleckley. This model has received support from studies demonstrating reduced autonomic and cortical reactivity to pleasurable stimuli in prisoners with high PCL-R scores (Verona et al., 2004; Williamson, Harpur, & Hare, 1991).

The other category of theories consists of those positing some sort of higher *cognitive* processing deficit in psychopathy. One proposed model entails deficits in psychopathic individuals' ability to shift attention from an ongoing action set to an alternative mode of response when appropriate (Newman et al., 1997; Patterson & Newman, 1993). A somewhat different perspective is that individuals with psychopathy have difficulty processing peripheral cues when attention is prioritized toward more central, goal-relevant cues—particularly under task conditions that promote activation of the left hemisphere (Kosson, 1996). Another cognitively oriented perspective is that the disjunction between words and actions in psychopathy reflects an underlying disturbance in language processing (Brinkley et al., 1999; Hare & McPherson, 1984).

A model that integrates these alternative etiologic models with reference to differing facets of psychopathy is the dual process model (Fowles & Dindo, 2009; Patrick & Bernat, 2009). The model posits that impairments in emotional response and in cognitive-attentional processing contribute differently to affective-interpersonal and antisocial deviance components of psychopathy. The two processes the model focuses on are dispositional fearlessness and weak inhibitory control. Dispositional fearlessness, considered more relevant to affective-interpersonal features, is theorized to reflect weakness of the brain's defensive motivational system, comprising the amygdala and affiliated structures. In contrast, the basis for the impulsive-antisocial component of psychopathy is theorized to be weak inhibitory control, or externalizing proneness. In neurobiological terms, this vulnerability is presumed to reflect deviations in the functioning of anterior brain systems including the prefrontal cortex and anterior cingulate cortex that operate to regulate emotion and guide decision-making and action.

Evidence for weak defensive reactivity underlying the affective-interpersonal features of psychopathy comes from research demonstrating a lack of startle reflex potentiation during explicit (visual) threat cuing in incarcerated offenders scoring high on Factor 1 of the PCL-R (cf. Patrick & Bernat, 2009). Other subsequent studies have replicated this finding in community participants scoring high on the Fearless-Dominance factor of the self-report based PPI (Benning, Patrick, & Iacono, 2005b). Regarding weak inhibitory control as a substrate for the antisocial deviance component of psychopathy, Patrick et al. (2005) documented a close association between scores on PCL-R Factor 2 and externalizing proneness, defined as the common factor linking child and adult symptoms of ASPD, substance-related problems, and disinhibitory personality traits. In turn, converging lines of evidence indicate that externalizing proneness reflects impairments in anterior brain systems that function to regulate affect and behavior in complex everyday contexts. One well-established brain response indicator of externalizing proneness is the P300 component of the event-related potential (Patrick et al., 2006a). Another is reduced amplitude of the error-related negativity (ERN), a cortical response that occurs following incorrect responses in a performance task (Hall et al., 2007). The finding of reduced ERN—indicative of a weakness in on-line error detection or performance monitoring—is reminiscent of Newman et al.'s (1997; Patterson & Newman, 1993) concept of deficient response modulation, entailing a failure to pause and reflect in the face of conflictual information or negative feedback.

A key question posed by this model is how to think about the contribution of these two distinct etiological processes to cases involving high scores on both factors of the PCL-R. Evidence addressing this question was provided by a study of offenders by Hicks et al. (2004), which identified two subgroups of high overall PCL-R scorers with markedly different personality profiles: a "stable" subgroup characterized by high agency (i.e., activity and social assertiveness) and low anxiousness, and an "aggressive" subgroup characterized by elevations on negative affective traits and reckless/impulsive tendencies. Notably, the personality profiles of these two subgroups mirrored patterns of traits known to be associated with boldness (as indexed by PPI-FD; Benning et al., 2003, 2005a) and externalizing proneness (Krueger, 1999a), respectively. The implication is that the two distinct mechanisms emphasized in the dual process model, dispositional fearlessness and externalizing proneness, are most relevant to the boldness and disinhibition facets, respectively, of the Triarchic model of psychopathy.

Less is known about the development and etiology of *meanness*, the third component of the Triarchic model. The major hypothesis that has been evaluated to date, in studies of children and adolescents, is that the variant of conduct disorder marked by the presence of callous-unemotional (CU) traits—a diagnostic concept similar to the construct of meanness—arises from a fearless temperament (Frick & Marsee, 2006), or a more general deficit in emotional capacity (Blair, 1995, 2006). However, it seems quite conceivable that a distinct process or set of processes related to the formation of empathy, affiliation, and nurturance also contributes to the emergence of meanness. The following section discusses existing evidence along these lines.

Distinct Neurobiological Substrates for Meanness

Externalizing disorders of various types in children, including attention deficit hyperactivity disorder (ADHD), substance use disorders, and conduct disorder (CD), have been

linked to deficits in inhibitory control (Iacono et al., 2008; Nigg, 2003). Extending previous knowledge regarding CD and other child externalizing disorders, major efforts have been devoted over the past 15 years to examining the role of affective-interpersonal features in CD that appear distinct from general externalizing proneness and reflect processes other than inhibitory deficits. In particular, systematic effort has been devoted to studying a distinct subgroup of youth who exhibit callous-unemotional (CU) traits in conjunction with impulsive conduct problems. As described earlier, the correlates of the CU factor of the APSD (Frick & Hare, 2001) suggest that the presence of CU traits is associated with diminished emotional reactivity on a variety of behavioral indices. In addition, functional neuroimaging studies have demonstrated reduced amygdala reactivity during viewing of fear faces in antisocial youth with CU traits (Marsh et al., 2008). Based on these and other findings, Frick and colleagues have hypothesized that CU traits in psychopathic youth represent a pathological expression of innate fearless temperament (Frick & Marsee, 2006).

Other researchers have postulated that psychopathy entails a more general deficit in emotional reactivity, as opposed to a specific deficit in fear. For example, Blair's violence inhibition mechanism model (1995) posits that psychopathy arises from a biologically-based disruption in the normal process of socialization. Being generally deficient in affective responding, psychopathic youth are unable to establish normal associative linkages between their own behaviors and distress cues such as fearful or sad facial expressions on the part of others. Consequently, they engage in antisocial behavior without distress and engage in instrumental forms of aggression without compunction. Supportive evidence for this model is provided by studies showing that psychopathic youth have difficulty in recognizing distress cues from others (e.g., fearful or sad vocalizations; Dolan & Fullam, 2006).

Research on the neurobiological bases of empathic concern is also of clear relevance to an understanding of processes that underlie callous-unemotionality or meanness. Decety and Jackson (2004) conceptualized empathy as a multifaceted tendency encompassing dissociable mechanisms, each underpinned by specific neural systems. According to these authors, the processes underlying empathy consist of: (1) reacting emotionally to the affective response of another person, (2) taking on the other's perspective, and (3) distinguishing one's own emotional response from that of the other. Disruptions in any of these processes can result in impaired empathy. For example, many individuals diagnosed with Autism Spectrum Disorders (ASDs) lack the cognitive capacity to understand the perspective of others. While able to resonate emotionally with others' suffering, they perform poorly on tasks that rely on intact 'Theory of Mind' (Baron-Cohen, 1989; Jones et al., 2010).

In contrast with findings for ASD children, Jones and colleagues (2010) have found, consistent with prior work on psychopathy (Dolan & Fullam, 2004; Richell et al., 2003), that boys high in psychopathy showed intact cognitive perspective-taking abilities, but deficient *emotional* empathy, as evidenced by lower levels of reported empathy for victims of aggression and lower reported fear when witnessing aggressive victimization. Deficits in emotional empathy as indexed by scores on the Questionnaire of Emotional Empathy (QEE; Mehrabian & Epstein, 1972), which assesses affective sensitivity to others, sympathetic tendencies, and the capacity to be moved by others' emotional experiences, have also been linked to higher scores on the IA factor of the PPI (Patrick et al., 2006b)—which indexes meanness to some degree along with disinhibition (Drislane et al., 2012). The Coldheartedness scale of the PPI, which (as noted earlier) is most clearly indicative of meanness, shows an even stronger relationship with QEE scores (Patrick et al., 2006b).

Decety and Jackson (2004) hypothesized that emotional empathy requires the activation of shared representations, that is, the activation of an internal representation pertaining to one's own behavior in the context of witnessing (or otherwise encountering) that same behavior in another person. Consistent with this, electrophysiological and neuroimaging studies have demonstrated that similar regions of the brain (in particular, regions of frontal and parietal cortex) are activated when imagining one's own behavior, when observing the same behavior by another person, and when imagining the same behavior by another (Clark et al., 2003; Hari et al., 1998; Ruby & Decety, 2001). Extrapolating from this perspective, it is conceivable that impairment in the ability to form shared representations of others' distress, or to activate these representations at appropriate times, contributes to phenotypic meanness.

Although the processes underlying shared representations are likely to involve multiple brain regions, certain brain regions may be particularly important for the capacity to form shared representations of others' distress. An example may be regions comprising the brain's pain system, including the thalamus and anterior cingulate cortex (ACC). Emotional resonance entails activating one's own pain evaluative networks in response to representations or depictions of the pain experienced by another person. Consequently, dysfunction in one's own pain network can compromise the ability to resonate with the pain or distress of others (Tucker, Luu, & Derryberry, 2005). Tucker et al. (2005) hypothesized further that brain regions comprising the pain network may also be involved in processes of affiliation and attachment—processes hypothesized to be impaired in individuals high in meanness (Patrick et al., 2009). Neuroscientific evidence indicates that the neural mechanism for pain in relation to social abandonment, which serves to motivate attachment behaviors, is similar to the mechanism of physical pain (Panksepp, 2003). Related to this, in the offspring of mammals, contact with the mother during times of distress induces endogenous opiate release, attenuating the experience of pain. From this perspective, one mechanism for the

lack of normal intrinsic motivation to seek out social contact in high callous-aggressive ("mean") individuals may entail dysfunction in pain processing networks.

A further key point is that neural systems implicated in the experience of pain and empathic capacity are subject to the influence of neuromodulatory hormones, including oxytocin and vasopressin (Donaldson & Young, 2008). In animal studies, these hormones have been shown to moderate social behaviors including social bonding, altruism, lactation, and sexual activity, and research has begun to delineate their role in more complex social behaviors like pair bonding. For example, recent work has implicated hormonal modulation of pathways related to reward and reinforcement, as well as those involved in the processing of social information, in elective pair bonding in rodent species such as prairie voles (Young & Wang, 2004). Thus, meadow voles who are by nature non-monogamous demonstrate the ability to form pair bonds if genetically engineered to have increased vasopressin receptor expression within the reward and reinforcement circuitry (Lim et al., 2004). In other work, Gobrogge et al. (2007) reported that, in male prairie voles that were pair-bonded for two weeks, dopamine and vasopressin expression in the anterior hypothalamus was associated with increased aggression toward other males and non pair-bonded females, presumably reflecting motives related to mate guarding. In humans, intranasal administration of oxytocin has been shown to improve men's ability to classify the emotional expressions of others (Domes et al., 2007), to increase gaze toward eye regions of human faces (Guastella et al., 2008), and to increase cooperative interactions in a "prisoner's dilemma" scenario (Kosfeld et al., 2005).

These findings suggest that neuromodulatory hormones including oxytocin and vasopressin may play an important role in the development of trust and close relationships, in humans as well as other mammals. Impairments in the production of such hormones, or hypoactivity of their receptor sites within the brain, could have a detrimental effect on affiliative processes. A valuable direction for future research will be to evaluate the role of hormones such as oxytocin and vassopressin in affective (i.e., deficient empathy), interpersonal (e.g., lack of close attachments), and behavioral tendencies (e.g., exploitativeness, proactive aggression) associated with the meanness component of psychopathy.

TRIARCHIC MODEL: IMPLICATIONS FOR TREATMENT OF PSYCHOPATHY

Historically, much of the discussion surrounding the treatment of psychopathy has been steeped in pessimism. As noted earlier, Cleckley (1976) expressed the belief that psychopathy was untreatable, and this perspective has persisted over the years (Salekin, 2002). Barriers to treatment do exist, particularly with regard to relatively lower motivation for traditional treatment among psychopathic individuals. However, a confluence of developments over the past several years makes the time ripe for reconsidering and advancing approaches to the treatment of psychopathy. First, advances in conceptualizing and measuring psychopathy and its distinguishable facets, and in identifying psychological, behavioral, and neurobiological correlates of these facets, suggest candidate mechanisms to target in treatment. Second, advances in the development of behavioral interventions to alter attentional processes and accompanying physiological activation patterns provide potential methods for targeting these mechanisms. This section focuses on approaches to intervention that may have potential utility for treatments of psychopathy, highlighting in particular those with ties to neurobiological concepts and findings. The proposed treatment approaches should be viewed, at this stage, as targets for research; none are ready for clinical implementation. Rather, the approaches discussed below are best viewed as avenues for applying advances made in psychophysiological and neurobiological research on psychopathy, a field that often seems divorced from practical applications.

New emerging perspectives on the etiology of psychopathy, including ideas about distinct processes underlying separable facets of the disorder, open up many new possibilities for treatment. Given limits of space, we limit our presentation here to two broad classes of intervention: (1) feedback-based response modification, and (2) cognitive and attentional bias retraining. We first define these interventions and then briefly review the current literature on each. We then provide some specific examples of how each type of intervention might be targeted toward altering specific processes associated with differing phenotypic facets of the Triarchic model—namely, boldness, disinhibition, and meanness.

Feedback-Based Response Modification

One class of intervention that may have relevance to the treatment of psychopathy is response modification with feedback provision. Feedback can be provided in a variety of forms, from information about behavioral performance to information regarding electrocortical (EEG/ERP) or functional brain (fMRI) response. Certain aspects of behavioral feedback distinguish it from biofeedback. Behavioral feedback entails the provision of information regarding performance on a particular trial sequence soon after the sequence is completed, or as it is being completed. Feedback of this type is commonly used in tasks that are cognitively demanding. One such task is the Flanker discrimination procedure, in which the participant is instructed to identify the direction of a central arrow that is surrounded either by arrows pointing in the same direction (< < < < <) or in the opposite direction (> > < > >), with feedback provided after trials in which an error is made. This represents an example of behavioral feedback.

Biofeedback, on the other hand, entails the online monitoring of some physiological or brain activity index and provision of information about activity to the participant in real-time (i.e., as activity occurs). This technique can be used

with any index of physiological activity that is amenable to real-time monitoring and display. The nature of physiological feedback provided varies widely, and may be as simple as having the participant view a screen displaying his or her raw heart rate (HR) and/or skin conductance response score (as in Steinberg & Schwartz, 1976). On the more complex end of the biofeedback spectrum, Ducharme and colleagues (2012) developed a HR biofeedback videogame modeled after the 1980s arcade game Space Invaders; in it the participant digitally moves a spaceship across the bottom of a screen and fires at a line of advancing enemy airships. The participant's HR is monitored as the game is played, and if HR rises above the level of a pre-task baseline, the participant's ship is disabled and cannot fire against oncoming enemies. The game was developed with the aim of reducing behavioral-affective anger problems in an adolescent participant, and preliminary success was reported with this single subject. Though still in its infancy, this type of paradigm is clearly novel, and deserves further investigation, particularly given that participants with high levels of disinhibition, such as those with psychopathy, may have difficulty engaging with more traditional forms of treatment.

Biofeedback paradigms focusing on EEG- or fMRI-based brain feedback have also been developed. In these tasks, individuals effortfully control designated brain responses with guidance provided by feedback. In the case of EEG biofeedback, participants receive ongoing information about amplitude or frequency characteristics of signals measured from the scalp surface as a referent for efforts to up- or down-regulate those signal characteristics. For example, in treatment of substance use disorders, alpha and theta frequencies have been targeted for up-regulation using biofeedback, with the aim of decreasing arousal in the face of alcohol or drug cues; this approach has been shown to increase periods of abstinence from drugs or alcohol (Sokhadze et al., 2008). EEG biofeedback has also been widely investigated as a treatment for anxiety disorders, using differing EEG frequency bands as targets for feedback (see Moore, 2001, for a review). The effortful control of alpha EEG frequencies has also been applied to the treatment of ADHD and traumatic brain injury (TBI). Essentially, any parameter of EEG response that can be quantified in scalar form continuously across time can be targeted for up- or down-regulation using EEG biofeedback.

Biofeedback using fMRI can enable participants to learn to effortfully control levels of activation in specific brain areas. Weiskopf and colleagues (2003) demonstrated that healthy volunteers were able to effectively modulate activity in rostral-ventral and dorsal anterior cingulate cortices—regions implicated in affective dysregulation and externalizing proneness (Hall et al., 2007; Patrick & Bernat, 2009). Because it can be used to teach people how to modify their brain reactivity in a concrete, quantifiable way, fMRI biofeedback holds potential for treatment of disorders that are characterized by increased or decreased brain reactivity in some distinct brain region. As a 'proof of concept,' fMRI biofeedback has been applied successfully in the treatment of chronic pain (deCharms et al., 2005), and is currently under investigation as a treatment for chronic tinnitus (Haller et al., 2010).

Attentional Retraining

Attentional retraining procedures, which focus on training individuals to modify attentional biases through provision of subtle reward cues, comprise a newer class of interventions that have been developed primarily in the context of treating anxiety disorders (see meta-analytic review by Hakamata et al., 2010). Procedures of this kind typically utilize the dot probe paradigm as a means of assessing attentional and processing biases. In the dot probe paradigm, two stimuli (e.g., pictures or words) from different categories (e.g., neutral versus unpleasant) are presented simultaneously on a screen for a brief interval (~500 ms), followed immediately by either one or two dots presented in the prior location of one of the two stimuli. Participants indicate, through button or keyboard press and as quickly as possible, whether one or two dots appeared after the offset of task stimuli. This process is repeated over multiple trials, with the dot probe presented in the location of stimuli from each category 50% of the time.

The tendency of participants to attend to stimuli of one type or the other is indexed by comparing average reaction time for trials when the dot probe is presented at locations of one (e.g., negatively valenced word) relative to the other (neutral word). A faster average reaction time for one type of stimulus compared to the other signifies that participants were generally attending more to stimuli of that type when the probe appeared. As one example of this effect, dot probe work has demonstrated, for example, that participants with generalized anxiety disorder, unlike healthy controls, preferentially attend to words that portray worrying content as compared to neutral words (Hazen, Vasey, & Schmidt, 2009).

Though the dot probe paradigm was designed to index existing attentional biases, the method can be used to modify such biases through inclusion of a retraining component. In attentional bias retraining, rather than presenting the probe at locations for each type of stimulus equally, the probe is presented most (e.g., 90%) of the time at the location of one type of stimulus versus the other. In this manner, attention is behaviorally shaped, with the probe's appearance serving as a confirmatory 'reward.' In the case of anxiety disorders, retraining is used to direct attention away from threatening or negatively valenced stimuli toward more neutral stimuli. Despite the apparent simplicity of the technique, a small number of sessions of this intervention have been shown to produce positive effects outside the treatment context, including symptom reduction, across a range of anxiety disorders (Hakamata et al., 2010).

The mechanisms accounting for symptomatic improvements using this approach are still under investigation (Koster et al., 2010), but are hypothesized to entail a cascade of

attentional, cognitive, and neural changes that together allow a client to reinterpret the overall stimulus context to emphasize neutral representations over threat-related representations (MacLeod et al., 2002). As an example, Eldar and Bar-Haim (2010) found that, among anxious participants trained to avoid threat-related stimuli through attentional retraining, electrocortical responses to threat stimuli were reduced—indicating an impact of retraining on neural processes underlying attentional bias.

Specific Interventions for Distinct Symptomatic Facets of Psychopathy

Boldness

Boldness as it relates to psychopathy is theorized to reflect the phenotypic expression of a heightened threshold for activation of the brain's defensive motivational system, resulting in reduced somatic and visceral activation to fear cues (e.g., Patrick & Bernat, 2009; Flor et al., 2002). Using fMRI biofeedback, Zotev and colleagues (2011) demonstrated that healthy volunteers are able to up-regulate activity in the region of the left amygdala through recollection of positive autobiographical memories. In view of Blair's (2006) hypothesis that psychopathy arises at least in part from dysfunction in the amygdala, these results point to the possibility of using fMRI biofeedback to promote enhancement of amygdala reactivity in high-psychopathic individuals. Training of this kind may be particularly effective if applied in contexts where defensive reactivity is seen to be diminished in psychopathy—for example, during viewing of threatening scenes or during anticipation of shock. However, because of the uncertain linkages between neurobiology and behavior, it cannot be assumed that up-regulation of amygdala reactivity will translate into reductions in psychopathic symptomatology; the link to behavior will need to before demonstrated empirically before interventions of this kind can be considered for implementation.

Autonomic arousal represents another potential target for treatment directed at deviations associated with the boldness component of psychopathy. It is well-known that psychopathic individuals show reduced skin conductance activation to emotionally-valenced stimuli, in particular aversive or threatening stimuli, in various contexts (Arnett, 1997; Patrick & Bernat, 2009). In a study conducted many years ago, Steinberg and Schwartz (1976) examined the ability of psychopathic individuals to self-regulate autonomic reactivity through use of electrodermal biofeedback and found that, after one 16-minute trial in which they were given auditory feedback to spontaneously occurring skin resistance responses, psychopathic participants were able to increase the amplitude of their skin conductance responses up to levels similar those of nonpsychopathic controls. It is conceivable that, with many more training sessions over a distributed period of time, autonomic reactivity could be durably enhanced, and perhaps affect daily behaviors in a positive manner as has been demonstrated with anxiety-related problems. For example, a variant of the biofeedback procedure developed by Ducharme et al. (2012), involving rewards in the form of points or in-game ability enhancement for large skin conductance responses to emotionally valenced stimuli, might be used for this purpose.

The literature on the benefits of attentional retraining to reduce bias toward threat cues in anxiety patients is of particular interest when considering the treatment of psychopathy. Conceptually, social anxiety can be viewed as the converse of psychopathy; behaviorally and physiologically, it appears to entail a *lower* rather than higher threshold for activation of the defensive motivational system (Stein et al., 2002). From this perspective, attentional retraining directed at establishing (versus eliminating) attentional biases toward threatening or aversive stimuli could prove beneficial as an intervention for psychopathy.

Disinhibition

The phenotypic construct of disinhibition can be indexed neurobiologically through deficiencies in top-down processes mediated by fronto-cortical brain structures. In particular, disinhibitory (externalizing) tendencies are associated with impairments in on-line detection of performance errors, as indexed by the ERN (Hall et al., 2007). The ACC, which is known to play a role in detection of conflict and online evaluation of performance, has been identified as the primary neural generator of the ERN. Although individuals high in externalizing proneness show reduced amplitude of ERN response in a standard flanker task, indicating reduced self-recognition of errors as they occur, other research demonstrates that high-disinhibited individuals do show normal amplitude of error-related negativity in response to explicit feedback about performance errors (Bernat et al., 2011). Given this evidence, it will be worthwhile to evaluate the potential effects of explicit feedback provided following erroneous choices over a training period (cf. Holroyd & Coles, 2002), enabling participants to learn over many trials which response options are correct versus incorrect. This might prove useful for ameliorating performance-monitoring deficits (and observed reduction of ERN) in high-disinhibited individuals. On a related note, work by Weiskopf et al. (2003) has demonstrated successful modulation of ACC activation through use of fMRI biofeedback in control participants. This suggests another method for normalizing brain processes relevant to performance monitoring in individuals with high levels of the disinhibition component of psychopathy.

Yet another cortical response variable with known associations to disinhibition is the P3 (or P300) component of the event-related potential. Amplitude of P3 response is known to be reduced in individuals who are high in general externalizing proneness (Patrick et al., 2006a). EEG biofeedback could provide a useful method for normalizing this component of brain response (and by extension, affiliated

cognitive-attentional processes). In practice, this could be tested by presenting of a reinforcing cue (such as an auditory tone, or an arrow pointing up) following occurrences of larger-than-average (that is, more normative) P3 responses during an oddball task procedure, with the aim of increasing P3 in high-disinhibited individuals. If this did lead to normalized P3 response, the next step would be looking for corresponding improvements in observable behavior outside the treatment context.

Meanness

As discussed earlier, one hypothesized pathway for the development of meanness consists of a lack of ability to form shared representations of pain and distress on the part of others. Accommodating this possibility, interventions aimed at ameliorating psychopathic meanness could focus on approaches that serve to foster emotional resonance. For example, provision of fMRI-based biofeedback to psychopathic individuals during viewing of negative emotional stimuli (e.g., fearful or sad faces, or depictions of victimization or injury) might prove effective for enhancing activation in specific brain regions related to pain and empathy, such as the amygdala, thalamus, and ACC. Along similar lines, psychopathic clients might be trained to increase their own emotional reactions to depictions of others' distress through provision of on-line feedback about autonomic (cardiac, electrodermal) arousal or somatic activation (e.g., reflex priming).

Another approach to intervention is suggested by evidence indicating that high callous-unemotional individuals have difficulty attending to and recognizing distress cues emitted by others (Dolan & Fullam, 2006). Attentional retraining therapy could prove valuable for helping psychopathic individuals, particularly children and adolescents, to recognize and attend to indicants of fear or sadness in the facial expressions of others. For example, a variant of the dot probe paradigm might be used in which probes occur systematically in the positions of distressed versus neutral face stimuli over the course of many trials. Training psychopathic individuals to attend to and resonate with the pain of others, in a procedure entailing direct brain measurement to confirm augmentation of activity in pain or distress regions of the brain as a function of treatment, could help to establish normative affective-representational capacities in such individuals and thereby curb behaviors directed at harming or exploiting other people.

SUMMARY AND CONCLUSIONS

To summarize major points covered in this review, compelling evidence exists for neurobiologically-based deficits in emotional reactivity and cognitive-attentional processing that contribute to observable (phenotypic) symptoms of psychopathy. The Triarchic Model of Psychopathy provides a useful framework for deconstructing psychopathy into constituent phenotypic components and clarifying neurobiological influences underlying these distinguishable components. Boldness, which is expressed phenotypically in the form of social poise, emotional resilience, and venturesomeness, has been linked experimentally to diminished responsiveness of the brain's defensive motivational system, comprising the amygdala and affiliated structures. As a function of a dispositionally fearless temperament, psychopathic individuals high in boldness fail to exhibit normal defensive mobilization when faced with aversive or threatening stimuli. In contrast, disinhibition, reflecting weak behavioral restraint and proness to externalizing problems of various types, is hypothesized to arise from impairments in anterior brain systems crucial to affective and behavioral control. High-disinhibited individuals perform poorly on tests of frontal lobe function and exhibit reduced brain potential responses (e.g. ERN, P300) in cognitive processing tasks. More speculatively, the meanness component of psychopathy, expressed phenotypically in terms of callous-aggressive behaviors and exploitative interpersonal relations, is hypothesized to reflect dysfunction in neural systems/processes that underlie emotional resonance and affiliative capacity. In sum, rather than comprising a discrete disease entity with a single underlying etiology, psychopathy appears to reflect the confluence of distinguishable symptomatic facets that reflect differing etiologic processes.

Advances in understanding neurobiological factors underlying distinguishable facets of psychopathy will contribute to refinement of existing approaches and development of new approaches to treating individuals diagnosed as psychopathic. In particular, methods utilizing feedback-based response modification and attentional retraining techniques may prove useful for modifying distinct symptomatic aspects of psychopathy through alteration of underlying neurobehavioral processes. For example, individuals high in dispositional boldness might be trained to up-regulate amygdala activity or attend more effectively to threat cues in the environment, as a means of countering deficiences in natural defensive reactivity. Similarly, interventions focusing on provision of immediate tangible feedback to high-disinhibited individuals regarding behavioral responses and affiliated neural processes may help to remedy impairments in effortful control. Correspondingly, interventions for meanness could be developed that incorporate developments in understanding of the neural bases of empathic concern and social affiliation. In particular, we encourage research directed at systematically evaluating the short- and long-term effects of attentional retraining and feedback-based response modifications on psychopathic features and affiliated behavioural outcomes. These treatments remain at the conceptual stage currently, but programmatic research directed at identifying distinguishable facets and distinct etiological (including neural) influences can provide an important vehicle for bridging the current gap between psychopathy's theoretical

underpinnings and its practical treatments. This is especially important given psychopathy's severity and its lingering reputation as untreatable.

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