Cognitive-Affective Processing Framework for Psychopathy Based on the Triarchic Psychopathy Model (TriPM)

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Date: 2021-04-22T00:00:00+00:00

Abstract

Psychopathy, as a significant predictor of violent crime, recidivism, and juvenile delinquency, has garnered considerable attention in clinical psychology and forensic domains. The triarchic model of psychopathy operationalizes the construct through three dimensions—boldness, meanness, and disinhibition—thereby reflecting cognitive-affective processing deficits at the neurobiological level. Specifically, low threat sensitivity constitutes the primary etiological foundation for the boldness dimension; impaired executive function, particularly attentional regulation deficits, underlies the disinhibition tendency; and empathy deficits linked to emotion recognition may represent the deep-seated cause of meanness. Future research must continue to address conceptual issues in psychopathy, investigate whether shared underlying etiological mechanisms exist across different psychopathic traits, and explore the early-life manifestations of these traits to facilitate timely and effective intervention.

Full Text

A Cognition-Affective Processing Framework for Psychopathy Based on the Triarchic Psychopathy Model (TriPM)

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Abstract

As an important predictor of violent crime, recidivism, and juvenile delinquency, psychopathy has received extensive attention in clinical psychology and forensic research. The Triarchic Model of Psychopathy operationalizes psychopathy

through three dimensions—boldness, meanness, and disinhibition—reflecting distinct cognitive-affective processing deficits at the neurobiological level. Specifically, low threat sensitivity constitutes the primary etiological basis for psychopathic boldness; impaired executive function, particularly deficits in attentional modulation, leads to disinhibited tendencies; and empathy deficits linked to emotion recognition may represent the underlying cause of meanness. Future research should continue to address conceptualization issues in psychopathy, investigate whether different psychopathic traits share common underlying etiological mechanisms, and explore the manifestation of psychopathic traits in early life stages to enable timely and effective intervention.

Keywords: Triarchic Model of Psychopathy, low threat sensitivity, impaired executive function, empathy deficits

Received: September 23, 2020
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Funding: No funding support

Psychopathy can be considered one of the "darkest"personalities in the domain of personality disorders. Cleckley (1964) described psychopathy as a syndrome and detailed 16 diagnostic criteria, primarily encompassing three aspects: (1) adaptive features that distinguish it from other mental disorders, such as normal or above-average intelligence, charm, absence of delusions, and low suicide risk; (2) behavioral deviations, including impulsive antisocial behavior, substance abuse, alcoholism, and lack of clear life plans; and (3) superficial emotions and insincerity, referring to characteristics of low anxiety, low guilt, lack of empathy, and inability to love. In psychology and psychiatry, psychopathy has garnered attention across numerous important research areas, such as interpersonal and affective deficits manifested as callousness, low anxiety, low fear, and impaired negative facial emotion perception (Hare & Neumann, 2008; Wilson et al., 2011), as well as attention deficits and hostile cognition leading to deviations from normal social norms in cognitive and behavioral patterns (Garofalo, Neumann, et al., 2020; Hare & Neumann, 2008; Smith & Lilienfeld, 2015). In applied forensic research, psychopathy also occupies a significant position. The Unified Theory of Crime (Delisi, 2016) even conceptualizes psychopathy as a precise manifestation of criminal behavior, involving violent crime, recidivism, and other serious socially impactful behaviors. This perspective treats psychopathy as a motivational factor for crime rather than a mere correlational relationship (Ireland et al., 2020). In social life, the cost imposed by psychopathic individuals on society is substantial—they frequently engage in antisocial behavior, and their likelihood of becoming recidivists is three to four times higher than that of ordinary criminals (Delisi, 2016). A global study of prison inmates found that psychopathy accounts for approximately 5–18% of male offenders, while another international study of adolescent male offender populations reported psychopathy diagnoses as high as 40% (Neumann et al., 2015). In community populations, although the proportion of psychopathy is only about 2.1%, these individuals are involved

in 18.7% of community violence incidents, with a population attributable risk of 16.6% (Coid & Yang, 2011).

Research on the etiology of psychopathy in clinical psychology can explain the pathological mechanisms underlying abnormal behavioral patterns in this population and enable effective intervention and treatment. Early scholars offered explanations for the etiology of psychopathy, with Karpman (1940, 1948) proposing two psychopathy subtypes: primary/idiopathic psychopathy, characterized by callousness, manipulativeness, extreme selfishness, and dishonesty; and secondary/symptomatic psychopathy, featuring more negative emotions, high harm avoidance, and impulsive tendencies when facing threats or danger. Anxiety serves as the key differentiator between these two subtypes. Primary psychopathy lacks negative emotional experiences such as anxiety and depression, whereas secondary psychopathy exhibits high trait anxiety levels. The two subtypes exhibit relative heterogeneity in underlying etiology, stemming from different developmental pathways. Primary psychopathy arises from specific genotypes and neurophysiological deficits causing idiopathic affective deficits, while the impulsivity and failed lifestyle of secondary psychopathy are more symptomatic, originating from childhood trauma and weak environmental adaptability.

Due to incomplete early conceptualization of psychopathy and lack of empirical research, many etiological explanations had inherent limitations. To this day, conceptualization issues in psychopathy remain controversial, such as the relationship between psychopathy and Antisocial Personality Disorder (ASPD) (Wygant et al., 2016), whether adaptive features like fearlessness, stress immunity, and low negative emotional experience belong to psychopathy (Miller & Lynam, 2012a, 2012b), and the heterogeneity of psychopathy or psychopathy subtypes (Bronchain et al., 2020; Lee & Salekin, 2010; Mokros et al., 2015). Such conceptual disagreements about the nature and boundaries of this disorder inevitably hinder progress in etiological research. Therefore, this paper proposes an integrated etiological framework for psychopathy based on the Triarchic Model of Psychopathy (Patrick et al., 2009) and related research. Before conducting this review, it is necessary to elaborate on some issues in the historical process of psychopathy conceptualization and introduce the triarchic model adopted in this paper to define psychopathy.

1.1 Psychopathy and Antisocial Personality Disorder

In the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association, 2013), Antisocial Personality Disorder is described as a pattern of psychological and behavioral disregard for or violation of others' rights, with core features of fraud and manipulation, lack of empathy, and exaggerated self-evaluation. Whether ASPD and psychopathy are largely similar or fundamentally reflect different constructs has been extensively discussed in psychopathology and clinical psychology. The divergence between psychopathy and ASPD emerged from an important challenge and re-

form in psychiatric diagnostic history—revising diagnostic criteria to improve the reliability of mental disorder diagnosis.

As shown in Figure 1 [Figure 1: see original paper], the divergence between these concepts can be traced back to the DSM-II era (American Psychiatric Association, 1968). Cleckley' s conceptualization of psychopathy was prominently reflected in DSM-II; however, considering the difficulty of measuring interpersonal and affective features, DSM-III (American Psychiatric Association, 1980) largely deleted these non-overt features, retaining only external indicators related to antisocial behavior to improve diagnostic reliability. This created two intertwined yet distinct branches in the clinical field: personality-based psychopathy and behavior-based ASPD from DSM-III.

Figure 1. Conceptualization history of psychopathy and antisocial personality disorder. Note: DSM = Diagnostic and Statistical Manual of Mental Disorders; PCL = Psychopathy Checklist; PCL-R = Psychopathy Checklist-Revised; SRP = Self-Report Psychopathy Scale.

Reviewing the research history of psychopathy, the shift from early clinical theory to empirical research was largely facilitated by Robert Hare' s work. His Psychopathy Checklist (PCL; Hare, 1980) and its revised version (PCL-R; Hare et al., 1990) perfectly captured Cleckley' s description of psychopathy and have been widely applied in prison and clinical populations. Researchers using the PCL-R to diagnose psychopathy in offender populations and comparing results with DSM-IV (American Psychiatric Association, 1994) ASPD diagnoses found that 50–80% of prisoners met DSM-IV ASPD criteria, while only 15% met PCL/PCL-R-defined psychopathy. Consequently, some researchers argued that ASPD and psychopathy might reflect two different measurement constructs.

This speculation was subsequently confirmed by empirical research. In numerous PCL-R-based studies, factor analyses revealed a relatively stable, replicable two-factor structure (Hare & Neumann, 2008; Hare et al., 1990). Factor 1 reflects interpersonal and affective deficits of psychopathy, including manipulative, deceptive, and narcissistic characteristics in interpersonal relationships, coupled with lack of deep emotion, empathy deficits, and callousness. Factor 2 comprises impulsivity, excitement-seeking, irresponsibility, parasitic lifestyle, and antisocial behavior. Although the two factors show moderate correlation, they actually reflect different psychopathic features and demonstrate differential external correlates. Specifically, Factor 1 is more associated with high selfishness, exploitation (e.g., narcissism, Machiavellianism, proactive aggression), and low empathy, while also showing some adaptive tendencies, such as positive correlation with social dominance and negative correlation with internalizing pathology like fear, anxiety, and depression. Factor 2, in contrast, expresses an anger-aggression tendency linked to externalizing pathology including impulsivity, substance abuse, alcoholism, and aggression. Compared to Factor 1, ASPD shows a stronger relationship with Factor 2. Therefore, before the DSM-5 personality disorder diagnostic system reform, many researchers considered ASPD distinct from psychopathy, likely reflecting the external behavioral

features associated with Factor 2 of psychopathy (Verona et al., 2001).

However, after the emergence of the DSM-5 Alternative Model for Personality Disorders, it innovatively proposed redefining existing personality disorder classifications through two new diagnostic criteria: Criterion A (Personality Functioning) and Criterion B (Personality Traits). In Criterion A, impairments in self and interpersonal personality functioning form different patterns of personality pathology. For ASPD populations, self-functioning impairment manifests as egocentrism, lack of internal prosocial standards, and inability to confirm the legitimacy of behavior; interpersonal functioning impairment appears as disregard for others, lack of empathy, exploitation, and maladaptive interpersonal interaction patterns using fraud, oppression, and manipulation to satisfy internal needs. In Criterion B, all personality disorders are embedded within five domains and 25 pathological personality traits included in the Pathological Personality Five-Factor Model. ASPD is described as a combination of six traits from antagonism (manipulation, callousness, hostility) and disinhibition (risk-seeking, impulsivity, irresponsibility) (American Psychiatric Association, 2013). These two new diagnostic criteria provided a foundation for researching conceptual issues between ASPD and psychopathy. Wygant et al. (2016) demonstrated that compared to the original categorical diagnosis, ASPD assessment under the DSM-5 alternative model showed higher correlation with PCL-R ($r = 0.59$ vs. 0.88). Thus, the definition in the alternative model partially bridges the split between ASPD and psychopathy.

1.2 The Multidimensional Nature of Psychopathy

Traditional personality disorder classification models conceptualize psychopathy as a syndrome, where all symptoms and features of psychopathy are moderately to highly correlated and share common etiological foundations. In reality, however, psychopathy may represent a combination of features with independent etiological bases that are not highly correlated with each other, indicating that the essence of psychopathy is multidimensional (Lilienfeld, 2018). Approaching psychopathy from a multidimensional perspective reveals potential limitations in previous research.

The first possible limitation lies in evaluating psychopathy using total scores, treating psychopathy as a unitary construct. Because previous two-factor models indicated that Factors 1 and 2 carry different psychopathic feature information with independent etiological bases. Theoretically (at least partially), Factor 1 originates from diminished capacity for fear experience, while Factor 2 stems from impaired impulse control (Patrick et al., 2009). In empirical studies, the two factors also show differential correlations with external outcomes, even exhibiting opposite external relationships. For instance, Factor 2 shows positive correlations with both internalizing and externalizing pathology and impulsivity, whereas Factor 1 is unrelated or weakly negatively correlated with distress, anxiety, and other negative emotions, and is associated with reduced impulsivity (Harpur et al., 1989; Hicks & Patrick, 2006). In a more refined

PCL-R-based four-facet study, reduced premeditation and perseverance-related impulsivity were found to be driven primarily by the interpersonal facet of Factor 1 rather than affective features. The unique information conveyed by these factors or their interaction effects would be obscured by total scores, potentially exposing a flawed research approach that overemphasizes the entire structure of psychopathy while paying insufficient attention to its subdimensions (Lilienfeld, 2018).

The second possible limitation involves overemphasizing callous-unemotional traits as the core feature of psychopathy. The Inventory of Callous-Unemotional Traits (ICU; Frick, 2004) comprises three related traits: callous, unconcerned, and unemotional (Waller et al., 2014). Callous-unemotional traits have been extensively studied in adolescent populations and confirmed as relatively stable traits across individual development that predict antisocial behavior in adulthood, representing important developmental precursors of psychopathy and conduct disorder (Flom & Saudino, 2017). However, callous-unemotional traits alone only reflect affective deficits in psychopathy, and overemphasizing this dimension neglects other valuable information. For example, when explaining the relationship between psychopathic traits and aggressive behavior, callous-unemotional traits and impulsivity influence aggression through different pathways. Preston and Anestis (2019) found that callous-unemotional traits promote proactive aggression through impaired empathy, whereas self-centered impulsivity increases both proactive and reactive aggression tendencies through reduced empathy and emotional dysregulation. Moreover, beyond callous-unemotional traits, the combination of multiple traits better captures the complete clinical presentation of psychopathy. Berg et al. (2017) invited participants with clinical psychology knowledge backgrounds to assess the similarity between a series of text descriptions and psychopathy, finding that when descriptions of adaptive traits (fearlessness, dominance) were added to typical maladaptive trait descriptions (callous-unemotional, impulsivity), participants' ratings of text-psychopathy similarity significantly increased.

Therefore, many researchers emphasize the importance of conceptualizing psychopathy as a multidimensional structure (Hare & Neumann, 2005; Lilienfeld et al., 2014; Patrick et al., 2009). In clinical assessment of psychopathy, combining multiple dimensions yields richer, more intervention-relevant clinical information.

1.3 Three Shifts in Psychopathy Measurement

Following the empirical research boom triggered by the PCL-R, the field has long been characterized by diverse schools of thought. Subsequently, influential psychopathy measures such as the Psychopathic Personality Inventory-Revised (PPI-R; Lilienfeld & Widows, 2005) and the Triarchic Psychopathy Measure (TriPM; Patrick, 2010) emerged from this controversy (Lilienfeld et al., 2012; Miller & Lynam, 2012a, 2012b), ushering psychopathy measurement into a new stage. Although these measures, like the PCL-R, are based on Cleckley' s tra-

ditional description of psychopathy, they represent three important shifts compared to earlier instruments.

First, the measurement population has shifted from clinical and prison populations to normal populations. Although psychopathy has historically been studied primarily in criminal populations, the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017) posits continuity of personality traits between normal and clinical populations, suggesting that personality pathology research needs to simultaneously examine pathological features conveyed in both abnormal and healthy populations (Kotov et al., 2017; Krueger et al., 2018). Latzman et al. (2017) also noted that psychopathy represents a multidimensional structure based on fundamental biobehavioral traits, reflecting differences in degree rather than category. From this perspective, examining the distribution of these fundamental dimensions across a range of populations including clinical and nonclinical samples, as well as similarities and differences between institutional and non-institutional populations, may provide further insights into the manifestation of psychopathy under normal circumstances, disorder-related underlying etiology, and specificity of adverse outcomes and treatment approaches.

Second, there has been a reduction in explicit behavioral assessment of antisocial behavior and a shift toward personality traits related to psychopathy. Increasingly, researchers recognize that antisocial/criminal behavior should be considered a possible consequence rather than a component of psychopathy (Boduszek & Debowska, 2016; Boduszek et al., 2017; Skeem & Cooke, 2010a, 2010b). Factor 2 of the PCL-R involves numerous antisocial behavior indicators, and using the PCL-R to assess psychopathy in prisons may overestimate psychopathy prevalence in offender samples (Boduszek & Debowska, 2016; Edens et al., 2001). When abandoning antisocial behavior content as measurement items and using trait-based psychopathy measures, the proportion of high psychopathy in both prison and university student populations is approximately 7% (Boduszek et al., 2019), indicating that prisoner populations do not show more severe psychopathy than non-prisoners, providing important empirical evidence against including criminal/antisocial behavior in psychopathy assessment. Therefore, using self-report, personality-based measures in nonclinical and non-prison populations may capture complete psychopathic traits while avoiding excessive focus on criminal or antisocial behavior.

Finally, emerging psychopathy measurement models provide an integrative path for psychopathy research, particularly the Triarchic Model. This model was designed to establish a theoretical structure that not only connects psychopathy concepts with broader dimensional models of normal personality and psychopathology, providing an integrative framework for understanding clinical psychopathy, criminal psychopathy, and successful psychopathy, but also offers insights for research on the etiology of psychopathy and core processes underlying observable symptoms (Patrick & Drislane, 2015). As shown in Figure 2 [Figure 2: see original paper], substantial empirical research has established a

rich network of relationships for the Triarchic Model with other domains, including the Five-Factor Model of personality, neurophysiology, genetic etiology, and developmental pathways (Dotterer et al., 2017; Latzman et al., 2017; Patrick & Drislane, 2015; Sleep et al., 2019; Tuvblad et al., 2018). Next, we will focus on introducing the Triarchic Model and integrating it with existing empirical research on cognition and affect in psychopathy to provide a relatively complete psychopathy cognition-affective processing framework.

Figure 2. Integrated psychopathy cognition-affective processing framework based on the TriPM model. Note: FFM = Five-Factor Model; PID-5 = Personality Inventory for DSM-5.

2.1 Boldness

Boldness describes the capacity to remain calm in the face of danger and threat, maintain self-possession, recover quickly from stressful events, exhibit high self-assurance and social potency, and tolerate danger and unfamiliarity (Patrick & Drislane, 2015; Patrick et al., 2009). In the personality domain, features included in boldness substantially overlap with low neuroticism and high extraversion (Hyatt et al., 2019). In psychopathological dimensions, boldness shows cross-loadings on externalizing (0.41) and internalizing (-0.33) factors (Latzman et al., 2020), with weak correlations with externalizing symptoms such as antisocial behavior, aggression, and substance abuse (average r = 0.10) and moderate negative correlations with internalizing symptoms like anxiety and depression (average r = -0.36), exhibiting personal characteristics of high sensation-seeking, self-esteem, and social potency (Sleep et al., 2019). In other psychopathy measures, boldness corresponds to the interpersonal facet of the PCL-R (facet 1) and the fearless-dominance dimension of the PPI (Patrick et al., 2009).

Controversy surrounding the Triarchic Model has focused primarily on this phenotypic structure. Miller and Lynam (2012a, 2012b) argued that psychopathy should represent a collection of maladaptive traits, while traits included in boldness are highly healthy and adaptive, unrelated to typical externalizing symptoms of psychopathy, thus questioning boldness as an important representation of psychopathy. Supporters countered that boldness reflects content already present in Cleckley' s description of psychopathy, representing the "mask" component of psychopathy (Lilienfeld et al., 2012), where the maladaptive core features of psychopathy are masked by adaptive appearances such as charm, lack of anxiety and neurotic symptoms, and social potential. Subsequent researchers quantitatively rated features of psychopathy cases documented by Cleckley, finding high scores for boldness, confidence, and low anxiety—precisely the features of boldness in the triarchic model (Crego & Widiger, 2016). Previous research overemphasizing typical maladaptive features of psychopathy (callous-unemotional and impulsivity) while ignoring boldness is biased; psychopathy comprises multiple complex mixed features reflecting surface-level health and internal psychological dysfunction, making simultaneous study of these features

necessary.

Patrick et al. (2009) proposed that boldness, as a phenotypic structure of psychopathy, represents an expression of deeper genotypic fearlessness. Fearlessness represents a genetically based fundamental tendency corresponding to the neurobehavioral dimension of threat sensitivity. Under normal circumstances, when individuals detect threat-related conditional cues in the environment, sensory information from sounds or images reaches the basolateral amygdala (BA) via the thalamus as a sensory input subsystem, then transmits to the action mobilization subsystem, the central nucleus of the amygdala (CeN). Through this neural pathway, when fear or threat-related conditioned stimuli appear, the amygdala-centered defense system automatically activates and produces avoidance responses. Boldness in the Triarchic Model is thought to reflect reduced sensitivity of the brain' s core defense system based on the amygdala and related structures to threat or punishment cues, manifesting as stress immunity, low fear, and insensitivity to punishment (Patrick & Drislane, 2015; Patrick et al., 2009). The high social potency, psychological resilience, and bravery exhibited by boldness can be considered mild expressions of the fearless genotype, phenotypically distinct from aggressive externalizing abnormal behavior and showing adaptive tendencies. In contrast, callous-unemotional/meanness represents a malignant expression of low fear, displaying highly maladaptive symptoms of interpersonal antagonism and externalizing behavior.

2.2 Disinhibition

Disinhibition in the Triarchic Model describes a phenotypic tendency toward impulse control problems, including lack of planning and foresight, poor regulation of emotion and impulse, insistence on immediate gratification, and lack of behavioral constraint, which can be viewed as a link between impulsivity and negative emotionality (Patrick & Drislane, 2015). From the Triarchic perspective, disinhibition must be combined with emotional detachment in the form of boldness or meanness to constitute diagnostic criteria for psychopathy. In the personality domain, disinhibition primarily reflects a trait combination related to low conscientiousness and high neuroticism (Hyatt et al., 2019). In the HiTOP model (Krueger et al., 2018), disinhibition corresponds to disinhibited externalizing within the two externalizing spectra, showing moderate positive correlations with externalizing symptoms such as antisocial behavior and aggression (average $r = 0.44$) and internalizing symptoms like anxiety and depression (average $r = 0.33$), exhibiting impulsive personal characteristics (Sleep et al., 2019). When compared with other psychopathy measures, disinhibition shows high correlation with the psychopathy impulsive-antisocial factor (Factor 2) (Patrick et al., 2009).

Patrick et al. (2009) suggested that the development of the disinhibition phenotypic structure can be traced to difficult temperament manifested in childhood, including early-onset high negative emotionality, irritability, hyperactivity, attention concentration deficits, and poor adaptability to novel environments.

Longitudinal research provides evidence for this hypothesis, indicating that psychopathic individuals show low response control characteristics as early as ages 3-5 (Dotterer et al., 2017). Meanwhile, substantial research demonstrates that poor self-control and disinhibition are relatively stable across individual development (Caspi et al., 2005; Patrick, 2018), and these inhibitory control deficits have high heritability. In Tuvblad et al.' s (2018) twin study, shared genetic effects explained 49% of variance in disinhibition. Venables et al. (2016) combined disinhibition-related scales with P300 data to form a composite indicator showing moderate phenotypic correlation with substance abuse symptoms (r = 0.44), with shared genetic factors explaining 89% of the covariance. These results indicate that beneath the impulse-related behavioral problems and abnormal neurophysiological indicators exhibited by psychopathy lies a highly heritable disinhibition tendency.

2.3 Meanness

Meanness in the Triarchic Model signifies lack of empathy and close relationships, contempt for others, predatory exploitation, and gaining power through cruel or destructive means. Meanness is central to Cleckley' s concept of psychopathy, typically manifested as arrogance and mockery, contempt for authority, lack of intimate interpersonal relationships, cruel abuse of people and animals, proactive aggression, manipulation and deception of others for profit, and excitement-seeking through destruction, reflecting interpersonal antagonism (Patrick & Drislane, 2015; Patrick et al., 2009). In the personality domain, meanness is associated with low agreeableness (Hyatt et al., 2019). In the HiTOP model (Krueger et al., 2018), it corresponds to another form of externalizing: antagonistic externalizing. Meanness shows moderate positive correlation with externalizing symptoms such as antisocial behavior and aggression (average r = 0.38) and weak association with internalizing symptoms like anxiety and depression (average r = 0.17), exhibiting personal characteristics of low empathy and low perspective-taking (Sleep et al., 2019). The meanness dimension of the Triarchic Model corresponds to the affective facet of the PCL-R (facet 2) and the callous-unemotional traits measured by the ICU (Patrick et al., 2009).

Patrick and Drislane (2015) proposed that meanness serves as "phenotypic glue" in the Triarchic Model, linking the three phenotypic structures. On one hand, meanness and boldness jointly reflect the potential influence of the fearless genotype on individual development. The insensitivity to punishment and threat cues caused by the fearless genotype not only develops into adaptive features like social confidence, leadership, and bravery but also limits individuals' capacity to form conditioned emotional responses to punishment cues, affecting internalization of rules or norms and hindering conscience development, thereby creating maladaptive consequences of meanness or interpersonal antagonism. On the other hand, both meanness and disinhibition are influenced by difficult temperament and postnatal rearing environments. Children with difficult temperament exhibit high negative emotionality and irritability, perform

poorly when attention needs to be maintained, and have difficulty adapting to environmental changes, which interact with parental rearing styles. If parental competence is insufficient, the process of establishing secure attachment relationships and internalizing social norms becomes extremely difficult, leading to inadequate behavioral inhibition and development of antagonistic interpersonal styles. Meanness represents an etiological intersection point among the three dimensions, genetically linked to inhibitory functions and environmentally shaped to connect with disinhibition and boldness (Latzman et al., 2017).

3. Linking Triarchic Psychopathy Structures to Cognitive-Affective Processing Deficits

The Triarchic Model hypothesizes that psychopathy consists of three distinct phenotypic structures: boldness, meanness, and disinhibition. Beneath these phenotypic structures lie deeper content related to etiology and developmental pathways, providing a broad framework for psychopathy research. Next, this paper attempts to connect some deficits in psychopathy' s cognitive-affective processing (low threat sensitivity, impaired executive function, and empathy deficits) with the three structures included in the Triarchic Model.

3.1 Boldness and Low Threat Sensitivity

Threat sensitivity is a general tendency to produce defensive responses to threatening stimuli (e.g., aversive, startling, punishing stimuli), thought to reflect individual differences in reactivity of the brain' s core defense system based on the amygdala and related structures. This represents the neurophysiological manifestation of boldness in the Triarchic Model (Patrick & Drislane, 2015). The stress immunity (low anxiety) and low harm avoidance (low fear) exhibited by psychopathic individuals may originate from low threat sensitivity resulting from impaired core defense systems. A long-standing view in psychopathy research holds that psychopathy involves deficits in fear responses (Patrick, 2018). Fear reflects reactions to imminent or unavoidable danger, with key brain regions being the lateral amygdala (LA) and central nucleus of the amygdala (CeN). Kramer et al. (2011) conducted exploratory factor analysis on a series of scales measuring fear or lack of fear in an adult twin sample, extracting a higher-order factor interpreted as threat sensitivity, indicating a more general tendency underlying lack of fear responses. In subsequent research, Yancey et al. (2016) established a series of neurophysiological indicators for threat sensitivity, such as startle potentiation, electromyographic responses, and heart rate under threat cues.

The notion that psychopathy involves low threat sensitivity was already reflected in the low-fear model (Lykken, 1995). The low-fear model posits that low fear/threat sensitivity reduces the reinforcing effectiveness of various threats or punishments that parents or caregivers use when attempting to shape behavior, instill prosocial attitudes, and form internal constraints (Lykken, 1995), which may manifest as reduced reactivity of the behavioral inhibition system.

Other psychopathy-related features such as excitement-seeking, lack of guilt, and superficial charm are subsequent chain symptoms triggered by low fear. Today, numerous empirical studies provide evidence for low threat sensitivity in psychopathy.

First, deficits related to threat sensitivity manifest at the neurophysiological level. Existing research examining amygdala structural characteristics in psychopathic and normal individuals has found that psychopathy is indeed associated with volume abnormalities in the lateral amygdala (LA) and central nucleus of the amygdala (CeN) (Boccardi et al., 2011; Yang et al., 2009). In terms of activation levels, psychopathic individuals also differ from normal individuals in amygdala activation, showing overactivation of the central amygdala (CeN) and underactivation of the basolateral amygdala (BA) (Moul et al., 2012).

Second, low threat sensitivity means psychopathic individuals less frequently respond to threat cues or fear-inducing stimuli across various contexts. Startle is an automatic defensive response to unconditional or conditional danger stimuli, widely used in research confirming low fear in psychopathy. In a series of controlled experiments, high psychopathy scorers showed less startle response than normal groups when facing fear-related cues (Baskin-Sommers et al., 2013; Justus & Finn, 2007; Sutton et al., 2002), while other danger-related automatic responses such as skin conductance also showed reduced reactivity (Thomson et al., 2019). When considering fearless features alone, participants with high scores on Factor 1 fearless dominance of the Psychopathy Personality Inventory (PPI; Lilienfeld & Andrews, 1996) also showed reduced response in aversive startle potentiation across different aversive cues (Benning et al., 2005; Dindo & Fowles, 2011; Lopez Penades et al., 2012). These studies demonstrate that psychopathic startle responses are negatively correlated with self-reported scores related to boldness or fearlessness, consistent with the low threat sensitivity hypothesis.

Third, research on threat-related conditioning also supports the low threat sensitivity hypothesis. In one study using aversive odors as unconditioned stimuli (US) to establish threat-related conditioning, psychopathic individuals showed impaired threat association learning (Flor et al., 2002). In another fear conditioning task using faces as conditioned stimuli (CS) and electric shock as unconditioned stimuli (US), psychopathic individuals also showed reduced conditioned response reactivity, with this abnormality primarily driven by interpersonal-affective (Factor 1) features. It can be inferred that normal individuals establish conditioned responses to environmental cues signaling danger, producing fear experiences when exposed to corresponding situations, while psychopathic individuals' deficits in danger association learning prevent them from generating conditioned fear responses in danger-related contexts.

Fourth, research on psychopathy subtypes also provides evidence for low threat sensitivity. Gill and Stickle (2016) identified the existence of a primary psychopathy subtype in an adolescent offender sample, showing far lower self-reported anxiety, depression, and Behavioral Inhibition System (BIS) scores

than ordinary criminals, indirectly reflecting reduced threat or punishment sensitivity caused by lack of fear. In numerous person-centered psychopathy studies, a subtype consistently emerges that matches theoretical descriptions of primary psychopathy, scoring significantly higher than other subtypes on interpersonal aspects (Poythress et al., 2010), fearless dominance (Garofalo, Sijtsema, et al., 2020; Lee & Salekin, 2010), and emotional stability (Bronchain et al., 2020), demonstrating high stress immunity and low harm avoidance tendencies—all manifestations of low threat sensitivity.

In summary, individuals with high boldness or fearlessness typically show lower reactivity levels in defense systems, manifesting as insensitivity to threat or fear-related cues. These individuals tend to exhibit fearless temperament in childhood, often developing social confidence, adventurousness, and emotional resilience in adolescence and adulthood. However, these reflect benign developmental outcomes of low threat sensitivity, such as social dominance and stress immunity, whereas core features of psychopathy involve more maladaptive outcomes like interpersonal antagonism and callousness. This raises the question of which etiological factors interact with low threat sensitivity to cause these maladaptive malignant outcomes, and the other structures of the Triarchic Model may contain this information.

3.2 Disinhibition and Impaired Executive Function

In neurobehavioral research, interaction between the striatum' s dopamine reward system and the prefrontal cortex enables individuals to constrain and guide behavior during goal-directed and emotion regulation processes. Impairment in these regions leads to difficulties in inhibiting behavior and regulating emotions when facing delayed goals (Patrick et al., 2012). These represent manifestations of impaired executive function, externally expressed as insufficient inhibitory control, constituting a common etiological basis for many externalizing pathologies, including disinhibition in the Triarchic Model (Patrick, 2018). The relationship between disinhibition in the Triarchic Model and executive function has been confirmed by numerous neurophysiological and behavioral studies.

First, individuals with high externalizing or disinhibition typically show abnormal brain responses related to cognitive-executive function impairment. One aspect is reduced P300 amplitude. P300 reflects inhibitory control at the neural level, with neuronal hyperexcitability as a common risk factor for general externalizing problems linked to reduced P300 amplitude (Patrick et al., 2006). Venables and Patrick (2014) used an oddball task to examine the relationship between PCL-R scores and P300 responses in a male offender sample, finding that increased scores on the impulsive-antisocial factor (Factor 2), highly related to disinhibition, were associated with reduced P300 amplitude. In another study of male offenders, Venables et al. (2015) examined relationships between cortical electrocortical responses to emotional and neutral pictures and psychopathy factors, finding that increased PCL-R impulsive-antisocial (Factor 2) scores were associated with reduced early P300 potential amplitude after picture viewing,

regardless of emotional valence. Interpersonal-affective (Factor 1) scores, however, were only associated with reduced late positive potential responses after viewing aversive pictures. These studies link disinhibition in the Triarchic Model to P300, indicating impaired inhibitory control function in highly disinhibited individuals.

Another aspect is reduced error-related negativity (ERN) amplitude. ERN is a brainwave component evoked by behavioral errors, often regarded as a neurophysiological indicator of behavioral monitoring function, with reduced amplitude indicating impaired behavioral monitoring. Previous research extensively explored relationships between psychopathic traits and behavioral and neurophysiological performance in go/no-go tasks, finding that individuals with high psychopathy scores typically make more error responses, with error frequency most closely linked to disinhibition traits. Paiva et al. (2020) examined event-related potentials during go/no-go tasks and found that disinhibition in the Triarchic Model was associated with reduced ERN amplitude. Ribes-Guardiola et al. (2020) conducted a more refined study comparing individual performance on go/no-go and flanker tasks, finding that high disinhibition participants only showed reduced ERN during no-go trials, not in flanker tasks. They speculated that disinhibition traits may reflect reduced brain reactivity during later refinement stages of processing motivationally significant events, manifesting as insufficient inhibition of prepotent response tendencies. These studies link disinhibition in the Triarchic Model to ERN, indicating impaired processing of motivational events and behavioral monitoring functions in highly disinhibited individuals.

Second, the influential Response Modulation Hypothesis (Newman, 1998) in psychopathy etiology research also supports impaired executive function in psychopathy. According to this hypothesis, the core deficit in psychopathy is essentially cognitive, particularly attentional deficits. Normal individuals' attention shifts periodically from current behavior to evaluation of environmental cues to determine whether current behavior is appropriate and adjust accordingly. For psychopathic individuals, the Response Modulation Hypothesis posits that when a dominant response set exists, this group' s attention is difficult to shift to other background cues, making it hard to adjust behavior based on other environmental information, thus showing impulsivity and behavioral inhibition difficulties. Dominant response set typically refers to primary tasks or primary attentional focus in experiments, while background or environmental cues involve any secondary experimental tasks or attentional foci (Smith & Lilienfeld, 2015). This hypothesis aligns with descriptions of impulsivity consequences from prefrontal cortex and reward system dysfunction in neurophysiological research, possibly reflecting impaired executive function and irrational pursuit of rewards in highly disinhibited individuals.

Regarding psychopathic individuals' insensitivity to punishment cues, the Response Modulation Hypothesis offers a different explanation than the low-fear model, suggesting that non-response to punishment is not due to lack of fear

but rather due to response perseveration in psychopathic individuals (Smith & Lilienfeld, 2015). Response perseveration represents a tendency to continue responding regardless of environmental cues (such as punishment). Psychopathic individuals can actually learn from punishment, provided that punishment avoidance is established as a dominant response and does not compete with reward events. To test this hypothesis, Newman and Kosson (1986) established two go/no-go task conditions. In the first trial, participants received monetary rewards for correct responses and monetary loss for incorrect responses. In the second trial, participants started with a certain amount of money, received no reward for correct responses, but incurred monetary loss for incorrect responses. Results supported the hypothesis: in the first task, because both reward and punishment mechanisms existed, psychopathic individuals established obtaining rewards as the dominant response, ignored punishment cues, and showed more error responses than the non-psychopathic group. In the second task, where only punishment avoidance existed, punishment avoidance became the dominant response, and psychopathic group performance showed no significant difference from the control group. This demonstrates the existence of response perseveration in psychopathic individuals, who have difficulty inhibiting dominant reward-seeking responses, providing evidence for executive function deficits in psychopathy.

Beyond inhibitory control difficulties in motivational (reward-seeking) events, evidence linking disinhibition traits to executive function impairment has also been observed in motivationally neutral events. Delfin et al. (2018) used the Cambridge Neuropsychological Test Automated Battery (CANTAB) to examine associations between psychopathic features and executive function. This battery includes four aspects of executive function: cognitive flexibility, working memory, inhibitory control, and planning/problem-solving, assessed through Intra-Extra Dimensional Set Shift (IED), Spatial Working Memory (SWM), Stop Signal Task (SST), and Stockings of Cambridge (SOC) tasks. Results found that the psychopathy impulsive-antisocial factor (highly related to disinhibition) was significantly associated with reduced initial thinking time in the planning/problem-solving task (SOC). The lack of planning and thoughtless action described by disinhibition traits was manifested through lower average initial thinking time. Similar results appear in other studies, where high disinhibition individuals perform poorly on planning and problem-solving tasks not involving reward or punishment (Bagshaw et al., 2014; Baskin-Sommers et al., 2015). This also provides evidence for the Response Modulation Hypothesis, suggesting that high disinhibition individuals have general attention deficits, making it difficult to shift attention to background environments once goal-directed behavior is established, showing inhibitory control difficulties (Smith & Lilienfeld, 2015). Motivational systems (especially reward) may exacerbate executive function deficits in high disinhibition individuals, making them perform worse in tasks.

An important external manifestation of disinhibition in the Triarchic Model is impulsive behavior leading to various maladaptive consequences (such as pun-

ishment), which is well explained by impaired executive function. However, the Triarchic Model defines psychopathy as multidimensional. Although the three phenotypic structures show weak to moderate correlations, they actually reflect three unique structures based on different etiological foundations. Moreover, research has found inconsistent associations between psychopathy Factor 1 (interpersonal-affective) and Factor 2 (impulsive-antisocial) with executive function. Psychopathy with high interpersonal-affective features shows normal or above-average frontal executive ability and P300 amplitude, while psychopathy with high impulsive-antisocial features shows reduced frontal executive ability and P300 amplitude (Pasion et al., 2017; Weidacker et al., 2017). This means that the entire clinical presentation of psychopathy cannot be explained through impaired executive function alone.

3.3 Meanness and Empathy Deficits

Verschuere et al. (2018) conducted network analyses based on the PCL-R across three cross-cultural samples, finding that lack of empathy among affective features is the core characteristic of psychopathy. Empathy includes affective empathy and cognitive empathy, where affective empathy refers to individuals' capacity to experience consistent vicarious emotions with others, while cognitive empathy refers to understanding others' emotions through association or perspective-taking processes (Cox et al., 2012). Existing research indicates that psychopathic individuals primarily lack affective empathy capacity (Brouns et al., 2013). Due to the complexity of interpersonal interactions, few etiological theories can fully summarize the development of meanness, but substantial research has identified unique links between callous-unemotional traits and empathy deficits, pointing to the etiology of psychopathic meanness or callous-unemotional traits as empathy deficits.

First, psychopathy involves reduced subjective emotional experience, which affects individuals' empathy capacity. The insula and anterior cingulate cortex (ACC) are crucial brain regions for subjective emotional experience (Hoppenbrouwers et al., 2016). The ACC has been emphasized as an important region for inhibiting excessive emotional responses, related to individuals' conscious pain experience (Viswanathan et al., 2013). The insula is typically activated in contexts involving self or others' experiences, whether the stimulus is motoric, painful, or emotional. The anterior insula (AI) has been shown to relate to individuals' pain processing and empathy (Kanel et al., 2019; Kogler et al., 2020). Evidence suggests that psychopathic individuals show lower physiological arousal to their own distress, such as reduced anterior cingulate cortex reactivity related to fear and anger (Flasbeck et al., 2019; Sethi et al., 2018). They also differ from normal individuals in brain structure and function related to emotional experience, including thinner insula in psychopathic individuals and reduced functional connectivity between insula and anterior cingulate cortex (Ly et al., 2012). These reductions in subjective experience may lead to empathy dysfunction, manifesting as psychopathic stress immunity and callous-unemotional

features (Shirtcliff et al., 2009).

Second, empathy deficits involve insensitivity to others' distress, and psychopathic individuals indeed show insufficient capacity to recognize distress cues. Numerous emotion recognition studies have shown that adolescents and adults with psychopathic traits have impaired emotion recognition functions, and this impairment is selective. For example, they have normal recognition ability for angry and disgusted expressions but show deficits in processing distress expressions (i.e., fear and sadness) and happy expressions (Gillespie et al., 2019; Moore et al., 2019; White et al., 2012). Impairment of the affective empathy system composed of the amygdala and brain structures related to vicarious experience, such as the anterior insula (AI) and anterior cingulate cortex (ACC), may constitute an important neurophysiological basis for psychopathic meanness or callous-unemotional traits. Blair (2013) suggested that the basolateral amygdala (BA) is involved in sensory input of emotion-related information and relates to facial emotion recognition deficits, while individuals with high callous-unemotional traits show reduced amygdala activation to distress-related cues, subsequently leading to insufficient capacity to empathize with others' pain. Seara-Cardoso et al. (2015) also found that increased psychopathy interpersonal-affective features (Factor 1) were associated with reduced sensitivity to others' pain, showing reduced activation in anterior insula cortex and anterior cingulate cortex, while the relationship between impulsive-antisocial features (Factor 2) and pain sensitivity was opposite to Factor 1. These results indicate that insufficient reactivity of brain structures related to affective empathy leads to reduced empathy sensitivity in psychopathic individuals, manifesting as callous-unemotional or mean external symptoms. Many genetic etiology studies have confirmed that meanness or callous-unemotional traits show relatively strong genetic effects (Latzman et al., 2017; Tuvblad et al., 2018), and callous-unemotional traits share partially common genetic etiology with distress cue recognition deficits (Moore et al., 2019; Petitclerc et al., 2019), indicating that the link between meanness traits and empathy deficits is genetically influenced.

Third, early insecure attachment affects empathy development, and psychopathic individuals indeed have difficulty establishing secure attachment relationships. The affective dimension of psychopathy—meanness or callous-unemotional traits—is associated with early insecure attachment styles, particularly avoidant attachment (low commitment, low empathy, interpersonal cynicism) (Christian et al., 2017; Jonason & Buss, 2012; Vanheule & De Ganck, 2015). Moreover, substantial research has found that insecure attachment patterns lead individuals to show lower cognitive and affective empathy capacity (Ardenghi et al., 2019; Stern & Cassidy, 2017), which promotes indifferent, exploitative attitudes toward others and gradually develops into meanness features (Patrick et al., 2009). It can be speculated that early difficult temperament and insensitive or unresponsive parenting lead to insecure attachment relationships, causing individuals to avoid emotions, dependence, and intimacy in later interpersonal interactions, pay less attention to others' emotional experiences, and show negative attitudes toward inter-

personal relationships (Patrick et al., 2009), which inevitably interacts with individuals' empathy development. From an attachment perspective, psychopathic individuals' meanness may be a manifestation of avoidant attachment style in interpersonal interactions—a preemptive counterattack to cope with expectations that others will exploit, dominate, and treat them cruelly—all reflecting low empathy sensitivity.

Impairment of emotion-related brain structure and function is an important neurobiological factor causing empathy deficits, while insecure attachment is an important environmental or familial factor leading to empathy deficits. Both may lead individuals to develop meanness features such as lack of intimate relationships and interpersonal antagonism. The Triarchic Model describes psychopathy' s social deficits as active disaffiliation, depicting a tendency to achieve goals by any means without regard for others' feelings (Patrick et al., 2009). This active disaffiliation may be driven by empathy deficits, representing the core etiology of meanness in the Triarchic Model.

4. Discussion and Future Research Directions

Existing research on cognitive and affective deficits in psychopathy is rich and diverse, drawing from evidence on antisocial personality disorder (Liu et al., 2019) and inspired by callous-unemotional trait research (Xiao et al., 2014). This paper attempts to integrate the currently influential Triarchic Model with low threat sensitivity, impaired executive function, and empathy deficits in psychopathy, providing a relatively complete psychopathy cognition-affective processing framework that offers useful information for psychopathy theoretical research and clinical treatment. However, this framework cannot explain all clinical features of psychopathy because factors influencing psychopathy feature development are multifaceted. Gender, race, age, sample type, and measurement methods are all important moderating variables in psychopathy research (Sleep et al., 2019) and may affect relationships between psychopathic traits and cognitive and affective deficits. The cognitive and affective deficits of psychopathic populations listed in this paper include substantial research findings from this field but cannot encompass all known cognitive and affective deficits. Here, we propose several key points requiring attention in future psychopathy research:

4.1 Discriminant Validity Among Triarchic Psychopathy Structures

The Triarchic Model assumes that the three phenotypic structures—boldness, meanness, and disinhibition—have different etiological bases. Substantial empirical research has tested this hypothesis with mixed results.

On one hand, existing evidence indicates significant exclusivity between low threat sensitivity reflected by high boldness and impaired executive function reflected by high disinhibition. Yancey et al. (2016) established a psychoneurometric definition of threat sensitivity through factor analysis of a series of scales

and physiological data, finding that threat sensitivity showed high correlations with fear-related disorders but weak or no correlations with substance use disorders and P300 potential in oddball tasks (both considered manifestations of disinhibition/executive function impairment) (Kramer et al., 2020; Nelson et al., 2016; Yancey et al., 2016). This evidence supports the discriminant validity of boldness and disinhibition structures and their corresponding processing deficits.

On the other hand, the Hierarchical Taxonomy of Psychopathology (HiTOP) attempts to redefine the existing DSM classification system, emphasizing the existence of a general psychopathology factor (P factor). This means that although all existing mental disorders have unique symptoms and etiological bases, there is indeed a P factor that influences all disorders. Current research suggests this P factor represents a cognitive ability deficit, particularly executive function (Krueger et al., 2018). In psychopathy, research has also demonstrated the existence of a P factor, with genetic effects of this general psychopathy factor reaching 64% (Latzman et al., 2017), suggesting potential common etiological bases among psychopathy structures. This is also reflected in the integrated etiological framework in this paper, such as both meanness and disinhibition structures involving cognitive deficits, while impairments in boldness and meanness both involve the amygdala at the neurophysiological level. Future research urgently needs to examine the discriminant validity of Triarchic structures from both phenotypic and etiological levels.

4.2 Psychopathy Subtypes

Personality disorder classification models define subtypes based on different measurement and diagnostic criteria. In contrast, researchers advocating psychopathy multidimensionality believe that subtypes reflect combinations of personality traits at different levels. Recently, increasing research following dimensional model perspectives has used cluster analysis or latent profile analysis on psychopathy-related traits to divide individuals into several relatively homogeneous groups based on trait-level similarity, matching them with theoretically described psychopathy subtypes. Such studies have almost universally identified two relatively stable subtypes corresponding to theoretically described primary and secondary psychopathy (Boduszek et al., 2019; Bronchain et al., 2020; Colins et al., 2018; Gill & Stickle, 2016; Hicks et al., 2004). The two subtypes have different etiological bases. The widely accepted view is that primary psychopathy represents genetic affective deficits with callousness, loss of attachment capacity, limited internalizing problems, high manipulativeness, and instrumental or proactive aggression. In contrast, secondary psychopathy is generally considered to result from adverse environments such as abuse, lack of parenting, rejection, or neglect, leading to emotional and behavioral regulation dysfunction, thus showing more impulsive and reactive aggression (Hicks et al., 2012; Lykken, 1995; Patrick, 2018; Preston & Anestis, 2019).

In Gill and Stickle' s (2016) study of psychopathy subtypes in youth, secondary

psychopathy showed more frequent and intense positive and negative emotional experiences than primary psychopathy, with these emotional experiences mediating the relationship between psychopathy subtype and depressive symptoms. Although secondary psychopathy also shows general antisocial and impulsive tendencies, it often exhibits neurotic characteristics of high anxiety and harm avoidance, with certain borderline personality features. In traditional psychopathy concepts, callous-unemotional is the core feature of psychopathy. In this sense, the concept of secondary or symptomatic psychopathy is problematic and misleading because it implies that this group also represents a type of psychopathy.

Ultimately, the emergence of subtypes actually reflects a core issue in psychopathy conceptualization: what constitutes true psychopathy. Although this issue remains unresolved, it is foreseeable that psychopathy is essentially a multidimensional structure. Future research must accept the complexity of its multidimensional structure, avoiding approaches that focus only on one subdimension (e.g., callous-unemotional) and methods that treat psychopathy as a single unitary construct (Lilienfeld, 2018).

4.3 Manifestation of Psychopathic Traits Across Different Life Stages

Current psychopathy research predominantly uses cross-sectional methods, focusing on adult offender, clinical, and community populations. Although boldness, meanness, and disinhibition can all be manifested through relatively stable external behavioral expressions (e.g., stress immunity, low empathy, impulsivity) and neurophysiological indicators (e.g., reduced P300, amygdala, frontal, and cingulate reactivity) in adult populations, whether these features are continuous across different life stages remains to be clarified. Dotterer et al. (2017) studied the developmental stability of boldness, meanness, and disinhibition in the Triarchic Model, examining external manifestations of psychopathic features across three key developmental periods (ages 3–5, 9–11, and 15–17). Results showed that boldness-related features demonstrated continuity across development, with high boldness adults showing low response control, fewer internalizing features, and greater resilience in early life. Disinhibition-related traits only showed continuity in late adolescence (ages 15–17), exhibiting low response control, more internalizing, externalizing, and substance use problems. Meanness was not stable across development, showing different manifestations at different ages, possibly because psychopathy features in this study were measured through conversion of normal personality scores, making it difficult to capture maladaptive features of psychopathy.

Conducting longitudinal psychopathy research using different types (clinical, nonclinical) and ages (children, adolescents, adults) of samples is necessary. For psychopathy treatment, better therapeutic effects are often shown in adolescent populations than in adults (Ribeiro da Silva et al., 2019). Stable and effective psychophysiological or behavioral indicators of psychopathy across different life stages are key to intervention, diagnosis, and treatment.

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Note: Figure translations are in progress. See original paper for figures.