The emergence and development of psychopathy

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Introduction

Psychopathic individuals are characterized by antisocial deviance and premeditated violent behavior, lack of empathy, remorse, or guilt, shallow affect, and poor emotional regulation and behavioral control (Hare, 1998; Hare & Neumann, 2008; Meloy, 1988, 2001; Salekin, 2017). Defined as a personality disorder, psychopathy is estimated to be between 0.6% and 4% in the general population and approximately in 20% of criminal violent offenders (Hare, 1991; Patrick, 2005), with a higher proportion of males (Thompson, Ramos, & Willett, 2014). Furthermore, it is considered a strong predictor of both nonviolent and violent recidivism (Hare, 2006; Skilling, Harris, Rice, & Quinsey, 2002).

The etiology of psychopathy, according to some, seems to be founded on an impaired emotional response that is manifest in the absence of fear and empathy, promoting the risk of antisocial behavior and hindering the development of moral conscience (e.g., Blair, 1995; Skeem, Johansson, Andershed, Kerr, & Louden, 2007; Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003). In a more general sense, limbic anomalies, centered on the amygdala and its connective pathways to higher cortical centers, are the focus of research attention. Many studies have also shown strong correlations between psychopathic traits and genetic factors in childhood and adulthood (Vernon, Villani, Vickers, & Harris, 2008; Viding, Jones, Paul, Moffitt, & Plomin, 2008), such work predicting antisocial behaviors in children (Viding et al., 2008; Viding, Blair, Moffitt, & Plomin, 2005), adolescents (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005), and also in adults (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006). A variation in antisocial behavior is explained by genetic factors when psychopathic traits are present, suggesting that antisocial behaviors in psychopathic individuals are somewhat rooted and stronger (Viding et al., 2005).

There are the two subtypes of psychopathy: primary and secondary. While primary psychopathy is mostly related to genetic features, associated with reduced neural activation to others’ emotions, secondary psychopathy—what used to be called sociopathy—develops mostly from environmental causes, such as parental abuse or rejection, resulting in neuroticism, aggression, impulsivity, and emotional reactivity (e.g., Blackburn & Maybury, 1985; Sethi et al., 2018).

It is not perfectly understood how the personality of a psychopath develops, but there are certain events in early life that may act as risk factors or predictors of psychopathy in adult life. The literature suggests an integrative approach between cognitive neuroscience and genetics to understand the development of psychopathy. We
intend to explore some aspects of the development of psychopathy by reviewing genetic, environmental, neurobiological, cognitive, and behavioral studies, enlightening how and why this disorder emerges and develops. Nevertheless, it is important to bear in mind that although we divided this chapter into different areas of research regarding possible psychopathy etiologies—for the sake of the organization—they are interconnected and related to each other.

Genetic and environmental research

The trajectory of each individual is determined by an interplay between genetic, social, and cognitive factors and even though the genome is quite important to the phenotypic expression, it is not a *sine qua non* condition of how an individual will be as an adult. The research on genetic influences in psychopathy requires the investigation of the interactions between genes and the social and contextual environment in order to improve the understanding of how genetic risks are associated with the disorder.

Although some psychopathic traits are observed in childhood, including the emotional impairment known as callous-unemotional (CU) traits, the psychopath label is not used to describe children. CU traits, however, do provide information for the comprehension of psychopathy during an individual’s life. They are related to a lack of empathy, guilt, or remorse, and are reported to be a developmental precursor to psychopathy (Frick & White, 2008). High CU traits are associated with lower levels of fear (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Petitclerc et al., 2019), and with a decrease in the response to negative emotional stimuli (Dadds et al., 2006), which are characteristics of psychopathic individuals.

Gene-association studies have focused on CU traits in children or adolescents that present some disruptive behavior (e.g., Fontaine, Rijsdijk, McCrory, & Viding, 2010; Moore et al., 2019; Muratori et al., 2016). Studies with twins have shown that individual differences in CU are estimated to be moderately to strongly heritable (e.g., Flom & Saudino, 2017; Viding & McCrory, 2015). In fact, recent genetic research suggests modest to high heritability of psychopathic traits (see Dhanani et al., 2018). Disruptive behaviors associated with high levels of CU traits appear to be strongly heritable, whereas disruptive behaviors with low levels of CU traits seem to have a major influence from environmental factors (Viding et al., 2005, 2008). For instance, negative parenting has been associated with higher levels of CU traits and disruptive behaviors, and a positive parental style has been associated with lower levels of CU traits and disruptive behaviors in children (see Waller, Gardner, & Hyde, 2013).

Longitudinal studies focusing on genetics have also investigated parenting and development of CU traits (Hyde et al., 2016; Waller, Shaw, & Hyde, 2016), and results have shown that the level of antisocial behavior and fearlessness in the biological mother predicted early CU behaviors in adopted children. Moreover, positive reinforcement provided by an adoptive mother was a protective factor against the development of CU traits even in the presence of significant biological risks, such as a biological mother with antisocial traits. Nonetheless, an adoptive mother with positive
reinforcement buffered the effects of heritable risk for CU behaviors; this may be explained by the high motivation levels and financial resources that adoptive parents habitually have, contrary to biological families, wherein parents of children with CU traits are more likely to have genetic and social risk factors involved.

Concerning the empathy and love from parents with children with high levels of CU traits, Dadds et al. (2012, 2014) found that compared with other children with disruptive behaviors, those with high levels of CU traits were less prone to establish eye contact with their mothers. The mothers of children with high levels of CU traits, on the other hand, did not differ from mothers of non-CU children with disruptive behaviors in terms of the amount of eye contact they had with their children. Furthermore, a recent cross-sectional study from Loney, Huntenburg, Counts-Allan, and Schmeelk (2007) found a significant association between maternal affective features of psychopathy and the CU traits of their children, wherein parenting dysfunction fully mediated the relationship.

Research has consistently revealed a significant overlap in genes that influence CU traits (Bezdjian, Raine, Baker, & Lynam, 2011; Forsman, Lichtenstein, Andershed, & Larsson, 2008) with the stability in CU traits being driven by genetics (Flom & Saudino, 2017; Fontaine et al., 2010). In order to explore the genetic and environmental overlap between psychopathic traits and antisocial behavior, Larsson et al. (2007) did a study with a longitudinal population based on 1480 twin adolescents. Multivariate twin methods were performed to analyze how genetic and environmental factors contributed to the associations between the psychopathic personality and antisocial behavior. Interestingly, a common genetic factor loaded substantially on both psychopathic personality traits and antisocial behavior, but a common shared environmental factor loaded solely on antisocial behavior. These observed shared environmental influences only in antisocial behavior may suggest a distinction between psychopathic personality dimensions and antisocial behavior etiologies (Larsson et al., 2007).

To further explore genetic influences, several authors (Beitchman et al., 2012; Dadds et al., 2014) have focused on variants of genes related to the serotonin and oxytocin systems. For example, Sadeh et al. (2010) found that the long allele of a serotonin transporter polymorphism—that was found to be related to a decreased amygdala reactivity—represented a genotype risk for elevated CU traits, but only in the context of lower socioeconomic backgrounds. This result demonstrates and reinforces the idea that the genetic vulnerability to CU traits is highly dependent on the deprived social environment in which the child is developing.

Besides serotonin, it has been theorized that there is a role for depleted oxytocin (OXT) in psychopathy (Bora, Yucel, & Allen, 2009; Dadds & Rhodes, 2008). Oxytocin is a neuropeptide, often called the “cuddly chemical,” and is strongly linked to prosocial behavior, empathy, and affiliation/attachment. Beitchman et al. (2012) have shown that common polymorphisms of common OXTR (oxytocin-receptor) single-nucleotide polymorphisms are associated with high levels of CU traits in samples that presented conduct problems. Subsequent studies have found that higher CU traits are associated with greater methylation of the oxytocin receptor gene (Cecil et al., 2014; Dadds et al., 2014).
More recently, Verona, Murphy, and Bresin (2018) suggested that oxytocin dysfunction may be related to the absence of loving and empathy in psychopathy. The authors examined the associations between three oxytocin-related single-nucleotide polymorphisms (SNP) and results showed that the rs53576 SNP on the oxytocin receptor and cumulative risk alleles across the 3 SNPs were associated with psychopathic traits. Moreover, the interaction between cumulative risk factors and a poor emotional environment was associated with affective deficits of psychopathy.

Tuvblad, Fanti, Andershed, Colins, and Larsson (2017) investigated the genetic and environmental sources among three psychopathic personality dimensions (grandiose-deceitful, callous-unemotional, and impulsive-need for stimulation) in 5-year-old children. The authors found that genetic and/or shared environment explained the majority of variance in all dimensions, and the proportions of the phenotypic correlations among these dimensions were mediated by genetic and shared environmental influences. Their results exhibited a moderate genetic influence (25%) and higher shared environmental influence (48%) on the callous-unemotional dimension, which goes in a contrary direction to what Viding et al. (2005) have found (i.e., high heritability [67%] for antisocial behavior in the presence of CU traits in a sample of 7-year-old twins, and no influence from shared environment).

A recent review of literature (Moore et al., 2019) presented 39 quantitative and/or molecular genetic studies on CU traits. Some of them included quantitative components, and the range of heritability was 25%–80%, depending on the study. Nevertheless, it is important to highlight that the percentage of heritability of CU traits in the general population of individuals in middle childhood, adolescence, and adulthood (between 36% and 67%) is not so different than most other temperament and personality traits of childhood and adolescence (see Polderman et al., 2015), a fact which maintains the debate concerning the genetic and environmental influences on CU traits.

Altogether, these findings provide support for the importance and interplay of both genetic and shared environmental risk factors (e.g., heritability, parenting style, social and contextual environment) in psychopathic personality traits. While Antisocial Personality Disorder (APD) shares a common genetic base with psychopathy (Larsson et al., 2007), environmental factors seem only to influence the development of APD, suggesting that APD is related to behavioral adaptations, whereas psychopathy may be innate (e.g., Freedman & Verdun-Jones, 2010). Further longitudinal and epigenetic research is needed, particularly with larger samples, in order to increase the understanding of psychopathy genetic and environmental etiology. Antisocial personality disorder may represent the phenotypic expression of the psychopathic genotype (Meloy, 2001).

**Family dynamics associated with intergenerational transmission of psychopathy**

Many theorists have examined the interaction between victimization at an early age and future offending, suggesting that victims have higher risk of future criminal behavior (CB) (e.g., Miley, Fox, Muniz, Perkins, & DeLisi, 2020). Adverse childhood
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experiences (ACEs), such as abuse and neglect during development, tend to promote negative behaviors as well as mental health problems throughout the lifespan (e.g., Baglivio & Epps, 2016; Craig, Piquero, Farrington, & Ttofi, 2017). In fact, data demonstrate that a large percentage of juvenile offenders have experienced trauma, abuse, neglect, or maltreatment in childhood, and the consequences of such increase significantly the chances of violence (see review Bland, Lambie, & Best, 2018).

A significant amount of research has studied both genetic and environmental risks involved in the transmission of CU traits in youth populations (e.g., Frogner, Gibson, Andershed, & Andershed, 2018). However, it is valuable to also examine the intergenerational similarity of these traits between parents and their children.

Intergenerational transmission (IT) can be explained by a combination of processes, such as social learning, criminogenic environments, biological proneness, and parenting dynamics that may result in disadvantage for the children. Literature in IT can be wide ranging, pointing to variables such as socioeconomic status (e.g., Bailey, Hill, Oesterle, & Hawkins, 2009), educational attainment (e.g., Wertz et al., 2018), substance use (Bailey et al., 2009), mental health (e.g., Serbin & Stack, 1998), parenting behaviors (e.g., Farrington, Coid, & Murray, 2009), and criminal behavior (e.g., Thornberry, 2005). The effects of parenting behaviors have been the most widely studied.

For instance, Kahn, Deater-Deckard, King-Casas, and Kim-Spoon (2016) have examined whether the association between parents and children, both with CU traits, was mediated by parenting behavior. The authors also assessed whether this association was moderated by household environment. Results showed that negative parenting mediated the association between parents and adolescents with CU traits in a high household chaos context. Additionally, household chaos intensified the effects of hostile parenting on CU traits in adolescents, resulting in susceptibility to IT of CU traits.

Other studies have shown that physical abuse, emotional neglect, and household violence or substance abuse predicted externalizing outcomes (e.g., Bonner, DeLisi, Jones-Johnson, Caudill, & Trulson, 2019; Muniz et al., 2019). Other problems experienced by the parents (e.g., unemployment, poverty, unsafe neighborhoods) may also impact children’s criminal behavior in the future.

Thornberry (2005, 2009) recognized a direct effect of antisocial behavior on one generation from the previous generation through genetic and social learning influences. He observed that delinquent adolescents confront more difficulties in transitioning into adulthood, and that criminal parents seem to use more severe parenting skills with their children, resulting in an increased children’s tendency for criminal behavior. Moreover, children with two criminal parents have an incremental risk of exhibiting antisocial behavior (Frick & Loney, 2002). However, criminal behavior is not directly conveyed from parents to offspring, but rather through a continuous pattern of criminogenic and antisocial features (e.g., Berti & Pivetti, 2019).

A systematic review made by Besemer, Ahmad, Hinshaw, and Farrington (2017) showed that children with criminal parents have about 2.4 times greater probability of criminal behavior in the future compared to children without criminal parents; the transmission seems strongest from mothers to daughters, followed by mothers to sons, fathers to daughters, and fathers to sons. In this regard, another interesting finding
highlighted from the literature is that a punitive environment (e.g., long incarceration sentences) might impact IT (see Besemer & Bui, 2019 for a meta-analysis).

Parental gender seems to be a critical factor in IT, since men and women typically do not engage in criminal behavior to the same degree and in the same way (e.g., Auty, Farrington, & Coid, 2015, 2017). Men are more likely to be involved in CB than women, and women who engage in criminal behavior might be more deviant compared with men, and are more likely to be the main caretakers of their children (Glaze & Maruschak, 2016). Similarly, young men and women tend to react differently to stressful life events such as parental criminal behavior. In general, young men show more externalizing problems (e.g., delinquency, antisocial behavior, aggression) while girls display more internalizing problems, such as depression and anxiety (Besemer et al., 2017; Glenn, 2019; Vieno, Nation, Pastore, & Santinello, 2009).

Studies have suggested that at least some of the origins of psychopathic traits are present in infancy and early childhood. But there is limited evidence about the relationship between parental and offspring psychopathy. A cross-sectional study by Loney et al. (2007) found a significant association between maternal affective features of psychopathy and the CU traits of their children, with parenting dysfunction fully mediating the relationship.

Auty et al. (2015) examined two consecutive generations in a large community sample in order to observe whether psychosocial risk factors can explain intergenerational transmission. Their findings provided the first evidence that the psychopathic personality traits of a community sample of men have a significant association with the psychopathic personality traits of their male and female offspring. This study also suggested that employment problems of the fathers played an important role in explaining the intergenerational transmission of psychopathic personality traits (Factor 1 scores) to both male and female offspring.

Schimmenti, Di Carlo, Passanisi, and Caretti (2015) reported a very high prevalence of emotional abuse among psychopathic offenders, and found a positive relationship between PCL-R Factor 1 and Factor 2 scores and emotional abuse history. The authors suggest that experiencing frequent emotionally abusive relationships in childhood may result in extreme levels of distress among children that ultimately reduce the ability of the child to relate to others. These experiences, in combination with other environmental risk factors, may emphasize dysfunctional affective and impulsive behavioral profile that typifies psychopathy.

Regarding the history of child abuse or neglect and its impact on psychopathy, Poythress, Skeem, and Lilienfeld (2006) proposed that both are positively but weakly related to global psychopathic features. Their findings revealed that abuse is unrelated to the core affective and interpersonal traits of psychopathy (Factor 1), but relates moderately to the impulsive and irresponsible lifestyle (Factor 2) or externalizing psychopathic features. Their findings suggest that early abuse and neglect may shut off affective responses, thereby resulting in individuals who possess the cold and callous features of primary psychopathy.

Gao, Raine, Chan, Venables, and Mednick (2010) demonstrated that poor parental bonding (lack of maternal care and low paternal overprotection) and childhood physical abuse were both associated with psychopathic personality. Those subjects
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separated from parents in the first three years of life were particularly characterized by low parental bonding and a psychopathic personality in adulthood; and the deviant behavior factor of psychopathy was related to lack of maternal care, whereas the emotional detachment factor was related to both lack of maternal care and paternal overprotection.

Dargis and Koenigs (2018) demonstrated that one subgroup of psychopathic offenders is characterized by a more extensive history of childhood maltreatment. More recently, it has been shown that men who experienced childhood abuse of any kind were more likely to demonstrate a fearful or preoccupied attachment style. Those who were securely attached were less likely to engage in antisocial behavior, and those who were more anxiously attached were less arrogant and deceptive in interpersonal relations (Grady, Looman, & Abracen, 2019).

Current evidence suggests that psychopathic tendencies in children are related to negative parenting styles that may have long-term effects on children’s future behaviors (Krupić, Ruićević, & Vučković, 2020). Since children may develop psychopathic tendencies by imitating their parents, this suggests that parents with psychopathic tendencies have a greater chance of raising children with propensities to psychopathy. However, it is worthy to mention that there are protective factors that decrease the effects of risk factors on later offending. Research indicates that early childhood interventions may not only help reduce future offending, but also increase the child’s chances of doing well in other life domains (Craig, Piquero, & Farrington, 2020).

**Neurobiological research**

Psychopathic traits are present in childhood, with psychological, behavioral, and temperamental differences detectable at an early age in individuals who develop psychopathic traits in adulthood (e.g., Frick, O’Brien, Wootton, & McBurnett, 1994; Glenn, Raine, Venables, & Mednick, 2007; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007).

Studies through the years have also been conducted to determine the psychophysiological functioning of the psychopathic individual (e.g., Hare, 1970). For instance, electroencephalography (EEG) studies have been performed in order to comprehend the neural network and the specific dynamics of individuals with psychopathic traits (e.g., Gao et al., 2018; Hare & Quinn, 1971). Clark, Bontemps, Batky, Watts, and Salekin (2019) reviewed 68 studies that focused on psychopathy and several components of EEG. Results show that psychopathic individuals have robust early selection processes paired with reduced or elaborative sustained processing of emotional information. This may suggest the existence of automatic and strategic aspects related to the information processing of the psychopathic individual. Those with higher psychopathic traits spend less time processing distress stimuli, which meets the idea that the LPP (Late Positive Potential) amplitude can be attenuated by voluntary top-down control (see Hajcak, MacNamara, & Olvet, 2010). Thus the psychopathic individual decides which stimuli to ignore or follow (Meloy, Book, Hosker-Field, Methot-Jones, & Roters, 2018).
We addressed in the previous section of this chapter that children with disruptive behaviors and CU traits show a lack of empathy, guilt, or remorse, and they engage in instrumental aggression (Viding & McCrory, 2015). The literature on children with disruptive behaviors and CU traits has focused mainly on the processing of emotions and in the relationship between children and their caregivers. In this regard, functional magnetic resonance imaging (fMRI) studies of children and adolescents with disruptive behaviors and CU traits have tried to understand the functioning of brain areas that are implicated in affective and reward processing (see Puzzo, Smaragdi, Gonzalez, Martin-Key, & Fairchild, 2016). These studies revealed that children with disruptive behaviors and high levels of CU traits display a reduced activity and altered connectivity in brain areas that are associated with empathy for others seen in healthy individuals.

Blair, Leibenluft, and Pine (2014) have shown a neural activity profile consistent with low emotional responsiveness to others suffering, and a weak ability to learn from reinforcement. It has been shown that dysfunction in the amygdala assumes a great importance in psychopathy (Blair, 2005; Waller et al., 2019). Specifically, reduced volume of the amygdala has been consistently reported with psychopathic individuals (Boccardi et al., 2011; Yang, Raine, Narr, Colletti, & Toga, 2009). These impairments likely occur early in life (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006), although it is not perfectly clear how and why this happens.

The amygdala is a neural structure implicated in aversive conditioning, necessary for enhancing attention to emotional stimuli, facilitating empathy for others, and the detection of threats (Blair, 2006; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002). It is also significant for the recognition of fearful facial expressions (Blair, Colledge, Murray, & Mitchell, 2001) and the augmentation of the startle reflex by visual threat stimuli (Levenston, Patrick, Bradley, & Lang, 2000). White et al. (2012) found unusually low amygdala activation to fear stimuli in children with disruptive behaviors and CU traits under low attentional load conditions, while fear stimuli elicited amygdala activation in healthy participants. Furthermore, reduced amygdala and insula activity have been observed in children with disruptive behaviors and high CU traits. These children engage in more complex forms of behaviors, such as social judgment regarding other people’s distress, making decisions about whether they will benefit themselves by harming others, or making decisions about appropriate responses to the distress of others (Marsh et al., 2011; Sakai et al., 2017; Sebastian et al., 2012, respectively). Other studies of children exhibiting disruptive behaviors and varying levels of CU traits have consistently reported atypical neural reactivity to the pain of others (Michalska, Zeffiro, & Decety, 2016; Yoder, Lahey, & Decety, 2016).

Since the amygdala represents a binding site for steroid hormones, one possible explanation for its impairment is that hormones imbalances, before birth or in early childhood, affect the development of subcortical structures, and may continue to influence functioning into adulthood (van Honk & Schutter, 2006). Because it also affects gene transcription, hormones have the ability to change functioning by increasing or decreasing the probability of certain behaviors in response to threat (Schulkin, 2003).

Likewise, there are other genetic and neurotransmitter features that may affect amygdala functioning. The amygdala has numerous serotonergic inputs and may be
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Sensitive to changes in serotonin transmission. In this context, Blair (2007) performed a study demonstrating that individuals who are homozygous for the long version of the serotonin transporter gene (5-HTTLPR) show significantly reduced amygdala responses to emotional expressions relative to those with the short-form polymorphism (Brown & Hariri, 2006), and behavioral impairment of emotional learning tasks that depend on the amygdala (Finger et al., 2007).

Serotonin neurotransmission has an effect on the hypothalamic-pituitary-adrenal (HPA) axis. Increased activity at serotonin receptor sites in the hypothalamus raise the production of cortisol, which is important since it mobilizes the resources in the body and provides energy in stressful times (Kudielka & Kirschbaum, 2005). Cortisol is also involved in potentiating the state of fear (Schulkin, Gold, & McEwen, 1998), sensitivity to punishment, and withdrawal behavior (Schulkin, 2003). When a disruption of serotonin neurotransmission happens, it will trigger off cortisol reactivity to a stress-inducing task (e.g., Sobczak, Honig, Nicolson, & Riedel, 2001). Hence, dysregulation of serotonin in the brain may contribute to the low cortisol levels observed in psychopathy.

Serotonin may also interact with testosterone levels to increase the probability of violent aggression (Birger et al., 2003). van Honk and Schutter (2006) propose that the underlying source of emotional deficits observed in psychopathy is a result of an imbalance of cortisol and testosterone. Testosterone is a product of the hypothalamic-pituitary-gonadal (HPG) axis and is associated with approach-related behavior, as well as reward sensitivity and fear reduction (Boissy & Bouissou, 1994). Testosterone and cortisol have antagonistic properties. On one hand, low serotonin levels combined with high testosterone levels increase the rates and intensity of aggression (Birger et al., 2003; Higley et al., 1996). On the other hand, cortisol suppresses the activity of the hypothalamic-pituitary-gonadal (HPG) axis on all levels, decreasing testosterone and inhibiting its effects (Tilbrook, Turner, & Clarke, 2000). And in turn, testosterone inhibits activity of the HPA axis (Vial, 2002). van Honk and colleagues have found that injections of testosterone reduce fearfulness (van Honk, Peper, & Schutter, 2005), promoting the response to angry faces (van Honk, Tuiten, et al., 2001), and shifting the balance from punishment to reward sensitivity (van Honk et al., 2004).

It has been hypothesized that deficits in the fronto-temporal neural circuit, particularly the ventromedial prefrontal cortex, dorsolateral prefrontal cortex, medial temporal regions, and cingulate cortex, may play important roles in the neurobiology of psychopathy (Kiehl, 2006). Indeed, abnormalities have also been observed in other subcortical regions such as the hippocampus. Raine et al. (2004) found asymmetries within the hippocampus in imprisoned psychopaths. Atypical brain asymmetries are thought in part to reflect disrupted neurodevelopmental processes (Best, 1988), thus structural asymmetries in psychopaths may reflect an interruption to the normal developmental process. Laakso et al. (2001) found psychopathy to be negatively correlated with the volume of the posterior hippocampus. The hippocampus has solid interconnections to the amygdala and prefrontal cortex, which are also implicated in psychopathy.

Prefrontal impairments have been linked to poor decision making, while deficits in the temporal regions seem to contribute to the lack of emotion and social dysfunction.
in psychopathic individuals. Raine, Lencz, Bihrlle, LaCasse, and Colletti (2000) observed a reduction in prefrontal gray matter volume in a group of individuals with antisocial personality disorder (11%) compared to normal and psychiatric control groups. The individuals with APD also showed reduced skin conductance activity during a social stress test, and those with low prefrontal gray showed particularly reduced stress reactivity. These results support evidence that prefrontal regions are involved in generating somatic states. In this regard, van Honk, Schutter, et al. (2001) provided further empirical evidence by using repeated transcranial magnetic stimulation (rTMS) to inhibit the activity of the orbitofrontal cortex and found significant reductions in skin conductance responses. In a study by Yang et al. (2005), reduced (22%) prefrontal gray matter was observed in a group of unsuccessful psychopaths.

Yang, Raine, Colletti, Toga, and Narr (2009) also identified differences in cortical gray matter thickness in psychopaths compared with nonpsychopathic controls. Psychopaths showed significant cortical gray matter thinning in the right frontal and temporal cortices compared with a nonpsychopathic control group. The orbitofrontal cortex is associated with the anticipation of punishment and reward, as well as with the response reversal during changing reinforcement contingencies, and social cognition (Mitchell, Colledge, Leonard, & Blair, 2002). Functional imaging studies have shown reduced activity associated with psychopathy in the orbitofrontal cortex during fear conditioning (Birbaumer et al., 2005; Veit et al., 2002), and during a socially interactive game (Rilling et al., 2007). Older research demonstrated that damage to the orbitofrontal cortex often resulted in pathological lying, promiscuous sexual behavior, shallow affect, lack of guilt or remorse, and irresponsibility (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999), all characteristics of psychopathy personality.

The orbital frontal cortex and the parietal lobes are responsible for working memory, the ability to plan and organize behavior, and behavioral inhibition. These structures also aid in the capacity to learn from experience (a deficit characteristic of psychopathic individuals) and are deeply entangled with the limbic system. In an fMRI study, Raine et al. (2000) found that individuals with APD had less gray matter within the prefrontal cortex compared to individuals who were not diagnosed with APD. Several studies have observed increased activation in higher cognitive areas such as the dorsolateral prefrontal cortex during emotional tasks in psychopaths compared to controls (Gordon, Baird, & End, 2004; Intrator et al., 1997; Rilling et al., 2007). In sum, significant gray matter thinning suggests that the structural correlates of psychopathy may be linked to the emotional deficits portraying psychopaths.

Psychopathy has also been associated with abnormalities in other regions. For example, reduced functioning in the anterior cingulate (connected with the amygdala and involved in emotional processing) has been observed during experimental conditioning (Birbaumer et al., 2005; Veit et al., 2002) in criminal psychopaths in the processing of emotional information (Müller et al., 2003). Deficits in the angular gyrus (posterior superior temporal gyrus) have been found in psychopathic and antisocial individuals, and deficits in functioning of the posterior cingulate may be involved in self-referencing and experiencing emotion (Kiehl et al., 2004). Reduced functioning of the insula has also been observed during fear conditioning (Birbaumer et al., 2005; Veit et al., 2002). Kiehl (2006) argued for a paralimbic system dysfunction of psychopathy, but in
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Reality, it is quite difficult to determine whether each region associated with psychopathy makes an exceptional and exclusive contribution to the disorder.

In addition to the abnormal functioning observed in certain brain areas of psychopathic individuals, some studies have also explored the connectivity between areas. Van Honk and Schutter (2006) hypothesize that disruptions in the connectivity between subcortical and cortical regions may contribute to psychopathy. Such connectivity, according to Damasio (1994), allows emotional information from subcortical regions to provide input to cortical regions, which is important for decision making and cognitive evaluation. Connectivity between the amygdala and orbitofrontal cortex may be important in the generation of somatic markers.

The orbitofrontal cortex receives emotional information from the amygdala and stores representations of certain stimuli that can be later retrieved. If an individual recalls or anticipates these stimuli, the orbitofrontal cortex triggers the somatic state, and if the connections between the amygdala and orbitofrontal cortex are disrupted, the orbitofrontal cortex will be unable to form representations, and anticipatory fear of aversive events will not be produced (Bechara, Damasio, & Damasio, 2003). Indeed, reduced connectivity between the orbitofrontal cortex and the amygdala has been associated with lower sensitivity to threat cues (Buckholtz et al., 2008). Thus poor connectivity between these areas would result in reduced regulation of subcortical structures by prefrontal areas, contributing to the disinhibition and reactive aggression observed in psychopathy. Psychopaths also display impaired connectivity between the two hemispheres. Hiatt and Newman (2007) found that the time required to transfer information from one hemisphere to the other is significantly lengthier in criminal psychopaths compared to criminal nonpsychopaths.

Lead (Pb) exposure has also been linked to behaviors that are related to psychopathy (Braun, Kahn, Froehlich, Auinger, & Lanphear, 2006; Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996). Lead exposure has been found to be associated with deficits in frontal lobe functioning. For instance, Trope, Lopez-Villegas, Cecil, and Lenkinski (2001), in a study with magnetic resonance spectroscopy, found that lead-exposed youths had a higher level of neuronal loss and damage within the frontal cortex than healthy participants. Meng, Zhu, Ruan, She, and Luo (2005) also found that lead exposure was associated with a lower density of neurons in the frontal lobe and hippocampus. An additional investigation with magnetic resonance imaging data from the Cincinnati Lead Study (Cecil et al., 2008) revealed that childhood lead exposure corresponded to significantly lower levels of adult gray matter within the frontal lobe. Thus results from brain imaging studies (e.g., Bellinger, 2008) demonstrate that lead exposure is related to deficits in brain structure and functioning, especially within the frontal lobe.

A longitudinal study (Dietrich, Ris, Succop, Berger, & Bornschein, 2001) revealed that prenatal and postnatal blood lead levels were related to both parent-reported and child-reported delinquent behaviors. In a follow-up investigation of these youths, results showed that prenatal and postnatal blood lead levels were associated with the number of criminal arrests in adulthood (Wright, Dietrich, Ris, Hornung, & Wessel, 2008). To further explore these effects and transpose to psychopathy, Wright, Boisvert, and Vaske (2009) investigated whether childhood blood lead concentrations were related
to psychopathy in adulthood. Results revealed that higher blood lead concentrations in early childhood were indeed associated with higher levels of psychopathic traits in adulthood, controlling for the effects of gender, race, mother’s intelligence quotient, child’s intellectual achievement, and the quality of the home environment. The authors also concluded that childhood lead levels predicted variation in Machiavellian Egocentricity, Social Potency, Impulsive Nonconformity, and Blame Externalization, dimensions that represent an individual with CU traits. Overall, these studies associate lead exposure to the etiology of psychopathy.

**Cognitive and behavioral research**

The increased amount of research in psychopathy has provided important advances in the understanding of this disorder. We started this chapter by pointing to some of the personality traits present in psychopathic individuals, taking the previous psychopathy definition and research work of Cleckley (1941, 1976) and Hare (1991, 2003). Research has continually yielded insights into personality traits in psychopathic individuals associated with proneness to impulse-related disorders and to recidivism (Hare, 2006).

According to Lilienfeld and Fowler (2006), individuals with clinical and/or criminal levels of psychopathy have the same personality profiles as their subclinical counterparts (nonclinical). Thus what really seem to differentiate one from another is the frequency, degree, and intensity with which the individuals engage in the deviant behaviors (LeBreton, Binning, & Adorno, 2006), suggesting that psychopathy is a continuum construct in which individuals can vary from low, moderate, to high levels of psychopathy (Lilienfeld & Fowler, 2006).

As mentioned earlier in this chapter, psychopathic personality traits include affective deficits (e.g., shallow affect, lack of remorse and empathy, callousness), as well as dysfunctional personality features associated with social functioning (e.g., manipulativeness, egocentricity, insincerity; Cleckley, 1941, 1976; Hare, 2003). Although distinct, it is important to keep in mind that psychopathy shares many traits similar to other personality disorders, in particular with APD.

As discussed earlier, CU traits have been studied as a symptomatic component of psychopathy. However, it cannot be considered a singular trait that predicts psychopathy. A recent study (Salekin, Andershed, Batky, & Bontemps, 2018) argued that psychopathy is more predictive of negative outcomes than CU traits by themselves, or CU traits + Conduct Problems (CP, such as breaking of norms, rules, or laws resorting or not to violence). Severe CP in childhood are associated with CU personality traits. Children with both CP and CU traits constitute a subgroup of CP children who exhibit a more severe and stable form of CP, more emotional and cognitive deficits, and also more psychiatric morbidity compared to children with CP without co-occurring CU traits (Frick, Ray, Thornton, & Kahn, 2014).

Salekin (2017) has emphasized the utility of considering interpersonal, affective, and lifestyle/antisocial dimensions of psychopathic traits when assessing youth with CD. It is well known that prosocial judgments and regulation capacities become more
developed in middle childhood (McKown & Weinstein, 2003). Children who are characterized by psychopathic traits have more difficulties in prosocial reasoning and self-regulation during middle childhood (Poythress et al., 2006).

The most common and widely used instrument to assess psychopathy in adults is the PCL-R (Hare, 1991, 2003), a 20-item rating scale that comprises two correlated factors: Factor 1 and Factor 2, described earlier. There is considerable empirical evidence linking psychopathy assessed by the PCL-R with criminality and violence (see Edens, Campbell, & Weir, 2007). Others have proposed a three-factor model (Cooke & Michie, 2001); however, cross-cultural research and the aggregation of large samples from various countries support a two-factor model with both factors (1 and 2) subdivided into two facets (Mokros et al., 2015; Neumann, Hare, & Pardini, 2015). There are also other instruments to assess the development risk for psychopathy in children and adolescents, particularly the risk of persistent antisocial behavior:

- Antisocial Process Screening Device (Frick & Hare, 2001),
- Youth Psychopathic Traits Inventory (Andershed, Gustafson, Kerr, & Stattin, 2002),
- Inventory of Callous Unemotional Traits (Essau, Sasagawa, & Frick, 2006),
- Child Problematic Traits Inventory (Colins et al., 2014).

None of the instruments would be relevant if there were no models and theories of personality to support them. The acknowledgment of the value and usefulness of models of personality such as the Five-Factor Model (FFM, McCrae & Costa, 1990), whereby personality disorders have been conceptualized, is also a major advance in the research of psychopathy.

FFM comprises five domains and thirty facets assessed by NEO PI-R. Although McCrae and Costa (2013) have noticed that there are traits that are not included within the FFM facets, the NEO PI-R is the most validated instrument to evaluate the personality profiles of normals cross-culturally.

In light of the FFM, psychopathy has been reliably described as the convergence of low agreeableness and low conscientiousness (Lynam, Leukefeld, & Clayton, 2003), regardless of the approach used to create the psychopathic profile (for translation of psychopathy measures into FFM traits, see Lynam & Derefeniko, 2006). Some studies supported that Factor 1 and Factor 2 were related to low agreeableness, but differed in Factor 2, which was more strongly related to low conscientiousness and high neuroticism (Lynam & Derefeniko, 2006).

Follow-up studies have shown fluctuating relations between Factor 1 measures and agreeableness and the other FFM dimensions (e.g., Seibert, Miller, Few, Zeichner, & Lynam, 2011). Relatedly, higher-order factors of Constraint and Negative Emotionality from Tellegen’s temperament-oriented personality model (Patrick, Curtin, & Tellegen, 2002; Tellegen & Waller, 2008) were associated with antisocial personality disorder and substance dependence, and both predict the occurrence of these situations (Krueger, 1999) and delinquency (Caspi et al., 1994).

Research on the stability of psychopathic traits during the life course of an individual has shown consistency in the structure and correlates of psychopathic traits as assessed with clinical measures of psychopathic traits in adolescence and in adulthood. Nevertheless, there is less agreement about the nature of psychopathic traits in...
childhood. As we presented in a previous section of this chapter, some studies suggest that psychopathic traits begin in early age and they are partly heritable and undeniably manifested in childhood (e.g., Viding & McCrory, 2018). According to Kosson et al. (2013), psychopathy in adolescence and in adulthood is underpinned by four correlated dimensions consisting of interpersonal traits, affective, lifestyle, and antisocial traits—the four-facet theory of Hare (Neumann et al., 2015). Psychopathy is characterized by several dimensions that can be identified in children as young as two or three years of age (e.g., Flom & Saudino, 2017). The combined research findings also suggest that psychopathic traits in youth may imply the development of the homogeneous disorder of psychopathy identified in adults.

Recently, Gorin et al. (2019) performed a study that aimed to understand the nature of psychopathic traits in youth by examining the fit of one-, two-, three-, and four-factor models of psychopathic traits during middle childhood (in the ages of 6, 7, and 8 years old). Additionally, they intended to examine the stability of the factor structure of psychopathic traits over time. The main results showed that a hierarchical three-factor model of psychopathic traits, consisting of interpersonal, affective, and lifestyle/antisocial factors, was the best fit to the data at all ages, thus providing evidence that the internal structure for psychopathic traits in middle childhood is similar but slightly less differentiated than the factor structure identified during adolescence. This study strongly suggests that psychopathy is a homogeneous syndrome with observed traits as early as six years of age.

Conclusions

This chapter aimed to explore the different etiologies of psychopathy, illustrated by scientific evidence throughout the decades. We presented several risk factors that underlie psychopathy related to genetic, social environment, family dynamics, neurobiological, and cognitive variables, which are all interconnected.

Inheritance of CU traits and the influence from environmental factors are well established. Research has consistently revealed a significant overlap in genes that influence CU traits (Bezdjian et al., 2011), with the stability in CU traits being driven by genetics. Moreover, studies have demonstrated the idea that genetic vulnerability to CU traits is highly dependent on the deprived social environment within which the child is developing. In a real sense, the environment shapes the expression of CU traits in the child.

Family dynamics also are extremely important as predictors of antisocial and criminal behavior in their offspring. Risk factors such as a poor lifestyle (poverty, unemployment, substance abuse, unsafe neighborhood), parent’s background, parenting styles, and their psychological structure have an enormous negative impact. The presented findings provide support for the importance and interaction of genetic and environmental risk factors (e.g., heritability, parenting style, social and contextual environment) in the development of psychopathic traits.

Psychopathy is a complex syndrome. Despite popular interest in the topic and the substantial body of new scientific research published each year, it remains unclear how genetic and environmental/social risk factors contribute to the development of a
pattern of neurocognitive processing that increases the risk for psychopathy. Likewise, we still do not know how that particular processing may influence the environmental and contextual settings of the individual’s life course.

There are more than 85,000 publications on psychopathy, and despite the continued writing and research, there is yet much to learn and to be discovered. In fact, what we already know about the different etiologies and expressions of this disorder are important markers that guide new research pathways in which new answers will be given and new questions will arise.

References


The emergence and development of psychopathy


Marques, 978-0-12-811419-3


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Abstract
Psychopaths are individuals with persistent antisocial deviance and often criminal behavior, characterized by a lack of empathy, poor behavioral control, and deficient emotional responses. It is not yet clear how the personality of a psychopath develops, but there are events in early life that may act as risk factors or predictors of psychopathy in adult life. The extant research suggests an integrative approach between cognitive neuroscience and genetics to understand the development of this personality. Although it may be hardwired from genetics, it also may be influenced by social environment and family dynamics. In this chapter, we intend to explore some aspects of the development of psychopathy by reviewing genetic, environmental, neurobiological, cognitive, and behavioral studies, enlightening how and why this condition emerges and develops.

Keywords: Psychopathy, Development, Genetics, Neurobiology, Behavior, Environment