Journal of Young Investigators RESEARCH REVIEW

The Fear Factor: Fear Deficits in Psychopathy as an Index of Limbic Dysregulation

Vasileia Karasavva[1]

Psychopathy is a constellation of distinct interpersonal (e.g., pathological lying), affective (e.g., lack of remorse), lifestyle (e.g., need for stimulation), and antisocial (e.g., poor behavioral controls) traits that contribute to a deceptive and exploitive personality profile. Current theoretical models attempting to explain the functional impairments and neural systems behind the behavioral profile of the disorder seem to converge on the idea of a fear deficit. The purpose of this essay was to investigate the positive relationship between psychopathy and fear deficits as well as the dysregulation of the limbic system in adults. Research shows that, in response to a threatening or fearful situation, psychopathy is associated with a reduced capacity to experience negative valence, diminished autonomic response, and difficulty in recognizing fear-related cues. In addition, psychopathy has also been implicated in abnormalities in the fear center of the brain, the limbic system. This includes structural, morphological, and functional alterations of limbic structures like the amygdala. Future research is needed to better explain the underlying causes of these brain abnormalities among psychopathic individuals and to investigate the contributing factors, whether innate or environmental, that lead to the development of the abnormal features.

INTRODUCTION

Since its initial conception by Phillippe Pinel, psychopathy has been recognized and diligently pursued in clinical, forensic and personality research (Serin et al., 2011). The term psychopathy refers to a personality disorder characterized by a collection of interpersonal, affective and behavioral deficits (Hare and Neumann, 2008). Specifically, psychopathic individuals often exhibit affective dullness, an egocentric and manipulative interpersonal style, and often engage in reckless and antisocial behaviors with little regard for any negative impact their actions might have on others (Hare et al., 1990). Although there is some overlap with those characteristics and the diagnostic criteria for antisocial personality disorder (APD), psychopathy is a separate and narrower construct that places greater emphasis on affective deficits (Blair et al., 2005). Thus, nearly all individuals with psychopathy can be diagnosed with APD (Serin et al., 2011), but only a third of those diagnosed with APD are psychopaths (Wynn et al., 2012). The most frequently-used diagnostic tool for the assessment and diagnosis of psychopathy is the Hare Psychopathy Checklist-Revised (PCL-R) (Hare et al., 2000). A score of 30 on the PCL-R is considered the accepted threshold for psychopathy (Hare et al., 1991) and it is suggested that it represents individuals who fall on the extreme tail of a continuously-distributed personality trait (Edens et al., 2006). Although the clinical and theoretical importance of psychopathy is well-established, a number of issues regarding the construct, including the factors that predict and explain psychopathic symptomatology, are yet to be resolved (Edens et al., 2006).

Psychopathic traits are highly compatible with risk-taking, antisocial behaviors. This association is reflected by the fact that, despite psychopathic individuals comprise less than 1% of the general population (Coid et al., 2009), approximately 10 to 25% of adult offenders can be classified as psychopaths (Serin et al., 2011). Offenders with psychopathic tendencies are often versatile in their offending, highly prolific, and have longer and more violent criminal careers compared to non-psychopathic offenders (Serin et al., 2011). Psychopathic tendencies are also positively correlated with sexual aggression (Porter et al., 2003), violent sexual offences (Brown and Forth, 1997), and sexual sadism (Knight and Guay, 2006), a paraphilia that includes sexual arousal to fantasies, urges, or acts of inflicting pain, suffering, or humiliation onto another person (American Psychiatric Association, 2013). Yet, not all psychopaths are criminals as many psychopathic individuals are able to function in society without offending (Serin et al., 2011). Rates of psychopathy in corporate positions have been estimated to be five times higher than those of the general population (Babiak et al., 2010) but this might still reflect negative societal effects as is echoed by Hare's sentiment that "[we] are more likely to lose our life savings to an oily tongued swindler than our lives to a steely-eyed killer" (Hare, 1993). This great negative influence that the psychopathic population exerts onto society has spurred interest in creating a transtheoretical model capable of explaining the deficits behind key traits that ordinarily buffer or moderate the antisocial or violent behaviors that characterize psychopathy (Serin et al., 2011).

[1] Department of Neuroscience, Carleton University, Ottawa, Ontario, Canada, K1S 5B6

*To whom correspondence should be addressed: vasiakaras@gmail.com

doi:10.22186/jyi.36.6.73-80

One of the earliest theoretical models of psychopathy, the punishment insensitivity hypothesis, suggests that psychopathic individuals have a deficit in the ability to avoid punishment and experience less negative valence in response to it (Serin et al., 2011). In contrast, Newman's response modulation hypothesis argues that psychopathic individuals possess an impaired ability to reallocate attention to environmental cues when engaging in goal-directed behaviors (Newman et al., 1990; Newman et al., 1987). Newman suggests that in psychopathy, the automatic shift of attention necessary to incorporate contextual stimuli is dysregulated (Newman et al., 1990). Thus, secondary information, including affective and fear-related stimuli, is ignored, unless it serves a crucial role for the pursuit of a specific goal (Blair, 2013; Newman et al., 2010). In other words, according to this model, psychopathy is characterized not by an inability to escape punishment but rather by an attention dysregulation that impairs threat or punishment detection (Blair, 2013). Finally, the low-fear hypothesis suggests that psychopaths have a core fear-processing deficit that is expressed by a low level of subjective experience of fear and a reduced impact of aversive stimuli on emotional centers of the brain (Fowles, 1980; Lykken, 1957; Moul et al., 2012). In turn, this is translated to a failure to correct dysfunctional actions, avoid fear-provoking situations or learn about fear (Moul et al., 2012). Thus, taken together, a clear pattern emerges as the major models of psychopathy converge on the notion of impaired threat detection and responsivity, or a fear deficit in psychopathy (Moul et al., 2012).

Fear refers to an emotion induced by a real or perceived threat that causes a change in metabolic and organ function and ultimately a change in behavior, including fleeing, fighting or hiding (Weiten and McCann, 2006). Fear has a conscious component that refers to the recognition of a threat and the negative valence that accompanies it, as well as an automatic component that refers to the response to the fear-related stimulus (Hoppenbrouwers et al., 2016). This response could be internal and physiological, like an increase in skin conductance and heart rate or an externalizing behavior such as freezing, fleeing or startling (Hoppenbrouwers et al., 2016). The fear center of the brain is thought to be the amygdala, one of the major structures of the limbic system (LeDoux, 2003).

In addition to responding to fear-related stimuli, the amygdala is also thought to regulate the encoding and storage of emotional and fear-related memories in complex vertebrates, including humans (Duvarci et al., 2009; Kilpatrick and Cahill, 2003; Richardson et al., 2004; Schage et al., 2000; Walker and Davis, 1997). Additionally, the amygdala is thought to be essential for the evaluation of the affective significance of stimuli, particularly of those pertaining to fear (LeDoux, 2003; Whalen, 2007). Two meta-analyses of human neuroimaging data have shown that the amygdala responds preferentially to fear stimuli compared to other categories of emotional cues (Phab et al., 2002; Murphy et al., 2003). Research has established that damage to the limbic system, particularly in the extended amygdala, results in reduced fear-conditioning (Nader et al., and LeDoux, 2001), startle reflex (Angrilli et al., 1996), avoidance learning (Alkire et al., 2001), and subjective experience of fear (Feinstein et al., 2011). This can leave the individual vulnerable to dangerous situations that could have been avoided due to fear (Brand et al., 2007; Nesse, 1994). Because psychopathy impairs performance in these tasks as well, it is hypothesized that limbic dysfunction is implicated in the disorder (Glenn et al., 2010; Hosking et al., 2017). In fact, changes in the amygdala have been identified as a likely source of deficient processing of fear-related cues in psychopathy (Glenn et al., 2010; Hosking et al., 2017; Schiffer et al., 2011; Yang et al., 2009). In addition, structural and functional alterations of the limbic system have been consistently observed in psychopathic populations (Glenn et al., 2010; Hosking et al., 2017; Schiffer et al., 2011; Yang et al., 2009). Converging evidence from electrophysiology (Kiel et al., 1999; Kiel, Hare et al., 1999), functional imaging (Birbaumer, et al., 2005; Kiehl et al., 2001), and lesion studies (Mallow et al., 1993) suggests that the limbic system of psychopathic individuals is dysfunctional. Therefore, increased rates of high-risk or antisocial behavior in psychopaths may suggest an impaired ability to experience fear and consequently, an underlying dysfunction of the limbic system. Further analysis of the brain abnormalities in psychopathy could provide a more nuanced understanding of the antisocial and often criminal behaviors that accompany the disorder to better inform the treatment practices used with psychopathic individuals.

The purpose of this review is to demonstrate that psychopathic tendencies in adults are positively correlated with fear deficits (Caes et al., 2012; Gillen et al., 2018; Marsh et al., 2011) and dysregulations of the limbic system (Boccardi et al., 2010; Boccardi et al., 2011). More specifically, this paper examines the relationship between fear and psychopathic symptomatology; explores brain abnormalities, particularly in the fear centers, in psychopaths; and identifies potential limitations, implications, and future research directions.

PSYCHOPATHY AND FEAR DEFICITS

Affective fearful responses and psychopathy

Aversive or threatening cues typically result not only in the mobilization of defensive actions via the activation of the autonomic nervous system but in the subjective experience of negative valence as well (Weiten and McCann, 2016). Researchers have investigated the affective fearful response of psychopathic individuals by asking participants to recall in great detail a recent instance in which they experienced fear (Marsh et al., 2011). Psychopathic participants reported experiencing fear less frequently and less strongly than their non-psychopathic peers (Marsh et al., 2011). Additionally, researchers have examined whether exposure to fear-evoking situations in a lab setting can produce feelings of fear and a negative emotional state in psychopathic individuals (Caes et al., 2012; Rothemund et al., 2012). In this line of research, aversive Pavlovian delay conditioning, a learning procedure in which an aversive stimulus, like a painful electrical shock, is paired with a neutral cue that acts as a pain signal, is often employed (Caes et

al., 2012; Rothemund et al., 2012; Veit et al., 2013). In this task, a participant could either experience the electrical shock themselves following the pain signal, or witness someone else experience the shock punishment (Caes et al., 2012; Rothemund et al., 2012; Veit et al., 2013). After the task, the participants were asked to rate to what extent they experienced fear during the pain signal (Caes et al., 2012; Rothemund et al., 2012; Veit et al., 2013). Compared to the non-psychopathic control group, on average, psychopathic participants reported lower levels of fear and negative valence regardless of whether the painful stimulus was directed towards themselves (Rothemund et al., 2012) or to someone else (Marsh et al., 2011; Veit et al., 2013). It should be noted that these reduced self-reported feelings of fear were not correlated with higher levels of pain tolerance (Caes et al., 2012), reduced perception of other's pain (Rothemund et al., 2012), or worse memory of fearful events (Marsh et al., 2011). Taken together, the results suggest that an affective, rather than a perceptual or a somatosensory, deficit is present in psychopathy (Caes et al., 2012; Rothemund et al., 2012; Veit et al., 2013). This finding supports the idea that psychopathic individuals experience depleted feelings of fear in response to threatening or aversive situations (Caes et al., 2012; Rothemund et al., 2012; Veit et al., 2013).

Somatic fearful responses and psychopathy

Since self-reports rely heavily on honesty and the introspective ability of the responder, they may not be the most valid method of measuring emotional experiences in individuals like psychopaths, who demonstrate shallow affect and are prone to lying (Krumpal, 2013). Therefore, research has also focused on obtaining more objective, quantitative measures of fearful responses based on the activation of the sympathetic division of the autonomic nervous system (ANS) (Caes et al., 2012; Marsh et al., 2011; Rothemund et al., 2012; Vaidyanathan et al., 2011). Such measures include increased heart rate, skin conductance, or event-related potentials that are routinely used as an index of ANS activation (Hoppenbrouwers et al., 2016). In studies employing the Pavlovian delay task, psychopathic traits were negatively correlated with scores in all these measures in response to the pain signal (Caes et al., 2012; Rothemund et al., 2012). Furthermore, when asked to describe recent fearful events that they were involved in, psychopathic participants reported experiencing fewer symptoms of sympathetic nervous system arousal at the time of the event (Marsh et al., 2011). Additionally, blink reflex potentiation is an automatic, defensive blinking of the eyelid in response to threatening stimuli that has been established in both humans and animals (Bradley et al., 2008). However, when viewing aversive, fear-evoking pictures, psychopathic traits were negatively correlated with blink reflex potentiation (Vaidyanathan et al., 2011). Thus, research based on objective, somatic symptomatology of sympathetic ANS activation collectively suggests that the fear-associated autonomic reflex is diminished or absent in psychopathy, possibly indicating that a psychopath's inability to experience fear has a physiological component (Bradley et al., 2008; Marsh et al., 2011; Vaidyanathan et al., 2011). This could potentially explain the blunted affective fearful response, since the interpretation of the ANS activation is crucial for the subjective experience of an emotion (Caes et al., 2012; Marsh et al., 2011; Weiten and McCann, 2016). If the arousal is unnoticed or not given any thought, then the individual will not experience emotion (Weiten and McCann, 2016). Therefore, under-arousal of the ANS in psychopathy could contribute to the dysfunction in fear responding seen in psychopathy and consequent less-aversive emotional response to threatening or fearful stimuli (Marsh et al., 2011). It is also hypothesized by some researchers that the reduced activation of the ANS is a result of hypoactivity of specific brain regions responsible in the fight-or-flight response, including the limbic system (Caes et al., 2012; Marsh et al., 2011; Rothemund et al., 2012).

Recognizing fearful responses in others and psychopathy

Not only is psychopathy associated with deficits in experiencing fear, but research also suggests that it involves a marked impairment in recognizing fear-related stimuli (Dadds et al.,, 2008; Gillen et al., 2018; Gillespie et al., 2015; Seara-Cardoso et al., 2012). To examine this concept, researchers employed a paradigm in which participants were shown pictures of people's faces and asked to label the emotion depicted (Dadds et al. 2008; Gillen et al., 2018; Gillespie et al., 2015; Seara-Cardoso et al. 2012). Psychopathic symptomatology is typically associated with poorer performance on this task compared to that of healthy control participants, especially when the cued pictures are of fearful faces (Dadds et al. 2008; Gillen et al., 2018; Gillespie et al., 2015; Seara-Cardoso et al. 2012). Expanding upon this idea, homologous tasks have been developed that ask participants to label fearful tone of voice (Gillen et al., 2018) or fearful, static body postures (Muñoz, 2009) in voice recordings and whole-body pictures, respectively. These studies also yielded a negative correlation between psychopathic traits and the ability to recognize signs of fear in others (Gillen et al., 2018; Muñoz, 2009). In addition to this impaired recognition of fearful emotions, researchers have explored whether these deficits are translated across frightening behaviors and situations (Marsh and Cardinale, 2012). Participants were asked to rate the emotional consequences of hypothetical social behaviors (Marsh and Cardinale, 2012). Psychopathy was associated with impaired judgements of which situations and statements could generally be characterized as frightening (Marsh and Cardinale, 2012). Collectively, this line of research provides support for the claim that psychopathic individuals have a reduced capacity of recognizing fear-related stimuli and suggests a fear-processing deficit in psychopathy (Gillespie et al., 2015; Marsh and Cardinale, 2012; Muñoz, 2009). Some researchers believe this reduced capacity to recognize fear-related stimuli is a due to lack of attention to emotionally-salient cues in the environment, including facial features of other people, like the eyes (Dadds et al., 2008; Muñoz, 2009). In other words, this could be a result of the difficulty in switching attention and incorporating contextual, affective stimuli when engaging in goal-directed behavior among individuals diagnosed with psychopathy (Dadds et al., 2008; Muñoz, 2009).

PSYCHOPATHY AND LIMBIC DYSFUNCTION

Volumetric properties of the limbic system in psychopathy

With the emergence of new technologies that allow greater insights into the workings of the nervous system, some researchers have attempted to establish the specific structural brain abnormalities that characterize psychopathy (Blair, 2013). Taking into consideration the great volume of research on fear deficits in psychopathy, the limbic system has been the focus of multiple different research avenues (Moul et al., 2012). In two similar studies, researchers examined the structural properties of the limbic system in the psychopathic brain using magnetic resonance imaging (MRI) (Contrereas- Rodríguez et al., 2015; Ermer et al., 2013). Limbic structures of psychopathic and non-psychopathic participants were compared using a single blind procedure while controlling for total brain volume, age, and sex (Contrereas- Rodríguez et al., 2015; Ermer et al., 2013). In both studies, psychopathy was associated with significantly fewer nerve cell bodies in limbic structures and other closely related paralimbic regions (Contrereas- Rodríguez et al., 2015; Ermer et al., 2013). More specifically, research using similar methodology has shown that psychopathic symptomatology is negatively correlated with the volume of nerve cell bodies in the amygdala (Yang et al., 2009) and the insula (de Oliviera-Souza et al., 2008). Altogether, MRI outcomes suggest the presence of significant atypical volumetric reductions in the limbic system of psychopathic individuals (Contrereas- Rodríguez et al., 2015; Yang et al., 2009).

Morphological properties of the limbic system in psychopathy

Additionally, MRI studies have also been used to assess morphological anomalies in specific limbic structures of psychopathic individuals (Boccardi et al., 2010; Boccardi et al., 2011). To do so, a single investigator, blind to the diagnosis, produced manual traces of limbic structures that were then used to generate a three-dimensional (3D) model of the limbic system (Boccardi et al., 2010; Boccardi et al., 2011). This 3D model was then used to map tissue differences of the amygdala (Boccardi et al., 2011) and hippocampus (Boccardi et al., 2010) between psychopathic and non-psychopathic participants. The results from these studies showed alternative structural morphologies in the psychopathic participants that consisted of both enlargement and reduction effects (Boccardi et al., 2010; Boccardi et al., 2011). Psychopathy was associated with an enlargement in the central nucleus of the amygdala, which is directly connected to the brain's "fight-or-flight" center (Boccardi et al., 2011). In contrast, a reduction in the basolateral nucleus of the amygdala, a region crucial for reinforcement learning, was observed (Boccardi et al., 2011). Although when compared to the non-psychopathic controls, psychopathic participants did not differ significantly in total hippocampal volume, they did in its distribution (Boccardi et al., 2010). Researchers noted an extensive enlargement of the lateral borders in the hippocampus that was accompanied with a depression along its midline (Boccardi et al., 2010). Even though the exact altered hippocampal sub-regions are not identifiable using current technology, these results suggest marked morphological alterations on hippocampus, a structure involved in the processing of aggressive behaviors, impulsivity and threat avoidance (Boccardi et al., 2010). Hence, these research findings suggest morphological changes in psychopathy pertaining to the limbic structures, which remain crucial for the affective and ANS response to threatening or fear-related stimuli (Boccardi et al., 2011; Boccardi et al., 2010).

Connectivity of the limbic system in psychopathy

Even though emotional deficits present in psychopathy, namely reduced negative valence or ANS activation in response to fear, could be at least partially accounted for by these limbic abnormalities, the difficulties in recognizing emotional cues, especially fear, may imply the involvement of higher cortical structures (Marsh et al., 2011; Motzkin et al., 2011). The prefrontal cortex is the executive function center of the brain and is considered to orchestrate thoughts or actions to inform and guide behavior (Del Arco and Mora, 2009). This is mainly achieved due to the high degree of interconnectedness of the prefrontal cortex with other cortical, subcortical, or brain stem sites (Del Arco and Mora, 2009). Thus, the prefrontal cortex relies on a high degree of connectivity with limbic areas to control the execution of goal-directed behaviors while simultaneously processing contextual and emotional information (Del Arco and Mora, 2009). Ineffective interconnections between the prefrontal cortex and limbic structures have gathered more attention as researchers believe these interconnections can better explain some of the deficits seen in psychopathy (Marsh et al., 2011; Motzkin et al., 2011). Using functional MRI (fMRI), researchers in two similar studies were able to assess the connectivity degree of limbic structures to the prefrontal cortex (Marsh et al., 2011; Motzkin et al., 2011). The severity of psychopathic symptomatology was negatively correlated with the functional connectivity at rest between the limbic system and the prefrontal cortex in both studies (Marsh et al., 2011; Motzkin et al., 2011). More specifically, at rest, there was an observed reduced connectivity between the amygdala and the orbitofrontal cortex (Mash et al., 2011) as well as the fronto-parietal cortices (Motzkin et al., 2011). Furthermore, this reduced connectivity between the cognitive and emotional centers of the brain might reflect how psychopathic traits affect an individual's ability to use information about valence to guide their behaviors (Mash et al., 2011). Thus, further research by Mash et al., 2011 and Motzkin et al., 2011 supports the central role of the limbic system in the neurobiological profile of psychopathy and suggests that the disorder might be characterized by atypical limbic function and structure.

Functional properties of the limbic system in psychopathy

Researchers have also been interested in the function of the limbic system and have investigated if there are functional differences in the regulation of limbic structures in psychopathy (Dolan and Fullam, 2009; Ewbank et al., 2018; Jones et al., 2009). Because of the limbic system's central role in fear regulation, researchers have employed experimental procedures typical of research in fear (Dolan and Fullam, 2009; Ewbank et al., 2018; Jones et al., 2009). For example, researchers have used fMRIs to measure amygdala activation while the participants are exposed to pictures of emo-

tional faces and are asked to label the emotion depicted (Dolan and Fullam, 2009; Ewbank et al., 2018; Jones et al., 2009). High psychopathy scores were not only negatively correlated with an ability to correctly label the depicted emotion, but were also associated with diminished amygdala responses to fearful, angry, or disgusted faces (Dolan and Fullam, 2009; Ewbank et al., 2018; Jones et al., 2009). Additionally, researchers found evidence of reduced connectivity between the amygdala and the rest of thebrain while processing threatening faces (Ewbank et al., 2018). This further supports the observed claim of reduced connectivity between the limbic system and higher cortical regions, which could result in a reduced capacity for threat detection and response among psychopathic individuals (Ewbank et al., 2018).

Researchers have also measured limbic function while the participants complete the Cambridge Decision-Making Task (CDMT) (Sutherland and Fishbein, 2017). In this task, participants were asked to choose between small, likely rewards or large, unlikely rewards (Sutherland and Fishbein, 2017). Psychopaths performing this task usually select the "riskier", large, and unlikely rewards more often than sex-matched, non-psychopathic controls (Sutherland and Fishbein, 2017). Moreover, positron emission tomography (PET) scans obtained while performing this task show reduced activation of the limbic system in psychopathic participants (Sutherland and Fishbein, 2017). These results suggest that psychopathy may be associated with an inability to use real-life negative experiences and adjust behavior accordingly (Sutherland and Fishbein, 2017). Psychopathy has also been associated with reduced amygdala activity during judgments of fear-evoking statements when participants performed this task in an fMRI imaging scanner (Marsh and Cardinale, 2012). Additionally, this reduced pattern of amygdala activation was positively correlated with a higher degree of leniency when deciding which statements could be generally described as frightening (Marsh and Cardinale, 2012). Thus, neuroimaging research suggests that the fear deficits observed in psychopathy are highly correlated with reduced limbic activation in response to fearful or threatening stimuli (Ewbank et al., 2018; Marsh and Cardinale, 2012; Sutherland and Fishbein, 2017).

Research strongly suggests that psychopathy is associated with characteristic structural and functional alterations in limbic structures (Ermer et al., 2013; Ewbank et al., 2018; Marsh and Cardinale, 2012; Yang et al., 2015; Yoder et al., 2015). However, it is not yet known if those changes are present at birth or if they emerge later on in life as a result of environmental factors(Ermer et al., 2013; Ewbank et al., 2018; Marsh and Cardinale, 2012; Yang et al., 2015; Yoder et al., 2015). This is mainly because research on the neurobiological underpinnings of psychopathy is in its infancy and is only recently attempting to show the existence of brain morphological alterations in psychopathy(Ewbank et al., 2018; Yang et al., 2015; Yoder et al., 2015). In fact, multiple studies cited in this paper were the first ones published in their line of research, suggesting there are still gaps in the psychopathy literature (Ermer et al., 2013; Ewbank et al., 2018; Marsh and Cardinale, 2012; Yang et al., 2015; Yoder et al., 2015).

DISCUSSION

The purpose of this paper was to demonstrate the fear deficits (Caes et al., 2012; Gillen et al., 2018; Marsh et al., 2011) and alterations of the limbic system that are associated with psychopathy (Boccardi et al., 2010; Boccardi et al., 2011; Yang et al., 2015). Evidence gathered from research articles collectively supports psychopathy's negative relationship on the subjective experience of fear (Caes et al., 2012; Rothemund et al., 2012; Veit et al., 2013). More specifically, psychopathy has been correlated with decreased negative valence and depleted feelings of fear when they are confronted with threatening or aversive situations suggesting an affective deficit is present in the disorder (Caes et al., 2012; Rothemund et al., 2012; Veit et al., 2013). In response to fear-related stimuli, psychopathic individuals did not show increased heart rate, skin conductance, event-related potentials, or blink reflex in response to aversive or threatening cues (Hoppenbrouwers et al., 2016; Marsh et al., 2011; Vaidyanathan et al., 2011). Since the interpretation of the somatic symptoms of the ANS activation is crucial for the experience of emotion, these results suggest that the fear deficits found in psychopathy could be due to diminished activation of the ANS (Marsh et al., 2011). Multiple studies have also shown that psychopathy is associated with a marked deficit in recognizing fear-related cues (Dadds et al., 2008; Gillen et al., 2018; Gillespie et al., 2015). This includes a difficulty recognizing fearful facial expressions (Dadds et al. 2008; Gillespie et al., 2015; Seara-Cardoso et al. 2015), tone of voice (Gillen et al., 2018), body postures (Muñoz, 2009) or statements that are generally considered to be frightening (Marsh and Cardinale, 2012). Some researchers believe this is due to an attentional deficit that leaves psychopathic individual "blind" to affective or emotional stimuli (Marsh and Cardinale, 2012; Muñoz, 2009).

Research studies also demonstrated that psychopathy is associated with marked deficits in the limbic system (Contreras- Rodríguez et al., 2015; Ermer et al., 2013; Yang et al., 2009). In particular, MRI studies have demonstrated that psychopathy might be linked with a reduction in the nerve cell body volume in the limbic system and other closely-linked paralimbic structures (Contreras-Rodríguez et al., 2015; Ermer et al., 2013; Yang et al., 2009). In addition, morphological differences in the limbic structures of psychopathic individuals have also been found (Boccardi et al., 2010; Boccardi et al., 2011). This includes changes in the shape and the distribution of neuronal volume in structures like the hippocampus and amygdala of psychopathic individuals (Boccardi et al., 2010; Boccardi et al., 2011). Psychopathy has also been associated with reduced connectivity between the limbic system and the prefrontal cortex, suggesting that the disorder might be marked by not only atypical limbic structure, but also function (Marsh et al., 2011; Motzkin et al., 2011). Researchers have used fMRI and PET scans to investigate limbic function in psychopathic individuals and found diminished amygdala activity in response to fear-related stimuli (Dolan and Fullam, 2009; Ewbank et al., 2018; Jones et al.,

2009; Sutherland and Fishbein, 2017). While the association between psychopathy and these brain deficits is yet to be explained, researchers are hopeful that recent technological will continue to reveal more about the neurobiological underpinnings of the disorder (Ewbank et al., 2018; Yang et al., 2015).

While these promising results support the idea of fear deficits and dysfunction of the limbic system in adults with psychopathy, they are not without limitations. Primarily, multiple studies discussed in this review employed only incarcerated offenders as participants, which raises questions about their generalizability (Boccardi et al., 2010; Boccardi et al., 2011; Ewbank et al., 2018; Yang et al., 2009). This is partially because the incarcerated sample population used included only male participants (Boccardi et al., 2010; Boccardi et al., 2011; Ewbank et al., 2018; Yang et al., 2009). In addition, incarcerated populations are more likely to have experienced a traumatic childhood event (Driesen et al., 2006) and have a history of substance dependence or another serious mental illness (Lynch et al., 2014). Concerns are also raised over the potential incidences of traumatic brain injury (TBI) that were not controlled for in any of the cited studies (Boccardi et al., 2010; Boccardi et al., 2011; Ewbank et al., 2018; Yang et al., 2009). traumatic brain injury (TBI), refers to an injury to the head that can result in significant neuropsychological abnormalities, including alterations in brain structures like the limbic system (Shiroma et al., 2010). It is particularly troubling to not control for TBI given that its prevalence in incarcerated populations is thirty times higher compared to the national average (Shiroma et al., 2010), subsequently raising concerns over the internal validity of the aforementioned studies. Future research should implement a more rigorous screening for TBI or head trauma history in this line of research. Furthermore, current studies could also be recreated in non-clinical populations to test the idea that psychopathy exists on a spectrum. If this hypothesis is validated, then brain alterations should also be observed in non-clinical samples, albeit to a lesser degree. Alternatively, if psychopathy is a categorical disorder, then brain changes should only observed after a threshold of symptoms is reached.

A second limitation includes flaws in the employed methodology. More specifically the experience of fear was often assessed after the experience of the fearful situation (Caes et al., 2012; Mash et al., 2011). Thus, it is not certain if the fear experience changed over time or the reported decreased negative valence and ANS activation are a result of a memory rather than an affective or somatic deficit (Caes et al., 2012; Mash et al., 2011). Furthermore, despite the fact that psychopathy has been associated with attentional deficits, only two studies controlled for possible influences of attention (Muñoz, 2009; Gillen et al., 2018). Thus, perhaps, psychopathic participants showed diminished emotional responses to fearful situations or had difficulties recognizing fearful emotions depicted in pictures was because they paid less attention to the task at hand (Muñoz, 2009; Gillen et al., 2018). Finally, the studies investigating the ability of psychopathic participants to recognize emotions from facial expressions used pictures of strangers (Caes et al., 2012; Mash et al., 2011). However, research has shown that often times, people pay more attention and are better able to recognize facial emotions of people they know (Caes et al., 2012). Thus, researchers could test if this also applies for psychopathic individuals by recreating this experimental design using pictures of people familiar with the participants.

Thirdly, the research findings presented in this paper focused on adult populations (Boccardi et al., 2010; Boccardi et al., 2011; Ewbank et al., 2018). However, psychopathy is often conceptualized as a personality disorder that emerges earlier in life, with psychopathic traits remaining relatively stable from childhood into adulthood (Serin et al., 2011). Researchers are not sure if the psychopathic tendencies precede, follow, or are concurrent with the brain alterations observed in adults, partially because very little imaging research has been done employing younger psychopathic participants (Boccardi et al., 2010; Boccardi et al., 2011; Ewbank et al., 2018). A longitudinal study on children who present psychopathic tendencies could clarify the time point when the brain abnormalities associated with psychopathy become apparent. In addition, through a longitudinal study, researchers could identify potential environmental factors, such as attachment styles, parental styles they experienced, or exposure to drugs and alcohol, that contribute to the development of psychopathic traits.

This literary review provides support that psychopathy is associated with fear deficits and dysregulation of the limbic system. Some researchers believe that such fear deficits are responsible for the high rate of antisocial and criminal behavior observed in psychopathic individuals. More research is needed to establish the neurobiological underpinnings of psychopathy and association with the diminished experience of fear seen in the disorder.

REFERENCES

Alkire, M. T., Vazdarjanova, A., Dickinson-Anson, H., White, N. S., and Cahill, L. (2001). Lesions of the basolateral amygdala complex block propofol-induced amnesia for inhibitory avoidance learning in rats. *Anesthesiology: The Journal of the American Society of Anesthesiologists*, 95(3), 708-71.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.).

Angrilli, A., Mauri, A., Palomba, D., Flor, H., Birbaumer, N., Sartori, G., and Paola, F. D. (1996). Startle reflex and emotion modulation impairment after a right amygdala lesion. *Brain*, 119(6), 1991-2004.

Babiak, P. (2000). Psychopathic manipulation at work. *The clinical and forensic assessment of psychopathy: A practitioner's guide*. Mahwah, NJ: Lawrence Earlbaum Associates.

Babiak, P., Neumann, C. S., and Hare, R. D. (2010). Corporate psychopathy: Talking the walk. *Behavioral Sciences and the Law*, 28(2), 174-193.

Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., and Flor, H. (2005). Deficient fear conditioning in psychopathy: a functional magnetic resonance imaging study. *Archives of General Psychiatry*, 62(7), 799-805.

Blair, R. J. R. (2013). Psychopathy: cognitive and neural dysfunction. *Dialogues in Clinical Neuroscience*, 15(2), 181.

Blair, R. J. R. and Mitchell, D. G. V. (2009). Psychopathy, attention and emotion. *Psychological Medicine*, 39(4), 543-555.

Boccardi, M., Ganzola, R., Rossi, R., Sabattoli, F., Laakso, M. P., Repo-Tiihonen, E., and Frisoni, G. B. (2010). Abnormal hippocampal shape in offenders with psychopathy. *Human Brain Mapping*, 31(3), 438-447.

Boccardi, M., Frisoni, G. B., Hare, R. D., Cavedo, E., Najt, P., Pievani, M., and Vaurio, O. (2011). Cortex and amygdala morphology in psychopathy. *Psychiatry Research: Neuroimaging*, 193(2), 85-92.

Boddy, C. R. (2014). Corporate psychopaths, conflict, employee affective well-being and counterproductive work behaviour. *Journal of Business Ethics*, 121(1), 107-121.

Bradley, M. M., Silakowski, T., and Lang, P. J. (2008). Fear of pain and defensive activation. *PAIN*, 137(1), 156-163.

Brand, M., Grabenhorst, F., Starcke, K., Vandekerckhove, M. M., and Markowitsch, H. J. (2007). Role of the amygdala in decisions under ambiguity and decisions under risk: evidence from patients with Urbach-Wiethe disease. *Neuropsychologia*, 45(6), 1305-1317.

Brown, S. L. and Forth, A. E. (1997). Psychopathy and sexual assault: Static risk factors, emotional precursors, and rapist subtypes. *Journal of Consulting and Clinical Psychology,* 65(5), 848.

Budhani, S. and Blair, R. J. R. (2005). Response reversal and children with psychopathic tendencies: success is a function of salience of contingency change. *Journal of Child Psychology and Psychiatry*, 46(9), 972-981.

Budhani, S., Richell, R. A., and Blair, R. J. R. (2006). Impaired reversal but intact acquisition: Probabilistic response reversal deficits in adult individuals with psychopathy. *Journal of Abnormal Psychology*, 115(3), 552.

Caes, L., Uzieblo, K., Crombez, G., De Ruddere, L., Vervoort, T., and Goubert, L. (2012). Negative emotional responses elicited by the anticipation of pain in others: psychophysiological evidence. *The Journal of Pain*, 13(5), 467-476.

Coid, J., Yang, M., Ullrich, S., Roberts, A., and Hare, R. D. (2009). Prevalence and correlates of psychopathic traits in the household population of Great Britain. *International Journal of Law and Psychiatry*, 32(2), 65-73.

Contreras-Rodríguez, O., Pujol, J., Batalla, I., Harrison, B. J., Soriano-Mas, C., Deus, J. and Pifarré, J. (2015). Functional connectivity bias in the prefrontal cortex of psychopaths. *Biological Psychiatry*, 78(9), 647-655.

Crooks, D., Anderson, N. E., Widdows, M., Petseva, N., Koenigs, M., Pluto, C., and Kiehl, K. A. (2018). The relationship between cavum septum pellucidum and psychopathic traits in a large forensic sample. *Neuropsychologia*, 112, 95-104.

Cummings, M. A. (2015). The neurobiology of psychopathy: recent developments and new directions in research and treatment. *CNS Spectrums*, 20(3), 200-206.

Dadds, M. R., El Masry, Y., Wimalaweera, S., and Guastella, A. J. (2008). Reduced eye gaze explains "fear blindness" in childhood psychopathic traits. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(4), 455-463.

Del Arco, A. and Mora, F. (2009). Neurotransmitters and prefrontal cortex–limbic system interactions: implications for plasticity and psychiatric disorders. *Journal of Neural Transmission*, 116(8), 941-952.

de Oliveira-Souza, R., Hare, R. D., Bramati, I. E., Garrido, G. J., Azevedo Ignácio, F., Tovar-Moll, F. (2008). Psychopathy as a disorder of the moral brain: fronto-temporo-limbic grey matter reductions demonstrated by voxel-based morphometry. *Neuroimage*, 40, 1202–1213.

Dolan, M. C. and Fullam, R. S. (2009). Psychopathy and functional magnetic resonance imaging blood oxygenation level-dependent responses to emotional faces in violent patients with schizophrenia. *Biological Psychiatry*, 66(6), 570-577.

Driessen, M., Schroeder, T., Widmann, B., von Schonfeld, C., and Schneider, F. (2006). Childhood trauma, psychiatric disorders, and criminal behavior in prisoners in Germany: a comparative study in incarcerated women and men. *Journal of Clinical Psychiatry*, 67(10), 1486-1492.

Duvarci, S., Bauer, E. P., and Paré, D. (2009). The bed nucleus of the stria terminalis mediates inter-individual variations in anxiety and fear. *Journal of Neuroscience*, 29(33), 10357-10361.

Edens, J. F., Marcus, D. K., Lilienfeld, S. O., and Poythress Jr, N. G. (2006). Psychopathic, not psychopath: taxometric evidence for the dimensional structure of psychopathy. *Journal of Abnormal Psychology*, 115(1), 131.

Ermer, E., Cope, L. M., Nyalakanti, P. K., Calhoun, V. D., and Kiehl, K. A. (2013). Aberrant paralimbic gray matter in incarcerated male adolescents with psychopathic traits. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(1), 94-103.

Ewbank, M. P., Passamonti, L., Hagan, C. C., Goodyer, I. M., Calder, A. J., and Fairchild, G. (2018). Psychopathic traits influence amygdala-anterior cingulate cortex connectivity during facial emotion processing. *Social Cognitive and Affective Neuroscience*, 1, 10.

Finger, E. C., Marsh, A. A., Blair, K. S., Reid, M. E., Sims, C., Ng, P., Pine, D. S., and Blair, R. J. R. (2011). Disrupted reinforcement signaling in the orbitofrontal cortex and caudate in youths with conduct disorder or oppositional defiant disorder and a high level of psychopathic traits. *American Journal of Psychiatry*, 168(2), 152-162.

Fishbein, D. H. and Sutherland, M. T. (2017). Higher Trait Psychopathy is associated with increased risky decision-making and less coincident insula and striatal activity. *Frontiers in Behavioral Neuroscience*, 11, 245.

Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology*, 17, 87–104.

Gillen, C. T., Lee, Z., Salekin, K. L., Iselin, A. M. R., Harrison, N. A., Clark, A. P., and Salekin, R. T. (2018). Psychopathic traits in adolescence: the importance of examining components in face processing, voice processing, and emotional skill. *Journal of Psychopathology and Behavioral Assessment*, 40(1), 50-59.

Gillespie, S. M., Mitchell, I. J., Satherley, R. M., Beech, A. R., and Rotshtein, P. (2015). Relations of distinct psychopathic personality traits with anxiety and fear: Findings from offenders and non-offenders. *PloS One*, 10(11), e0143120.

Glenn, A. L., Raine, A., Yaralian, P. S., and Yang, Y. (2010). Increased volume of the striatum in psychopathic individuals. *Biological Psychiatry,* 67(1), 52–58.

Hare, R.D. (1993). *Without conscience: The disturbing world of the psychopaths amongst us*. New York, NY: Pocket Books.

Hare, R. D., Clark, D., Grann, M., and Thornton, D. (2000). Psychopathy and the predictive validity of the PCL-R: An international perspective. *Behavioral Sciences and the Law*, 18(5), 623-645.

Hare, R. D., Hart, S. D., and Harpur, T. J. (1991). Psychopathy and the DSM-IV criteria for antisocial personality disorder. *Journal of Abnormal Psychology*, 100(3), 391.

Hare, R. D., Harpur, T. J., Hakstian, A. R., Forth, A. E., Hart, S. D., and Newman, J. P. (1990). The revised Psychopathy Checklist: Reliability and factor structure. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 2(3), 338.

Hare, R. D. and Neumann, C. S. (2008). Psychopathy as a clinical and empirical construct. *Annual Revisions Clinical Psychology,* 4, 217-246.

Hoppenbrouwers, S. S., Bulten, B. H., and Brazil, I. A. (2016). Parsing fear: A reassessment of the evidence for fear deficits in psychopathy. *Psychological Bulletin*, 142(6), 573.

Hosking, J. G., Kastman, E. K., Dorfman, H. M., Samanez-Larkin, G. R., Baskin-Sommers, A., Kiehl, K. A., Newman, J. P., Buckholtz, J. W. (2017). Disrupted prefrontal regulation of striatal subjective value signals in psychopathy. *Neuron*, 95(1), 221.e4–231.e4.

Iria, C. and Barbosa, F. (2009). Perception of facial expressions of fear: Comparative research with criminal and non-criminal psychopaths. *Journal of Forensic Psychiatry and Psychology*, 20, 66 –73.

Jones, A. P., Laurens, K. R., Herba, C. M., Barker, G. J., and Viding, E. (2009). Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. *American Journal of Psychiatry*, 166(1), 95-102.

Kiehl, K. A., Hare, R. D., Liddle, P. F., and McDonald, J. J. (1999). Reduced P300 responses in criminal psychopaths during a visual oddball task. *Biological Psychiatry*, 45(11), 1498-1507.

Kiehl, K. A., Hare, R. D., McDonald, J. J., and Brink, J. (1999). Semantic and affective processing in psychopaths: An event-related potential (ERP) study. *Psychophysiology*, 36(6), 765-774.

Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., and Liddle, P. F. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Biological Psychiatry*, 50(9), 677-684.

Kilpatrick, L. and Cahill, L. (2003). Amygdala modulation of parahippocampal and frontal regions during emotionally influenced memory storage. *Neuroimage*, 20(4), 2091-2099.

Knight, R. A. and Guay, J.-P. (2006). The role of psychopathy in sexual offenders against women. In C. J. Patrick (Ed.), *Handbook of Psychopathy*. New York: The Guilford Press.

Krumpal, I. (2013). Determinants of social desirability bias in sensitive surveys: a literature review. *Quality and Quantity*, 47(4), 2025-2047.

LeDoux, J. (2003). The emotional brain, fear, and the amygdala. *Cellular and Molecular Neurobiology*, 23(4-5), 727-738

López, R., Poy, R., Patrick, C. J., and Moltó, J. (2013). Deficient fear conditioning and self-reported psychopathy: The role of fearless dominance. *Psychophysiology*, 50(2), 210-218.

Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *Journal of Abnormal and Social Psychology*, 55, 6 –10.

Lynch, S. M., DeHart, D. D., Belknap, J. E., Green, B. L., Dass-Brailsford, P., Johnson, K. A., and Whalley, E. (2014). A multisite study of the prevalence of serious mental illness, PTSD, and substance use disorders of women in jail. *Psychiatric Services*, 65(5), 670-674.

Malloy, P., Bihrle, A., Duffy, J., and Cimino, C. (1993). The orbitomedial frontal syndrome. *Archives of Clinical Neuropsychology*, 8(3), 185-201.

Marsh, A. A. and Cardinale, E. M. (2012). When psychopathy impairs moral judgments: neural responses during judgments about causing fear. *Social Cognitive and Affective Neuroscience*, 9(1), 3-11.

Marsh, A. A., Finger, E. C., Fowler, K. A., Jurkowitz, I. T., Schechter, J. C., Henry, H. Y., Pine, D.S., and Blair, R. J. R. (2011). Reduced amygdala–orbitofrontal connectivity during moral judgments in youths with disruptive behavior disorders and psychopathic traits. *Psychiatry Research: Neuroimaging*, 194(3), 279-286.

Marsh, A. A., Finger, E. C., Schechter, J. C., Jurkowitz, I. T., Reid, M. E., and Blair, R. J. R. (2011). Adolescents with psychopathic traits report reductions in physiological responses to fear. *Journal of Child Psychology and Psychiatry*, 52(8), 834-841.

Motzkin, J. C., Newman, J. P., Kiehl, K. A., and Koenigs, M. (2011). Reduced prefrontal connectivity in psychopathy. *Journal of Neuroscience*, 31(48), 17348-17357.

Moul, C., Killcross, S., and Dadds, M. R. (2012). A model of differential amygdala activation in psychopathy. *Psychological Review*, 119(4), 789.

Muñoz, L. C. (2009). Callous-unemotional traits are related to combined deficits in recognizing afraid faces and body poses. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(5), 554-562.

Nesse, R. M. (1994). Fear and fitness: An evolutionary analysis of anxiety disorders. *Ethology and Sociobiology*, 15(5-6), 247-261.

Newman, J. P., Patterson, C. M., and Kosson, D. S. (1987). Response perseveration in psychopaths. *Journal of Abnormal Psychology*, 96(2), 145.

Newman, J. P., Curtin, J. J., Bertsch, J. D., and Baskin-Sommers, A. R. (2010). Attention moderates the fearlessness of psychopathic offenders. *Biological Psychiatry*, 67(1), 66-70.

Newman, J. P., Patterson, C. M., Howland, E. W., and Nichols, S. L. (1990). Passive avoidance in psychopaths: The effects of reward. *Personality and Individual Differences*, 11(11), 1101-1114.

Porter, S., Woodworth, M., Earle, J., Drugge, J., and Boer, D. (2003). Characteristics of sexual homicides committed by psychopathic and nonpsychopathic offenders. *Law and Human Behavior*, 27(5), 459.

Richardson, M. P., Strange, B. A., and Dolan, R. J. (2004). Encoding of emotional memories depends on amygdala and hippocampus and their interactions. *Nature Neuroscience*, 7(3), 278.

Rothemund, Y., Ziegler, S., Hermann, C., Gruesser, S. M., Foell, J., Patrick, C. J., and Flor, H. (2012). Fear conditioning in psychopaths: event-related potentials and peripheral measures. *Biological Psychology*, 90(1), 50-59.

Schafe, G. E., Atkins, C. M., Swank, M. W., Bauer, E. P., Sweatt, J. D., and LeDoux, J. E. (2000). Activation of ERK/MAP kinase in the amygdala is required for memory consolidation of pavlovian fear conditioning. *Journal of Neuroscience*, 20(21), 8177-8187.

Schiffer, B., Müller, B. W., Scherbaum, N., Hodgins, S., Forsting, M., Wiltfang, J., Gizewski, E. R., and Leygraf, N. (2011). Disentangling structural brain alterations associated with violent behavior from those associated with substance use disorders. *Archives of General Psychiatry,* 68(10), 1039–1049.

Schultz, D. H., Balderston, N. L., Baskin-Sommers, A. R., Larson, C. L., and Helmstetter, F. J. (2017). Corrigendum: psychopaths show enhanced amygdala activation during fear conditioning. *Frontiers in Psychology*, 8, 1457.

Seara-Cardoso, A., Neumann, C., Roiser, J., McCrory, E., and Viding, E. (2012). Investigating associations between empathy, morality and psychopathic personality traits in the general population. *Personality and Individual Differences*, 52(1), 67-71.

Serin, R., Forth, A., Brown, S., Nunes, K., Bennell, C., and Pozzulo, J. (2011). Psychology of criminal behaviour: A Canadian perspective, Second Canadian Edition. Pearson Toronto.

Sutherland, M. T. and Fishbein, D.H. (2017). Higher Trait Psychopathy is associated with increased risky decision-making and less coincident insula and striatal activity. *Frontiers in Behavioral Neuroscience*, 11, 245.

Sylvers, P. D., Brennan, P. A., and Lilienfeld, S. O. (2011). Psychopathic traits and preattentive threat processing in children: a novel test of the fearlessness hypothesis. *Psychological Science*, 22(10), 1280-1287.

Vaidyanathan, U., Hall, J. R., Patrick, C. J., and Bernat, E. M. (2011). Clarifying the role of defensive reactivity deficits in psychopathy and antisocial personality using startle reflex methodology. *Journal of Abnormal Psychology*, 120(1), 253.

Veit, R., Konicar, L., Klinzing, J. G., Barth, B., Yilmaz, Ö., and Birbaumer, N. (2013). Deficient fear conditioning in psychopathy as a function of interpersonal and affective disturbances. *Frontiers in Human Neuroscience*, 7, 706.

Walker, D. L. and Davis, M. (1997). Double dissociation between the involvement of the bed nucleus of the stria terminalis and the central nucleus of the amygdala in startle increases produced by conditioned versus unconditioned fear. *Journal of Neuroscience*, 17(23), 9375-9383.

Weiten, W. and McCann, D. (2016). "Psychology: themes and variations", Fourth Canadian edition. Toronto: Nelson Education.

White, S. F., Marsh, A. A., Fowler, K. A., Schechter, J. C., Adalio, C., Pope, K., Sinclair, S. Pine, D. S., and Blair, R. J. R. (2012). Reduced amygdala response in youths with disruptive behavior disorders and psychopathic traits: decreased emotional response versus increased top-down attention to nonemotional features. *American Journal of Psychiatry*, 169(7), 750-758.

Wynn, R., Høiseth, M. H., and Pettersen, G. (2012). Psychopathy in women: theoretical and clinical perspectives. *International Journal of Women's Health*, 4, 257.

Yang, Y., Raine, A., Narr, K. L., Colletti, P., and Toga, A. W. (2009). Localization of deformations within the amygdala in individuals with psychopathy. *Archives of General Psychiatry*, 66(9), 986-994.