Psychological Bulletin
2016, Vol. 142, No. 6, 573–600

0033-2909/16/$12.00 http://dx.doi.org/10.1037/bul0000040

Parsing Fear: A Reassessment of the Evidence for Fear Deficits in Psychopathy

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Psychopathy is a personality disorder characterized by interpersonal manipulation and callousness, and reckless and impulsive antisocial behavior. It is often seen as a disorder in which profound emotional disturbances lead to antisocial behavior. A lack of fear in particular has been proposed as an etiologically salient factor. In this review, we employ a conceptual model in which fear is parsed into separate subcomponents. Important historical conceptualizations of psychopathy, the neuroscientific and empirical evidence for fear deficits in psychopathy are compared against this model. The empirical evidence is also subjected to a meta-analysis. We conclude that most studies have used the term "fear" generically, amassing different methods and levels of measurement under the umbrella term "fear." Unlike earlier claims that psychopathy is related to general fearlessness, we show there is evidence that psychopathic individuals have deficits in threat detection and responsivity, but that the evidence for reduced subjective experience of fear in psychopathy is far less compelling.

Keywords: fear, threat, psychopathy, conscious experience, emotion

Psychopathy is a severe personality disorder that entails affective dullness, a deceitful and entitled interpersonal style, and often impulsive, irresponsible, and reckless behavior. The interpersonal and affective components are believed to lie at the core of psychopathy, whereas the antisocial features are shared with other disorders. Cleckley (1976) metaphorized the psychopathic individual to resemble the monkey that, given infinity, will write Shakespeare's works on a typewriter: Whereas Shakespeare's verses are the result of a "prodigiously high level of human values" (p. 383), the psychopath uses his rational powers to mimic a sane and sociable human being, and the end product is a cold and mechanical copy of what he observes in others. Admittedly, this may be the most extreme conceptualization of the lack of emotionality of psychopathic individuals in Cleckley's work. Although Cleckley did ascribe the experience of fleeting and superficial emotions to psychopathic individuals, the role of emotions in psychopathy, and particularly fear, remains a subject of debate. Specifically, a lack of fear has been proposed to be one of the driving forces behind the callous and antisocial behavior of psychopathic individuals. However, postulating that a lack of fear is one of the most important features of psychopathy involves a number of premises. First, such a statement presupposes that it is known exactly what psychopathy is. However, there is currently a debate about how psychopathy should be conceptualized. Many researchers have employed a categorical approach in which psychopathy is differentiated from generic antisociality in forensic populations, whereas others have employed a dimensional approach in which psychopathic features are measured along a continuum in both forensic and general populations (for a detailed review, see Koenigs, Baskin-Sommers, Zeier, & Newman, 2011). More recently, another framework has been formulated in which psychopathy is described in terms of three interacting phenotypic dimensions (Patrick, Fowles, & Krueger, 2009). From a neurocognitive perspective, all of these approaches have led to the discovery of important links between psychopathy, emotion, and cognition. This further supports their validity but also makes it difficult to assign superiority to one framework (Brazil, 2015). Importantly, contemporary views on psychopathy are the product of a concept that was first used roughly 200 years ago and which, throughout this time, has been used to describe different types of patients that do, however, share characteristics. Second, such a statement also presupposes that fear is a unitary construct or has a clear definition. This idea has recently been challenged. One of the main problems of studies on psychopathy and fear is the use of different operationalizations of

This article was published Online First February 8, 2016.

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We are thankful to Drs. Roy Kessels, Roald Maes, Dennis Schutter, and Stefan van der Stigchel and the reviewers for their valuable comments and suggestions.

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fear and different methods of measuring it, such as questionnaires and autonomic measures. As a consequence, it remains unclear whether psychopathic individuals have problems in the subjective experience of fear or whether their "fearlessness" can be mostly attributed to disturbed automatic bodily responsivity to threat. In sum, claims such as "psychopathic individuals are fearless" require scrutinization of the involved concepts. This article presents a conceptual analysis and meta-analysis of the evidence for fear deficits in psychopathy.

Goals

In this review, the evidence for fear deficits in psychopathy will be assessed from multiple perspectives. First, we will highlight the role of fear in the conceptualization of psychopathy from a historical perspective. With this step, we intend to elucidate whether theorists have consequently emphasized fear as a key feature of psychopathy. This is a crucial step, as the definition of the construct has major consequences for the empirical evidence for fear deficits in psychopathy. In other words, how psychopathy is defined may determine whether fear deficits will be observed in individuals with psychopathic characteristics. As such, although fear is currently held to be central to psychopathy, this may not have been the case throughout history, and fear deficits could be a product of the currently prevailing conceptualizations. Second, we will provide a targeted review in which we consider fear from a neurobiological perspective. Neurobiological findings on fear in psychopathy will be discussed in light of a model of fear in which automatic processing of threat and the conscious experience of fear are separated. Finally, we will test the validity of this model by conducting two meta-analyses. With the first meta-analysis, we aim to test whether the experience of fear or the automatic response to emotive stimuli is compromised in adult psychopathy. A secondary meta-analysis was conducted to assess whether fear experience differs from the experience of other emotions in adult samples.

The Definition of Fear

An exact definition of fear is of paramount importance. In this light, two issues deserve attention: (a) the difference between fear and anxiety, and (b) the difference between threat reactivity and conscious fear. The difference between fear and anxiety has long been debated and may be one of the most important distinctions in the study of fear and psychopathy. Consistent with a growing body of research, there is evidence that fear and anxiety are different emotions. Although a recent meta-analysis showed a moderate relationship between trait fear and trait anxiety, certain conceptual features differentiate between fear and anxiety (Sylvers, Lilienfeld, & LaPrairie, 2011). An important defining feature of fear is that it is a surge of physiological activity in response to clear and impending danger (Grillon, 2008). For instance, Sylvers and colleagues (2011) note that "avoidance behaviours across several situations characterize trait fear, whereas sustained vigilance and prolonged hyperarousal while approaching several situations characterized trait anxiety" (p. 133). In line with this, Grillon (2008) notes that unpredictability evokes anxiety: Potential or unseen threats induce behavior that can be characterized as risk assessment. On the other hand, fear arises during specific, aversive, short-lived arousal in which the individual is motivated to escape a specific and awaiting threat (e.g., with a fight, flight, or freeze response). In contrast, trait anxiety is a more diffuse emotion in which the individual approaches an uncertain threat and the arousal is sustained, even after the removal of the threat. Anxiety can also arise in situations in which there is no concrete event in the environment leading to an emotional reaction. The distinction between fear and anxiety has also been made on the neurobiological level. For instance, there is evidence that the bed nucleus of the stria terminalis is heavily involved in the expression of anxiety-driven autonomic responses via the lateral central amygdala, but that these areas are not involved in fear expression (Robinson, Overstreet, Allen, Pine, & Grillon, 2012). Robinson et al. (2012) also found that acute tryptophan depletion affected anxiety-based responding but had no effect on fear. In addition, anxiety can likely be treated with benzodiazepines, whereas a physiological response to clear and imminent danger is less sensitive to benzodiazepines (Blanchard, Yudko, Rodgers, & Blanchard, 1993). Although there is still no consensus about the exact definitions of "fear" and "anxiety" and how the differences between these emotions are embedded in neurobiology, the evidence supports the idea that fear and anxiety are different emotions and are mediated by different neural systems (for a review, see Grillon, 2008). Thus, fear and anxiety are related, but dissociable, emotions at the conceptual, psychological, and neurobiological levels, and this distinction allows the focus on the unique role of fear in psychopathy.

Second, the importance of disentangling built-in bodily reactions to imminent threat (i.e., threat responses) and the conscious psychological experience of fear has recently been advocated (LeDoux, 2013, 2014). More specifically, the argument is that the term *fear* has been used inappropriately to describe automatic processing and responding to threat rather than the psychological phenomenon of fear. With regard to conscious experience of fear, LeDoux (2013, 2014) has recently argued that the system that detects and responds to threat is different from the system that gives rise to the conscious experience of fear. An automatic response to threat does not necessarily indicate whether an organism experiences fear. In addition, defensive reactions to threat recruit different brain areas than fearful feelings (Phelps, 2006). In addition, although the amygdala is highly involved in automatic responses to threatening stimuli, a recent meta-analytic review showed that that there is little evidence that the amygdala is activated during the conscious experience of fear (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012). In people with visual-cortex-based blindness, visual threat cues induce amygdala and sympathetic activity without any conscious experience of fear (LeDoux, 2014). There is thus evidence that the activation of automatic mechanisms triggered by threat does not necessarily lead to fear experiences. Although the conscious experience of fear is likely to be (indirectly) influenced by those brain mechanisms that are involved in the rapid detection of, and response to, threat, there is a difference between threat detection and defensive reactions, on the one hand, and the conscious experience of fear, on the other.

In order to determine whether there is truly reduced fear experience in psychopathy or whether current findings reflect impaired reactivity to threat, a distinction has to be made between (a) automatic threat responding, and (b) conscious experience of fear (see Figure 1). The first aspect, that is, *threat responsivity*, denotes

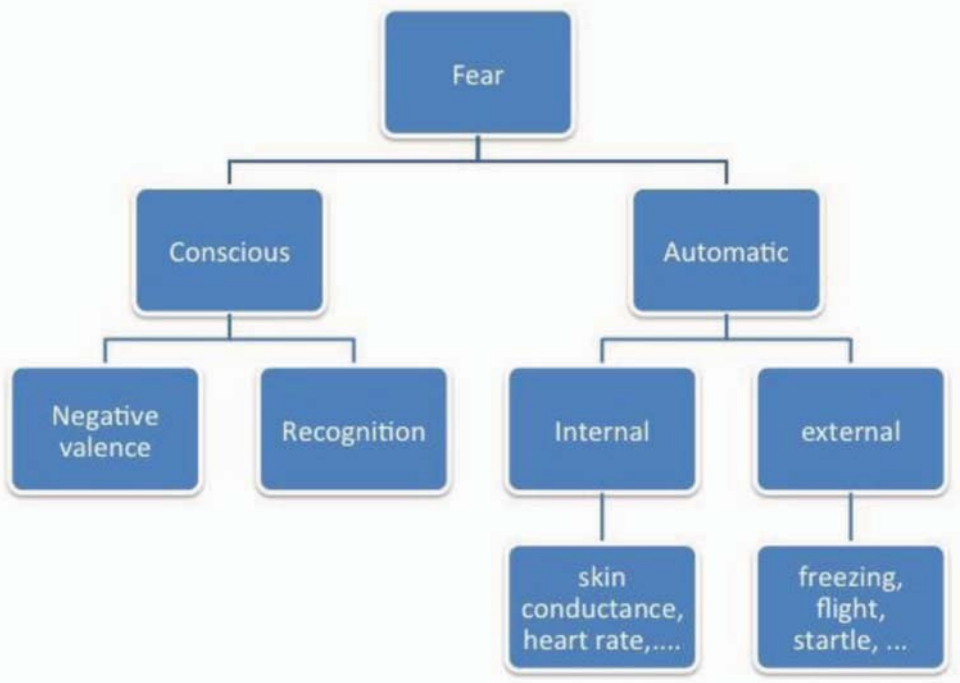

Figure 1. A schematic depiction of the model including the different elements of fear. See the online article for the color version of this figure.

the activation of a neurobiological mechanism that prepares an organism to react appropriately to imminent threat in order to survive (LeDoux, 2013, 2014). It is important to realize that these behaviors can be measured as visceral reactions or as externally observable automatic behavioral adjustments. We define *visceral reactions* as measurable changes in the organism's internal state that are not observed externally, such as changes in heart rate, respiration, skin conductance, and body temperature. In contrast, *automatic threat responses* include externally observable automatic behaviors in response to a threatening stimulus, such as freezing, avoidance behavior (i.e., flight), the startle reflex, and facial mimicry reactions (e.g., Moody, McIntosh, Mann, & Weisser, 2007). Importantly, in contrast to the conscious experience of fear, both internal and external responses to threat occur automatically and do not require the organism to experience fear (LeDoux, 2013, 2014).

The conscious experience of fear entails at least two important features, namely, negative valence and identification of emotion. The *valence* of an emotion refers to a longstanding philosophical debate on qualia, that is, the subjective evaluation of experiencing an emotion. Empirical research on this topic is particularly difficult and can only be tackled using questionnaires. The negative valence of fear denotes the unenjoyable state of experiencing it. Another aspect of the conscious experience of fear is the ability to correctly identify when one is experiencing fear. For instance, individuals with alexithymia are limited in their capacity to describe emotions when they experience these emotions and are unable to correctly identify them in others (Taylor, 2000). In addition, the capability of separating feelings from physiological arousal is also reduced. Taken together, these distinctions lead to the model described in Figure 1. Importantly, we do not claim that the levels of the model operate independently. For instance, the emergence of the fear as an affective state could be reliant on input from the visceral domain to some extent, but whether the cognitive computations involved occur serially or in parallel still has to be established (see also Balleine & Killcross, 2006).

Fear in the Genealogy of Psychopathy

At the present time, a good portion of research on psychopathy is conducted in nonclinical samples, based on the notion that psychopathic traits vary along a continuum and are also present in the general population, but as less extreme expressions of psychopathy compared with the clinical population (Seara-Cardoso & Viding, 2014). However, throughout most of history, psychopathy has been regarded as a clinical condition typified by an unstable and antagonistic lifestyle, but there has also been a strong emphasis on the affective problems associated with the disorder. In order to better understand the origins of the importance of fear for psychopathy, we need to examine the role that fear has played in clinical theories of psychopathic personality throughout history. In this section, we will examine the development of psychopathy as a clinical and personality construct (as opposed to one described in terms of cognition and/or neurobiology; see Anderson & Kiehl, 2012; Koenigs et al., 2011) in order to establish if and when fear became a prominent feature of this condition. A complete historical timeline of clinical theories of psychopathy is given in Table 1, along with descriptive quotes or statements for each hypothesis that capture the essence of the theory.

To be clear, scrutinizing various definitions of the construct of psychopathy throughout history is important, as (a) the evolution of the construct determines the key topics that are subjected to

Table 1
Historical Timeline of the Clinical Conceptualizations of Psychopathy, Comparted by the Inclusion of the Concept of Fear in the Theories

Author	Source	Abstract
Pinel	(Pinel, 1806)	*Manie sans delire* [Insanity without delusion]; I was surprised to find many maniacs who at no period gave evidence of any lesion of the understanding, but who were under the dominion of instinctive and abstract fury, as if the faculties of affect alone had sustained injury. Werlinder (1978) states that in the examples given by Pinel, "violent uncontrolled emotion" is the common denominator.
Rush	(Rush, 1812)	Cognitively intact; antisocial behavior is explained by moral derangement: "In all these cases of innate, preternatural moral depravity, there is probably an original defective organization in those parts of the body, which are occupied by the moral faculties of the mind" (p. 360).
Prichard	(Prichard, 1835)	*Moral insanity*; these individuals are cognitively intact but affect is disordered (Hervé & Yuille, 2007, p. 33). It is important to note that "moral" does not denote "ethical." With "moral insanity," Prichard meant to say that the deficits lie in the emotional and conative side of the psyche, not in the intellectual.
Campagne	(Campagne, 1869)	Basis is an egoistic character. As children, they are disobedient and cannot be influenced by parents or teachers. No respect for authority. They lack friends (unless they can profit from them) and are unable to experience love in any other form than the directly sensual. Provoke others constantly. Cognitively intact, although they often lack true originality in their thoughts. They cannot endure the same job for long periods.
Maudseley	(Maudseley, 1874)	Cognitively intact (no illusions, delusions, or hallucinations); symptoms are mainly exhibited in the active and moral powers, namely, feelings, affections, propensities, temper, habits, and conduct. Affective life is profoundly deranged. All impulses and desires are egoistic and there is no desire to resist them.
Koch	(Koch, 1891)	*Psychopathische Minderwertigkeiten* [Psychopathic inferiorities]; first to see psychopathy as a personality disorder but also very overinclusive.
Birnbaum	(Birnbaum, 1909)	Importance for the concept of psychopathy: (a) established the term *Psychopathische Persönlichkeiten* [Psychopathic personalities], (b) argued that individuals with psychopathy were born with an abnormal personality, and (c) concentrated on the tendency of the psychopathic individual to engage in criminal behavior.
Kraepelin	(Kraepelin, 1915)	Several subtypes: the *born criminals* (morally blind individual, lacks social feelings or remorse), the *unstable* (lacking drive to carry out tasks), the *morbid liars and swindlers* (superficial subjects that enjoy deception), *the psuedo-quarulants* (self-centered egocentric individuals with subclinical forms of paranoia), the *excitable* (individuals with labile and dramatic emotions), the impulsive (impulsive or compulsive actions), and the *eccentrics* (lack of uniformity or consistency in mental lives).
Partridge	(Partridge, 1930)	Antisocial behaviors; immature values, interests, and activities; emotional instability; disturbed social emotions. Importantly, Partridge is the first to stress that chronic social maladjustment is one of the most important features of psychopathy.
Henderson	(Henderson, 1939)	Those with psychopathy cannot live in society because they lack social emotions. They are antisocial, unstable and irresponsible, impulsive, explosive, egocentric, unempathic, and entitled.
Schneider	(Schneider, 1923)	Several subtypes of psychopathy-like disorders, including the self-assertive (entitled, boastful, manipulative, and deceptive), the explosive (unprovoked impulsive, explosive affective outbreaks), and the affectionless (callous, remorseless, deceptive, incorrigible, emotional dullness, propensity for criminal behavior).
Karpman	(Karpman, 1941, 1948a, 1948b)	Antisocial lifestyle; need for immediate gratification; lack of anxiety, guilt, or remorse; grandiose and entitled; callous; impulsive; irresponsible. Also noted the child-like immaturity in social emotions. "They only experience simple emotions like tension, worry, frustration that have no future implications" (Hervé & Yuille, 2007, p. 33), and therefore they are likely to act in the spur of the moment. Karpman also divided the construct into two different types: the symptomatic and the idiopathic.
Arieti	(Arieti, 1963)	Need for immediate gratification, callousness; lack of anxiety or guilt; grandiosity; irresponsibility; inability to learn from experience; lack of loyalty to group, persons, or code; and antisociality. *Short-circuited anxiety/emotion*: Individuals with psychopathy experience superficial emotions that are related to current situations. The lack of long-circuited emotions (related to future events) makes them act at the spur of the moment, which relieves any tension they may experience and therefore reinforces this behavior. Again, the emotional system is thought to be immature. Like Karpman, he speaks of symptomatic and idiopathic individuals with psychopathy.
McCord & McCord	(McCord & McCord, 1964)	Dangerous, maladaptive personality disorder with a deep-rooted lack of social emotions (empathy, love, guilt, remorse), egocentric manipulative attitude, callous, aggressive, impulsive. Individuals with psychopathy are prone to tension and frustration and experience intense but transitory emotions: The emotional deficits in psychopathy are confined to long-circuited emotions. According to Werlinder (1978), two features are most important: guiltlessness and lovelessness.
Craft	(Craft, 1966)	Identified traits that he thought were distinctive of psychopathy: *Positive*—Primary features: (a) lack of feeling quality to other humans (affectionless), (b) liability to act on impulse and without forethought. Secondary derived features: (a) aggression, (b) lack of shame or remorse, (c) inability to learn from experience (e.g., punishment), (d) lack of drive or motivation, (e) viciousness/will to damage things or persons. *Negative*—(a) lack of psychoses (schizophrenia or depression), (b) lack of pure intellectual deficit, (c) lack criminal motivation of planning of actions in the light of risks.

Table 1 (*continued*)

Author	Source	Abstract
Cleckley	(Cleckley, 1976)	Cleckley's description of psychopathy resembles the one described by Karpman and Partridge (p. 164 Werlinder, 1978). Cleckley Checklist: (a) superficial charm and good "intelligence," (b) absence of delusions, (c) absence of nervousness, (d) unreliability, (e) untruthfulness and insincerity, (f) lack of remorse or shame, (g) inadequately motivated antisocial behavior, (h) poor judgment and failure to learn by experience, (i) pathological egocentricity and incapacity for love, (j) general poverty in major affective reactions, (k) specific loss of insight, (l) unresponsiveness in general interpersonal relationships, (m) fantastic and uninviting behavior with/without drink, (n) suicide rarely carried out, (o) sex life impersonal, (p) failure to follow any life plan.
Hare	(Hare, 2003)	Psychopathy Checklist (PCL-R). Starting from the Cleckley checklist, Hare constructed a 20-item list capturing interpersonal-affective and antisocial lifestyle features.
		Theories including a fear deficit
Lykken	(Lykken, 1957)	Primary psychopathy is typified reduced fearfulness, ultimately leading to the development of psychopathy.
Patrick et al.	(Patrick, Fowles, & Krueger, 2009)	The triarchic model of psychopathy includes boldness, meanness, and disinhibition. Boldness includes toleration of stressful situations, self-confidence, and social assertiveness.

empirical research, and (b) the definition of the construct will influence whether empirical evidence for fear deficits in psychopathy will be observed.

Pinel (1806) formulated one of the earliest theories of psychopathy and stated that one of the core elements of psychopathy is a profound affective derangement, "as if the faculties of affect alone had sustained injury" (as cited in Werlinder, 1978, p. 29). A few decades later, Prichard (1835) spoke of "moral insanity" and argued that the essence of the disorder lies in the emotional and conative side of the psyche, rather than in the intellectual. Campagne (1869) described individuals with an intensely egoistic and disobedient character who could not be influenced by parents or teachers. It appears that Campagne may have been one of the first to note the limited range of emotionality of individuals with psychopathy. In 1915, Kraepelin (eighth edition) stated that, in the antisocial personalities, there is a lack of deep emotional reaction (Kraepelin, 1915), also suggesting that there is a limited range of emotionality in psychopathy (Millon, Simonsen, Birket-Smith, & Davis, 1998, p. 10). Schneider (1923) described the *affectionless*, who were callous, remorseless, and emotionally dull individuals. Importantly, however, in all of these earlier theories, it does not appear that a specific lack of fear is seen as a hallmark feature of psychopathy. Individuals with psychopathy were regarded as emotionally shallow, and, by inference, it could be said that these theorists may have meant that individuals with psychopathy also have a lack of fear, but this has not been stated clearly. After the 1950s, Cleckley, Karpman, and Arieti were among the first to propose that psychopathy is associated with a lack of anxiety, although not necessarily fear. Clearly, the hallmark study of Lykken (1957) provided the first empirical evidence for fear deficits in individuals with psychopathy. McCord and McCord (1964) stressed that individuals with psychopathy lack social emotions but are not completely devoid of emotions, and also claimed that individuals with psychopathy are "thick-skinned" and quite resistant to anxiety (though not necessarily fear). A later substantial influence on the concept of psychopathy was Hare's Psychopathy Checklist (PCL-R; Hare, Hart, & Harpur, 1991), which does not specify a lack of fear, although the PCL-R has been argued to account for fearlessness (Neumann, Johansson, & Hare, 2013). The PCL-R operationalizes psychopathy as a condition characterized by two interacting dimensions encompassing disturbed Interpersonal-Affective Functioning (Factor 1) and Antisocial Lifestyle (Factor 2). Subsequent research has also identified three-factor solutions (Cooke & Michie, 2001) and a four-facet solution (Hare & Neumann, 2008). Importantly, the PCL-R does not specify a lack of fear, although the PCL-R has been argued to account for fearlessness (Neumann et al., 2013). More recently, Patrick and colleagues (2009) introduced the triarchic model of psychopathy, which encompasses three (neurobiologically anchored) personality constructs, termed Boldness, Meanness, and Disinhibition. Fearlessness is considered to be a genotypic precursor for phenotypical boldness, which refers to "a capacity to remain calm and focused in situations involving pressure or threat, an ability to recover quickly from stressful events, high self-assurance and social efficacy, and a tolerance for unfamiliarity and danger" (Patrick et al., 2009, p. 926). In other words, *fearlessness* is defined as an abnormality in the system that should detect and respond to threat, and not necessarily in the conscious experience of the emotion.

In sum, the limited range of emotionality attributed to individuals with psychopathy was described relatively early (Campagne, 1869). But with the exception of Lykken's (1957) hypothesis and the triarchic model, there has been no clinically oriented theory positioning fear as a key feature of a psychopathic personality. This is a striking finding given the great role that fear has come to play in contemporary research on psychopathy. Importantly, some contemporary theories aim to characterize psychopathy from a neurobiological perspective (see Anderson & Kiehl, 2012; Koenigs et al., 2011). In the next section, we will shortly review the current state of the art of neurobiological and physiological research on fear in adult psychopathy.

The Neuroscience of Threat and Fear Experience

The past few decades have seen significant advances in the study of neurobiological underpinnings of human behavior. In the following sections, we will first provide a brief description

of the key components of the brain network believed to be involved in fear processing. We will differentiate between a network involved in automatic threat processing and one involved in conscious experience of fear, and review the evidence for or against a dysfunction in psychopathy in each network (for other detailed reviews, see Anderson & Kiehl, 2012; Gao & Raine, 2010; Koenigs et al., 2011). The main goal is to examine whether the neuroscientific literature offers support for the distinction between fear experience and threat processing proposed in our model and a differential relationship with psychopathy. Note that although many of the individual brain structures involved are often highly interconnected, they are discussed separately for the sake of clarity.

The Threat-Processing Network

The most prominent approach to study the (neuro)biological correlates of fear has been the employment of conditioning paradigms in which an aversive stimulus (unconditional stimulus [US]) is paired with a neutral stimulus (conditional stimulus [CS]). Through this process, the aversive properties of the US are transferred to the CS, resulting in a learned defensive response to the previously neutral CS (henceforth referred to as *threat conditioning*; LeDoux, 2014). A large body of evidence has pinpointed the amygdala as a key region involved in the acquisition, expression, and extinction of these aversive associations (Maren & Quirk, 2004; Olsson & Phelps, 2007). The amygdala is a limbic structure composed of 13 interconnected nuclei (Sah, Faber, Lopez De Armentia, & Power, 2003). The precise function of each nucleus in humans is not fully known (partly because of technical limitations), and most research on human fear has focused on the amygdala as a unitary structure or has parceled its nuclei into broader categories (Bzdok, Laird, Zilles, Fox, & Eickhoff, 2013; Moul, Killcross, & Dadds, 2012). The nuclei are often grouped into the central nuclei (CeN), the lateral amygdala (LA), and the basal amygdala (BA). In the psychopathy literature, the LA and the BA are often grouped together into the basolateral amygdala (Blair, 2005; Moul et al., 2012).

The Amygdala and Threat: Associative Learning and Autonomic Reactivity

Humans and animals generally learn to avoid threats through experience. This is commonly held to imply that the organism learns to associate the affective state of fear with a specific threat and the corresponding bodily reactions. This associative learning process is usually referred to as *fear conditioning*, but we will refer to this process as *threat conditioning*, as has been suggested by LeDoux (2014). The LA receives input from other brain structures and relays information to the CeN and the BA (Maren & Quirk, 2004). The CeN are subsequently involved in the modulation of behavioral and physiological autonomic reactions such as changes in heart rate (Kapp, Frysinger, Gallagher, & Haselton, 1979) and the startle reflex (Walker & Davis, 1997; Figure 2; but for an alternative model, see Balleine & Killcross, 2006), although recent

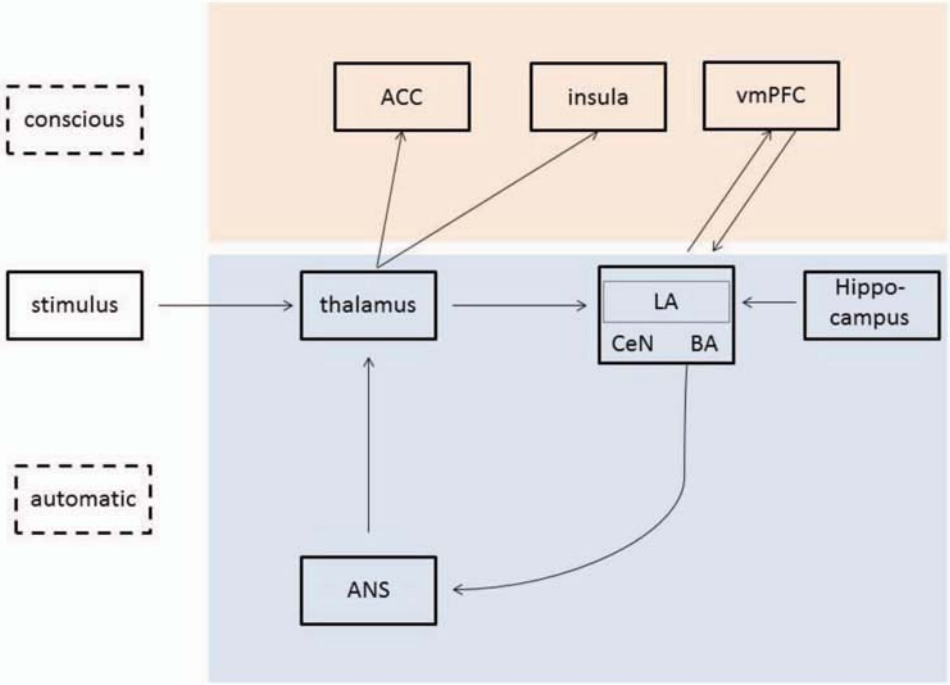

Figure 2. Simplified representation of an amygdala-centered threat network, in combination with the depiction of crucial nodes in the network subserving the conscious experience of fear. vmPFC = ventromedial prefrontal cortex; ACC = anterior cingulate cortex; LA = lateral amygdala; CeN = central nucleus of the amygdala; BA = basolateral amygdala; ANS = autonomic nervous system. See the online article for the color version of this figure.

evidence also suggests a role in threat conditioning (see Maroun, 2013).

The Hippocampus, Thalamus, and Prefrontal Cortex

Although the amygdala has received the most attention in the literature, threat conditioning also relies on other brain regions (see Figure 2). The LA receives input from the hippocampus, another key structure implicated in the coding of contextual information during aversive conditioning (Anagnostaras, Gale, & Fanselow, 2001; Maren, Phan, & Liberzon, 2013; Milad et al., 2007). That is, the hippocampus encodes information about the environment in which threat conditioning takes place (e.g., the room in which aversive shocks are administered), and when an organism is placed in the same environment again, these memories are reactivated. Recent animal work suggests that the dorsal portion of the hippocampus is specifically involved in the encoding of spatial features during threat conditioning (Zelikowsky, Hersman, Chawla, Barnes, & Fanselow, 2014).

The LA also receives input from the thalamus (Maren & Quirk, 2004). The involvement of the thalamus is particularly important, given its role in auditory threat conditioning. In auditory threat conditioning paradigms, the CS consists of an auditory cue, such as a tone, which is associated with the aversive event. This acoustic information is processed in substructures within the thalamus, such as the medial geniculate nucleus, and then passed on to the LA, where conditioning takes place (LeDoux, Cicchetti, Xagoraris, & Romanski, 1990; LeDoux, Sakaguchi, & Reis, 1984).

The thalamus and the amygdala are well connected to the prefrontal cortex (Behrens et al., 2003; Maroun, 2013). Various subregions of the prefrontal cortex are involved in the regulation and extinction of fear responses. For example, the ventromedial prefrontal cortex is thought to inhibit the amygdala during fear extinction, and the lateral prefrontal cortex has been linked to the employment of cognitive strategies that regulate fear responses (Delgado, Nearing, LeDoux, & Phelps, 2008).

Taken together, there is a large amount of animal and human literature showing the key roles of the amygdala in establishing associations during threat conditioning, the hippocampus in learning of contextual information related to threat, the thalamus as a relay station, and the prefrontal cortex as a modulator of threat responses. Importantly, there is also ample evidence indicating that this threat network processes information about threats automatically, and that it is unlikely that these responses are accompanied by the conscious experience of fear (LeDoux, 2014; Mineka & Öhman, 2002). With respect to psychopathy, this implies that disturbances within brain regions and functions located within this network would be indicative of disturbed threat processing instead of lack of fear. In the next sections, we will review the psychopathy literature for each of the components of the threat network discussed here in order to synthesize the general pattern of findings in the existing literature.

The Amygdala and Psychopathy

There is a rich literature linking psychopathy to dysfunctions in the amygdala. With regard to the associative learning of threat that takes place in the LA, Birbaumer and colleagues (2005) conducted one of the earliest fMRI studies reporting reduced amygdala activation during threat conditioning in a paradigm employing electric shocks as US. Associative threat learning was the topic of some of the earliest experimental work done in psychopathy in the 1960s and 1970s (Hare, 1965; Hare, Frazelle, & Cox, 1978). These studies found impaired associative learning of threat in relation to psychopathy, a finding that has since been replicated using different methodologies (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Rothemund et al., 2012; Veit et al., 2013). Interestingly, one of these studies reported more general aversive conditioning impairments in psychopathy (Flor et al., 2002). In this study, an obnoxious odor was used as a US instead of electric shocks. As such a stimulus is not expected to induce fear, the latter finding is in accordance with the notion that there is a generic threat circuitry that responds to various types of aversive stimuli, and that it is threat processing, not fear processing, that is impaired in psychopathy. Importantly, there is still no direct evidence showing that the LA shows reduced activation in relation to psychopathy during threat conditioning, but the disturbances in threat conditioning seen in the literature makes it likely that this region is indeed impaired in psychopathy. In addition, there are recent studies that examined the structural properties of the amygdala that indicate that psychopathy is related to volumetric abnormalities in a number of nuclei, including the LA and the CeN (Boccardi et al., 2011; Yang, Raine, Narr, Colletti, & Toga, 2009). However, Yang and colleagues (2009) reported reduced volumes, whereas Boccardi et al. (2011) reported increased volumes, and the precise functional significance of these contradicting results still needs to be addressed in these populations. Taken together, there is evidence indicating that psychopathy is related to diminished automatic threat conditioning, most probably driven by deficiencies in the lateral part of the amygdala, but it remains to be examined whether the LA is indeed directly responsible for these impairments.

The pattern of results from studies tapping into the functions of the CeN in psychopathy has been somewhat clearer. These studies employed various psychophysiological methods to establish more easily observable autonomic markers for reduced threat reactivity, such as skin conductance level (SCL; Arnett, Howland, Smith, & Newman, 1993; Dindo & Fowles, 2011a), heart rate, and startle reflex (Patrick, 1994). The startle is an automatic defensive reflex to natural or conditioned threat and has become increasingly meaningful in research on psychopathy (see, e.g., (Vaidyanathan, Patrick, & Bernat, 2009). It has been demonstrated that startle response decreases as a function of the interpersonal-affective component of psychopathy in community samples (Vanman, Mejia, Dawson, Schell, & Raine, 2003), as well as in incarcerated offenders with psychopathy (Patrick, 1994). In addition to the startle response, there is also a negative relationship between psychopathy and other measures of autonomic responses to threat, such as reduced SCL (Dindo & Fowles, 2011a) and decreased blood pressure (Pham, Philippot, & Rime, 2000). The findings for heart rate have been paradoxical, with reports of both lower heart rate (e.g., Patrick, Cuthbert, & Lang, 1994) and increased heart rate (Hare et al., 1978). Either way, whether heart rate is decreased or increased, these findings are still indicative of disturbed autonomic responding. These autonomic responses to threat have been shown to be modulated by the CeN (LeDoux, Iwata, Cicchetti, & Reis, 1988). With a few exceptions (e.g., Birbaumer et al., 2005; Flor et al., 2002), a relatively large body of work shows impairments in various types of autonomic responses to threat in psy-

chopathy, which is in accordance with the hypothesis that CeN functioning is aberrant in these individuals. Furthermore, a recent imaging study has shown a relationship between structural decrements in parts of the amygdala corresponding to the CeN and the affective component of psychopathy (Yang et al., 2009). Thus, there are reasons to believe that at least some of the functions carried out by the CeN are impaired in psychopathy when it comes to threat responsivity (for an alternative view, see Blair, Mitchell, & Blair, 2005).

The Hippocampus and the Thalamus in Psychopathy

The hippocampus has received much less attention in research on psychopathy. There are, however, indications from other lines of research suggesting that the hippocampus is compromised in psychopathy (e.g., Kiehl et al., 2001), but there are no functional MRI studies assessing its direct role in threat conditioning in psychopathy. However, studies assessing its morphology in psychopathy have found reduced hippocampal gray matter (Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012), smaller volumes (Laakso et al., 2001), an abnormal shape (Boccardi et al., 2010), and increased structural asymmetry (Raine et al., 2004). Although morphological abnormalities do not necessarily translate to functional impairment, these findings suggest that the hippocampus also plays a role in explaining the diminished threat conditioning associated with psychopathy. The thalamus is a severely understudied brain region in psychopathy research, but a recent study reported reduced fractional anisotropy (white matter conductivity) in psychopathic offenders that involved the anterior thalamic radiation (Hoppenbrouwers, Nazeri et al., 2013). In sum, the general pattern of findings indicates that the hippocampus and the thalamus seem to be impaired in psychopathy, but this conclusion is drawn with great caution, given the small amount of studies on these brain regions.

The Prefrontal Cortex in Psychopathy

In addition to the amygdala, the prefrontal cortex has received a lot of attention in research on psychopathy, with a focus on areas involved in cognition, such as the dorsolateral prefrontal cortex (Hoppenbrouwers et al., 2013; Hoppenbrouwers et al., 2014), and social and affective processing, such as the ventromedial prefrontal cortex, the anterior cingulate, and the insula (for more detailed reviews, see Anderson & Kiehl, 2012; Kiehl, 2006). The ventromedial prefrontal cortex has been implicated in many of the dysfunctions seen in psychopathy, ranging from selective behavioral impairments during probabilistic learning (Blair, 2008) to disturbances in more complex processes, such as moral reasoning (Blair, 2007). Importantly, the ventromedial prefrontal cortex is also involved in exerting inhibitory control over the amygdala in response to threat (Delgado et al., 2008). In their imaging study on threat conditioning in psychopathy, Birbaumer and colleagues (2005) found abnormal activation in the ventromedial prefrontal cortex. Moreover, recent studies assessing connectivity in psychopathy also found abnormal connectivity between limbic regions and the ventromedial prefrontal cortex (Contreras-Rodríguez et al., 2015; Motzkin, Newman, Kiehl, & Koenigs, 2011). Together, the body of findings points toward impaired functioning of the ventromedial prefrontal cortex, and there is direct evidence of functional abnormalities in processing threat information.

Interim Summary: Impaired Threat-Processing Network in Psychopathy

Our review of the literature points toward disturbances within the threat network. We found evidence showing functional and structural impairments in the amygdala as well as the prefrontal cortex in relation to psychopathy. However, the hippocampus and the thalamus have received much less attention in research on psychopathy, but the limited extant data seems to suggest abnormalities in these regions, too. Thus, the general pattern of the findings supports our proposal that threat processing is disturbed in psychopathy, but this claim has to be made with care, given that some brain regions are currently understudied in psychopathy. The targeted review of the neurobiological results highlights that there has been a relatively large focus on studying the amygdala in psychopathy, perhaps at the expense of other important brain regions in the network. This tendency is probably driven by the currently dominant beliefs that this structure plays a key role in threat processing and that reduced fear experience is central to psychopathy. However, it is important to realize that the affective and cognitive computations carried out by the brain are unlikely to occur in isolated brain structures (Lindquist et al., 2012), and that other parts of the network need to be studied, too, in order to truly understand the source of the threat processing impairments. Regarding our model in which we separate fear experience from threat processing, the review points out that there are indeed disturbances within the threat-processing network in psychopathy. However, as the more fine-grained parsing of fear has not been made before in the psychopathy literature, these impairments have often been (mis)interpreted as evidence for reduced conscious fear experience. Our model postulates that threat processing and fear experience rely on dissociable, but overlapping, neural circuits. Therefore, it is also necessary to determine the current state of the art in psychopathy research in relation to the circuit involved in conscious fear experience. In the following section, we will examine the neuroscientific literature on conscious experience of fear and relate the findings to the psychopathy literature. The goal is to determine whether there is neuroscientific support for abnormalities in conscious fear experience in psychopathy.

The Neuroscience of the Subjective Experience of Fear

Neurobiological research on the subjective experience of fear is scarce, in general, and even more so in psychopathy. However, peripheral feedback theories such as the James-Lange theory, the somatic marker hypothesis (Bechara, Damasio, Tranel, & Damasio, 1997), and the neurovisceral integration theory (Thayer & Lane, 2000) make important predictions about how the valenced affective component of emotions arises. The common denominator in these theories is that an emotive stimulus activates the autonomic nervous system via the central nervous system (as detailed above). This autonomic activity is then projected back to the central nervous system, where these motivational internal states are centrally represented (Critchley, 2005). The incorporation of

such peripheral feedback is extremely important for the affective component of emotions. Here, an important concept in the subjective experience of emotion is interoception. *Interoception* refers to the sense of self, and is superimposed on an accurate representation and recognition of the physiological state of the body (Craig, 2003). Recently, Terasawa, Fukushima, and Umeda (2013) suggested that interoception may underlie the subjective experience of one's emotional state, echoing the ideas of earlier researchers (Bechara et al., 1997; Critchley, 2005). As such, interoception is likely a necessary prerequisite for subjective experience.

The neurobiological network that allows for interoception entails various brain areas that receive feedback from, and project to, the autonomous nervous system. The crucial parts are the vagal nerve, the anterior cingulate cortex (ACC), and the insula. For instance, the efferent part of the vagal nerve affects heart rate, blood pressure, and sweating. As the vagal nerve has an important cardioinhibitory influence, it is a crucial pathway mediating the transition from stress-fueled behaviors to resting behaviors (i.e., a transition from the sympathetic to the parasympathetic nervous system; Porges, 2001, 2003). The close connection between the autonomic nervous system and emotion is further evidenced by findings showing that, in healthy controls, amygdala responses to fearful facial expressions depend on the phase of heartbeats (systole vis-á-vis diastole; Garfinkel et al., 2014). Vagal tone, which is measured as heart rate variability (HRV; i.e., differences in beat-to-beat alterations; Park, Vasey, Van Bavel, & Thayer, 2013), also affects emotional self-regulatory processes (Porges, Doussard-Roosevelt, & Maiti, 1994). Individuals with higher tonic HRV demonstrate context-appropriate responses, whereas individuals with higher phasic HRV responses facilitate emotional regulatory processes (Thayer & Lane, 2009). Thayer and Lane (2009) have described how cortical areas modulate the sympathetic nervous system via the vagal nerve. However, the vagal nerve is also a major afferent tract conveying physiological information regarding the state of the body. Via the nucleus of the solitary tract, this information reaches the (hypo)thalamus and, subsequently, the insula and the ACC.

The insula and (dorsal) ACC are crucially important brain areas for the subjective experience of emotion (Craig, 2005, 2011). The ACC has been highlighted as an area that is important for the inhibition of excessive emotional responding (Thayer & Lane, 2000), and cingulotomy results in a decrease in the conscious experience of pain (Viswanathan, Harsh, Pereira, & Aziz, 2013). A seminal study in healthy controls by Critchley, Wiens, Rotshtein, Öhman, and Dolan (2004) has shown that activity in the right anterior insula correlates strongly with heartbeat detection accuracy, and also with anxiety and negative affect. Phan and colleagues (2004) showed that the insula and amygdala are active when nondisabled subjects rate how intense certain emotional stimuli are. In addition, alexithymia (i.e., deficient identification and recognition of emotion) correlates with ACC activity (Gundel et al., 2004). A recent review suggested that alexithymia corresponds to brain alterations in the amygdala, insula, and cingulate cortex, although this correspondence depends on the type of emotional task that was being done (van der Velde et al., 2013). In line with this, Goerlich-Dobre, Bruce, Martens, Aleman, and Hooker (2014) showed that the right insula is larger in subjects with higher rates of the cognitive dimension of alexithymia (analyzing and verbalizing feelings), whereas the emotional reactivity dimension related more strongly to larger volumes in the right cingulate. A recent experiment showed that the representation of the body is more strongly influenced by affective touch (touch that is subjectively rated as pleasant) compared with regular touch (van Stralen et al., 2014), further underscoring the importance of the insula in the formation of integrated representations of bodily signals (i.e., metarepresentations). Affective touch is subserved by so-called C-tactile fibers. These slow-conducting unmyelinated fibers convey important affective information regarding the body, and terminate in the insula, showing the importance of the insula for the representation of affective bodily information.

Taken together, these findings indicate that the subjective experience of an emotion involves a metarepresentation of the physiological state of the body. In short, the mechanism here is that environmental information is processed by sensory systems (such as the thalamus), which relay this input to brain areas involved in threat detection and responsivity, for example, the amygdala. Hereafter, autonomic threat responses are initiated, resulting in physiological arousal (e.g., increased heart rate, skin conductivity, and pupil dilation). Autonomic feedback is then projected to the insula and ACC, where this neuronal input leads to visceral awareness (see Figure 2; Bechara & Naqvi, 2004).

Behavioral Evidence for Psychopathic Deficits in Identifying and Recognizing Emotions

Behavioral evidence indicates that psychopathic individuals may suffer from problems in recognizing and identifying (certain) emotions (e.g., Dawel, O'Kearney, McKone, & Palermo, 2012; Marsh & Blair, 2008). As noted earlier, alexithymia refers to the incapacity to identify and describe feeling and emotions. This personality trait is common among offenders and correlates positively with psychopathy (Gori et al., 2014; Grieve & Mahar, 2010; Lander, Lutz-Zois, Rye, & Goodnight, 2012; Louth, Hare, & Linden, 1998), suggesting that psychopathic individuals have difficulty recognizing their feeling and emotions. It is interesting that two studies observed that the deviant lifestyle component of psychopathy relates to alexithymia (Lander et al., 2012; Louth et al., 1998). Alexithymia is subdivided into two types; simply put, there is a type in which subjects have difficulty describing their emotional responses, and a type that is characterized by little autonomic and emotional reactivity to emotional situations (Bird & Viding, 2014). It is currently unclear to which type psychopathy is related. However, at the level of personality traits, psychopathic individuals may possess traits that make it difficult for them to identify and recognize their emotions and feelings, suggesting reduced conscious experience of fear.

Reminiscent of Cleckley's (1976) idea of semantic aphasia, a recent investigation applied the somatic marker hypothesis to psychopathy, stating that individuals with psychopathy may have trouble recognizing bodily sensations during emotions (Gao, Raine, & Schug, 2012). Gao and colleagues (2012) found that individuals with psychopathy do not adequately identify and recognize their bodily sensations during a social stressor experiment. A similar finding has also been observed recently by another research group (Nentjes, Meijer, Bernstein, Arntz, & Medendorp, 2013). The somatic marker hypothesis has also been directly tested in relation to psychopathy (Schmitt, Brinkley, & Newman, 1999). This study showed that the level of anxiety predicted deficits in

using somatic markers in becoming risk aversive, whereas psychopathic traits did not. Therefore, in addition to alexithymia, some findings suggest that psychopathic individuals also have difficulty recognizing and processing bodily signals that are related to emotions.

Insula and psychopathy. There is some evidence that the insula is affected in psychopathy. In studies looking at empathic responding in psychopathy, the insula is more active in psychopathic individuals than in nonpsychopathic individuals (Decety, Skelly, & Kiehl, 2013; Decety, Skelly, Yoder, & Kiehl, 2014). By contrast, psychopathy has also been reported to be related to reduced spontaneous responding to emotional or painful video clips in the insula and cingulate cortex (Meffert, Gazzola, den Boer, Bartels, & Keysers, 2013). Birbaumer and colleagues (2005) found reduced activation during threat conditioning in a limbic circuit including the insula and anterior cingulate. Decreased gray matter in insula has been observed in psychopathic individuals (de Oliveira-Souza et al., 2008), which is in line with other findings showing reduced insular thickness in psychopathy (Ly et al., 2012). During a probabilistic response reversal task, men with an antisocial personality disorder and psychopathy showed increased activation in the posterior cingulate cortex and anterior insula when punished errors were made in the reversal phase (Gregory et al., 2015). In psychopathy, reduced functional connectivity between the amygdala, on the one hand, and ACC, insula, striatum, and ventromedial prefrontal cortex, on the other hand, has also been recently reported during moral judgments (Yoder, Harenski, Kiehl, & Decety, 2015). Others have also observed (functional) insula alterations in relation to psychopathic traits (Seara-Cardoso, Sebastian, Viding, & Roiser, 2015; Viera et al., 2015; Yoder, Porges, & Decety, 2015) or psychopathy (Decety, Chen, Harenski, & Kiehl, 2015; Philippi et al., 2015; Sitaram et al., 2014). In contrast, Glenn, Raine, Schug, Young, and Hauser (2009) found no relationship between psychopathy and insular activity during moral decision making.

Cingulate cortex and psychopathy. Reduced activity in response to affective information has been found in the ACC in criminal psychopathic individuals (Kiehl et al., 2001; Müller et al., 2003). Motzkin and colleagues (2011) found differential resting-state ACC activity in psychopathic individuals when compared with nonpsychopathic individuals. In a prisoner's dilemma game, elevated psychopathic tendencies in an undergraduate sample were associated with reduced activity in the dorsolateral prefrontal cortex and rostral ACC when choosing to defect (Rilling et al., 2007). Reduced connectivity between the dorsal ACC and lateral parietal cortex has been related to overall psychopathy severity (Philippi et al., 2015). Psychopathic emotional detachment was recently found to relate to reduced fractional anisotropy in the dorsal cingulate (Sethi et al., 2015). During a social exchange game, White and colleagues (2013) also observed malfunction of the dorsal ACC in psychopathy. A recent study showed that individuals with elevated psychopathic traits showed reduced cingulate activation to a no-go task (Kim & Jung, 2014). Although reduced gray matter in various limbic regions, including the posterior cingulate cortex (Ermer et al., 2012) and subregions of the ACC (Ly et al., 2012), has been reported, others found no volumetric differences in the ACC in psychopathic individuals (Glenn, Yang, Raine, & Colletti, 2010). Glenn and colleagues (2010) hypothesized that reduced functionality in the ACC in psychopathy may be accounted for by reduced input from other areas such as the amygdala and orbitofrontal cortex. The latter suggestion is in line with general theories that distinguish between an "affective portion" and a "cognitive portion" in the cingulate cortex (Bush, Luu, & Posner, 2000). One hypothesis that still needs to be tested systematically is whether all key functions of the cingulate are affected in psychopathy, or whether the functional impairments primarily concern the affective subdivision of the ACC and are more selective in the cognitive subdivision (cf. Brazil et al., 2009, 2011).

Interim Summary: Subjective Experience of Fear and Psychopathy

Taken together, there is some evidence that the insula is affected in psychopathy, but this evidence cannot be taken to be conclusive. It should be noted, however, that there seems to be an increase in studies reporting insula deficits in psychopathy in the last few years. This pattern of results concerning the ACC seems to point out that some of the functionality of the cingulate cortex is affected in psychopathy. Whether these functional alterations in the cingulate cortex also relate to a different, or reduced, subjective experience of emotion remains unknown. The neurocognitive mechanism of interoception delineated above may explain awareness of physiological processes, but it should be noted that interoception, or peripheral feedback, cannot be equated with subjective experience (of fear), although it likely is a prerequisite. From the findings on brain regions and functions subserving emotion experience, it can be inferred that individuals with psychopathy may experience fear as less intense. That is, the deficient threat responsivity in the central nervous system of individuals with psychopathy leads to little autonomic activity. Feedback to the insula and ACC may then also be decreased, resulting in less intense awareness of visceral states, which may be accompanied by a less intense subjective experience of fear. To conclude, the neuroscientific findings point toward impairments within the threat network, and some leads have emerged that indicate deficits in the network involved in subjective experience of fear, although the body of evidence indicating the latter is significantly smaller.

Importantly, the reviewed neurobiological findings cannot provide conclusive evidence as to which components of fear are affected in psychopathy. Therefore, to further examine the evidence for fear deficits in psychopathy in light of our model, we conducted a systematic literature search and classified the studies based on the distinction between threat and conscious fear. We then performed a meta-analysis of the findings to directly quantify the evidence in favor of or against our model. The results are discussed in the following sections.

Literature Search and Meta-Analyses

A literature search was conducted in order to identify extant literature and investigate whether individuals with psychopathy have impairments in all subcomponents of fear (as formulated in our model). PubMed and Web of Science were used to search for articles using the combination of the terms "fear" and "threat" with "psychopathy." In order to determine whether potential impairments in the experience of fear may reflect general disturbances in conscious affective experience (Dawel et al., 2012; Marsh & Blair, 2008), we also searched for studies on the experience of sadness,

happiness, anger, and surprise. Based on the title and abstract, we selected the relevant articles based on the following inclusion criteria:

1. The studied population should *only contain adults*, that is, at least 18 years of age. In light of recent findings indicating that lack of fear also plays a role in female samples (e.g., (Brazil et al., 2013; Seara-Cardoso, Dolberg, Neumann, Roiser, & Viding, 2013; Verona, Bresin, & Patrick, 2013), we did not include gender as an exclusion criterion.

2. All samples were allowed, for example, offender samples, community samples, and undergraduates.

3. A validated measure was used to index psychopathy, for example, the PCL-R, Psychopathic Personality Inventory (PPI) or Self-Report Psychopathy Scale (SRP). (Hare, 2003; Lilienfeld & Andrews, 1996; Paulhus, Neumann, & Hare, in press)

4. *Fear* or *threat* should be the main focus of the article. For additional analysis including other emotions, the article should include direct measures for one or more of the emotions of happiness, sadness, anger, fear, disgust, or surprise.

5. Articles should be original research published in peer-reviewed journals.

6. Articles should be written in English.

Exclusion criteria were as follows:

1. Review papers, commentaries, and conference proceedings.

2. Subjects presenting with cognitive impairments, brain injuries, or major Axis I psychiatric disorders.

Meta-Analyses of Literature Search Results

The following criteria were employed for the meta-analyses:

1. Only studies reporting scorable results using measures directly related to threat processing/responding and conscious experience of emotions were included. Studies reporting MRI coordinates of blood-oxygen-level activation or electroencephalography measures without a strong established link with threat processing and/or conscious fear experience were excluded.

2. Only studies with samples sizes >10 were included (Hedges, 1982).

3. When more than two groups were used in a study, we compared the psychopathic group against the combination of the two other groups. This was done when the needed data was available or could be retrieved by contacting the authors. If a study did not include the statistics for the group comparisons but did report all relevant means, standard deviations, and sample sizes, effect sizes were calculated based on these of descriptive statistics. For studies employing dimensional measures of psychopathy, only zero-order correlations were included. Studies only reporting effects after statistical correction (e.g., for IQ) or methodology-related factors (e.g., order effects) that reduce comparability with the main body of research were not included.[1]

4. Effect sizes were calculated as Pearson's r using MetaCalc (Rosenberg, Adams, & Gurevitch, 2000) and transformed to Fisher's z for the analyses.

5. All relevant data had to be available. Authors of articles with missing data were contacted in order to retrieve data.

6. There were two coders (Sylco S. Hoppenbrouwers and Inti A. Brazil) who independently extracted relevant data from all articles. Interrater agreement was excellent (Spearman's $r = 1.0$).

7. A random effects model was used as implemented in MetaWin (Rosenberg et al., 2000). Heterogeneity was assessed using the Q statistic (Cochran, 1954), and Rosenthal's fail-safe number was used for file drawer analyses with alpha = 0.05 (Rosenthal, 1979).

The selected articles were also hand searched to locate other studies that were not detected in our automatic search as well as studies that provided data for multiple emotions. Based on these criteria, 18 articles were found suitable for threat processing, and 16 for the experience of fear, 15 for anger, seven for sadness, six for happiness, three for surprise, and one for disgust. After inclusion, we examined each study on fear and threat and categorized the type of outcome measure(s) used according to our model and meta-analyzed the results. For example, the employment of the startle reflex as an outcome measure would be classified as "Automatic: Internal," whereas fear recognition would be "Conscious: Identification." The results are presented in Table 2. The included articles for the additional analysis on the conscious experience of fear and other emotions are depicted in Table 3.

Results of the Meta-Analyses

Fear versus threat. The mean effect size for the conscious component was $r = 0.097$ and not significant. In contrast, the mean effect size for the automatic component was moderate ($r = 0.21$) and significant (see Table 4). No relationship was found between psychopathy dimension scores, subjective fear, and threat (see Footnote 1). Thus, although the overall effect size for threat

[1] We also meta-analyzed the effect size for the psychopathy dimensions. If a study provided results for psychopathy dimensions/factors, these were also included in order to assess the relationships with threat and the experience of emotions. A two-factor solution is commonly found across different instruments, so only scores based on the traditional two-factor solution were included. All the findings were nonsignificant. Importantly, the amount of suitable studies including information about the factors was small, so these effect sizes cannot be estimated reliably and we therefore refrain from interpreting the findings.

Table 2
Studies Included in the Meta-Analysis of Threat and Fear

Study	Methods and measures	Participants	Psychopathy measure	Main findings	Deficit?	Place in model
(Hare, 1965)	Fear-conditioning using auditory cues Generalization of conditioning stimuli was also investigated. Skin conductance was measured.	24 offenders	Cleckley's checklist	Nonpsychopathic offenders showed more conditioned responses than offenders with psychopathy. Conditioned fear responses generalized less in offenders with psychopathy than in nonpsychopathic offenders.	Yes	Automatic: internal
(Hare, 1965a)	Fear conditioning paradigm	20 students assigned to a high- or - low-psychopathy group (10 each)	MMPI Psychopathic Deviance scale	Shock imminence induced greater, earlier, and more rapid increase in skin conductance in the low-scoring group when compared with the high-scoring group.	Yes	Automatic: internal
(Hare, 1965b)	Skin conductance was monitored while the numbers 1–12 were consecutively presented on a memory drum	11 psychopathic, 11 nonpsychopathic criminals, and 11 noncriminal controls	Cleckley's checklist	Log conductance increases in the interval prior to shock were significantly smaller and began later for the psychopathic than for the nonpsychopathic subjects. The individuals with psychopathy were less responsive to shock than were the nonpsychopathic criminals but did not differ significantly from the noncriminal controls. There were no significant differences among groups in the rate of recovery from the effects of shock.	No	Automatic: internal
(Schmauk, 1970)	Aversive-conditioning using skin conductance	30 primary psychopathic offenders vs. 30 neurotic psychopathic offenders vs. 30 normal controls	Psychopathic deviance scale of the MMPI	Primary psychopathic offenders showed less SCR in anticipation to shock	Yes	Automatic: external
(Hare, Frazelle, & Cox, 1978)	Heart rate, respiration rate, digital and forehead pulse amplitude, and skin conductance were measured during threat anticipation	64 offenders assigned to groups based on median split on psychopathy and socialization score	Cleckley's checklist	No tonic differences in autonomic activity between offenders. Most individuals with psychopathy (high-psychopathy/low-socialization) had an increase in heart rate but little increase in SC activity	Inconclusive	Automatic: internal
(Sterling & Edelmann, 1988)	Psychopathic and nonpsychopathic subjects rated emotionality of situations	17 offenders and 15 nonoffenders	Socialization scale	The psychopathic group rated situations as more fear-inducing	No	Conscious: negative valence and recognition
(Ogloff & Wong, 1990)	Countdown toward shock experiment	32 offenders	PCL	Less anticipatory SCR in response to shock onset	Yes	Automatic: external
(Patrick, Bradley, & Lang, 1993)	Emotional picture viewing task with startle response, SCR, EMG, and heart rate measures	54 male sexual offenders, divided in three groups	PCL-R	No differences in negative valence, but autonomic measured differed between groups	Automatic: Yes Conscious: No	Automatic: internal and external Conscious: negative valence

Table 2 (*continued*)

Study	Methods and measures	Participants	Psychopathy measure	Main findings	Deficit?	Place in model
(Patrick, Cuthbert, & Lang, 1994)	Questionnaire Upon Mental Imagery, Fear Survey Schedule Neutral, and emotional sentences	54 male sexual offenders, divided in three groups	PCL-R	No group differences in fearfulness, mental imagery, or imagery experience. Individuals with high psychopathy scores had lower SC responsivity during fearful imagery.	No Yes	Conscious: negative valence Automatic: internal
(Blair, Jones, Clark, & Smith, 1997)	IAPS	18 psychopathic and 18 nonpsychopathic offenders	PCL-R	Psychopathic offenders showed normal responses to threatening stimuli	No	Automatic: external (SCR)
(Schmitt & Newman, 1999)	Self-report measures of anxiety and fear. Fear was measured through the Constraint scale and the Harm Avoidance scale of the Multidimensional Personality Questionnaire	217 offenders	PCL-R	Psychopathy and the questionnaire measures did not correlate	No	Conscious: negative valence
(Levenston, Patrick, Bradley, & Lang, 2000)	Affective viewing task with IAPS pictures and white noise probes were used to measure startle, facial EMG, and SCR	36 male offenders	PCL-R	Individuals with psychopathy showed inhibited startle to victim scenes and little startle potentiation to threat	Yes (automatic)	Automatic: internal
(Herpertz et al., 2001)	Number of psychophysiological tests (SC, corrugator EMG activity) to measure emotional dimensions of valence and arousal	25 male psychopathic offenders, 18 male offenders with borderline personality disorder (BPD), and 24 controls	PCL-R	Individuals with psychopathy had lower SC responses during emotional and neutral pictures compared with BPD and controls	Yes (automatic)	Automatic: internal
				More individuals with psychopathy had no startle reflex. Psychopathic group also did not show a modulation of startle by emotional slides. No difference in subjective ratings of valence and arousal.	No (conscious)	Conscious: recognition
(Blair et al., 2002)	Recognition of vocal affect	39 offenders	PCL-R	Influence of IQ on vocal affect recognition. Individuals with psychopathy recognized less emotions, in particular, sadness and fear.	Yes	Conscious: recognition
(Sutton, Vitale, & Newman, 2002)	IAPS	87 nonpsychopathic and 21 psychopathic offenders	PCL-R	Deficient startle modulation at 2 s in psychopathic offenders	Yes	Automatic: external
(Blair, Mitchell, Peschardt, et al., 2004)	Multimorph emotion recognition task	19 offenders with low PCL-R scores and 19 high-scoring offenders	PCL-R	Intact recognition for all emotions except fear	Yes	Conscious: recognition
(Benning, Patrick, & Iacono, 2005)	Picture viewing task with fear potentiated startle and skin conductance using an extreme groups approach	A community sample of 307 young male twins	PPI	Participants high on fearless dominance showed deficits in fear potentiated startle. Participants high on impulsive antisociality had skin conductance deficits.	Yes	Automatic: internal and external
(Dolan & Fullam, 2006)	Face affect recognition task	49 offenders with diagnosis of dissocial personality disorder and 49 healthy controls	PCL-SV	No evidence for recognition deficits of fearful facial expression in offenders or relationship to psychopathy.	No	Conscious: identification

(*table continues*)

Table 2 (*continued*)

Study	Methods and measures	Participants	Psychopathy measure	Main findings	Deficit?	Place in model
(Book, Quinsey, & Langford, 2007)	Japanese and Caucasian Facial Expressions of Emotion stimulus set and Neutral Faces task	72 inmates and 60 controls	LSRP and PCL-R	Intact recognition for all emotions	No	Conscious: recognition
(Del Gaizo & Falkenbach, 2008)	DANVA-2-AF for facial emotion recognition and DANVA-2-AP for vocal affect recognition	175 undergraduates	PPI	Deficit in recognizing fearful facial expression	Yes	Conscious: recognition
(Justus & Finn, 2007)	Startle response to IAPS pictures with different probe presentation times	108 community subjects	Psychopathic deviate scale of MMPI and PPI short form/Fear Survey Schedule to index fearfulness	High levels of psychopathy in men was related to reduced augmentation of the startle response to aversive stimuli	Yes	Automatic: external
(Hastings, Tangney, & Stuewig, 2008)	Face affect recognition task	154 male inmates	PCL-SV	Negative correlation between accuracy and overall psychopathy score. Significant negative correlations between psychopathy and recognition of sad and happy facial expression. No evidence for fear recognition deficits.	No	Conscious: recognition
(Vaidyanathan, Patrick, & Bernat, 2009)	Various fear-related self-report questionnaires including the Fear Survey Schedule, the Fearfulness subscale of the EAS Temperament Survey, the Tridimensional Personality Questionnaire – Harm Avoidance scale and the Sensation Seeking Scale	88 undergraduates	PPI	A positive correlation was found between threat responsivity and the fearlessness scale of the PPI	Yes	Automatic: external
(Baskin-Sommers, Newman, Sathasivam, & Curtin, 2011)	Fear potentiated startle, SRP-III, Brief MPQ	92 psychopathic offenders	PCL-R	Fear deficits (predicted by different theories) were not present in African American offenders	No	Automatic: external
(Dindo & Fowles, 2011)	Self-report, skin conductance	131 Students	PPI	Results indicated that PPI-1 (low fear) was associated with reduced SC during anticipation of an aversive noise and PPI-2 (regulatory dysfunction) was associated with enhanced SC reactivity during presentation of a speech about one's faults.	Yes	Automatic: internal
(Lishner et al., 2012)	Affective empathy task	Study 2: 60 offenders	PCL-SV		No	Conscious: recognition
(Marsh & Cardinale, 2014)	Moral judgment task requiring recognition of emotions	38 undergraduates	PPI	Psychopathy correlated negatively with the identification of fear-causing statements	Yes	Conscious: recognition

Table 2 (*continued*)

Study	Methods and measures	Participants	Psychopathy measure	Main findings	Deficit?	Place in model
(Rothemund et al., 2012)	Aversive Pavlovian delay conditioning: Event-related potentials, startle response potentiation, skin conductance response, corrugator activity, and heart rate were assessed, along with valence, arousal, and contingency ratings of the CS and US	11 criminal psychopathic individuals vs. 11 healthy matched controls	PCL-SV	The psychopathy group showed associative learning deficits as indexed by a lack of differential startle response, lack of increased skin conductance, and a lack of increased corrugator activity to the CS.	Yes	Automatic: internal
(Brook & Kosson, 2013)	Empathic accuracy task and self-reported empathy	103 inmates	PCL-R	Psychopathy was associated with reduced empathic accuracy for negatively valenced emotions only (fear and sadness)	Yes	Conscious: recognition
(López, Poy, Patrick, & Molto, 2013)	Fear conditioning paradigm	74 undergraduates	PPI-R	PPI-factor fearless dominance was related to deficient acquisition of fear, irrespective of scores on the impulsive antisocial scale	Yes	Automatic: internal
(Baskin-Sommers, Curtin, & Newman, 2013)	Emotional picture viewing task with fear potentiated startle.	136 male inmates	PCL-R	Individuals with psychopathy had startle response deficit during emotion modulation by novel pictures but not by familiar pictures.	Yes	Automatic: internal
(Veit et al., 2013)	Event-related potentials, skin conductance, and subjective evaluation during delayed fear conditioning experiment	14 male individuals with psychopathy	PCL-R	During early acquisition, high psychopathy was negatively associated with differential skin conductance responses and N100 amplitude. The affective facet of the PCL-R explained most variance in startle response deficits.	Yes	Automatic: internal

Note. For each study, the columns provide information about the first author, the methods used, the instrument used to assess psychopathy and the number of participants included, the relevant main findings of each study, whether there was a deficit or not, and the type of outcome measure according to our model. MMPI = Minnesota Multiphasic Personality Inventory; SCR = skin conductance response; SC = skin conductance; PCL = Psychopathy Checklist; PCL-R = Psychopathy Checklist-Revised; IAPS = International Affective Picture System; EMG = electromyography; IQ = intelligence quotient; PPI = Psychopathic Personality Inventory; LSRP = Levenson's Self-report Psychopathy Scale; DANVA-2-AF = Diagnostic Analysis of Nonverbal Accuracy-form 2; EAS = Emotionality-Activity-Sociability Scale; MPQ = Multidimensional Personality Questionnaire; SRP-III = Self-report Psychopathy Scale III; PCL-SV = Psychopathy Checklist-Screening Version.

Table 3
All Studies That Included a Measure of the Conscious Experience of an Emotion and Were Incorporated into the Meta-Analysis

Study	Happy	Sad	Fear	Anger	Surprise
Sterling & Edelmann, 1988			X	X	
Patrick, Bradley, & Lang, 1994			X		
Schmitt & Newman, 1999			X		
Levenston, Patrick, Bradley, & Lang, 2000			X		
Herpertz et al., 2001			X		
Blair, Mitchell, Peschardt, et al., 2004	X	X	X	X	X
Babcock, Green, Webb, & Yerington, 2005				X	
Hicklin & Widiger, 2005				X	
Cale & Lilienfeld, 2006				X	
Derefinko & Lynam, 2006				X	
Dolan & Fullam, 2006			X		
Book, Quinsey, & Langford, 2007			X		
Justus & Finn, 2007			X		
Del Gaizo & Falkenbach, 2008	X	X	X	X	
Eisenbarth, Alpers, Segrè, Calogero, & Angrilli, 2008		X			
Hastings, Tangney, & Stuewig, 2008	X	X	X	X	
Bagley, Abramowitz, & Kosson, 2009	X	X		X	X
Edens & McDermott, 2010				X	
Dindo & Fowles, 2011			X		
Hornsveld, Muris, & Kraaimaat, 2011				X	
Lishner et al., 2012	X	X	X	X	
Marsh & Cardinale, 2014	X		X		
Swogger, Walsh, Homaifar, Caine, & Conner, 2012				X	
Rothemund et al., 2012					
Brook & Kosson, 2013		X	X		X
Veit et al., 2013			X		
Coccaro, Lee, & McCloskey, 2014				X	
Gilbert, Daffern, Talevski, & Ogloff, 2015				X	

was moderate, these findings provide quantitative support for our model and show that psychopathy is characterized by impaired automatic threat processing and responsivity, and not by disturbances in the conscious experience of fear. Importantly, these results do not necessarily show that the impairments in the subjective experience of fear are completely absent. Instead, they indicate that the body of evidence suggesting disturbances in the conscious experience of fear is relatively small, and that lack of fear in psychopathy is less of a given than is commonly believed.

Psychopathy and the experience of emotions. In addition to fear, we also meta-analyzed the experience of other emotions in adult psychopathy (see Table 5). We found that psychopathy is related to increased experience of anger and reduced experience of happiness. Thus, there seem to be no impairments in the experience of sadness, fear, and surprise. Disgust could not be meta-analyzed, given that only one study was found to be suitable. Importantly, however, care is needed when interpreting the results for sadness and surprise, given that a relatively small number of studies were included ($3 \leq N \leq 7$). In theory, a meta-analysis can be conducted on as few as two studies, but the effect sizes found in the individual studies varied and even tended to go in opposite directions (see also confidence intervals in Table 5). The analyses of fear and anger included a larger amount of studies and are therefore more reliable.

Fail-safe numbers and heterogeneity. The fail-safe number indicates how many unpublished studies with nonsignificant re-

Table 4
Results From the Meta-Analyses for the Conscious Experience of Fear and Automatic Threat Detection and Responsivity

Subcomponent of fear	Mean effect size	95% CI	Fail-safe number	Heterogeneity *Q*
Automatic	.21[a]	.09, .32	88.2	16.66
Conscious	.097	−.07, .26	—	15.76

Note. Mean effect sizes are reported as Pearson's *R* with their corresponding 95% confidence interval (CI), Rosenthal's fail-safe number, and heterogeneity statistic. Note that a positive correlation (i.e., effect size) indicates a deficit.
[a] Denotes a significant finding at $\alpha = .05$.

Table 5
Effect Sizes for the Conscious Experience of Emotion in Relation to Psychopathy

Emotion	Mean effect size	95% CI	Fail-safe number	Heterogeneity *Q*
Happy	.194[a]	.013, .37	13.6	6.40
Sad	.085	−.10, .28	—	6.95
Fear	.097	−.07, .26	—	15.76
Anger	−.15[a]	−.32, .01	7.1	10.2
Surprise	.14	−.29, .56	—	2.12

Note. Mean effect sizes are reported as Pearson's R with their corresponding 95% confidence interval (CI), Rosenthal's fail-safe number, and heterogeneity statistic. Note that a positive correlation (i.e., effect size) indicates a deficit.
[a] Denotes a significant finding at $\alpha = .05$.

sults are required in order to make the effects found in a meta-analysis nonsignificant. In our case, 88.2 unpublished studies with null findings are needed to make the effect for threat processing and responding nonsignificant, 13.6 studies for happy, and 7.1 studies for anger. Rosenthal (1979) suggested that the minimum number of unpublished studies required should exceed the threshold of $5n + 15$, with n representing the number of studies included in the meta-analysis. The fail-safe numbers for threat (88.2), happy (13.6), and anger (7.1) do not exceed these respective thresholds. Thus, this general rule of thumb would not allow classification of these fail-safe numbers as robust. Still, roughly 4 times as many unpublished zero-effect studies as already published studies would be needed to nullify our findings for threat. Finally, the nonsignificant heterogeneity statistics indicated that the samples formed homogeneous groups.

Discussion

Threat Processing and Conscious Experience of Emotions in Psychopathy: Meta-Analytic Evidence

The results of the second meta-analysis assessing conscious experience of emotions in psychopathy show that only the experience of happiness seems to be reduced, whereas anger was found to be increased in psychopathy. The effect sizes for happiness and anger were moderate but significant. Note, however, that the fail-safe numbers were very low for happiness (13.6) and anger (7.1). None of the emotions were significantly related to the psychopathy dimensions. Of more importance for the main goal of our study, the experience of fear was not compromised in psychopathy. These findings are not entirely in accordance with those obtained in some of the previous meta-analyses on conscious emotion processing. Marsh and Blair (2008) focused on emotion recognition in the general antisocial population and found that the recognition of fear and sadness was compromised, but that there were no impairments for the recognition of happiness, anger, surprise, and disgust. These effects, however, were independent of the level of psychopathy. Other meta-analyses by Wilson, Juodis, and Porter (2011) and Dawel et al. (2012) suggested a general impairment in recognizing emotions. Importantly, Dawel et al. also tested whether adults and youths with psychopathic tendencies differed in their ability to recognize emotions. In line with our findings, Dawel et al. found that there was no fear deficit and that only the recognition of happiness and surprise was impaired in adults. Finally, Derefinko (2014) tested the roles of anxiety, fear, and constraint in psychopathy, and reported a weak effect ($r = .05$) for a fear deficit in psychopathy. Thus, the pattern of results in previous meta-analyses on emotion recognition is inconsistent. One likely reason is that reliability tends to be low, given the limited amount of studies that are often included for each emotion. A second issue is the aggregation of samples of adults, children, and adolescents scored on some measure of psychopathy in prior meta-analyses. It still remains to be established whether psychopathy can be collapsed across these different age groups (Hart, Watt, & Vincent, 2002). Our study is the first to specifically focus on emotion experience in psychopathy in adult populations. In fact, the need to target adult populations was one of the issues explicitly highlighted by Dawel et al.

When aggregating adult and youth samples, it is necessary to keep in mind that these populations differ in their cognitive and affective capabilities, and that these discrepancies may have unexpected and unwanted effects on multiple levels. In the case of emotion experience, it is crucial to consider that children need to learn to recognize and differentiate between emotions, and that this process is influenced by both genetic and environmental factors, such as physical abuse and neglect (Camras, Grow, & Ribordy, 1983; Pollak, Cicchetti, Hornung, & Reed, 2000). In general, abused and neglected children tend to show impairments in the decoding and experience of emotions, although they seem to differ in which emotions are afflicted. It is often highlighted that children with psychopathic tendencies show affective impairments because of biological factors, such as reduced amygdala functioning (for a review see Blair, 2013). However, these children are also at elevated risk of experiencing abuse and/or neglect, making it likely that detrimental environmental factors promote aberrant learning of emotion categories (Kimonis, Fanti, Isoma, & Donoghue, 2013). Impaired performance in experimental settings could reflect an abnormal or delayed learning trajectory that might be partially corrected by the time they reach adulthood. This would be an explanation for the different patterns of emotion deficits between children/adolescents and adults reported by Dawel et al. (2012), and also further highlights that aggregating these different types of populations reduces specificity and may not be an optimal approach. Future longitudinal studies should aim to establish the role of environmental factors in the acquisition of emotion categories and charting the trajectory of the development and stability of emotion experience in children with psychopathic tendencies. Finally, our meta-analysis of conscious fear experience was not limited to emotion recognition but also included measures of negative valence. As postulated by our model, the experience of fear extends beyond recognition of others' emotions and also involves the experience of a negatively valenced internal state. Negative valence is one aspect of fear that has been overlooked in prior meta-analyses. By incorporating negative valence, we tested a more complete operationalization of fear experience, which is also a source of discrepancy with previous findings.

Threat Processing Impairments, Not Fearlessness: Possible Implications for Three Influential Contemporary Models of Psychopathy

There are currently three major theories on the underlying cognitive and emotional mechanisms of psychopathy. All three theories partly explain the "fear" deficit in psychopathy, but do so in different ways. We will describe these models succinctly and compare them. We will also discuss them in relation to the results of the meta-analysis on threat processing and conscious fear experience.

Blair: Integrated emotion systems (IES). The IES model (Blair et al., 2005) is a neurobiological account of psychopathy that focuses on a brain network heavily involved in processing social and affective information as well as reinforcement-based learning. This network includes the basolateral and central amygdala, insula, ventromedial prefrontal cortex, motor cortex, and sensory association cortex. The amygdala plays a pivotal role in this model, as it is thought to be of paramount importance for the formation of stimulus–reinforcement associations, which is im-

paired in psychopathy. Furthermore, disturbances in associative learning are predicted for both aversive and appetitive stimuli, but may be more pronounced for aversive stimuli (Blair, Mitchell, Leonard et al., 2004). The brain network that lies at the core of the IES includes the threat processing and responding circuit, and as a result of deficiencies in key brain areas in psychopathic individuals, such impairments are expected to lead to reduced threat responsivity. Moreover, amygdala dysfunctions are held to also impair recognition of sad and fearful facial expressions of others. Fearful and sad expressions convey information about another individual's distress. Reduced recognition of these emotions in psychopathy is believed to be play a key role in disturbed associative learning of the distress signals with the actions that led to the negative outcomes. Taken together, the IES predicts that psychopathic individuals have impairments in appropriate processing and responding to both threats and distress in others. It should be noted, however, that Blair et al. also state that "fear" is an umbrella term that incorporates different behaviors stemming from dissociable brain systems, and that there is no unified fear network in the brain (Patrick, 2006).

Newman: Response modulation hypothesis (RMH). The RMH proposes that individuals with psychopathy have difficulty in temporarily suspending a dominant response set in order to use peripheral information to evaluate the predominant response set and modulate behavior if necessary (Newman & Baskin-Sommers, 2011). The RMH differs from other theories in that it proposes that attention moderates the emotion deficits in psychopathy, rather than ascribing all emotional problems to a deficit in affective reactivity per se. Although not primarily centered on fearlessness, an important implication of the RMH is that abnormalities in both fear and inhibition (e.g., in the context of passive avoidance learning) vary as a function of attentional focus. According to Newman and colleagues, individuals with psychopathy suffer from an early attention bottleneck "that interferes with the processing of information that is incongruent with a current goal rather than limitations in later stages of selection" (Newman & Baskin-Sommers, 2011, p. 427). That is, once a goal-directed focus of attention is established, all seemingly irrelevant information is gated out, resulting in rigid perseverance of the initiated behavior. As a result, individuals with psychopathy do not process, and are therefore relatively insensitive to, stimuli that would typically induce a fearful response (Newman, Curtin, Bertsch, & Baskin-Sommers, 2010). However, a recent study also suggested that it may be the integration of contextual information that is impaired in psychopathy, even when contextual information was goal-relevant (Hoppenbrouwers, Van der Stigchel, Slotboom, Dalmaijer, & Theeuwes, 2015).

We find it interesting that a recent meta-analysis (Smith & Lilienfeld, 2015) of the RMH showed a similar effect size to the one observed here for threat processing, $r = .20$. As such, accounts suggesting disturbed modulation of attention or threat processing impairments in psychopathy seem to outperform other frameworks postulating that a lack of fear experience is central to psychopathy.

Patrick: The dual-process model of psychopathy. Patrick and Bernat (2009) have proposed a model of psychopathy in which two etiologically distinct pathways lead to Factor 1 and 2 of the PCL-R (Hare et al., 1991), respectively. Regulatory control deficits (e.g., low constraint associated with a myriad of externalizing disorders) are associated with the antisocial lifestyle factor (Hervé & Yuille, 2007). On the other hand, the core characteristics (affective/interpersonal) of psychopathy relate to an aversive/defensive system that interacts with an appetitive approach system. This model proposes that approach behavior will be terminated when environmental cues reach a certain threshold, after which the aversive defensive system outweighs the approach system. Naturally fearless individuals (e.g., psychopathic individuals) suffer from a weak defensive system, and, as a consequence, behavioral avoidance is less likely to occur in the presence of environmental cues signaling danger (Hervé & Yuille, 2007). This model also proposes that individuals with psychopathy are normally responsive to aversive stimuli (Flor et al., 2002), but do not form associations between a conditioning stimulus and an aversive event, suggesting a primary deficit in aversive learning (López, Poy, Patrick, & Molto, 2013; Rothemund et al., 2012). The underlying substrate is thought to include the amygdala, typified as the core of the defensive fear system (Hervé & Yuille, 2007).

Comparison of Models in Relation to Fear

The review of all empirical articles regarding fear and psychopathy suggests that, for psychopathy, the evidence is more in favor of the notion that there are deficits in threat detection and responsivity rather than reduced experience of fear. It also shows that many studies that use the term *fear* in fact measure threat detection and threat responsivity, and do not tap into the psychological phenomenon of (experiencing) fear. All three models converge on the notion of impaired threat detection and responsivity. For instance, the dual-process model of psychopathy suggests that defensive reactivity is a key problem in psychopathy. Our review and meta-analysis corroborates the idea of impaired defensive reactivity. In the same vein, the IES is focused on amygdala-based deficiencies, which have a detrimental effect on threat processing and responding, emotion recognition, and aversive learning. An important claim of the IES is that individuals with psychopathy should be impaired in the identification of fearful facial expression in others. Although this may be true for antisocial populations in general (Marsh & Blair, 2008), we were unable to find conclusive evidence that this is indeed the case in adult psychopathy, and neither have other recent meta-analyses on related topics (Dawel et al., 2012; Derefinko, 2014). Importantly, the IES postulates a general fear processing deficit that is independent of circumstances and manifests itself in both automatic threat processing and conscious fear experience. By contrast, the RMH suggests that fear deficits relate to situation-specific information processing demands. The RMH would predict threat detection and responsivity problems, but not necessarily any deficits in the subjective experience of fear. This prediction is in line with our review of the available empirical evidence. However, in assessing the relevance of the RMH for the low-fear hypothesis, one potential issue that deserves attention is that some key studies do not contain a baseline condition in which there is no manipulation of attention (Newman et al., 2010). Although the effect of attention deployment on threat reactivity (i.e., the startle response) has been shown consistently, it is unclear what the strength of this effect is. It could be argued that startle response tasks require subjects to attend to less frequently occurring aversive probes, and there will therefore always be engagement of attention. As such, it could be argued that there is no meaningful way of incorporating a baseline con-

dition in such experiments. However, as the low-fear hypothesis postulates a pan-situational fear deficit, it is crucial that studies are regarded in that light. Thus, as no baseline condition (with minimal demands for attention) is present in these studies, it remains to be elucidated to what extent top-down deployment of attention normalizes threat reactivity.

However, the RMH does not appear to be completely at odds with the dual-process model of psychopathy or the IES when it comes to threat reactivity. In fact, the RMH is premised on the idea that there is indeed a spontaneous threat detection problem related to psychopathy, likely to be amygdala-based, and that attention deployment moderates the deficiency in threat detection and responsivity (Larson et al., 2013). Patrick & Bernat's (2009) dual-process model is similar to the Blair et al. (2005) IES model in postulating a deficient emotion-related defensive system, and in relating this deficit to a specific component of psychopathy (interpersonal-affective traits). Patrick also distinguishes this deficit from other components of emotion processing, including the subjective assessment and arousal components. Thus, all three influential models focus on somewhat different aspects of fear processing in psychopathy, but converge on the notion that threat detection and responsivity is impaired in psychopathy. Although they are clearly differentiable in specific, important, and testable ways, they do not appear to be incompatible with each other with regard to predictions about threat-based impairments in psychopathy.

Next, the models are discussed in light of predictions on which they differ.

IES Versus RMH

The IES predicts that psychopathic individuals are impaired in learning associations between negative (affective) outcomes and the stimuli that led to these outcomes. Imagined threat scenes evoke less SCRs in psychopathic individuals compared with nonpsychopathic individuals (Patrick et al., 1994). The IES would attribute this finding to amygdala-based deficits in CS-affect representations. However, two RMH studies (Baskin-Sommers, Curtin, & Newman, 2011; Newman et al., 2010) have used a threat-potentiated startle paradigm in which subjects were informed a priori about what the threat contingency was. It is therefore unclear whether the attentional manipulation had an influence on top-down representation of the threat or on the Pavlovian conditioning that may have occurred during the experimental block. That is, during the experimental block, subjects may have started to learn that a red square signaled threat because they were repeatedly shocked when a red square was presented. As such, from the perspective of the IES, an important line of further research for RMH can be distilled. Attention may moderate Pavlovian conditioning, which relies on intact amygdala functioning, but it may also affect CS-affect representations, which constitutes a different class of amygdala-based associative learning. At this point, both mechanisms are likely intertwined, making it difficult to elucidate on which mechanism attention has an influence.

RMH versus dual-process theory. According to Patrick & Bernat's (2009) dual-process theory, Factor 1 is related to normal higher order cognitive processes and a major problem in core defensive reactivity. Factor 2 is related to normal defensive reactivity and a failure to process complex environmental cues, including aversive cues (Patrick in Hervé & Yuille, 2007). This account of psychopathy would predict that normalizing threat processing deficits, for instance, by focusing attention on the cue that indicates the threat contingency (e.g., Newman et al., 2010), would interact with Factor 2. However, Newman et al. (2010) showed that better processing of environmental threat cues interacts with Factor 1, whereas Baskin-Sommers, Curtin, & Newman (2011) showed that it interacts with both factors. The models therefore seem to diverge on the predictions about the relationships between the threat system and the psychopathy factors.

IES versus dual-process model. The dual-process model is based on the idea that disinhibition associated with Factor 1 is related to a normal appetitive system, but a deficient defensive system. As such, the dual-process model differs from the IES model in that it assumes the existence of a unitary fear system, without differentiation for the processing of different types of threat and the conscious experience of fear. In addition, in our view, it also posits a rather unitary view of the approach or appetitive system. The IES framework argues against this, stating that both aversive and appetitive associative learning are impaired in psychopathy, but that the case may be more severe for aversive associative learning. A recent study (Gregory et al., 2015) found that reward-based learning is also affected in psychopathy. Therefore, the dual-process model cannot accommodate recent findings regarding deficient reward learning in psychopathy, whereas IES does make predictions about this possibility.

Future Directions: Hidden Confounds

The main aim of this study was to review studies addressing fear in psychopathy and to assess whether it is threat processing rather than the experience of fear that is impaired in psychopathy. One intriguing conclusion of our review and meta-analysis is that the subjective experience of fear does not appear to be compromised in psychopathy. Although the neurobiological evidence would not necessarily contradict impaired subjective experience of fear in psychopathy, empirical evidence does indicate that it is relatively intact in psychopathy. Importantly, studies targeting the conscious subjective experience of emotions commonly rely on self-report measures (e.g., Phan et al., 2004). One confound that is often overlooked in studies on the conscious experience of fear is that sociocultural factors shape the expression of fear, as cultures differ in when and to what extent emotions are allowed to be expressed (Butler, Lee, & Gross, 2007; Elfenbein & Ambady, 2002). Importantly, these environmental limitations and expectations also have an impact on neurobiology. For example, there are findings indicating that amygdala activation increases after viewing fearful faces portrayed by members of the participant's own culture relative to those from another culture, thus showing that culture modulates the processing of fearful faces (Chiao et al., 2008). Studies on the impact of environmental variables on the neurobiological mechanisms involved in the expression of threat in humans are relatively scarce, but evidence from animal research might provide some indications. For instance, maternal behavior in the first postnatal week influences the development of the central nervous system in rats. More specifically, pups of doe rats showing high amounts of licking, grooming, and arched-back nursing during the first postnatal week exhibited less fearful behavior in adulthood. This diminished behavioral expression of fear was

related to altered sensitivity of tissue in the CeN, partly triggered by the increased mother–pup interactions (Caldji et al., 1998). The latter points out that the mechanisms for fear experience and threat processing may overlap to some extent. Another study found that increased maternal interaction reduced fearful behavior in a strain of rats known to be excessively fearful (Anisman, Zaharia, Meaney, & Merali, 1998). With regard to conscious experience of fear in humans, studies also point toward environmental differences in the experience and expression of fear. There are cultural differences in recognizing emotional facial and vocal expressions (Gendron, Roberson, van der Vyver, & Barrett, 2014a, 2014b). In addition, using a standard fear survey schedule, cross-cultural differences in the numbers, content, pattern, and level of fear have been observed (Ollendick, Yang, King, Dong, & Akande, 1996). Together, these results highlight the fact that the social environment has profound effects on neurobiological development, the behavioral expression, and subjective experience of fear. Further assessment of the interactions between neurobiology and environmental factors may have great explanatory value for the deficiencies seen in psychopathy.

A second important feature concerns the influence of the relativity of the subjective experience of emotions. To illustrate, happiness has been argued to be a process of hedonic adaptation (Lykken & Tellegen, 1996). That is, the temporal stability of positive affect in humans may oscillate around a certain individual homeostatic baseline. Lifetime events will influence the experience of happiness, but people may tend to get back to their individual baseline. The crucial point here is the potential existence of a baseline. In psychopathy, the baseline of the subjective experience of fear may be lower than in nonpsychopathic individuals: They may consider minor deviations from this baseline as a full fearful experience, or vice versa. Thus, on questionnaires to assess fear, psychopathic individuals will use a different "internal ruler" that reflects their own range of emotionality, which may not be comparable with that of nonpsychopathic individuals. As an analogy, although the absolute deviation from this baseline may be smaller in psychopathy, the relative change may be similar to nonpsychopathic individuals. In addition, psychopathic individuals have a low tolerance for frustration that may lead them to appraise such a deviation in a negative manner, which would account for relatively normal appraisals of the negative valence of fear. Note also that the accumulation of additional sensory information to compensate for a lower baseline is consistent with findings showing that directing attention to peripheral information can make startle deficits disappear in psychopathy. In summary, the influence of environmental factors on subjective experience of fear and potential differences in individual baseline for experiencing emotions may be some of the confounds that have received little attention in psychopathy research and could form a source of heterogeneity within the current body of evidence.

As was outlined earlier, Newman and colleagues (2010) have shown that attention moderates potentiated startle responses, possibly via top-down upregulation of amygdala activity (Larson et al., 2013). An interesting option is that this technique may also affect the subjective experience of fear in psychopathy. That is, top-down deployment of attention may affect brain areas involved in threat responsivity, which could potentially affect the subjective experience. In addition, the present review highlights the need for fine-grained dissection of the threat circuitry in psychopathy. It is important to realize that, given the current indications that multiple structures included in the threat circuitry show abnormalities in psychopathy, functional impairments could arise at multiple stages of processing. Measures such as the startle response or blood-oxygen-level dependent signal during conditioning reflect the end result of this processing, thus occluding the source of the functional disturbance. Therefore, a certain amount of care is needed when making claims about functional causality. One interesting future avenue would be to employ formal computational models to directly quantify the computations carried out by this circuit in individuals with psychopathy. Such models could be used to estimate variables known to affect learning in general, such as the rate at which learning occurs (i.e., learning rate) and the amount of uncertainty in the available information during threat conditioning (Stephan & Mathys, 2014). This would allow the further specification as well as the quantification of the cognitive computations carried out by the network components during threat conditioning (for a similar notion, see, e.g., Maia & Frank, 2011).

The Consideration of Novel Theoretical Frameworks

Our final suggestion for future research is that there may be great gains to be made by employing and systematically studying predictions based on theoretical frameworks that have not been prominent in research on psychopathy (Brazil, 2015). For instance, the periaquaductal gray (PAG) and ventromedial prefrontal cortex have been argued to play key roles in threat processing based on the distance between the organism and the threat (Blair, 2006). That is, when a threat is distal, other brain structures are activated than when threat is proximal. Threat detection at close range involves the ACC and medial orbitofrontal cortex (Mobbs et al., 2007). When a threat is distant, the ventromedial prefrontal cortex becomes active, possibly suggesting the weighting of alternative behavioral responses. More imminent threats, however, shift activation to the PAG, which is involved in freeze, flight, and fight behaviors. There are currently no studies that have looked at brain activations in individuals with psychopathy in response to proximal versus distal threats, nor are there studies that directly indicate abnormalities in the PAG in psychopathic individuals in this context.

Another example is a framework that has recently been proposed in which the prelimbic (PL) regions of the prefrontal cortex mediate the expression of threat responses (for more details, see Sotres-Bayon & Quirk, 2010). The PL is reciprocally connected to the BA, and activations in these two areas covary such that involvement of the PL appears necessary for the expression of threat responses. Sotres-Bayon and Quirk (2010) propose the existence of a BA-centered network that is capable of switching between states of high and low threat reactivity. Within this network, the prefrontal cortex receives input from other structures, such as the hippocampus and orbitofrontal cortex, integrates this information, and relays to output to the BA, which in turn regulates threat responses in the medial portion of the CeN. With respect to psychopathy, impaired threat responsivity could thus be a consequence of functional disturbances in the prefrontal cortex, the BA, and/or the CeN and their mutual interactions. Such an account would also be consistent with recent findings pointing toward reduced functional connectivity between the amygdala and prefrontal cortex in psychopathy (Motzkin et al., 2011) and altered

(neural) flow of information in psychopathy (Yang et al., 2012). From this perspective, one tentative hypothesis would be that impairments in this circuit could lead to an inability to switch between high and low threat modes, and therefore individuals with psychopathy would be neurobiologically predisposed to remain "stuck" in the low threat mode.

Limitations: Highlighting the Need for Precision

It may be argued that the proposed model does not include all aspects of fear and that the categorization of the physiological outcome measures included at the lower level (freeze, startle, etc.) is somewhat arbitrary. Admittedly, there may be other ways of categorizing these automatic reactions. More importantly, we do not believe that the classification at this level affects the overarching distinction between automatic threat reactivity and conscious experience of fear. Nor is it likely that a somewhat different model would negate the presently observed findings. Another limitation is the lack of sufficient studies to reliably assess the relationships between psychopathy-related personality dimensions, threat, and the experience of emotions. This scarcity highlights the need to further assess the potential explanatory role of the dimensions in the future. However, one foreseeable problem is that there are currently multiple theoretical frameworks about psychopathy as a personality construct that differ in the nature and content of the dimensions (Cooke & Michie, 1999; Hare, 2003; Lilienfeld & Andrews, 1996; Lynam & Widiger, 2001; Patrick et al., 2009). Another complicating factor is that psychopathy studies in offender populations often employ group comparisons, but differ in how the groups are defined. In contrast, studies conducted in nonoffender populations are often correlational. Thus, although diversity certainly has many advantages, it also makes the integration of the findings across frameworks increasingly challenging. One solution would be to employ designs that allow both group comparisons and correlation analyses in offender samples (Anderson & Kiehl, 2012), and, if possible, multiple measures of psychopathy that differ in their operationalization of the dimensions (e.g., self-report psychopathy list vs. the triarchic measure of psychopathy).

Another issue is that even though we have tried to dissociate fear and anxiety based on different theoretical angles, in some studies, it remains extremely difficult to characterize the exact nature of the construct that is being measured. This also may have had an impact on our analysis of threat processing, as it can be difficult to determine whether the paradigm used in some studies taps into threat processing in relation to fear or anxiety. For instance, some of the studies employed a countdown paradigm in which an aversive stimulus was presented when the target count was reached (e.g., Hare et al., 1978). Anticipatory autonomic responses could be regarded as anxiety-based responses, given their prolonged nature, but the response to the aversive stimulus itself can be seen as a fear-based response because it is short-lived. Although the predictability of the shock would argue for a fear response (as we noted in the introduction), the potential influence of subjective processes on the fear response cannot be excluded. As such, the outcome measures used to describe automatic processing may, in some cases, be contaminated with the conscious components of fear. Because of this issue and the modest number of studies included in the meta-analysis, we opted not to exclude such studies. Nonetheless, dissociating between anxiety-driven and fear-driven autonomic processing is essential in order to advance our understanding of the exact impairments that characterize psychopathy. Related to the issue of mislabeling, our meta-analyses show that many studies claiming to have measured fear in fact seem to have quantified the engagement of the defensive threat mechanism and not the subjective component of fear. As such, future studies are advised to clearly define what is being measured. Whether the focus lies on associative threat learning, threat detection, threat anticipation, the identification of fear as an emotion, or the subjective experience of the negative valence of fear, it is crucial to use the correct terminology. Although challenging, the distinctions described in our model should still be accounted for in future studies.

General Summary and Conclusions

In this review, we strictly defined fear and endeavored to parse fear into various components. We assessed the importance of fear for historical conceptualizations of psychopathy. This step was pivotal, as the definition of the construct is likely to determine the extent to which empirical evidence corroborates the notion of fearlessness in psychopathy. The results indicate that earlier conceptualizations of psychopathy do not explicitly include fear as a feature of psychopathy. Patrick and colleagues' (2009) influential dual-process model and the triarchic model are, however, very much in line with the concept of fear as we use it at the present time. Subsequently, a conceptual model was formulated in which fear was parsed based on recent neuroscientific insights, in which the conscious experience of fear and automatic threat processing were disentangled. This framework was used to examine the current neurobiological and empirical evidence on fear deficits in psychopathy from a different perspective. The findings suggest that psychopathic individuals have deficiencies in threat detection and responsivity (with a medium effect size), both at the behavioral and neurobiological levels. In contrast, they seem to be able to consciously experience fear as an emotion, which contradicts the general notion that lack of fear is a core component of psychopathy. The general evaluation of the low-fear hypothesis may therefore have been too superficial, which likely stems from the faulty assumptions that fear is a unitary construct, and that various measures of fear are all equally indicative of a general fear deficit in psychopathy.

To assess how selective the intact experience of fear was, we conducted a second meta-analysis for the subjective experience of other emotions. We found that psychopathy is related to reduced experience of happiness, but increased anger. Reduced experience of happiness is consistent with results of previous meta-analyses on a related topic (Dawel et al., 2012; Marsh & Blair, 2008), whereas increased anger concords with earlier findings that psychopathy is related to angry hostility. The small reliability of the effect sizes for these emotions (i.e., fail-safe numbers below the amount of studies included) do not allow for strong conclusions, but it does appear to be the case that intact experience of fear does not occur in isolation. In psychopathy, sadness and surprise may also be experienced relatively normally.

From a neurobiological perspective, there is some evidence for impairments in crucial cortical nodes underlying the subjective experience of fear, but these impairments arise in various emo-

tional tasks that are not specifically tailored toward measuring the subjective experience of emotions such as fear. There are, however, two important factors to consider. Given the deficiencies in threat detection and responsivity, and concomitant reduced physiological reactivity, it is possible that these deficiencies indirectly lead to less intense subjective experiences. It may be the case that focusing attention on threatening cues may normalize threat detection and responsivity and, by inference, the subjective experience of fear. Second, as it is particularly difficult to measure subjective experiences, and only questionnaires are currently available to index these, there are important environmental factors that make the interpretation of such questionnaires difficult. Thus, not only do empirical studies indicate equivocal evidence for a difference between healthy and psychopathic individuals in the subjective experience of fear—there are also important obstacles in measuring this subjective experience that need to be overcome before any conclusive tests can be applied. Although most historical conceptualizations do not include fear as a feature of psychopathy, it is important to note that current notions of psychopathy (e.g., PCL-R) do delineate a patient population that has impairments in various components of fear, mainly threat detection and responsivity. One may speculate that the same holds for earlier conceptualizations.

Taken together, this review highlights the importance of evaluating evidence in light of novel or alternative theoretical frameworks such as the one proposed here. By increasing conceptual precision, even ideas that have become established may prove to be less well founded than is believed. Importantly, although we have applied this model to psychopathy, research in other psychiatric disorders may also benefit from using such an approach. Empirical work in patient populations suffering from conditions related to pathological levels of fear, such as posttraumatic stress disorder or phobias, could employ a similar approach and categorize different studies according to the various subcomponents of fear to obtain a more nuanced perspective of the differing roles of fear and threat processing in these disorders. The latter could have important implications not only for empirical research but also for clinical practice, as targeting an over- or hypoactive threat circuit versus atypical experience of fear will likely require different therapeutic approaches.

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Received July 11, 2014
Revision received October 15, 2015
Accepted November 6, 2015 ■