Psychopathy is essentially a disorder of decision making. For decades researchers have aimed to identify the psychobiological mechanisms that underlie the psychopath’s profound decision-making impairment. Still, a comprehensive answer to a simple question (Why do psychopaths do the things they do?) remains elusive. The implications of a veritable answer to this question are far-reaching, including how criminals are evaluated, sentenced, and potentially rehabilitated. In this chapter, we (1) discuss clinical and research evidence that justifies our conceptualization of psychopathy as a disorder of decision making; (2) characterize the attentional abnormalities associated with psychopathy to highlight a distinction between active and passive decision making and the implications of this distinction for culpability; (3) selectively review evidence from neuroscientific studies for the purpose of evaluating the relevance of different neurobiological models of psychopathy; and (4) conclude with a brief discussion of the potential implications of our review for legal considerations in psychopathy.

PSYCHOPATHY AS A DECISION-MAKING DISORDER

One of the earliest and most compelling depictions of psychopathic behavior was conveyed through the accumulation of narrative case descriptions in Hervey Cleckley’s book *The Mask of Sanity* (Cleckley, 1941, 1976). Over the multiple editions of this text, Cleckley assimilated his clinical experiences with scores of psychopathic cases to distill the essential features of the disorder. As a testament to the acumen of his observations, many of the core elements of psychopathy he described decades ago are incorporated into the current standard for evaluating psychopathy. With respect to the decision-making capability of psychopaths, Cleckley emphasized a blatant and enduring impairment:

To say the least, the pattern of [the psychopath’s] actions over any fairly long range of time indicates little that the observer can understand as what a human being would consciously choose. (pp. 261–262)

Despite his excellent rational powers, the psychopath continues to show the most execrable judgment about attaining what one might presume to be his ends... This exercise of execrable judgment is not particularly modified by experience, however chastening his experiences may be... It is my opinion that no punishment is likely to make the psychopath change his ways. (pp. 345–346)

A number of the laboratory paradigms that have been used to distinguish the performance of psychopathic and nonpsychopathic individuals highlight this decision-making deficit. Building on the clinical descriptions of psychopathy, researchers modeled the apparent insensitivity to punishment and impaired decision making (i.e., behavioral choices) in a laboratory paradigm known as “passive-avoidance learning.”
In one version of this task, the subject attempts to complete a mental maze through a series of choice points. At each choice point the subject has four possible choices. Only one choice is correct (leading to advancement in the maze), while one of the three error choices is associated with an additional punishment (e.g., painful electric shock). Multiple studies demonstrate that compared to nonpsychopaths, who show preferential avoidance of the punished (shocked) choice, psychopaths are more likely to commit the punished choice, a so-called error in passive avoidance (Lykken, 1957; Schmauk, 1970).

These results lend themselves to at least two possible interpretations. One interpretation holds that the psychopaths' passive-avoidance errors arise from a fundamental deficit in the generation of emotion. If the punishment does not elicit a subjectively aversive feeling, or if the aversion tied to past punishment experiences is not evoked during the contemplation of a subsequent action in a similar context, then the psychopath may not exhibit learning on the basis of punishment. A second interpretation is that the psychopath may simply be oblivious to the association between certain choices and their associated consequences. In other words, psychopaths may rigidly attend to learning the correct choices in the maze (as per task instructions) and fail to process (and/or link with their responses) the incidental punishments that are not directly germane to their instructed goal. Indeed, these differing interpretations for the passive-avoidance data can be extrapolated to the psychopaths’ decision making more generally. At this level, the question can be posed succinctly: Is the psychopath's decision-making impairment due primarily to a deficit in the generation of emotion or primarily to a deficit in the allocation of attention? Cleckley seemed to acknowledge both possibilities as explanations for his clinical observations. At one point he proposed that "...despite [the psychopath's] otherwise perfect functioning, the major emotional accompaniments are absent or so attenuated as to count for little" (p. 371). At another point he proposed that "...[the psychopath's] difference from the whole or normal or integrated personality consists of an unawareness and a persistent lack of ability to become aware of what the most important experiences of life mean to others" (p. 371). The former quote seems to suggest a primary deficit of emotional processing, whereas the latter seems to implicate processes specifically related to attention and awareness.

These two competing explanations have spawned a wealth of theoretical and empirical work. In clinical and real-world settings, it can be difficult, if not impossible, to discern the relative validity of these different interpretations. However, it is often possible to parse these causal factors in laboratory experiments. In the following sections, we consider two lines of research on the psychobiological mechanisms of psychopathic decision making: one highlighting the role of emotion and the other highlighting the role of attention. We conclude this section with a brief integration of the findings.

**Psychopathic Decision Making as a Deficit of Emotion**

One of the earliest and most influential theoretical accounts of the primacy of an emotional deficit in psychopathy was formalized by David Lykken in his "low-fear hypothesis" (Lykken, 1957, 1995), which proposes simply that a deficit in the generation of fear may underlie psychopathic behavior. This idea has clear appeal in its parsimony and experimental tractability. Initial laboratory support for this proposal included the passive-avoidance data described previously, as well as a host of studies employing galvanic skin response (GSR) as an index of autonomic physiological arousal. In these studies, GSR is taken as an objective proxy for the experience of fear or anxiety (though some have argued that it is more accurately regarded as an index of arousal) (e.g., Patrick, Bradley, & Lang, 1993). In normal (nonpsychopathic) subjects, the repeated pairing of an emotionally neutral stimulus (e.g., an auditory tone) with an emotionally aversive stimulus (e.g., electric shock) leads to a state of "conditioned fear," in which the presentation of the previously neutral stimulus alone is sufficient to elicit a fear response (as measured with GSR). Psychopaths, however, exhibit abnormally low GSR to the conditioned stimulus (Hare & Quinn, 1971; Lykken, 1957). In subsequent years,
psychophysiological recording techniques have been used to document attenuated autonomic reactivity in psychopaths for a variety of experimental conditions, including passive-avoidance learning (Schmuck, 1970), hearing loud noises (Hare, 1978), anticipation of an aversive stimulus such as loud noise or shock (Hare, Frazelle, & Cox, 1978; Ogloff & Wong, 1990; Tharp, Maltzman, Syndulko, & Ziskind, 1980), viewing scenes of distress (Blair, Jones, Clark, & Smith, 1997), and imagining fearful situations (Patrick, Cuthbert, & Lang, 1994).

Although prolific from an experimental standpoint, the low-fear hypothesis has clear limitations in explaining the full spectrum of psychopathic traits. In the domain of emotions alone, psychopaths exhibit conspicuously diminished guilt, shame, embarrassment, empathy, and love, among others (Cleckley, 1976; Eilenfeld & Arkowitz, 2007). It seems implausible that these various manifestations of restricted affect are reducible to a root deficiency in fear. Furthermore, the lack of a measurable physiological response in psychopaths could conceivably be due to a failure to appropriately engage or attend the threatening or aversive stimuli, a possibility that is discussed at greater length in the text that follows.

A more comprehensive account of the role of emotion in decision making has been proposed by Antonio Damasio in his “somatic marker hypothesis” (Damasio, 1994). The central idea of this hypothesis is that physiological processes, including those that constitute emotion, may act as signals to influence behavior. More specifically, through experience humans develop associations between various situations and the corresponding somatic states (i.e., emotions). The recurrence of a particular situation triggers the reactivation of neural patterns depicting the associated emotion, which marks potential outcomes as good or bad. The poor judgment and decision making of psychopaths could be seen as a consequence of weak somatic markers due to the underlying defect in emotional reactivity. This hypothesis thus posits a causal relationship between emotion and decision making, without necessarily stipulating the preeminence of any particular emotion, such as fear.

Psychopathic Decision Making as a Deficit of Attention

As previously mentioned, it is possible that the poor decision making and even the conspicuously blunted affect associated with psychopathy could arise from abnormalities in the allocation of attention. This hypothesis is consistent with early clinical descriptions of psychopaths, which note a lack of “the active, searching attention and organizing process that normally puts [relevant] information to use” (Shapiro, 1965, p. 149), as well as with psychopaths’ own statements: “I always know damn well I shouldn’t do these things...it’s just that when the time comes I don’t think of anything else. I don’t think of anything but what I want now.” (Grant, 1965). These descriptions suggest an inability to flexibly reallocate attention away from a dominant goal.

Experimental evidence for this type of attentional defect in psychopathy can be found in the early passive-avoidance learning studies. In the initial such study (Lykken, 1957), the instructed goal of the task was to complete the maze successfully; thus learning the one correct choice at each choice point was the “manifest” task, whereas learning to avoid the shocked incorrect choice was the “latent” task (in that it was not directly related to the goal of the task). Interestingly, psychopathic subjects performed normally on the “manifest” task despite their insensitivity to the “latent” task. In fact, unlike nonpsychopathic subjects, the majority of psychopathic subjects did not report any awareness that the shocks were contingent upon any particular incorrect choice (Schmuck, 1970). In a modified version of the passive-avoidance task, subjects were endowed with a sum of money at the start of the task, and one of the errors was punished with a loss of money rather than shock. Importantly, subjects were allowed to keep the money at the end of the test, and therefore learning the punished error was arguably an explicit goal of the test (i.e., a “manifest” task). In this condition, psychopaths demonstrated both good awareness of the shock contingency and normal passive-avoidance learning (Schmuck, 1970).

The task dependence of the psychopaths’ defect in learning from punishment was further
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demonstrated in a go/no-go discrimination learning task (Newman & Kosson, 1986). In one condition of this task, correct responses were rewarded with monetary gain and incorrect responses were punished with monetary loss. A second condition featured only punishment to incorrect responses. Psychopaths made more commission errors (responding to the “no-go” stimuli) than nonpsychopaths in the reward + punishment condition, but exhibited normal performance in the punishment-only condition. In a related study of decision making, subjects selected cards from a deck, with each card indicating a gain or loss of money (Newman, Patterson, & Kosson, 1987). The subject was free to stop playing the cards at any time and collect whatever money he had accumulated. The key experimental manipulation was that initially the cards were almost always rewarding (and hence established a dominant response set), but as the task progressed the cards became increasingly more punishing. When no restrictions were placed on the subjects’ responding, the psychopaths played more cards and won less money than nonpsychopaths. However, when subjects were able to view their cumulative net earnings and also required to pause briefly before choices, psychopaths performed normally. Taken together, these data suggest that the psychopath’s inability to inhibit a punished behavior depends critically on the context of the situation, which argues against a pervasive insensitivity to punishment. More generally, the psychopath’s decision making may reflect a deficit in the natural human tendency to reflect on the various consequences of previous actions. In a subsequent study, Newman and colleagues addressed this issue directly (Newman, Patterson, Howland, & Nichols, 1990). Once again using a go/no-go test with reward and punishment, they found that following a monetary loss (punishment), psychopaths did not pause as long as nonpsychopaths before initiating their next choice. Importantly, the duration of the post-punishment pause was shown to be predictive of learning to avoid the punished response.

Integrating the aforementioned clinical and experimental observations, Newman and colleagues developed a theoretical framework that highlights the role of attention in psychopathic decision making: the “response modulation hypothesis” (Newman & Lorenz, 2003; Patterson & Newman, 1993). The essence of response modulation is the “temporary suspension of a dominant response set and a brief concurrent shift of attention from the organization and implementation of goal-directed responding to its evaluation” (Patterson & Newman, 1993, p. 717). In the context of decision making, this involves attending to the remote or secondary consequences of one’s actions as well as to immediate or primary considerations to guide decision making. In recent years, Newman and colleagues have tested the role of attention in decision making among psychopaths in various ways. Here we summarize a number of the key studies.

One study investigated psychopaths’ capacity for attentional focus using Stroop tests. In the classic color-word Stroop test, the subject sees a color word (e.g., “red”) with the letters printed in a different color (e.g., green). Confronted with a written word, the natural or “pre-potent” impulse is to read the word. However, in the Stroop test the subject’s task is to name the color of the letters. Longer reaction times are interpreted as greater interference of the distractor task (reading the word) on the instructed task (naming the color). Psychopaths exhibit normal interference effects in the color-word Stroop test (Hiatt, Schmitt, & Newman, 2004; Smith, Arnett, & Newman, 1992). In a modified version of the test, the picture-word Stroop, the subject sees an object word (e.g., “chair”) superimposed on a picture outline of another object (e.g., table). The subject’s task is to name the picture while ignoring the word, and again, longer reaction times are interpreted as greater interference. Unlike nonpsychopaths, psychopaths were insensitive to interference in this variant of the task (Hiatt et al., 2004; Newman, Schmitt, & Voss, 1997). In another modification of the test, the spatially segregated color-word Stroop test, the subject sees a color word (e.g., “red”) printed in white letters and surrounded by a rectangular frame of a different color (e.g., green). The subject’s task is to name the color of the frame while ignoring the word. Again, the psychopaths showed reduced interference effects (Hiatt et al.,
These results demonstrate that psychopaths’ attention is relatively impervious to distraction when the task-irrelevant information is spatially distinct from the deliberately attended goal-relevant stimuli. However, when the irrelevant information is spatially integrated within the attended stimuli the psychopaths perform normally. These results point to a dysfunction in attending/processing certain types of contextual information. Importantly, the behavioral differences documented in these studies occur for emotionally neutral stimuli, indicating that psychopaths’ defects are not restricted to the affective domain.

A second study examined whether this apparent defect in the integration of contextual information is also evident in psychopaths’ memory function (Glass & Newman, 2009). This study consisted of three memory tasks. In each memory task, subjects saw a series of words (some neutral, some emotionally arousing) presented one at a time and were instructed to remember each word as it appeared. Each of the three tests involved an additional piece of contextual information: word location, color of a rectangular frame around the word, or color of the font. Subjects were later tested on their ability to recall the words as well as the associated contextual information. As predicted by the response modulation hypothesis, psychopaths and nonpsychopaths exhibited similar performance on the word recall task (both groups demonstrated memory bias for the emotional words), but unlike nonpsychopaths, who also demonstrated memory bias for the contextual information associated with the emotional words, psychopaths exhibited no such bias for the contextual information. These data indicate a specific impairment of integrating contextual information in memory, rather than a global impairment in memory for emotionally salient information.

A third study investigated whether psychopaths’ physiological responses of fear are also dependent on attentional focus (Newman, Curtin, Bertsch, & Baskin-Sommers, 2010). In this study, subjects underwent a fear conditioning paradigm in which red letters were sometimes associated with electric shocks, whereas green letters were never associated with shocks. Attentional focus was manipulated with one of three concomitant behavioral tasks: indicating the color of the letter, indicating whether the letter was lowercase or uppercase, or indicating whether or not the letter matched the letter that appeared two letters back. Thus the color identification task requires the subject to attend to the property of the stimulus that predicts the painful stimulus (“threat-focus”), whereas the other two tasks require the subject to attend to other aspects of the stimulus (“alternative-focus”). Startle responses to sudden bursts of loud noise were measured as electrical activity in the facial muscles mediating eyeblinks. As predicted by the response modulation hypothesis, psychopaths exhibited normal startle responses in the threat-focus condition, but significantly diminished startle responses in the alternative-focus condition. These data demonstrate that focus of attention is a critical factor in determining the psychopaths’ physiological fear response (see also Arnett, Smith, & Newman, 1997).

Overall, the results presented in this section argue for the primacy of an attention-related defect in psychopathy. The evidence suggests that psychopaths are not globally impaired in generating fear responses or in fear-related learning, but rather that the emotional hyporesponsiveness and impaired decision making are consequences of the psychopaths’ abnormal deployment of attention. This conclusion is further supported by the studies revealing attention-related defects for certain types of nonaffective information as well. In the following section we elaborate on the relationship between these cognitive deficits and the concept of intentionality.

**SPECIFYING THE COGNITIVE DEFICIT IN PSYCHOPATHY: IMPLICATIONS FOR INTENTIONALITY**

The decision-making perspective on psychopathy highlights the potential importance of multiple information-processing stages. Decisions vary in complexity and, as a result, vary in the range of information processing skills required. In general, however, decision making will be affected by the quality of a person’s perception
(i.e., attention to all relevant stimuli), the accessibility of relevant memories and prior learning, and the integration of all relevant considerations during response selection. Similarly, poor decision making may reflect problems in perception, memory, and/or response selection.

An important question highlighted by this information processing perspective on decision making concerns the extent to which psychopaths’ poor decision making reflects deliberate and callous choices as opposed to a deficit in information processing that systematically prevents consideration of all relevant information. Writing about a similar consideration, Shapiro (1965) wrote “conscience and moral values are not elemental psychological faculties, but involve and depend on a number of cognitive and affective functions” (p. 163). According to this view, differences in moral conduct “are not primarily matters of moral scruple on the part of the normal person or the lack of them on the part of the psychopathic character; they are matters of interest and automatic cognitive tendency” (p. 166).

“In the normal person, the whim or the half-formed inclination to do something is the beginning of a complex process, although, if all is well, it is a smooth and automatic one” (p. 140). The process entails integrating current experience or whims with preexisting values and provides a perspective on behavior that goes beyond one’s immediate concerns. In the absence of this perspective, it is difficult to develop long-term goals or resist impulses, so that a person’s thoughts and goals tend to shift erratically. Moreover, in providing a context for the person’s commitment to a course of action, the process of integrating current motivations with more stable goals enhances one’s ability to tolerate frustration, endure boredom, and accept responsibility for one’s behavioral choices. By contrast, psychopaths’ difficulty integrating current whims with past experience interferes with their ability to appreciate the emotional and moral significance of events as well as their ability to objectify their own behavior and exercise critical judgment. In the following section we consider experimental evidence that informs the question of whether the psychopaths’ decision-making deficit reflects automatic or deliberate processes.

Early versus Late Selective Attention Deficits in Psychopathy

An important distinction in attention research pertains to early- versus late-selective attention. Early selection typically reflects a perceptual bottleneck and results in an unintentional failure to process all relevant information. Conversely, late selection is typically intentional and involves the application of capacity limited resources to focus one’s attention in a particular direction while ignoring “goal-irrelevant” information. The standard color-word Stroop task described previously is a classic example. Even though reading words is “pre-potent,” late selection may be used to attend preferentially to the color of the stimulus and ignore the color word. Factors that impair cognitive capacity (e.g., alcohol consumption) will also impair the consistent application of late-section (Curtin & Fairchild, 2003), resulting in the disinhibited expression of the pre-potent but incorrect response. Early selection is generally thought to reflect limitations in perception that limit the processing of distracting peripheral information. In contrast to late selection, secondary information is not suppressed using limited capacity resources. Rather, such information simply receives less attention and, thus, has minimal impact on behavior regardless of limitations on cognitive processing resources (Arnett et al., 1997; Bishop, Jenkins, & Lawrence, 2007; Lavie, Hirst, de Fockert, & Viding, 2004).

The relevance of the early and late selection distinction for psychopathy concerns its implications for judging whether psychopaths deliberately ignore behaviorally relevant information post-perception (late selection) or fail to perceive behaviorally relevant information unintentionally (early selection). Although the consequences for self-regulation will generally be the same, the distinction may have important implications for their culpability. Two recent studies from the Newman laboratory are germane.

The first study employed a modified version of the Eriksen Flanker task. In a flanker task, participants are instructed to focus on a character that appears in the “target location” and make one of two responses depending upon the category of the target (e.g., letter vs. number). The task
typically employs three conditions: congruent trials in which the target and distracters belong to the same category; incongruent trials in which the target and distracters belong to the opposite categories; and control trials in which the targets are paired with a neutral stimulus (e.g., *). The principal dependent measure is interference, which is calculated by subtracting response times for control trials from response times for incongruent trials. This measure is useful for quantifying the extent to which conflicting peripheral information (distracters) modulates a person’s response to the goal-relevant cues (targets).

Zeier, Maxwell, and Newman (2009) modified the standard flanker task to evaluate the effects of early versus late selection on response incongruent distracters (Zeier et al., 2009). In this task, targets were either a letter (H, G) or a number (5, 8) and were presented to the left or right of a central arrow (i.e., > or <) along with one other letter, number, or neutral stimulus (*). The direction of the arrow indicated the location of the target stimulus. For example, G < 5 would be a letter trial. To address the early versus late selection issue, each display was preceded by a cue display that either directed attention to the location of the target or directed attention to the location of both the target and distracter. In the exogenous cuing condition, for instance, an open square appeared at the location for 100 milliseconds before the target display and was placed so that the eventual target would fill the square. This cue elicits an involuntary attention response prior to presenting the target/distracter display. In the opposing condition, two squares appear for 100 milliseconds so that both the target and distracter locations are highlighted. The single cue condition facilitates early selection of the target location whereas the double cue procedure facilitates processing of the distracter as well as the target and thus increases the demand for late selection.

The results of Zeier et al. (2009) showed that psychopathic offenders displayed significantly less interference than nonpsychopathic offenders in the single-cue condition, whereas they displayed at least as much interference as nonpsychopathic controls in the double-cue condition. These findings show that psychopaths and controls are equally sensitive to information that conflicts with their goal-directed behavior once it has been perceived but that they are insensitive to the identical information if their attention is already focused on goal-relevant stimuli. By this account, their obliviousness to peripheral information appears to be involuntary (i.e., relatively automatic) rather than deliberate.

The second study evaluated sensitivity to fear-related distracters rather than response conflict but yields a similar conclusion. Following up on the fear conditioning study described previously (Newman et al., 2010), Baskin-Sommers and colleagues (2011) examined fear-potentiated startle either before or after the presentation of goal-relevant cues. The results of this study showed that deficits in fear-potentiated startle to threat cues were specific to the condition that presented threat cues after an alternative focus had been established. Thus, as in the Zeier et al. study, psychopaths appear to be normally responsive to secondary information unless their attention is already focused on goal-relevant stimuli. Such findings strongly suggest that their failure to inhibit inappropriate responses or weaker responses to peripheral emotion cues in laboratory studies is an unintentional consequence of an attentional abnormality that restricts processing of secondary information once their attention is engaged in goal-relevant processing. Analogously, it would seem to follow that psychopaths would be impaired in their ability to process all relevant information and exercise good decision making in real-world contexts once focused on achieving an immediate goal.

To this point, our brief review suggests that psychopathy may be usefully understood as a deficit in decision making and that this deficit is mediated (at least in part) by dysfunction in the allocation of attention that starts at an early stage of the information-processing stream and appears to be involuntary. More generally, the results offer empirical support for a circumscribed cognitive or psychological basis for psychopathic behavior. A separate but related question is whether there is evidence for a neurobiological basis for psychopathy.
IS THERE A NEUROLOGICAL BASIS FOR PSYCHOPATHY?

Of central importance for the present discussion is whether psychopathic behavior can be attributed directly to an organic biological defect. If a neurobiological substrate for psychopathy could indeed be identified and ultimately treated through clinical intervention (pharmacologic, neurosurgical, or otherwise), it would seem to have significant implications for how psychopaths are handled by our legal system. Here we consider neuroscientific evidence related to two brain areas that have been theorized as central components of the neural substrate for psychopathy: the amygdala and ventromedial prefrontal cortex.

As previously discussed, much attention in psychopathy research has been paid to the psychopaths' experience (or non-experience) of fear—recall the "low-fear hypothesis" (Lykken, 1957, 1995). If a lack of fear is indeed a contributing factor underlying psychopathy, there is ample reason to suspect that psychopathy would be associated with dysfunction within the amygdala, an almond-shaped subcortical structure within the anterior temporal lobe. A wealth of neuroscientific data implicates the amygdala in fear-related processing. The amygdala is necessary for the acquisition of conditioned fear responses (Bechara et al., 1995; LaBar, LeDoux, Spencer, & Phelps, 1995) as well as the recognition of facial expressions of fear (Adolphs, Tranel, Damasio, & Damasio, 1994) and the allocation of attention to fear-related information (Adolphs et al., 2005). In recent years, brain imaging techniques such as positron emission tomography (PET) and magnetic resonance imaging (MRI) have allowed researchers to determine whether psychopaths exhibit abnormalities in the amygdala in terms of structure or function. One structural brain imaging study reports lower amygdala volumes in psychopaths (Yang, Raine, Narr, Colletti, & Toga, 2009). Among the functional imaging studies that compare psychopaths with nonpsychopaths, three report abnormalities in amygdala activity, one finding abnormally low levels of activity during a word identification test (Kiehl et al., 2001), one finding abnormally low levels of activity during a fear conditioning task (Birbaumer et al., 2005), and the other finding abnormally high levels of activity in response to positive emotion pictures (Muller et al., 2003). A related pair of studies have demonstrated that greater levels of "psychopathic" traits among the normal population are associated with lower levels of amygdala activity during socioaffective tasks such as moral judgment (Glenn, Raine, & Schug, 2009) and social cooperation (Rilling et al., 2007). However, viewing fearful facial expressions, which reliably elicits amygdala activation in normal subjects (Adolphs, 2002), did not elicit an abnormally diminished amygdala response in psychopaths (Deeley et al., 2006). One caveat for interpreting these brain imaging results is that neuroimaging data are inherently correlational; brain imaging data alone cannot address whether any observed neural abnormality is a cause or consequence of the disorder. Taken together, these studies offer some intriguing preliminary data relating amygdala dysfunction to psychopathy, but as of yet the data do not seem to provide unequivocal support for a hypo-responsive amygdala as the primary neurobiological basis of psychopathy.

A second candidate brain region for the neuropathological basis of psychopathy is the ventromedial prefrontal cortex (vmPFC). The putative connection between vmPFC dysfunction and psychopathy has long been recognized in the field of behavioral neurology. In 1975, Blumer and Benson coined the term "psychopathy" to summarize the personality changes ("the lack of adult tact and restraints") observed in their vmPFC-damaged patients (Blumer & Benson, 1975). A decade later, Damasio began a series of clinical and laboratory evaluations of such patients that would offer novel insight into the neural mechanisms of emotion and decision making. Damasio and colleagues' case descriptions of vmPFC patients typically noted the following "psychopathic" traits: lack of empathy and guilt, generally blunted affect, poor long-term planning, irresponsibility, marked lack of insight or concern, and defective decision making despite seemingly intact intellect (Anderson, Barrash, Bechara, & Tranel, 2006; Barrash, Tranel, & Anderson, 2000; Eslinger &
Damasio, 1985). Expanding on these clinical similarities, a number of laboratory paradigms have demonstrated parallel deficits between psychopaths and vmPFC lesion patients. Examples include reversal learning (Budhani, Richell, & Blair, 2006; Hornak et al., 2004), gambling tasks (Bechara, Damasio, Tranel, & Damasio, 1997; Mitchell, Colledge, Leonard, & Blair, 2002) (but see also Losel & Schmucker, 2004; Schmitt, Brinkley, & Newman, 1999), smell identification (Jones-Gotman & Zatorre, 1988; Lapiere, Braun, & Hodgins, 1995), and autonomic physiological responses to emotional stimuli (Blair et al., 1997; Damasio, Tranel, & Damasio, 1990; Patrick et al., 1994). These intriguing similarities hint that vmPFC dysfunction may contribute to psychopathic behavior. However, one notable difference is that the clinical reports of vmPFC patients do not typically feature criminal or violent behavior to the same degree as in psychopaths. An important consideration in this regard is the age at lesion onset in the vmPFC lesion patients. The majority of vmPFC patients described in the literature suffered their brain damage in middle age or later adulthood from a medical condition such as stroke, tumor, or aneurysm. This means that adult-onset vmPFC lesion patients had “normal” psychosocial development through childhood and early adulthood that perhaps mitigates their antisocial behavior following the lesion. A valuable source of data to inform this issue is individuals who suffered vmPFC damage very early in life (i.e., before age 2). Only a handful of such cases have been reported (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Anderson, Wisonowski, Barrash, Damasio, & Tranel, 2009), but the results clearly indicate that early vmPFC damage leads to a pattern of behavior through adolescence and young adulthood that is even more reminiscent of psychopathy. Unlike the adult-onset vmPFC lesion patients, the early-onset patients exhibit stereotypical “psychopathic” antisocial behaviors such as petty theft, physical assaults, sexual promiscuity, and chronic lying. Taken together, the data from lesion patients indirectly support developmental dysfunction within vmPFC as a putative neurobiological mechanism of psychopathy.

As a noteworthy aside, we point out that the “somatic marker hypothesis” (Damasio, 1994) (described earlier in the chapter), which figures prominently in psychopathy research (Losel & Schmucker, 2004; Schmitt et al., 1999; van Honk, Hermans, Putman, Montagne, & Schutter, 2002), was actually developed to account for the decision-making impairments observed in vmPFC patients.

While initial studies on the neurobiology of psychopathy focused largely on individual brain structures with well-established roles in social-affective processing (e.g., amygdala and vmPFC), more recent neuroimaging work has associated psychopathy with reduced connectivity between more widely distributed networks of brain areas (Ly et al., 2012; Philippi et al., in review). Interestingly, the comparatively weak functional connections identified in psychopaths in these studies correspond to cortical networks that have been implicated in shifting or maintaining attentional state (Dosenbach et al., 2007; Fox et al., 2005; Raichle et al., 2001). Hence, this emerging line of neuroimaging research, which highlights the coordinated activity within large-scale cortical networks as the neural basis for attention and information integration, suggests a putative neurobiological mechanism for the observed abnormalities in attentional processing in psychopathy.

In sum, the results of neuroscientific studies suggest intriguing neuropathophysiological models of psychopathy, but at present there are insufficient data to conclude with certainty that psychopathy arises as a direct consequence of dysfunction in a particular area (or areas) of the brain. However, the field of cognitive neuroscience has been making rapid progress in identifying brain-based mechanisms underlying information processing and decision-making competence. We are optimistic that continuous advancements in technological and theoretical precision will ultimately reveal a biological basis for psychopathy.

In particular, one area that we believe warrants more precise and rigorous investigation is the distinction between “primary” and “secondary” psychopaths. This distinction reflects the long-theorized possibility that the extreme affective and behavioral traits that characterize the disorder could arise through different causal
mechanisms. In other words, psychopaths may consist of phenotypically similar, but etiologically distinct subtypes (Lykken, 1957, 1995). In the "primary" subtype, psychopathy is presumed to arise directly from some fundamental intrinsic deficit, likely involving innate dysfunction in basic affective and/or attentional mechanisms. By contrast, "secondary" psychopathy is thought to arise as an acquired disturbance of social and affective processing—an indirect consequence of environmental or psychosocial factors such as parental abuse, socioeconomic disadvantage, poor intellect, substance abuse, or neurotic anxiety (Blackburn, Logan, Donnelly, & Renwick, 2008; Cleckley, 1976; Karpman, 1946, 1948; Lykken, 1995; Skeem, Johansson, Andershed, Kerr, & Louden, 2007). Clearly this theoretical distinction could have profound implications for research on the psychobiological basis of the disorder.

If there are indeed multiple, distinct causal mechanisms for psychopathy, then one may expect the different etiological subtypes to exhibit distinct psychological and neurobiological profiles within the context of similarly flagrant antisocial behaviors. The question, then, is how to differentiate primary psychopaths from secondary psychopaths for the purposes of research. A number of previous studies have differentiated primary and secondary psychopaths based on levels of trait anxiety (Arnett et al., 1997; Blackburn, 1975; Brinkley, Newman, Widiger, & Lyman, 2004; Fagan & Lira, 1980; Hiatt et al., 2004). This practice is supported by ample theoretical and empirical work. In his seminal clinical descriptions, Cleckley stressed the importance of considering anxiety levels for the classification of psychopathy: "...primary psychopaths are sharply characterized by the lack of anxiety...I do not believe that [primary] psychopaths should be identified with the psychoneurotic group" (Cleckley, 1976, p. 257). Following Cleckley's recommendation of distinguishing low-anxiety individuals from those with high (neurotic) levels of anxiety, a large and growing number of laboratory studies demonstrate abnormal behavioral results for low-anxious (primary) psychopaths but not necessarily for high-anxious (secondary) psychopaths (Arnett, Howland, Smith, & Newman, 1993; Arnett et al., 1997; Chesno & Kilmann, 1975; Fagan & Lira, 1980; Lykken, 1957; Newman, Kosson, & Patterson, 1992; Newman et al., 1990, 1997; O'Brien & Frick, 1996; Schmitt et al., 1999; Skeem et al., 2007; Smith et al., 1992; Zeier et al., 2009). Despite this substantial literature, none of the recent brain imaging studies on psychopathy has differentiated subjects on the basis of anxiety. Assuming that the low- and high-anxiety psychopathy subtypes do indeed correspond to etiologically distinct conditions with unique psychological and neurobiological profiles, then the combination of the two subtypes in a single group of psychopaths for the purposes of a research study may result in muddled and inconsistent results. Thus we believe that the primary/secondary distinction is an important, but too often overlooked, consideration for investigating the biological basis of psychopathy.

**SUMMARY AND CONCLUSION**

The aim of this chapter is to outline the current state of knowledge on the psychological and neurobiological mechanisms that underlie psychopathy so that legal experts may incorporate this information as they craft policy to best ensure public safety and welfare. We assert that it is reasonable and instructive to conceptualize psychopathy broadly as a disorder of decision making and to consider the specific deficits that may contribute to the overall decision-making impairment. To this end, we have reviewed theoretical and empirical work suggesting that diminished emotional reactivity and a defect in the flexible allocation of attention are likely critical factors. We propose that psychopathy may entail a primary deficit in attention, as an attentional defect could theoretically account for the observed abnormalities in processing both affective and nonaffective information. Importantly, the attentional deficit in psychopathy appears to operate at an early, relatively automatic stage of information processing, suggesting that psychopathic behavior may reflect a lack of decision-making competence rather than a deliberate intention to harm others. To make this conclusion more definitively, future research will need to be carefully designed to parse and specify the information-processing capability of psychopaths.
Regarding the brain mechanisms of psychopathy, there are multiple neural structures that have been proposed as likely candidates, notably the amygdala and vmPFC. Both brain areas have been linked to psychopathy, albeit largely through indirect evidence at this point. Although there is currently no clear consensus on the biological root of the disorder, technological advances in brain imaging have clearly made the pursuit of a neural basis for psychopathy a tractable field of inquiry. As the science progresses, we expect such data to more frequently arise in court cases, specifically with respect to questions of culpability, likelihood of future offense, and prospects for rehabilitation. To establish the appropriate framework for incorporating psychopathy research into the legal system, legal scholars will have to consult closely with scientists as they further elucidate the psychological and neurobiological mechanisms of psychopathy.

REFERENCES


