Brain Imaging in Psychopathy
Julia R. Lushing, Lyn M. Gaudet, and Kent A. Kiehl

The use of functional magnetic resonance imaging (fMRI) techniques to study mental health and disease began in the 1990s. A relative newcomer in the history of mental health research, functional neuroimaging has nevertheless made a significant impact in dozens of disciplines in a short amount of time. Researchers in every major mental health and developmental field—from normal adolescent development to schizophrenia to autism to memory—use neuroimaging to advance their understanding of their condition of interest, specifically by identifying the neural mechanisms at work. The field of psychopathy has seen a rapid growth in the use of neuroimaging to understand the condition. Neuroimaging work spans studies relating self-reported psychopathic traits in undergraduate populations to clinical forensic populations using the Hare Psychopathy Checklist—Revised. This chapter will be limited to studies of individuals with clinical levels of psychopathy, defined as a score of 30 or greater on Hare’s PCL-R (Hare, 2003).

Individuals with clinical levels of psychopathy frequently come into contact with the criminal justice system. In addition, psychopathy is associated with high levels of violent and sexually violent recidivism (e.g., Hare & Vertommen, 2003; Harris, Rice, & Cormier, 1991). Accordingly, this chapter includes a brief section on the legal relevance of the condition, taking into account a summary of recent research on the neurobiological evidence supporting diagnosis of psychopathy and its effect on legal decision-making in mock trial settings. The chapter concludes with an example of when neuroimaging evidence was presented in corroboration of a clinical assessment of psychopathy in a capital sentencing hearing.

Assessing Psychopathy in Forensic Contexts and Imaging Research

The gold standard for diagnosing psychopathy is the Hare PCL-R (Hare, 2003). Raters are professionally trained to conduct an extensive semi-structured interview and a collateral file review that entails a review of criminal files, school files, employment files, and any other available corroborating documents. Although the PCL-R has both dimensional and categorical properties, meaning one can exhibit psychopathic traits without meeting the diagnostic threshold for psychopathy, the PCL-R manual designates scores of 30 and above as indicating clinical levels of psychopathy (Hare, 2003).

If a psychopathy score is needed in a forensic context, such as for an adversarial legal proceeding or risk assessment, the PCL-R is the recommended assessment method, as it has demonstrated...
reliability and validity in identifying psychopathic traits. It is also the assessment method of choice in imaging studies, because, if one is trying to link imaging data to a psychological test instrument, the neuroscience data are only going to be as robust as the assessment tool one uses to quantify the construct of interest. In other words, the more accurate the psychological measurement, the more robust the relationship to neuroscience data. For further discussion of psychopathy assessment, see Chapters 12–16.

Models of Psychopathy

Research on the neurological origins of psychopathy has led to two prominent models of the condition, proposed by Blair and Kiehl, respectively (Blair, 2007a; Kiehl, 2006). Blair and Kiehl both hypothesize pathological function and structure in specific emotional processing areas of the brain. Blair’s model argues that psychopathy-related symptoms arise from dysfunction of the amygdala and orbital frontal cortex. Kiehl’s model includes the amygdala and orbital frontal cortex, but also includes additional paralimbic regions, including the anterior superior temporal gyrus (temporal pole) and anterior and posterior cingulate cortex. Another notable theory of psychopathy is Newman’s response modulation hypothesis. Newman argues that psychopathy is fundamentally a disorder of attention and was originally based on animal models of septal lesions (Newman & Lorenz, 2003). This model is discussed in more detail in Chapter 4.

Blair Model

Blair (2007b) posited that dysfunction in two brain regions is primarily constitutive of psychopathy: the ventromedial prefrontal cortex (also known as the orbital frontal cortex) and the amygdala.

The amygdalae are two almond-shaped structures located deep within the left and right medial temporal lobes of the brain. The amygdala has many functions, including amplifying salient stimuli (e.g., fearful face expressions) and stimulus-reinforcement learning (LeDoux, 1998). The basolateral nucleus of the amygdala receives sensory input from potential conditioned stimuli, which allows associations between conditioned and unconditioned stimuli to develop (Campeau & Davis, 1995). These associations are then transmitted via the central nucleus of the amygdala to the hypothalamus and brainstem, which dictate behavioral, autonomic, and neuroendocrine responses (Campeau & Davis, 1995). Research has shown that psychopaths present with a diminished capacity to process emotional information (e.g., fearful faces) and fail to form normal stimulus-reinforcement associations (Blair, 2007b). In particular, much research has investigated aversive stimuli processing in psychopaths (see for example Hare, 1972; Hare, Frazelle, & Cox, 1978; Müller et al., 2003; Patrick, 1994). For example, Kosson et al. found that psychopaths were less accurate in identifying facial expressions than non-psychopaths (Kosson et al., 2002). Marsh et al. showed reduced amygdala activation in response to fearful faces in children and adolescents with callous and unemotional traits, which can be precursors to psychopathy (Marsh et al., 2008). These findings suggest that psychopaths have specific deficits in processing nonverbal emotional cues, which may contribute to their apparent disregard for the feelings of others.

A second line of research involving amygdala dysfunction in psychopathic subjects relates to the consequences of emotional distractors on attention. In a typical population, one would expect to see modulations in reaction time to a stimulus based on the presence of emotional distractors (Dolcos & McCarthy, 2006). Healthy subjects have faster response times when no emotional distractors are present (Dolcos & McCarthy, 2006). Imaging studies confirm increased activation in the amygdala and ventrolateral prefrontal cortex during tasks when emotional distractors are present (Dolcos & McCarthy, 2006). Moreover, response times tend to be slower in the presence of emotional distractors (Yamasaki, 2002). These findings suggest that emotional processing interferes with cognitive processing in normal populations. Similar tests conducted on the psychopathic population...
showed that emotional distracters do not interfere with attention to nonemotional cues in short-term recollection tasks (Mitchell, Richell, Leonard, & Blair, 2006). This suggests emotional processing deficits in psychopaths can be linked to dysfunctional amygdala.

Another study employed a lexical decision task to investigate emotional processing in psychopaths (Lorenz & Newman, 2002). During lexical decision tasks, participants are required to determine whether a letter string forms a word or not (Meyer & Schvaneveldt, 1971). Prior studies show that emotionally valenced words are processed faster and more accurately in lexical decision tasks than neutral words (Kanske & Kotz, 2007). Specifically, positively and negatively valenced words are processed faster than neutral words (Kanske & Kotz, 2007). Psychopaths did not show the expected facilitation in response times to emotional relative to nonemotional words compared with non-psychopaths (Lorenz & Newman, 2002; Williamson, Harpur, & Hare, 1991). These findings suggest that processing of emotional content in language is impaired in psychopaths. In the first fMRI study of criminal psychopathy, Kiehl et al. (2001) found that the amygdala failed to show normal engagement during processing of emotional words in psychopaths compared with non-psychopaths. In sum, behavioral and neuroimaging data support the notion that psychopaths “know the words, but not the music” (Blair et al., 2006).

In addition to the amygdala, the ventromedial prefrontal cortex (vmPFC) has been a focus in psychopathy research since early lesion studies documented that patients with vmPFC injuries developed psychopathic traits (Bechara, Tranel, & Damasio, 2000). The vmPFC is implicated in emotional regulation (Phillips, 2003) and contributes to decision-making processes by representing value information (Blair, 2007b). Both functions are, in part, believed to be dependent on amygdala activity that feeds forward reinforcement–expectancy information to the vmPFC (Schoenbaum & Roesch, 2005). This connectivity is crucial to behavioral extinction and reversal learning (Budhani, Marsh, Pine, & Blair, 2007). Psychopathic populations show a marked deficiency in both reversal learning (Mitchell et al., 2006) and behavioral extinction (Newman, Patterson, & Kosson, 1987). Specifically, psychopaths are more likely than controls to revert to a previously rewarded response when it is no longer a “correct” response (Budhani, Richell, & Blair, 2006).

These findings may explain why psychopaths have a general disregard for the well-being of others. Normal humans experience complex interactions between cognitive and emotional parts of the brain that ultimately influence moral behavior. For example, a normal person not only knows that murder is wrong, but also experiences negative emotions associated with the act. Although psychopaths tend to have intact cognitive abilities and normal IQs, there is critical disruption in the communication between emotions and cognition that may explain their pervasive and consistent immoral behavior.

The key points of the Blair model of psychopathy are as follows:

- Amygdala and ventromedial prefrontal cortex abnormalities are constitutive of psychopathy.
- These brain regions are important for the communication between “feeling” and “thinking” centers of the brain.
- Psychopaths can “think” normally, but do not integrate “feelings” appropriately in decision-making.

Paralimbic Hypothesis of Psychopathy

The paralimbic hypothesis asserts that psychopathy is related to aberrant structure and function of the core limbic and surrounding paralimbic regions (Kiehl, 2006). This model is based on cytoarchitectural organization of the brain and was originally developed, in part, in extensive studies of the behavioral changes associated with acquired brain injuries that resulted in psychopathic traits (for detailed review, see Kiehl, 2006, 2014).
Studies of patients with damage to the bilateral orbitofrontal cortex (i.e. brain tissue just above the eyes) led researchers to coin the terms “pseudopsychopathy” (Blumer & Benson, 1975) and “acquired sociopathic personality” (Tranel, 1994). Symptoms associated with bilateral orbital frontal damage include: disturbances in reactive aggression, impulsivity, irresponsibility, behavioral inhibition (Malloy, Bihrl, Duffy, & Cimino, 1993; Stuss et al., 1983), and, in some cases, grandiosity (Blumer & Benson, 1975). However, the symptomology associated with orbital frontal damage cannot wholly explain the constellation of symptoms associated with psychopathy. For example, orbital frontal patients infrequently display instrumental aggression or callousness, both key features of psychopathy (Blair, 2001; Hare & Hart, 1993). Interestingly, two case reports of orbital frontal brain damage early in life were associated with elevated aggression levels later in life (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999). These findings suggest a strong developmental course to some symptoms of psychopathy (Anderson, Barrash, Bechara, & Tranel, 2006).

Lesion studies of the anterior cingulate may also provide clues to the neurobiology of psychopathy (Kiehl, 2006). The anterior cingulate is a central brain structure commonly divided into two parts: the rostral and caudal divisions (Devinsky, Morrell, & Vogt, 1995). The rostral anterior cingulate is involved in pain perception and emotional regulation (Bush, Luu, & Posner, 2000), whereas the caudal anterior cingulate is implicated in response conflict, error monitoring, and task switching (Kiehl, Liddle, & Hopfinger, 2000). Isolated anterior cingulate damage is associated with lack of concern with others’ emotions (Mesulam, 2000), aggression, disagreeableness, and irresponsible behaviors (Devinsky et al., 1995). Additional research has shown a positive correlation between right anterior cingulate volume and harm avoidance behaviors (Pujo et al., 2002). Psychopaths notoriously score low on harm avoidance tasks (Hare, Hart, & Harpur, 1991). Accordingly, aberrations in the anterior cingulate may account for the traits and behaviors associated with the affective and lifestyle factors of psychopathy. These traits include: lack of remorse, callousness, impulsivity, and stimulation seeking (Hare & Neumann, 2005).

In addition to the aforementioned regions of the brain, the temporal lobes may also be connected to certain symptoms of psychopathy (Kiehl, 2006). Klüver–Bucy syndrome was first documented in monkeys with medial temporal lobe lesions and is characterized by inappropriate fear processing, hyperactivity, and disinhibited or hypersexuality (Klüver & Bucy, 1938, 1939). Additionally, early studies of personality problems in patients with temporal lobe epilepsy indicate high levels of psychopathic behaviors (Blumer & Benson, 1975; Hill, Pond, Mitchell, & Falconer, 1957). Temporal lobe epilepsy is typically associated with problems in the hippocampus, parahippocampal gyrus, amygdala, and anterior superior temporal gyrus (Kiehl, 2006). Elective surgeries to remove both the amygdala and anterior temporal lobes have, generally, resulted in reduced violence and aggression (Bagshaw et al., 1972; Hill et al., 1957; Lee et al., 1988, 1998).

Furthermore, the amygdala, a medial temporal lobe structure and part of the limbic circuit, is implicated in determining the emotional salience of linguistic stimuli (Anderson & Phelps, 2001; Funayama, Grillon, Davis, & Phelps, 2001; Isenberg et al., 1999; Strange, Henson, Friston, & Dolan, 2000) and aversive conditioning (Bechara, et al., 1995; Funayama et al., 2001; LaBar, LeDoux, Spencer, & Phelps, 1995). Psychopaths do not show typical facilitation in processing emotional words in affective lexical decisions tasks (Williamson et al., 1991). Moreover, psychopaths show deficits in the processing of distressful faces (Blair, Jones, Clark, & Smith, 1997), which is thought to rely on amygdala function (Blair, 1995). The temporal lobe, including the amygdala, may therefore account for traits and behaviors associated with the affective, lifestyle, and antisocial facets of psychopathy. To date, several structural and functional imaging studies corroborate this hypothesis and are discussed in greater detail in their respective sections of this chapter (see Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012, 2013).

The paralimbic hypothesis suggests that there is a developmental course to psychopathy that has pervasive effects on the paralimbic and limbic structures in the brain (Kiehl, 2006). The convergence
evidence from early neuroimaging and lesion studies indicates that these parts of the brain are critical for the social and emotional behaviors that are disrupted in psychopathy.

The key points of the paralimbic hypothesis of psychopathy are as follows:

- Psychopathy is the result of structural and functional abnormalities in the paralimbic structures of the brain.
- This suggests that psychopathy has a developmental course.

Response Modulation Hypothesis

The concept of response modulation first arose from the literature on dysfunctional septal-limbic systems in animals (e.g., McCleary, 1966; Newman et al., 1987). Response modulation is the suspension of a dominant response in reaction to negative or unexpected stimuli (Newman & Lorenz, 2003). Deficits in response modulation are often associated with poor behavioral controls, failure to learn from corrective feedback, and failure to reflect on response strategies (Newman & Lorenz, 2003).

Newman and Wallace argue that there is more than one pathway to deficient response modulation (Newman & Wallace, 1993). In one path, individuals demonstrate a generalized response modulation deficiency apparent while engaged in any goal-directed behaviors (see Bernstein, Newman, Wallace, & Luh, 2000). Other individuals specifically show response modulation deficits during periods of high emotional arousal (Wallace & Newman, 1997; Wallace, Newman, & Bachorwski, 1991). In both instances, however, individuals with modulation response deficits are less likely to consider relevant secondary stimuli and inhibit responses accordingly (Newman & Lorenz, 2003).

One of the most salient characteristics of psychopathic behavior, described by Hervey Cleckley in *The Mask of Sanity*, is the psychopath’s apparent insensitivity to punishment (Cleckley, 1941/1976). Researchers eventually modeled this particular decision-making impairment in an experimental paradigm known as “passive-avoidance learning,” which involves choosing among four stimuli, where three result in punishment (e.g., painful electric shock), and one results in reward (e.g., advancement in the maze). Psychopaths, compared with non-psychopaths, were more likely to choose the punished choice (Lykken, 1957; Schmauk, 1970). Newman argues that these results lend themselves to two hypotheses: (1.) psychopaths have a primary deficit in the production of emotion (e.g., psychopaths do not experience the aversive emotion associated with punishment, or (2.) psychopaths fail to associate aversive emotional responses with the punishment (Pujara, Motzkin, Newman, Kiehl, & Koenigs, 2013).

The key points from the response modulation hypothesis are as follows:

- Psychopathic emotional processing deficits are largely restricted to the automatic (attentional) integration of emotional and nonemotional cues.
- Although response modulation is critical to understanding impulsivity (Wallace et al., 1991), a key feature of psychopathy, it may not account for the affective traits of psychopathy, e.g., callousness, lack of empathy, and failure to take responsibility etc.

Functional Neuroimaging

Modern imaging techniques have allowed researchers to investigate the neural networks associated with psychopathic traits. Functional MRI, which measures changes in the magnetic properties of oxygen levels in the blood, is one of those techniques. Functional MRI relies on the premise that activity in specific parts of the brain requires relative increases in blood flow to the area, thereby causing changes in the relative oxygenation of blood during performance of some experimental task. Measuring these changes in oxygenation is referred to as blood oxygenation level-dependent
(BOLD) contrast. BOLD data can be subjected to statistical tests, with the results then superimposed on structural MRIs to provide neuroanatomical maps of where the brain activity took place. BOLD-generated brain images can provide unsurpassed spatial resolution (less than a cubic millimeter). However, BOLD signal changes typically occur slowly, on the order of seconds, and thus the fMRI technique has limited temporal resolution relative to electrophysiological techniques. Carefully designed fMRI tasks can allow for meaningful measurements of how brain activity in specific regions corresponds with discreet cognitive and emotional functions, and how those systems are impaired in psychopathology in general, and psychopathy in particular.

In sum:
- fMRI measures blood flow in the brain, which indicates which parts of the brain are activated during certain tasks.
- fMRI can tell us where in the brain activity is taking place better than any other technology, but still lacks temporal specificity.

Empathy

A hallmark symptom of psychopathy is lack of empathy. Empathy is typically operationalized as the ability to affectively experience the inferred emotional states of others. Empathy is critical to interpersonal relationships and social cooperation (Decety, 2007).

An important research question has always concerned the nature of the empathic abnormalities characteristic of psychopaths and uncovering their associated neural networks. Decety, Chen, Harenski, and Kiehl (2013) and Decety, Skelly, and Kiehl (2013) examined brain activation patterns of incarcerated psychopaths and non-psychopaths while exposing them to stimuli depicting people experiencing pain. Psychopaths, relative to non-psychopaths, showed significantly less activation in the ventromedial prefrontal and orbitofrontal cortices, as well as the periaqueductal gray matter. Interestingly, insula activation was positively correlated with PCL-R scores (Decety, Chen et al., 2013; Decety, Skelly et al., 2013). Based on the extensive research correlating insula activation with empathy for pain (Lamm, Decety, & Singer, 2011), it is surprising that psychopaths would show increased activation in this area, given the psychopath’s seeming disregard for other people’s pain. One interpretation of this finding is that the insula is implicated in cognitive assessments, as opposed to affective processing, of pain (Decety, Chen et al., 2013; Decety, Skelly et al., 2013). Psychopaths do not have the normal limbic input to process the emotional components of pain and they overly rely on cognitive systems when processing empathy stimuli.

Additional fMRI studies suggest that empathy deficits in psychopaths stem from developmental abnormalities in the amygdala, a brain structure associated with salience detection (Decety & Moriguchi, 2007). Psychopaths show processing deficits with respect to fearful and sad faces, while showing no abnormalities in connection with displays of positive emotions (Blair, 2001). This hypothesis is corroborated by the fact that psychopaths perform normally on “Theory of Mind” tasks. This suggests that psychopaths are capable of taking the cognitive perspectives of others, but lack the affective processes associated with empathy (Hare & Hart, 1993). In other words, psychopaths have an intellectual understanding of what is physical and emotional pain and can even identify it in others. However, psychopaths apparently do not have the visceral experience of other people’s pain, which is evidently what actually motivates moral behavior. This distinction is particularly relevant in the legal context, in which insanity defenses require that the person not appreciate that their actions are wrong. This point is discussed further later in the chapter.

Psychopathy and Moral Decision-Making

A key feature of psychopathy is “moral insensitivity,” in terms of both the psychopath’s willingness to commit moral transgressions and the lack of guilt or remorse from doing so (Harenski, Harenski,
Table 3.1 Summary of fMRI Studies in Psychopathy.

<table>
<thead>
<tr>
<th>First Author</th>
<th>Psychopathy Assessment</th>
<th>Comparison Groups</th>
<th>Type of Analysis/Task</th>
<th>Results (Psychopaths vs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birbaumer et al. (2005)</td>
<td>PCL-R, (M = 24.89,)</td>
<td>(N = 10) healthy controls</td>
<td>fMRI, aversive differential Pavlovian delay conditioning paradigm</td>
<td>No activity in limbic-prefrontal circuit (amygdala, OFC, anterior cingulate, insula) during aversive conditioning</td>
</tr>
<tr>
<td>Contreras-Rodriguez et al. (2014)</td>
<td>PCL-R ((M = 27.8, SD = 4.5),)</td>
<td>(N = 22) controls</td>
<td>fMRI, emotional face-matching task</td>
<td>↑ Activation in visual and prefrontal areas; latter associated with Factor 1; † functional connectivity between lt. amygdala and visual and prefrontal cortices</td>
</tr>
<tr>
<td>Cope et al. (2014)</td>
<td>PCL-R used as continuous measure, (N = 44) male inmates; (N = 93) female inmates</td>
<td>N/A</td>
<td>fMRI, drug-related stimuli, craving ratings</td>
<td>↓ ACC, PCC, hippocampus, amygdala, caudate, globus pallidus, and parts of the PFC while viewing drug cues. Results specific to Factor 2, Facet 3, &amp; Facet 4</td>
</tr>
<tr>
<td>Decety, Skelly et al. (2013)</td>
<td>PCL-R ≥ 30, (N = 37) inmates</td>
<td>(N = 44) medium psychopathy inmates ((21–29); N = 40) low psychopathy inmates ((&lt; 210))</td>
<td>fMRI, viewing painful and nonpainful stimuli while adopting self-perspective or other-perspective</td>
<td>In self-perspective condition, dysfunction in emotional pain network, including insula, anterior midcingulate cortex, SMA, IFG, somatosensory cortex, and rt. amygdala. When imaging others, atypical activity in insula, amygdala, OFC, and vmPFC. Results driven by Factor 1 scores</td>
</tr>
<tr>
<td>Decety, Skelly et al. (2013)</td>
<td>PCL-R ≥ 30, (N = 27) inmates</td>
<td>(N = 28) low psychopathy ((&lt; 21 \text{ PCL-R})) and (N = 25) medium psychopathy ((21–29 \text{ PCL-R}))</td>
<td>fMRI, pain interactions and pain expression task</td>
<td>↓ vmPFC, lateral OFC, and PAQ; † insula. Deficits in vmPFC and OFC regardless of stimulus type. Impairment in regions associated with mentalizing while processing facial cues</td>
</tr>
<tr>
<td>Decety (2014)</td>
<td>PCL-R ≥ 30, (N = 27) inmates</td>
<td>(N = 28) low psychopathy ((&lt; 21 \text{ PCL-R})) and (N = 25) medium psychopathy ((21–29 \text{ PCL-R}))</td>
<td>fMRI, viewing emotional stimuli and asked to identify gender</td>
<td>↓ IFG, OFC, and vmPFC in response to all four categories of facial expression; † insula to fear, sadness, and pain</td>
</tr>
<tr>
<td>Deeley et al. (2006)</td>
<td>PCL-R &gt; 25, (N = 9) forensic mental health patients</td>
<td>(N = 9) healthy controls</td>
<td>fMRI, viewing fear or happy faces with two intensities; identify gender</td>
<td>↓ Fusiform and extrastriate cortices when processing both facial emotions</td>
</tr>
<tr>
<td>Harenski et al. (2010)</td>
<td>PCL-R ≥ 30, (N = 16) inmates</td>
<td>PCL-R &lt; 30, (N = 56)</td>
<td>fMRI, moral processing task</td>
<td>During moral violations, ↓ ventromedial PFC and anterior temporal. Strength of positive association between amygdala and severity ratings reduced. Strength of negative association between posterior temporal activity and severity ratings increased</td>
</tr>
<tr>
<td>Harenski et al. (2014a)</td>
<td>PCL-YV used as continuous measure, (N = 119) detained youth</td>
<td>N/A</td>
<td>fMRI, moral processing task</td>
<td>While viewing unpleasant pictures with and without moral transgressions, † amygdala</td>
</tr>
<tr>
<td>Authors</td>
<td>PCL-R Used As</td>
<td>N =</td>
<td>Task/Condition</td>
<td>Note</td>
</tr>
<tr>
<td>------------------</td>
<td>---------------</td>
<td>-----</td>
<td>-------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>Harenski et al.</td>
<td>PCL-R used as continuous measures, N = 164 inmates</td>
<td>46</td>
<td>fMRI, moral processing task</td>
<td>↓ Rt. amygdala and rACC during emotion contrast; ↓ rt. TPJ during moral contrast</td>
</tr>
<tr>
<td>Juárez et al.</td>
<td>PCL-R ≥ 30, N = 17 inmates</td>
<td>37</td>
<td>fMRI/ICA, auditory oddball task</td>
<td>Frontoparietal and visual/posterior cingulate network correlated with Factor 2 and total scores. Factor 1 correlated with DMN. No evidence of aberrant PCC function</td>
</tr>
<tr>
<td>Kiehl et al.</td>
<td>PCL-R, M = 32.8, SD = 2.9, N = 8 inmates</td>
<td>8</td>
<td>fMRI, affective memory task</td>
<td>↓ Amygdala/hippocampal formation, parahippocampal gyrus, ventral striatum, and in the anterior and posterior cingulate gyri; ↑ bilateral fronto-temporal cortex for processing affective stimuli</td>
</tr>
<tr>
<td>Kiehl et al.</td>
<td>PCL-R, M = 32.8, SD = 2.9, N = 8 inmates</td>
<td>8</td>
<td>fMRI, lexical decision task</td>
<td>↓ Behavioral performance for processing abstract words. Aberrant neural differentiation between abstract and concrete stimuli rt. anterior temporal gyrus and surrounding cortex</td>
</tr>
<tr>
<td>Larson et al.</td>
<td>PCL-R, M = 31.3, SD = 2.2, N = 24 inmates</td>
<td>8</td>
<td>fMRI, fear-potentiated startle</td>
<td>↓ Amygdala only when attention was engaged in goal-relevant task prior to presenting threat-relevant information; ↑ selective-attention regions of the LPFC, which mediated decreased amygdala activation. When explicitly attending to threat, amygdala activation did not differ</td>
</tr>
<tr>
<td>Meffert et al.</td>
<td>PCL-R ≥ 26, N = 18 forensic psychiatric patients</td>
<td>26</td>
<td>fMRI, empathy task (watched hands while experiencing similar interactions)</td>
<td>Temporal, insular, parietal, and frontal lobes</td>
</tr>
<tr>
<td>Müller et al.</td>
<td>PCL-R ≥ 30, N = 6 high-security psychiatric patients</td>
<td>6</td>
<td>fMRI, instructed to feel the emotions IAPS pictures presented</td>
<td>↑ Prefrontal regions and amygdala during negative emotion condition; ↓ rt. subgenual cingulate and the temporal gyrus, and lt. dACC and parahippocampal gyrus; ↑ lt. OFC regions during positive emotion condition; ↓ medial frontal and medial temporal regions</td>
</tr>
<tr>
<td>Müller et al.</td>
<td>PCL-R &gt; 28, N = 10 forensic psychiatric patients</td>
<td>10</td>
<td>fMRI, Simon task</td>
<td>Aberrant PFC and temporal brain activation. Disturbed superior temporal gyrus</td>
</tr>
<tr>
<td>Pujara et al.</td>
<td>PCL-R ≥ 30, N = 18 female inmates</td>
<td>18</td>
<td>fMRI, passive gain or loss of money</td>
<td>↑ Lt. ventral striatum correlated with total PCL-R score among psychopaths only</td>
</tr>
<tr>
<td>Sommer et al.</td>
<td>PCL-R &gt; 28, N = 14 forensic psychiatric patients</td>
<td>14</td>
<td>fMRI, presented cartoons where character's intention was fulfilled or not</td>
<td>No increase in emotion attribution in mirror neuron systems; ↑ outcome monitoring and attention, such as OFC, medial frontal cortex, and temporal-parietal areas</td>
</tr>
</tbody>
</table>

Note: All results are described as levels of brain activity in psychopaths compared to the indicated comparison group(s).
Accordingly, Harenski et al. examined moral decision-making among incarcerated psychopaths and non-psychopaths and their neural substrates. A set of moral, immoral, and amoral images were selected and shown to participants. Participants were asked to determine whether the picture depicted a moral violation and to rate the severity of the moral violation on a scale of 1–5. Results showed that psychopaths and non-psychopaths had similar determinations and ratings of moral and non-moral violations. However, analysis of fMRI data indicated significant differences in neural activity during moral decision-making between psychopaths and non-psychopaths. Psychopaths showed reduced activity in the ventromedial prefrontal cortex and anterior temporal cortex during moral/nonmoral determinations compared with non-psychopaths. Additionally, a positive association was found between amygdala activity and severity ratings of moral transgressions that was stronger in non-psychopaths than in psychopaths. Finally, a negative association between posterior temporal activation and severity ratings was stronger in psychopaths than non-psychopaths (Harenski et al., 2010).

These results provide solid evidence that psychopaths show reduced activity in areas that have been previously associated with moral decision-making (e.g., the amygdala; Blair, 2007a; Kiehl, 2006), and psychopaths potentially rely on different brain regions when rating the severity of moral transgressions more than non-psychopaths (e.g., the posterior temporal area) (Harenski et al., 2010).

A salient point here is that psychopaths showed similar determinations and ratings as non-psychopaths, while relying on different neural networks to do so. Once again, these findings are particularly relevant to criminal justice determinations of guilt, which rely on nonscientific characterizations of mental states (e.g., did the defendant “know” what he was doing was wrong?). These findings support the notion that the psychopath knows what is wrong; s/he just may not have the capacity to care.

In sum:

- Psychopaths can identify pain, but may not rely on the same neural substrates as non-psychopaths when doing so.
- Psychopaths can identify the difference between a moral right and wrong, but may not experience the appropriate emotional reaction to moral wrongs.
- The psychopath’s ability to know the difference between right and wrong may undermine potential insanity defenses.

### Functional Connectivity

Functional connectivity is the study of relationships between neuronal activity and spatially remote regions of the brain (Friston, 1994). To date, three functional connectivity studies have been published in psychopathy. Studies frequently use independent component analyses (ICAs) to analyze functional connectivity. When ICA is applied to fMRI task data, it provides measures of both connectivity and task-relatedness (Calhoun et al., 2001). Because of its ability to identify the relationships between multiple brain regions and the neural networks connecting them, it has become an extremely popular way to analyze fMRI data (Erhardt et al., 2011). The first study to assess functional connectivity in a sample with clinical levels of psychopathy also measured white matter integrity using diffusion tensor imaging (DTI; Motzkin, Newman, Kiehl, & Koenigs, 2011). The functional connectivity findings are reviewed here, and the DTI findings are discussed in a later section of this chapter. The rest fMRI data revealed lower vmPFC–amygdala BOLD signal correlation among psychopaths than non-psychopaths (Motzkin et al., 2011). Based on this finding, the authors sought to determine if amygdala–vmPFC connectivity could also distinguish between psychopathy subtypes. They used a $2 \times 2$ (psychopathy high/low–anxiety high/low) ANOVA. They found a significant interaction between psychopathy and anxiety, such that
higher anxiety was associated with greater connectivity among non-psychopaths, and lower connectivity among psychopaths. Post hoc tests found no significant difference in connectivity between high- and low-anxious non-psychopaths, but significantly greater connectivity in low-anxious (“primary”) psychopaths than in high-anxious (“secondary”) psychopaths. Accordingly, amygdala–vmPFC connectivity was not only able to distinguish between psychopaths and non-psychopaths, but was also able to differentiate among psychopathy subtypes (Motzkin et al., 2011).

A subsequent study that examined functional brain connectivity in adults with psychopathy found that the condition was associated with aberrant functional connectivity in the paralimbic system (Juárez et al., 2013). Analyses revealed different task-related activity in several brain networks constitutive of the limbic and paralimbic regions in individuals with psychopathy, compared with controls. These findings are consistent with previous research identifying paralimbic abnormalities in psychopathy (Juárez et al., 2013; Kiehl, 2006).

Functional connectivity measures have also been used to investigate the neural correlates of impulsivity in juvenile offenders (Shannon et al., 2011). Shannon et al. examined the relationships between functional connectivity in the pre-motor areas of 107 youths incarcerated in a high-security juvenile facility. Impulsivity scores were assessed using Factor 2 PCL:YV scores. The findings yielded distinct patterns of functional connectivity between low-impulsive juveniles and controls compared with high-impulsive juveniles. In low-impulsive juveniles and controls, pre-motor areas showed connectivity with areas in the brain associated with spatial attention and executive control. In high-impulsive juveniles, pre-motor areas showed stronger correlations with brain areas associated with spontaneous, unconstrained, self-referential cognition. Shannon et al. (2011) additionally explored the relationship between pre-motor areas and their associated resting state networks in 95 normally developing individuals between the ages of 7 and 31. Similarly, younger subjects showed patterns comparable with high-impulsive juvenile offenders, whereas older subjects displayed functional connectivity patterns more similar to low-impulsive juveniles. These findings suggest that impulsivity among juvenile offenders is not distinct, but rather indicative of developmental delay. This bodes well for potential treatment interventions that would encourage development of inhibitory circuits.

**Structural MRI**

MRI is a powerful tool that generates images of the brain using noninvasive radio waves and magnetic fields. These three-dimensional images are able to distinguish between gray matter (i.e., cell bodies or thinking areas of the brain), white matter (i.e., myelin or wiring of the brain), and cerebral spinal fluid (i.e., ventricles or fluid-filled spaces of the brain), picking up on gross structural anomalies. Psychopathy does not appear to be related to gross structural abnormalities. However, detailed analyses of structural MRI density data have consistently linked gray matter abnormalities to psychopathy. The prime technique used to show these gray matter abnormalities is known as voxel-based morphometry (VBM). VBM is an automated and replicable technique used to analyze structural MRI scans for differences in both gray matter volume and density across subjects. Studies have shown that VBM assessments of gray matter volume decrease with age (Smith et al., 2007), are positively correlated with measures of IQ (Narr et al., 2007), and have substantial utility for understanding psychopathology (see e.g., Koutsouleris et al., 2008; Vasic, Walter, Höse, & Wolf, 2008). In addition, recent VBM analyses of psychopathy have produced highly consistent findings.

In the first large-scale ($N = 296$) VBM study of incarcerated males, Ermer et al. (2012) reported significant reductions in gray matter volumes in broad regions of the paralimbic system associated with Hare PCL-R scores. The regions showing abnormalities were: orbital frontal cortex, bilateral medial (amygdala and hippocampus) and lateral (temporal pole) temporal lobe, insula, and posterior cingulate. These findings are highly consistent with the hypothesis that psychopathy is related to abnormalities in the paralimbic and limbic regions (Kiehl, 2006).
The Ermer et al. (2012) VBM study of adult men raised a number of important questions. One question was whether the aberrant brain structures were due to developmental, or perhaps even genetic, factors, or whether they were due to atrophy related to nonuse. In other words, are psychopaths born with abnormal brains, or do their brains develop abnormally owing to specific environmental conditions (e.g., nonuse)?

Accordingly, Ermer et al. (2013) performed a VBM analysis on a large sample (N = 200) of maximum-security incarcerated male adolescents. Consistent with the adult findings, male adolescent offenders with high Hare PCL:YV scores showed gray matter reductions across several key paralimbic structures, including: orbitofrontal cortex, bilateral temporal poles, amygdala, and posterior cingulate cortex (Ermer et al., 2013). The brain gray matter abnormalities did not appear to change with age—that is, the youth and the adults showed very similar brain gray matter effects. These results were interpreted as support for developmental or genetic accounts of psychopathic traits, rather than environmental hypotheses.

Another question raised by the Ermer et al. (2012) studies was whether the gray matter abnormalities are also found in females. Cope, Ermer, Nyalakanti, Calhoun, & Kiehl (2014) reported that maximum-security incarcerated female youth with elevated Hare PCL:YV scores showed reduced gray matter across paralimbic structures, including: orbitofrontal cortex, right parahippocampal cortex, and temporal pole. Importantly, all of these latter VBM gray matter analyses carefully controlled for variables such as age, IQ, and substance abuse histories. These data suggest psychopathy-related gray matter deficits span both male and female samples and ages.

In another study, Gregory et al. (2012) used VBM to analyze structural gray matter differences in adult male violent offenders with and without psychopathy. When comparing violent offenders
with psychopathy with healthy non-offenders, the former showed reduced gray matter in the bilateral anterior rostral medial prefrontal cortex, bilateral anterior temporal areas, and bilateral anterior insula (Gregory et al., 2012). When violent offenders without psychopathy were compared with healthy controls, no statistically significant differences in gray matter were detected. Finally, when violent offenders with psychopathy were compared with violent offenders without psychopathy, the former showed gray matter reductions bilaterally in the anterior rostral medial prefrontal and temporal pole regions (Gregory et al., 2012). These findings not only lend support to the paralimbic hypothesis of psychopathy, but also affirm psychopathy as a construct distinct from general propensity to violence.

In sum:
- Psychopaths have reduced gray matter volumes in key emotional processing areas hypothesized to be relevant to psychopathy.
- These results are also differentiated among non-psychopathic violent offenders and psychopathic ones, suggesting that psychopathy is a distinct construct.
- These results are further evidence that psychopathy is a developmental disease.

**Diffusion Tensor Imaging**

Diffusion tensor imaging (DTI; or diffusion tensor MRI) is a type of MRI that allows the mapping of the diffusion process of molecules, mainly water in biological tissues, in vivo (Jones & Leemans, 2011). Water molecule diffusion patterns can reveal microscopic details about tissue architecture. When used in conjunction with fMRI, DTI can reveal abnormalities in white matter fiber structure and provide models of brain connectivity. DTI is an extremely sensitive measure and has been incorporated into research and clinical practice for over two decades. It has been used to document abnormalities in white matter in a variety of conditions, such as brain ischemia (Le Bihan et al., 2001), multiple sclerosis (Filippi, Cercignani, Inglese, Horsfield, & Comi, 2001), stroke, schizophrenia, Alzheimer's (Rose et al., 2000), and Parkinson's disease (Vaillancourt et al., 2009). Below is a summary of the DTI findings in samples with clinical levels of psychopathy.

The first study to use DTI tractography to assess white matter structural integrity in individuals with elevated levels of psychopathic traits (psychopaths defined as PCL-R scores > 25) was conducted by Craig et al. (2009). Their study found that individuals with higher psychopathy scores had significantly lower DTI values \( (P < .003) \) in the right uncinate fasciculus (UF) compared with IQ- and age-matched controls. The UF is the primary, bidirectional white matter tract that connects structures within the limbic system to the lateral orbitofrontal cortex and Brodmann area 10 with the anterior temporal lobes (Von Der Heide, Skipper, Klobusicky, & Olson, 2013). Understanding the function of the UF has considerable clinical relevance, because it has been implicated in a number of developmental and psychiatric disorders. A recent review of the psychiatric implications proposed that the big-picture role of the UF is to allow temporal-based mnemonic associations to influence behavior by communicating with the lateral orbitofrontal cortex (Von Der Heide et al., 2013). Under this view, damage to the UF can cause difficulties with the ability to use memories to inform decision-making, as well as disrupt learning and memory processes, and even manifest itself in social–emotional problems. This interpretation of UF’s role and function is consistent with deficits seen in individuals with elevated levels of psychopathic traits, because a hallmark characteristic of psychopathy is a seeming inability to use emotionally salient information to guide decision-making and, thus, behavior. Further, difficulties in assessing and appropriately acting on social–emotional cues is consistent with the impaired moral decision-making and other affective deficits seen in individuals with psychopathy.

Motzkin et al. (2011) examined both structural and functional connectivity in a sample with clinical levels of psychopathy (PCL-R score ≥ 30) compared with non-psychopathic controls.
Table 3.2 Summary of Structural Psychopathy Studies.

<table>
<thead>
<tr>
<th>Author</th>
<th>Psychopathy Assessment</th>
<th>Comparison Groups</th>
<th>Type of Analysis</th>
<th>Results (Psychopaths vs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boccardi et al.</td>
<td>PCL-R high, (M = 34.6), N = 12 medium, (M = 25.9)</td>
<td>N = 25 healthy controls</td>
<td>Structural MRI</td>
<td>High psychopathy group showed reductions along the dorsal and ventral hippocampal axis. High and medium psychopathy groups showed enlargement of lateral borders in the left and right hippocampi</td>
</tr>
<tr>
<td>(2010)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boccardi et al.</td>
<td>PCL-R high (M = 34.6), N = 12 medium, (M = 25.9), N = 14 inmates</td>
<td>N = 25 healthy controls</td>
<td>Structural MRI</td>
<td>Psychopaths had 20% reduction in OFC and midline structures, 30% tissue reduction in basolateral nucleus, and 10–30% enlargement in central and lateral nuclei of amygdala</td>
</tr>
<tr>
<td>(2011)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boccardi et al.</td>
<td>PCL-R high (M = 34.6), N = 12, medium (M = 25.9), N = 14 inmates</td>
<td>N = 25 healthy controls</td>
<td>Structural MRI</td>
<td>Caudate and putamen volumes normal. Abnormal morphology in rt. dorsal putamen. Nucleus accumbens was 13% smaller</td>
</tr>
<tr>
<td>(2013)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contreras-</td>
<td>PCL-R (M = 27.8), N = 22 inmates</td>
<td>N = 22 healthy controls</td>
<td>Structural MRI/functional resting state</td>
<td>Reduced gray matter in ventral, lateral, and medial aspects of PFC, ACC, PCC, insula-operandum, amygdala-hippocampus, and fusiform gyrus. Deficits in medial-dorsal frontal cortex. Factor 1 negatively correlated with anatomic measures. Factor 2 positively correlated with connectivity measures</td>
</tr>
<tr>
<td>Rodríguez et al.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2014)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ermer et al.</td>
<td>PCL-R used as continuous measure, N = 77 parolees and participants in drug treatment</td>
<td>N/A</td>
<td>Structural MRI</td>
<td>Positive correlation with gray matter in bilateral OFC and right ACC. Rt. hippocampus and lt. OFC explained 21.8% of the variance. Gray matter volume increases correlated with functional impairment</td>
</tr>
<tr>
<td>(2012)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cope et al.</td>
<td>PCL:YV used as a continuous measure, N = 39 youth detainees</td>
<td>N/A</td>
<td>Structural MRI</td>
<td>Negative correlation with gray matter in limbic and paralimbic regions, including the OFC, parahippocampal cortex, temporal poles, and left hippocampus, which is consistent with juvenile and adult male results</td>
</tr>
<tr>
<td>(2014)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reference</td>
<td>Methodology</td>
<td>Sample Size</td>
<td>Modality</td>
<td>Findings</td>
</tr>
<tr>
<td>--------------------</td>
<td>------------------------------------------</td>
<td>----------------------</td>
<td>-------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Ermer et al. (2012)</td>
<td>PCL-R used as a continuous measure, N = 296 inmates</td>
<td>N/A</td>
<td>Structural MRI</td>
<td>Decreased gray matter in paralimbic and limbic areas, including bilateral parahippocampal, amygdala, and hippocampal regions, bilateral temporal pole, PCC, and OFC</td>
</tr>
<tr>
<td>Ermer et al. (2013)</td>
<td>PCL:YV used as a continuous measure, N = 191 youth detainees</td>
<td>N/A</td>
<td>Structural MRI</td>
<td>Decreased gray matter volumes in the OFC, bilateral temporal poles, and PCC</td>
</tr>
<tr>
<td>Gregory et al. (2012)</td>
<td>PCL-R ≥ 25, N = 17 violent offenders</td>
<td>N = 17 violent offenders with ASPD</td>
<td>Structural MRI</td>
<td>Reduced gray matter volumes in bilateral rostral PFC and temporal poles. Psychopathy distinct from ASPD</td>
</tr>
<tr>
<td>Kolla et al. (2013)</td>
<td>PCL-R ≥ 25, N = 9 violent parolees</td>
<td>N = 15 non-psychopathic parolees; N = 13 healthy controls</td>
<td>Structural MRI</td>
<td>Reduced gray matter volumes in bilateral temporal poles, right uncus, and right lobule VI of the posterior cerebellum</td>
</tr>
<tr>
<td>Laakso et al. (2001)</td>
<td>PCL-R used as continuous measure, N = 18 pretrial psychiatric patients</td>
<td>N/A</td>
<td>Structural MRI</td>
<td>Negative correlations with posterior half of the hippocampus bilaterally</td>
</tr>
<tr>
<td>Ly et al. (2012)</td>
<td>PCL-R (M = 31.8; SD = 1.7), N = 21 inmates</td>
<td>N = 31 non-psychopathic inmates</td>
<td>Structural MRI/ functional resting state</td>
<td>Thinner cortex in: lt. insula and dACC precentral gyri, anterior temporal cortices, and rt. IFG. Reduced functional connectivity between lt. insula and lt. dorsal ACC</td>
</tr>
<tr>
<td>Tiibonen (2008)</td>
<td>PCL-R (M = 34.6, SD = 3.1), N = 12 forensic psychiatric patients</td>
<td>N = 14 patients with ASPD</td>
<td>Structural MRI</td>
<td>Reduced gray matter in postcentral gyri, frontopolar cortex, and OFC</td>
</tr>
</tbody>
</table>

*Note: All results are described in terms of psychopathy group versus indicated control or comparison group.*
(PCL-R score < 20). Replicating the finding from Craig et al. (2009), individuals with clinical levels of psychopathy showed a reduction of white matter integrity in the right UF. The latter effect was most robust in individuals with high psychopathic traits and low anxiety.

In 2013, a group in the Netherlands replicated the finding of white matter abnormalities in a sample of 11 offenders with psychopathy, compared with 11 matched healthy controls (Hoppenbrouwers et al., 2013). In addition, Hoppenbrouwers et al. (2013) found that voxel-based tractography revealed significant differences between the two groups in the amygdalo-prefrontal network, as well as a striato-thalamic-frontal network. Although disruption in the former network had been discovered previously, the second finding was novel and unique to this study. Disruption in this latter white matter pathway that connects the ventral tegmental area with the nucleus accumbens is an important part of reward circuitry and was positively correlated with Factor 2 scores on the PCL-R (Hoppenbrouwers et al., 2013). The authors proposed that the disruption of striato-thalamic-frontal reward circuitry is linked to impulsivity, violence, and substance abuse.

In sum:

- Functional imaging findings have shown psychopathy is associated with reduced functional connectivity between vmPFC and amygdala, as well as between vmPFC and medial parietal cortex.
- Together with the diffusion findings, these results provide strong support for the hypothesis that reduced vmPFC connectivity is an underlying neurobiological characteristic of clinical levels of psychopathy.

**Neuroscience of Psychopathy in Court**

In cases where the state seeks the death penalty against a criminal defendant, due process requires an adequate investigation into a defendant’s mental history when appropriate and, if justified, additional testing to assist in evaluating the defendant’s condition. “Additional testing” has come to include neuroimaging evaluations, as medical experts state with increasing frequency that such technology enables them to make a more complete and accurate assessment of the defendant’s neurological and psychological state (e.g., Hoskins v. State, 2007).

In 2010, fMRI, as well as structural MRI density analyses, was admitted in the DuPage County Illinois capital sentencing trial of Brian Dugan (Gaudet, Lushing, & Kiehl, 2014; Kiehl, 2014). Dugan had a well-documented history of serious behavioral problems from a very early age and he met clinical criteria for psychopathy using the PCL-R (99th percentile). In Illinois, an “extreme mental or emotional disturbance” is a statutory mitigating factor, and, if the defendant presents evidence of such a disturbance, the jury must consider it during their deliberations. As summarized in the preceding sections, there is a robust peer-reviewed literature supporting the proposition that psychopathy is associated with abnormalities in areas of the brain responsible for the processing of emotional information. Structural and functional MRI data were collected on Dugan to evaluate whether he suffered from similar structural and functional brain abnormalities as found in the peer-reviewed literature. The defense’s argument was that Dugan suffered from a developmental disorder of emotion (i.e., psychopathy), and this constituted evidence supporting the statutory mitigating condition of “extreme emotional disturbance.” The Illinois court conducted a Frye hearing—the admissibility standard that requires that the proffered scientific evidence be generally accepted in the relevant scientific community before the evidence is allowed to be introduced in court—and found the proffered evidence to be generally accepted and thus admissible.

To our knowledge, the Dugan trial is the first time that fMRI was used in a legal proceeding and the first time that MRI data consistent with a diagnosis of psychopathy were admitted in court and presented to a jury. For complete details on this case and the outcomes, see Kiehl (2014).
Empirical Research of Legal Assumptions

At the heart of the intersection of science and law is an effort to translate scientific research into effective legal practice and policy. Our modern legal system still maintains a great many practices from the British common law, a result of which is that our trial courts create an environment where a great deal of weight and a great deal of deference can be given to certain types of evidence, for no other reason than that particular type of evidence has been admitted, and relied upon, since common law. One important step toward the goal of integrating research into more informed policy is to test a legal practice, or what can be referred to as a legal assumption, and design an experiment to see if the assumption holds true. For example, prior to the discovery of DNA, eyewitness testimony and confessions were considered the best evidence that the accused individual indeed committed the crime in question. Decades of research have shown that eyewitness testimony is fraught with problems (Loftus, 1996) and people confess to things they did not do (Gudjonsson, 1992).

Relating specifically to neuroscience evidence, there was a considerable commentary in both the psychological and legal literature about the feared prejudicial impact of neuroimaging evidence on a jury (e.g., Baskin, Edersheim, & Price, 2007; Brown & Murphy, 2010; Gurley & Marcus, 2008). In 2011, Schweitzer et al. tested the prejudicial assumption and found it to be false. Across four experiments, more than 1,400 mock jurors evaluated written summaries of criminal cases that included mental health expert testimony designed to be exculpatory. Neuroimages did not affect jurors’ judgments of verdicts, sentence recommendations, or judgments of the defendant’s culpability above and beyond verbal neuroscience-based testimony (Schweitzer et al., 2011). Thus, brain images were not considered to be prejudicial in those studies.

Saks, Schweitzer, Aharoni, and Kiehl (2014) conducted another set of experiments designed to test the impact of neuroimaging on juror decision-making, also using a large cohort (more than 820) of jury-eligible, death-qualified mock jurors and also during the penalty phase of a capital murder trial (Saks et al., 2014). Both experiments compared the introduction of neuroimages with three other forms of evidence that led to the same diagnosis: (1.) neuroscience expert testimony without brain images, (2.) expert testimony of the defendant’s genetic abnormality, and (3.) expert testimony based on a clinical psychological examination. The researchers also presented the case to a control group of jurors, who saw the facts without the introduction of any expert testimony.

The first experiment evaluated the four types of expert testimony presented and the absence of expert testimony when crossed with a defendant diagnosed with psychopathy, schizophrenia, or “healthy” as additional independent variables. They found that, for defendants diagnosed with psychopathy, the introduction of neuroimaging evidence did decrease the jurors’ judgment of the defendant’s responsibility, and it slightly decreased the imposition of the death penalty. The authors also found that neuroimaging evidence of psychopathy did not alter the future dangerousness assessment. These latter data empirically confirmed the logic used by the defense in the Dugan trial.

The second experiment added an additional element to the same structure as the first experiment: whether the expert testimony was offered by the prosecution as evidence of an aggravating factor, supporting a death verdict, or if it was offered by the defense as mitigating evidence, in hopes of a sentence of life-in-prison as opposed to death. Interestingly, if the neuroimaging evidence was presented by the defense, it did have the effect of reducing the rate of death sentences in comparison with presentations of clinical, genetic, and nonimage neurological evidence. If the evidence was offered by the prosecution, the jury was more likely to impose the death penalty.

Based on that finding, Saks et al. (2014) concluded that, “[f]or both advocates [prosecution and defense], then, the neuroimaging evidence was the most persuasive, advancing their arguments, and moving verdicts in the direction they sought.”

In another interesting study, Aspinwall, Brown, and Tabery (2012) investigated whether evidence of a biomechanical cause of a defendant’s psychopathy would affect state trial judges’ (N = 181) determinations regarding the aggravating and mitigating impact of the evidence and, in turn, their
sentencing determinations. It did. The researchers reported that evidence of a biomechanical cause for a defendant’s antisocial behaviors had the effect of reducing the likelihood that the defendant’s psychopathy would be considered aggravating—in other words, it was considered mitigating—and resulted in a significant reduction in the defendant’s sentence.

When presented with just the facts of the crime, 29.7 percent of judges found at least one mitigating factor, and 86.7 percent found at least one aggravating factor. However, if the prosecution added evidence of biomechanism for the defendant’s psychopathy to the same fact pattern, it only marginally increased the percentage of judges who found at least one aggravating factor. But, when the defense presented a biomechanism for the defendant’s psychopathy, the number of judges who found at least one mitigating factor doubled (from 29.7 percent to 47.8 percent). Together, these finds suggest that, when evidence supporting a biological/neurobiological mechanism for psychopathy is presented, it has the potential to be considered mitigating if proffered by the defense.

As the studies above demonstrate, neuroimaging evidence can be relevant and persuasive evidence of a psychiatric diagnosis. Of course, exactly how relevant scientific research will be to any given legal argument requires a detailed and nuanced analysis of both the science and legal argument in question (Gaudet et al., 2014). For decades, attorneys have been using neuroimaging modalities such as MRI, EEG, and SPECT/PET and making a variety of arguments in all phases of criminal trial (competency, guilt, and mitigation). For the most part, these arguments have not been successful, but it is not because of any shortcomings of the imaging techniques, but because there is often no peer-reviewed literature that supports the connection between the imaging data and the legal argument in question (Gaudet et al., 2014). As the neuroscientific research of legally relevant conditions (e.g., psychopathy, schizophrenia) continues to grow, and researchers continue to conduct studies to test the impact of different areas of research in legal contexts, there will be an increasing number of attorneys who seek to support their legal arguments with such evidence. Neuroimaging evidence generally and neuroimaging of psychopathy evidence specifically are examples of types of evidence that have been tested to see their purported influence in mock juror and judge decision-making contexts. Some of the results are surprising, such that evidence of a neurobiological cause of psychopathy can be considered mitigating rather than aggravating evidence. However, this finding is actually right in line with a number of beliefs about how certain types of evidence would be received that have simply turned out not to be true—such as the alleged reliability of eyewitness testimony and the belief that people would never confess to something they did not do.

Conclusion

The greatest challenge in psychopathy research is developing effective evidence-based treatments in adolescents and adults with clinical levels of psychopathy. Currently, there are promising programs that have been found to greatly improve outcomes in youth on a trajectory towards developing clinical levels of psychopathic traits as adults (see Kiehl, 2014). Imaging is being applied to help understand the neurobiology associated with elevated levels of psychopathic traits in both adult and adolescent populations, as well as the neurobiological changes in high-risk adolescents as they go through treatment. Identifying the neurobiological differences in individuals with these traits is important, because it will provide a target to focus treatment interventions. Given the disproportionate impact that individuals with psychopathy have on society in terms of the social, emotional, and fiscal costs of crime, even a modest improvement in behavior could translate into far fewer crimes and victims and result in millions, if not billions, of dollars of cost savings across the country. Lastly, early identification of these traits from a developmental perspective will likely be the key to prevention—the ultimate goal.
References


