A Social Neuroscience Perspective on the Neurobiological Bases of Aggression

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The discovery of brain regions and mental processes that contribute to aggressive behavior has long been a significant concern in psychology. Although much progress has been made, identification of the underlying neural mechanisms remains elusive (Davidson, Putnam, & Larson, 2000). It is thought that neural functioning mediates aggressive behavior by biasing mental processes toward aggressive responses to social situations (Raine, 2008). Relying on advances in the emerging field of social neuroscience, this chapter reviews research that has increased our understanding of the neural mechanisms associated with human aggression. This review contains a brief discussion of relevant brain anatomy, followed by a review of structural and functional brain abnormalities in highly aggressive populations. Next, I shall discuss the application of a recent social neuroscience dual-process model, the X- and C-systems, to the study of anger and aggression. This is followed by a discussion of social neuroscience research that is consistent with social psychological theory. Finally, I discuss the role played by top down executive control in determining aggressive behavior.

Anatomy

Reviews of anger and aggression converge on the importance of the prefrontal cortex (PFC) underlying these phenomena (Davidson et al., 2000; Raine, 2008; Siever, 2008). Indeed, the PFC is broadly involved in the regulation and control of affect and behavior. Within the PFC, four regions are particularly relevant to aggressive behavior. The dorsolateral PFC is involved in planning and behavioral control. The ventral PFC, which encompasses the ventromedial PFC, ventrolateral PFC, and the orbitofrontal cortex (OFC), is involved in emotion regulation. The dorsal region of medial PFC (referred to here as the mPFC) is involved in introspection and the awareness of emotion as well as emotion regulation. Finally, the dorsal anterior cingulate cortex (dACC) is involved in the detection of conflict and triggering activity in top-down control regions (see Figure 1).

In terms of neurotransmitter involvement, converging evidence suggests that serotonin has a prominent role in facilitating and inhibiting anger and hostile aggression via 5-HT_2 receptors in the PFC (Davidson et al., 2000; Rhee & Waldman, Chapter 9, this volume; Siever, 2008). Because a detailed discussion of neurotransmitter modulation is beyond the scope of this chapter, the purpose here is to review the most current research on brain regions implicated in anger and aggression.

Evidence from Abnormal Populations

Early examinations of patients with brain lesions revealed that abnormalities in the PFC were associated with aggressive and antisocial behavior. The classic case of Phineas Gage, who suffered trauma to his orbitofrontal cortex, is illustrative of the dramatic within-person change from agreeable and conscientious to hostile and antagonistic that is associated with trauma to this region. A fairly large study of 279 Vietnam War veterans with brain lesions revealed that those who suffered injury to the orbitofrontal cortex and medial PFC were more irritable, hostile, and aggressive than control participants (Grafman et al., 1996).

More recent neuroimaging studies have examined structural differences in groups of individuals known to be highly aggressive compared with less aggressive matched controls. Several studies have identified prefrontal structural differences in the brains of aggressive individuals, such as violent offenders and psychopaths, relative to matched controls (for reviews, see Raine, 2008; Yang, Glenn, & Raine, 2008). For instance, individuals with antisocial personality disorder have an 11% to 14% deficit in prefrontal gray matter relative to normal controls, substance-dependent individuals, and individuals with other psychiatric disorders (Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000).

These differences are not limited to brain structure. Numerous studies document functional impairment in frontal and limbic regions such as the ventral PFC, dorsolateral PFC, mPFC, anterior cingulate cortex (ACC), posterior cingulate cortex (PCC), hippocampus, and amygdala (Raine, 2008; Raine & Yang, 2008). For instance, a positron-emission tomography (PET) study reported reduced glucose metabolism in the PFC in a group of murderers compared with a matched control group (Raine, Buchsbaum, & LaCasse, 1997). Another study found that when processing emotional versus neutral words, criminal psychopaths show less activation in the lateral PFC, ACC, PCC, and amygdala than non-criminal psychopaths and normal controls (Kiehl et al., 2001). In summary, violence-prone groups show deficits in the structure and functioning of key brain regions involved in emotion regulation and behavioral control. Although these data are correlational, presumably these abnormalities reduce the ability to regulate and control angry feelings and aggressive behavior.

A Social Neuroscience Framework: The X- and C-Systems

Although research on abnormally aggressive individuals is instructive, most social and personality psychologists are primarily interested in the normal spectrum of human behavior.

Indeed, decades of social psychological and personality research demonstrate that we are all capable of behaving aggressively under certain circumstances, and that some individuals within the normal range tend to be more aggressive than others across a variety of contexts (e.g., Anderson & Bushman, 2002; and DeWall & Anderson, Chapter 1, Huesmann, Dubow, & Boxer, Chapter 8, and Slotter & Finkel, Chapter 2, this volume). Social neuroscience is an emerging field that examines the neural correlates of social psychological phenomena. Because functional neuroimaging technology, especially functional magnetic resonance imaging (fMRI), is relatively widely available, we are now able to examine neural processes in normal individuals using experimental methods drawn from social and cognitive psychology. Tools such as fMRI enable us to explore previously unquantifiable aspects of human functioning that are directly relevant to social psychological questions.

In an effort to integrate findings from the rapidly expanding field of social neuroscience, Lieberman and colleagues (Lieberman, 2007; Lieberman, Gaunt, Gilbert, & Trope, 2002; Satpute & Lieberman, 2006) proposed a dual-process framework known as the X- and C-systems model. The *X* component of the model, which we likely share with other animals, represents our continuous stream of current experience (Lieberman et al., 2002, p. 204). The *X* stands for the *x* in *reflexive*. The X-system is phylogenetically older than the C-system, operates quickly, supports spontaneous processes, and engages in parallel processing. Brain regions that make up the X-system are the ventromedial PFC, dACC, amygdala, basal ganglia, and lateral temporal cortex. Social psychological phenomena associated with this region include implicit prejudice, emotional pain resulting from social rejection, and intuition-based self-knowledge (see Lieberman, 2007).

In contrast with the X-system, the C-system underlies reflective and control processes. The *C* stands for the *c* in *reflective*. The C-system is phylogenetically newer than the X-system, operates slowly, is associated with volitional processes, utilizes serial processing (i.e., not parallel processing), and involves abstract thought (Lieberman, 2007). We experience activity in the C-system during reflective thought. Thus, current experience is the result of the X-system, whereas reflecting on this experience is supported by the C-system. The C-system often becomes active when the X-system detects a problem. Brain regions that make up the C-system include the lateral and medial PFC, lateral and medial parietal cortex, medial temporal lobe, and ventrolateral PFC. Social psychological phenomena associated with the C-system include explicit attitudes, reflecting on current experiences, impulse control, reappraising emotional events, and moral reasoning (see Lane, Fink, Chau, P. & Dolan, 1997; Lieberman, 2007; MacDonald, 2008). Although research on anger and aggression was not considered in Lieberman’s (2007) review, the X- and C-systems provide a valuable framework for discussing the neural bases of anger and aggression. Specifically, many of the processes associated with the X- and C-systems are relevant for understanding aggressive behavior.

Social Neuroscience Research on Anger and Aggression

Anger

Most functional imaging studies of anger in nonclinical samples have exposed participants to angry faces or asked them to recall and re-experience anger-inducing events. Two recent meta-analyses of nine PET and fMRI studies revealed that some of the most prominent areas of frontal and limbic brain reactivity were the mPFC, ventromedial PFC, ACC, PCC, lateral PFC, and thalamus (Murphy, Nimmo-Smith, & Lawrence, 2003; Phan, Wager, Taylor, &

Liberzon, 2002). Interestingly, whereas the amygdala has a prominent role in fear, it was not implicated in these studies of angry faces and memories (Phan et al., 2002).

My colleagues and I recently examined the neural correlates of anger more directly by exposing participants to an interpersonal insult (Denson, Pedersen, Ronquillo, & Nandy, 2009). Anderson and Bushman (2002) described such a provocation as “perhaps the most important single cause of human aggression” (p. 37). In this fMRI study, participants were asked to complete difficult anagrams and state the answer aloud or say “no answer” if they did not know the answer. Following two polite prompts to speak louder, the experimenter then insulted participants by saying in an irritated and condescending voice, “Look, this is the third time I’ve had to say this! Can’t you follow directions?” We found that, relative to baseline, participants showed increased activation in many of the same regions active during exposure to angry faces and autobiographical recall of anger experiences. Moreover, we found that a component of the X-system, the dACC, played a special role in the subjective experience of anger. Specifically, self-reported anger was correlated with dACC activation ($r = .56$). Activity in the dACC was also correlated with scores on the Buss and Perry (1992) Aggression Questionnaire, a measure of general trait anger, hostility, and aggression ($r = .61$). Consistent with the claim that the dACC is involved in the subjective experience of anger, individuals who have had portions of the ACC removed demonstrate decreased anger (Cohen, Botvinick, & Carter, 2001).

Angry Rumination

Reflective processes also play a role in anger and aggression. Regions of the C-system that have been implicated in anger and aggression include the lateral PFC, mPFC, and medial parietal cortex. As noted above, following provocation, participants demonstrated increased activity in regions of the C-system as well as the X-system. One important reflective process that can influence aggressive behavior is the way one regulates emotions. One particularly pernicious form of emotion regulation is angry rumination. Immersive rumination on anger-inducing experiences increases anger, aggression, cardiovascular arousal, and cortisol levels (Bushman, 2002; Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005; Denson, Fabiansson, Creswell, & Pedersen, 2009; Denson, Pedersen, & Miller, 2006; Ray, Wilhelm, & Gross, 2008; Rusting & Nolen-Hoeksema, 1998).

In addition to these negative consequences of angry rumination, our recent research demonstrates that angry rumination also increases activity in parts of the C-system (Denson et al., 2009). In the second part of the experiment described above, following the provocation, participants were asked to engage in a "memory task," which served as a rumination manipulation. Using a modified within-participants rumination task from prior research (Bushman et al., 2005; Denson et al., 2006; Rusting & Nolen-Hoeksema, 1998), during the rumination task, participants were asked to think about what had occurred in the experiment so far, who they interacted with, and their current mood. During the distraction period, participants were asked to think about neutral events. Relative to distraction, rumination increased activity in regions of the C-system such as medial and lateral PFC, insula, precuneus, and PCC. Furthermore, self-reported rumination was correlated with activity in the mPFC ($r = .42$) as were scores on the Displaced Aggression Questionnaire, an individual difference measure of trait displaced aggression (Denson et al., 2006, 2009). The relationship between the medial PFC and the displaced aggression measure was likely due to the fact that when provoked, individuals high in trait displaced aggression tend to ruminate about the event rather than immediately retaliate against the provocateur.

We expected that the mPFC would be especially relevant to angry rumination because it supports many of the reflective processes at work during rumination. For example, the medial PFC is activated during tasks that require the self-awareness of emotions and self-relevant cognition (Lane et al., 1997; Lieberman, 2007; Ochsner et al., 2004). This region is also active when monitoring one's emotional state, reflecting on feelings, and reappraising emotional responses to distressing stimuli (Amodio & Frith, 2006; Ochsner et al., 2004; Ochsner, Bunge, Gross, & Gabrieli, 2002). Furthermore, Ray et al. (2005) reported that when participants were asked to decrease their negative affective responses to aversive photographs, a composite measure of trait rumination was correlated with medial PFC activity.

Cognitive Neoassociation Theory

As mentioned above, activity in the dACC was linearly related to self-reported anger following provocation (Denson et al., 2009). The dACC is of social psychological interest because it is involved in at least two additional negative emotional states that have been shown to increase aggression. This is relevant because a core tenet of Berkowitz's (1993) cognitive neoassociationistic model of aggression states that any form of negative affect can increase aggression. A large body of evidence supports Berkowitz's supposition. For example, physical pain and social rejection both increase aggression *and* activation in the dACC (Berkowitz, Cochran, & Embree, 1981; Eisenberger et al., 2003; Rainville, Duncan, Price, Carrier, & Bushnell, 1997; Twenge, Baumeister, Tice, & Stucke, 2001). Together, these and the anger findings converge to suggest the existence of a common neural mechanism underlying the process Berkowitz proposed, although the mediating role of dACC activation between anger, pain, social rejection, and actual aggressive behavior remains to be investigated. Future neuroimaging research examining additional aversive stimuli known to increase aggression, such as noxious odors and heat, might demonstrate increased activity in the dACC as well.

Social Learning, Media Violence, and Script Theory

According to social learning perspectives on aggression, individuals learn how and when to behave aggressively by observing others either in person or vicariously in the media (Bandura, 1973). A tremendous amount of research supports this notion (e.g., Anderson et al., 2003). Expanding upon social learning theory, Huesmann (1998) proposed that individuals learn behavioral scripts from aggressive media exposure. Scripts are closely connected concepts in memory that can become strengthened by rehearsal and chronic exposure to violent media. When activated, such scripts can increase the likelihood of aggressive behavior (Huesmann et al. Chapter 8, this volume).

Only a handful of social neuroscience studies have examined neural responses during exposure to violent media (for a review, see Carnagey, Anderson, & Bartholow, 2007). In one study, in an attempt to identify children most responsive to violent media, children who showed heart rate acceleration to violent media on a pretest were exposed to violent (i.e., boxing) and nonviolent (i.e., animal) scenes while functional images were acquired (Murray, Liotti, Mayber, Pu, Zamarripa, & Liu, 2006). The strongest activity was located in the right PCC and the right precuneus (in the medial parietal cortex). Because of its role in memory, these authors speculated that activity in the PCC might correspond to the activation of stored aggressive scripts. This is consistent with our research showing that the PCC was active during angry rumination, because the revenge planning that occurs during angry rumination likely involves the activation of aggressive scripts (Denson et al., 2009). Indeed, factor analytic work indicates that angry rumination involves rehearsing acts of revenge (Caprara, 1986; Denson et al., 2006; Sukhodolsky, Golub, & Cromwell, 2001).

Two additional studies found that violent media influence brain activity in what is thought to be a maladaptive manner. Specifically, in an investigation of the hypothesis that chronic exposure to violent media desensitizes individuals to aggressive content, one study examined the P300 component of the event-related brain potential (ERP) in people who played relatively high levels of aggressive video games and those who played aggressive video games less often (Bartholow, Bushman, & Sestir, 2006). When exposed to violent images in the laboratory, chronic exposure to violent video games was associated with decreased P300 amplitude to violent images, but not to negative or neutral images. These findings support the notion that violent video game play can desensitize basic neural responses to violent stimuli. Furthermore, the P300 deficit predicted increased aggression as assessed by choosing to deliver loud noise blasts to a fictitious participant. Although EEG methods do not allow for precise localization of brain processes, presumably the automatic response of the P300 reflects activity in the X-system, suggesting that chronic exposure to media violence can alter even quite rudimentary information processing such that individuals with high levels of exposure actually *experience* violent media differently than those low in exposure.

A recent fMRI study investigated brain activity during actual violent video game play (Weber, Ritterfeld, & Mathiak, 2006). In a sophisticated frame-by-frame analysis of violent game play, these authors demonstrated that activity in the dACC preceded suppression in the rostral ACC (rACC), which is involved in affective information processing, and in the amygdala during aggressive “search and destroy” sequences. Recall that the dACC is associated with the subjective experience of anger (Denson et al., 2009). This suppression of the rACC by the dACC when committing acts of video game violence suggests that the dACC overrides affective input from the rACC. Consistent with the suppression function of the dACC, when participants were in danger, under attack, or using a weapon, the dACC was more active than when participants were passive or safe.

Aggressive Behavior

Only two neuroimaging studies have investigated brain activity during actual acts of aggression. The first such fMRI study examined brain activity in 14 men high and low in psychopathy during their performance in a Taylor (1967) aggression paradigm that was modified such that participants could see a fictitious participant receive the allocated bursts of physically painful pressure to their hand (Lotze, Veit, Anders, & Birbaumer, 2007). Of primary interest, activity in the mPFC was positively correlated with the intensity of pain participants chose to have administered to the confederate. This latter finding is consistent with the role of the mPFC in angry rumination, emotion regulation, attributions, and theory of mind (e.g., Amodio & Frith, 2006; Denson et al., 2009; Harris, Todorov, & Fiske, 2005; Ochsner et al., 2002).

A second study of normal young adults utilized a modified white-noise Taylor (1967) paradigm in which the decision phase (i.e., deciding what noise level to choose) and the outcome phase (i.e., the aggressive act) were analyzed separately (Krämer, Jansma, Tempelmann, & Münte, 2007). Furthermore, participants were given the opportunity to aggress against highly provocative and less provocative bogus participants, as well as against a computer. This allowed the authors to identify the neural mechanisms specifically associated with aggression rather than social interaction *per se*. Under high provocation, the dACC and mPFC were active during the decision phase, suggesting the presence of anger and rumination (e.g., Denson et al., 2009).

Perhaps the most intriguing result of this study is that a component of the reward system – the dorsal striatum – was activated during the decision phase in which participants chose the level at which to blast the highly provocative participant with noise. This finding, which the authors called the “sweetness of revenge” (p. 209), suggests that aggression can be inherently rewarding, and thereby provides a neural basis that might partially explain why it is difficult to reduce retaliatory aggressive behavior. During the outcome phase, activation in another region of the reward system, the ventral striatum, was also observed. However, the authors concluded that this was most likely due to relief derived from the successful avoidance of the noise blast.

In summary, the brain regions associated with anger, angry rumination, and actual aggressive behavior, as well as issues addressed by cognitive theories of aggression and media violence, involve elements of both the X- and C-systems. The concept of a dual system harkens back to the days of Freud, who posited an innate form of destruction motivation (i.e., Thanatos) emanating from the *id* that resists control by the *ego* and *superego*. This notion of conflict between primitive aggressive urges and control of these urges remains with us today (Slotter & Finkel, Chapter 2, this volume). However, the X- and C-systems framework differs markedly from Freud’s notion of destructive drives. The X- and C- systems are compatible. When the situation calls for it, the C-system intervenes. For example, when someone cuts us off on the freeway while making an obscene gesture, the dACC sounds the neural alarm and snaps us out of our placid stream of experience. Our subsequent behavior, aggressive or otherwise, will depend on a number of factors, one of which is the activation in top-down control regions of the brain. I discuss this in more detail below.

The Role of Top-Down Control and Emotion Regulation Mechanisms

For the aggressor, aggressive behavior can have positive consequences (e.g., self-defense, achievement of dominance) and negative consequences (e.g., developing a bad reputation, instigating retaliation, and even being killed). Evolutionary theorists argue that in our ancestral past, aggression was a risky strategy, but when successfully executed, aggression likely increased reproductive success (e.g., MacDonald, 2008; and Sell, Chapter 3, and Shaver, Segev, & Mikulincer, Chapter 4, this volume). Although he did not use the X- and C-system framework, in a review of the literature on effortful control, MacDonald (2008) argued that a conscious system located in the PFC (C-system) allows humans to inhibit prepotent impulses toward aggression stemming primarily from limbic structures (X-system). When the X-system cannot solve a problem, it calls on the C-system. It is the C-system that allows individuals to make "explicit appraisals of costs and benefits" (MacDonald, 2008, p. 1014) that are "only…available through explicit processing" (MacDonald, 2008, p. 1015). These explicit appraisals play a key role in determining whether aggression will or will not occur, and it is precisely these appraisals that distinguish human aggression from the purely reflexive aggression observed in other animals.

The General Aggression Model (GAM; Anderson & Bushman, 2002; DeWall & Anderson, Chapter 1, this volume) highlights the importance of the explicit decision-making process. According to the GAM, appraisals and decision-making processes precede thoughtful or impulsive action. One key implication of the GAM and other models of effortful control is that individual differences in impairment of self-control should be related to aggression and impulsivity in general. Indeed, individual differences in trait aggression, executive functioning, and impulsivity are inter-related (MacDonald, 2008). Moreover, temporary experimental impairment in self-control increases aggression (DeWall, Baumeister, Stillman, & Gailliot, 2007;

Stucke & Baumeister, 2006; and Slotter & Finkel, Chapter 2, this volume). Acute alcohol intoxication has similar effects, likely via altereed activity in the dorsolateral PFC (Dao-Castellana et al., 1998).

In the modern world, aggression is still risky. One might even argue that the negative consequences (e.g., imprisonment, legal fees, social rejection) are typically a more likely outcome following aggression that any positive consequences. Thus, the ability to effectively weigh the costs and benefits of aggression is critical (Sell, Chapter 3, this volume). This is not to say that the C-system flawlessly functions in our best interest. Indeed, in modern society we still find that most aggressive acts are impulsive acts. For example, the majority of homicides and other aggressive crimes occur when people are provoked and angry and either explicitly decide to aggress or are simply unable to resist the motivation to behave aggressively. Thus, the immediate cause of many acts of aggression is often a loss of self-control (DeWall et al., 2007).

Substantial neuropsychological and imaging research indicates that the PFC is the seat of self-control (Banfield, Wyland, Macrae, Munte, & Heatherton, 2004), and integral parts of the circuit that underlie self-control are the dACC, dorsolateral PFC, and mPFC (Cohen, Botvinick, & Carter, 2000). The neural substrates of emotion regulation include the medial, ventrolateral PFC, and ventromedial PFC. The lateral and medial regions of the PFC share rich connectivity with cortical and limbic structures such as the dACC and ventromedial PFC, and have been implicated in emotion regulation and behavioral control (Inzlicht & Gutsell, 2007). Accordingly, Davidson et al. (2000) proposed that impaired functioning of an emotion regulation circuit involving the dACC, ventromedial PFC, and the dorsolateral PFC predisposes individuals to aggressive behavior. Indeed, engaging in self-control or completing neuropsychological measures that rely on inhibitory ability recruits the dACC and dorsolateral PFC (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Richeson et al., 2003). As noted above, both provocation and subsequent angry rumination recruit neural regions underlying executive control and emotion regulation mechanisms (Denson et al., 2009).

Part of the anger and aggression circuit, the dACC, has been dubbed a “neural alarm system” because of its role in detecting conflict (Eisenberger & Lieberman, 2004; Kross, Egner, Ochsner, Hirsch, & Downey, 2007). In the presence of an unjustified wrongdoing, there is likely a conflict between how people feel they should be treated and how they were actually treated (Sell, Chapter 3, this volume). This is consistent with the associations of the dACC with self-reported social distress and anger following ostracism and provocation (Denson et al., 2009; Eisenberger et al., 2003). Because there are costs and benefits to aggression, there is also likely to be a conflict between motivation to aggress and motivation not to aggress. As Krämer et al. (2007) suggest, when a person is unjustly wronged, it is likely that the dACC initiates regulatory behavior via activity in the dorsolateral PFC.

There is some evidence to suggest that exposure to media violence impairs top-down control mechanisms. Matthews et al. (2005) examined adolescents who had been diagnosed with a disruptive behavior disorder, including aggressive features, and matched controls who had either high or low levels of exposure to media violence. During neuroimaging, participants completed the Stroop task, which typically activates the dACC and dorsolateral PFC. The aggressive group demonstrated decreased activity, and this activation was not different from that in the normal adolescents who had been exposed to high levels of media violence. Only the normal adolescents with low levels of exposure to media violence demonstrated the typical pattern of lateral PFC and dACC activity during the Stroop task.

Conclusion and Future Directions

The evidence reviewed here implicates a network of neural regions that underlie anger, angry rumination, aggression, and media violence. Activity in these regions and the processes that they support are consistent with social psychological models of aggression, such as cognitive neo-association theory, script theory, and the GAM. The hostile aggression circuit described here implicates limbic and top-down prefrontal regions, which support both reflective and reflexive processes. Anger, pain, and social rejection are mediated by activity in the dACC, whereas angry rumination is mediated by the mPFC. There is also evidence that high levels of exposure to media violence are associated with abnormal functioning in the aggression circuit. Furthermore, during actual acts of aggression, neural regions involved in reward processing are active. This finding partially explains the difficulty associated with effectively reducing aggression.

Much work remains to be done. For instance, because alcohol is involved in a large number of aggressive acts, neuroimaging research investigating the pathways by which alcohol influences reactions to provocation seems worthy of investigation. Future research could also investigate genetic markers that might influence neural reactivity to provocation such as the MAOA polymorphism. Additional work could also investigate the connection between neural activity, other systems (e.g., cardiovascular, endocrine), and actual aggressive behavior in more detail. Furthermore, one might also examine the effects of interventions known to reduce aggression (e.g., distraction, self-control training) on long-term changes in neural reactivity. By grounding social psychological theory in brain processes, future research will expand our understanding of situational and personological influences on aggressive behavior.

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Acknowledgements

This chapter was supported by the Australian Research Council's *Discovery Projects* funding scheme (DP0985182). Thank you to Ajay Satpute for comments on an earlier draft of this chapter.