

# Intentional and Incidental Self-Control in Ventrolateral PFC

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The ability to exert self-control over one's thoughts and behaviors is crucial to successfully navigating the real world in a variety of domains, such as motor control (remaining in your seat during a boring lecture instead of jumping up and running outside), control over risky behavior (taking a sure option so as not to risk losing money), control over immediate temptation (choosing to delay a payment so as to receive a larger one at a later date), and emotional control (remaining composed and suppressing the desire to yell at someone who angered you). Each of these examples requires different actions to successfully exert control over the more desirable yet detrimental action. There are multiple clinical disorders that are related to impairments in control such as ADHD, substance abuse, and pathological gambling. Given the serious problems that may occur if one has difficulty exerting behavioral or affective self-control, it is critical to understand the mechanisms behind successful self-control and how they are changed when self-control ability is impaired.

As can be noted by the above examples, self-control is a broad concept that has been defined in many different manners. A good, general definition is that self-control is "the overriding or inhibiting of automatic, habitual, or innate behaviors, urges, emotions, or desires that would otherwise interfere with goal directed behavior" (Muraven et al., 2006). As is noted from this definition, many different methods can be used to study self-control, ranging from inhibiting a motor response to regulating an emotion to suppressing the temptation to eat sweets. In addition to these explicit, intentional forms of self-control, it is possible to exert control without an explicit goal to do so (i.e., automatically or incidentally) given the right situation. For example, in priming paradigms participants are not explicitly aware that they saw a prime, but the implicit encoding of primes can cause incidental behavioral control. Additionally, it is possible to implicitly or incidentally regulate an affective response without awareness (for a review, see Berkman & Lieberman, 2009).

It has been asserted that self-control ability may be like a muscle: it is a limited resource that can be fatigued with use, or trained to increase stamina (Muraven, 2010; Muraven & Baumeister, 2000; Muraven et al., 1999). Evidence for this assertion can be found in studies in which participants were required to first exert self-control in one domain, and then subsequently exert self-control in a different domain. The domains used were quite varied and included motor control (the stop-signal paradigm or squeezing a handgrip), controlling one's temptation to eat sweets or drink alcohol, and emotional control. It was consistently found that participants who were required to exert control two times in a row were worse on the second control task than were those who performed a difficult task that did not require self-control as their first task (such as solving mathematical problems or typing a paragraph quickly

without feedback; for a review, see Muraven & Baumeister, 2000). Moreover, not only was this self-control fatigue alleviated when participants practiced exerting self-control over an extended period of time (Muraven et al., 1999), but baseline self-control ability improved with practice (Muraven, 2010). It is important to note that the type of self-control practiced did not matter; self-control was improved across domains.

This research implies that multiple forms of self-control may be subsumed under one general control mechanism. Therefore, it is natural to turn to brain systems to determine whether different forms of control utilize the same, or at least overlapping, neural networks.

This chapter reviews the literature exploring the neural basis of self-control and asserts that the right ventrolateral prefrontal cortex (rVLPFC) is a neural region commonly recruited across many different forms of self-control. As used here, self-control is operationalized as inhibitory impulse control. This is one of multiple subprocesses of executive, or cognitive, control (Lenartowicz et al., 2010; Sabb et al., 2008). This operationalization is motivated by the hypothesis addressed here that the rVLPFC underlies inhibitory control, thus for the remainder of this chapter we will use the term “self-control” to refer to inhibitory impulse control.

It is important to note that “ventrolateral prefrontal cortex” is a broad term that covers a wide swath of brain (Figure 1). We purposely use such a broad term to be consistent with the literature, which refers to a range of distinct brain regions that fall into the ventral and the lateral prefrontal cortex. These regions include the inferior frontal junction (IFJ), the inferior frontal gyrus (IFG), the lateral orbitofrontal gyrus (OFG), and the ventral anterior middle frontal gyrus (MFG). The IFG can be further divided into three subregions: the pars opercularis, pars triangularis, and pars orbitalis (Duvernoy, 1991). The IFJ is defined as the junction of the inferior frontal sulcus and the inferior precentral sulcus (Derrfuss et al., 2004). When studies we discuss below report activity in subregions within the VLPFC we specify where in the VLPFC the activity was localized.

In this chapter we will first discuss the common activation of the rVLPFC across many different forms of self-control. We will first focus on explicit, intentional self-control such as motor control, control over risky behavior, the ability to delay gratification, and intentional emotion regulation. While self-control literature mostly limits itself to discussing these and other examples of intentional self-control, we argue that implicit, incidental processes can be considered self-regulation, or self-control, as well. Therefore, we will then focus on incidental self-control including incidental affect regulation, incidental behavioral control, and incidental pain regulation. Subsequently, we will discuss relevant anatomical and functional connections of the rVLPFC. Finally, we will review other hypothesized roles of the rVLPFC and attempt to resolve conflicting theories. We will conclude by stating that the rVLPFC is a brain region central to executive control that has different subdivisions with different roles. One main role of the rVLPFC is to exert self-control over behaviors.

## **1 The rVLPFC and Intentional Self-Control**

The rVLPFC is a strong candidate for a brain region that is central to exerting self-control. It is commonly activated across many different tasks requiring different forms of behavioral and affective self-control. It is also in a central location and well-connected to regions that may carry out control-related phenomena, such as motor control or emotional control. Historically, there has been a focus on the role of the rVLPFC in intentional forms of self-control. While activity in healthy participants is sometimes bilateral, the VLPFC is significantly active in the left hemisphere less often than in the right hemisphere (see Figure 2 and Table 1), and lesion and TMS studies point to the right, but not the left, VLPFC as being necessary for control (Aron et al., 2003; Chambers et al., 2007). Thus, while we mention relevant left VLPFC

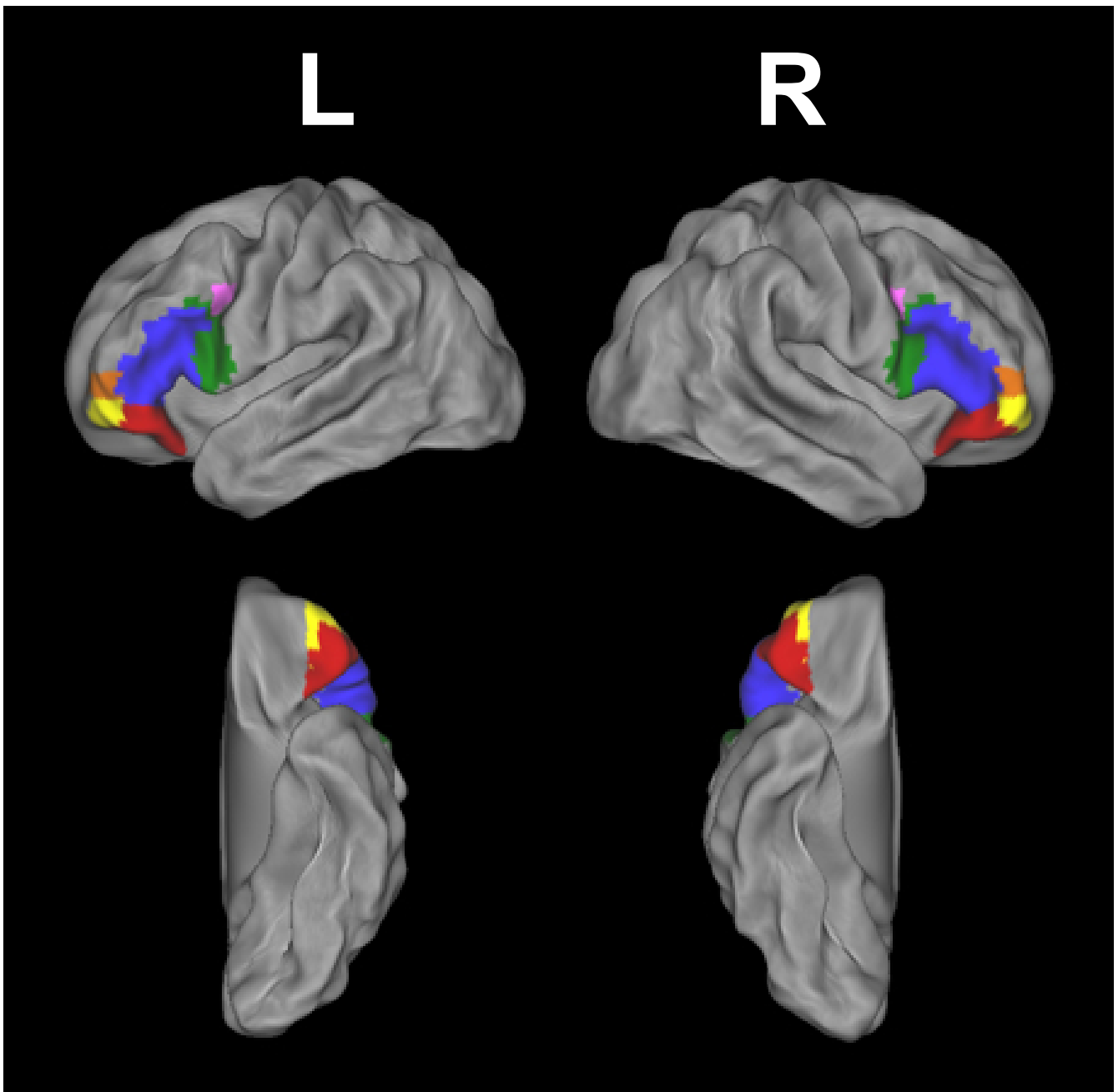


Figure 1: Lateral and ventral views of the anatomical subdivisions of the ventrolateral prefrontal cortex (VLPFC), which includes the inferior frontal junction (IFJ; in pink); the three subdivisions of the inferior frontal gyrus (IFG): pars opercularis (in green), pars triangularis (in blue), and lateral pars orbitalis (lateral to  $x = \pm 32$ ; in red); the lateral portion of the orbitofrontal gyrus (OFG; lateral to  $x = \pm 32$ ; in yellow); and the ventral anterior portion of the middle frontal gyrus (MFG; ventral to  $z=10$ ; in orange). These subdivisions of the VLPFC were taken from the Automated Anatomical Labeling (AAL) atlas (Tzourio-Mazoyer et al., 2002).

activation, our focus remains on the role of the rVLPFC. Moreover, it is important to note that other prefrontal brain regions are often activated during tasks that require self-control, such as the dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC), medial prefrontal cortex (mPFC), and or-

bitofrontal cortex (OFC). However, these regions may be recruited for other, non-inhibitory self-control-related task demands, such as rule monitoring (DLPFC; Bunge, 2004), performance/conflict monitoring (ACC; Botvinick et al., 2004), or the processing of emotions or rewards (mPFC/OFC; Elliott et al., 2000; Ochsner & Gross, 2005). Therefore, the role of these regions in self-control related tasks will not be discussed in depth here.

## 1.1 Motor Control

The control of motor responses is an oft-studied form of self-control. Generally, motor response inhibition is studied using the go/no-go (Casey et al., 1997b) and the stop-signal (Logan, 1994) tasks, in which an intended motor response simply has to be suppressed. Motor control can also be more complex, however, requiring the substitution of a novel response in addition to the suppression of the intended response. This is typically studied using response switching, or stop-change, paradigms (De Jong et al., 1995) or reversal learning paradigms (Clark et al., 2004).

Simple response inhibition tasks require participants to exert motor self-control by inhibiting a button press to a stimulus when they perceive a signal to stop their response. By altering the proportion of stimuli that are associated with stop-signals, the level of prepotency of responding can be manipulated. It becomes more difficult to inhibit a response when there are fewer stop-signals interspersed among the go stimuli. The dependent variables in the go/no-go task are number of commission errors (i.e., responding to a no-go stimulus) and number of omission errors (i.e., not responding to a go stimulus). The dependent variable in the stop-signal task is stop-signal reaction time (SSRT). SSRT is a measure of the time a participant needs to be able to inhibit his or her intended response. While go/no-go and stop-signal tasks are fairly similar, there is one key difference between them: the signal to stop. In the go/no-go task, the stop-signal is the stimulus itself (i.e., an “X” in a string of other letters that require a response). In the stop-signal task, the stop-signal is a signal that occurs after the onset of the primary go stimulus (i.e., a color change or an auditory tone). Given the difference in the stop-signals of the two tasks, it has been asserted that they may measure slightly different forms of motor control. It is possible that the go/no-go task may actually evaluate response selection ability, since the signal to withhold a response is given before the response is initiated. The stop-signal task, on the other hand, does not produce the signal to stop until after the go stimulus has been displayed, and consequently an intended motor response has already been initiated. This task may thus assess response inhibition ability (Rubia et al., 2001). Regardless of these differences, both tasks require motor control and neuroimaging results are quite similar across them.

Research in monkeys has pointed to a critical role of the VLPFC for motor control. In one study, lesions to the inferior frontal convexity, which corresponds to the human VLPFC, but not to the mPFC, impaired performance on go/no-go tasks (Iversen & Mishkin, 1970). In another study, single-cell recording in macaque monkeys found that inferior DLPFC neurons (analogous to the human VLPFC) responded selectively to either go or to no-go stimuli (Sakagami & Niki, 1994).

Lesion and transcranial magnet stimulation (TMS) studies in humans have confirmed the animal research that the rVLPFC is necessary in order to exert motor control. It has been found that lesions in the right inferior frontal gyrus (IFG) of the VLPFC impaired motor control and, critically, the extent of the lesions was positively correlated with longer SSRTs. The extent of the damage to no other regions in the frontal lobes, including those of the left IFG, correlated with SSRT (Aron et al., 2003). Other studies in patients with focal lesions to the frontal lobes have implicated the pre-supplementary motor area (preSMA) as a second area necessary for successful response inhibition performance (Floden & Stuss, 2006; Picton et al., 2007). A series of TMS studies have confirmed the results from the lesion studies. They have found that temporary disruption of the right IFG, but not middle frontal gyrus (MFG),

angular gyrus, dorsal premotor area, or left IFG, impairs SSRT on the stop-signal task (Chambers et al., 2007, 2006; Verbruggen et al., 2010). Literature is inconsistent whether disruption of the preSMA via TMS impairs (Chen et al., 2009) or does not impair (Verbruggen et al., 2010) response inhibition.

Human neuroimaging studies, while not speaking to the necessity of the rVLPFC in motor control, have consistently found the involvement of rVLPFC during successful performance on the go/no-go task (Buchsbaum et al., 2005; Garavan et al., 2002, 1999; Konishi et al., 1998; Liddle et al., 2001; Menon et al., 2001; for a review, see Chikazoe, 2010) and the stop-signal task (Aron & Poldrack, 2006; Boehler et al., 2010; Chevrier et al., 2007; Congdon et al., 2010; Kenner et al., 2010; Rubia et al., 2003; for a review, see Aron et al., 2004). Significantly, the involvement of the rVLPFC in simple response inhibition holds across different modalities, such as eye movements (Chikazoe et al., 2007) and speech production (Xue et al., 2008a). It should be noted that VLPFC involvement in motor control may be bilateral, but is often predominantly right-lateralized. Additionally, greater rVLPFC activity has been associated with lower, and therefore better, SSRT (Aron & Poldrack, 2006; Congdon et al., 2010).

It is currently under debate whether rVLPFC activity reflects a self-control mechanism (Aron, 2011; Chikazoe, 2010; Verbruggen et al., 2010) or an attentional mechanism (Hampshire et al., 2010; Sharp et al., 2010). This debate will be addressed further below. It is important to note that other prefrontal and subcortical regions, the DLPFC, ACC, preSMA, and subthalamic nucleus (STN) in particular, are also often associated with successful motor control. These regions are likely involved in certain aspects of cognitive control during these tasks, although since this chapter is focusing on the role of the rVLPFC in inhibitory self-control, they are not discussed further.

Literature examining simple motor control with the go/no-go and stop-signal tasks consistently and fairly specifically implicates the rVLPFC in the self-control of motor responses (for a review, see Aron et al., 2007b). The rVLPFC is similarly implicated in more complex forms of motor control that require both response inhibition and the substitution of a different response. Response switching paradigms, for example, combine a simple stop-signal task with the requirement to push a new button after response withholding (De Jong et al., 1995). It has been found that a very similar network, including the rVLPFC, is involved in response switching as compared to the simple stop-signal task (Kenner et al., 2010). Moreover, TMS to the rVLPFC impairs one's response switching performance (Verbruggen et al., 2010).

Reversal learning is another complex form of motor control that has been studied more extensively than response switching. It requires the inhibition of a prepotent response and the substitution of that response with an alternative that participants previously were told to avoid. Often, reward and punishment are used to develop prepotent response tendencies and to alert participants to the need to replace those response tendencies (Clark et al., 2004).

The lateral OFC, which lies within the larger VLPFC, has been implicated as necessary for reversal learning in research with animals. OFC lesions consistently impair reversal learning in rats and primates (Clark et al., 2004; Ragozzino, 2007). Lesions of the medial OFC, anterior OFC, and DLPFC, on the other hand, do not impair reversal learning in rhesus monkeys (Butter, 1969; Dias et al., 1996; Iversen & Mishkin, 1970).

Similar to the animal literature, human lesion studies have found that the OFC is critical for successful reversal learning but not initial learning (Fellows & Farah, 2003; Hornak et al., 2004; Mitchell et al., 2006; Rolls et al., 1994). Lesions in humans tend to be less focal than those in animals since they cannot be caused in a controlled setting, thus a lateral/medial distinction has not been made with the human lesion studies. Neuroimaging studies with healthy participants have been useful in more specifically determining the brain regions involved in reversal learning in humans.

Reversal learning studies with healthy adult participants generally use probabilistic reward contingencies such that participants are given incorrect feedback on a percentage of responses, often 20% to 30%. This method increases task difficulty and therefore the number of post-reversal errors that can then be an-

alyzed in event-related fMRI designs. Moreover, reversals tend to occur after a range of correct responses in a row (e.g., anywhere between 10 and 15 correct responses). Taken together, these approaches ensure that a reversal is not predictable (Cools et al., 2002). Reversal learning studies consistently find that the last incorrect post-reversal trial before a successful response switch (a final reversal error) activates the lateral OFC/VLPFC more than correct trials, incorrect trials where participants did not subsequently change their response, or control tasks not requiring a decision to be made (Cools et al., 2002; Freyer et al., 2009; Kringelbach & Rolls, 2003; O'Doherty et al., 2003; Remijnse et al., 2005). VLPFC activity has also been noted when looking at all incorrect post-reversal trials as compared to correct trials (Mitchell et al., 2009). In one of these studies that focused on the neural response to errors, it was found that the rVLPFC was active only for the last incorrect trial before a behavioral reversal as compared to correct trials, but not for initial errors after a reversal (when the response was not subsequently changed) or for probabilistic errors as compared to correct trials (Cools et al., 2002). In other words, the rVLPFC was active only when the participants realized that their prepotent response had to be inhibited, but not for errors generally. Instead of specifically focusing on final reversal errors, some studies have examined epochs of reversal learning tasks and compared neural activity on post-reversal trials with that during initial learning, when there is no prepotent response that must be suppressed. These studies have also found that there is more rVLPFC activity for post-reversal trials as compared to initial learning trials (Ghahremani et al., 2010; Xue et al., 2008b). Taken together, these studies indicate that the role of the rVLPFC in reversal learning, like that in simple motor control tasks, may be to exert behavioral self-control over a prepotent motor response.

The reversal learning literature supports the theory that the ventral PFC, and specifically the OFC, can be functionally separated into lateral and medial regions. It is theorized that the medial OFC tracks dynamic reward contingencies, while the lateral OFC exerts behavioral control based on the realization that those contingencies have changed (Elliott et al., 2000).

As can be noted from the above review of motor control literature, evidence consistently supports a role for the rVLPFC, the IFG and lateral OFC in particular, in the behavioral control of prepotent responses, whether the task requirements are simple and only entail the suppression of a motor response, or more complex and additionally necessitate the substitution of a novel motor response.

## **1.2 Risk-Taking Behavior**

Risk-taking behavior can manifest itself in many different manners, such as substance use, gambling, or driving without a seatbelt. There is a sense that people who engage in any risky action are behaving impulsively, or lacking the self-control necessary to take the more difficult but more responsible action. While some of the processes behind control over risk-taking behavior may be similar to those required in motor control (i.e., relatively rapid decision-making and suppression of the prepotent, easier, or more desirable response), a major difference between these two types of control is the addition of external rewards in risk-taking. This may change the subjective experience of the participants, as well as the strategies used to exert self-control. Additionally, while the decision-making is fairly rapid in both motor control and control over risk-taking behavior, motor control usually occurs on the order of milliseconds, while control over risky behavior can occur on the order of milliseconds or seconds. Risk-taking is often studied in the laboratory using tasks that invoke gambling behavior because they can use simple stimuli and the potential for being rewarded with money is universally appealing. While participants can alternately be given self-report questionnaires, many do not fill them out accurately due to lack of insight or self-presentational concerns (Lejuez et al., 2002).

The Iowa Gambling Task (IGT; Bechara et al., 1994) is one of the earliest gambling tasks used to assess risky behavior. In this task, participants choose cards from four different decks. Two of these

decks are “advantageous” and associated with small rewards and small losses, with an overall gain. The other two decks are “disadvantageous” and associated with large rewards and large losses, but an overall loss. Patients with ventromedial prefrontal cortex (VMPFC) lesions tend to make risky choices that result in potentially higher gains in the short term but an overall lower payoff (Bechara, 2004; Bechara et al., 1994, 1998). It has been hypothesized that this tendency to make maladaptive, risky decisions is due to the impairment of emotional circuitry after VMPFC lesions (the somatic marker hypothesis; Bechara, 2004; Damasio, 1994). This theory states that healthy decision-making requires a link between autonomic responses to risky and emotional stimuli and control regions in the brain, such as the VMPFC. However, poor performance on the IGT has also been seen in patients with DLPFC lesions (Fellows & Farah, 2005; Manes et al., 2002), a region not associated with risky decision-making or emotional circuitry. Therefore, whether this poor performance is due to risky impulses or to other processes hypothesized to be involved in the task, such as learning outcome probabilities, long-term strategy development (Manes et al., 2002; Wu et al., 2004), or reversal learning (Dunn et al., 2006; Fellows & Farah, 2005) is under debate. However, it is understood that this is not a task that purely measures the lack of self-control associated with risky behavior. Even given the potential confounds of the IGT, a few neuroimaging studies have been conducted examining the neural regions involved in successful performance. In a PET study, overall earnings on the IGT were correlated with the magnitude of regional cerebral blood flow in the rVLPFC, as well as in the right anterior insula and the right head of the caudate nucleus (Ernst et al., 2002). In other words, participants who were able to suppress the impulsive urge to choose short-term higher gains so that they could maximize long-term higher gains utilized a right-lateralized network including the rVLPFC more than participants who responded based on those impulsive urges. As mentioned before, it is important to realize that self-control over risky behavior may be confounded with learning, strategy development, or reversal learning in this task.

In an attempt to separate the processes involved in risky decision-making from the confounding processes found in the IGT, Rogers and colleagues developed the Cambridge Gamble Task (CGT; Rogers et al., 1999a) and the Cambridge Risk Task (CRT; Rogers et al., 1999b). In these tasks, each trial is independent so there is no learning or strategy development that can occur. A token (worth a variable amount of points) is hidden behind one of many red and blue boxes on the computer monitor. Participants must choose the color of the box it is hidden behind. The proportion of red:blue boxes is manipulated in order to make some choices riskier than others. Importantly, in order to maximize one’s winnings, a participant must inhibit the risky but more appealing choice of gaining more points in order to make the safer bet. The researchers found that patients with OFC lesions (including lateral OFC) were slower and made more risky, maladaptive decisions on the CGT than did healthy control participants and patients with DLPFC and mPFC lesions, who performed equivalently to controls (Rogers et al., 1999a). A subsequent study found that patients with both VMPFC and insula lesions (including the posterior VLPFC) were riskier than control participants, but that only the patients with insula lesions did not adjust their risk-taking based on probabilities and as a result went bankrupt more than both controls and patients with VMPFC lesions (Clark et al., 2008). In a PET study with the CRT, greater rVLPFC activity was associated with decision-making on trials that involved making a decision about riskier options (i.e., trials in which the ratio of red:blue boxes was 4:2 or 5:1) as compared to safer options (i.e., trials in which the ratio was 3:3; Rogers et al., 1999b). Unfortunately, no analyses were conducted based on participant choice, so no conclusions can be drawn regarding whether this rVLPFC activity was related to choosing the safe option, choosing the risky option, or the decision-making process in general.

Other studies examining risky decision-making with gambling tasks appear to have somewhat inconsistent results. In a meta-analysis, Krain and colleagues (2006) concluded that the lateral OFC and the medial PFC are both generally involved in risky decision-making, but neither are associated specifically with making risky choices (i.e., with more impulsivity) or safe choices (i.e., with more self-control).

Contrary to this conclusion, it has been found that regions within the VLPFC were active specifically when participants made safe, as compared to risky, choices (Matthews et al., 2004). Moreover, a study with lesion patients found that participants with VLPFC lesions (some were bilateral and some were confined to the left hemisphere) made riskier choices than participants with non-frontal lesions and controls (Floden et al., 2008). Alternately, however, it has also been found that the right OFC/VLPFC was more active for risky as compared to safe trials (Cohen et al., 2005; Ernst et al., 2004; Eshel et al., 2007). It is critical to point out, however, that many studies exploring risky decision-making either focus on the decision phase without taking choice into account (Ernst et al., 2004), or control for the expected value of the decision, meaning that it is not detrimental to choose the riskier option. These studies, therefore, may be measuring risk preference more than control over negative, risky impulses (Cohen et al., 2005). To support this, studies specifically examining risk-preference (as opposed to the neural correlates of risk-taking when it is accompanied by negative consequences) have found that there is more activity in bilateral lateral OFC/IFG (Engelmann & Tamir, 2009) and more connectivity between the right IFG and the anterior insula (Cox et al., 2010) in risk-seeking individuals than in risk-averse individuals. Another study examining risk preference found that the rIFG was more active when making less risky choices, but only in risk-averse participants, and that it was more active if people were more risk-averse than risk-seeking, but only on trials that were relatively low-risk (Christopoulos et al., 2009). These findings imply that it is critical to take risk preference into account, as the decision-making process is different in risk-averse and risk-seeking individuals.

In addition to examining the overall neural response during risky decision making, some studies have examined individual differences and have found that lateral OFC activity was negatively correlated with number of risky choices (Eshel et al., 2007) and positively correlated with risk aversion (Tobler et al., 2007), both relationships indicating that a tendency toward making safer choices is related to lateral OFC activity.

Another task that has been used to explore risky decision-making is the Balloon Analogue Risk Task (BART; Hunt et al., 2005; Lejuez et al., 2002). In this task, participants are told to inflate a balloon worth a small amount of money (e.g., 10 cents) with a button press. The button press inflates the balloon and increases its worth by a constant amount (e.g., 5 cents). When it is inflated too much, however, the balloon explodes and the participant loses all the money gained on that trial. If the participant ends the trial before the balloon explodes, the balloon's worth is added to a pool of winnings. The average number of pumps before an explosion and the amount of money each inflation is worth can be varied to study the nuances of risky behavior. This task is an appealing alternative to gambling tasks because it is simpler, provides immediate feedback, and, as sometimes occurs in the real world, risky behavior is rewarded up to a point before it is punished (Lejuez et al., 2002). Behaviorally, number of pumps has been associated with a variety of self-reported risk-taking and impulsive behaviors in healthy adults, such as smoking, drinking, drug use, gambling, stealing, unprotected sex, not using seatbelts, and impulsivity-related subscales of the Barratt Impulsiveness Scale, the Eysenck Impulsiveness Scale, and the Sensation Seeking Scale (Lejuez et al., 2002). Crucially, the relationship between risky performance on the BART and responses on the self-report scales was specific to risk-taking; it was not correlated with anxiety, depression, or empathy (Lejuez et al., 2002).

The neural correlates of the BART are beginning to be explored. There has been one published study exploring risky decision-making on the BART in healthy adults (Rao et al., 2008). The purpose of this study was to examine the differences between active risky behavior and passive risky behavior (when the computer instructed participants what action to take). Therefore, the authors did not investigate the differences in safe versus risky decisions. Some preliminary data suggests that the rVLPFC is active, along with the ACC, DLPFC, parietal and occipital regions, the basal ganglia, and hippocampus, when suppressing risky responding in order to cash out on the BART (Cohen & Poldrack, 2009). Therefore,

preliminary data from the BART and data from other risk-taking tasks imply that the rVLPFC, especially the lateral OFC, is involved in decision-making when confronted with risky choices. While the findings are not entirely consistent, most of the literature supports the theory that the rVLPFC is specifically involved in suppressing risky choices (i.e., in exerting control over the urge to take risks). However, it is important that future research takes the expected value of the risky options and risk-taking preferences into account in order to more completely understand the role of the rVLPFC in risky decision-making.

### **1.3 Temporal Discounting**

Temporal discounting is a phenomenon that is often used to measure impulsive behavior and therefore a lack of self-control. It is appealing because the tendency to temporally discount rewards can be measured both in animals and in humans. In animals, temporal discounting is usually measured in studies where the animals are allowed to push a button or a lever at will, but are administered larger rewards of food or drink if they wait longer between button or lever pushes. In some studies with humans identical procedures are utilized (e.g., thirsty participants are given the option of whether to receive a smaller juice award immediately or a larger juice award at a delay). Because more abstract concepts can be measured in humans than in animals, other temporal discounting studies will give humans a choice between receiving a smaller amount of money immediately (e.g., \$5) or a larger amount of money at a delay (e.g., \$20). Amounts of candy, drugs, or anything else deemed rewarding can be manipulated as well, with the common goal of measuring how steeply participants discount the subjective value of future rewards or how well they are at delaying gratification and waiting for a larger, future reward. Animals and humans who tend to prefer smaller amounts of a reward immediately are thought to be impulsive, or lacking self-control, because their desire for an immediate payoff cannot be controlled even though it would provide long-term benefit to do so. This form of self-control differs from motor control and control over risk-taking behavior because it is more slow and deliberate and assesses long-term, instead of immediate, impulsivity. In other words, decisions are made about stimuli with outcomes sometime in the future, as opposed to having instantaneous consequences.

Based on research in animals such as pigeons, rats, and primates, it has been hypothesized that rewards are discounted temporally in a hyperbolic fashion, meaning that the tendency to choose immediate rewards drops off steeply with time. These studies often focus on the effects of lesions on temporal discounting behavior. Focal lesion studies have identified two regions that are associated with impulsive discounting behavior: the nucleus accumbens core and the OFC (Cardinal, 2006; Mobini et al., 2002). Additionally, single cell recordings in intact nidopallium caudolaterale in pigeons, which corresponds to the human prefrontal cortex, have identified cells that fire during the delay between decision and reward when choosing the larger, delayed option. Moreover, when the larger, delayed reward is chosen over the smaller, immediate reward, activity in these cells has been shown to be negatively correlated with delay length (Kalenscher et al., 2005). Cells with similar firing patterns have also been identified in rhesus monkeys (Roesch & Olson, 2005). In both pigeons and monkeys, these delay-sensitive cells also fire more for greater reward magnitudes. In other words, these OFC cells appear to code for the subjective value of the rewards, incorporating both delay, which decreases subjective value, and reward, which increases subjective value (Kalenscher et al., 2005; Roesch & Olson, 2005). It has been demonstrated that cells in other prefrontal areas in the monkey, such as the DLPFC, frontal eye fields, supplementary eye fields, premotor area, and supplementary motor area, do not code for delay length, implying that sensitivity to subjective value is specific to the OFC (Roesch & Olson, 2005).

There have not been many functional neuroimaging studies in healthy humans exploring the neural systems underlying the exertion of control over impulsive behavior during temporal discounting. In an early temporal discounting study in humans, McClure and colleagues (2004) found two dissociable neural

systems involved when participants were choosing between a smaller monetary reward sooner or a larger monetary reward later. They found one network, including limbic areas such as the ventral striatum, medial OFC, and mPFC, that was active for all trials in which an immediate choice was available. They found another network, including the rVLPFC/lateral OFC and DLPFC, that was active for all trials in which two delayed options were offered. Crucially, these areas were more active during difficult, as compared to easy, decisions. Difficult trials were defined as those in which the magnitude of the two options was relatively similar and there was more variability in participant responses. When comparing the relative activation of these two networks during trials in which one option was immediate, the lateral prefrontal network was more active than the limbic network when the delayed option was chosen, while there was a trend toward more activity in the limbic network as compared to the lateral prefrontal network when the immediate option was chosen. Similar regions were found to underlie temporal discounting behavior when participants were deciding between smaller primary rewards sooner (juice or water) or larger primary rewards later (McClure et al., 2007).

In a second study, methamphetamine abusers were compared to healthy adult control participants. Similarly to the study conducted by McClure and colleagues (2004), when difficult choices were compared to easy choices there was significantly more activity in the rVLPFC for difficult choices in both participant groups. Furthermore, participants who discounted delayed options less on difficult trials had greater activity in the VLPFC (in the left hemisphere) than those with steeper discounting curves (Monterosso et al., 2007). In a third study that also explored the relationship between one's tendency to discount rewards temporally and VLPFC activity, it was found that participants who exhibited less temporal discounting of rewards had more bilateral VLPFC activity (localized to the IFG) than those who discounted delayed rewards more (Wittmann et al., 2007). A fourth key study confirmed those findings; it was found that the lateral OFC was the only brain region to correlate with the tendency to choose larger, delayed rewards (Boettiger et al., 2007).

Lastly, it was found in a delayed reward task that participants who chose to delay the receipt of large immediate rewards to ultimately avoid large losses and end up with larger rewards at a delay had neural activity in a network including bilateral VLPFC, as well as DLPFC, dorsal premotor area, parietal regions, and subcortical regions (Tanaka et al., 2004).

While only a small number of neuroimaging studies have explored the neural correlates of temporal discounting in healthy humans, what does exist suggests that the rVLPFC is involved when exerting self-control over impulsive urges and behavior and a decision is made to delay gratification for a larger payoff in the future.

## **1.4 Emotion Regulation**

It is often adaptive to be in touch with and be able to express one's own emotions. However, there are some situations in which that is not appropriate, such as if a person falls and hurts him or herself in a manner that a bystander finds amusing. In that situation, it is beneficial to be able to exert self-control over one's emotions. Emotion regulation, the process by which people influence their emotional experience and expression (Gross, 1998), is a method that has been studied in order to understand the mechanisms behind self-control over affective processes. Prefrontal lesions, specifically VMPFC lesions, have been shown to cause impairments in both emotion expression and emotion regulation (Anderson et al., 2006; Barrash et al., 2000). Moreover, it is possible that some mental disorders, such as anxiety or depression, may in part be caused by the inability to regulate affect. Thus, there has been much interest in discovering how successful emotion regulation can be exerted (Gross, 1998). Emotion regulation differs from the afore-mentioned types of intentional self-control because there is no behavioral outcome measure.

The regulation of one's emotions can be explicit and intentional, or implicit and unintentional (Mauss

et al., 2007). This section will focus on intentional emotion regulation, in which participants are actively trying to regulate their emotional experiences. Multiple strategies may be used in order to control emotions. Gross (1998; 2002) developed a process model of emotion regulation that separates control over one's feelings into two broad categories: antecedent-focused and response-focused. Antecedent-focused strategies are used to alter one's appraisal of a situation before an emotion is experienced. These may include strategies that are not directly relevant to emotion regulation per se, such as avoiding a situation that may bring about an emotional response or changing a situation so an emotional response is not elicited. Other strategies more specifically act on inhibiting the occurrence of a soon-to-be experienced emotion, such as the deliberate deployment of attentional resources away from the emotion-eliciting stimulus or the cognitive reappraisal of a situation so it is not as emotionally salient. Response-focused strategies such as distraction, on the other hand, focus on changing an emotion after it has already been experienced. This can be achieved via direct modulation of a current affective state, either by suppression or enhancement (Gross, 1998, 2002). This section will focus on literature examining both antecedent-focused and response-focused strategies in intentional emotion regulation that are utilized after the emotional stimulus has been experienced (i.e., cognitive reappraisal of emotional stimuli to reduce their emotionality or suppression of already-experienced negative emotions, but not avoidance of an emotional stimulus before it has been experienced). Unintentional emotion regulation will be discussed below.

There is a large amount of literature focusing on the neural correlates of intentional emotion regulation. Most commonly, participants view images that are neutral or elicit negative emotions and are asked to use a technique called cognitive reappraisal to decrease the intensity of the emotion felt toward the negative images (Goldin et al., 2008; Harenski & Hamann, 2006; Kim & Hamann, 2007; Levesque et al., 2003; McRae et al., 2010; Ochsner et al., 2004; Phan et al., 2005; Wager et al., 2008). In cognitive reappraisal, participants are trained to redefine an image in a non-emotional, less negative manner. For example, an image of a person with a gruesome bullet wound may be described as an image of an actor in a movie covered in fake blood. In these studies, the rVLPFC, including the lateral OFC and IFG, is consistently implicated when suppressing as compared to maintaining a negative emotional reaction to an image (Harenski & Hamann, 2006; Kim & Hamann, 2007; McRae et al., 2010; Ochsner et al., 2004; Phan et al., 2005; Wager et al., 2008). Similar involvement of the rVLPFC has been noted when reappraising sad or negatively valenced films (Goldin et al., 2008; Levesque et al., 2003) and when suppressing anxiety resulting from the anticipation of shocks (Kalisch et al., 2005). This finding is consistent not only across different emotions and stimuli, but across strategies as well. In one study comparing cognitive reappraisal to expressive suppression (the inhibition of facial expressions and verbal utterances related to an emotion), the rVLPFC was involved during both reappraisal and suppression. Interestingly, the timecourse of rVLPFC involvement was different across the two strategies. In this study, participants were shown film clips for 15 seconds and told to either reappraise or suppress their natural emotional reaction to the film clips. While the rVLPFC was involved early in the trial for reappraisal (0-4.5 sec), it was involved later in the trial for suppression (10.5-15 sec; Goldin et al., 2008). Another study compared cognitive reappraisal with cognitive distraction (participants were asked to perform a concurrent memory task). This study found that the rVLPFC was involved in both techniques and there was no difference in rVLPFC activity across reappraisal and distraction (McRae et al., 2010). These studies indicate that while the rVLPFC is involved during emotional self-control utilizing multiple strategies, it is differentially involved based on the specific strategy used; presumably it is only active when the self-control is being implemented. All the above studies highlight that the rVLPFC may play a role in exerting self-control across a variety of forms of emotion regulation, in addition to a variety of forms of self-control. It is important to note that the rVLPFC is not the only brain area active during emotional self-control. Other prefrontal regions such as the mPFC, ACC, and DLPFC are commonly active, as are subcortical regions such as the amygdala. Additionally, there is often activity seen in the left VLPFC as well (Goldin

et al., 2008; Harenski & Hamann, 2006; Kalisch et al., 2005; Kim & Hamann, 2007; Levesque et al., 2003; McRae et al., 2010; Ochsner et al., 2004; Phan et al., 2005; Wager et al., 2008). However, a recent review (Berkman & Lieberman, 2009) indicates that the rVLPFC is the most commonly activated region across different emotion regulation tasks.

Importantly, these findings are specific to decreasing emotions. In one study where participants were instructed to increase negative emotions there was less activity in the right-lateralized network seen when decreasing negative emotions, and more activity in the left amygdala. This upholds findings that the amygdala is involved in the subjective experience of negative emotions (Ochsner et al., 2004). Interestingly, a negative relationship has been found between rVLPFC and amygdala activity, implying that the rVLPFC may play a role in suppressing the amygdala's natural response to negative emotions (Banks et al., 2007; Hariri et al., 2000, 2003; Lieberman et al., 2007; Phan et al., 2005; Wager et al., 2008). A second study comparing decreasing and increasing both negative and positive emotions found that rVLPFC was involved in decreasing both negative and positive emotions (although more so for negative emotions), but not for increasing negative or positive emotions (Kim & Hamann, 2007).

It has been asserted that many of the neural activations that are thought to be associated with cognitive reappraisal may in fact be due to eye movements and not emotion regulation (van Reekum et al., 2007). However, this study was done in older adults (ages 61-65), who have been shown to have different patterns of brain activity (Wincoff et al., 2011) and functional connectivity (Urry et al., 2006) during emotion regulation than younger adults. Additionally, participants were cued whether to reappraise the emotional stimuli four seconds after each stimulus was initially presented, while most emotion regulation studies give participants instructions before the stimuli appear on the screen (Goldin et al., 2008; Harenski & Hamann, 2006; Kalisch et al., 2005; Kim & Hamann, 2007; Levesque et al., 2003; McRae et al., 2010; Ochsner et al., 2004; Phan et al., 2005; Wager et al., 2008). This procedural difference could have caused a strategy shift and therefore the eye gaze results may be specific to the age group and procedure utilized in this study (van Reekum et al., 2007).

Critically, a relationship between the magnitude of rVLPFC activity and self-reported decrease in negative emotions has been found, indicating that this region is involved in the control of emotions (Ochsner et al., 2004; Phan et al., 2005; Wager et al., 2008). Increased negative coupling between the VLPFC and the amygdala during cognitive reappraisal has also been associated with less self-reported negative affect, indicating that there is a behavioral correlate to the antagonistic relationship between these two regions (Banks et al., 2007). Therefore, the rVLPFC is not only consistently active when people suppress their emotions in a variety of contexts, but its magnitude is related to the degree of emotional self-control as well.

## **2 The rVLPFC and Incidental Self-Control**

Traditional theories of self-control assume that it is a deliberate act. In this view, engaging in an act of self-control requires at least: an intention to regulate one's behavior, awareness of the fact that self-control is occurring, and expenditure of limited top-down cognitive control resources (here called "effortful" processes). However, these assumptions have recently been challenged based on both behavioral (Custers & Aarts, 2010) and neural (van Gaal et al., 2010) evidence. There is now a growing body of evidence that self-control (and particularly emotional self-control) can be engaged without attention, outside of awareness, and with little effortful processing. This section will briefly review studies providing insights into the neural bases of this kind of incidental self-control, which we termed earlier as "incidental self-regulation" (Berkman & Lieberman, 2009).

## 2.1 Defining Incidental Self-Control

A preliminary challenge is simply defining incidental self-control. This is particularly important given that participants by definition cannot report on something that occurred outside of their awareness. As such, self-report can only provide an indirect measure that self-control occurred by indexing pre- to post-control change in a response channel. If incidental self-control occurs outside of subjective awareness, how are we to measure whether it occurred at all?

Drawing insight from affective science, the best evidence for incidental control can be obtained using a combination of multimethod assessment, peripheral, and central physiology (cf., Cacioppo et al., 2000; Gross & John, 1998). We maintain that emotion reactivity and regulation can be measured without self-report so long as other channels of emotion responding are isolated by the task and measured. Nonetheless, researchers must be cautious in determining the presence of or change in an emotional state based on neuroimaging data alone. Many of the studies reviewed in this chapter do employ multimethod assessment, but many do not. In light of this, there are four special considerations for determining that incidental self-control has occurred (Berkman & Lieberman, 2009).

1. The habitual or prepotent response must be established in the relevant domain during some trials not requiring self-control. For example, participants must produce a go response in a go/no-go task or an affective response in an emotion regulation task following some stimulus (e.g., the go signal or an affective image) in the absence of attempts to exert control. This is particularly important in the affective domain, as researchers occasionally employ nominally “affective stimuli” without a manipulation check and assume that viewing those stimuli will produce an affective response.
2. A condition measuring self-control should produce reductions of the prepotent response (e.g., no-go trials or reductions in affective responding) under conditions where those responses would have otherwise occurred.
3. An “incidental manipulation check” should be used following the experiment to ensure that participants (1) did not intend to control the prepotent response and (2) were not aware that self-control might have occurred. This second condition—awareness—is important for eliminating the effects of demand characteristics. For example, participants who are repeatedly asked about their distress level following exposure to affective stimuli may become aware that some aspect of the task is expected to reduce their distress, even if they do not intend to control it.
4. The alternative explanations of task difficulty and distraction in particular, but also several others, must be ruled out as possible causes of the self-control effect. For example, demonstrating that participants successfully withhold a go response on no-go trials when they are also blasted with extremely loud bursts of white noise is not evidence that white noise produces incidental no-go responses. Likewise, showing that participants who view affective stimuli during a complicated split-attention paradigm fail to produce an amygdala response on those trials is not evidence that split-attention unintentionally reduces emotional reactions to emotional stimuli outside of awareness.

It should be noted that the processes that launch incidental self-control need not occur entirely outside of awareness; we claim only that incidental self-control remains unintentional and outside of awareness. In many cases, an entirely intentional process results in unintended and incidental self-control. In this sense, incidental processes are similar to behavioral automaticities, which are thoughts or behaviors that occur outside of awareness and without deliberate intent but can be initiated by consciously-perceived primes such as a word search (Bargh, 1984). As with incidental self-control, what matters is not whether

the stimulus or action that triggers the automatic behavior is perceived, but whether the actor knows the relationship between the trigger and the automatic behavior.

Below, we review neuropsychological studies on a variety of topics that meet these four criteria for incidental self-control. Nearly all of the studies reviewed find activation in the rVLPFC during incidental self-control. The topics include: incidental affect regulation produced by labeling, contextual task demands, or trait-driven spontaneous regulation; incidental behavioral control using priming of inhibition or relationship maintenance motives; and incidental regulation of pain responses based on beliefs about a placebo or use of religious prayer.

## 2.2 Incidental Self-Control of Emotion

One of the most direct demonstrations of incidental affect regulation comes from studies using the affect labeling paradigm (Hariri et al., 2000; Lieberman et al., 2007). In this paradigm, participants are presented with an emotion face target and are instructed to identify the emotion depicted in the face by matching it to a similar emotional face ('match') or to a linguistic label for the emotion ('label'). In both cases the comparison is made by selecting one of two options (either emotional faces or emotion words) that best corresponds to the target face. A task matching geometric shapes is used as a control condition. In both cases participants are attending to affective features of the stimulus. The critical difference between them is whether the comparison emotional information is represented visually (faces) or linguistically (words).

Results from these studies consistently find greater activation in rVLPFC during 'label' than 'match', and greater bilateral amygdala activity in 'match' than 'label'. In one study, connectivity analyses revealed greater inverse connectivity between the amygdala and the rVLPFC during 'label' than 'match' (Hariri et al., 2000). Together, this pattern of findings suggests an inhibitory relationship between amygdala and rVLPFC that is specifically engaged during the processing of linguistic affective information.

How is affect labeling a form of emotion regulation? It is not, according to conventional definitions of emotion regulation, because it lacks both the intention to reduce affective experience and the awareness that the reduction is occurring. However, although those differences qualify affect labeling as a distinct strategy phenomenologically, we suggest that it is similar to intentional emotion regulation in several key ways. Across studies, labeling has been shown to reduce affective responding in three response channels. Labeling elicits similar reductions in subsequent self-reported affect and physiological responding (Lieberman et al., 2011; Tabibnia et al., 2008), and shares a similar (but not identical) pattern of neural activity with intentional emotion regulation.

Another type of incidental affect regulation occurs when contextual factors alter an affective response outside of awareness. Even without self-reports of emotional experience, this effect can be observed using neuroimaging by identifying limbic system activity in one condition, and then a pattern of decreased activity in those same limbic structures coupled with increased prefrontal cortical activity in the other condition. This conclusion would be further bolstered by showing parallel effects in additional affect response channels. Together, these findings would suggest that some process beyond simple disengagement from the affective stimuli is involved in the limbic reductions.

For example, Berkman and colleagues examined the impact of intentional control of a motor response (i.e., a form of behavioral self-control) on the brain activity of participants while they viewed affective images (Berkman et al., 2009). In this study, participants viewed negative emotional images while performing a go/no-go task. Importantly, the affective content of the images—facial expressions—was entirely irrelevant to the task, which required behavioral responses based on the gender of the face. The contextual factor of whether intentional motor control was engaged produced incidental affect regulation. Specifically, the amygdala responses that were otherwise present when participants viewed negative im-

ages without motor control (on go trials) were significantly reduced during motor control (on successful no-go trials). Additionally, rVLPFC activity was increased during motor control trials relative to non-control trials, and was significantly and negatively correlated with amygdala activity during motor control trials in the negative emotional condition. The fact that the behavioral self-control seemed to “spill-over” into the affective domain via rVLPFC activation supports the hypothesis of that region acting as a central and domain-general locus of self-control in the brain.

In another example of how contextual factors can induce incidental affect regulation, Hare, Tottenham, Davidson, Glover, and Casey (2005) used an emotion go/no-go task to examine the effect of contextual information on neural responses to fear stimuli. In the task, the go trials were fearful face stimuli. Those fearful go faces were intermixed with occasional no-go trials, which were indicated by either neutral or happy face stimuli. The authors observed robust amygdala activation in response to the fearful faces in blocks when they were paired with neutral no-go stimuli. However, in blocks when the fearful faces were paired with happy no-go faces, the authors found a relative reduction in amygdala activity and an increase in caudate and VLPFC during the fear trials. There was also an increase in response time to the fearful trials in this condition. Here, a contextual factor—whether the occasional no-go trials were neutral or happy—altered the neural affective response to fearful face stimuli that were identical across the conditions. This study provides intriguing evidence that contextual factors can produce regulation-like effects in the absence of any intention to control one’s emotions.

In a recent study using a novel paradigm, researchers showed that another contextual factor—one’s own facial expression—may also generate incidental reductions in affective responding (Lee et al., 2008). Participants in this study produced an emotional facial expression that was either congruent or incongruent with one that they were viewing. For example, smiling while looking at a happy face is congruent, whereas smiling while looking at a fear face is incongruent. This incongruence automatically produces what the authors termed “emotional expression interference” between the participants’ own expression and the one they are viewing. Despite the fact that participants were not instructed to nor reported intentionally regulating their emotion, the emotional expression interference task produced inhibition of emotional expressions (measured with facial electromyography; EMG) and also recruited a brain network implicated in motor self-control, including rVLPFC and preSMA. This line of research is consistent with the facial feedback hypothesis (James, 1890; Strack et al., 1988) that mimicking a facial expression can unintentionally alter emotion experience.

We note that the rostral ACC has also been implicated in incidental affect regulation (Egner et al., 2008; Etkin et al., 2006; Vuilleumier et al., 2001) and in regulation of pain (see below), but we will not discuss these studies further here as they are not relevant to the role of the rVLPFC in incidental self-control.

One final way of measuring incidental affect regulation is to leverage individual differences in the tendency to spontaneously engage in emotion regulation, particularly in a context when there is no explicit instruction or awareness of emotion regulation. People who repeatedly engage in intentional emotion regulation in a particular situation (e.g., when interacting with one’s boss) over time may develop a cue-response association whereby incidental emotion regulation is triggered by contextual cues in a relatively automatic fashion and outside of awareness in a process similar to habit formation (Aarts & Dijksterhuis, 2000). Though these individuals still undoubtedly engage in deliberate emotion regulation in their everyday lives, they may also use incidental emotion regulation at a greater level than others. Additionally, studies comparing those high in daily emotion regulation use to those low in daily use can be useful not only in elucidating the neural systems involved in incidental emotion regulation, but may also further our understanding of the difference between the capacity to self-regulate (i.e., the effectiveness of self-regulation when one is prompted to engage it) and the tendency to self-regulate (i.e., the likelihood of self-regulation being engaged during daily life, regardless of one’s capacity to wield it; Berkman &

Lieberman, 2009).

For example, the effect of the “emotional expression interference” in the study described above (Lee et al., 2008) was moderated by trait-level emotion regulation such that those who report higher levels of daily emotion regulation show even greater activation in rVLPFC and even more behavioral inhibition of emotional facial expressions than those who report lower levels of daily emotion regulation. A related study found that those who report higher daily levels of one form of emotion regulation, reappraisal, show reduced amygdala activity and increased rVLPFC activity during passive viewing of negative emotional faces compared to those who report lower levels (Drabant et al., 2009). One suggestion of studies like these is that those who tend to engage in emotion regulation in daily life may over time develop the ability to engage in relatively low-effort and unintentional incidental self-regulation.

### **2.3 Incidental Self-Control of Behavior**

Several studies show that incidental self-control of behavior also recruits the rVLPFC. The majority of these use an unconscious priming paradigm, whereby a cue that has been consciously associated with motor inhibition during training (e.g., response inhibition during a stop-signal task) is subsequently displayed very briefly (e.g., for 32 ms) during an ongoing task as brain activity is recorded. Experimenters using this paradigm typically ensure that participants are unable to see the primes by using backwards masking and/or a visual discrimination task. In a series of such studies, van Gaal and colleagues used electroencephalography (EEG) to record neural activity as participants completed a go/no-go task with no-go trials that were both above and below the visual discrimination threshold (van Gaal et al., 2008). In other words, on some no-go trials participants could consciously recognize the no-go signal and on others the no-go signal was only accessible unconsciously. Consistent with the idea that behavioral inhibition can be primed outside of awareness, the researchers found that participants withheld a behavioral response more frequently on unconscious no-go trials than on go trials and that response latency during unconscious no-go trials was significantly slower compared to conscious go trials. Source localization identified increases in right lateral prefrontal cortex during unconscious no-go trials and furthermore, the magnitude of this activation was correlated with the magnitude of the behavioral slowing of the go response on unconscious no-go trials. In a subsequent study, the same group replicated these findings using fMRI and found that unconscious no-go trials recruited activation in bilateral VLPFC as well as the preSMA (van Gaal et al., 2010). Another study also found that left VLPFC was active during unconsciously presented incongruent behavioral primes (Lau & Passingham, 2007).

A recent study capitalized on the tendency for people in committed romantic relationships to automatically devalue potential alternative partners in order to study incidental control over romantic attraction (Meyer et al., 2011). Individuals who are committed to maintaining a romantic relationship report being disinterested when shown images of possible alternative partners. This occurs outside of subjective awareness (Ritter et al., 2010). Meyer and colleagues used fMRI to record neural activity while romantically committed participants viewed a series of images of potential attractive relationship partners and made a judgment about each one. The rVLPFC was more active on trials when committed participants successfully derogated the alternative option compared to when they failed to engage in romantic self-control. Furthermore, a greater level of commitment to one’s current romantic partner was associated with greater levels of control-related rVLPFC activation. This study not only provides another example of how the rVLPFC is recruited for self-control outside of awareness, but also illustrates the practical value of incidental self-control in protecting pair-bonding relationships. To the extent that encounters with potential alternative partners are common and that conscious exertions of self-control draw upon a limited resource, it would be highly adaptive to be able to recruit self-control without deliberate intent and outside of awareness in the service of maintaining long-term relationships.

## 2.4 Incidental Self-Control of Pain

Placebo analgesia refers to self-induced pain relief that one attributes to an external source that actually has no effect. By definition, then, placebo effects are a form of self-control that occurs without intention and outside of awareness. Numerous studies using fMRI have implicated the rVLPFC and other regions (e.g., the rostral ACC) as being critical for placebo-induced pain relief. For example, an early PET study observed increased activation in rVLPFC and rostral ACC during painful heat stimulation with a placebo compared to without (Petrovic et al., 2002). Another PET study found that rVLPFC activation increased significantly across a three-week placebo intervention for irritable bowel syndrome and was correlated with the magnitude of reductions in participant pain reports (Lieberman et al., 2004). Intriguingly, one study found an analgesic effect of viewing religious images among devout Catholics (compared to atheists) during painful stimulation, and that this effect was associated with rVLPFC activation (Wiech et al., 2008).

Several other studies have replicated these results implicating rVLPFC using various manipulations of momentary pain stimulation (Kong et al., 2006; Wager et al., 2004), and even the distress associated with viewing aversive images (Petrovic et al., 2005). Each of these studies stresses the importance of subjective expectations prior to the experience of pain or distress over deliberately attempting to control it after the fact. One potential implication is that placebo effects are a kind of implicit “pre-regulation” of distress, which occur without intention, typically outside of awareness, and may be less effortful than post-hoc deliberate self-control.

## 2.5 Summary

We reviewed studies on incidental self-control in three domains: affect, behavior, and pain. Among these, we surveyed studies using a variety of experimental paradigms including affect labeling, emotional expression interference, threat to close relationships, visual masking, and placebo manipulations. In each case, some prepotent response (affective, behavioral, or pain) that would have otherwise been present was shown to be either reduced or absent, and these effects occurred with no instruction to engage in self-control and outside of the conscious experience of the participant. The rVLPFC was recruited in nearly all of these cases of incidental self-control, and the magnitude of rVLPFC activation was frequently associated with reductions in the various response channels when they were measured. In several studies, the rostral ACC was observed to be co-active with the rVLPFC during instances of incidental self-control.

What do these results tell us about the role of the rVLPFC in self-control more broadly? First, and most importantly, they provide diverse examples of how self-control can be successfully recruited without intention and outside of awareness, and implicate the rVLPFC in this process. It is possible that self-control is similar to the intention to act, in that it is often, but not necessarily, accompanied by the subjective experience of awareness (e.g., Libet et al., 1983). The fact that the rVLPFC is recruited regardless of whether the act of self-control rises to the level of awareness or “feels” intentional implies that this brain region may be involved in other aspects of self-control.

For example, it is consistent with the data reviewed above that the rVLPFC represents the concept of self-control and flexibly applies that concept as appropriate to an ongoing task. The data from priming studies in particular (van Gaal et al., 2008, 2010) suggest that possibility. This observation converges with a wealth of behavioral evidence (for a review, see Custers & Aarts, 2010) that self-control (and cognitive control more generally) can become automated over time through repeated use (akin to motor learning) and associated with antecedent cues. Once this happens, self-control may be triggered automatically by cues in the environment and proceed outside of awareness. The studies reviewed here demonstrate the varied forms that those cues may take, such as priming of a learned stop-signal, viewing of images that

threaten one's romantic relationship, or an exposure to a painful stimulus combined with a placebo cue that one believes to be associated with analgesia.

If it is true that rVLPFC activation indexes the representational "strength" of the concept of self-control but not awareness or intention of that self-control, an important unanswered set of questions regards how and where the conscious experience of intentional self-control is activated. Might there be a threshold of activation in the rVLPFC, above which self-control becomes effortful, or is effort only experienced upon the recruitment of another region that is only involved in intentional, and not incidental, self-control? The answer to these questions might bear on another set of questions about the difference in quality between intentional and incidental forms of self-control. For example, can incidental self-control be as effective as intentional self-control though it recruits a different or possibly reduced network? Future studies can adapt the paradigms reviewed above to address these questions and many others regarding the neural pathways underlying incidental self-control and their relation to those involved in intentional forms.

### 3 Synthesizing the Literature

As is noted above, the rVLPFC is commonly active in a variety of forms of both intentional and incidental self-control (for a summary of all VLPFC maxima for all discussed forms of self-control, see Figure 2 and Table 1). Just as behavioral literature has theorized that self-control is like a muscle and can be fatigued (or trained) across domains (for a review, see Muraven & Baumeister, 2000), some neuroimaging literature has focused on the common role of the rVLPFC in multiple forms of self-control. Moreover, there is a body of literature exploring whether populations across the lifespan (children do not yet have a fully developed PFC and older adults have prefrontal atrophy) and populations with impaired self-control (e.g., people with ADHD, people who abuse substances, or compulsive gamblers) have impaired rVLPFC activity on tasks requiring acts of self-control.

Most of this research focuses on motor control and its relation to other forms of self-control. For example, conjunction analyses have found overlap within the rVLPFC when comparing response inhibition on the go/no-go task to a flanker task, which requires the suppression of irrelevant distracting information (Bunge et al., 2002; Wager et al., 2005), to set shifting during the Wisconsin Card Sorting Test (Konishi et al., 1999), and to an incompatible stimulus-response task that requires participants to press left for a right arrow and vice versa (Wager et al., 2005). Additionally, a meta-analysis found overlap in the rVLPFC across the Wisconsin Card Sorting Test, task-switching, and go/no-go tasks across 49 studies (Buchsbaum et al., 2005).

Preliminary research conducted by Cohen and Poldrack (unpublished data) has attempted to equate three different forms of self-control in 24 healthy adult participants: motor control (using the stop-signal task), risk-taking (using the BART), and emotion regulation (using a cognitive reappraisal paradigm). BOLD data during each of these tasks was examined within the rVLPFC, which was defined as in Figure 1. When contrasting rVLPFC activity during successful response inhibition with that during successful response execution during the stop-signal task, we found significant activity in a large portion of the rVLPFC ROI. When exploring self-control on the BART (defined as cashing out on the current balloon as compared to continuing to inflate the balloon regardless of the increasing risk of an explosion), again most of the rVLPFC was active, save for the pars triangularis portion of the IFG and the most anterior VLPFC subregions. For the emotion regulation task we limited our analysis to the 21 participants who rated the images they were to regulate as at least a 5 out of 7 in a post-scan rating of negativity. This was to ensure that viewing the images was a sufficiently negative experience so cognitive reappraisal could be used to decrease that initial negative reaction. We focused on rVLPFC regions that increased with greater

Study	# rVLPFC Foci	# lVLPFC Foci	Study	# rVLPFC Foci	# lVLPFC Foci
<b>Intentional Motor Control</b>			<b>Temporal Discounting</b>		
Aron & Poldrack (2006)	5	0	Boettiger et al. (2007)	1	0
Boehler et al. (2010)	2	2	McClure et al. (2004)	1	0
Buchsbaum et al. (2005)	1	0	Tanaka et al. (2004)	1	0
Chikazoe et al. (2007)	2	0	Total	<b>3</b>	<b>0</b>
Congdon et al. (2010)	2	0	<b>Intentional Emotion Regulation</b>		
Cools et al. (2002)	1	0	Goldin et al. (2008)	4	3
Freyer et al. (2009)	0	1	Ochsner et al. (2004)	4	4
Garavan et al. (1999)	1	0	Phan et al. (2005)	2	1
Ghahremani et al. (2010)	1	0	Wager et al. (2008)	3	1
Kenner et al. (2010)	0	1	Total	<b>13</b>	<b>9</b>
Kringelbach et al. (2003)	1	2	<b>Incidental Affect Regulation</b>		
Liddle et al. (2001)	1	1	Hare et al. (2005)	1	1
Menon et al. (2001)	0	1	Hariri et al. (2000)	2	0
Mitchell et al. (2009)	1	1	Lieberman et al. (2007)	4	0
ODoherty et al. (2003)	1	0	Total	<b>7</b>	<b>1</b>
Remijnse et al. (2005)	2	2	<b>Incidental Pain Regulation</b>		
Rubia et al. (2003)	2	0	Kong et al. (2006)	4	2
Xue et al. (2008a)	4	0	Lieberman et al. (2004)	2	0
Xue et al. (2008b)	1	2	Petrovic et al. (2002)	1	0
Total	<b>28</b>	<b>13</b>	Petrovic et al. (2005)	2	2
<b>Incidental Behavioral Control</b>			Wager et al. (2004)	1	1
Meyer et al. (2011)	1	1	Wiech et al. (2009)	1	0
vanGaal et al. (2010)	1	0	Total	<b>11</b>	<b>5</b>
Total	<b>2</b>	<b>1</b>	<b>Risk-Taking</b>		
<b>Risk-Taking</b>			Christopoulos et al. (2009)	1	0
Christopoulos et al. (2009)	1	0	Ernst et al. (2002)	4	1
Ernst et al. (2002)	4	1	Eshel et al. (2007)	1	0
Eshel et al. (2007)	1	0	Tobler et al. (2007)	1	0
Tobler et al. (2007)	1	0	Total	<b>7</b>	<b>1</b>
Total	<b>7</b>	<b>1</b>			

Table 1: List of numbers of right and left VLPFC foci for all studies included in this chapter that have foci within the VLPFC as defined in Figure 1. This list is not exhaustive but is intended to be a representative sample of studies within each self-control domain. While most domains include both right and left VLPFC foci, this table indicates that the rVLPFC is more consistently involved across domains and across studies than the left VLPFC. See Figure 2 for a pictorial representation of each of the foci.

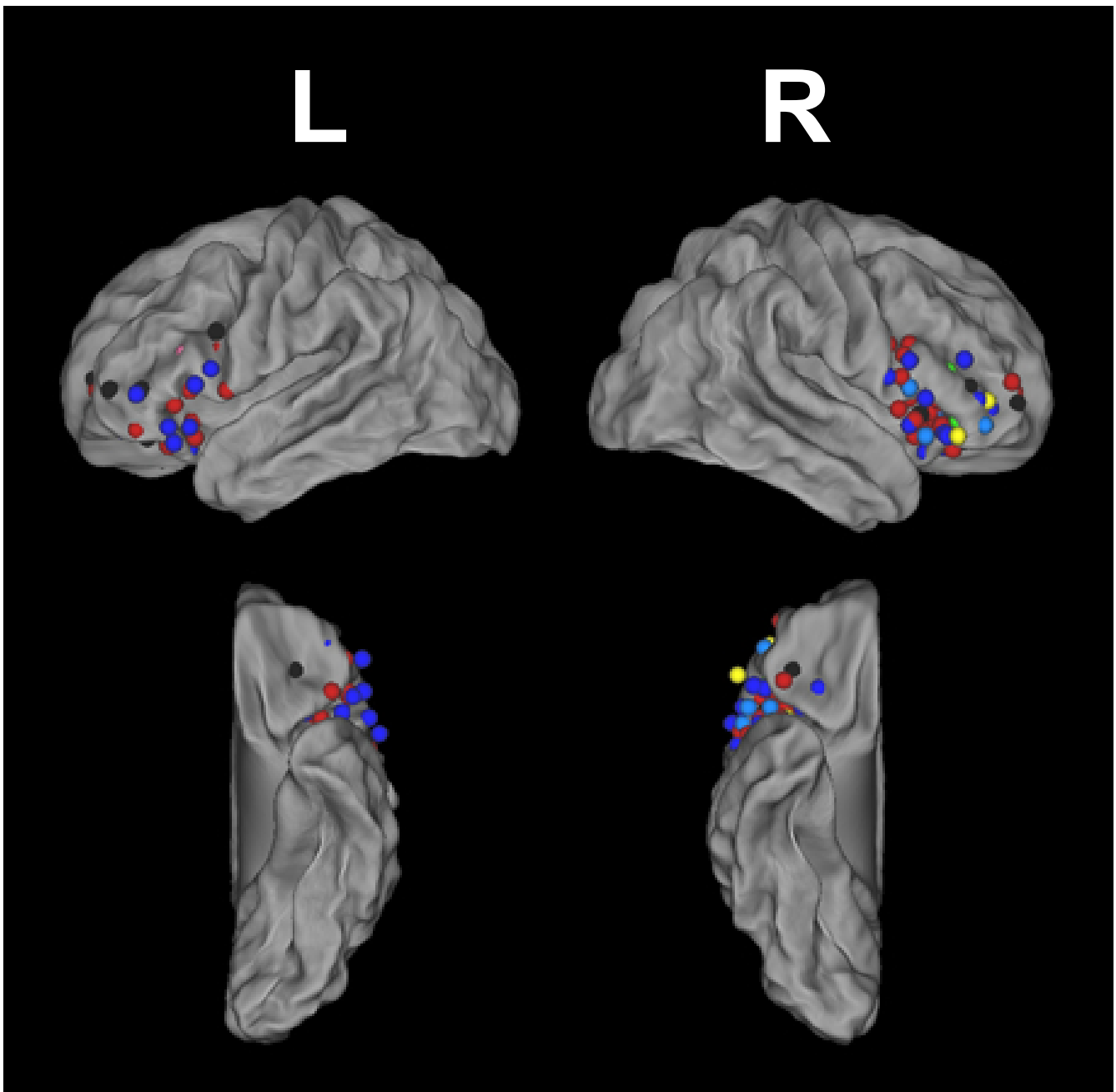


Figure 2: Three-dimensional surface rendering of all reported VLPFC foci (left and right) during intentional and incidental self-control in the studies discussed in this chapter. Red = intentional motor control, pink = incidental behavioral control, green = risk-taking, yellow = temporal discounting, blue = intentional emotion regulation, light blue = incidental affect regulation, black = incidental pain regulation. Foci were only included if they fell within the VLPFC as defined in Figure 1. See Table 1 for details about lateralization of VLPFC foci.

self-reported regulation on negative images participants were to suppress. We incorporated self-reported regulation into this analysis given the variability of non-regulated reactions to the negative images in order to most closely model actual, as opposed to assumed, emotion regulation. This analysis found a region in the lateral OFG that increased with increasing self-reported emotion regulation. A conjunction analysis confirmed that a region in the right lateral OFG was commonly active in all three self-control

tasks, with a greater overlap across the rVLPFC between the stop-signal and the BART (Figure 3a).

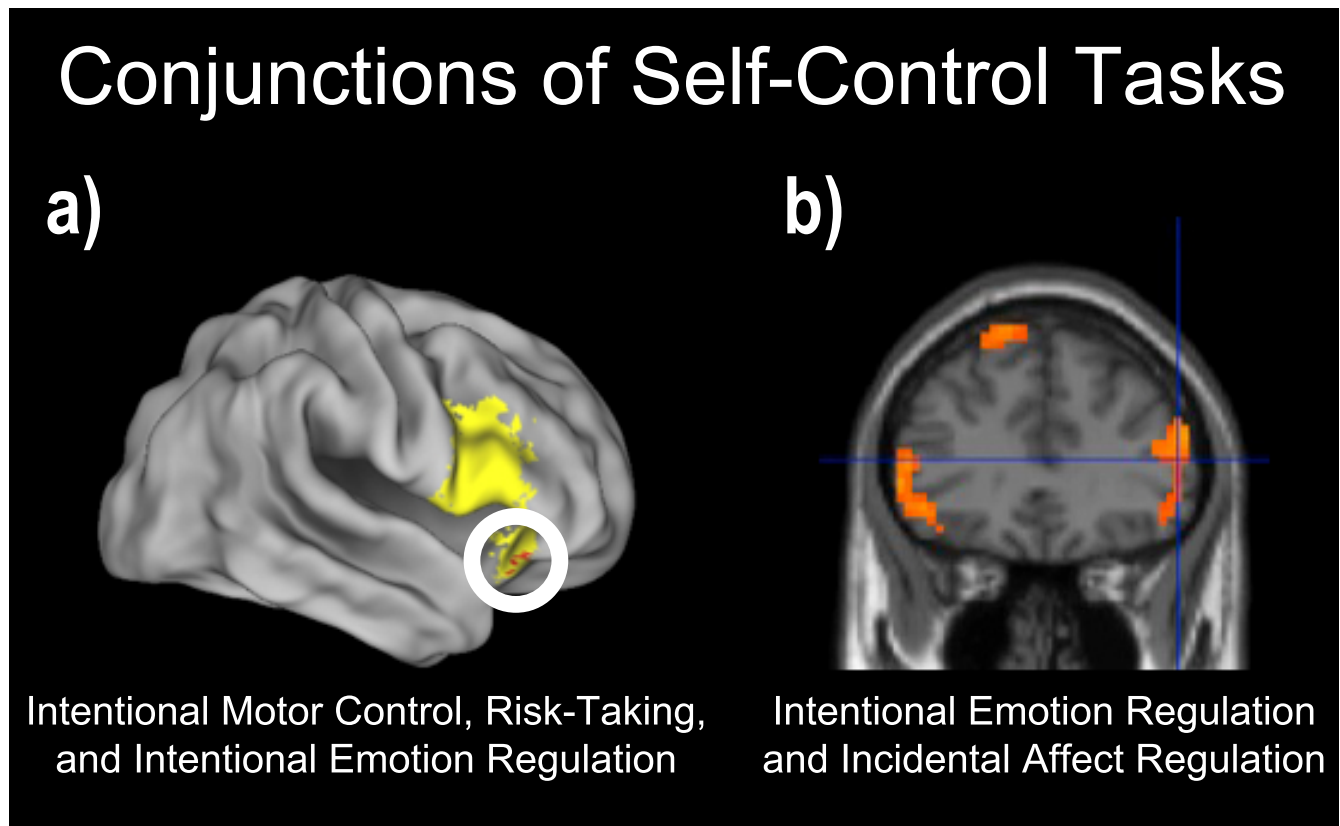


Figure 3: a) Conjunction analysis of self-control-related activation within the rVLPFC during motor control (stop-signal task), risk-taking (BART), and emotion regulation (cognitive reappraisal task). Red = conjunction of all three tasks (within circled area), yellow = overlap between motor control and risk-taking. b) Whole-brain conjunction analysis of intentional emotion regulation and incidental affect regulation ( $y = 30$ ). Neural overlap between the two tasks is limited to rVLPFC, left VLPFC, and posterior DMPFC.

In a study exploring the overlap between intentional emotion regulation and incidental affect regulation, participants viewed negative images from the International Affective Picture System (IAPS; Lang et al., 1993). Only three regions were commonly significantly active for both types of affective self-control: the rVLPFC, the left VLPFC, and the posterior dorsomedial PFC (DMPFC; Burklund, Creswell, Irwin & Lieberman, unpublished data; Figure 3b).

The prefrontal cortex, including the rVLPFC, is not fully developed until adulthood (Giedd et al., 1999). Therefore, developmental research has explored the effects of immature prefrontal function on various indices of self-control. It is well known that children and adolescents have poorer motor control than adults (Casey et al., 1997a; Durston et al., 2002; Williams et al., 1999), act in a more risky manner than adults (adolescents in particular; Crone & van der Molen, 2004; Gardner & Steinberg, 2005; for a review, see Steinberg, 2008), and discount delayed rewards more than adults (Christakou et al., 2011; Mischel & Metzner, 1962; Mischel et al., 1989; Olson et al., 2009; Scheres et al., 2006). Intentional emotion regulation paradigms have not been used to directly compare the emotion regulation ability of children or adolescents with that of adults, but implicit paradigms have found that incidental affect regulation improves with age (Lewis et al., 2006). In addition to behavioral improvements in self-control with age, it has been found that children have immature rVLPFC activity as compared to adults on a variety

of self-control tasks. For example, children have been shown to have decreased rVLPFC activity as compared to adults during successful simple motor control (Bunge et al., 2002; Durston et al., 2002; Rubia et al., 2007). Furthermore, a recent study found that neural activity in the rVLPFC, along with other regions such as the striatum and right STN, predicted both age and response inhibition ability (as measured by SSRT) on the stop-signal task (Cohen et al., 2010). This indicated that the rVLPFC is critically involved in successful motor control and that rVLPFC activity is decreased in children, who have not yet fully developed their self-control ability. In literature examining the development of reward-seeking behavior, children and adolescents have immature patterns of activity in the lateral OFC as compared to adults (Eshel et al., 2007; Galvan et al., 2006). The developmental literature implies that the development of the rVLPFC is related to the increase in self-control ability seen as children and adolescents become adults.

On the other end of the aging spectrum, it has been found that as people age their brains atrophy, particularly in the prefrontal cortex (Cabeza, 2001). Therefore, some aging research has examined the effects of prefrontal degeneration on self-control. Much of the research has focused on either motor control or emotion regulation. Older adults have longer SSRTs in the stop-signal task (Williams et al., 1999) and fewer correct inhibitions in the go/no-go task (Nielson et al., 2002) than younger adults. Further, brain imaging results from one study revealed decreased activity in multiple brain regions, including rVLPFC, in older adults as compared to younger adults (Nielson et al., 2002). In contrast, older adults had greater activity in left VLPFC than younger adults, potentially indicating the recruitment of additional regions, specifically the reduction of hemispheric asymmetry (Cabeza, 2002), as compensation for poorer functioning in the rVLPFC. These results were specific to response inhibition, as there were no differences in prefrontal activity during go trials across age (Nielson et al., 2002). In the field of emotion processing it has been noted that older adults show a “positivity effect” when remembering stimuli, in that they selectively remember positive, as compared to negative, stimuli more so than younger adults. One theory is that this positivity effect occurs due to increased emotion regulation in older adults (Mather & Carstensen, 2005; Nashiro et al., In Press). Very little research has directly compared the neural regions involved in emotion regulation in older and younger adults. A recent study that did compare the two groups found that attempted emotion regulation resulted in greater activity in left, but not right, IFG in younger as compared to older adults (Wincoff et al., 2011). However, older adults also reported less successful emotion regulation than younger adults, so future studies where equivalent regulation is found across age groups should be conducted before drawing conclusions about neural differences due to aging in emotion regulation.

In addition to the normal functional trajectory of the rVLPFC during acts of self-control across the lifespan, it has been shown that rVLPFC activity has important implications for a variety of psychiatric illnesses that are related to impulsivity. Research has demonstrated decreased rVLPFC activity during simple motor control tasks in people with ADHD (Booth et al., 2005; Durston et al., 2006; Rubia et al., 1999) and obsessive compulsive disorder (Roth et al., 2007). A recent review of the simple response inhibition literature noted that not only is right IFG activity decreased in people with ADHD as compared to controls, but that right IFG volume is also reduced relative to controls (Chambers et al., 2009). Additionally, it was demonstrated that compulsive gamblers performed worse on a reversal learning task and had less rVLPFC activity than did healthy controls (de Ruiter et al., 2009). Furthermore, individuals who abuse methamphetamine were shown to have less rVLPFC activity on an incidental affect labeling task (Payer et al., 2008), as well as a trend toward lower right IFG gray matter concentration in individuals who abuse methamphetamine than in healthy control participants (Payer et al., 2011). This literature underscores that not only do people with impulsivity-related disorders have behavioral problems with self-control, but the neural mechanisms underlying self-control may be impaired as well.

## 4 rVLPFC Connections

The rVLPFC is well positioned to be a key neural region involved in exerting self-control. It is anatomically associated with other prefrontal control regions, such as the DLPFC, mPFC, ACC, and OFC (Miller & Cohen, 2001). Research utilizing Diffusion Tensor Imaging (DTI) in humans has indicated that there are white matter connections between the rVLPFC and the ventral caudate (Leh et al., 2007), the preSMA, and the STN (Aron et al., 2007a). A study comparing functional to structural connectivity similarly found that the rVLPFC and the preSMA were connected using both functional connectivity and diffusion-weighted techniques (Johansen-Berg et al., 2004). The preSMA has been associated with conflict detection and the basal ganglia, including the caudate and the STN, have been associated with the control of motor responses. Connections between these regions, therefore, could be the means through which the rVLPFC becomes aware of a conflict between a goal-directed intention and a prepotent impulse (through the preSMA) and then sends a signal to exert behavioral self-control over the impulse (through the basal ganglia). Functional connectivity analyses using Granger causality mapping provided evidence that not only are the preSMA and the rVLPFC connected, but that the preSMA, along with the cerebellum and the thalamus, cause changes related to conflict/error processing in rVLPFC activity (Ide & Li, 2011). Moreover, Granger causality supports the theory that the rVLPFC projects to the preSMA, which in turn projects to the basal ganglia/STN (Duann et al., 2009).

Additionally, there is evidence that the rVLPFC is functionally connected to the amygdala, possibly via the mPFC, which has reciprocal connections with both structures (Banks et al., 2007; Hariri et al., 2000, 2003; Lieberman et al., 2007). Lastly, there is evidence that the lateral OFC is functionally connected to a large network of dorsal prefrontal and dorsal parietal regions (Cohen et al., 2005).

Studies with macaque monkeys have more directly examined structural brain connections. Evidence exists that the cytoarchitecture of the macaque VLPFC is similar to that in humans, specifically the macaque inferior arcuate sulcus and its surrounding cortex (Ongur & Price, 2000; Petrides et al., 2005). It is therefore assumed that this area in macaques is the monkey homologue to the human VLPFC and that the anatomical connections found in this region in macaques may also be found in humans. In the monkey, the inferior arcuate sulcus is connected to the lateral and medial OFC, the DMPFC, the DLPFC, the ACC, the insula, the supplementary, premotor and primary motor areas, and areas of the superior temporal lobe (Deacon, 1992). Moreover, there are architectonic and connectivity subdivisions within the macaque VLPFC, implying that different subregions may have different functional roles (Gerbella et al., 2010).

Other research investigating the primate lateral OFC, which is a part of the larger VLPFC, has found that the lateral OFC receives sensory input directly from the primary taste cortex, indirectly from visual areas via the inferior temporal cortex, and from somatosensory areas such as the primary and secondary sensory cortices and the insula. The lateral OFC sends output to the hypothalamus, the periaqueductal gray area, and the striatum, especially the ventral caudate. Furthermore, it has reciprocal connections with the amygdala, cingulate cortex, premotor area, and DLPFC (Kringelbach & Rolls, 2004). To further support the theory that the connections found in monkeys also exist in humans, research has demonstrated that connections with some of these regions, such as the ACC, DLPFC, preSMA, amygdala, and ventral caudate, have been noted in humans as well (Aron et al., 2007a; Leh et al., 2007; Lieberman et al., 2007; Miller & Cohen, 2001).

## 5 Other rVLPFC Roles

It is important to point out that there are multiple theories about the function of the rVLPFC. Activity in this region has been associated with cognitive processes as diverse as self-control (as discussed in this chapter), attention to unexpected stimuli (Corbetta & Shulman, 2002; Hampshire et al., 2010; Iaria et al., 2008; Sharp et al., 2010), various aspects of memory (Courtney et al., 1996; Dove et al., 2008; Kostopoulos & Petrides, 2003; Rizzuto et al., 2005), and the interpretation of emotions (Kober et al., 2008). For example, it has been proposed that rVLPFC activity is specifically related to stimulus-driven, bottom-up attention and automatic alerting to unexpected, salient stimuli (Corbetta & Shulman, 2002; Iaria et al., 2008). Recent research comparing stop-signal performance to performance on equivalent tasks that do not require motor inhibition but still have random (i.e., unexpected), infrequent “signals” has found equivalent rVLPFC activity when inhibiting one’s response in reaction to a stop-signal as when encountering a signal giving instructions to respond (Hampshire et al., 2010) or to be ignored (Sharp et al., 2010). However, participants responded more slowly when they heard a signal that was to be ignored (Sharp et al., 2010), leaving open the possibility that a response inhibition mechanism was occurring (and then overcome) on those trials. There is also evidence that the rVLPFC is involved in target detection and thus responds to the relevant aspects of a stimulus (Hampshire et al., 2009). In line with this finding but in a different cognitive domain, the rVLPFC has been implicated during memory retrieval when one must differentiate between relevant and irrelevant aspects of a stimulus (Kostopoulos & Petrides, 2003) or when participants are engaging in active, goal-directed retrieval (Dove et al., 2008). Additionally, the rVLPFC has been associated with both object-oriented (Courtney et al., 1996) and spatially oriented (Rizzuto et al., 2005) working memory.

Clearly the rVLPFC has been associated with many different roles. These are not necessarily mutually exclusive. First, there is evidence that different foci within the rVLPFC are associated with motor control (specifically the right ventral IFG) and attention to unexpected stimuli (specifically the right dorsal IFG/inferior frontal junction; Aron, 2011; Chikazoe, 2010; Chikazoe et al., 2009; Verbruggen et al., 2010). A recent meta-analysis further supports the finding that across many studies the locus of rVLPFC activity during motor control and during reorienting to unexpected stimuli, while partially overlapping, can be dissociated (Levy & Wagner, 2011). The possibility of functional subdivisions within the rVLPFC is strengthened by the finding that there are different structural patterns within VLPFC subdivisions in the macaque (Gerbella et al., 2010). Second, many purported roles of the rVLPFC involve goal-directed selection, be it whether or not to respond to a stimulus, how to regulate one’s emotions, whether to behave in a risky or impatient manner, or selectively remembering relevant items. Thus, it is possible that the rVLPFC is generally involved in goal-directed behavior, while different subregions are specifically involved in various processes that fall under that umbrella.

In addition to the afore-mentioned theories about the specific function(s) of the rVLPFC, general theories of prefrontal functioning have been proposed that may explain the varied processes that the rVLPFC appears to be involved in. McIntosh (2000; 2004) has proposed a theory of “neural context” that states that dynamic neural interactions, which can rapidly and transiently change based on the current environment, are more important for cognition than the magnitude of neural activity of any single region. This theory would predict that the role of an individual region such as the rVLPFC can change based on its current pattern of functional connectivity (i.e., what other brain regions to which it is connected and with which it is interacting). This is a distinct possibility with the rVLPFC, given that it is highly interconnected with other brain regions (see “rVLPFC Connections” section above). Other theories have similarly proposed that the role of prefrontal cortical regions can change based on the current cognitive environment (Duncan, 2001; Miller & Cohen, 2001). Evidence supporting all of these connectivity theories comes in part from single unit recordings demonstrating that prefrontal neurons can adaptively

change their activity patterns and influence on downstream brain regions (Miller & Cohen, 2001) and in part from functional connectivity studies in humans that demonstrate that the same region can have different patterns of connectivity in different cognitive contexts (McIntosh, 2000, 2004). See Asp and Tranel (Chapter 26 of this book) for an alternate theory of general prefrontal functioning.

## 6 Conclusions

As has been discussed throughout this chapter, while the rVLPFC has been theorized to have multiple functional roles, one major role is its centrality to a variety of forms of both intentional and incidental self-control. Just as behavioral literature has theorized that self-control is like a muscle and can be fatigued (or trained) across domains (for a review, see Muraven & Baumeister, 2000), neuroimaging literature commonly points to the involvement of the rVLPFC in many different forms of self-control. For example, the rVLPFC is involved during tasks involving motor control, control over risky behavior, delay of gratification, intentional emotion regulation, incidental affect regulation, incidental behavioral control, and incidental pain regulation.

While the activity is often bilateral, there is a tendency for the rVLPFC to be more consistently involved in many self-control processes than the left VLPFC (for reviews, see Aron et al., 2004; Chikazoe, 2010). As can be seen in Table 1, in 6/7 of the intentional and incidental self-control domains examined here there are at least twice as many foci in the rVLPFC as compared to the left VLPFC (and in the last domain, intentional emotion regulation, there are approximately 1.5 times the number of rVLPFC foci than left VLPFC foci). Alternately, the left VLPFC has been associated more often with the cognitive control specifically of memory and language, in particular the resolution of competition among possible representations to select only relevant representations (for reviews, see Badre & Wagner, 2007; Blumenfeld & Ranganath, 2007; Novick et al., 2010). This right/left distinction indicates a functional dissociation between right and left VLPFC, but it is important to note that the processes in each hemisphere may be similar (i.e., the goal-directed suppression of irrelevant actions or memories in order to select the most appropriate); it may be the modality, not the process, that is different across hemispheres.

The majority of the literature exploring the role of the rVLPFC in self-control focuses on a single self-control related task in a single population, although some literature has attempted to synthesize the results from these different forms of self-control to more directly examine the common role of the rVLPFC across domains. This literature has demonstrated that overlapping but partially distinct regions in the rVLPFC are generally related to the exertion of self-control (Buchsbaum et al., 2005; Bunge et al., 2002; Cohen & Poldrack, unpublished data; Konishi et al., 1999; Wager et al., 2005). Moreover, it has emphasized that compromised rVLPFC function, whether it's healthy but immature (Bunge et al., 2002; Cohen et al., 2010; Durston et al., 2002; Eshel et al., 2007; Galvan et al., 2006; Rubia et al., 2007), atrophied due to normal aging (Nielson et al., 2002), or mature but impaired (Booth et al., 2005; Chambers et al., 2009; de Ruiter et al., 2009; Durston et al., 2006; Payer et al., 2008; Roth et al., 2007; Rubia et al., 1999), results in decreased performance on tasks requiring self-control.

Given the centrality and the necessity of being able to exert self-control throughout one's daily life, much research has been focused on how self-control is exerted and what fails when self-control is impaired. The literature to date implies that the rVLPFC is, at least in part, an important brain region underlying successful self-control ability: its level of activity is often related to self-control ability and when it has abnormal levels of activity, self-control ability is often impaired. This seems to hold true in intentional and in incidental laboratory tasks, as discussed in this chapter, and in the real world, as can be noted by the relationship between impaired rVLPFC activity and impulsive behavior in multiple neuropsychiatric disorders. Even with this knowledge, there are still open questions about the role of

particular subsections of the rVLPFC and other roles that the rVLPFC may have, and how inconsistencies in the literature may be resolved. Moreover, it is still unknown whether the process of incidental self-control uses the same or different rVLPFC-related networks as intentional self-control. Therefore, further research more thoroughly exploring the generality of the rVLPFC as a self-control mechanism, and how this relates to other hypothesized roles of the rVLPFC, remains to be conducted.

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