

61 Self-Regulation and its Failures

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ABSTRACT The ability to flexibly alter behavior in the service of future goals is one of the key evolutionary adaptations that has enabled humankind to flourish. Self-regulation refers to a set of mental processes for overriding impulses, selectively attending to goal-relevant information, and monitoring thoughts and behavior for signs of failure. Although self-regulation is of fundamental importance to an individual's success, failures of self-regulation are common. In this chapter we focus on the role of exposure to tempting cues, negative emotions, and limited cognitive resources in bringing about self-regulation failure. We review evidence from neuropsychology and functional neuroimaging that successful self-regulation depends on the interaction between brain structures in the prefrontal cortex involved in representing goals and directing attention away from goal-irrelevant stimuli and cortical and subcortical structures involved in representing the value of rewards during decision making. When the balance between these countervailing systems is disrupted as a result of overwhelming impulses, negative affect, or deficient top-down control, self-regulation failure ensues.

More than any other species, humans are especially talented at controlling their own behavior in order to follow their goals and abide by rules and laws. Outside of certain psychiatric and neurological conditions, even the most impulsive human is still leaps and bounds more capable of not blowing off work, of not eating everything in the pantry, or of not stealing their spouse's food than are the most precocious of nonhuman primates. Despite humankind's enormous advantage in this domain, successful self-regulation remains difficult and failures are common (Baumeister & Heatherton, 1996; Wagner & Heatherton, in press). How is it, then, that humans are capable of inhibiting urges and pushing aside temptations in order to pursue their goals?

One commonly held view is that humans have evolved specific mental faculties that allow for superior planning and behavioral flexibility. These adaptations underlie humans' apparent superiority at self-regulation, and it has been hypothesized that they arose from a disproportionate amount of cortical expansion of the prefrontal cortex (PFC) over the course of human evolution (Rilling, 2006). Given the known role of the PFC in self-control, it was reasonable to assume that the brain would show specific structural changes to support these putative cognitive adaptations. However, recent work suggests that relative larger size of the PFC in humans compared to nonhuman primates may have been

overstated (Semendeferi, Lu, Schenker, & Damasio, 2002). Instead, it has been suggested that, rather than overall size, the human PFC demonstrated increased white matter connectivity (i.e., Schoenemann, Sheehan, & Glotzer, 2005; although see Barton & Venditti, 2013). Regardless of whether the human PFC shows evidence of specialized morphological enlargement, that humans possess a unique capacity for planning and self-regulation compared to all other animals appears irrefutable.

Or does it? Just how unique is the human capacity for self-regulation? Comparative psychological research on nonhuman primates and other animals has generally found that, with the exception of some domesticated animals (e.g., Miller, Pattison, DeWall, Rayburn-Reeves, & Zentall, 2010), most non-human animals display remarkably poor ability to inhibit prepotent responses in order to obtain later, larger rewards (i.e., delay of gratification). For example, most nonhuman animals, including many primates, will tolerate delays of only a few seconds before consuming a desired food item (Green, Myerson, Holt, Slevin, & Estle, 2004; Ramseyer, Pelé, Dufour, Chauvin, & Thierry, 2006). There are, however, some rare exceptions. For instance, great apes and some species of birds will tolerate delays as long as a few minutes provided the expected reward greatly exceeds the value of the currently available item (e.g., Beran, 2002; Dufour, Wascher, Braun, Miller, & Bugnyar, 2012). Given the studies discussed above, it appears that outside of certain primates and species of birds, evidence of self-regulation-like behavior is sparse among nonhuman animals. Why is it that humans have evolved this complex capacity to self-regulate?

The importance of self-regulation for human social groups

Unlike many animals, humans display a prolonged period of development and are unable to care for themselves for the first decade of life. Being a member of a social group brings with it tremendous advantages, from sharing the burden of child rearing to cooperative hunting and food sharing (Buss & Kenrick, 1998). As human safety and survival has long depended on living in groups, it has been suggested that humans have a

fundamental need to belong (Baumeister & Leary, 1995) that motivates them to avoid behaviors that could lead to their expulsion from the group (e.g., theft of common resources) as this would greatly lessen their chances for survival (Goodall, 1986; Heatherton, 2011). Among humans and possibly some great apes (i.e., Jensen, Call, & Tomasello, 2007), cheaters who fail to share with other members of the group could face expulsion from the group (Kurzban & Leary, 2001). In fact, it is precisely this punishment of cheating behavior that enables cooperation to be an evolutionarily sound strategy for some social animals (Boyd, Gintis, & Bowles, 2010). Thus, by allowing individuals to inhibit impulses and bring their behavior in line with group standards, self-regulation has permitted humans to benefit from all the perks of living in social groups, such as cooperation and food sharing but also the transmission of knowledge and culture.

What is self-regulation?

At its core, self-regulation is concerned with starting, stopping, or modifying thoughts, emotions, or behavior in order to pursue goals or stay in line with societal norms. Self-regulation encompasses both internal modes of control, such as when people regulate their thoughts or attempt to change their emotional states, and also external ones, such as when people initiate or stop a behavior (i.e., starting work or stopping oneself from over-eating). Although many models of self-regulation exist, most share a similar framework characterized by the capacity to set goals, regulate thoughts, behaviors, or emotions, and monitor for signs of failure (Bandura, 1991; Baumeister & Heatherton, 1996; Carver & Scheier, 1981; Heatherton, 2011; Metcalfe & Mischel, 1999). As will be apparent, self-regulation shares many similarities with components of executive function in cognitive psychology and neuropsychology (e.g., Norman & Shallice, 1986) such as working memory and attention control. Indeed, aspects of executive function have since become incorporated into theories of self-regulation (Hofmann, Schmeichel, & Baddeley, 2012).

Cognitive neuroscience of self-regulation failure

Self-regulation failures typically occur upon exposure to a desired stimulus or following some precipitating event, such as emotional distress, alcohol consumption, or exhaustion of self-regulatory resources. Successful self-regulation relies on a delicate balance between the strength of urges and impulses on the one hand, and the capacity to keep them in check on the

other. Self-regulation failure then can occur due to a particularly strong impulse or when the capacity to engage in self-control is impaired or the motivation is absent. Below, we review three common threats to this balance: exposure to tempting cues (e.g., food, drugs), emotional and social distress, and depletion of self-regulatory resources.

CUE EXPOSURE AND IMPULSE INHIBITION Modern-day humans are surrounded by a wealth of indulgences. At no other time in history has such a plethora of pleasures and vices been so readily available. It is perhaps little wonder, then, that the dominant form of self-regulation in daily life is impulse control (see Hofmann, Baumeister, Förster, & Vohs, 2011). In the psychological literature, an impulse typically refers to an urge or desire to consume a particular item or engage in a pleasurable behavior. With some exceptions, impulses are inherently rewarding behaviors that invade people's attention and require effort to inhibit (Metcalf & Mischel, 1999). One of the most common ways an impulse can arise is from viewing an activating stimulus, such as food advertisements or the sight and smell of a cigarette. Studies show that physiological measures such as heart rate and salivary responses are increased following exposure to food cues in dieters (Brunstrom, Yates, & Witcomb, 2004) and cigarette cues in smokers (Drobes & Tiffany, 1997). Perhaps not surprisingly, then, exposure to desired items also increases craving for and consumption of the substance (e.g., Carter & Tiffany, 1999; Federoff, Polivy, & Herman, 1997; Sayette, Martin, Wertz, Shiffman, & Perrott, 2001). Perhaps more perniciously, being exposed to tempting substances can also have effects that people many not be consciously aware of. For example, tempting cues can capture people's attention, even when presented incidentally as part of a film (Lochbuehler, Voogd, Scholte, & Engels, 2011) and may activate motor schemas for using the substance (e.g., the action of holding and smoking a cigarette; see Tiffany, 1990). This last point is especially interesting in light of recent functional neuroimaging work suggesting that, among smokers, viewing scenes of other people smoking activates brain regions associated with representing goal-directed actions (Wagner, Dal Cin, Sargent, Kelley, & Heatherton, 2011).

Nonhuman animal neurophysiology studies show that consuming rewards (foods, drugs) or engaging in rewarding activities (e.g., sex) is associated with activation of the mesolimbic dopamine system (e.g., the ventral tegmental area and nucleus accumbens/ventral striatum) and the orbitofrontal cortex (Damasio, Pfaus, Wenkstern, Phillips, & Fibiger, 1992; Kringelbach, 2005; Schilström, Svensson, Svensson, & Nomikos, 1998).

In humans, functional neuroimaging research has similarly shown that activity in the ventral striatum and orbitofrontal cortex increases when consuming (Gottfried, O'Doherty, & Dolan, 2003; Kringelbach, O'Doherty, Rolls, & Andrews, 2003) or viewing cues associated with appetitive rewards such as food or attractive faces, as well as abstract rewards such as money (Cloutier, Heatherton, Whalen, & Kelley, 2008; Knutson, Taylor, Kaufman, Peterson, & Glover, 2005; van der Laan, de Ridder, Viergever, & Smeets, 2011). Given the role of the reward system in motivating behavior, there has been considerable effort aimed at investigating whether people who are at risk for obesity or substance-abuse problems show any abnormalities in reward processing. So far the evidence seems to indicate that increased striatal responses to food (Demos, Heatherton, & Kelley, 2012) or drug cues (Janes et al., 2010; McClernon, Kozink, & Rose, 2008) are predictive of real-world behavior. For instance, people who are rated as being particularly sensitive to rewards show heightened food cue-related activity in the ventral striatum and orbitofrontal cortex (Beaver et al., 2006). A similar finding was demonstrated by Demos and colleagues (2012), in which individual differences in ventral striatal responses to food cues and erotic scenes predicted subsequent weight gain and degree of sexual activity in a six-month follow-up. Taken together, these findings suggest that individual differences in cue reactivity may reflect a stable sensitivity to rewards, the excess of which makes it more difficult for some individuals to maintain self-control. An extreme example of this comes from a study by Casey and colleagues (2011), which showed that people who had difficulty delaying gratification as children exhibited heightened activity in the ventral striatum when viewing appetitive stimuli over 40 years later.

Thus far we have discussed findings in which passive viewing of food or drug cues led to heightened reward-related neural responses in the striatum and orbitofrontal cortex. However, an important question for understanding self-regulation failure is what happens when people are explicitly engaging in self-control in order to reduce their craving for appetitive stimuli. In general, it has been found that explicit regulation of cravings and desires involves the lateral PFC and the anterior cingulate cortex. For instance, the lateral PFC and ACC show increased activity when attempting to down-regulate responses to cigarette paraphernalia among smokers (Brody et al., 2007; Kober et al., 2010), to food cues among dieters (Siep et al., 2012), drug cues among substance abusers (Volkow et al., 2010), and to monetary rewards for most people (Delgado, Gillis, & Phelps, 2008). Importantly, across all these

studies, engaging in explicit self-regulation, whether through cognitive reappraisal or other means, resulted in decreased activity in the striatum and orbitofrontal cortex.

SOCIAL AND EMOTIONAL DISTRESS A frequently reported cause of self-regulation failure is the experience of emotional and social distress. For example, negative affect often precedes binge eating and binge drinking episodes (Haedt-Matt & Keel, 2011; Witkiewitz & Villarreal, 2009). Laboratory inductions of negative mood or social distress (e.g., social rejection) similarly show that experiencing negative affect leads to disinhibited behavior (e.g., Twenge, Baumeister, Tice, & Stucke, 2001). For instance, negative mood inductions lead dieters to subsequently overeat (Heatherton, Herman, & Polivy, 1991; Heatherton, Strieppe, & Wittenberg, 1998) and smokers to crave smoking (Willner & Jones, 1996). Similarly, inducing social rejection has been shown to increase consumption of unhealthy foods, reduce task persistence, and interfere with the ability to sustain attention (Baumeister, DeWall, Ciarocco, & Twenge, 2005).

A number of mechanisms have been proposed to explain how emotional and social distress influence self-regulation (for a review, see Wagner & Heatherton, 2013b). Common among them is the notion that people are motivated to repair their mood and change their negative emotional state. This often takes the form of increased efforts to regulate emotions, which can come at a cost to self-regulation since the increased cognitive load incurred is thought to impair monitoring of ongoing behavior (e.g., Johns, Inzlicht, & Schmeider, 2008) and reduce cognitive resources that could otherwise be used in the service of self-regulation.

Another mechanism whereby negative affect can lead to self-regulation failure comes from research suggesting that negative affect increases the perceived reward value of temptations, rendering them more difficult to inhibit. For instance, negative affect has been shown to reduce people's ability to delay gratification, biasing them toward accepting immediate monetary rewards over waiting for larger delayed payments (e.g., Lerner, Li, & Weber, 2013; Mischel, Ebbesen, & Zeiss, 1973; Twenge et al., 2001). In addition, experiencing negative affect is associated with increased cravings for carbohydrate-rich foods (Christensen & Pettijohn, 2001) and, among smokers, increases both the intensity of smoking (McKee et al., 2011) and the amount of pleasure people report from smoking a cigarette (Zinser, Baker, Sherman, & Cannon, 1992). Together, these studies suggest that negative affect may serve to ramp up the gain on temptations and pleasurable activities.

Indeed, research in nonhuman animals suggests that emotional distress (usually via social isolation) can elicit reward-seeking behavior owing to a stress-induced sensitization of brain regions involved in reward processing (e.g., Peciña, Schulkin, & Berridge, 2006; Piazza & Le Moal, 1996; Ramsey & van Ree, 1993). Research in humans provides converging evidence in the form of affect-related modulation of activity in the ventral striatum and orbitofrontal cortex (OFC) to appetitive cues (e.g., Killgore & Yurgelun-Todd, 2006; Wagner, Boswell, Kelley, & Heatherton, 2012). For instance, the experimental induction of negative mood has been shown to lead to increased food-cue related activity in the OFC (Wagner et al., 2012).

Research on social rejection provides another means for examining the role of emotional distress on self-regulation. As mentioned above, a number of studies have shown that social rejection can bring about self-regulation failure (Baumeister et al., 2005). Although there is considerable research on the neural correlates of experiencing social rejection (for a review, see Eisenberger, 2012), there are far fewer cognitive neuroscience studies examining the link between social exclusion and self-regulation failure. Of the few extant studies, results show that social rejection leads to reduced activity in the PFC during executive function tasks. For example, social rejection was found to reduce activity in the lateral PFC as well as impair accuracy when completing complex math problems (Campbell et al., 2006). Similarly, Peake and colleagues (2013) demonstrated that social exclusion increased risk-taking behavior (operationalized as amount of crashes in a driving simulator) and reduced lateral PFC activity during crash trials.

Taken together, the results of these studies suggest two mechanisms whereby the experience of emotional and social distress impairs self-regulation. First, the experience of negative affect may serve to sensitize the ventral striatum and OFC to the incentive value of appetitive rewards (e.g., Peciña et al., 2006; Piazza & Le Moal, 1996). And second, negative affect may also act directly on self-control, reducing the capacity to engage in sustained attention or inhibit prepotent responses (e.g., Campbell et al., 2006; Peake, Dishion, Stormshak, Moore, & Pfeifer, 2013).

DEPLETION OF LIMITED SELF-REGULATORY RESOURCES

In the preceding section, we briefly touched upon the notion that one of the means by which negative affect can sabotage self-control is through imposing a cognitive load as individuals juggle regulating affect with regulating their behavior in other domains (e.g., food or drug consumption). One consequence of this

sustained mental effort is that it can temporarily deplete cognitive resources required for self-regulation, thereby leaving people vulnerable to temptations. This view of self-regulation as being resource-limited and subject to fatigue is the central tenant of the limited resource, or *strength*, model of self-regulation (e.g., Baumeister & Heatherton, 1996). Since its formulation, the strength model has received support from a large number of studies within the laboratory (for a meta-analysis, see Hagger, Wood, Stiff, & Chatzisarantis, 2010) as well as more naturalistic experiments using experience-sampling methods (Hofmann et al., 2011). For example, engaging in a prior effortful self-control task can subsequently make people more vulnerable to temptations such as food and alcohol (Muraven, Collins, & Nienhaus, 2002; Vohs & Heatherton, 2000), less able to control their emotions (Schmeichel, 2007), and more likely to violate social norms (DeBono, Shmueli, & Muraven, 2011; Vohs, Baumeister, & Ciarocco, 2005).

In recent years, there has been several efforts aimed at defining the mechanisms underlying self-regulatory depletion effects, with researchers suggesting that depletion reflects temporary decreases in circulating blood glucose availability (Gailliot et al., 2007), lay beliefs in self-control (Job, Dweck, & Walton, 2010), or shifts in motivation and attention away from effortful control and toward more rewarding activities (Beedie & Lane, 2012; Inzlicht & Schmeichel, 2012). More recently, it has been suggested that the strength model may have overemphasized the role of self-regulatory depletion impairing top-down control, neglecting the possibility that depletion may also increase the strength of temptations and impulses, thereby making them more difficult to resist. Evidence for this last conjecture comes from research demonstrating that, following self-regulatory depletion, people rate emotions as more extreme, pain as more intense, and desires as more strongly felt than nondepleted individuals (Vohs, Baumeister, Mead, Ramanathan, & Schmeichel, Manuscript Submitted for Publication).

Research on the neural basis of self-regulatory depletion is still in its infancy, with only a handful of studies investigating the effects of depletion on the neural systems involved in self-control. Within the cognitive domain, three studies have examined the effects of self-regulatory depletion on the subsequent recruitment of prefrontal brain regions during self-control tasks. The first of these studies measured the error-related negativity, an index of conflict monitoring thought to originate in the ACC, and found that, relative to a control group, depleted participants showed a reduced error-related negativity during a subsequent task requiring self-control, and that this effect mediated behavioral

performance impairments (Inzlicht & Gutsell, 2007). Two other experiments examined the aftereffect of depletion using functional neuroimaging. In both cases it was found that the right lateral PFC showed reduced activity in participants who first completed an effortful self-control task, compared to control subjects (Friese, Binder, Luechinger, Boesiger, & Rasch, 2013; Hedgcock, Vohs, & Rao, 2012).

As mentioned above, recent behavioral work suggests that another possible aftereffect of self-regulatory depletion is the intensification of desires and emotions (e.g., Schmeichel, 2007; Vohs et al., 2013). Consistent with this theory are results from two functional neuroimaging studies, one in the emotional domain and the other using tempting food cues. In the first of these, participants were depleted using a difficult attention-control task and subsequently exposed to a series of emotional scenes. Compared to nondepleted individuals, depleted subjects viewing negatively valenced emotional scenes showed an exaggerated response in the amygdala, a region involved in the detection of threat (Wagner & Heatherton, 2013a). Moreover, this was accompanied by reduced functional connectivity between the amygdala and the ventromedial prefrontal cortex, a region commonly involved in emotion regulation, when viewing negatively valenced emotional scenes (Wagner & Heatherton, 2013a). Within the appetitive domain, another study found that dieters who completed a prior self-regulation task subsequently showed an exaggerated reward response in the OFC when viewing highly appetizing food cues. Moreover, compared to nondepleted dieters, depleted dieters showed reduced functional connectivity between the OFC and the lateral PFC during appetizing food trials (Wagner, Altman, Boswell, Kelley, & Heatherton, 2013). Together, the results of these two studies suggest that self-regulatory depletion may serve to impair the functional connectivity between regions important for self-regulation, such as the lateral PFC for appetitive stimuli (Delgado et al., 2008; Kober et al., 2010; Somerville, Hare, & Casey, 2011) and the ventromedial prefrontal cortex for emotional stimuli (e.g., Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007; Somerville et al., 2012). This breakdown in functional connectivity leaves responses in reward and emotion related-brain regions unchecked, thereby leading to an exaggerated response to appetitive (Wagner et al., 2013) and emotionally charged stimuli (Wagner & Heatherton, 2013a).

Conclusion

Based on the research reviewed above, successful self-regulation can be conceptualized as involving a balance

between impulse strength and the capacity to override or otherwise control them. This “balance” model (Heatherton & Wagner, 2011) suggests that when people experience strong urges brought on by exposure to temptations, to the degree that these urges outweigh the capacity to control them, self-regulation failure becomes more likely. Conversely, when the capacity to engage in self-regulation is impaired either by negative affect or prior expenditure of self-regulatory resources, then temptations can exert a greater sway over behavior, again leading to self-regulatory failure. Evidence from cognitive neuroscience suggests that this tug-of-war involves brain regions important for representing the reward value of temptations and the intensity of emotions as well as lateral and medial regions of the PFC important for implementing goal-directed behavior and inhibiting cravings and desires. This relatively simple model of self-regulation failure shares much in common with developmental models of self-control that emphasize differential maturation rates between the prefrontal cortex and regions involved in representing rewards and emotions (e.g., Somerville, Jones, & Casey, 2010). For instance, in this line of research it has been found that compared to adults adolescents show exaggerated responses to appetitive and rewarding stimuli in regions such as the ventral striatum (May et al., 2004; Somerville et al., 2011), and that this is largely due to a failure to appropriately recruit regions of the lateral PFC.

Further evidence for a causal role of the lateral PFC in response inhibition comes from research using transcranial magnetic stimulation as well as transcranial direct current stimulation, which has shown that inactivation of the right lateral PFC increases impulsive behavior and risky decision making (Chambers et al., 2006; Knoch, Pascual-Leone, Meyer, Treyer, & Fehr, 2006) whereas stimulating the function of this region with transcranial direct current stimulation pushes people to become risk-averse and better able to inhibit impulses (Fecteau et al., 2007; Jacobson, Javitt, & Lavidor, 2011). In line with the imaging work reviewed above demonstrating a role for the lateral PFC in regulating cravings and desires (e.g., Delgado et al., 2008; Kober et al., 2010; Wagner et al., 2013), these studies suggest any event that impairs lateral PFC function may precipitate self-regulation failure, particularly when an individual is faced with strong temptations or desires. Moreover, the work reviewed in this chapter suggests that absent the inhibitory influence of the lateral PFC, brain regions involved in representing reward value, emotional valence, or both may become sensitized to environmental triggers, which in turn bias individuals toward seeking out rewards, acting on

desires, and ultimately abandoning their long-term goals.

Finally, it is important to note that, although we have taken the view that cue exposure, mood, and self-regulatory depletion all interfere with the ability to initiate top-down control over impulses and desires, the data from cognitive neuroscience are largely consistent with alternate interpretations such as motivational explanations, which state that the failure to engage in self-control may, in some cases, reflect a conscious choice on the part of individuals to abandon long-term goals in favor of instant gratification. Arbitrating between these competing accounts of how cues, negative affect, and resource depletion bring about self-regulatory collapse presents an important next step for theories of self-regulation failure—one in which, we believe, cognitive neuroscience promises to play a pivotal role.

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