(2024). *Journal of Experimental Psychology: General*, *153*(6), 1582–1604.

Just Do It: A Neuropsychological Theory of Agency, Cognition, Mood, and Dopamine

F. Gregory Ashby[1], Heidi A. Zetzer[2],
Collie W. Conoley[3], and Alan D. Pickering[4]
[1]Department of Psychological & Brain Sciences, University of California, Santa Barbara
[2]Department of Counseling, Clinical, and School Psychology, University of California, Santa Barbara
[3]Department of Psychology, California State University, Long Beach
[4]Department of Psychology, Goldsmiths, University of London

Agency is the sense that one has control over one's own actions and the consequences of those actions. Despite the critical role that agency plays in the human condition, little is known about its neural basis. A novel theory proposes that increases in agency disinhibit the dopamine system and thereby increase the number of tonically active dopamine neurons in the ventral tegmental area. The theory, called ADDS (Agency Disinhibits the Dopamine System), proposes a specific neural network that mediates these effects. ADDS accurately predicts a variety of relevant neuroscience results, and makes many novel predictions, including that increases in agency will 1) increase motivation, 2) improve executive function, 3) facilitate procedural learning, but only in the presence of immediate trial-by-trial feedback, 4) have little or no effect on learning-related effects of stimulus repetition or on standard eyeblink conditioning, 5) facilitate the development of automatic behaviors, but have little or no effect on the production of behaviors that are already automatized, 6) amplify the cognitive benefits of positive mood, and 7) reduce pain. The implications of this new theory are considered for several purely psychological theories that assign prominent roles to agency, including self-efficacy theory, hope theory, and goal-focused positive psychotherapy.

Keywords: agency; dopamine; positive mood; feedback contingency; self-efficacy theory

Author Notes. A preliminary version of this manuscript was posted on PsyArXiv. Gregory Ashby served as lead for conceptualization, investigation, visualization, writing-original draft, and writing-review and editing. Heidi Zetzer, Collie Conoley, and Alan Pickering contributed to conceptualization, investigation, and writing-review and editing.

Correspondence concerning this article should be addressed to F. Gregory Ashby, Department of Psychological & Brain Sciences, University of California, Santa Barbara 93106 USA. email: fgashby@ucsb.edu

Public Significance Statement. Agency is the sense that one has control over one's own actions and the consequences of those actions. A strong sense of agency is critical for optimal performance and mental health. Despite the important role that agency plays in the human condition, little is known about how the brain responds to changes in agency. We propose a novel theory, which predicts that increases in agency increase baseline levels of dopamine throughout the brain and amplify the brain's dopamine response to salient environmental events. The theory describes possible pathways for improving human performance and mental health.

1. Introduction

Agency is the sense that one has control over one's own actions and the consequences that result from those actions (Moore, 2016). An enormous literature suggests that agency plays a critical role in a wide variety of human behaviors (e.g., Bandura, 2006; Mele, 2003; Russell, 2013) and in the efficacy of the psychotherapeutic process (e.g., Bandura, 1977). Despite its importance, there are no theories of how agency influences behavior at the neural level. A number of articles have identified brain regions that become more active when agency is increased (e.g., Crivelli & Balconi, 2017; Haggard, 2017; Sperduti et al., 2011), but none of these attempted to provide a theoretical account of the neural effects of agency.

This article proposes a novel neuropsychological theory of how agency affects cognition and how it interacts with positive mood. Specifically, the theory predicts that increases in agency disinhibit the dopamine (DA) system and thereby

increase the number of tonically active[1] DA neurons in the ventral tegmental area (VTA). This increase raises tonic levels of DA in frontal cortical regions that support creative problem solving, working memory, and more generally, executive function, and also amplifies the DA response to feedback with either positive or negative valence (i.e., positive or negative feedback). The theory, which we refer to as ADDS (Agency Disinhibits the Dopamine System), proposes a specific neural circuit that mediates these effects (described in Figure 1 below) and makes many novel predictions at both the neural and behavioral levels.

One challenge to developing a neuropsychological theory of agency is that the existing literature on the neural basis of agency is arguably too sparse to support the development of a rigorous theory. This is largely because there is no consensus on behavioral signatures that co-occur with changes in the level of agency. For this reason, there are almost no studies with the stated goal of studying the neural basis of agency in nonhuman animals. Even so, we believe a large neuroscience literature has studied the neural effects of manipulating an independent variable that strongly affects agency – namely feedback contingency. Feedback is information received from the environment that follows a behavior (e.g., a reward or punishment). When feedback is noncontingent on behavior, then there is no behavior that can change the feedback, and therefore, agency should be low. When feedback is contingent on behavior, then the agent controls the outcome via their behavior, and therefore agency should be high. For this reason, we propose that manipulations of feedback contingency also manipulate agency, even if this latter manipulation is unintentional. As a result, our approach supplements the existing literature on the neural basis of agency with behavioral and neural studies of feedback contingency. Understanding the neural changes that occur when feedback contingencies are altered can provide invaluable clues about the neural changes that occur when agency is increased or decreased.

The article is organized as follows. The next (i.e., second) section establishes the relationship between agency and feedback contingency. This section provides much of the initial, empirical justification for hypothesizing that agency affects the DA system. The third section describes our novel theory, the fourth section describes neuroscience tests, the fifth describes the theory's predicted effects on motivation, and the sixth describes behavioral predictions, with a specific focus on executive function, skill learning, perceptual priming, eyeblink conditioning, and the effects of agency on automatic behaviors. The seventh section describes predictions of the theory for positive mood, the eighth section describes predictions for how changes in agency should affect pain, and the last section closes with a general discussion and some conclusions.

2. Agency and Feedback Contingency

According to one prominent theory, a sense of agency requires that outcomes are consistent with expectations (Wegner & Wheatley, 1999). When a cue ball that we have hit strikes an object ball in billiards, we have a strong expectation that the object ball will move as soon as it is struck and of the direction in which the object ball will travel. Any deviation from these expectations reduces our sense of agency. The actual time and direction that the object ball moves are feedback that the environment provides about our action of striking the cue ball. Environmental feedback that matches our expectations tends to increase agency, whereas mismatching feedback tends to lower agency.

When feedback is noncontingent on behavior, then prediction errors – that is, the difference between actual and expected outcomes – will tend to be extreme, no matter what the behavior, and consequently, we expect agency to be low. In contrast, feedback that is contingent on behavior allows the agent the opportunity to change the consequences of the behavior (i.e., the feedback) by changing the behavior. This allows agency to be high. Formally, we define feedback contingency as the correlation between actual and expected outcomes.[2] With noncontingent feedback, the correlation is zero because actual and expected feedback will match only by chance, whereas with feedback that is contingent on behavior, the correlation is high. A central thesis of this article is that changes in an agent's estimate of feedback contingency will change the agent's sense of agency.

Note that a prediction error is a property of a single event, whereas estimating contingency requires estimating the correlation over time between actual and expected outcomes, which like any correlation, requires many trials of data. The fact that contingency requires some number of events to estimate is one reason why it is critical to distinguish between agency and feedback contingency. Consider a scenario is which contingency and agency are both high, so that the agent feels in control of their environment. Now suppose that, unbeknownst to the agent, the environment suddenly changes and outcomes become noncontingent on the agent's behavior. Even with optimal statistical methods, some number of events will be needed to detect this change, and during this readjustment period, the sense of agency will be high, even though feedback contingency is low. Thus, agency is purely a psychological property of the actor, whereas feedback contingency depends on statistical properties of the environment.

Although very few neuroscience studies have manipulated agency directly, many have studied the neural networks that

[1] A neuron is tonically active if its baseline firing rate is above zero.

[2] The correlation could be either between the time when feedback is delivered and when it is expected, or between the valence of the obtained feedback and its expected valence.

respond to changes in feedback contingency. Thus, this article focuses on what feedback contingency can tell us about agency because this link allows us to mine the neuroscience literature for studies that might inform a neural theory of agency. But it is important to note that sense of agency depends on more than just feedback contingency. For example, Bandura (1977) suggested that our sense of agency might also be influenced by our observation of the behavior of others (i.e., by vicarious experience), by verbal persuasion, and by our physiological state (e.g., fear can reduce agency). So agency is a multifactorial construct. This article proposes a theory that describes the neural consequences of changes in agency – no matter what variables induce that change. The theory is largely based on neuroscience studies of feedback contingency because 1) there are many such studies; 2) all psychological theories of agency predict that changes in feedback contingency should affect agency; and 3) there are virtually no neuroscience studies that manipulated agency directly.

Many neuroscience studies that examined feedback contingency used tasks in which the feedback is reward related. These studies are so common that in the remainder of this article, when we speak of contingency or feedback contingency, we will almost always mean reward-related feedback contingency. It makes sense to focus on reward-related feedback because rewards are highly salient and predictions about future rewards are thought to occur automatically in human and nonhuman animals. In these studies, a prediction error is a *reward prediction error* or RPE, which is defined as the value of the obtained reward, which can be denoted as R, minus the value of the predicted reward, denoted as P; so RPE = R – P. Positive RPEs signal that the outcome was better than expected, whereas negative RPEs signal that the outcome was worse than expected.

Even a cursory glance at the large neuroscience literature on reward-related feedback contingency leads immediately to the well-known hypothesis that RPE is a major driver of DA neuron firing (Schultz, 1998; Schultz et al., 1997). When the obtained reward is better than predicted, RPE is positive and DA neuron firing increases above baseline. In contrast, when the obtained reward is worse than predicted, RPE is negative and DA neuron firing decreases below baseline.

A central thesis of this article is that studies that manipulated reward-related feedback contingency also manipulated agency. Several lines of evidence provide initial, immediate support for this prediction. First, humans are highly sensitive to variables that affect their own agency (e.g., Metcalfe & Greene, 2007), so they should also be highly sensitive to changes in reward-related feedback contingency. Many studies support this prediction, in both humans and nonhuman animals (Alloy & Abramson, 1979; Chatlosh et al., 1985; Rescorla, 1968). For example, in instrumental conditioning tasks, extinction can be induced simply by suddenly making the time of reward noncontingent on the behavior (Balleine & Dickinson, 1998; Boakes, 1973; Nakajima et al., 2002; Woods & Bouton, 2007). A second prediction is that because there is overwhelming evidence that the DA system is sensitive to changes in RPE, the DA system should also be implicated in more traditional studies of agency. In fact, several studies have linked agency to brain DA levels (e.g., Aarts et al., 2012; Hassall et al., 2019; Render & Jansen, 2019).

2.1 Feedback contingency and associative learning

The main thesis of this article is that changes in agency that depend on reward predictions affect brain DA levels. How could one test this hypothesis? There are a variety of invasive techniques for measuring brain DA levels. In the case of humans however, indirect tests are required. DA is known to play a key role in the synaptic plasticity that mediates certain forms of associative learning (e.g., Steinberg et al., 2013), so one testable prediction is that if agency affects DA levels then it should also affect those forms of associative learning.

DA plays an especially important role in instrumental or operant conditioning and what is known in cognitive psychology as procedural (or skill) learning. An enormous literature implicates the striatum in these forms of associative learning (e.g., Miyachi et al., 2002; O'Doherty et al., 2004; Yin et al., 2005). More specifically, this type of learning is thought to be mediated by long-term, structural changes in the synapses between cortical and striatal neurons that increase the efficacy of synaptic transmission. Collectively, such changes are referred to as synaptic plasticity.

Cortical-striatal synapses are glutamatergic, as are the synapses that subserve almost all excitatory networks in the brain. When glutamate is released presynaptically, it diffuses across the synapse, and then binds to a number of different types of postsynaptic receptors. The most important of these for synaptic plasticity is arguably the N-methyl-D-aspartate (NMDA) receptor. NMDA-receptor activation initiates a number of chemical cascades that can affect synaptic plasticity. One of the most important and best understood is the pathway that phosphorylates calcium/calmodulin-dependent protein kinase II (CaMKII). When calcium enters the cell through the activated NMDA receptor, it binds to calmodulin and the calcium/calmodulin complex phosphorylates CaMKII. When fully phosphorylated, CaMKII initiates a variety of processes that eventually increase the efficacy of the synapse (e.g., Lisman et al., 2002). During the time when CaMKII is partially phosphorylated, it serves as a memory trace, which signals that the synapse was recently active. This trace only lasts a few seconds, and DA plays an important role in these processes because if it enters the synapse while the trace is active then it can potentiate the phosphorylating effects of calcium/calmodulin (via D1 receptor activation) and thereby potentiate synaptic efficacy (Yagishita

et al., 2014).[3] As these neural timing effects predict, many studies have reported that feedback delays of even a few seconds impair associative learning (e.g., Grice, 1948; Maddox & Ing, 2005; Maddox et al., 2003).

A large literature shows that DA neurons in the VTA and substantia nigra pars compacta (SNpc) increase their firing above baseline following unexpected rewards (when RPE > 0) and decrease their firing below baseline following failure to receive an expected reward (when RPE < 0; Hollerman & Schultz, 1998; Mirenowicz & Schultz, 1994; Schultz, 1998. In fact, as mentioned, a popular proposal is that the phasic DA response to feedback is proportional to the RPE (Schultz et al., 1997). This phasic DA response facilitates associative learning. On trials when the RPE is positive, DA levels in the striatum rise above baseline and strengthen recently active synapses. In contrast, on trials when the RPE is negative, DA levels in the striatum fall below baseline and weaken recently active synapses (e.g., Ashby & Crossley, 2011; Houk et al., 1995; Wickens, 1993). Inglis et al. (2021) proposed that the population of DA neurons that are active enough to respond to any given RPE increases with feedback contingency, and therefore contingency serves to act as a gain or amplifier on the DA response to RPE. When contingency is high, the population of tonically active DA neurons is large, and therefore many DA neurons are able to respond to a positive RPE. However, when contingency is low, the population of tonically active DA neurons is small, and therefore fewer DA neurons are able to respond to the same positive RPE. As a result, there is less synaptic plasticity during periods of low contingency than when contingency is high. This model therefore proposes a mechanism via which increased levels of feedback contingency can improve associative learning.

2.2 Evidence that agency alters the DA response to feedback

A wide variety of evidence supports the hypothesis that contingency affects associative learning by altering brain DA levels in all targets of the two most prominent DA-producing regions in the brain (i.e., the VTA and SNpc). First, as previously noted, extinction can be induced in instrumental conditioning tasks simply by suddenly making the time of reward noncontingent on the behavior (Balleine & Dickinson, 1998; Boakes, 1973; Nakajima et al., 2002; Rescorla & Skucy, 1969; Woods & Bouton, 2007). This is predicted by the DA/contingency hypothesis, but it provides only weak support. Extinction could occur even if there is no change in the DA response to reward simply by reinforcing competing behaviors.

Stronger support comes from several studies that have reported that the rate of human procedural learning is reduced when feedback contingency is degraded (Ashby & Vucovich, 2016; Crossley et al., 2013). In the first of these, Crossley et al. (2013) trained adults to classify single lines that varied across trials in length and orientation into one of four categories. The categories were constructed in a way known to recruit procedural learning. The experiment included three phases of 300 trials each: acquisition, intervention, and reacquisition. During acquisition and reacquisition, every response was followed by valid feedback. In the random feedback condition, the intervention feedback was completely random and therefore noncontingent on the participant's behavior. Random feedback is unpredictable and therefore generates large RPEs, so if the DA response is unaffected by changes in feedback contingency then participants should have learned the random stimulus-response associations that were reinforced during the intervention phase in this condition. Since these random associations are incompatible with the correct classification responses that were learned during acquisition, reacquisition therefore should have been at least as slow as original acquisition. In contrast to this prediction, reacquisition was significantly faster than acquisition – suggesting that learning was depressed during the random feedback intervention, as predicted by the assumption that the DA response to any given RPE is attenuated in low contingency conditions.

Ashby and Vucovich (2016) tested the effects of varying feedback contingency on category learning more directly (see also Vucovich, 2016). They compared learning in three conditions that differed in feedback contingency (low, medium, or high). All conditions used category structures known to recruit procedural learning, they all used exactly the same stimuli, required the same classification strategy for optimal performance, and had the same optimal accuracy. The results showed that the amount of learning in the three conditions increased with feedback contingency. Learning was good when feedback contingency was high, significantly worse in the medium-contingency condition, and almost absent in the low-contingency condition. This result is counter to many prevailing theories of learning because low-contingency feedback produces large RPEs, which according to many current theories should result in high levels of learning. In summary, the relevant behavioral studies strongly suggest that the DA response to RPE is modulated by feedback contingency.

At the neural level, there is also evidence that changing feedback contingency changes how the brain responds to RPEs. For example, functional magnetic resonance imaging (fMRI) studies in humans have shown that activity in the dorsal striatum – that is, in the region most strongly implicated in operant conditioning and procedural learning – is correlated with RPE when feedback is contingent on behavior, but not when feedback is independent of behavior (Haruno & Kawato, 2006; O'Doherty et al., 2004).

[3] DA cannot affect these processes when the CaMKII either has no bound phosphate or is fully phosphorylated. For these reasons, CaMKII serves as a trace of recent activity when it is partially phosphorylated.

3. A Theory of How Agency Affects Brain DA Levels

Reward and feedback processing recruit diverse brain networks that include the limbic system and prefrontal and sensory cortices (Faget et al., 2016; Haber, 2016; Liu et al., 2011; Takahashi et al., 2016; Tian & Uchida, 2015; Watabe-Uchida et al., 2012). Multiple brain regions respond to obtained and predicted rewards (Bromberg-Martin et al., 2010; Humphries & Prescott, 2010; Sesack & Grace, 2010), and this redundancy led to the formulation of many alternative theories to explain the mechanisms via which DA firing is modulated by RPE (Brown et al., 1999; Contreras-Vidal & Schultz, 1999; Hazy et al., 2010; Houk et al., 1995; Joel et al., 2002; Kawato & Samejima, 2007; Morita et al., 2013; O'Reilly et al., 2007; Schultz, 1998; Schultz et al., 1997; Stuber et al., 2008; Sutton & Barto, 1998; Tan & Bullock, 2008). Despite all this work, we know of no theories that account for the effects of agency on DA neuron firing. As a result, our focus here is on the subnetwork via which changes in agency (e.g., feedback contingency) might cause changes in DA neuron firing.

First, even when rewards are delivered noncontingently, electrolytic lesions of the hippocampus cause rats to behave "as if a dependency exist(s) between pellet delivery and their behavior" (p.721, Devenport, 1979). In other words, hippocampal lesions appear to disrupt an animal's ability to compute reward contingency. Corbit et al. (2002) later showed that this result is caused by damage to the entorhinal cortical efferent fibers passing into hippocampus. In particular, they showed that lesions of the entorhinal cortex, but not the dorsal hippocampus, reduce sensitivity to changes in reward contingency. Second, disruption of the DA-mediated interaction between the entorhinal cortex and the striatum reduces sensitivity to changes in reward contingnecy (Lex & Hauber, 2010). Third, the medial prefrontal and obitofrontal cortices have been implicated in enabling flexible behavioral adjustments when contingency is degraded (Izquierdo et al., 2004; Jackson et al., 2016) and during explicit detection of causal or temporal relationships between actions and rewards (Jocham et al., 2016; Tanaka et al., 2008; Walton et al., 2010). Interestingly, rats with hippocampal lesions have been found to be less flexible in the face of changing contingencies, which could mean that without hippocampal encoding of reward contingency, the rats default to less flexible associative-learning systems (Kosaki & Watanabe, 2012).

In summary, considerable evidence links the hippocampus and other medial temporal lobe (MTL) structures to contingency estimation. As noted above, (reward-related) feedback contingency is a correlation between the numerical values of predicted and obtained reward (i.e., between P and R), and there is a variety of other evidence that the MTL are well suited to such computations. In particular, the hippocampus appears to compute similar correlations between spatial and nonspatial cues (e.g., object identity and time; Eichenbaum et al., 2016; Lee & Lee, 2013; Redish & Touretzky, 1997), the entorhinal cortex encodes general properties of the current context (Jacobs et al., 2010), and the parahippocampal cortex has a general role in contextual binding (Aminoff et al., 2013). Furthermore, the entorhinal cortex receives almost all of its cortical inputs from polymodal association areas, including cingulate, orbitofrontal, and parahippocampal cortices, making it well situated for integrating diverse inputs (Insausti et al., 1987).

Other regions are also likely to contribute to the contingency estimates that drive agency, including anterior cingulate and prefrontal cortices. For example, the anterior cingulate increases activity when a commitment is made to act (Blanchard et al., 2015; Ma et al., 2014) and this activity increases with the effort required by that action (Cowen et al., 2012; Hillman & Bilkey, 2010). Furthermore, following electrical stimulation of the anterior cingulate (i.e., in the anterior midcingulate cortex), awake behaving humans report "anticipation of challenge coupled with strong motivation to overcome it" (Parvizi et al., 2013, p. 1364).

In summary, sense of agency is likely computed by a widespread network that includes contingency-sensitive regions of the anterior cingulate, prefrontal cortex, and hippocampus. The cerebellum also likely contributes by detecting discrepancies between predicted and actual outcomes of motor behaviors (Welniarz et al., 2021). The output node of this complex network is likely to be in the ventral subiculum (vSub), since this is the main output structure of the hippocampus. For the present purposes, a more detailed model is not needed, since our goal is not to model how agency is computed, but rather to understand the neural changes that occur when the sense of agency increases or decreases.

Synofzik et al. (2008) distinguished between what we are referring to as the sense of agency, which they defined as "the non-conceptual, low-level feeling of being the agent of an action" and what they called the judgment of agency, which they defined as "the conceptual, interpretative judgment of being an agent" (p. 222). By this account, judgment of agency, which is a high-level conscious interpretation of the sense of agency, presumably recruits a more widespread and complex neural network than the one described in this section.

We adopt the Inglis et al. (2021) model of how feedback contingency gates the DA response to RPE, and we extend this model to agency. The proposed theory, referred to as ADDS (Agency Disinhibits the Dopamine System), is described in Figure 1. The right half of the network instantiates the standard RPE model. The idea is that reward sensitive units in regions such as prefrontal and orbitofrontal cortex contribute to the RPE DA signal by providing excitatory inputs to the pedunculopontine tegmental nucleus (PPTN) (Hong & Hikosaka, 2014; Kobayashi & Okada, 2007; Okada & Kobayashi, 2013) and lateral habenula (Hong et al., 2011;

Matsumoto & Hikosaka, 2007, 2009; Tian & Uchida, 2015). Through these circuits, positive RPEs excite DA neurons via the PPTN, whereas negative RPEs inhibit DA activity via the lateral habenula (and the rostromedial tegmental nucleus).

The left half of Figure 1 is more novel and also more relevant to the current article. The idea is that an extensive network that includes regions in the anterior cingulate, prefrontal cortex, and hippocampus continuously updates estimates of agency, and that the outputs of this network gate the amount of DA release via projections through the hippocampal vSub (via projections proposed by Grace et al., 2007). We assume that any variable that increases the sense of agency – be it feedback contingency, observational learning, verbal persuasion, or physiological state – will increase the output of this network, and therefore increase activity in vSub.

The projections from vSub to the nucleus accumbens are excitatory and the projections from the accumbens to the ventral pallidum and from the ventral pallidum to the DA neurons of the VTA are inhibitory. Even so, a key feature of this neuroanatomy is that the tonic firing rate of ventral pallidal neurons is much higher than the tonic firing rate of nucleus accumbens neurons. As a result, many DA neurons in VTA are silent due to tonic inhibition by the ventral pallidum. Estimates suggest that because of this inhibition, only about half of VTA DA neurons are spontaneously active under control conditions, and these tonically firing neurons are the only ones available to respond to RPEs (Lodge & Grace, 2006). ADDS predicts that when agency is high, vSub excites the nucleus accumbens, which inhibits the ventral pallidum. This releases VTA DA neurons from tonic inhibition, which increases the number of tonically firing VTA DA neurons, thereby raising tonic DA levels in all VTA target brain regions and enlarging the pool of DA neurons that can respond to RPEs. In contrast, if agency suddenly drops, ADDS predicts that the vSub excitation of the nucleus accumbens will decrease, which reduces inhibition on the ventral pallidum, and that the resulting subsequent increase in pallidal activity will increase inhibition of the VTA DA neurons, thereby reducing the number that are tonically active.

ADDS therefore makes two fundamental and novel neuroscience predictions. First, increases in agency should increase tonic DA levels in all VTA target regions (e.g., frontal cortex). Second, increases in agency should increase the number of DA neurons available to respond to feedback and thereby amplify the DA response to any given RPE. In other words, the higher the agency, the higher the DA levels will rise above baseline for any given positive RPE and the lower the DA levels will fall below baseline for any given negative RPE.[4] Furthermore, these predictions are causal in the sense that ADDS predicts that any increase in agency, no matter what the cause, will lead to these predicted DA effects. In contrast, ADDS makes no predictions about how changes in tonic DA might affect the sense of agency (i.e., note in Figure 1 that the VTA DA neurons are downstream from the network that computes agency). As we will shortly see, these very specific neural predictions allow for many novel and strong neural and behavioral predictions.

4. Neural Tests of the Theory

4.1 Tests of the proposed neural circuitry

Inglis et al. (2021) built a computational model of the Figure 1 network constructed from mathematical models of spiking neurons, and they tested this model against a variety of different neuroscience results. These tests made no assumptions about the psychological construct(s) that was modulating vSub output, and therefore say nothing about the possible role that agency plays in the Figure 1 model. Even so, they provide strong tests of the validity of the rest of the network.

The first test, which is summarized in Figure 2, examined the ability of the model to account for results reported by Lodge and Grace (2006). In this experiment, the authors activated the vSub, the pedunculopontine tegmental nucleus, or both of these structures (via NMDA infusion, in rats), and they then examined how these excitations affected the number of VTA DA neurons that were firing tonically and the average firing rate of all currently active VTA DA neurons. They also compared these results to results from a control condition in which neither structure was activated. The results are shown in the left column of Figure 2. The right column shows predictions of the computational version of the Figure 1 model under these same conditions. Note that the model accurately predicts that vSub activation increases the number of tonically active VTA DA neurons without increasing the firing rate of active neurons, whereas activation of the PPTN results in the opposite profile – that is, the firing rate of active DA neurons is increased but not the number of tonically active neurons.

As a second test, Inglis et al. (2021) showed that the model is consistent with results reported by Bayer and Glimcher (2005), in which DA neuron firing increased linearly with RPE between minimal and maximal values. Third, Inglis et al. (2021) showed that the model could also account for the data of Hart et al. (2014), in which extracellular DA concentrations were a linear function of RPE and that positive and negative RPEs were encoded symmetrically.

These tests support the validity of the Figure 1 model and therefore, greatly sharpen our possible predictions. For example, without the Figure 1 network, we would be left only

[4] This latter prediction follows because increasing the number of tonically active DA neurons increases the number than can respond to inhibitory input from the RMTN on negative RPE trials (i.e., see Figure 1). In other words, raising the tonic DA level increases the range over which DA levels can drop in response to negative feedback.

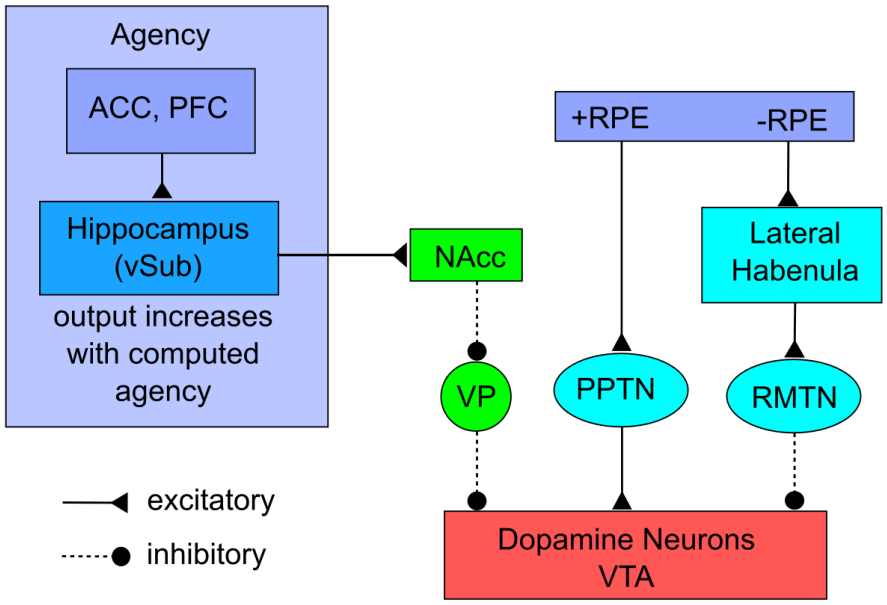

Figure 1

Proposed theory (ADDS) of how agency modulates the firing of dopamine neurons. ACC = anterior cingulate cortex; PFC = prefrontal cortex; vSub = ventral subiculum; RPE = reward prediction error; NAcc = nucleus accumbens; VP = ventral pallidum; PPTN = pedunculopontine tegmental nucleus; RMTN = rostromedial tegmental nucleus; VTA = ventral tegmental area. Adapted from Inglis, Valentin, and Ashby (2021).

with the prediction that agency affects brain DA levels in some unknown way. With this network however, we can make the strong prediction that increases in agency will increase the number of DA neurons that are tonically active without affecting the average firing rate of DA neurons that are active. The specificity of this prediction allows many of the strong behavioral predictions of ADDS that are derived and tested in later sections of this article.

Inglis et al. (2021) did not test their model against any behavioral data, and they made no strong claims about the possible cortical and hippocampal inputs to vSub. Instead they hypothesized only that these inputs were sensitive to some modulating variable that might affect neural learning rates. They speculated that this variable could be expected uncertainty, unexpected uncertainty, volatility, environmental uncertainty, the covariance between predicted reward and past RPEs, or feedback contingency. Importantly, however, they made no mention of agency. The key contribution of the present article therefore, is to link the Inglis et al. (2021) network to agency, and to explore implications of this hypothesis.

4.2 Neural tests of ADDS

The studies considered so far in this section test the validity of the neural circuitry described in Figure 1, but they do not test the core assumption of ADDS that the key driver of vSub activity is estimated agency. Because ADDS is a new theory, there are no published neuroscience studies directly testing this key assumption. Even so, a number of published neuroscience results are highly relevant.

ADDS predicts that a cortical network that includes regions of the anterior cingulate continuously updates agency and that these computations alter the size of the population of tonically active DA neurons in the VTA via the neural network described in Figure 1. A strong test of this prediction was provided by Elston and Bilkey (2017), who implanted recording electrodes in the anterior cingulate, the VTA, and the dorsal CA1 subregion of the hippocampus of rats, and then trained the animals to run laps around a track for a fixed food reward. Importantly, the physical effort required for each lap was varied by sometimes including a barrier over which the animals had to climb. The hippocampal recordings are important because the dorsal CA1 subregion sends a prominent projection to vSub. As predicted by the Figure 1 network, the results suggested that anterior cingulate activity had a causal influence on both hippocampal and VTA activity. Furthermore, because agency presumably increases with effort, ADDS also correctly predicts that this influence was modulated by the amount of effort required (for more details on ADDS's predicted effects on effort, see the next section).

The VTA DA neurons project to the ventral striatum (i.e., nucleus accumbens) and all of frontal cortex, so ADDS predicts that changes in agency should affect DA levels in any of these target areas. All theories of agency predict that

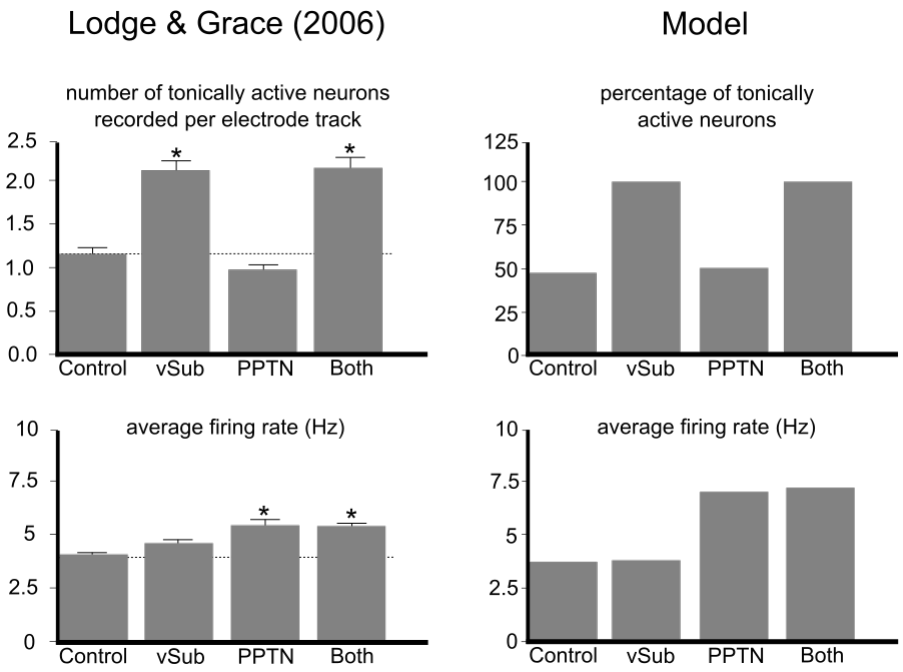

Figure 2

*Fits of a computational version of the Figure 1 model to the data of Lodge and Grace (2006). vSub = ventral subiculum; PPTN = pedunculopontine tegmental nucleus; RMTN = rostromedial tegmental nucleus. * = statistically signficant difference from control ($p < 0.05$). Error bars indicate one standard error. Adapted from Inglis, Valentin, & Ashby (2020).*

agency should be higher when a reward is received following a motor action than when a reward is received passively (e.g., Frith, 2005; Moore, 2016). However, ADDS makes the additional and specific neural prediction that DA levels will be higher in VTA target areas in the former case than in the latter. In support of this prediction, after presentation of a reward-predicting stimulus, DA levels are higher in the nucleus accumbens of rats on trials when the animal initiates an action than on trials when no motor response is required (Syed et al., 2016).

Related results have been found with humans. In particular, Hassall et al. (2019) reported that the reward-related positivity, which is an event-related brain potential that is widely thought to index RPEs, is greatly reduced in a (two-armed bandit) gambling task when the participant no longer needs to act. From this they concluded that "agency ... affects the generation of a neural prediction error signal" (p. 1463). Furthermore, other studies have reported that the increased agency that results from active choice versus passive viewing enhances declarative memory and that these enhancements are associated with trial-by-trial interactions between the striatum and the hippocampus (Murty et al., 2015).

Other neuroscience tests of ADDS come from studies of the partial reinforcement extinction effect (PREE). In a typical experiment, reinforcement is used to train a behavior in two groups of animals (e.g., maze running). One group receives continuous reinforcement (CR) and the other receives partial reinforcement (PR; e.g., reward might be given on a random 50% of trials). After training, both groups enter an extinction phase in which all rewards are withheld. The PREE refers to the fact that the PR group extinguishes the learned behavior more slowly than the CR group.[5]

Note that during extinction, the CR group could theoretically detect the statistical change in reward contingency after a single non-rewarded response, whereas the PR group would require many non-rewarded responses. This statistical difference between CR and PR provides a well-known account of the PREE (Capaldi, 1967). In addition, however, note that all theories of agency predict higher agency with CR than with PR. As a result, ADDS predicts that agency differences between the two groups could also contribute to the PREE. Specifically, ADDS predicts that the lower levels of agency for the PR group mean fewer active DA neurons during extinction, and therefore slower unlearning of the behavioral response.

ADDS also makes neuroscience predictions about the PREE. Specifically, ADDS predicts that the PREE should be disrupted by hippocampal lesions and by dopaminergic drugs. There is good support for both of these predictions.

[5]For a detailed description of the PREE and the multiple theories advanced to explain it, see McNaughton (1989, pp. 80-92).

Large lesions of the hippocampus abolish the PREE (see Gray et al., 2002, for references). Most importantly, the effects of septo-hippocampal lesions on the PREE seem to derive specifically from damage to the pathway from the vSub to the ventral striatum, exactly as ADDS predicts (Rawlins et al., 1989). DA agonists (e.g., amphetamine) also abolish the PREE under certain conditions, as do DA antagonists, (e.g., haloperidol; see Gray et al., 2002, for references). On the other hand, it should be noted that the PREE did not show any sensitivity to amphetamine when tested in human participants (Gray et al., 2002).

5. Predicted Effects of Agency on Motivation

One straightforward prediction of ADDS is that increases in agency should cause increases in motivation. At first glance, this prediction seems obvious since it is also made by all theories of agency. The difference however, is that ADDS is unique in that it specifies the specific neural events that mediate these motivational effects. The key to deriving this prediction comes from the neural literature on response vigor. This literature is relevant to motivation because it interprets the term "vigor" as a behavioral manifestation of the energizing effects of motivation (e.g., Niv et al., 2006). In a typical experiment, a hungry rat is placed in a maze that includes two baited arms – one containing a large reward and one containing a small reward. Critically, access to the large reward is blocked by a barrier that the rat must climb over, whereas the arm with the small reward is unguarded. So the animal must choose between a large reward that requires a large effort and a small reward that requires considerably less effort. These studies are relevant to ADDS because a large literature suggests that DA plays a key role in the rat's choice (for reviews, see, e.g., Niv, 2007; Salamone et al., 1994). For example, untreated, control rats often choose the high-effort, high-reward arm, whereas a number of studies have reported that administering a DA antagonist or depleting DA from the nucleus accumbens sharply reduces the choice of the high-effort arm (Cousins et al., 1996; Salamone et al., 1994). DA depleted rats still seek reward, but now they choose the smaller reward that requires less effort – in other words, they display classic signs of reduced motivation.

Many similar results have been reported with humans. For example, a variety of studies have reported that Parkinson's disease patients, who have reduced brain DA levels, have lower levels of motivation and display reduced levels of vigor, compared to age-matched controls (e.g., Chong et al., 2015; Mazzoni et al., 2007). Furthermore, medications that increase brain DA levels reduce these deficits (Chong et al., 2015; Czernecki et al., 2002; McGuigan et al., 2019).

One prominent theory is that these motivational effects depend predominantly on tonic DA levels, rather than on any phasic DA response (Niv et al., 2007). According to this account, the higher the levels of tonic DA, the more effort an agent will be willing to expend to secure a reward. Subsequent research indicates that phasic DA responses also contribute to motivation (Hamid et al., 2016; Howe et al., 2013; Salamone & Correa, 2012). For example, tonic DA levels change slowly, over the course of many trials. So if motivation only depended on tonic DA, changes in motivation should also be slow. In contrast to this prediction, animals become more reluctant to re-engage in a task immediately after receiving a reward, and both motivation and striatal DA levels quickly ramp up as an animal approaches a reward (Howe et al., 2013). ADDS predicts that increases in agency increase tonic DA levels as well as the phasic DA responses that contribute to these effects, and therefore ADDS predicts that increases in agency will increase motivation and vigor.

A huge literature suggests that agency increases motivation (e.g., Heckhausen et al., 2019; Mele, 2003). These results are consistent with almost all theories of agency. Even so, among these theories, ADDS is unique because it also specifies the neural network that mediates these effects. For example, ADDS correctly predicts that DA levels in the nucleus accumbens should be especially important in regulating vigor and motivation (as established, e.g., by Salamone et al., 1994). Specifically, ADDS predicts that reduced DA levels in the nucleus accumbens will reduce the accumbens inhibition of the ventral pallidum (see Figure 1), which will increase the pallidal inhibition of the VTA, which will reduce the size of the tonically active VTA DA population, and finally that this reduction will cause decreases in motivation.

6. Behavioral Predictions

ADDS predicts that changes in the sense of agency could affect any behavior that is sensitive to DA. Even so, some behaviors seem unaffected by DA, and among the DA-sensitive behaviors, DA can affect different behaviors in qualitatively different ways (Ashby et al., 2015). There are several reasons for this. First, DA neurons project to much, but not all of the brain, so behaviors mediated within brain regions that are not DA targets will be relatively unaffected by changes in DA compared with behaviors mediated within brain regions that receive rich DA projections. Second, phasic DA bursts increase DA in the synapses of all DA neuron targets, but the persistence of these DA elevations is very different in say, basal ganglia versus prefrontal cortex, and as a result, ADDS predicts that changes in agency should have different effects on behaviors mediated primarily in these different brain regions.

This section derives behavioral predictions of ADDS for a variety of different types of behaviors. Because these predictions are, for the most part, novel, none of them have been rigorously tested. Even so, in each case, some published studies offer promising preliminary support for ADDS's predictions.

6.1 Executive function

Executive functions encompass a variety of skills that are mediated primarily within the prefrontal cortex and that include working memory, creative problem solving, selective attention, and self-control (e.g., Gilbert & Burgess, 2008). The prefrontal cortex receives a dense DA projection from the VTA and a wide range of evidence suggests that modest increases in DA facilitate executive function (for reviews, see e.g., Ashby et al., 2015; Ott & Nieder, 2019).[6] On the other hand, the evidence also suggests that there is an optimal DA level for executive function, and that performance is compromised if DA levels are either higher or lower than this optimal level (e.g., Vijayraghavan et al., 2007). A thorough review of this literature is beyond the scope of this article. Instead, just a few suggestive results will be described. For example, several studies have reported improvements in the working memory of healthy humans who were given small doses of a drug that mimics the effects of DA (i.e., a DA agonist; e.g., Luciana et al., 1992; U. Müller et al., 1998). Similarly, a drug that slows DA degradation in prefrontal cortex by inhibiting the action of catechol-O-methyltransferase (COMT) was found to improve performance on a variety of executive function tasks (Apud et al., 2007). In addition, several non-human animal studies have confirmed that over-stimulation of prefrontal DA receptors impairs working memory (Lauzon et al., 2013; Sawaguchi & Goldman-Rakic, 1991), supporting the hypothesis that executive function is optimized when cortical DA levels are below their maximum value.

ADDS predicts that an increased sense of agency will amplify these results.[7] And it also predicts that these effects should be relatively reward independent. This latter prediction follows for two reasons. First, an increased sense of agency is predicted to increase the number of tonically active DA neurons, regardless of how the increased agency occurs. For example, even in the absence of reward, an increase in agency caused by explicit instruction or via the agent's expectations about the environment should cause tonic DA levels in prefrontal cortex to increase, resulting in improved executive function (assuming baseline DA levels were below the level required for optimal performance).

Second, prefrontal cortex has negligible concentrations of dopamine transporter (DAT), a molecule that binds to free DA and quickly clears it from the synapse (e.g., Varrone & Halldin, 2014). Instead, free DA in prefrontal cortex is slowly degraded by the enzyme COMT. As a result, any phasic burst of VTA DA neurons is likely to cause elevated DA in prefrontal cortex that persists for many minutes. For example, the delivery of a single food pellet to a hungry rat increases prefrontal DA levels for approximately 30 minutes (Feenstra & Botterblom, 1996). As a result, any unexpected reward when agency is high should raise prefrontal DA levels – and therefore facilitate executive function – for approximately 20-30 minutes.[8]

In support of these predictions, Loyola-Navarro et al. (2022) reported that a higher sense of agency improves working memory. In this study, participants completed a modified version of the Sternberg (1966) memory-scanning task during EEG recording. Sense of agency was manipulated by varying the participant's control of stimulus presentation. As predicted by ADDS, accuracy was higher with higher agency (i.e., when participants had more control of stimulus presentation) and the EEG results also suggested that agency improved selective attention (i.e., the attention-related P200 evoked potential occurred earlier in the conditions with higher agency).

Other studies also reported that increases in agency improved selection attention. In particular, Kumar et al. (2015) reported enhanced selective attention during visual search on trials when participants initiated the onset of the search display, compared to trials when participants had no control over display onset.

In summary, ADDS predicts that an increased sense of agency will facilitate all forms of executive function for a period of 20–30 minutes (i.e., assuming cortical DA levels are below optimal levels when agency is increased), and that this effect should persist even if the sense of agency decreases during this period of time (i.e., because cortical DA levels change so slowly).

6.2 Skill or procedural learning

Procedural learning is a type of learning that requires repeated practice that includes immediate and consistent feedback (e.g., Willingham, 1998). Improvements are incremental and typically include little conscious recollection or even awareness of what was learned. The most widely cited behaviors that are acquired via procedural learning are motor skills, such as playing a musical instrument, riding a bicycle, or putting a golf ball (known colloquially as muscle memories). Even so, there is now abundant evidence that many more purely cognitive behaviors also recruit procedural learning and memory systems (e.g., Ashby & Maddox,

[6] A popular model is that DA elevations improve executive function because these skills are largely mediated by glutamatergic networks and DA potentiates the glutamate response through NMDA receptors (via DA D1 receptor activation) while simultaneously depressing the glutamate response through non-NMDA receptors (via DA D2 receptor activation; e.g., Ashby & Casale, 2003). Because NMDA receptors have a higher threshold for activation than non-NMDA receptors, these DA effects serve to increase the signal-to-noise ratio of affected synapses, thereby boosting performance of the network.

[7] For the most part, we mean that increases in agency should benefit executive function. However, in the rare cases in which cortical DA levels are so high that executive function is compromised, ADDS predicts that increases in agency will actually impair executive function. To our knowledge, however, there are no empirical tests of this prediction.

[8] "Unexpected" is critical here because if the reward is expected, then RPE = 0, and the DA neurons will not fire a phasic burst.

2005; Ashby & Valentin, 2017; Ashby & Waldron, 1999).

There is now overwhelming evidence that procedural learning depends on the basal ganglia, and especially on the striatum. This evidence comes from single-unit recording studies in non-human animals (Carelli et al., 1997; Merchant et al., 1997), animal lesion experiments (Eacott & Gaffan, 1991; Gaffan & Eacott, 1995; Packard & McGaugh, 1992), neuropsychological patient studies (Filoteo et al., 2001, 2005; Knowlton et al., 1996), and human neuroimaging studies (Nomura et al., 2007; Seger & Cincotta, 2005; Waldschmidt & Ashby, 2011).

The basal ganglia receive a dense DA projection, from both the SNpc and the VTA. The striatal regions thought to mediate procedural learning, however (i.e., the dorsal striatum), receive DA primarily from the SNpc. Furthermore, unlike cortex, these striatal regions are rich in DAT. As a result, phasic bursts of SNpc DA neurons cause DA levels in the striatum to rise, but only for a few seconds.[9]

Procedural learning requires immediate feedback. In fact, a feedback delay of only a few seconds can abolish almost all procedural learning (Maddox & Ing, 2005; Maddox et al., 2003; Yagishita et al., 2014). This is thought to be because feedback interpreted as rewarding generates a positive RPE, which causes striatal DA levels to rise above baseline, which causes recently active synapses to be strengthened.[10] In contrast, the failure to receive an expected reward generates a negative RPE, which causes DA levels to fall below baseline, which causes recently active synapses to be weakened (e.g., Calabresi et al., 1996; Reynolds & Wickens, 2002). Both of these effects are needed to learn the arbitrary stimulus-response associations that characterize procedural learning.

Therefore, ADDS predicts that an increase in agency will have no effect on procedural learning in the absence of immediate trial-by-trial feedback. However, if immediate trial-by-trial feedback is available, and if the learner interprets the positive feedback received as rewarding, then ADDS predicts that increasing the sense of agency will facilitate procedural learning because the larger population of tonically active DA neurons will act to increase the gain on the DA response to both positive and negative RPEs. In other words, ADDS predicts that the same positive RPE will result in a larger increase in striatal DA concentrations the higher the sense of agency, while the same negative RPE will result in a greater decrease in striatal DA concentrations below their baseline level, and furthermore, that this increase in the dynamic range of the DA response will facilitate synaptic plasticity and therefore procedural learning.[11]

A large literature supports this prediction. At least one study directly examined the relationship between sense of agency and motor learning. In particular, van der Wel et al. (2012) reported that higher agency ratings were associated with greater accuracy in a motor-learning task where immediate feedback was provided by visual observation. In addition though, many other studies have provided more indirect tests, by comparing motor learning in groups of participants who are allowed to make choices about their training conditions – for example, about how feedback is delivered or about the use of physical assistive devices – to the learning that occurs in groups who train under the exact same conditions, but without any choice. All theories of agency predict that the act of choosing increases agency. These studies overwhelmingly report better learning by the choice groups than by the no-choice groups (for reviews, see Sanli et al., 2013; Wulf, 2007). In fact, the benefits of choice even extend to features that are irrelevant to the actual motor skill. For example, Lewthwaite et al. (2015) had two groups of participants practice a golf-putting task. One group was allowed to choose the color of the golf balls they used during practice and the other group was given the same colored balls as the choice group. The results showed that this simple act of choice led to higher putting accuracy that persisted for at least 24 hours, relative to the no-choice group, even though that choice was irrelevant to the skill being learned.

In summary, ADDS predicts that an increased sense of agency should facilitate all forms of procedural learning by amplifying the response of the DA system to any given RPE. In other words, ADDS predicts that increases in agency should cause striatal DA levels to rise higher to any given positive RPE and fall lower to any given negative RPE, thereby causing more synaptic plasticity. Therefore, there should be more synaptic strengthening following successful behaviors and more synaptic weakening following unsuccessful behaviors when agency is high. Even so, it is important to note that the these predictions assume immediate feedback is given after every behavior. If the feedback is delayed by even a few seconds, then the molecular trace of the synaptic activity (e.g., partially phosphorylated CaMKII) will be gone and it will be impossible to determine which synapses were responsible for the emitted behavior. As a result, under these conditions, the level of agency should be irrelevant because all forms of procedural learning should be impossible.

It is also important to note that although ADDS predicts that increases in agency should benefit both executive func-

[9]The Figure 1 model describes how agency could increase the number of tonically active DA neurons in the VTA, whereas the predictions in this section assume that agency has similar effects on SNpc DA neurons. Therefore, the predictions in this section must be more tentative than the other predictions discussed in this article. Even so, there are reasons to believe that agency would have similar effects on VTA and SNpc DA neurons. For example, the basal ganglia have a well-known ascending spiral architecture that allows activity in the VTA to affect activity in the SNpc (Haber et al., 2000).

[10]Recently active synapses are those in which a trace is still active (e.g., partially phosphorylated CaMKII).

[11]By strengthening synapses that led to successful behaviors (i.e., trials when the RPE > 0) and weakening synapses that led to unsuccessful behaviors (i.e., trials when the RPE < 0).

tion and procedural learning, the theory makes the strong and more subtle prediction that the timecourse of these effects should be very different. Because the DA reuptake molecule DAT is almost absent from prefrontal cortex, ADDS predicts that the benefits of an increase in agency on executive function should persist for 20 minutes or so, even if the sense of agency drops during this time. In contrast, the dorsal striatum is rich in DAT and as a result, any DA-related facilitation in procedural learning should closely track increases and decreases in the sense of agency.

6.3 Perceptual priming and eyeblink conditioning

This subsection highlights two behaviors that ADDS predicts should be relatively unaffected by changes in agency. In both cases, this prediction follows because the evidence suggests that the behaviors are mediated primarily in brain regions that do not receive a prominent DA projection, and therefore as a result, there is no direct way for a change in agency to alter the acquisition or expression of these behaviors. This subsection is important because it demonstrates the precision of ADDS – specifically, it shows that ADDS does not simply predict that increases in agency should benefit all behaviors. Instead, ADDS makes qualitatively different predictions for different kinds of behaviors.

Perceptual priming

Perceptual priming is the phenomenon in which mere exposure to a stimulus influences a future response to that stimulus, or to a similar stimulus, even in the absence of any conscious awareness. The behavioral effects of perceptual priming can be observed after only a single stimulus repetition (e.g., Wiggs & Martin, 1998), and the effects of a single stimulus (i.e., picture) presentation can persist for as long as 48 weeks (Cave, 1997).

Although there is no consensus theory that provides a complete description of the neural basis of perceptual priming, there is widespread agreement that perceptual priming is associated with a reduced neural response in sensory cortex to stimulus repetitions (e.g., Schacter & Buckner, 1998; Wiggs & Martin, 1998). Visual and auditory cortex receive almost no DA input. As a result, a change in tonic DA levels will have no direct effect on processing in visual or auditory cortex. Support for this view comes from studies showing that perceptual priming is intact in Parkinson's disease (e.g., Koivisto et al., 1996; Vingerhoets et al., 2005), despite the DA reductions that characterize the disease. For this reason, ADDS predicts that changing levels of agency should have little or no effect on perceptual priming. An equivocation that agency could have some effect on priming is needed because virtually all of sensory cortex receives prominent top-down inputs from prefrontal cortex. As we have seen, prefrontal cortex receives a prominent DA input and is a likely mediator of many agency-related effects. Therefore, in the absence of a more complete theory of priming, it is impossible at this time to rule out a small agency-related effect on priming that is mediated by top-down input from prefrontal cortex. Even so, in contrast to the other behaviors considered in this section, ADDS predicts that agency should have little or no effect on any learning that is due to perceptual priming. This null prediction is important because it demonstrates that ADDS does not predict that agency will have the same beneficial effects on all types of learning.

We know of no priming studies that directly tested this ADDS prediction. Even so, there is some strongly supportive evidence. For example, several studies have reported that perceptual priming is intact even under general anesthesia. In one of these, Iselin-Chaves et al. (2005) repeatedly read lists of 40 words to two groups. One group included 48 surgical patients who were administered general anesthesia in preparation for their surgeries. After they lost consciousness, and therefore presumably all sense of agency, they were read the lists of words. Thirty-six hours later, their memory for the words was tested in a word-stem completion task and the data were analyzed using process-dissociation logic (Jacoby, 1991). In addition, a control group of 24 participants who were matched on age, sex, and level of education completed the same task while fully conscious. The results showed that both groups displayed equal amounts of learning that could be attributed to (implicit) perceptual priming, even though the anesthetized patients had little or no explicit memory of any of the stimulus words.

A variety of other studies have reported similar results (e.g., Deeprose & Andrade, 2006; Flouda et al., 2013; Quan et al., 2013). In addition, learning-related effects of perceptual priming appear to also be intact during sleep (Oniz et al., 2015). On the other hand, some studies have failed to find any learning-related priming effects during anesthesia (e.g., Bejjani et al., 2009). One possible explanation of these apparently contradictory results is that there appears to be a dose-response curve for the effects of anesthesia on perceptual priming. Specifically, several studies have reported that perceptual priming is intact during light and moderate anesthesia, but abolished during deep anesthesia (Flouda et al., 2013; Iselin-Chaves et al., 2005; Quan et al., 2013). Note that ADDS makes no predictions about any effects of deep anesthesia. The anesthesia data are relevant only because they allow a test of the ADDS prediction that changing levels of agency should have little or no effect on priming-related learning. The important point is that in all these studies, patients are unconscious and therefore, presumably have no agency, even when anesthesia levels were classified as light. As a result, overall this literature strongly supports the ADDS prediction.

The interesting and provocative reports that perceptual priming is intact during light and moderate anesthesia and during sleep provide promising preliminary support for the

ADDS prediction that agency should have little or no effect on learning due to perceptual priming. Even so, a more rigorous test must await experiments that examine the effects of stimulus repetition on learning when sense of agency is directly manipulated. Ideally, any such study would also include a suitable comparison task for which ADDS predicts that increasing levels of agency should improve learning (e.g., such as one of the tasks discussed earlier in this section).

Eyeblink conditioning

Eyeblink conditioning is a well-studied form of classical conditioning.[12] In a standard experiment, a conditioned stimulus (CS) is presented – typically an auditory or visual cue – and then a short time later an unconditioned stimulus (UCS) is presented – typically a puff of air delivered to the cornea. The UCS elicits a reflexive eyeblink, which is the unconditioned response (UR). After repeated pairings of the CS and UCS, the CS itself begins to elicit a learned eyeblink (the conditioned response; CR) – which occurs before the UCS is presented.

There are two widely used eyeblink conditioning tasks. In the standard version (also called delay-eyeblink conditioning), the CS and UCS presentations overlap in time and the CS typically remains on until the UCS ends. In trace-eyeblink conditioning, there is a short delay between the offset of the CS and the onset of the UCS. This distinction is important because there is good evidence that standard- and trace-eyeblink conditioning are mediated by different neural circuits and have different phenomenologies. Standard-eyeblink conditioning, which can occur without conscious awareness (Manns et al., 2002), is mediated primarily within the cerebellum and does not depend on hippocampus or on frontal cortex (e.g., Freeman & Steinmetz, 2011; Schmaltz & Theios, 1972). In contrast, trace-eyeblink conditioning depends on the hippocampus as well as the cerebellum (e.g., McGlinchey-Berroth et al., 1997) and does depend on conscious awareness of the relationship between the CS and the UCS (e.g., Manns et al., 2000).

The cerebellum does not receive a prominent DA projection and therefore ADDS makes the strong prediction that changes in agency should have little or no effect on standard-eyeblink conditioning. This prediction is strongly supported by studies showing that standard-eyeblink conditioning does not depend on conscious awareness (for a review, see Manns et al., 2002). For example, participants who show poor awareness of the relationship between the CS and UCS acquire and retain standard conditioned eyeblink as well as participants who show good awareness of this relationship (Clark et al., 2001; Manns et al., 2000). All theories of agency predict higher levels of agency in the presence of awareness than in its absence, and therefore these results suggest that, as predicted by ADDS, changes in agency have little or no effect on standard-eyeblink conditioning.

The story is quite different in the case of trace-eyeblink conditioning. Critically, in contrast to standard-eyeblink conditioning, trace-eyeblink conditioning depends on the hippocampus (McGlinchey-Berroth et al., 1997), which does receive a prominent DA projection. ADDS predicts that increases in agency should potentiate hippocampal function, because of the increased availability of DA, and therefore could facilitate trace-eyeblink conditioning. We know of no studies that have tested this prediction directly, but several studies have reported that the strength of trace-eyeblink conditioning increases with the participant's awareness of the CS-UCS relationship (Manns et al., 2000, 2002) – a result that is consistent with this ADDS prediction.

6.4 Automatic behaviors

After long periods of practice, almost any behavior can be executed quickly, reliably, and with little or no conscious deliberation. At this point, we say that the behavior has become automatic. A strong case can be made that most behaviors performed by adults are automatic. When we sit in a chair, pick up a cup of coffee, or identify an object as a square, our actions are almost always automatic. The evidence is good that automatic behaviors do not depend on executive function or on the neural networks that mediate procedural learning (Ashby & Crossley, 2012). For example, patients with Parkinson's disease have impaired executive function and procedural learning (e.g., Knowlton et al., 1996; McKinlay et al., 2010), but they are often able to emit an automatic motor response fluidly when presented with a familiar visual cue (e.g., kicking a ball, riding a bicycle), despite difficulties in initiating novel voluntary movements (e.g., Asmus et al., 2008).

Ashby et al. (2007) proposed that skills learned procedurally are mediated entirely within cortex after they become automatized, and that the development of automaticity is associated with a gradual transfer of control from the striatum to cortical-cortical projections from the relevant sensory areas directly to the premotor areas that initiate the behavior. Kovacs et al. (2021) proposed a similar account of how rule-guided behaviors are automatized in which the prefrontal cortex trains the cortical circuits that implement the automatic behaviors. Therefore, according to these accounts, a critical function of the neural networks that mediate executive function and procedural learning are to train purely cortical representations of automatic behaviors. Both of these theories assume that automatic behaviors are acquired when synapses are strengthened between direct cortical-cortical projections from the relevant areas of sensory cortex onto targets in premotor and motor cortex. So by these accounts, prefrontal cortex and basal ganglia have little or no role in

[12] For example, a Google Scholar search of "eyeblink conditioning" returns 12,000 results.

initiating automatic behaviors, and both theories predict that automatic behaviors are almost entirely feedback independent. Visual cortex receives almost no DA input. The VTA does send a prominent projection to premotor cortex, however, so ADDS does predict that a strong sense of agency should facilitate the development of automaticity. Even so, it also predicts that a sense of agency should have little or no effect on the production of behaviors that are already automatized.

A classic hallmark of automatic behaviors is that they can occur without conscious awareness (Shiffrin & Schneider, 1977). A key feature that is often included in definitions of agency is intentionality (e.g., Bandura, 2006), which presumably requires conscious awareness. If so, then by definition, the production of automatic behaviors is unrelated to sense of agency. For example, Wu (2013) argued that "automaticity appears to eliminate agency" (p. 245). On the other hand, to our knowledge, the prediction that agency should facilitate the development of automaticity has not been tested directly. Even so, a famous claim is that true expertise in any complex skill requires 10,000 hours of *deliberate* practice, in which deliberate practice is assumed to include "the subjects' motivation to attend to the task and exert effort to improve their performance" (p.367, Ericsson et al., 1993). In other words, Ericsson et al. (1993) assumed that deliberate practice requires a high sense of agency, and therefore they cite considerable evidence that agency facilitates the development of expertise. On the other hand, expertise is not the same as automaticity. Expertise implies a high level of skill, but automatic behaviors are not necessarily skillful, and may even be maladaptive (e.g., as in many addictions). Furthermore, not all expert behaviors are automatic. Therefore, the data on deliberate practice and expertise provides promising initial support for ADDS, but much more research is needed to test the theory's predictions rigorously.

7. Positive Mood

Twenty-five years ago, Ashby et al. (1999) proposed that events that induce mild positive mood elevate cortical DA levels for a period of 20 – 30 minutes, and that this DA increase facilitates executive functions such as creative problem solving and working memory. Today this proposal is widely accepted. For example, it plays a central role in the popular broaden-and-build theory of positive emotions (Fredrickson, 2001), which asserts that positive emotions broaden thought-action repertoires, which builds resources. The initial step of feeling a positive emotion causes a broadened state that promotes learning, creativity, and engaging with the environment. The building process exploits the broadened state to develop new knowledge, skills, or relationships. The results increase the probability of more positive emotions, thus fueling a reoccurrence of the broaden-and-build process (i.e., an upward spiral).

ADDS predicts that the effects of positive mood on executive function should be amplified by increases in agency. Events that elevate mood frequently include an unexpected reward and therefore generate a positive RPE. For example, a mild elevation in mood might occur after receiving an unexpected gift, being the target of a kind act by a stranger, or unexpectedly running into an old friend. In fact, the evidence is good that the same reward improves mood more when it is unexpected than when expected (Mellers et al., 1997; Rutledge et al., 2014; Shepperd & McNulty, 2002). Furthermore, these effects are not limited to laboratory studies, but are also prominent in everyday life (Otto & Eichstaedt, 2018). When rewards that elevate mood are unexpected, they generate a positive RPE, and as we have seen, an enormous literature predicts that this causes the tonically active DA neurons to fire phasic bursts (via the PPTN pathway in Figure 1). As a result, DA levels will rise in all VTA (and SNpc) targets. This includes the nucleus accumbens (and more generally, the ventral striatum) and all of frontal cortex.[13]

As mentioned earlier, in much of the basal ganglia, sudden DA elevations are quickly cleared from synapses via DAT. However, there are negligible concentrations of DAT in cortex (e.g., Varrone & Halldin, 2014), and DA elevations may take 20 – 30 minutes to return to baseline levels. For this reason, Ashby et al. (1999) predicted that the improvements in executive function caused by elevated mood should also persist for 20 – 30 minutes.

ADDS predicts that increases in agency should amplify these effects. In other words, the theory predicts that the greater the agency experienced during the events that elevate mood, the greater the improvements in executive function caused by those mood elevations – at least, up until the DA level associated with optimal performance is reached (Akbari Chermahini & Hommel, 2012).

The neural network illustrated in Figure 1 is proposed to mediate changes in DA levels as agency changes, but there is overwhelming evidence that DA does not mediate the pleasant feelings associated with positive mood. For example, Berridge has argued that reward is associated with functionally separate motivational and feeling components (which he calls "wanting" and "liking"; Berridge, 1996; Berridge et al.,

[13] ADDS also predicts that increases in agency should amplify the amount by which DA levels drop below baseline following a failure to receive an expected reward (i.e., when the RPE is negative). To the extent that such a failure will lead to disappointment and negative mood, ADDS would therefore seem to predict that higher levels of agency might increase this disappointment. Although this seems plausible, deriving testable predictions from ADDS about negative moods is well beyond the scope of this article because the evidence is strong that positive and negative affect are not simple opposites, but instead are mediated by separate neural networks (e.g., Ashby et al., 1999; George et al., 1995). Therefore, deriving predictions from ADDS about negative mood requires marrying ADDS to some separate theory that describes the neural networks that mediate negative mood.

2009). He proposes that DA mediates the motivational component of reward and that the pleasant feelings that result from unexpected rewards are mediated by endogenous opioids. ADDS predicts that events that elevate mood will increase cortical DA levels for a period of 20 – 30 minutes, and that increasing levels of agency will amplify these effects. But the theory does not predict that the pleasant feelings must necessarily persist for this long. Once a phasic burst from VTA DA neurons causes prefrontal DA levels to rise, there is no mechanism to decrease these levels quickly. Specifically, the protein that quickly removes DA from synapses in the basal ganglia (i.e., DAT) is almost absent in prefrontal cortex (e.g., Varrone & Halldin, 2014). Instead, free DA in prefrontal cortex is removed from synapses either by diffusion or by slow degradation via the enzyme COMT. As a result, ADDS predicts that the cognitive effects of positive mood induction will persist for 20 – 30 minutes, even if the pleasant feelings do not. For example, suppose some unexpected reward is obtained that elevates positive mood. This should cause prefrontal DA levels to rise. Now suppose that just a few minutes later, some new event – for example, the failure to receive an expected reward – causes the positive mood to be lost. Even though the mood has changed, the DA elevations caused by the earlier events that induced the positive mood should remain in prefrontal cortex for 20 – 30 minutes. Therefore, for example, ADDS predicts that the broadening component of the broaden-and-build theory could persist longer than the subjective experience of the positive mood that triggered the broadening process (Fredrickson, 2004; Fredrickson & Joiner, 2002).

The hedonic component of positive mood is thought to be mediated by a widespread, but related network that includes medial prefrontal cortex, orbitofrontal cortex, anterior cingulate, insula, the amgydala, and the nucleus accumbens (e.g., Alexander et al., 2021). A glance back at Figure 1 reveals considerable overlap of these two networks. The critical region of convergence could be the nucleus accumbens since this region of the ventral striatum is closely associated with reward and pleasure. For example, rats regularly self-stimulate when an electrode is placed in the accumbens (e.g., Wise, 1996) and human patients with deep brain stimulation (DBS) electrodes implanted in the accumbens report mood improvements with stimulation that increases with voltage (Giordana et al., 2015; Haq et al., 2011).

DBS has similar behavioral benefits as lesion therapy. For example, for many years it was common to treat the bradykinesia that frequently occurs with Parkinson's disease by pallidotomy – a surgical procedure in which part of the globus pallidus is removed. More recently however, pallidotomy is being replaced by DBS of the subthalamic nucleus, which results in similar behavioral improvements but has the advantage of not requiring any lesions. The subthalamic nucleus excites the globus pallidus, so the fact that DBS of the subthalamic nucleus causes similar reductions in bradykinesia as pallidotomy suggests that DBS reduces subthalamic nucleus excitation of the globus pallidus (and thereby reduces the pallidal inhibition of motor thalamus). Although the exact physiological effects of DBS are still being investigated, it is becoming increasingly clear that DBS prevents the implanted brain region from responding in its usual way (e.g., Chiken & Nambu, 2016). So the fact that DBS of the nucleus accumbens elevates mood suggests that mood improvements should be associated with *reduced* accumbens activity. In fact, a prominent proposal is that reward reduces accumbens activity, whereas aversive stimuli have the opposite effect (Carlezon Jr & Thomas, 2009).

A likely mediator of these effects is medial prefrontal cortex, which sends a prominent excitatory projection to the nucleus accumbens. In particular, mood improvements are associated with a reduction of this excitatory input. For example, rats will self-stimulate to reduce prefrontal excitation of the accumbens (by self-administering an NMDA antagonist into prefrontal cortex; Carlezon Jr & Wise, 1996). Similarly, animals will self-stimulate with an electrode implanted in this region (e.g., Tzschentke, 2000).

Figure 3 describes a popular model of these effects that was proposed by Grace et al. (2007). The idea is that both ventral subiculum and medial prefrontal cortex send excitatory projections to nucleus accumbens, but the postsynaptic effects of the ventral subiculum inputs are mediated by the response of DA D1 receptors, whereas the prefrontal input is mediated by the response of DA D2 receptors. D1 receptor activation potentiates the glutamate response through NMDA receptors, whereas D2 activation depresses the glutamate response through non-NMDA receptors (e.g., Surmeier et al., 2007). This model therefore predicts that activation of nucleus accumbens D2 receptors attenuates excitatory input from medial prefrontal cortex (Goto & Grace, 2005; O'Donnell & Grace, 1994). In contrast, D1 receptor activation enhances the response of accumbens neurons to inputs from ventral subiculum, without affecting the response to inputs from medial prefrontal cortex (Goto & Grace, 2005; West & Grace, 2002).

Consider now the predictions for the relationship between agency and mood that result from combining the networks described in Figures 1 and 3. First, consider a scenario in which an unexpected reward occurs when agency is high. The Figure 1 model predicts that this event will increase the number of tonically active VTA DA neurons and therefore raise DA levels in all VTA target sites – including the nucleus accumbens. The Figure 3 model then predicts that the resulting elevated DA levels in the accumbens will depress the inputs from medial prefrontal cortex, thereby elevating mood. Next, suppose an unexpected reward occurs when agency is low. The Figure 1 model predicts that the lack of agency will reduce the number of tonically active DA neurons in VTA

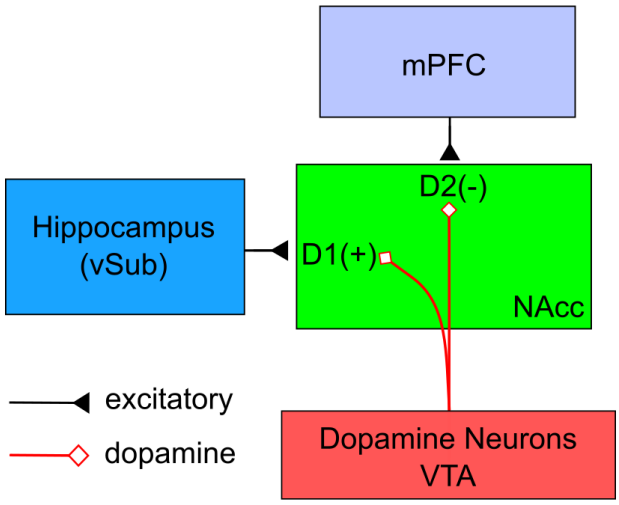

Figure 3

The Grace, Floresco, Goto, and Lodge (2007) model that is adapted here to predict how agency affects mood. mPFC = medial prefrontal cortex; vSub = ventral subiculum; NAcc = nucleus accumbens; D2 = dopamine D2 receptors; D1 = dopamine D1 receptors; VTA = ventral tegmental area.

and therefore lower tonic DA levels in all VTA targets. A reduction of DA in the accumbens will potentiate the glumatergic input from medial prefrontal cortex and thereby reduce the mood effects of the unexpected reward. Although mood should be elevated in this scenario regardless of agency, the model that results from combining the networks described in Figures 1 and 3 predicts that the amount that mood is elevated should increase with agency – specifically, agency will not only increase the DA response to unexpected rewards (via the Figure 1 network), but also increase the effects of that reward on positive mood (via the Figure 3 network).

To our knowledge, this prediction has not been rigorously tested. Even so, some preliminary results are encouraging. First, Fritz et al. (2013) reported that listening to music during exercise enhanced mood more when the music was generated by the exercise movements than when it was presented passively. Second, Jallais and Gilet (2010) compared the efficacy of two methods for inducing positive mood. In one, participants were asked to recall a happy memory and to write down as many details of this event as possible. In the other, participants first passively listened to 4 minutes of happy music (Delibes's Coppélia and Bach's Brandenburg Concerto No. 2) and then they were asked to imagine as vividly as possible a happy scenario described to them in text. Note that the sense of agency should be considerably higher in the former method than in the latter. In the autobiographical-memory method, the positive mood was induced by recalling a real event from the participants' lives, and this memory was also used to elicit behavior (i.e., describing the memory in writing). In contrast, in the music-imagery method, no behavior was ever elicited and the happy scenario described in text might have never occurred at any time in the participants' lives. As a result, ADDS predicts that, all else being equal, the former method should be more efficacious at improving mood than the latter. In support of this prediction, the autobiographical-memory method improved self-reported ratings of happiness more than the music-imagery method.

8. Pain

Events that elevate mood cause DA release, but so do aversive and stressful events. For example, many studies have reported that nociceptive stimuli, such as a shock or pinch of a rat's tail, cause an increase in DA release in a variety of brain regions, including, for example, the nucleus accumbens and medial prefrontal cortex (e.g., Abercrombie et al., 1989; Bassareo et al., 2002; Budygin et al., 2012). In fact, the evidence is now good that there are separate populations of DA neurons in the ventral tegmental area – one type that responds to unexpected rewards and one type that responds to unexpected nociceptive stimuli (e.g., Brischoux et al., 2009; Bromberg-Martin et al., 2010; de Jong et al., 2019). Furthermore, these populations have distinct targets in the basal ganglia and cortex. For example, DA neurons that respond to nociceptive stimuli project to the shell of the nucleus accumbens, but not the core, whereas DA neurons that respond to rewards project to the core, but not the shell (Badrinarayan et al., 2012; de Jong et al., 2019).

For our purposes, the most relevant of these anatomical differences is that the DA neurons tuned to nociceptive events project heavily to medial prefrontal cortex (Abercrombie et al., 1989; Bassareo et al., 2002) – a brain region that has been characterized as a "central hub" for pain processing (Kummer et al., 2020). Although a widespread collection of brain regions are activated during pain perception (e.g., Apkarian et al., 2005), the medial prefrontal cortex is especially critical for our affective and cognitive responses to acute pain (Bushnell et al., 2013; Kummer et al., 2020).

DA plays a key role in pain perception. In particular, DA acts as an analgesic, in the sense that, for the same nociceptive stimulus, the higher the brain DA levels, the less intense the pain (Huang et al., 2020; Potvin et al., 2009; Tiemann et al., 2014; Wood, 2006; Wood et al., 2007). Therefore, note that ADDS makes a strong prediction about the effects of agency on pain – the higher the sense of agency when the nociceptive stimulation occurs, the lower the level of pain that should be experienced. High levels of agency mean many tonically active DA neurons, which means there are more DA neurons available to respond to the nociceptive stimulation, which means more DA in cortical regions that mediate pain (e.g., such as the medial prefrontal cortex), which means less pain. In other words, ADDS predicts that a self-inflicted nociceptive stimulus should be less painful than the same stim-

ulus delivered by someone or something else.

In support of ADDS, a number of studies have reported that self-administering a nociceptive stimulus elicits lower levels of judged pain than when the same stimulus is externally administered (Karsh et al., 2018; M. J. Müller, 2012; Wang et al., 2011). Despite these positive results, a few studies have reported no difference in judged pain intensity of self-administered versus externally-administered nociceptive stimulation (Karsh et al., 2018; Mohr et al., 2005). Even so, it is important to view these null results through the lens of some recently discovered complicating factors.

Most critically, recent research suggests that it is essential to distinguish between the sensation of pain and the emotional response to that sensation. Pain has both a sensory-discriminative component, which is similar to other senses, and an affective-motivational component (Auvray et al., 2010; Treede et al., 1999). Many studies fail to distinguish between the two, but some evidence suggests that the primary effect of DA is on the affective-motivational component of pain. For example, Tiemann et al. (2014) reported that temporary DA depletion caused human participants to rate nocioceptive stimuli as more unpleasant, but not as more intense, than when DA was at control levels.[14] Therefore, studies that do not unambiguously focus on unpleasantness could miss a significant effect of agency on pain. For example, among the studies that reported that manipulations of agency did not affect pain, Mohr et al. (2005) asked participants to rate pain intensity, rather than any affective response to the nociceptive stimulation. Furthermore, this was an fMRI study and the authors found many brain regions that responded differently in the high- versus low-agency conditions, which suggests that agency did affect pain processing. As another example, although Karsh et al. (2018) reported lower pain levels when agency was high in three different experiments, this effect was significant in only two of their experiments. In all three experiments, participants were asked "To what degree did you feel that the stimulation was painful?", which could be interpreted by some participants as a question about sensory magnitude, rather than a question about the participant's hedonic response to the stimulus. In summary, although more research is needed, the available evidence is generally supportive of the ADDS prediction that increasing levels of agency reduce pain.

9. Discussion

Agency is the sense that one has control over one's own actions and the consequences of those actions. Despite the critical role that agency plays in the human condition, its neural basis is only beginning to emerge. Several previous studies have identified brain regions that become more active when agency is increased (e.g., Crivelli & Balconi, 2017; Haggard, 2017; Sperduti et al., 2011), and several others have linked agency to brain DA levels (e.g., Aarts et al., 2012; Hassall et al., 2019; Render & Jansen, 2019). This article extends this empirical work by proposing a neuropsychological theory of agency that makes many novel neural and behavioral predictions.

One reason for the sparse literature on the neural basis of agency is that there are virtually no nonhuman animal studies with the stated goal of studying agency. This has severely limited progress because much of what we know about the human brain comes from studies with nonhumans. Our approach to overcoming this limitation was to appeal to the large neuroscience literature on feedback contingency. When feedback is noncontingent on behavior, then there is no behavior that can increase or decrease the reward rate, and as a result, agency should be low. When rewards are contingent on behavior, then the agent controls the reward rate via their behavior, and therefore agency should be high. As a result, we believe that studying the neural networks that are sensitive to feedback contingency can shed invaluable light on the neural basis of agency.

A review of the large neuroscience literature on feedback contingency motivated the ADDS theory proposed here, and specifically, the neural network described in Figure 1. This model proposes that a cortical network, which includes regions of the anterior cingulate and prefrontal cortex, computes contingency in real time and that the output of this network is in the hippocampal vSub. Increases in agency, and hence the output of this network, excite the nucleus accumbens, which leads to the disinhibition of DA neurons in the VTA. This disinhibition increases the number of tonically active DA neurons, causing tonic levels of DA in all VTA targets to rise and effectively increasing the gain on the DA response to any given RPE.

ADDS accurately predicts a variety of relevant neuroscience results, including the differential effects on the number of tonically active DA neurons in the VTA that result from stimulating the ventral subiculum, the pedunculopontine tegmental nucleus, or both. These results support the validity of the specific neural network illustrated in Figure 1, which was used to derive all of the behavioral predictions described in this article.

ADDS also makes many novel and testable behavioral predictions, including that increases in agency will 1) increase motivation, 2) improve executive function, 3) facilitate procedural learning, but only in the presence of immediate trial-by-trial feedback, 4) have little or no effect on learning-related effects due to perceptual priming or on the acquisition or expression of standard-eyeblink conditioning, 5) facilitate the development of automatic behaviors, but have little or no effect on the production of behaviors that are already automatized, 6) amplify the cognitive benefits of positive mood, and 7) reduce pain. In almost all cases,

[14] DA was depleted in this study by selectively restricting dietary intake of precursor amino acids that are needed for DA synthesis.

there is preliminary support for these predictions. Even so, the support did not generally come from studies specifically designed to test these predictions. As a result, much more research is needed to test ADDS.

Despite these strong predictions, it is important to note that individual differences should be expected in the magnitude of these effects, and for some predictions these differences could be significant. In particular, there is considerable evidence that certain major personality traits, such as extroversion, are associated with specific individual differences in the functioning of brain DA systems (e.g., Pickering, 2004; Smillie et al., 2010). For example, there is evidence that extroverts exhibit a larger DA response than introverts to the same RPE (Cooper et al., 2014; Pickering & Pesola, 2014; Smillie et al., 2011, 2019). As a result, the magnitude of the predicted behavioral effects of changes in agency could be different in extroverts and introverts.

9.1 Implications for psychological theories of agency

Although no other theories describe the neural networks that mediate agency and its effects on behavior, a number of purely psychological and highly influential theories assign prominent roles to agency. Included in this list are: 1) self-efficacy theory (Bandura, 1977), 2) hope theory (Snyder, 1989), and 3) goal-focused positive psychotherapy (GFPP; Conoley & Scheel, 2017).[15] All of these theories were developed as accounts of real-world behaviors – including social behaviors – that are far more complex than the simple, mostly laboratory, studies we have considered so far. Complex, real-world human behaviors almost certainly recruit more elaborate neural, cognitive, and affective processes than the simpler studies that were used to derive ADDS. For example, Bandura's self-efficacy theory considers agency to include intentionality, forethought, self-reactiveness, and self-reflectiveness (Bandura, 2006), which is a far more elaborate view of agency than we have so far considered.

ADDS is not inconsistent with any of these psychological theories, and therefore should not be viewed as an alternative. ADDS overlaps with these theories, in the sense that there are many scenarios in which ADDS makes the same predictions as the existing psychological theories – for example, that agency increases motivation. Whereas the psychological theories provide a much more elaborate description of the cognitive and affective consequences that result from increasing or decreasing agency, ADDS provides a much more detailed picture of the neural consequences. As a result, we believe that rather than replacing any existing psychological theory, ADDS should be considered as a supplement to those theories. Marrying ADDS to a purely psychological theory has a number of potential benefits. First, it can sharpen predictions of the psychological theory – for example, by allowing it to make subtly different predictions for different kinds of behaviors. Second, by appealing to underlying neural mechanisms, it can strengthen predictions of the psychological theory by providing more rigorous justification. Third, it might allow the psychological theory to make novel predictions. And fourth, it can link two large and historically disparate literatures – namely, the psychological literature on agency and the neuroscience literature on dopamine, reward, and feedback contingency.

The remainder of this section considers in more detail the implications of marrying ADDS to self-efficacy theory, hope theory, goal-focused positive psychotherapy, and the broaden-and-build theory of positive emotions.

Self-efficacy theory

Bandura (1977) proposed that a fundamental attribute of psychological health is self-efficacy, which he defined as "beliefs in one's capabilities to organize and execute the courses of action required to produce given levels of attainments" (p. 16, Bandura, 2000). According to this account, self-efficacy is closely related to agency, which Bandura (2006) defined as the ability "to influence intentionally one's functioning and life circumstances" (p. 164). So according to self-efficacy theory, self-efficacy is a belief, whereas agency is an ability. Furthermore, Bandura (1977) is clear that self-efficacy is task specific and is not a generalized construct or trait. Therefore, virtually all of the conditions considered earlier in this article that were hypothesized to increase agency would also likely increase self-efficacy as defined by Bandura (1977). For example, a meta-analysis examining a variety of predictors of self-efficacy reported that the most efficacious was the direct experience of accomplishing a goal (Byars-Winston et al., 2017).

Self-efficacy theory is highly influential. According to Google Scholar, the 1977 article proposing the theory has been cited more than 108,000 times. Self-efficacy is a cornerstone of Bandura's more general social cognitive theory (Bandura, 1986), and research on self-efficacy has spread to a wide variety of topics, including choice of career (Choi et al., 2012; Rottinghaus et al., 2003), educational success (Pajares, 1996; Robbins et al., 2004), health-related behaviors (Sheeran et al., 2016), job burn-out (Shoji et al., 2016), and addiction issues (Gwaltney et al., 2009).

Bandura (2012) identified four ways to boost self-efficacy: 1) by increasing mastery (i.e., performance accomplish-

[15] Agency plays a key role in many other psychological theories, but often more indirectly. For example, according to self-determination theory (Deci & Ryan, 2000, 2002), competence is one of three basic psychological needs that develops as one acquires mastery of a task or skill. Agency surely affects competence, but competence also likely depends on other factors. Space limitations prevent us from discussing the many theories, including self-determination theory, in which agency plays a key, but indirect role. Even so, it is important to realize that many of the implications highlighted in this section that ADDS has for these three psychological theories are also relevant for the many other theories in which agency plays a more indirect role.

ments), 2) by making choices, 3) via social modeling (e.g., vicarious experience), and 4) via social (e.g., verbal) persuasion. Of these, ADDS is most relevant to the former two – that is, mastery and choice. For example, all of the predictions we derived about the effects of agency on learning and executive function could be interpreted as predictions about mastery and choice.

Hope theory

Hope theory assumes that hope is created when 1) a desired goal is identified, 2) agency is high, and 3) one or more pathways to achieving the desired goal are envisioned (Snyder, 1989). The repeated successful attainment of a goal leads to agentic beliefs such as "I made this happen," accompanied by happiness that creates a more welcoming attitude toward goals (Snyder, 2002). A large literature demonstrates that hope predicts mental well-being (Bailey et al., 2007; Carver & Scheier, 2002; Magaletta & Oliver, 1999; Snyder, 2002). Snyder's measure of agency in hope moderately correlates with measures of self-efficacy, optimism, and the ability to identify pathways (Bryant & Cvengros, 2004; Magaletta & Oliver, 1999). Furthermore, Gallagher and Lopez (2009) found that hope significantly correlated with the concepts of subjective well-being (Diener, 1994), psychological well-being (Ryff, 1989), and social well-being (Keyes, 1998), as well as each of the concepts' factors.

Hope, and by extension agency, also has been celebrated as the central purpose of psychotherapy (Frank & Frank, 1993) and a universal goal in all psychotherapies (Yalom & Leszcz, 2020). Hope therapy teaches the skills of developing agency and pathway thinking (Cheavens & Guter, 2018; Lopez et al., 2000). Agency is increased primarily via self-talk, as used for example, in cognitive-behavioral therapies. Hope therapists encourage clients to increase agency via statements such as "I believe I can do this" and "I know the best way to accomplish this goal," (p. 135, Cheavens & Guter, 2018).

A key contribution of hope theory is to emphasize the importance of finding pathways to achieve the desired goal (Snyder, 1989, 2002; Snyder et al., 1991). Hope is created when agency is high and one or more pathways are identified. Identifying a pathway to a goal requires creative problem solving – a generalized skill that depends on executive processes such as exploration and cognitive flexibility. Exploration is needed to generate novel candidate pathways and cognitive flexibility is required to switch to a different pathway if the current pathway proves unsuccessful. These are executive functions that ADDS predicts are enhanced by agency and by positive mood. Therefore, ADDS predicts that the core tenets of hope theory – namely, agency and finding a pathway to a desired goal – are interdependent. Specifically, ADDS predicts that increases in agency and/or positive mood should facilitate the search for pathways and therefore increase hope.

Goal-focused positive psychotherapy, and the broaden-and-build theory of positive emotion

Goal-focused positive psychotherapy (GFPP; Conoley & Scheel, 2017) is a theory of psychotherapy rooted in the broaden-and-build theory of positive emotions (Fredrickson, 2001). GFPP highlights the beneficial role of positive emotions in psychotherapy and their contributions to psychotherapy outcome. Using a strengths-oriented approach to elicit positive emotions, GFPP therapists seek to increase a client's sense of hope and subjective well-being in order to address problematic issues from a position of increased confidence or agency.

GFPP therapists increase client agency by affirming a client's strengths and underscoring the association between the client's actions and the production of positive outcomes (e.g., a client takes a healthy risk and experiences positive emotion and an increased sense of agency as a result). This approach is consistent with reports that inducing positive affect increases the belief that positive events are more likely to occur (Isen et al., 1988; Johnson & Tversky, 1983). One way that a GFPP therapist might increase client agency is through capitalization – that is, by celebrating client strengths (e.g., a healthy desire, behavior, or thought; Gable et al., 2018). The celebration increases agency by attributing ownership of the strength to the client and by the explicit labeling of the strength as important by an expert (i.e., the therapist).

ADDS predicts that unexpectedly praising a client and affirming their strengths will increase tonic DA levels in prefrontal cortex for a period of about 20 minutes, and that this effect will be more pronounced with higher levels of agency. Furthermore, ADDS predicts that this increased cortical DA will improve all forms of executive function. As a result, ADDS predicts that nearly optimal benefits should result if the therapist improves mood and increases agency about every 15 minutes or so. Specifically, a therapist can expect that such an intervention should provide the client with around 20-30 minutes of greater cognitive flexibility, which the therapist can then use to promote psychotherapeutic change.

9.2 Conclusions

Sense of agency is fundamental to the human experience. It increases motivation, improves many forms of learning, and is vital to well-being. Not surprisingly, increased agency is a universal goal of psychotherapy. One significant barrier to achieving this goal has been a poor understanding of the neural basis of agency and of the neural changes that occur when agency is increased or decreased. We hope that ADDS can reduce this barrier by sharpening the predictions of psychological theories of agency, and by making novel predictions that can be used to motivate new research. In fact, the

greatest contribution of ADDS might be the many new avenues for research that it suggests.

References

Aarts, H., Bijleveld, E., Custers, R., Dogge, M., Deelder, M., Schutter, D., & van Haren, N. E. (2012). Positive priming and intentional binding: Eye-blink rate predicts reward information effects on the sense of agency. *Social Neuroscience*, *7*(1), 105–112.

Abercrombie, E. D., Keefe, K. A., DiFrischia, D. S., & Zigmond, M. J. (1989). Differential effect of stress on in vivo dopamine release in striatum, nucleus accumbens, and medial frontal cortex. *Journal of Neurochemistry*, *52*(5), 1655–1658.

Akbari Chermahini, S., & Hommel, B. (2012). More creative through positive mood? Not everyone! *Frontiers in Human Neuroscience*, *6*, 319.

Alexander, R., Aragón, O. R., Bookwala, J., Cherbuin, N., Gatt, J. M., Kahrilas, I. J., Kästner, N., Lawrence, A., Lowe, L., Morrison, R. G., et al. (2021). The neuroscience of positive emotions and affect: Implications for cultivating happiness and wellbeing. *Neuroscience & Biobehavioral Reviews*, *121*, 220–249.

Alloy, L. B., & Abramson, L. Y. (1979). Judgment of contingency in depressed and nondepressed students: Sadder but wiser? *Journal of Experimental Psychology: General*, *108*(4), 441–485.

Aminoff, E. M., Kveraga, K., & Bar, M. (2013). The role of the parahippocampal cortex in cognition. *Trends in Cognitive Sciences*, *17*(8), 379–390.

Apkarian, A. V., Bushnell, M. C., Treede, R.-D., & Zubieta, J.-K. (2005). Human brain mechanisms of pain perception and regulation in health and disease. *European Journal of Pain*, *9*(4), 463–484.

Apud, J. A., Mattay, V., Chen, J., Kolachana, B. S., Callicott, J. H., Rasetti, R., Alce, G., Iudicello, J. E., Akbar, N., Egan, M. F., et al. (2007). Tolcapone improves cognition and cortical information processing in normal human subjects. *Neuropsychopharmacology*, *32*(5), 1011–1020.

Ashby, F. G., & Maddox, W. T. (2005). Human category learning. *Annual Review of Psychology*, *56*, 149–178.

Ashby, F. G., & Casale, M. B. (2003). A model of dopamine modulated cortical activation. *Neural Networks*, *16*(7), 973–984.

Ashby, F. G., & Crossley, M. J. (2011). A computational model of how cholinergic interneurons protect striatal-dependent learning. *Journal of Cognitive Neuroscience*, *23*(6), 1549–1566.

Ashby, F. G., & Crossley, M. J. (2012). Automaticity and multiple memory systems. *Wiley Interdisciplinary Reviews: Cognitive Science*, *3*(3), 363–376.

Ashby, F. G., Ennis, J. M., & Spiering, B. J. (2007). A neurobiological theory of automaticity in perceptual categorization. *Psychological Review*, *114*(3), 632–656.

Ashby, F. G., Isen, A. M., & Turken, A. (1999). A neuropsychological theory of positive affect and its influence on cognition. *Psychological Review*, *106*(3), 529–550.

Ashby, F. G., & Valentin, V. V. (2017). Multiple systems of perceptual category learning: Theory and cognitive tests. In H. Cohen & C. Lefebvre (Eds.), *Handbook of categorization in cognitive science, Second edition* (pp. 157–188). Elsevier.

Ashby, F. G., Valentin, V. V., & von Meer, S. S. (2015). Differential effects of dopamine-directed treatments on cognition. *Neuropsychiatric Disease and Treatment*, *11*, 1859–1875.

Ashby, F. G., & Vucovich, L. E. (2016). The role of feedback contingency in perceptual category learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *42*(11), 1731–1746.

Ashby, F. G., & Waldron, E. M. (1999). On the nature of implicit categorization. *Psychonomic Bulletin & Review*, *6*(3), 363–378.

Asmus, F., Huber, H., Gasser, T., & Schöls, L. (2008). Kick and rush: Paradoxical kinesia in Parkinson disease. *Neurology*, *71*, 695.

Auvray, M., Myin, E., & Spence, C. (2010). The sensory-discriminative and affective-motivational aspects of pain. *Neuroscience & Biobehavioral Reviews*, *34*(2), 214–223.

Badrinarayan, A., Wescott, S. A., Vander Weele, C. M., Saunders, B. T., Couturier, B. E., Maren, S., & Aragona, B. J. (2012). Aversive stimuli differentially modulate real-time dopamine transmission dynamics within the nucleus accumbens core and shell. *Journal of Neuroscience*, *32*(45), 15779–15790.

Bailey, T. C., Eng, W., Frisch, M. B., & Snyder, C. (2007). Hope and optimism as related to life satisfaction. *The Journal of Positive Psychology*, *2*(3), 168–175.

Balleine, B. W., & Dickinson, A. (1998). Goal-directed instrumental action: Contingency and incentive learning and their cortical substrates. *Neuropharmacology*, *37*(4), 407–419.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, *84*(2), 191–215.

Bandura, A. (1986). The explanatory and predictive scope of self-efficacy theory. *Journal of Social and Clinical Psychology*, *4*(3), 359–373.

Bandura, A. (2000). Self-efficacy: The foundation of agency. In W. J. Perrig & A. Grob (Eds.), *Control of human behavior, mental processes, and consciousness: Essays in honor of the 60th birthday of August Flammer* (pp. 16–31). Lawrence Erlbaum Associates, Inc.

Bandura, A. (2006). Toward a psychology of human agency. *Perspectives on Psychological Science*, *1*(2), 164–180.

Bandura, A. (2012). On the functional properties of perceived self-efficacy revisited. *Journal of Management*, *38*(1), 9–44.

Bassareo, V., De Luca, M. A., & Di Chiara, G. (2002). Differential expression of motivational stimulus properties by dopamine in nucleus accumbens shell versus core and prefrontal cortex. *Journal of Neuroscience*, *22*(11), 4709–4719.

Bayer, H. M., & Glimcher, P. W. (2005). Midbrain dopamine neurons encode a quantitative reward prediction error signal. *Neuron*, *47*(1), 129–141.

Bejjani, G., Lequeux, P.-Y., Schmartz, D., Engelman, E., & Barvais, L. (2009). No evidence of memory processing during propofol-remifentanil target-controlled infusion anesthesia with bispectral index monitoring in cardiac surgery. *Journal of Cardiothoracic and Vascular Anesthesia*, *23*(2), 175–181.

Berridge, K. C. (1996). Food reward: Brain substrates of wanting and liking. *Neuroscience & Biobehavioral Reviews*, *20*(1), 1–25.

Berridge, K. C., Robinson, T. E., & Aldridge, J. W. (2009). Dissecting components of reward: "liking","wanting", and learning. *Current Opinion in Pharmacology*, *9*(1), 65–73.

Blanchard, T. C., Strait, C. E., & Hayden, B. Y. (2015). Ramping ensemble activity in dorsal anterior cingulate neurons during persistent commitment to a decision. *Journal of Neurophysiology*, *114*(4), 2439–2449.

Boakes, R. A. (1973). Response decrements produced by extinction and by response-independent reinforcement. *Journal of the Experimental Analysis of Behavior*, *19*(2), 293–302.

Brischoux, F., Chakraborty, S., Brierley, D. I., & Ungless, M. A. (2009). Phasic excitation of dopamine neurons in ventral VTA by noxious stimuli. *Proceedings of the National Academy of Sciences*, *106*(12), 4894–4899.

Bromberg-Martin, E. S., Matsumoto, M., & Hikosaka, O. (2010). Dopamine in motivational control: Rewarding, aversive, and alerting. *Neuron*, *68*(5), 815–834.

Brown, J., Bullock, D., & Grossberg, S. (1999). How the basal ganglia use parallel excitatory and inhibitory learning pathways to selectively respond to unexpected rewarding cues. *Journal of Neuroscience*, *19*(23), 10502–10511.

Bryant, F. B., & Cvengros, J. A. (2004). Distinguishing hope and optimism: Two sides of a coin, or two separate coins? *Journal of Social and Clinical Psychology*, *23*(2), 273–302.

Budygin, E. A., Park, J., Bass, C. E., Grinevich, V. P., Bonin, K. D., & Wightman, R. M. (2012). Aversive stimulus differentially triggers subsecond dopamine release in reward regions. *Neuroscience*, *201*, 331–337.

Bushnell, M. C., Čeko, M., & Low, L. A. (2013). Cognitive and emotional control of pain and its disruption in chronic pain. *Nature Reviews Neuroscience*, *14*(7), 502–511.

Byars-Winston, A., Diestelmann, J., Savoy, J. N., & Hoyt, W. T. (2017). Unique effects and moderators of effects of sources on self-efficacy: A model-based meta-analysis. *Journal of Counseling Psychology*, *64*(6), 645–658.

Calabresi, P., Pisani, A., Mercuri, N. B., & Bernardi, G. (1996). The corticostriatal projection: From synaptic plasticity to dysfunctions of the basal ganglia. *Trends in Neurosciences*, *19*(1), 19–24.

Capaldi, E. J. (1967). A sequential hypothesis of instrumental learning. In K. W. Spence & J. T. Spence (Eds.), *Psychology of learning and motivation* (pp. 67–156, Vol. 1). Elsevier.

Carelli, R. M., Wolske, M., & West, M. O. (1997). Loss of lever press-related firing of rat striatal forelimb neurons after repeated sessions in a lever pressing task. *Journal of Neuroscience*, *17*(5), 1804–1814.

Carlezon Jr, W. A., & Thomas, M. J. (2009). Biological substrates of reward and aversion: A nucleus accumbens activity hypothesis. *Neuropharmacology*, *56*, 122–132.

Carlezon Jr, W. A., & Wise, R. A. (1996). Rewarding actions of phencyclidine and related drugs in nucleus accumbens shell and frontal cortex. *Journal of Neuroscience*, *16*(9), 3112–3122.

Carver, C. S., & Scheier, M. F. (2002). The hopeful optimist. *Psychological Inquiry*, *13*(4), 288–290.

Cave, C. B. (1997). Very long-lasting priming in picture naming. *Psychological Science*, *8*(4), 322–325.

Chatlosh, D., Neunaber, D., & Wasserman, E. (1985). Response-outcome contingency: Behavioral and judgmental effects of appetitive and aversive outcomes with college students. *Learning and Motivation*, *16*(1), 1–34.

Cheavens, J. S., & Guter, M. M. (2018). Hope therapy. In M. W. Gallagher & S. J. Lopez (Eds.), *The Oxford handbook of hope* (pp. 133–142). Oxford University Press Oxford.

Chiken, S., & Nambu, A. (2016). Mechanism of deep brain stimulation: Inhibition, excitation, or disruption? *The Neuroscientist*, *22*(3), 313–322.

Choi, B. Y., Park, H., Yang, E., Lee, S. K., Lee, Y., & Lee, S. M. (2012). Understanding career decision self-efficacy: A meta-analytic approach. *Journal of Career Development*, *39*(5), 443–460.

Chong, T. T.-J., Bonnelle, V., Manohar, S., Veromann, K.-R., Muhammed, K., Tofaris, G. K., Hu, M., & Husain, M. (2015). Dopamine enhances willingness to exert effort for reward in Parkinson's disease. *Cortex*, *69*, 40–46.

Clark, R. E., Manns, J. R., & Squire, L. R. (2001). Trace and delay eyeblink conditioning: Contrasting phenomena of declarative and nondeclarative memory. *Psychological Science*, *12*(4), 304–308.

Conoley, C. W., & Scheel, M. J. (2017). *Goal focused positive psychotherapy: A strengths-based approach*. Oxford University Press.

Contreras-Vidal, J. L., & Schultz, W. (1999). A predictive reinforcement model of dopamine neurons for learning approach behavior. *Journal of Computational Neuroscience*, *6*(3), 191–214.

Cooper, A. J., Duke, É., Pickering, A. D., & Smillie, L. D. (2014). Individual differences in reward prediction error: Contrasting relations between feedback-related negativity and trait measures of reward sensitivity, impulsivity and extraversion. *Frontiers in Human Neuroscience*, *8*, 248.

Corbit, L. H., Ostlund, S. B., & Balleine, B. W. (2002). Sensitivity to instrumental contingency degradation is mediated by the entorhinal cortex and its efferents via the dorsal hippocampus. *Journal of Neuroscience*, *22*(24), 10976–10984.

Cousins, M., Atherton, A., Turner, L., & Salamone, J. (1996). Nucleus accumbens dopamine depletions alter relative response allocation in a T-maze cost/benefit task. *Behavioural Brain Research*, *74*(1-2), 189–197.

Cowen, S. L., Davis, G. A., & Nitz, D. A. (2012). Anterior cingulate neurons in the rat map anticipated effort and reward to their associated action sequences. *Journal of Neurophysiology*, *107*(9), 2393–2407.

Crivelli, D., & Balconi, M. (2017). The agent brain: A review of non-invasive brain stimulation studies on sensing agency. *Frontiers in Behavioral Neuroscience*, *11*, 229.

Crossley, M. J., Ashby, F. G., & Maddox, W. T. (2013). Erasing the engram: The unlearning of procedural skills. *Journal of Experimental Psychology: General*, *142*(3), 710–741.

Czernecki, V., Pillon, B., Houeto, J., Pochon, J., Levy, R., & Dubois, B. (2002). Motivation, reward, and Parkinson's disease: Influence of dopatherapy. *Neuropsychologia*, *40*(13), 2257–2267.

Deci, E. L., & Ryan, R. M. (2000). The "what" and "why" of goal pursuits: Human needs and the self-determination of behavior. *Psychological Inquiry*, *11*, 227–268.

Deci, E. L., & Ryan, R. M. (2002). *Self-determination research: Reflections and future directions*. University of Rochester Press.

Deeprose, C., & Andrade, J. (2006). Is priming during anesthesia unconscious? *Consciousness and Cognition*, *15*(1), 1–23.

de Jong, J. W., Afjei, S. A., Dorocic, I. P., Peck, J. R., Liu, C., Kim, C. K., Tian, L., Deisseroth, K., & Lammel, S. (2019). A neural circuit mechanism for encoding aversive stimuli in the mesolimbic dopamine system. *Neuron*, *101*(1), 133–151.

Devenport, L. (1979). Superstitious bar pressing in hippocampal and septal rats. *Science*, *205*(4407), 721–723.

Diener, E. (1994). Assessing subjective well-being: Progress and opportunities. *Social Indicators Research*, *31*(2), 103–157.

Eacott, M., & Gaffan, D. (1991). The role of monkey inferior parietal cortex in visual discrimination of identity and orientation of shapes. *Behavioural Brain Research*, *46*(1), 95–98.

Eichenbaum, H., Komorowski, R., MacDonald, C. J., Kraus, B. J., & Robitsek, J. (2016). How does the hippocampus support the spatial and temporal attributes of memory? In P. A. Jackson, A. A. Chiba, R. F. Berman, & M. E. Ragozzino (Eds.), *The neurobiological basis of memory* (pp. 39–57). Springer.

Elston, T. W., & Bilkey, D. K. (2017). Anterior cingulate cortex modulation of the ventral tegmental area in an effort task. *Cell Reports*, *19*(11), 2220–2230.

Ericsson, K. A., Krampe, R. T., & Tesch-Römer, C. (1993). The role of deliberate practice in the acquisition of expert performance. *Psychological Review*, *100*(3), 363–406.

Faget, L., Osakada, F., Duan, J., Ressler, R., Johnson, A. B., Proudfoot, J. A., Yoo, J. H., Callaway, E. M., & Hnasko, T. S. (2016). Afferent inputs to neurotransmitter-defined cell types in the ventral tegmental area. *Cell Reports*, *15*(12), 2796–2808.

Feenstra, M. G., & Botterblom, M. H. (1996). Rapid sampling of extracellular dopamine in the rat prefrontal cortex during food consumption, handling and exposure to novelty. *Brain Research*, *742*(1), 17–24.

Filoteo, J. V., Maddox, W. T., & Davis, J. D. (2001). A possible role of the striatum in linear and nonlinear category learning: Evidence from patients with Hungtington's disease. *Behavioral Neuroscience*, *115*(4), 786–798.

Filoteo, J. V., Maddox, W. T., Salmon, D. P., & Song, D. D. (2005). Information-integration category learning in patients with striatal dysfunction. *Neuropsychology*, *19*(2), 212–222.

Flouda, L., Pandazi, A., Papageorgiou, C., Perrea, D., Krepi, E., & Kostopanagiotou, G. (2013). Comparative effects of sevoflurane and propofol based general anaesthesia for elective surgery on memory. *Archives of Medical Science*, *9*(1), 105–111.

Frank, J. D., & Frank, J. B. (1993). *Persuasion and healing: A comparative study of psychotherapy*. Johns Hopkins University Press.

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist*, *56*(3), 218–226.

Fredrickson, B. L. (2004). The broaden–and–build theory of positive emotions. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *359*(1449), 1367–1377.

Fredrickson, B. L., & Joiner, T. (2002). Positive emotions trigger upward spirals toward emotional well-being. *Psychological Science*, *13*(2), 172–175.

Freeman, J. H., & Steinmetz, A. B. (2011). Neural circuitry and plasticity mechanisms underlying delay eyeblink conditioning. *Learning & Memory*, *18*(10), 666–677.

Frith, C. (2005). The self in action: Lessons from delusions of control. *Consciousness and Cognition*, *14*(4), 752–770.

Fritz, T. H., Halfpaap, J., Grahl, S., Kirkland, A., & Villringer, A. (2013). Musical feedback during exercise machine workout enhances mood. *Frontiers in Psychology*, *4*, 921.

Gable, S. L., Reis, H. T., Impett, E. A., & Asher, E. R. (2018). What do you do when things go right? The intrapersonal and interpersonal benefits of sharing positive events. In H. T. Reis (Ed.), *Relationships, well-being and behaviour* (pp. 144–182). Routledge.

Gaffan, D., & Eacott, M. (1995). Uncinate fascicle section leaves delayed matching-to-sample intact, with both large and small stimulus sets. *Experimental Brain Research*, *105*(1), 175–180.

Gallagher, M. W., & Lopez, S. J. (2009). Positive expectancies and mental health: Identifying the unique contributions of hope and optimism. *The Journal of Positive Psychology*, *4*(6), 548–556.

George, M. S., Ketter, T. A., Parekh, P. I., Horwitz, B., Herscovitch, P., Post, R. M., et al. (1995). Brain activity during transient sadness and happiness in healthy women. *American Journal of Psychiatry*, *152*(3), 341–351.

Gilbert, S. J., & Burgess, P. W. (2008). Executive function. *Current Biology*, *18*(3), R110–R114.

Giordana, B., Benoit, M., Darmon, N., Yelnik, J., Millet, B., & Fontaine, D. (2015). Acute and reproducible mood improvement due to nucleus accumbens deep brain stimulation. *Brain Stimulation*, *8*(4), 842–843.

Goto, Y., & Grace, A. A. (2005). Dopaminergic modulation of limbic and cortical drive of nucleus accumbens in goal-directed behavior. *Nature Neuroscience*, *8*(6), 805–812.

Grace, A. A., Floresco, S. B., Goto, Y., & Lodge, D. J. (2007). Regulation of firing of dopaminergic neurons and control of goal-directed behaviors. *Trends in Neurosciences*, *30*(5), 220–227.

Gray, N. S., Pickering, A. D., Snowden, R. J., Hemsley, D. R., & Gray, J. A. (2002). The partial reinforcement extinction effect in humans: Effects of schizophrenia, schizotypy and low doses of amphetamine. *Behavioural Brain Research*, *133*(2), 333–342.

Grice, G. R. (1948). The relation of secondary reinforcement to delayed reward in visual discrimination learning. *Journal of Experimental Psychology*, *38*(1), 1–16.

Gwaltney, C. J., Metrik, J., Kahler, C. W., & Shiffman, S. (2009). Self-efficacy and smoking cessation: A meta-analysis. *Psychology of Addictive Behaviors*, *23*(1), 56–66.

Haber, S. N. (2016). Corticostriatal circuitry. *Dialogues in Clinical Neuroscience*, *18*(1), 7–21.

Haber, S. N., Fudge, J. L., & McFarland, N. R. (2000). Striatonigrostriatal pathways in primates form an ascending spiral from the shell to the dorsolateral striatum. *Journal of Neuroscience*, *20*(6), 2369–2382.

Haggard, P. (2017). Sense of agency in the human brain. *Nature Reviews Neuroscience*, *18*(4), 196–207.

Hamid, A. A., Pettibone, J. R., Mabrouk, O. S., Hetrick, V. L., Schmidt, R., Vander Weele, C. M., Kennedy, R. T., Aragona, B. J., & Berke, J. D. (2016). Mesolimbic dopamine signals the value of work. *Nature Neuroscience*, *19*(1), 117–126.

Haq, I. U., Foote, K. D., Goodman, W. G., Wu, S. S., Sudhyadhom, A., Ricciuti, N., Siddiqui, M. S., Bowers, D., Jacobson, C. E., Ward, H., et al. (2011). Smile and laughter induction and intraoperative predictors of response to deep brain stimulation for obsessive-compulsive disorder. *NeuroImage*, *54*, S247–S255.

Hart, A. S., Rutledge, R. B., Glimcher, P. W., & Phillips, P. E. (2014). Phasic dopamine release in the rat nucleus accumbens symmetrically encodes a reward prediction error term. *Journal of Neuroscience*, *34*(3), 698–704.

Haruno, M., & Kawato, M. (2006). Different neural correlates of reward expectation and reward expectation error in the putamen and caudate nucleus during stimulus-action-reward association learning. *Journal of Neurophysiology*, *95*(2), 948–959.

Hassall, C. D., Hajcak, G., & Krigolson, O. E. (2019). The importance of agency in human reward processing. *Cognitive, Affective, & Behavioral Neuroscience*, *19*(6), 1458–1466.

Hazy, T. E., Frank, M. J., & O'Reilly, R. C. (2010). Neural mechanisms of acquired phasic dopamine responses in learning. *Neuroscience & Biobehavioral Reviews*, *34*(5), 701–720.

Heckhausen, J., Wrosch, C., & Schulz, R. (2019). Agency and motivation in adulthood and old age. *Annual Review of Psychology*, *70*, 191–217.

Hillman, K. L., & Bilkey, D. K. (2010). Neurons in the rat anterior cingulate cortex dynamically encode cost–benefit in a spatial decision-making task. *Journal of Neuroscience*, *30*(22), 7705–7713.

Hollerman, J. R., & Schultz, W. (1998). Dopamine neurons report an error in the temporal prediction of reward during learning. *Nature Neuroscience*, *1*(4), 304–309.

Hong, S., & Hikosaka, O. (2014). Pedunculopontine tegmental nucleus neurons provide reward, sensorimotor, and alerting signals to midbrain dopamine neurons. *Neuroscience*, *282*, 139–155.

Hong, S., Jhou, T. C., Smith, M., Saleem, K. S., & Hikosaka, O. (2011). Negative reward signals from the lateral habenula to dopamine neurons are mediated by rostromedial tegmental nucleus in primates. *Journal of Neuroscience*, *31*(32), 11457–11471.

Houk, J., Adams, J., & Barto, A. (1995). A model of how the basal ganglia generate and use neural signals that predict reinforcement. In J. L. Houk J. C. Davis & D. G. Beiser (Eds.), *Models of information processing in the basal ganglia* (pp. 249–270). Cambridge, MA: MIT Press.

Howe, M. W., Tierney, P. L., Sandberg, S. G., Phillips, P. E., & Graybiel, A. M. (2013). Prolonged dopamine signalling in striatum signals proximity and value of distant rewards. *Nature*, *500*(7464), 575–579.

Huang, S., Zhang, Z., Gambeta, E., Xu, S. C., Thomas, C., Godfrey, N., Chen, L., M'Dahoma, S., Borgland, S. L., & Zamponi, G. W. (2020). Dopamine inputs from the ventral tegmental area into the medial prefrontal cortex modulate neuropathic pain-associated behaviors in mice. *Cell Reports*, *31*(12), 107812.

Humphries, M. D., & Prescott, T. J. (2010). The ventral basal ganglia, a selection mechanism at the crossroads of space, strategy, and reward. *Progress in Neurobiology*, *90*(4), 385–417.

Inglis, J. B., Valentin, V. V., & Ashby, F. G. (2021). Modulation of dopamine for adaptive learning: A neurocomputational model. *Computational Brain & Behavior*, *4*(1), 34–52.

Insausti, R., Amaral, D., & Cowan, W. (1987). The entorhinal cortex of the monkey: II. cortical afferents. *Journal of Comparative Neurology*, *264*(3), 356–395.

Iselin-Chaves, I. A., Willems, S. J., Jermann, F. C., Forster, A., Adam, S. R., & Van der Linden, M. (2005). Investigation of implicit memory during isoflurane anesthesia for elective surgery using the process dissociation procedure. *The Journal of the American Society of Anesthesiologists*, *103*(5), 925–933.

Isen, A. M., Nygren, T. E., & Ashby, F. G. (1988). Influence of positive affect on the subjective utility of gains and losses: It is just not worth the risk. *Journal of Personality and Social Psychology*, *55*(5), 710–717.

Izquierdo, A., Suda, R. K., & Murray, E. A. (2004). Bilateral orbital prefrontal cortex lesions in rhesus monkeys disrupt choices guided by both reward value and reward contingency. *Journal of Neuroscience*, *24*(34), 7540–7548.

Jackson, S. A., Horst, N. K., Pears, A., Robbins, T. W., & Roberts, A. C. (2016). Role of the perigenual anterior cingulate and orbitofrontal cortex in contingency learning in the marmoset. *Cerebral Cortex*, *26*(7), 3273–3284.

Jacobs, J., Kahana, M. J., Ekstrom, A. D., Mollison, M. V., & Fried, I. (2010). A sense of direction in human entorhinal cortex. *Proceedings of the National Academy of Sciences*, *107*(14), 6487–6492.

Jacoby, L. L. (1991). A process dissociation framework: Separating automatic from intentional uses of memory. *Journal of Memory and Language*, *30*(5), 513–541.

Jallais, C., & Gilet, A.-L. (2010). Inducing changes in arousal and valence: Comparison of two mood induction procedures. *Behavior Research Methods*, *42*(1), 318–325.

Jocham, G., Brodersen, K. H., Constantinescu, A. O., Kahn, M. C., Ianni, A. M., Walton, M. E., Rushworth, M. F., & Behrens, T. E. (2016). Reward-guided learning with and without causal attribution. *Neuron*, *90*(1), 177–190.

Joel, D., Niv, Y., & Ruppin, E. (2002). Actor–critic models of the basal ganglia: New anatomical and computational perspectives. *Neural Networks*, *15*(4-6), 535–547.

Johnson, E. J., & Tversky, A. (1983). Affect, generalization, and the perception of risk. *Journal of Personality and Social Psychology*, *45*(1), 20–31.

Karsh, N., Goldstein, O., & Eitam, B. (2018). Evidence for pain attenuation by the motor system-based judgment of agency. *Consciousness and Cognition*, *57*, 134–146.

Kawato, M., & Samejima, K. (2007). Efficient reinforcement learning: Computational theories, neuroscience and robotics. *Current Opinion in Neurobiology*, *17*(2), 205–212.

Keyes, C. L. M. (1998). Social well-being. *Social Psychology Quarterly*, *61*(2), 121–140.

Knowlton, B. J., Mangels, J. A., & Squire, L. R. (1996). A neostriatal habit learning system in humans. *Science*, *273*(5280), 1399–1402.

Kobayashi, Y., & Okada, K. (2007). Reward prediction error computation in the pedunculopontine tegmental nucleus neurons. *Annals of the New York Academy of Sciences*, *1104*(1), 310–323.

Koivisto, M., Portin, R., & Rinne, J. O. (1996). Perceptual priming in Alzheimer's and Parkinson's diseases. *Neuropsychologia*, *34*(5), 449–457.

Kosaki, Y., & Watanabe, S. (2012). Dissociable roles of the medial prefrontal cortex, the anterior cingulate cortex, and the hippocampus in behavioural flexibility revealed by serial reversal of three-choice discrimination in rats. *Behavioural Brain Research*, *227*(1), 81–90.

Kovacs, P., Hélie, S., Tran, A. N., & Ashby, F. G. (2021). A neurocomputational theory of how rule-guided behaviors become automatic. *Psychological Review*, *128*, 488–508.

Kumar, N., Manjaly, J. A., & Sunny, M. M. (2015). The relationship between action-effect monitoring and attention capture. *Journal of Experimental Psychology: General*, *144*(1), 18–23.

Kummer, K. K., Mitrić, M., Kalpachidou, T., & Kress, M. (2020). The medial prefrontal cortex as a central hub for mental comorbidities associated with chronic pain. *International Journal of Molecular Sciences*, *21*(10), 3440.

Lauzon, N. M., Bechard, M., Ahmad, T., & Laviolette, S. R. (2013). Supra-normal stimulation of dopamine D1 receptors in the prelimbic cortex blocks behavioral expression of both aversive and rewarding associative memories through a cyclic-AMP-dependent signaling pathway. *Neuropharmacology*, *67*, 104–114.

Lee, I., & Lee, C. H. (2013). Contextual behavior and neural circuits. *Frontiers in Neural Circuits*, *7*, 84.

Lewthwaite, R., Chiviacowsky, S., Drews, R., & Wulf, G. (2015). Choose to move: The motivational impact of autonomy support on motor learning. *Psychonomic Bulletin & Review*, *22*(5), 1383–1388.

Lex, B., & Hauber, W. (2010). Disconnection of the entorhinal cortex and dorsomedial striatum impairs the sensitivity to instrumental contingency degradation. *Neuropsychopharmacology*, *35*(8), 1788–1796.

Lisman, J., Schulman, H., & Cline, H. (2002). The molecular basis of CaMKII function in synaptic and behavioural memory. *Nature Reviews Neuroscience*, *3*(3), 175–190.

Liu, X., Hairston, J., Schrier, M., & Fan, J. (2011). Common and distinct networks underlying reward valence and processing stages: A meta-analysis of functional neuroimaging studies. *Neuroscience & Biobehavioral Reviews*, *35*(5), 1219–1236.

Lodge, D. J., & Grace, A. A. (2006). The hippocampus modulates dopamine neuron responsivity by regulating the intensity of phasic neuron activation. *Neuropsychopharmacology*, *31*(7), 1356–1361.

Lopez, S. J., Floyd, R. K., Ulven, J. C., & Snyder, C. (2000). Hope therapy: Helping clients build a house of hope. In C. R. Snyder (Ed.), *Handbook of hope* (pp. 123–150). Elsevier.

Loyola-Navarro, R., Moënne-Loccoz, C., Vergara, R. C., Hyafil, A., Aboitiz, F., & Maldonado, P. E. (2022). Voluntary self-initiation of the stimuli onset improves working memory and accelerates visual and attentional processing. *Heliyon*, *8*(12), e12215.

Luciana, M., Depue, R. A., Arbisi, P., & Leon, A. (1992). Facilitation of working memory in humans by a D2 dopamine receptor agonist. *Journal of Cognitive Neuroscience*, *4*(1), 58–68.

Ma, L., Hyman, J. M., Phillips, A. G., & Seamans, J. K. (2014). Tracking progress toward a goal in corticostriatal ensembles. *Journal of Neuroscience*, *34*(6), 2244–2253.

Maddox, W. T., Ashby, F. G., & Bohil, C. J. (2003). Delayed feedback effects on rule-based and information-integration category learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *29*, 650–662.

Maddox, W. T., & Ing, A. D. (2005). Delayed feedback disrupts the procedural-learning system but not the hypothesis testing system in perceptual category learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *31*(1), 100–107.

Magaletta, P. R., & Oliver, J. (1999). The hope construct, will, and ways: Their relations with self-efficacy, optimism, and general well-being. *Journal of Clinical Psychology*, *55*(5), 539–551.

Manns, J. R., Clark, R. E., & Squire, L. R. (2000). Awareness predicts the magnitude of single-cue trace eyeblink conditioning. *Hippocampus*, *10*(2), 181–186.

Manns, J. R., Clark, R. E., & Squire, L. R. (2002). Standard delay eyeblink classical conditioning is independent of awareness. *Journal of Experimental Psychology: Animal Behavior Processes*, *28*(1), 32–37.

Matsumoto, M., & Hikosaka, O. (2007). Lateral habenula as a source of negative reward signals in dopamine neurons. *Nature*, *447*(7148), 1111–1115.

Matsumoto, M., & Hikosaka, O. (2009). Representation of negative motivational value in the primate lateral habenula. *Nature Neuroscience*, *12*(1), 77–84.

Mazzoni, P., Hristova, A., & Krakauer, J. W. (2007). Why don't we move faster? Parkinson's disease, movement vigor, and implicit motivation. *Journal of Neuroscience*, *27*(27), 7105–7116.

McGlinchey-Berroth, R., Carrillo, M. C., Gabrieli, J. D., Brawn, C. M., & Disterhoft, J. F. (1997). Impaired trace eyeblink conditioning in bilateral, medial-temporal lobe amnesia. *Behavioral Neuroscience*, *111*(5), 873–882.

McGuigan, S., Zhou, S.-H., Brosnan, M. B., Thyagarajan, D., Bellgrove, M. A., & Chong, T. T. (2019). Dopamine restores cognitive motivation in Parkinson's disease. *Brain*, *142*(3), 719–732.

McKinlay, A., Grace, R. C., Dalrymple-Alford, J. C., & Roger, D. (2010). Characteristics of executive function impairment in Parkinson's disease patients without dementia. *Journal of the International Neuropsychological Society*, *16*(2), 268–277.

McNaughton, N. (1989). *Biology and emotion*. Cambridge University Press.

Mele, A. R. (2003). *Motivation and agency*. Oxford University Press.

Mellers, B. A., Schwartz, A., Ho, K., & Ritov, I. (1997). Decision affect theory: Emotional reactions to the outcomes of risky options. *Psychological Science*, *8*(6), 423–429.

Merchant, H., Zainos, A., Hernández, A., Salinas, E., & Romo, R. (1997). Functional properties of primate putamen neurons during the categorization of tactile stimuli. *Journal of Neurophysiology*, *77*(3), 1132–1154.

Metcalfe, J., & Greene, M. J. (2007). Metacognition of agency. *Journal of Experimental Psychology: General*, *136*(2), 184–199.

Mirenowicz, J., & Schultz, W. (1994). Importance of unpredictability for reward responses in primate dopamine neurons. *Journal of Neurophysiology*, *72*(2), 1024–1027.

Miyachi, S., Hikosaka, O., & Lu, X. (2002). Differential activation of monkey striatal neurons in the early and late stages of procedural learning. *Experimental Brain Research*, *146*(1), 122–126.

Mohr, C., Binkofski, F., Erdmann, C., Büchel, C., & Helmchen, C. (2005). The anterior cingulate cortex contains distinct areas dissociating external from self-administered painful stimulation: A parametric fMRI study. *Pain*, *114*(3), 347–357.

Moore, J. W. (2016). What is the sense of agency and why does it matter? *Frontiers in Psychology*, *7*, 1272.

Morita, K., Morishima, M., Sakai, K., & Kawaguchi, Y. (2013). Dopaminergic control of motivation and reinforcement learning: A closed-circuit account for reward-oriented behavior. *Journal of Neuroscience*, *33*(20), 8866–8890.

Müller, M. J. (2012). Will it hurt less if I believe I can control it? Influence of actual and perceived control on perceived pain intensity in healthy male individuals: A randomized controlled study. *Journal of Behavioral Medicine*, *35*(5), 529–537.

Müller, U., von Cramon, D. Y., & Pollmann, S. (1998). D1-versus D2-receptor modulation of visuospatial working memory in humans. *Journal of Neuroscience*, *18*(7), 2720–2728.

Murty, V. P., DuBrow, S., & Davachi, L. (2015). The simple act of choosing influences declarative memory. *Journal of Neuroscience*, *35*(16), 6255–6264.

Nakajima, S., Urushihara, K., & Masaki, T. (2002). Renewal of operant performance formerly eliminated by omission or noncontingency training upon return to the acquisition context. *Learning and Motivation*, *33*(4), 510–525.

Niv, Y. (2007). Cost, benefit, tonic, phasic: What do response rates tell us about dopamine and motivation? *Annals of the New York Academy of Sciences*, *1104*(1), 357–376.

Niv, Y., Daw, N. D., Joel, D., & Dayan, P. (2007). Tonic dopamine: Opportunity costs and the control of response vigor. *Psychopharmacology*, *191*(3), 507–520.

Niv, Y., Joel, D., & Dayan, P. (2006). A normative perspective on motivation. *Trends in Cognitive Sciences*, *10*(8), 375–381.

Nomura, E., Maddox, W., Filoteo, J., Ing, A., Gitelman, D., Parrish, T., Mesulam, M., & Reber, P. (2007). Neural correlates of rule-based and information-integration visual category learning. *Cerebral Cortex*, *17*(1), 37–43.

O'Doherty, J. P., Dayan, P., Schultz, J., Deichmann, R., Friston, K., & Dolan, R. J. (2004). Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science*, *304*(5669), 452–454.

O'Donnell, P., & Grace, A. A. (1994). Tonic D2-mediated attenuation of cortical excitation in nucleus accumbens neurons recorded *in vitro*. *Brain Rresearch*, *634*(1), 105–112.

Okada, K., & Kobayashi, Y. (2013). Reward prediction-related increases and decreases in tonic neuronal activity of the pedunculopontine tegmental nucleus. *Frontiers in Integrative Neuroscience*, *7*, 36.

Oniz, A., Inanc, G., Guducu, C., & Ozgoren, M. (2015). Explicit and implicit memory during sleep. *Sleep and Biological Rhythms*, *13*, 380–386.

O'Reilly, R. C., Frank, M. J., Hazy, T. E., & Watz, B. (2007). PVLV: The primary value and learned value Pavlovian learning algorithm. *Behavioral Neuroscience*, *121*(1), 31–49.

Ott, T., & Nieder, A. (2019). Dopamine and cognitive control in prefrontal cortex. *Trends in Cognitive Sciences*, *23*(3), 213–234.

Otto, A., & Eichstaedt, J. (2018). Real-world unexpected outcomes predict city-level mood states and risk-taking behavior. *PLoS ONE*, *13*(11), e0206923.

Packard, M. G., & McGaugh, J. L. (1992). Double dissociation of fornix and caudate nucleus lesions on acquisition of two water maze tasks: Further evidence for multiple memory systems. *Behavioral Neuroscience*, *106*(3), 439–446.

Pajares, F. (1996). Self-efficacy beliefs in achievement settings. *Review of Educational Research*, *66*, 543–578.

Parvizi, J., Rangarajan, V., Shirer, W. R., Desai, N., & Greicius, M. D. (2013). The will to persevere induced by electrical stimulation of the human cingulate gyrus. *Neuron*, *80*(6), 1359–1367.

Pickering, A. D. (2004). The neuropsychology of impulsive antisocial sensation seeking personality traits: From dopamine to hippocampal function. In R. M. Stelmack (Ed.), *On the psychobiology of personality: Essays in honor of Marvin Zuckerman* (pp. 453–476). Elsevier.

Pickering, A. D., & Pesola, F. (2014). Modeling dopaminergic and other processes involved in learning from reward prediction error: Contributions from an individual differences perspective. *Frontiers in Human Neuroscience*, *8*, 740.

Potvin, S., Grignon, S., & Marchand, S. (2009). Human evidence of a supra-spinal modulating role of dopamine on pain perception. *Synapse*, *63*(5), 390–402.

Quan, X., Yi, J., Ye, T., Tian, S., Zou, L., Yu, X., & Huang, Y. (2013). Propofol and memory: A study using a process dissociation procedure and functional magnetic resonance imaging. *Anaesthesia*, *68*(4), 391–399.

Rawlins, J., Feldon, J., Tonkiss, J., & Coffey, P. (1989). The role of subicular outputs in the development of the partial reinforcement extinction effect. *Experimental Brain Research*, *77*, 153–160.

Redish, A. D., & Touretzky, D. S. (1997). Cognitive maps beyond the hippocampus. *Hippocampus*, *7*(1), 15–35.

Render, A., & Jansen, P. (2019). Dopamine and sense of agency: Determinants in personality and substance use. *PloS One*, *14*(3), e0214069.

Rescorla, R. A. (1968). Probability of shock in the presence and absence of CS in fear conditioning. *Journal of Comparative and Physiological Psychology*, *66*(1), 1–5.

Rescorla, R. A., & Skucy, J. C. (1969). Effect of response-independent reinforcers during extinction. *Journal of Comparative and Physiological Psychology*, *67*(3), 381–389.

Reynolds, J. N., & Wickens, J. R. (2002). Dopamine-dependent plasticity of corticostriatal synapses. *Neural Networks*, *15*(4-6), 507–521.

Robbins, S. B., Lauver, K., Le, H., Davis, D., Langley, R., & Carlstrom, A. (2004). Do psychosocial and study skill factors predict college outcomes? A meta-analysis. *Psychological Bulletin*, *130*(2), 261–288.

Rottinghaus, P. J., Larson, L. M., & Borgen, F. H. (2003). The relation of self-efficacy and interests: A meta-analysis of 60 samples. *Journal of Vocational Behavior*, *62*(2), 221–236.

Russell, J. (2013). *Agency: Its role in mental development*. Psychology Press.

Rutledge, R. B., Skandali, N., Dayan, P., & Dolan, R. J. (2014). A computational and neural model of momentary subjective well-being. *Proceedings of the National Academy of Sciences*, *111*(33), 12252–12257.

Ryff, C. D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology*, *57*(6), 1069–1081.

Salamone, J. D., & Correa, M. (2012). The mysterious motivational functions of mesolimbic dopamine. *Neuron*, *76*(3), 470–485.

Salamone, J. D., Cousins, M. S., & Bucher, S. (1994). Anhedonia or anergia? Effects of haloperidol and nucleus accumbens dopamine depletion on instrumental response selection in a T-maze cost/benefit procedure. *Behavioural Brain Research*, *65*(2), 221–229.

Sanli, E. A., Patterson, J. T., Bray, S. R., & Lee, T. D. (2013). Understanding self-controlled motor learning protocols through the self-determination theory. *Frontiers in Psychology*, *3*, 611.

Sawaguchi, T., & Goldman-Rakic, P. S. (1991). D1 dopamine receptors in prefrontal cortex: Involvement in working memory. *Science*, *251*(4996), 947–950.

Schacter, D. L., & Buckner, R. L. (1998). Priming and the brain. *Neuron*, *20*(2), 185–195.

Schmaltz, L. W., & Theios, J. (1972). Acquisition and extinction of a classically conditioned response in hippocampectomized rabbits (Oryctolagus cuniculus). *Journal of Comparative and Physiological Psychology*, *79*(2), 328–333.

Schultz, W. (1998). Predictive reward signal of dopamine neurons. *Journal of Neurophysiology*, *80*(1), 1–27.

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, *275*(5306), 1593–1599.

Seger, C. A., & Cincotta, C. M. (2005). The roles of the caudate nucleus in human classification learning. *Journal of Neuroscience*, *25*(11), 2941–2951.

Sesack, S. R., & Grace, A. A. (2010). Cortico-basal ganglia reward network: Microcircuitry. *Neuropsychopharmacology*, *35*(1), 27–47.

Sheeran, P., Maki, A., Montanaro, E., Avishai-Yitshak, A., Bryan, A., Klein, W. M., Miles, E., & Rothman, A. J. (2016). The impact of changing attitudes, norms, and self-efficacy on health-related intentions and behavior: A meta-analysis. *Health Psychology*, *35*(11), 1178–1188.

Shepperd, J. A., & McNulty, J. K. (2002). The affective consequences of expected and unexpected outcomes. *Psychological Science*, *13*(1), 85–88.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological Review*, *84*(2), 127–190.

Shoji, K., Cieslak, R., Smoktunowicz, E., Rogala, A., Benight, C. C., & Luszczynska, A. (2016). Associations between job burnout and self-efficacy: A meta-analysis. *Anxiety, Stress, & Coping*, *29*(4), 367–386.

Smillie, L. D., Cooper, A. J., & Pickering, A. D. (2011). Individual differences in reward–prediction–error: Extraversion and feedback-related negativity. *Social Cognitive and Affective Neuroscience*, *6*(5), 646–652.

Smillie, L. D., Cooper, A. J., Proitsi, P., Powell, J. F., & Pickering, A. D. (2010). Variation in DRD2 dopamine gene predicts extraverted personality. *Neuroscience Letters*, *468*(3), 234–237.

Smillie, L. D., Jach, H. K., Hughes, D. M., Wacker, J., Cooper, A. J., & Pickering, A. D. (2019). Extraversion and reward-processing: Consolidating evidence from an electroencephalographic index of reward-prediction-error. *Biological Psychology*, *146*, 107735.

Snyder, C. R. (1989). Reality negotiation: From excuses to hope and beyond. *Journal of Social and Clinical Psychology*, *8*(2), 130–157.

Snyder, C. R. (2002). Hope theory: Rainbows in the mind. *Psychological Inquiry*, *13*(4), 249–275.

Snyder, C. R., Irving, L. M., & Anderson, J. R. (1991). Hope and health. *Handbook of Social and Clinical Psychology: The Health Perspective*, *162*, 285–305.

Sperduti, M., Delaveau, P., Fossati, P., & Nadel, J. (2011). Different brain structures related to self-and external-

agency attribution: A brief review and meta-analysis. *Brain Structure and Function*, *216*(2), 151–157.

Steinberg, E. E., Keiflin, R., Boivin, J. R., Witten, I. B., Deisseroth, K., & Janak, P. H. (2013). A causal link between prediction errors, dopamine neurons and learning. *Nature Neuroscience*, *16*(7), 966–973.

Sternberg, S. (1966). High-speed scanning in human memory. *Science*, *153*(3736), 652–654.

Stuber, G. D., Klanker, M., De Ridder, B., Bowers, M. S., Joosten, R. N., Feenstra, M. G., & Bonci, A. (2008). Reward-predictive cues enhance excitatory synaptic strength onto midbrain dopamine neurons. *Science*, *321*(5896), 1690–1692.

Surmeier, D. J., Ding, J., Day, M., Wang, Z., & Shen, W. (2007). D1 and D2 dopamine-receptor modulation of striatal glutamatergic signaling in striatal medium spiny neurons. *Trends in Neurosciences*, *5*(30), 228–235.

Sutton, R. S., & Barto, A. G. (1998). *Reinforcement learning: An introduction*. MIT Press.

Syed, E. C., Grima, L. L., Magill, P. J., Bogacz, R., Brown, P., & Walton, M. E. (2016). Action initiation shapes mesolimbic dopamine encoding of future rewards. *Nature Neuroscience*, *19*(1), 34–36.

Synofzik, M., Vosgerau, G., & Newen, A. (2008). Beyond the comparator model: A multifactorial two-step account of agency. *Consciousness and Cognition*, *17*(1), 219–239.

Takahashi, Y. K., Langdon, A. J., Niv, Y., & Schoenbaum, G. (2016). Temporal specificity of reward prediction errors signaled by putative dopamine neurons in rat VTA depends on ventral striatum. *Neuron*, *91*(1), 182–193.

Tan, C. O., & Bullock, D. (2008). A local circuit model of learned striatal and dopamine cell responses under probabilistic schedules of reward. *Journal of Neuroscience*, *28*(40), 10062–10074.

Tanaka, S. C., Balleine, B. W., & O'Doherty, J. P. (2008). Calculating consequences: Brain systems that encode the causal effects of actions. *Journal of Neuroscience*, *28*(26), 6750–6755.

Tian, J., & Uchida, N. (2015). Habenula lesions reveal that multiple mechanisms underlie dopamine prediction errors. *Neuron*, *87*(6), 1304–1316.

Tiemann, L., Heitmann, H., Schulz, E., Baumkötter, J., & Ploner, M. (2014). Dopamine precursor depletion influences pain affect rather than pain sensation. *PLoS One*, *9*(4), e96167.

Treede, R.-D., Kenshalo, D. R., Gracely, R. H., & Jones, A. K. (1999). The cortical representation of pain. *Pain*, *79*(2-3), 105–111.

Tzschentke, T. (2000). The medial prefrontal cortex as a part of the brain reward system. *Amino Acids*, *19*(1), 211–219.

van der Wel, R. P., Sebanz, N., & Knoblich, G. (2012). The sense of agency during skill learning in individuals and dyads. *Consciousness and Cognition*, *21*(3), 1267–1279.

Varrone, A., & Halldin, C. (2014). Human brain imaging of dopamine transporters. In P. Seeman & B. Madras (Eds.), *Imaging of the human brain in health and disease* (pp. 203–240). Amsterdam: Elsevier.

Vijayraghavan, S., Wang, M., Birnbaum, S. G., Williams, G. V., & Arnsten, A. F. (2007). Inverted-U dopamine D1 receptor actions on prefrontal neurons engaged in working memory. *Nature Neuroscience*, *10*(3), 376–384.

Vingerhoets, G., Vermeule, E., & Santens, P. (2005). Impaired intentional content learning but spared incidental retention of contextual information in non-demented patients with Parkinson's disease. *Neuropsychologia*, *43*(5), 675–681.

Vucovich, L. (2016). *The role of feedback contingency in perceptual category learning: An investigation of neurobiological and behavioral mechanisms* [Doctoral dissertation, University of California, Santa Barbara].

Waldschmidt, J. G., & Ashby, F. G. (2011). Cortical and striatal contributions to automaticity in information-integration categorization. *NeuroImage*, *56*(3), 1791–1802.

Walton, M. E., Behrens, T. E., Buckley, M. J., Rudebeck, P. H., & Rushworth, M. F. (2010). Separable learning systems in the macaque brain and the role of orbitofrontal cortex in contingent learning. *Neuron*, *65*(6), 927–939.

Wang, Y., Wang, J.-Y., & Luo, F. (2011). Why self-induced pain feels less painful than externally generated pain: Distinct brain activation patterns in self- and externally generated pain. *PloS One*, *6*(8), e23536.

Watabe-Uchida, M., Zhu, L., Ogawa, S. K., Vamanrao, A., & Uchida, N. (2012). Whole-brain mapping of direct inputs to midbrain dopamine neurons. *Neuron*, *74*(5), 858–873.

Wegner, D. M., & Wheatley, T. (1999). Apparent mental causation: Sources of the experience of will. *American Psychologist*, *54*(7), 480–492.

Welniarz, Q., Worbe, Y., & Gallea, C. (2021). The forward model: A unifying theory for the role of the cerebellum in motor control and sense of agency. *Frontiers in Systems Neuroscience*, *15*, 644059.

West, A. R., & Grace, A. A. (2002). Opposite influences of endogenous dopamine D1 and D2 receptor activation on activity states and electrophysiological properties of striatal neurons: Studies combining *in vivo* intracellular recordings and reverse microdialysis. *Journal of Neuroscience*, *22*(1), 294–304.

Wickens, J. (1993). *A theory of the striatum*. Pergamon Press.

Wiggs, C. L., & Martin, A. (1998). Properties and mechanisms of perceptual priming. *Current Opinion in Neurobiology*, *8*(2), 227–233.

Willingham, D. B. (1998). A neuropsychological theory of motor skill learning. *Psychological Review*, *105*, 558–584.

Wise, R. A. (1996). Addictive drugs and brain stimulation reward. *Annual Review of Neuroscience*, *19*(1), 319–340.

Wood, P. B. (2006). Mesolimbic dopaminergic mechanisms and pain control. *Pain*, *120*(3), 230–234.

Wood, P. B., Patterson II, J. C., Sunderland, J. J., Tainter, K. H., Glabus, M. F., & Lilien, D. L. (2007). Reduced presynaptic dopamine activity in fibromyalgia syndrome demonstrated with positron emission tomography: A pilot study. *The Journal of Pain*, *8*(1), 51–58.

Woods, A. M., & Bouton, M. E. (2007). Occasional reinforced responses during extinction can slow the rate of reacquisition of an operant response. *Learning and Motivation*, *38*(1), 56–74.

Wu, W. (2013). Mental action and the threat of automaticity. In A. Clark, J. Kiverstein, & T. Vierkant (Eds.), *Decomposing the will* (pp. 244–261). Oxford University Press.

Wulf, G. (2007). Self-controlled practice enhances motor learning: Implications for physiotherapy. *Physiotherapy*, *93*(2), 96–101.

Yagishita, S., Hayashi-Takagi, A., Ellis-Davies, G. C., Urakubo, H., Ishii, S., & Kasai, H. (2014). A critical time window for dopamine actions on the structural plasticity of dendritic spines. *Science*, *345*(6204), 1616–1620.

Yalom, I. D., & Leszcz, M. (2020). *The theory and practice of group psychotherapy*. Basic Books.

Yin, H. H., Ostlund, S. B., Knowlton, B. J., & Balleine, B. W. (2005). The role of the dorsomedial striatum in instrumental conditioning. *European Journal of Neuroscience*, *22*(2), 513–523.