

Reward and Punishment Processing in Depression

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Abstract: *Depression is an intricate and assorted disorder whose reason is inadequately grasped. Theories on the mechanisms of the disease have regularly spotlighted either its neurobiology or its cognitive and behavioural interpretations. Lately, studies investigating how depressed patients process reward and punishment have related these two aspects jointly. It has been proposed that individuals with a dysfunction in a specific network of brain areas are not capable to develop emotional information to conduct behaviour. Deficits in this aptitude might incline such individuals to develop depression, while consequent reinstatement of this aptitude, whether throughout pharmacological or behavioural treatments—might permit healing from the disorder. Here we analysis behavioural, neuroimaging, and computational findings pertinent to this hypothesis. There is good evidence that depressed patients reveal aberrant behavioural in response to rewards and punishments and that these propensities correspond to abnormal function in frontostriatal systems adapted by the monoamine systems. In addition, computational studies have drawn testable projections for how these neural indications and neurochemical aberration might contribute to the hallmarks of depression. Merging these approaches—as well as molecular and behavioural task in animals—offers great promise for extending our knowledge of this ordinary and incapacitating disease.*

Keywords: *Behaviour, computational, depression, feedback, neuro-imaging, reinforcement*

Depression is a principal cause of morbidity and mortality universally. Fifteen percent of people will develop depression over their lifetimes, making it a greater encumbers to wellbeing than angina, arthritis, asthma, and diabetes (Moussavi S, et al, 2007). Presently, the fourth chief cause of disability, it is predictable to become the second by 2030 (Mathers CD, Loncar D, 2006).

Even if low mood is the traditional signs of depression, anhedonia (reduced interest or pleasure) and cognitive dysfunction are equally central to the disorder (Clark L, et al & Roiser JP, et al, 2009). The diagnostic factors for Major Depressive Disorder (MDD) comprise such cognitive symptoms as indecisiveness and reduced concentration (American Psychiatric Association, 2000) and experimental tasks have shown depression-associated deficits in attention, memory, and psychomotor pace (Roiser JP, et al, 2009). Also, cognitive dysfunction outlines a center aspect of several psychological models of MDD, from Seligman's learned neural abnormalities to Beck's cognitive model (Beck AT, 1979). These theories have marked the starting point of cognitive therapies, which are a universal and effective means to fight depression. The cognitive deficits in depression are most prominent in the perspective of affective information processing. Emotionally "hot" tasks, which check responses to positively or negatively valenced incentives provoke more vigorous differences between MDD patients and healthy individuals than do "cold" tasks (e.g., motor function) (Roiser JP, 2009). These behavioral findings accord with convergent neuroimaging evidence putting forward that the same brain areas that function aberrantly in depressive patients (e.g., orbitofrontal cortex [OFC], medial frontal cortex, ventral striatum [VS], amygdala, and hippocampus) (Clark L, 2009 & Ebmeier KP, 2006) are vital for reinforcement processing (Schultz W, 2000). Taken jointly, these results advocate that imbalances in a different network of areas, mainly those innervated by monoamines, distract patients' ability to construct affective stimuli. This distraction could then provoke symptoms of depression. Such a two-stage process—in which neural abnormalities provoke impaired cognitive function, which in turn predisposes individuals to depression—bears out recent theories of antidepressant drug action (Harmer CJ, 2008).

To confirm these cognitive hypotheses, it is crucial to demonstrate that depressed patients have difficulty with reinforcement learning and decision-making and that this difficulty corresponds with aberrations in reward-related brain systems. In this non- thorough review, we spotlight selected evidence for these claims.

We start with behavioral results, shedding light on tasks in which patients are given reward or punishment throughout task performance. We then review human neuroimaging studies that match with the animal neurobiology literature (Nestler EJ, 2006), exploring the neural correlates of these behavioural effects. Finally, we probe computational modeling work, which has said to afford a potent account for the neural signals putting emphasize on depressed individuals' performance.

1. BEHAVIORAL STUDIES

It has long been confirmed that depressed patients undergo cognitive impairments as well as low mood (Seligman ME,1972). A number of paradigms have been employed to review patients' ability to process information in the framework of reward or punishment.

Two major conclusions have been drawn from this literature: that depressed individuals reveal maladaptive responses to punishment (negative feedback) and hyposensitive responses to reward (positive feedback).

2. MALADAPTIVE RESPONSE TO PUNISHMENT

Along with the first observations in the literature on cognitive function in depression was that patients, as projected by models of learned helplessness (Seligman ME ,1972) demonstrate dysfunctional responses to negative feedback. Beats *et al.* (13) asked depressed patients and control subjects to perform the Tower of London planning task.

The groups completed easy problems equally good, but for complicated problems, depressed patients needed more steps than did control subjects. Significantly, the depressed group was not only bad at planning; in spite of difficulty, both groups answered the same number of problems perfectly. Nevertheless, when patients made an error on a test, their behavior deteriorated immediately, which the researchers coined a “catastrophic response to perceived failure” (Beats BCet al, 1996)

Some successive studies proved this abnormal response to negative feedback (Elliott R, Sahakian, 1996& Steffens DC, 2001), though not all studied underlined the effect (Shah PJ, 1999). The shortfall was revealed to associate with the severity of depression (Elliott R et al, 1996) and to be precise to depressed patients and not patients with schizophrenia, Parkinson's disease, or brain injuries (Elliott R,et al, 1997). Though, every one of these patient groups generally behaved worse than control participants, only depressed patients demonstrated an augmented conditional probability of making an error, given an error on the earlier test. In addition, depressed patients persisted to exhibit an aberrant response to negative feedback, though their overall test behaviour had enhanced (Elliott R,et al, 1997), proffering additional evidence that this deficit is not merely secondary to poor overall behaviour.

Along the lines of Beck's psychological theory of depression (Beck AT, 1979), one reading of these findings is that perceived failure on a task could activate further failure-correlated thoughts, integrating with consequent behaviour. Therefore, patients could be hypersensitive to punishment. Yet, a substituted reading, is that depressed patients failed only to use negative feedback to perk up future behaviour (Elliott R,et al, 1997& Steele JD, et al, 2007) which might give the impression of punishment hyposensitivity. Holmes and Pizzagalli (Holmes AJ et al, 2007) uncovered that, after errors, a population of participants with high scores on the Beck Depression Inventory (BDI) adapted their responses importantly less than those with low scores. Such a failure in later behaviour adaptations could reveal underlying deficits in motivation or performance monitoring or an overall blunted response to strengthening rather than hypersensitivity.

3. HYPOSENSITIVITY TO REWARD

In addition to maladaptive reactions to aversive stimuli, depressed individuals show blunted responses to rewarding information, probably exhibiting a shortfall in the approach- connected or

appetitive system (Bylsma LM, et al, 2008). For instance, McFarland and Klein (23) asked participants to rate their mood before and after a block of puzzles wherein right performance was rewarded with money or the evasion of physical punishment (60 sec with their hand placed in a freezing cooler). The researchers discovered that depressed participants were considerably less happy than healthy control subjects when predicting reward, although no difference in concern to predictable punishment.

What is the effect of this diminished approach system on performance? Henriques et al. (1994&2000) asked participants to carry out a memory task with three conditions: one where correct responses were rewarded, one where incorrect responses were punished, and one without reward or punishment. To probe their results, the researchers used signal detection theory, which supplies orthogonal measures of sensitivity (the ability to remember stimuli) and response bias (the general tendency to respond “yes” or “no”). Though participants with low BDI scores relaxed their response bias in the reward condition, hence maximizing their earnings, subjects with high BDI scores (24) or MDD (Henriques JB, et al, 2000) supported a conventional bias, exhibiting indifference to reward.

More recently, Pizzagalli et al. (Pizzagalli DA, et al, 2005&2009) employed similar signal-detection techniques to investigate the responsiveness to reward of depressed patients. They planned a task in which correct responses to one objective were three times more probable to be rewarded than correct responses to another objective. Healthy individuals maintained a strong preference for the highly rewarded stimulus, while participants with high BDI scores (Pizzagalli DA, 2005) and MDD (Pizzagalli DA, et al, 2009) did not, a shortfall that associated with depressive symptoms (Pizzagalli DA, et al, 2005). These results advocate that depressed patients are less capable to adjust behaviour according to prior strengthening. It is critical to note that, though these studies show the presence of reward-processing deficits in depressed individuals, they cannot demonstrate whether such deficits are fundamental.

Nonetheless, both recovered depressive subjects (28) and girls whose mothers were depressed (29) have exhibited impairments in recognizing emotional expressions. Likewise, recovered depressive subjects have exhibited blunted neural responses to positive stimuli, even while their subjective ratings of such stimuli correspond those of control subjects (McCabe C, 2009). These results, in line with pharmacological studies that have disconnected changes in reward processing from changes in mood (Robinson OJ, et al, 2009&, Roiser JP, et al, 2005), infer an underlying aberrant in emotional processing that could award susceptibility to depression, despite further research is required in this context.

The results reviewed in the previous text make out that depressed individuals respond aberrantly to both punishments and rewards, though some opposing findings have been drawn. These propensities might provoke or aggravate their depression. Particularly, if individuals are not capable to modulate their behaviour in response to reinforcements, they might practice fewer rewards and more punishments, in a self-maintaining ferocious cycle. In the next section, we discuss the likely neural substrates of these aberrations.

It has been identified for more than 40 years that individuals that boost monoamine levels improve some of the symptoms of depression (Coppin A, 1967). This unexpected finding led to the hypothesis that a deficiency in monoamine neurotransmitters is at the origin of the disease (Hirschfeld RM, 2000). Though, there are crucial limitations to this hypothesis, there is now wide consent that each of the three major monoamines—serotonin, norepinephrine, and dopamine—might interfere to the symptoms of depression (Nutt DJ et al, 2008 & Ruhe HG et al, 2007). In parallel, a considerable body of literature has related the function of these monoamines to reinforcement processing. Specifically, controlling serotonin levels through either tryptophan reduction or selective serotonin reuptake inhibitor (SSRI) administration alters how healthy individuals respond to rewards and punishments, regardless any changes in mood.

4. COMPUTATIONAL MODELS

A significant growth in the research of human learning and decision-making is the advent of computational modeling. Neuroscientists and computer scientists have gathered attempts, developing formal mathematical algorithms to offer impending into the nature of brain signals while processing reinforcement stimuli (Dayan P, et al, 1996). This approach has not yet been

used broadly to study depression, but the limited published studies have provided novel projections and a more specific knowledge of the neural computations that might go slanted in MDD.

Though some computational studies have employed a neural network approach (Siegle GJ, et al, 2002), here we concentrate on reinforcement-learning (R-L) modeling. The R-L models, which derived from the artificial intelligence field, describe how an agent learns to maximize reward in a complex and uncertain environment (58). An important concept in R-L is that of prediction error, the difference between expected and actual outcomes. Electrophysiological studies in monkeys convincingly linked prediction errors with phasic activity in midbrain dopamine neurons (59). Later neuro-imaging studies reported prediction error-related responses in a network of regions in the human brain extensively innervated by the monoamine system (56) and implicated in MDD (8), including OFC, ACC, amygdala, VS, and hippocampus.

In one of the first studies using R-L modeling in depression, patients and control subjects played a monetary decision-making task during fMRI (60). Participants had to choose between two cards displayed on a screen, resulting in monetary gain or loss control subjects a probabilistic learning task and found that computationally derived learning rates correlated negatively with self-reported anhedonia: regardless of depressed status, anhedonia was associated with a blunted ability to use reinforcement to alter behavior. These studies demonstrate that computational modeling might be useful not only to understand the causes for depressive symptoms but also potentially to detect, monitor, or assess the disease process. Computational approaches have also shed light on the neurochemistry of depression. Kumar et al. (63) scanned depressed patients and control subjects while they performed a Pavlovian conditioning task, which used water as a primary reinforcer (in thirsty participants). Importantly, the control subjects were scanned both in an unmedicated state and after 3 days of SSRI treatment. Compared with healthy control subjects, medicated depressed patients showed blunted prediction error-related responses (reduced activations and deactivations) in the VS, ACC, retrosplenial cortex, and hippocampus as well as increased responses in the ventral tegmental area. The general blunting pattern observed was consistent with the previous study of this group (60). Interestingly, medicated control subjects showed blunting in a similar network, with a pattern of responses intermediate between unmedicated control subjects and depressed patients.

This result suggests that blunted responses might be partly related to medication, as reported in other studies in healthy volunteers (38,64), although SSRIs do not appear to explain the ventral tegmental area difference. However, there are other explanations for why control subjects given SSRIs might exhibit responses similar to those of depressed patients. One is that short-term antidepressant treatment could have induced a temporary reduction in serotonin transmission through inhibitory presynaptic autoreceptors (65,66). Alternatively, normal reward processing might require a narrow window of serotonin activity, such that any departure from optimal levels would cause dysfunction (67). In other words, the effects of SSRIs on reward learning could depend on the underlying state of the serotonin system; the same dose that helps depressed patients could harm healthy control subjects. Whatever the explanation, the results underscore the important role of serotonin in processing rewards as well as the need to consider the impact of medication in studies of depressed patients. Finally, Dayan and Huys (68, 69) demonstrated how computational modeling might aid our understanding of the specific roles of monoamines in depression. In particular, they suggested that serotonin might inhibit behaviors associated with adverse consequences. Individuals with normal serotonin levels should reflexively inhibit or “prune” choices with poor expected outcomes, thus underexploring negative environments and forming an overly optimistic view of the world. Although such pruning might be adaptive in promoting resilience, any drop in serotonin levels (e.g., preceding the onset of depression) would compromise reflexive inhibition, resulting in the unexpected experience of more negative events. To date, this model has only been tested with computational simulations. It will be important in future work to demonstrate that healthy individuals exhibit greater pruning than depressed patients and that pruning is altered after serotonin treatment.

5. CONCLUSIONS

Depression is characterized by impairments in reinforcement processing and the use of affective information to guide behavior. Research over the past decade has begun to describe the nature and

neural mechanisms of these abnormalities experimentally. Common findings include that depressed patients respond maladaptively to punishment and hyposensitively to reward and that these behavioral phenotypes correspond to abnormal function in a circumscribed network of brain regions, particularly, frontostriatal systems innervated by monoamines. Computational studies have generated testable hypotheses of how such neural signaling and neurochemical abnormalities might underlie the behavioral findings. Together, the evidence suggests that deficits in reinforcement processing are important in the development of MDD and are a worthy target for treatments. We believe that testing this hypothesis through a combination of approaches will provide crucial insight into the mechanisms of this deadly and prevalent disease.

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