ELSEVIER

Physiology & Behavior 86 (2005) 717 – 730

PHYSIOLOGY & BEHAVIOR

Neural bases of food-seeking: Affect, arousal and reward in corticostriatolimbic circuits☆

Bernard W. Balleine *

Department of Psychology and the Brain Research Institute, University of California, Box 951563, Los Angeles, CA 90095-1563, United States

Received 28 July 2005; accepted 25 August 2005

Abstract

Recent studies suggest that there are multiple 'reward' or 'reward-like' systems that control food seeking; evidence points to two distinct learning processes and four modulatory processes that contribute to the performance of food-related instrumental actions. The learning processes subserve the acquisition of goal-directed and habitual actions and involve the dorsomedial and dorsolateral striatum, respectively. Access to food can function both to reinforce habits and as a reward or goal for actions. Encoding and retrieving the value of a goal appears to be mediated by distinct processes that, contrary to the somatic marker hypothesis, do not appear to depend on a common mechanism but on emotional and more abstract evaluative processes, respectively. The anticipation of reward on the basis of environmental events exerts a further modulatory influence on food seeking that can be dissociated from that of reward itself; earning a reward and anticipating a reward appear to be distinct processes and have been doubly dissociated at the level of the nucleus accumbens. Furthermore, the excitatory influence of reward-related cues can be both quite specific, based on the identity of the reward anticipated, or more general based on its motivational significance. The influence of these two processes on instrumental actions has also been doubly dissociated at the level of the amygdala. Although the complexity of food seeking provides a hurdle for the treatment of eating disorders, the suggestion that these apparently disparate determinants are functionally integrated within larger neural systems may provide novel approaches to these problems.

Keywords: Goal-directed action; Habit learning; Instrumental conditioning; Pavlovian conditioning; Incentive learning; Motivation; Striatum; Thalamus; Prefrontal cortex; Amygdala

1. Introduction

There has been a recent trend towards identifying the processes involved in obesity with those associated with addictive behavior generally and with drug addiction in particular. For example, in a recent series of papers, Volkow and colleagues have established that binding at the dopamine D2 receptor in obese subjects, i.e., those with a body mass index over 30, is reduced in similar fashion to that of individuals addicted to drugs of abuse [119–122]. A feature of these, and similar [24], accounts is that, often in the interests of a simple story, they focus on one factor, brain dopamine, as the causal factor, not just in pathological food intake but in its sequelae, notably in food seeking or pursuit. The operation of the reward system is commonly argued to link intake and pursuit and, indeed, since the discovery of self-stimulation, students of neuroscience have felt strongly predisposed to the view that there is a central reward system in the brain, that it is monolithic and that it involves midbrain dopaminergic neurons and particularly their projection via the medial forebrain bundle to limbic structures in the ventral forebrain [61,87,137].

It has appeared, therefore, to be a reasonable leap to propose that pathologies of brain dopamine are associated, more or less directly, with pathologies of the 'reward system' and so with pathological food seeking [44]. Indeed, evidence that, in addition to reduced D2 receptor binding, drug addicts have increased genetic variation associated with the D2 receptor has raised the specter of a 'reward gene' [25,26]. Of course, it is equally possible that this evidence points to a corollary of addiction rather than its efficient cause. But these issues aside, the real problem with this approach is that it over-simplifies our

☆ The preparation of this manuscript was supported by NIMH grant #56446.
* Tel.: +1 310 825 7560 (office), +1 310 825 2998 (lab); fax: +1 310 206 5895.
E-mail address: balleine@psych.ucla.edu.

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doi:10.1016/j.physbeh.2005.08.061

understanding of the complex nature of the processes that contribute to both normal and abnormal food seeking. A number of recent papers have, as a consequence, unnecessarily conflated the processes that contribute to the compulsive pursuit of food with those that control goal-directed actions [63,66,85] and still further with those that control responses elicited by stimuli associated with food [74]. Although the operation of these processes objectively affects the rate of food seeking, recent evidence suggests that they each have distinct determinants. This review will attempt to tease these various influences apart with reference to recent research that has identified not one but potentially five 'reward' or 'reward-related' processes in the brain; that is to say, five systems that function to influence food seeking either directly, through learning, or indirectly, by modifying performance.

2. Reward and reinforcement

The recent literature concerning drug seeking in addicts has focused attention on the compulsive or habitual nature of these responses revealed particularly in their persistence, even in the face of sometimes quite extreme negative consequences, and their sensitivity to drug-related cues, an observation that has informed various theories of relapse [29,54,95,107]. Many of the ideas that have been expressed in these recent papers have their root in now classical theories of habit learning, associated most notably with Hull [76], that explain the acquisition of actions instrumental to gaining access to rewarding events in terms of the operation of a stimulus–response/reinforcement (S–R) architecture. From this perspective, addictive drugs reinforce or strengthen associations between contiguously active sensory and motor processes allowing the sensory process subsequently to elicit the motor response in a manner that is no longer regulated by its consequences.

Although it is a straightforward matter to apply these ideas to drug addiction, it is much less clear whether and to what degree they apply directly to activities associated with natural rewards like food. Although S–R theorists regarded food seeking, like compulsive drug seeking, as a form of habit, what evidence there is for this claim has really only emerged relatively recently in studies assessing the effect of post-training reinforcer devaluation on instrumental performance. For example, Holman [75] was able to show that lever press responses in thirsty rats reinforced on an interval schedule by access to a saccharin solution were maintained in extinction even after the saccharin had been devalued by pairing its consumption with illness. It is important to recognize how maladaptive the lever pressing was in Holman's rats. Although the pairing with illness resulted in the rats no longer consuming or even contacting the previously palatable (but now poisonous) saccharin, their subsequent extinction performance on the lever continued at a rate comparable to that of rats for which the saccharin was not devalued.

Several years later in a replication of Holman's experiment, Adams and Dickinson [1] found, in contrast, that, when lever pressing in hungry rats was reinforced either continuously or on a ratio schedule by sugar pellets, devaluation of the pellets strongly attenuated subsequent performance on the lever. Although several features of the two studies differed, Dickinson, Nicholas and Adams [51] later showed that interval schedules of reinforcement were particularly apt to produce habitual responses; i.e., responses that are no longer dependent on the current value of their consequences; when previously reinforced by sugar on a ratio schedule lever pressing was sensitive to devaluation whereas when reinforced on an interval schedule it was not. These findings provide direct evidence that, over and above a habit or S–R process, the performance of instrumental actions can also be goal-directed; it can reflect encoding of the relationship between action and outcome. Furthermore, they show that both processes can be engaged depending on the relationship between instrumental performance and reward delivery. When reward delivery is constrained by time so that changes in the rate of performance have little if any effect on the rate of reward, actions tend to become habitual. When rate of reward is proportional to the rate of performance, however, actions tend to be goal-directed. It is also worth noting that the fact that the same event, sucrose in this case, could serve both as the goal of an action and to reinforce S–R associations must raise immediate questions regarding the notion of single or monolithic 'reward' system responsible for all changes in instrumental performance.

Recent experimentation has only made these questions more pointed. For example, several recent studies have found evidence that damage to the lateral region of the dorsal striatum (DLS) renders rats incapable of developing simple S–R solutions to various maze discrimination problems suggesting that this region may be important in the formation of associations of this kind [46,92,99]. Anatomically, the DLS appears to be well suited to this functional role, maintaining strong connections with sensorimotor regions of the neocortex [93]. Furthermore, this region receives a dense projection from the midbrain dopaminergic neurons that electrophysiological studies suggest may play a reinforcing role, modulating plasticity between converging cortical afferents [105]. In a recent study we attempted to provide more direct evidence for the involvement of the DLS in habit learning by assessing the effect of cell-body lesions of this area on the acquisition and performance of instrumental actions trained on an interval schedule of reinforcement as well as on sensitivity of performance to the devaluation of the instrumental outcome [134]. The strong view that the DLS mediates S–R learning predicts that acquisition and subsequent performance of actions reinforced on interval schedules should be severely attenuated. Against this prediction we found that acquisition was normal and subsequent performance was only moderately affected by the lesion. The most striking effect was, however, the change in the influence of outcome devaluation. Whereas the instrumental performance of sham-lesioned controls showed no sensitivity whatever to outcome devaluation by conditioned taste aversion, replicating previous findings, the DLS-lesioned group showed clear sensitivity to this treatment [134]. This result was specific to the DLS; lesions of the dorsomedial striatum did not increase sensitivity to outcome devaluation. The lesions of the DLS, therefore, effectively abolished