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Short Communication

Food reinforcement

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Abstract

The reinforcing value of food, measured by how hard someone is willing to work to obtain food, is influenced by food palatability, food deprivation and food variety, and may be a more powerful determinant of food intake than hedonics or liking. The reinforcing value of food is mediated in part by dopaminergic activity. Genotypes that influence dopamine transport and the density of dopamine D2 receptors interact with food reinforcement to influence eating behavior, and D2 receptor genotypes may influence food reinforcement and weight gain after smoking cessation. Inhibition of dopamine transport increases brain dopamine concentrations, which may influence weight gain after smoking cessation and can reduce energy intake in obese adults.

Keywords: Food reinforcement; Dopamine; Deprivation

Food reinforcement

Food is a primary reinforcer, i.e. no learning is required for food to motivate behavior. Food is also a very powerful reinforcer, and in some circumstances may be more reinforcing than drugs of abuse. For example, heroin dependent baboons reduced their responding for heroin when they had to choose between heroin and food and opportunities to obtain heroin or food were decreased, while responding for food was maintained (Elsmore, Fletcher, Conrad, & Sodetz, 1980). Food reinforcement is an important determinant of human food ingestion (Epstein & Saelens, 2000). The reinforcing value of food describes how hard someone is willing to work to gain access to food, and is usually defined in terms of how many responses will be made on a particular schedule of reinforcement to obtain food (Epstein & Saelens, 2000). Reinforcing value can be measured when food is the only alternative, or the relative reinforcing value can be measured when food or different types of foods are alternative choices in a concurrent schedule paradigm. This paper will present research from our laboratory that has focused on factors that influence food reinforcement, the influence of food reinforcement on intake, and dopamine and food intake in humans.

Research from our laboratory has focused primarily on the relative reinforcing value of food using choice methodologies, rather than how hard someone will work only for access to food. This is done for three reasons. First, there is ecological validity to a choice paradigm, since in the real world eating is almost always a choice and studying only one option may not provide a realistic appraisal of how reinforcing food is. Second, when there is only one option, subjects may continue to respond for food because there is nothing else to do, and they are seeking stimulation. Third, the value of many reinforcers depends in part on the alternatives that are available, in addition to the value of food. Something that may seem very reinforcing in isolation may not be as reinforcing when an alternative is available.

Factors that influence food reinforcement and choice of food

In a choice situation the reinforcing value depends on the reinforcing value of the alternatives as well as the constraints on access to food. For example, when given a choice between preferred versus less preferred foods, subjects initially chose to work for the more preferred food, but as constraints for this food increased, adult and

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child subjects chose to work for the less preferred food (Lappalainen and Epstein, 1990; Smith and Epstein, 1991). Likewise, when provided a choice between preferred snack foods versus fruits and vegetables or pleasurable sedentary activities, subjects chose snacks, but shifted their choice to the alternatives as the work needed to gain access to snacks increased (Goldfield and Epstein, 2002). These studies illustrate two important principles of behavioral choice theory. First, increasing the behavioral cost to obtain a food reduces responding for that food. Second, increasing the cost to obtain a food can increase responding for a different food, and the commodity that is associated with increased responding is called a substitute. The substitute may not be as reinforcing but can substitute when the cost to obtain the alternative becomes too high.

Food also may have complementary relationships with other behaviors. When access to a behavior decreases, and a different behavior also decreases, this is evidence for a complementary relationship. For example, food intake is often associated with television watching, and reducing television watching can reduce food intake (Epstein, Roemmich, Paluch, & Raynor, 2005). Thus, food intake can be reduced by increasing the cost to obtain the food, providing reinforcing alternatives to eating, or reducing access to variables that are reliably associated with eating.

Motivation to eat is influenced by food deprivation (Raynor & Epstein, 2003). The increase in food consumption that occurs as a function of deprivation may be particularly relevant for weight regulation. Diets usually require some deprivation, which may increase motivation to eat, which may be a particular problem for obese persons. When non-dietary restrained female college students were given a choice between food and a variety of pleasurable sedentary behaviors, the obese worked harder for access to food than the non-obese (Saelens and Epstein, 1996). Food variety also increases energy intake (Raynor and Epstein, 2001) and the reinforcing value of a variety of food in comparison to repeated presentation of the same food (Myers & Epstein, 2002). Introducing a new food is associated with an increase in motivated responding to obtain food (Epstein, Saad, Handley, Roemmich, Hawk & McSweeney, 2003).

Food reinforcement is a powerful motivator to eat and may be a more powerful determinant of eating than the subjectively rated liking or hedonic value of food. Food reinforcement is objectively measured by how much work a subject will engage in to obtain food. Liking (or related concepts such as palatability or hedonics), is subjectively measured using rating sheets or questionnaires and is separate from the reinforcing value of food, and we have shown that increasing the reinforcing value of food in humans via food deprivation does not alter the liking of selected foods (Epstein, Truesdale, Wojcik, Paluch, & Raynor, 2003). In smokers, the reinforcing value of food is a better predictor of food intake in the laboratory setting than is subjectively rated food hedonics (Epstein, Wright, Paluch, Leddy, Hawk and Jaroni, 2004a).

Dopamine and food reinforcement

Dopamine is important in understanding food reinforcement. Food reinforcement's action in the brain depends in part upon the release, transport (reuptake), and receptor binding of synaptic dopamine. Positive reinforcers such as food and drugs of abuse stimulate the release of brain dopamine (Berridge, 1996; Robinson and Berridge, 2000), which acts at two general classes of dopamine receptors termed D1-like and D2-like (Sibley, Monsma, McVittie, Gerfen, Burch and Mahan, 1992). Dopamine is released in anticipation of eating and binds to D2 receptors in the lateral hypothalamus, motivating animals to search for food (Kiyatkin and Gratton, 1994). Dopamine is released again when food is consumed and its activity in the nucleus accumbens rewards the feeding behavior (Kiyatkin & Gratton, 1994). Dopamine cell activity is responsive to environmental stimulation in proportion to the saliency of the stimulus (Horvitz, 2000), and dopamine release may accompany the cues that anticipate reinforcement, so that over time with repeated ingestion dopamine release shifts from food delivery (i.e. the pleasure of ingesting food) to cues associated with wanting food (for example, the sight and smell of food) (Volkow, Wang, Fowler, Logan, Jayne and Franceschi, 2002), increasing the incentive salience of food (Berridge, 1996).

We have been interested in two genotypes that may influence dopaminergic activity and food reinforcement. The dopamine transporter gene (*SLC*6A3) codes for a dopamine transporter protein (DAT) that reduces synaptic dopamine levels and the magnitude and duration of dopamine receptor activation (Bannon, Poosch, Xia, Goebel, Cassin and Kapatos, 1992). There are three common polymorphisms: 9/9, 9/10 and 10/10. The DAT 10-repeat allele may be associated with increased dopamine transporter density and therefore to lower levels of postsynaptic dopamine (Heinz, Goldman, Jones, Palmour, Hommer and Gorey, 2000) when compared with polymorphisms containing a 9 allele. Recent data suggest that DAT may have a role in body weight regulation. Elevated levels of DAT have been found in obese rats (Figlewicz, Patterson, Johnson, Zavosh, Israel and Szot, 1998), and food deprivation (Patterson, Brot, Zavosh, Schenk, Szot and Figlewicz, 1998) and food restriction (Bello, Sweigart, Lakoski, Norgren, & Hajnal, 2003) increase DAT mRNA in rats. In humans, the DAT10/10 allele has been associated with obesity in African American but not Caucasian smokers (Epstein, Jaroni, Paluch, Leddy, Vahue and Hawk, 2002).

The dopamine D2 receptor gene (*DRD*2) has three allelic variants (A1/A1, A1/A2, and A2/A2). The A1 allele is associated with fewer D2 receptors than the A2 allele (i.e. reduced brain dopamine signaling) (Noble, Noble, Ritchie,