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Food deprivation and emotional reactions to food cues: implications for eating disorders

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Abstract

Two studies examined emotional responding to food cues. In experiment 1, normal college students were assigned to 0-, 6- or 24-h of food deprivation prior to presentations of standard emotional and food-related pictures. Food deprivation had no impact on responses elicited by standard emotional pictures. However, subjective and psychophysiological reactions to food pictures were affected significantly by deprivation. Importantly, food-deprived subjects viewing food pictures showed an enhanced startle reflex and increased heart rate. Experiment 2 replicated the food deprivation effects from experiment 1, and examined participants reporting either a habitual pattern of restrained (anorexia-like) or binge (bulimia-like) eating. Food-deprived and binge eater groups showed startle potentiation to food cues, and rated these stimuli as more pleasant, relative to restrained eaters and control subjects. The results are interpreted from the perspective that startle modulation reflects activation of defensive or appetitive motivation. Implications of the data for understanding eating disorders are considered.

Keywords: Emotion; Eating disorder; Anorexia; Bulimia; Startle

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1. Introduction

The Russian physiologist Pavlov stated early in the last century that 'The most essential connection between the animal organism and the surrounding world is that brought about by certain chemical substances which constantly enter into the composition of the given organism, i.e. the food connection.' (Pavlov, 1909). Subsequently, Pavlov's (1927) classic demonstration of conditioning used the salivary gland reflex of the dog: After a neutral cue was repeatedly paired with food, the gland showed increased flow to subsequent presentations of the cue. Humans may experience the same conditioned salivation when seeing a sign advertising a 'bakery'. Such conditioned reactions to salient food cues (which include other responses — somatic and autonomic) are useful adaptations that generally facilitate survival; however, learning may also modulate appetitive response patterns in less salutary ways that can lead to eating disorders and threaten health.

Since Pavlov's early work, considerable animal and human research has supported the role of conditioning in mediating appetitive reflex modulation and motivation for food intake (e.g. Capaldi et al., 1983; Laberg et al., 1991; Lappalainen et al., 1994; Wooley and Wooley, 1973). In the present study, we sought to investigate normal and pathological forms of food motivation by examining verbal, physiological, and behavioral responses to salient food cues in normal participants systematically deprived of food, and in participants who report habitually deviant eating patterns.

1.1. Motivational priming and the startle reflex

Drawing on ideas developed by Konorski (1967), Lang and coworkers (Lang, 1995; Lang et al., 1990, 1992, 1997) proposed that affects are determined by the individual's motivational state. Two brain circuits are postulated, one determining appetitive responding (e.g. approach, attachment, consumption) and positive, pleasant affects, and the other prompting defense (e.g. avoidance, fight-flight) and unpleasant affects. These systems can be co-active (see Miller, 1944), and the motive significance of cues may be modified by experience. However, emotion and mood (pleasant or unpleasant) at any given time are determined by the dominant motive system (appetitive or defensive).

A feature of this biphasic view is that an individual's affective state (positive or negative valence) can be inferred by evoking a reflex that is consistent or inconsistent with the dominant motive system. Thus, several investigations (e.g. Vrana et al., 1988; Bradley et al., 1990, 1991; Cook et al., 1991) have shown that the defensive startle reflex is potentiated when it is elicited in the context of an unpleasant foreground stimulus. Pleasant foregrounds, on the other hand, prompt an appetitive emotional state that is inhibitory of the defensive startle reflex.

Animal researchers have also consistently shown startle reflex augmentation in an aversive experimental context (see Davis, 1989; Davis et al., 1987). Furthermore, these investigators have elegantly delineated the neural circuitry underlying startle