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Cue reactivity and regulation of food intake

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Abstract

A robust finding in eating research is the so-called counterregulation in restrained eaters. This means that while normal subjects eat less during a taste test, after they consumed a preload, restrained eaters consume more. An explanation is that food exposure causes stronger physiological preparatory reactivity in the restrained eaters. This reactivity is experienced as craving and leads to an increased food intake. To test this theory, 46 high and low restrained eaters were exposed to food or soap, while physiological measurements were made. Afterwards, the subjects performed a taste test, during which food intake was secretly measured. Unrestrained eaters showed an increase in heart rate, gastric activity, and saliva during food exposure; however, restrained eaters did not. Gastric activity significantly correlated with food intake. Group or exposure type did not influence food intake. It can be concluded that unrestrained eaters prepared for food intake, whereas the restrained eaters did not. A possible explanation is that restrained eaters used cognitive suppression to block physiological responding, thereby controlling their food intake. © 2001 Elsevier Science Ltd. All rights reserved.

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1. Introduction

A large group of people is struggling with their food intake, trying to restrict it, but often fail to do so. Herman and Mack (1975) performed an elegant experiment, using the so-called preload paradigm, in which this phenomenon is shown. In that experiment, unrestrained eaters showed normal regulation of their eating behavior; after they ate a preload (milkshake), they ate less ice cream during a subsequent taste test than when they ate no preload. Restrained eaters,

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however, defined as subjects who want to restrict their food intake, consumed less during a taste test when they did not eat a preload, but they consumed more during the taste test when they did eat a preload. This illogical behavior is called counterregulation and Herman and Mack's experiment initiated a large body of research. Restrained eaters repeatedly showed abnormal regulation of food intake (for an overview, see Boon, 1998). How can this eating pattern of restrained eaters be explained? A widespread cognitive explanation stems from the boundary model (Herman & Polivy, 1984). In this model, biological boundaries determine when a person feels hungry or satiated and thereby when a person starts and stops eating. Restrained eaters have another self-imposed boundary, marking their maximum desired consumption, the so-called diet boundary. As long as this boundary is not broken, the restrained eater succeeds in restricting food intake. When this diet boundary is broken, for example, after eating the preload, restrained eaters have disinhibitory thoughts, like "I've already blown my diet, I might as well continue to eat," and start overeating. However, restrained eaters also showed abnormal regulation after exposure to the sight, smell, or thought of food, without actually eating it (Fedoroff, Polivy, & Herman, 1997; Jansen & Van den Hout, 1991). These findings cannot be explained with the disinhibitory thoughts of the boundary model; the "preload" was not consumed, and, thus, no diet rule was broken. Furthermore, no evidence of disinhibitory thoughts was found when restrained eaters were instructed to think aloud during the preload condition (Jansen, Merckelbach, Oosterlaan, Tuiten, & van den Hout, 1988).

An alternative explanation for the counterregulation phenomenon stems from the cue reactivity theory. This theory states that when a person regularly has eating binges, and these binges are reliably preceded by certain cues (e.g., the sight, smell and taste of the food, environment, cognitions, and emotions), these cues become predictors of the start of a binge. Exposure to these cues induces conditioned physiological reactivity, which can prepare the person for the intake of food. In normal subjects, physiological responses to food cues are widely documented and called cephalic phase responses (Mattes, 1997; Nederkoorn, Smulders, & Jansen, 2000). Binge eaters are expected to show even larger cephalic phase responses because the enormous amount of food intake during a binge threatens the homeostatic balances of the body, and anticipation is more important compared to normal food intake. In addition, more intense unconditioned stimulus, in this case the food intake, strengthens the conditioning. The cue reactivity is experienced as craving for the food and triggers an eating binge (Jansen, 1994, 1998; Wardle, 1990). The theory originates in the addiction field, where increased psychophysiological reactivity and craving are found when the substance abuser is exposed to drug/alcohol cues (Glautier & Remington, 1995; Robbins, Ehrman, Childress, & O'Brien, 1997), and studies suggest that craving for a substance is a classical conditioned response (Drummond, Tiffany, Glautier, & Remington, 1995). At least a part of the restrained eaters shows an eating pattern of dieting and overeating, which resembles the eating pattern of subjects with eating binges. Applied to the preload paradigm, the cue reactivity model of binge eating states that exposure to a preload, either by seeing and smelling or actually eating it, will elicit conditioned physiological responses and craving in the restrained eater. This, in turn, leads to increased food intake and counterregulation. From the model, it also follows that, when not exposed to the preload or other binge cues, the restrained eater is able to control food intake and eats less.

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