Eating rate in the treatment of eating disorders and obesity

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The cause of eating disorders and obesity

Outcome of the treatment of anorexia and bulimia nervosa remains suboptimal [1], possibly because it is assumed that eating disorders are caused by anxiety disorders [2] although interventions targeting these disorders are not effective [3]. An alternative approach is based on the observation that eating disorder symptoms emerge in healthy people who volunteered [4] or were forced [5] to starve. Most patients have a history of reduced food intake; bulimic behavior, for example, is elicited by food deprivation [4,5]. Starvation changes eating behavior [4] and we hypothesize that the change mediates between physical state and cognitive state in the patients. Similarly, obesity may causally depend a change in eating behavior [6].

Experimental control of eating rate

Recording weight loss of a plate during meals yields a measure of cumulative food intake. Curves for eating rate displayed on a screen provide visual feedback during meals and subjects can follow these curves, because they see their eating rate appearing on the screen during meals. This method makes it possible to increase or decrease eating rate experimentally. Display of a scale, ranging from 0 - 100, on the screen allows simultaneous rating of satiety [7]. On the hypothesis that eating behavior is a cause of some aspects of eating disorders and obesity, this method is used to treat these conditions.

Linear eating: a risk for eating disorders and obesity

The cumulative food intake was fitted to a quadratic equation: $y = ax^2 + bx + c$, where a = change in the slope of the curve over time, b = initial rate of eating, and c = food intake at the start of the meal, i.e., 0. The cumulative satiety curve was fitted to a two-parameter sigmoid curve: $y = \alpha/(1+e^{-(x-x\ )/\beta})$, where $\alpha = 100$, i.e., the maximum of the satiety rating scale, β = steepness of the curve and x = time at which satiety has reached the half maximal value, i.e., the inflection point of the curve. These models fitted the experimental data satisfactorily; all square correlations were $r^2 \geq 0.99$. The averages of observed values are reported as medians; ranges and details of statistical analysis are omitted for clarity of presentation.

Subjects ate ordinary food (400kJ, 4.5 g protein, 18 g fat and 15 g carbohydrate/100 g) at lunchtime. Three weekly tests of 30 normal weight (body mass index, BMI = 22.2 kg/m^2), healthy men and women (aged 21.2) showed that intake and satiety is inter-individually stable. Forty women (similar BMI and age) were divided into linear; a = -0.1 (n=30) and decelerated; a = -2.4 (n=17) eaters in a control meal unassisted by visual feedback. Linear eaters ate at an initially lower rate (b = 29 g/min) than decelerated eaters (b = 44.2 g/min), but food intake (292 vs 288 g) and meal duration (10.9 vs 9.3 min) were similar. Satiety fitted the sigmoid curve in both groups; the inflexion point visible only among decelerated eaters, who reached a higher level of satiety (65.2) than linear eaters (51.4).

The women were challenged to eat 40% more food than they ate in the control test in the same period of time; this was achieved by asking them to follow the curve that they had generated in the control test and that was displayed on the screen. Decelerated eaters failed to adapt to the higher rate, ate less food (217 g) and reached a lower level of satiety (51.8). Linear eaters, however, ate more food (355 g) and reached about the same level of satiety (52.2). When challenged to eat 30% less food than they ate in the control test in the same period of time, the decelerated eaters ate about the same amount of food (274 g) and reached about the same level of satiety (64.2). Linear eaters, however, ate less food (249 g) and reached a higher level of satiety (60.2).

The results suggest that linear eaters, who eat at a constant rate, are unable to adjust their intake when challenged to eat at a rate that differs from their baseline rate. This is referred to as disinhibition, which is shown by those at risk for both eating disorders (too little food when eating slowly) and obesity (too much food when eating quickly). The default rate of eating may be decelerated, which may provide protection from the effect of disinhibition and therefore disordered eating.

Seventeen linear eaters (a = -0.2; b = 26.4), who overate by 16% in a test of disinhibition, practised eating at a decelerated rate three times/week during eight weeks by adapting to a decelerated curve; the women put food on their plate and an algorithm in the computer generated a decelerated curve with a = -1.7 and b = 53. After training, they ate about the same amount of food (261 vs 273 g) in about the same time (11.2 vs 12 min) as before training. However, they maintained a decelerated rate of eating (a = -3.9; b = 43) in the absence of visual feedback during the meal, and they did not overeat when challenged in a test of disinhibition (-6%).

These results suggest that linear eating, a possible risk for disordered eating, can be reduced by practicing eating at a decelerated rate [8], opening the possibility of using eating rate to treat eating disorders and obesity.

Treating eating disorders and obesity

A randomised controlled trial showed that the method described above had a significant effect on outcome, that an estimated 75% of 168 patients with anorexia, bulimia or unspecified eating disorder went into remission in on average 14 months and that 90% of 83 patents treated to remission remained free of symptoms in on average one year [9]. An interim analysis of an ongoing randomised controlled trial adapting the same principles to the treatment of childhood obesity showed that 31% of 36 patients lost > 0.5 BMI standard deviation scores (10-15 kg) compared to 11% among children treated as usual ($P < 0.001$).

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