1. Introduction

It was suggested long ago that restrained eating is a cognitively controlled eating style associated with obesity [1]. Restrained eaters are concerned with how they eat and what they eat and they are thought to be more sensitive to external cues than non-restrained eaters [1]. If challenged by changes in these cues, restrained eaters lose control, enter a state of disinhibition and overeat, which may put them at risk of becoming overweight [1]. The postulated relationship between restrained eating and obesity has since been modified and it is now hypothesized that those who score high on rating scales of restrained eating are at risk of becoming obese [2]. While questions concerning the proper measurement remain [3–6], restrained eating is still considered a risk factor for obesity [78]. In addition, restrained eating has been suggested to be a risk for the disordered eating associated with binge eating and bulimia nervosa [910] and also anorexia nervosa [1112].

A recent review pointed out that it is not yet known whether the many endocrine concomitants to restrained eating are cause or consequence of the altered pattern of eating [13]. Similarly, while restrained eating is thought to be a “self-imposed resistance to the internal and external cues that regulate eating behavior” [9] we don’t know whether restrained eating is a cause or consequence of the pattern of eating behavior. The pattern of eating can be determined by the cumulative food intake curve, which has been modeled by a quadratic equation $y=ax^2+bx+c$; where $a$=change in the slope of the curve over time, which equals the initial eating rate and $c$=food intake at the start of the meal, i.e., $0$ [1417]. Subjects can be divided into decelerated ($a<0$) and linear ($a=0$) eaters, with $b$-value higher among decelerated than linear eaters [17]. Thus, decelerated eaters start eating at a higher rate than linear eaters and their rate of eating decreases gradually during the meal, but there is no difference in total intake among decelerated and linear eaters and although meal duration is somewhat longer among linear eaters, the difference is not statistically significant [17]. Linear eaters score higher than decelerated eaters on a scale of restrained eating [1718] and if asked to eat at a higher rate, they overeat; we have suggested that this challenge is a behavioral test of disinhibition [17]. In contrast to linear eaters, decelerated eaters do not overeat in this test; in fact they eat less and may therefore be protected from becoming obese and possibly also from developing disordered eating [17].

We have found that as the rate of eating normalizes, mental functions such as depression, anxiety and obsessionalism normalize in patients with anorexia nervosa [19]. The pattern of eating rather than the actual body weight may be the important factor in changing the mental state in anorexia [20]. In the present study, we extend this hypothesis by examining if the restrained eating of linear eaters is an effect of eating at a linear rate that can be modified by practicing eating at a decelerated rate.

2. Methods

2.1. Subjects

Thiry-four women were recruited from a nearby college campus. Their median (range) body mass index was 22.1 (19.8–24.4) kg/m² and their age was 21.1 (19.1–24.2) years. They were selected from a bigger group for eating at a constant rate, i.e., $a=0$ in the cumulative food intake.

The text continues with further details about the study's methodology and findings.
curve tested using Mandometer® [17] (see Apparatus below) and are therefore referred to as linear eaters. All women completed a questionnaire to ensure that they were healthy. We study women, because ultimately, our aim is to understand eating disorders, which mainly affect women. The present study, however, does not specifically address the issue of eating disorders.

2.2. Apparatus

The women were tested for food intake by use of Mandometer® [17], which is a scale (IDEMA 750, IDEMA, Gävle, Sweden) connected to an IBM compatible PC, that reads the scale every 2 s with an accuracy of 1 g. The woman eats from a plate on the scale and the computer records the weight loss of the plate.

Mandometer® has a 15” TFT touch screen and the woman is asked to follow curves which are displayed on the screen during the meal. This is possible because she sees her own rate of eating appearing on the screen during the meal. Thus, Mandometer®, which is a development of the original methods of Jordan [21], Pudel [15] and Kissileff [22], allows experimental manipulation of eating rate [17].

2.3. Procedure

The women filled in the Dutch Eating Behavior Questionnaire (DEBQ), as a measure of restrained, emotional, and external eating [23]. They were challenged with a test of disinhibition 3–5 days later [17]. In this test, the women were asked to eat at an increased rate by placing 40% more food on their plate compared to the amount they had eaten in the first test, which discriminated them as linear eaters. They were requested to follow the cumulative curve of food intake they had generated in the first test, which was displayed on the touch screen. Linear eaters are able to comply with this procedure [17].

Seventeen randomly selected women were asked to follow a decelerated eating curve displayed on the touch screen in a training meal three times every week during eight weeks. The women put 350 (280–460) g of food on their plate in these tests and an algorithm in the computer generated a corresponding decelerated curve in which $a=-1.7$ ($-2.9$ to $-1.5$) and $b=53$ (51–55).

Seven days after the training period, the women were tested without assistance from the training curves, challenged with the test of disinhibition 3–5 days later and asked to fill in the DEBQ. The 17 other women were also re-tested but they did not practice eating during the intervening eight weeks.

It is sufficient to test subjects once, because eating behavior is individually stable when tested under the present conditions [17].

The women ate various dishes of normal food (Findus, Bjus, Sweden, 400 kJ, 4.5 g protein, 18 g fat and 15 g carbohydrate/100 g) in all meals. The food was prepared fresh before each meal and served at a temperature of 65 °C after heating in an oven.

The procedures were approved by the ethics committee of the Karolinska Institute and the women gave written consent to participate.

2.4. Statistical analysis

Observed values are presented as box plots in the figures. ANOVA for repeated measures was used for analyzing effects of experimental condition and eating pattern; $t$-tests were subsequently used for within and between group comparisons. STATISTICA 6 (StatSoft Inc, Tulsa, TX) was used for the analyses.

3. Results

3.1. Linear eaters turned decelerated

There was a significant effect of practicing eating at a decelerated rate on the rate of deceleration [$F(1,64)=21.7, p<0.001$] and on the initial eating rate [$F(1,64)=18.6, p<0.001$] (Fig. 1). However, food intake [$F(1,64)=1.69, ns$] and the duration of the meal [$F(1,64)=0.1, ns$] were not significantly affected (Fig. 1).

Within group comparison showed that there was a significant increase in the rate of deceleration [$t(16)=5.6, p<0.001$] and the initial eating rate [$t(16)=-5.3, p<0.001$] after training. These measures were unaffected in the women in the no training group ($t$-values not shown).

3.2. Reversal of the effect of disinhibition

There was a significant effect of practicing eating at a decelerated rate on the effect of disinhibition [$F(1,64)=5.56, p=0.021$] (Fig. 2). All women overate when asked to eat at a higher rate before training [$t(16)=3.9, p<0.001$] and [$t(16)=4.9, p<0.001$], for the training and no training group, respectively (Fig. 2). This effect of disinhibition was eliminated by eight weeks of practice to eat at a decelerated rate; women who had

![Fig. 1. Rate of deceleration (a), initial eating rate (b), food intake and meal duration in women before and after training to eat at a decelerated rate for eight weeks and in women who were not trained. There were 17 women in each group and the values are box plots. *p<0.001, t-test after ANOVA.](image)

![Fig. 2. Food intake in a test of disinhibition, e.g., eating 40% more food than in the control condition (ER+), in women before and after training to eat at a decelerated rate for eight weeks and in women who were not trained. There were 17 women in each group and the values are box plots. *p<0.001, t-test after ANOVA.](image)
developed a decelerated pattern of eating ate less food in the test of disinhibition \([t(16)=3.2, p<0.005]\) (Fig. 2). The effect of disinhibition was unaffected among the women in the no training group; they still overate in response to disinhibition \([t(16)=-4.2, p<0.001]\) (Fig. 2).

### 3.3. Reduction of restrained eating

There was a significant reduction of restrained eating \([t(16)=4.98, p<0.001]\) and external eating \([t(16)=4.01, p=0.001]\), but not emotional eating \([t(16)=0.29, ns]\) after practicing eating at a decelerated rate (Fig. 3). These measures were unaffected in the no training group \(r\)-values not shown) (Fig. 3).

### 4. Discussion

A recent review pointed out that the concept of dietary restraint has dominated “... the understanding of eating behaviour,” and that disinhibition, which was suggested to be “a set of endurable characteristics”, plays an important role as well [24]. Thus, restrained eating and disinhibition are thought to be trait-related determinants of eating behavior. However, the same review raised the question of cause and effect, because disinhibition has been found to diminish with weight loss, behavioral therapy, drugs or surgery [24]. An endurable trait should probably resist such interventions. In addition, restrained eating and disinhibition are thought to be interrelated, although this relationship can have a variable outcome on food intake: someone who scores high on a scale for restrained eating may eat more or less food depending upon several external factors which are thought to influence disinhibition [25]. Variability in behavioral outcome and in response to available questionnaires has raised concerns about concept validity [26,27]. However, the critique was rebutted by a creator of one of the questionnaires, who argued that restrained eating is a matter of the “desire to eat” rather than the “amount of food eaten”. In fact, “how much the restrained eater eats is irrelevant” the important measure is “... how much the restrained eater does not eat”, which, surprisingly, was “impossible to determine”, at least by a direct measure [25,28]. This situation was interpreted to show that “a restrained eater might (a) eat a lot, (b) eat more than required ... but (c) still eat less than desired... “. calling for development of “a more complex notion of how dietary restraint interacts with circumstances that affect food intake” [25]. This analysis departs from the more common, 500 year old, scientific tradition “Entia non sunt multiplicanda praeter necessitatem”, in other words, to “make everything as simple as possible, but not simpler.”

The results reported here raise the possibility that restrained eating is a cognitive effect of the pattern of eating behavior. When linear eaters had practiced eating at a decelerated rate for eight weeks, they continued eating at a decelerated rate when tested in the absence of the feedback that assisted them during training. Confirming our previous study, linear eaters ate more food when eating at a higher rate; we have suggested that this challenge is a test of disinhibition [17]. Interestingly, those who had practiced eating at a decelerated rate resisted overeating in this test of disinhibition; in fact, they ate somewhat less food, also confirming our previous results [17]. In addition, linear eaters turned decelerated scored lower on the DEBQ scales of restrained and external eating. In our previous study, score on the DEBQ restraint scale did not predict the amount of food eaten [17].

The present parsimonious behavioral analysis of the causal relationship among linear eating, disinhibition and restrained eating contrasts to the notion that restrained eating is a self-imposed cognitive strategy, dissociated from physiology and related to a personality trait such as impulsivity [15,29]. In further contrast, eating behavior has been found to be less dependent upon the cognitive characteristics of the individual and more dependent upon the effort necessary to obtain food. When that effort is minimal, humans overeat to the extent that their eating behavior appears “mindless” [30]. Moreover, findings in the neurobiology of mental causation and cognitive science question the notion of conscious cognitive control of behavior [31]. In this analysis, the experience of conscious will is a feeling rather than a cause [31].

Food intake among restrained eaters is thought to depend on external cues [23], but this notion extends to most types of eater [30]. Interestingly, cue-conditioning easily overrides physiological concomitants of eating and as the neurobiology of cue-conditioned eating emerges [32], it will probably replace intervening cognitive constructs such as restrained eating. Meanwhile, we suggest that the distinction between restrained and unrestrained eating is replaced by behavioral data such as linear and decelerated eating. Eating behavior cannot only be conveniently measured and changed as demonstrated here; it can also be used as a clinical intervention. Thus, by use of the external support provided by Mandometer®, eating disorders such as anorexia and bulimia nervosa can be treated [19]. Preliminary evidence suggests that morbidly obese children and obese patients with binge eating disorder can be similarly treated [33]. These results offer further verification of the hypothesis tested here and elsewhere [20], that eating behavior is a cause rather than a consequence of the psychological and cognitive changes in anorexia, bulimia and possibly obesity. However, as we have pointed out, the details of the precise pattern of eating to be used for clinical interventions need to be further examined [17].

On the basis of these results it is tempting to suggest a possible way to prevent eating disorders, including obesity. Thus, by training young people to eat at a decelerated rate, they may develop resistance to disinhibition and hypothetically therefore to disordered eating. Obviously, however, follow-up of the effect reported here is required to further test this hypothesis. It has been correctly pointed out that eating behavior should be studied for prolonged periods of time [24]; the test of disinhibition used in the present study may be temporary. We also need to study if a decelerated pattern of eating as a result of practicing eating using Mandometer® affects food intake and eating behavior in an everyday setting. In addition, cognitive characteristics other than restrained eating many very well turn out to be important determinants of human eating behavior. Clearly, further testing of the hypothesis presented here is required.
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References