Vision and Eating Behavior in Obese Subjects

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Abstract

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Objective: Vision is one of a number of factors influencing the amount of food consumed during a meal. The purpose of this study was to investigate the impact of vision on the microstructure of the eating behavior of obese subjects.

Research Methods and Procedures: Eighteen obese subjects with a body mass index (mean ± SD) of 39.1 ± 6.3 kg/m^2 twice consumed a standardized test meal in excess, once with and once without a blindfold. The microstructure of the eating behavior was registered by VIKTOR, a computerized eating monitor. Subjective motivation to eat (i.e., desire to eat, hunger, satiety, and prospective consumption) was rated by visual analogue scales (VASs) before, immediately after, and then hourly up to 3 hours after the test meals.

Results: The obese subjects ate 24% less food when blindfolded (359 ± 194 g vs. 472 ± 179 g; $p < 0.01$). Despite a smaller amount of food consumed when blindfolded, there were no significant differences with or without the blindfold for any of the VASs measuring subjective motivation to eat after test meals.

Discussion: The importance of vision in regulating our eating behavior was demonstrated in this study. The obese subjects ate 24% less food blindfolded without feeling less full. Eating blindfolded could be tested as a didactic tool to make obese subjects aware of what factors affect the termination of eating.

Key words: appetite, eating behavior, universal eating monitor, vision

Introduction

The mechanisms controlling eating behavior are very complex and involve a number of factors, including internal physiological signals and external signals arising from cognitive cues, social factors, and cultural rules, as well as cues from the actual food being eaten (1). Vision is one component in this complex system regulating the food intake. Vision may stimulate, but also contribute to, satiety cognitions that terminate intake. The importance of visual signals in regulating food intake was recently demonstrated by us in a study on normal-weight subjects eating with and without a blindfold, using the VIKTOR equipment, which measures the microstructure of the eating behavior (2). Eating blindfolded decreased the intake of food by 22% without affecting the eating rate. Interestingly, the reported feeling of fullness was identical to that reported after the larger meal without the blindfold. Manipulation of the visual cues of a meal might be an interesting new approach in the treatment of obesity. Thus, we extended our studies from normal-weight subjects by using the VIKTOR set up to analyze the microstructure of the eating behavior and subjective motivation to eat in obese subjects eating with and without blindfolds.

Research Methods and Procedures

Eighteen obese subjects (9 women and 9 men; ages 46 ± 14 years) with a body mass index (mean ± SD) of 39.1 ± 6.3 kg/m^2 (range 29 to 52 kg/m^2) were recruited from the waiting list of the Obesity Unit at Huddinge University Hospital.

The microstructure of the eating behavior was measured by VIKTOR (Cabmek, Stockholm, Sweden), a universal eating monitor, originally developed by Kissileff et al. (3). Our version of the universal eating monitor has been previously validated and used in several studies (4–12). Briefly, the VIKTOR equipment consists of a table with a hidden scale connected to a computer that registers bite-by-bite the total intake of food (in grams), the duration of consumption (in minutes), the eating rate (grams per minute), and the rate of deceleration, which is defined as the reduction in eating rate that normally occurs toward the end of a meal. The eating curve is fitted to a polynomial, and the two-fold quadratic coefficient represents the rate of deceleration. A negative coefficient illustrates a decelerating eating curve and a positive coefficient illustrates an accelerating eating curve. The most common type of eating curve is the decelerated eating curve, which has also been called

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the "biological satiation curve" (4). In a previous study of children, only 10% of the normal-weight subjects had accelerating eating curves, whereas 30% of the otherwise healthy obese children and 56% of children with obesity attributable to a hypothalamic dysfunction (e.g., Prader-Willi syndrome) had accelerating eating curves (5).

The subjective motivation to eat, defined as desire to eat, hunger, satiety, and prospective consumption were measured by conventional 100-mm visual analogue rating scales (VASs)[1] (6,7). Subjects rated with a cross on each scale how hungry, full, etc. they felt from 0 to 100 mm, where 0 represents "not at all" and 100 represents "very, very much." The pleasantness of the test meal was checked by using an additional VAS at the end of the test meals.

Experimental Procedure

Before the study, subjects were informed that the effect of vision on "the experience of appetite and of eating" was to be studied. No information of eating behavior measurements was given until after the last meal on VIKTOR.

All subjects were instructed to eat their breakfast according to ordinary habits and not to eat anything else between breakfast and lunch. All subjects were asked to maintain the same exercise habits before test days. All these requirements were checked in detail by a dietitian interviewing the subjects on arrival in the laboratory.

All subjects had lunch twice, with and without a blindfold in counterbalanced order, on the VIKTOR set up. The two occasions were separated by at least 7 days for men ($n = 9$) and the menopausal woman ($n = 4$). For premenopausal women ($n = 5$), the two occasions were separated by 4 weeks to take hormonal effects on eating behavior into consideration. All subjects had the test meal on the VIKTOR equipment at the same hour they were supposed to have their ordinary lunch meal. They were served a glass of water with an excess portion (900 g for men and 700 g for women, frozen weight) of a microwave-heated Swedish hash (150 kcal/100 g) consisting of diced meat and mixed and fried onions and potatoes (Nestlé AB, Bjuv, Sweden). The blindfold was put on the subjects before they entered the room where the test meal was served. A project leader (experienced in working with blind subjects) led the subjects into the test-meal room, seated them, led their hand to the utensil, the glass of water, and around the plate. When the subjects felt familiar with this setting, they were instructed to eat as much as they wished of the meal and to drink the glass of water. Before, immediately after, and hourly up to 3 hours after the lunch test meals, the subjects rated their subjective motivation to eat with VAS (see above).

Table 1. Eating behavior characteristics of obese subjects eating with and without a blindfold

Characteristics	Blindfolded	Without blindfold	Statistics
Total intake of food (g)	359 ± 194	472 ± 179	$p < 0.01$
Time of consumption (min)	10.7 ± 11.1	10.2 ± 5.2	NS
Eating rate (g/min)	41 ± 15	51 ± 18	$p < 0.01$
Deceleration (g/min^2)	−0.7 ± 4.6	−1.8 ± 3.0	NS

NS, not significant.
Mean ± SD are shown.

Data Analysis

Statistical analyses of the VIKTOR data and the VAS were carried out by using Student's t tests for paired comparisons. For VAS, the ratings at the four occasions post-meal were summarized and then compared with Student's t test to prevent mass significance, given that ANOVA was not applicable because of the within-subject design of the study. When comparing the number of decelerated eating curves in each group, we used Fisher's exact test. $p < 0.05$ was considered statistically significant. Approval of the study was obtained by the local Ethics Committee, and all subjects gave their informed consent to participate.

Results

The total food intake, the duration of consumption, the eating rate, and the rate of deceleration are shown in Table 1. The obese subjects ate 24% less food blindfolded than without the blindfold ($p < 0.01$). The order of the meals (i.e., blindfolded the first meal vs. blindfolded the second meal) did not affect the intake of food. The duration of the meal was the same with and without the blindfold, but the eating rate was faster when not blindfolded ($p < 0.05$). There was no difference in the rate of deceleration with and without the blindfold. Thirteen of 18 subjects exhibited decelerated eating curves when eating blindfolded, and 12 of 16 subjects (data on deceleration was missing for 2 subjects) had decelerated eating curves when not blindfolded.

There were no differences in motivation to eat before either test meal; thus, subjects arrived in the same "hunger state" before both meals. In Figure 1, VAS ratings at the different time-points are shown. Although the obese subjects ate less when blindfolded, there were no significant

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[1] Nonstandard abbreviation: VAS, visual analogue scale.

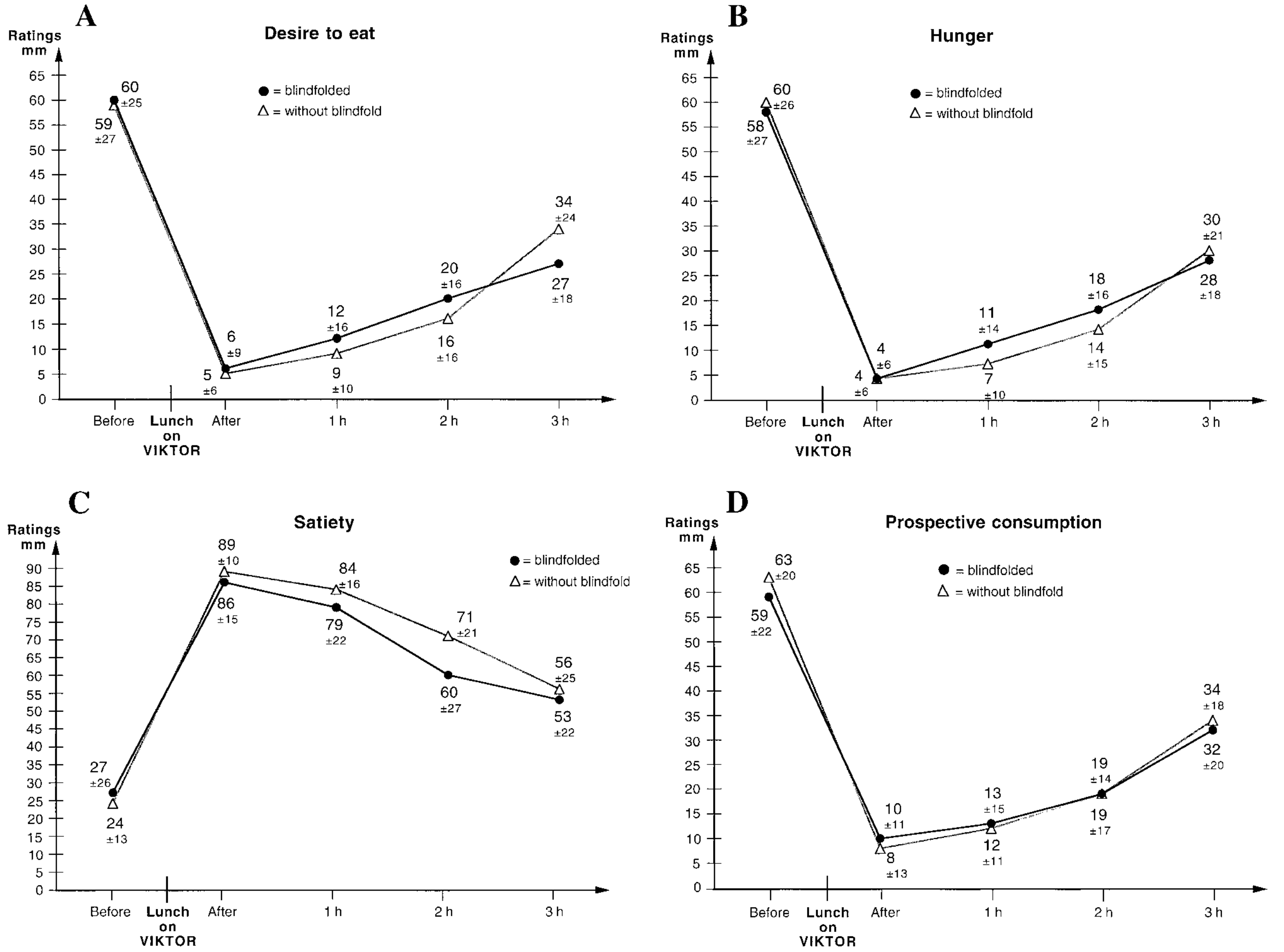

Figure 1: Subjective ratings of motivation to eat, i.e., desire to eat (A), hunger (B), satiety (C), and prospective consumption (D), measured by VASs (100 mm) at different time-points when obese subjects ($N = 18$) were eating lunch test meals with and without the blindfold. Means ± SD are given.

differences in any of the VASs measuring subjective motivation to eat after test meals when comparing with and without the blindfold. The palatability of the test meal was equally rated at the two eating occasions (59 ± 26 mm and 65 ± 26 mm ratings with and without the blindfold, respectively).

Discussion

The importance of vision in regulating our eating behavior was demonstrated in this study. The obese subjects ate 24% less food when blindfolded without feeling less full, compared with eating with vision. These results are in accordance with our previous study on normal-weight subjects using the same measurements and study design (2), although in the present study we extended the use of VAS ratings with hourly measurements up to 3 hours after the test meal.

The use of the VIKTOR equipment as a tool for measuring the microstructure of the eating behavior has been found to be useful for identifying differences in single-meal eating behavior between clinical groups (5,7–10), as well as in studies with within-subject design to test the effects of manipulations on satiety (11–14).

One hypothetical explanation for the decreased food intake is that eating blindfolded could be a normal variability of eating behavior. However, this is not likely because data on the reproducibility of VIKTOR measurements have shown that variables such as eating rate and amount of food eaten are stable, if physical habits and eating patterns are controlled before test meals (7). In the present study, the subjects were asked not to change their breakfast and exercise habits before test meals. To decrease the hormonal influences on the results, menstruating women had their two test meals four weeks apart at identical phases of their menstrual cycles.

When eating without the blindfold, the obese subjects in the present study had a higher eating rate (51 g/min) compared with the normal-weight subjects in the previous study (39 g/min) (2).

In contrast to the normal-weight subjects in the previous study, who ate with the same eating rate when blindfolded (39 g/min on both occasions) (2), in the present study, the eating rate was significantly slower among obese subjects when they ate blindfolded (51 g/min vs. 41 g/min). Some of the obese subjects told us that they felt it was more complicated to eat blindfolded and prevent spilling. Could the slower eating rate explain why subjects ate less food blindfolded? In many of the behavioral-modification programs, subjects are told to slow down the eating rate because it is a widely held belief that slowing of eating rate should result in reductions of the food intake by allowing time for post-ingestive feed-back. However, this assumption lacks scientific proof. All of the post-ingestive, pre-absorptive components of satiety that signal a halt in eating are present from the first bite, and there are no data available showing that signals need a certain time interval function. A study by Yeomans et al. (15) on the effect of the eating rate on the amount of food consumed showed that subjects consumed the smallest amount of food when they ate with a higher eating rate in comparison to when they ate more slowly by taking pauses between bites.

What could be the plausible explanations for the considerable (24%) decrease in food intake with removal of the vision of food? A number of internal factors contribute to the termination of eating; examples are gastric distension and release of intestinal peptides affecting neural mechanisms (16). External factors such as vision may also affect neural mechanisms in the brain involved in the termination of eating. The cephalic phase of digestion in response to the sight and smell of food eliciting salivation, insulin release, and gastric acid secretion is a well-known phenomenon (17). The removal of sight may, therefore, influence the cephalic phase, which, in turn, could affect satiation (the willingness to stop eating).

There is an old Swedish expression "eating with your eyes," i.e., eating is governed more by the visual signals from an appetizing dish than by physiological needs signaled from the gastrointestinal tract and brain. When eating without vision, subjects may be forced to rely more on internal signals. These internal signals may lead subjects to eat the amount of food needed to receive comfortable satiety, whereas vision may have a additional stimulating effect on the appetite and, thus, increase the intake of food. A comment from two of the obese subjects in the present study was, actually, "seeing the food increased the appetite."

It is well known that a variety of foods (e.g., "smorgasbord" vs. a single dish) stimulate and enhance intake. This phenomenon has been called "sensory-specific satiety." Sensory-specific satiety refers to an enhancement of food intake when a variety of foods with different sensory and nutritional properties are served, as compared with a situation with only one or a few food components (18). Sensory-specific satiety also occurs for the visual appearance of food, such as shape and color. For example, when pasta with three different shapes was served, subjects ate 14% more compared with when they were offered their favorite single shape (19). Apparently a variety in appearance through vision also stimulates the intake of food. This indicates that vision vs. no vision could stimulate the intake of food, as seen in the present study.

Another explanation could be that vision does contribute to the estimation of the satiating potential of foods. When we see familiar food, we have some experience of how satisfying it will be and will make a judgement about how it will satisfy us by designing our meal plan. Without vision, the meal plan of the sighted food is lost, and one element of within-meal satiation is absent. Vision may thus stimulate, but also contribute to, satiety cognitions that terminate intake.

What is the clinical application of these findings? These are results from single meal intakes, and we do not know what happens at later meals. To suggest that obese subjects consume all of their meals blindfolded to limit their intake of food, "the blindfold diet," is obviously pointless. However, as part of a behavioral-modification program for obese subjects, it might be worthwhile to test whether a didactic tool such as eating blindfolded might make obese subjects aware of what factors affect the termination of eating.

In summary, in this study, the importance of visual signals to regulate food intake was demonstrated. Eating blindfolded decreased the intake of food among obese subjects without making subjects feeling less full.

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References

1. **Rodin J.** Determinants of food intake regulation in obesity. In: Björntorp P, Brodoff B, eds. *Obesity*. Philadelphia, PA: J.B. Lippincott Company; 1992:220–30.
2. **Linné Y, Barkeling B, Rössner S, Rooth P.** Vision and eating behavior. *Obes Res*. 2002;10:92–5.
3. **Kissileff HR, Klingsberg G, Van Itallie TB.** Universal eating monitor for continuous recording of solid or liquid consumption in man. *Am J Physiol*. 1980;238:R14–R22.
4. **Meyer J-E, Pudel V.** Experimental studies on food intake in obese and normal weight subjects. *J Psychosom Res*. 1972; 16:305–8.
5. **Lindgren AC, Barkeling B, Hägg A, Ritzén M, Marcus C, Rössner S.** Eating behaviour in Prader-Willi syndrome, normal weight and obese control groups. *J Pediatr*. 2000;137: 50–5.
6. **Blundell JE, Rogers PJ, Hill AJ.** Evaluating the satiating power of foods: implications for acceptance and consumption.

In: Solms J, ed. *Chemical Composition and Sensory Properties of Food and their Influence on Nutrition.* London: Academic Press; 1988:205–19.

7. **Barkeling B, Rössner S, Sjöberg A.** Methodological studies on single meal food intake characteristics in normal weight and obese men and women. *Int J Obes*. 1995;19:284–90.
8. **Barkeling B, Ekman S, Rössner S.** Eating behaviour in obese and normal weight 11-year-old children. *Int J Obes*. 1992;16:355–60.
9. **Hylander B, Barkeling B, Rössner S.** Eating behaviour in continuous ambulatory peritoneal dialysis and hemodialysis patients. *Am J Kidney Dis*. 1992;6:592–7.
10. **Hylander B, Barkeling B, Rössner S.** Changes in patients' eating behavior: in the uremic state, on continuous ambulatory peritoneal dialysis treatment, and after transplantation. *Am J Kidney Dis*. 1997;29:691–8.
11. **Barkeling B, Rössner S, Björvell H.** Effects of a high-protein meal (meat) and a high-carbohydrate meal (vegetarian) on satiety measured by automated computerized monitoring of subsequent food intake, motivation to eat and food preferences. *Int J Obes*. 1990;14:743–51.
12. **Barkeling B, Granfelt Y, Björck I, Rössner S.** Effects of carbohydrates in the form of pasta and bread on food intake and satiety in man. *Nutr Res*. 1995;15:467–76.
13. **Näslund E, Barkeling B, King N, et al.** Energy intake and appetite are suppressed by glucagon-like peptide-1 (GLP-1) in obese men. *Int J Obes*. 1999;23:304–11.
14. **Rössner S, Barkeling B, Erlanson-Albertsson C, Larsson P, Wåhlin-Boll E.** Intravenous enterostatin does not affect single meal food intake in man. *Appetite*. 1995;24:37–42.
15. **Yeomans MR, Gray RW, Mitchell CJ, True S.** Independant effects of palatability and within-meal pauses on intake and appetite ratings in human volunteers. *Appetite*. 1997;29:61–76.
16. **Blundell JE, Hill AJ, Lawton CL.** Neurochemical factors involved in normal and abnormal eating in humans. In: Shepherd R, ed. *Handbook of the Psychophysiology of Human Eating.* New York: John Wiley & Sons; 1989:85–112.
17. **Powley TH, Berthoud H-R.** Diet and cephalic phase insulin responses. *Am J Clin Nutr*. 1985;42:991–1002.
18. **Rolls BJ.** Sensory-specific satiety. *Nutr Rev*. 1986;44:93–101.
19. **Rolls BJ, Rowe EA, Rolls ET.** How sensory properties of foods affect human eating behavior. *Physiol Behav*. 1982;29:409–417.