The Eating Disorders
Edited by BJ Blinder, BF Chaitin, &R Goldstein

Chapter 11

Physiological Basis of Food Preferences in Eating Disorders

Adam Drewnowski

Both cognitive and physiological factors determine taste preferences and dietary choices in humans. Food intake may be primarily determined by hunger and metabolic status of the organism, but it is also influenced by nutritional beliefs and attitudes toward weight and dieting [1]. This delicate balance between human behavior and metabolism is disturbed in eating disorders, where a conscious denial of calories can override the body's metabolic needs. Dramatic weight loss seen in anorexia nervosa is often associated with the rejection of specific foods, most often fats, starches and sweet desserts—a phenomenon often described as "carbohydrate phobia" [2].

Anorexia nervosa is also characterized by a variety of metabolic and neuroendocrine dysfunctions, including amenorrhea, hypothyroidism, hypotension, and increased adrenal activity [3]. Some of these changes are the likely consequence of metabolic starvartion resulting from the relentless pursuit of extreme thinness, since many neuroendocrine abnormalities are reversed following weight regain [4]. However, certain symptoms of the anorectic syndrome have been attributed to a basic primary hypothalamic disorder [5]. For example, amenorrhea occurs in at least one third of anorectic patients before any weight loss has occurred, and it may persist after the return to normal body weight, suggesting that some disturbances in the hypothalamic-pituitary-ovarian axis are not fully accounted for by the malnourished state [6]. Furthermore, indirect evidence has linked anorexia nervosa to affective disorders. It has been suggested that mood swings and the binge-purge cycles that are common in anorectic patients are the result of hypothalamic fluctuations in alpha-noradrenergic function and therefore susceptible to pharmacological intervention [7]. Treatment studies of anorexia nervosa with antidepressants have been conducted to eveluate this hypothesis [6].

Central neurotransmitter mechanisms may influence eating behaviors and weight status in different ways. Psychopharmacological studies [7,8] have examined the role of adrenergic, dopaminergic, serotonergic, and opiate receptor systems in the regulation of caloric intake and in the selection of specific macronutrients in the rat. Studies on the noradrenergic system in the medial hypothalamus suggest that chemical blockade of noradrenergic activation or irreversible damage in this area produces, in the rat, a variety of symptoms similar to those of anorectic patients [7]. These symptoms include a decrease in total food intake, a reduction in meal size and rate of eating, a disturbed diurnal pattern of eating, and hyperactivity. In contrast, medial hypothalamic injections of norepinephrine are likely to cause a rebound toward overeating of a carbohydrate-rich diet. The abnormally large eating response of satiated rats following medial hypothalamic injections of norepinephrine have been compared to bulimic epi-

sodes shown by anorectic patients [7,9]. Compulsive binge eating of sugar and fat-containing foods may also be linked to abnormal function of the endogenous opioid system. Animal studies have documented that exogenous opiate agonists increase the consumption of palatable diets, while opiate antagonists suppress intake and prevent dieting-induced obesity in the laboratory rat. Sensory studies suggest further that opiates may be involved in mediating the pleasure response to sugar and possibly fat [10], explaining perhaps the uncontrollable cravings for sweet taste often reported by patients with eating disorders.

Clinical studies of eating disorders have only recently began to focus on the interaction between metabolic and behavioral variables underlying food palatability and diet selection [1,11-13]. Because neuroendocrine disorders, physiological factors, and the overriding obsession with food and dieting are so closely intertwined, it is difficult to assess their precise contribution to dietary restriction and other abnormalities of eating behavior. However, there is indirect evidence that binge-purge or binge-starvation cycles have a physiological as well as a psychological basis. While binge eating may be viewed as a periodic breakdown of dietary restraint, it may be that cravings for sweet-tasting foods are metabolically determined. Preferences for sweet taste may be influenced by body-weight status, a range of physiological variables, or adipose tissue metabolism [12]. The patient, aware of abnormal craving for sweets, may use fasting or purging in a conscious attempt to regulate a grossly disturbed physiological mechanism [7].

It is worth noting that "carbohydrate phobia" and starch avoidance is not a permanent trait. Forbidden foods such as ice cream, cookies, and desserts are typically consumed to excess during binge eating episodes shown by up to 50% of anorectic patients [14]. During such episodes, large amounts (>2,000 kcal) of easily ingested and usually sweet-tasting foods are consumed in a short space of time. The sensation of satiety is reportedly absent, and eating is accompanied by breathlessness, sweating, racing pulse, and other symptoms of sympathetic activiation. Bulimic episodes are generally terminated by abdominal pain, sleep, social interruption or self-induced vomiting, and are followed by guilt and depression and by prolonged periods of severely restrictred dieting [15,16].

MALNUTRITION AND TASTE

The diet of anorectic patients is typically low in calories, but it includes often excessive amounts of green vegetables, salads, and fruits, sometimes supplemented by daily vitamins [2,17]. Protein intake is generally adequate, but fats and carbohydrates, including sweets and desserts are strictly avoided. Consumption of a relatively high-protein diet produces an unusual type of malnutrition. Extreme emaciation follows from caloric restriction, but protein metabolism is relatively preserved. Russell [2] studied hospitalized patients, including four men, with severe anorexia nervosa to assess the relative importance of protein and calorie malnutrition to the disease state. Weight regained by subjects was primarily fat, indicating that only a small amount of protein depletion had to be corrected by refeeding. The typical diet of anorectic patients has been reported as low in carbohydrate but adequate in both protein and fat [2].

Although the typical diet is not vitamin deficient, it may lack trace minerals, especially copper and zinc. Trace metals, including zinc, cooper, and nickel are thought to have a role in normal taste and smell, and zinc deficiency can depress appetite and facilitate abstinence from food. This possibility was explored in a study [17] of 30 hospitalized women, all meeting Feighner's criteria for anorexia nervosa. Trace metals, vitamins, and other biochemical parameters were measured in an attempt to relate them to taste function, biochemical changes, and clinical signs of the disorder. Plasma and urinary zinc in anorectic patients were moderately lower than in normal controls. The majority of anorectic patients had subnormal taste acuity for bitter and sour substances; taste acuity for salt and sweet was less disturbed. However, taste recognition scores failed to correlate with plasma zinc levels. Appetite was suppressed but not absent unless the patient totally abstained from food, indicating that the role of zinc during total abstinence from food should be further investigated. Nutritional therapy including zinc supplementation and concommitant psychotherapy was reported to improve the patients' emotional well-being and led to substantial weight gain [17].

PERCEPTION OF INTERNAL STATES

The palatability of a given food often depends on the internal states of hunger and satiety [1]. In fact, satiety occurring within a meal has been operationally defined as the point at which food stops tasting good [18]. Anorectic patients are reported to show normal awareness of hunger, but often show disturbances in satiety. These may include postprandial sensation of nausea, distension, and bloating, tense and irritable moods, as well as cognitive preoccupation with food [19,20]. Studies on the relationship between sensations of hunger and gastric distension or gastric motility [21-22] reported that although most anorectic patients correctly identified

stomach contractions, some did not interpret them as hunger. One clinical study [22] compared the rates of gastric emptying in ten anorexic patients and in 12 healthy volunteers fed a sample meal of cornflakes, milk, and sugar. Anorectic patients showed significantly slower gastric emptying and their perception of extreme fullness may well reflect abnormal gastrointestinal function. Attitudinal studies [23] have shown that eating disorder patients tend to dislike calorie dense foods—that is foods rich in fat. It may be that such foods, which delay gastric emptying even further, cause acute discomfort to the anorectic patient.

The perception of satiety is likely to have a cognitive component [1]. In a study of 26 anorectic women and 16 controls, Garfinkel et al [20] provided the subjects with meals that were viewed as either high or low calorie, although the meals were in fact identical (400 kcal). Anorectics reported feeling fuller than normals pre-meal, and they stayed fuller longer after eating the high-calorie-connotation meal. The findings that the expectation of satiety affects the perception of internal state are generally consistent with comparable data obtained for normal-weight and obese individuals [1].

RESPONSIVENESS TO SWEET TASTE

Scientific studies on taste responsiveness have primarily focused on the perception of sweetness and the reported pleasantness of sweet solutions [11]. In contrast to the extensive literature on the role of sweet taste in human obesity [24], there are relatively few studies on the responsiveness to sweet taste of extremely underweight individuals [12]. Previous studies of threshold detection, stimulus recognition, and magnitude estimation of sweetness intensity have generally failed to show differences in sensory function between obese and normal-weight individuals [24,25]. In a study of six anorectic patients and six age-matched controls [26], sensitivity to the sweet taste of sucrose was examined using a two-alternative forced-choice method. Anorectic patients appeared to show a lower taste sensitivity for 0.175% sucrose, but this difference was not statistically significant. Patients gaining weight on a high-calorie, high-carbohydrate diet appeared to show lower sensitivity to sucrose than patients maintaining a stable weight on a low-calorie diet. The study found no major differences in sensory function between anorectic patients and controls, concluding that small differences in the threshold detection of sweet taste may be a consequence of sucrose avoidance in both anorectic and dieting individuals [26].

Studies on the hedonics of sweet taste have generally shown that the pleasure response to sweetness can be influenced by changes in metabolic status [24]. One measure thought to link the subject's taste responsiveness with internal state is the test commonly known as the satiety aversion to sucrose [27]. In early applications of this test, normal-weight subjects were reported to find sweet solutions less pleasant after ingesting 100 ml of concentrated glucose solution. Glucose preloading was assumed to result in "satiety," causing a temporary aversion to the sweet taste of sucrose [27]. Dieting individuals at below-normal body weight reportedly failed to show the satiety aversion to sucrose, which returned following weight regain. Early studies on obese subjects showed that the obese rated sweet solutions as equally pleasant both before and after the ingestion of glucose preload [28], although this finding was not confirmed by other investigators [11,24]. There has been, however some agreement that sustained caloric deprivation or sustained weight loss do lead to enhanced pleasantness ratings for sweet stimuli. Formerly obese patients, or overweight teenage girls following several weeks of weight reduction, showed higher preference ratings for sweetened KoolAid or sweet milkshakes [29,30]. Since reponsiveness to sweet taste is affected by the long-term metabolic status of the organism, it has been thought to serve as a behavioral measure of a physiological "set-point" [27].

A few recent studies have suggested that the relationship between preferences for sweet taste and body weight status may be more complex. One study [31] compared responses to sucrose preloads in underweight, normal-weight, and dieting moderately overweight subjects (at 10% or more above ideal body weight). Overweight subjects rated sucrose solutions higher at the outset than did the other groups, but there were no differences among subjects in the change of pleasantness ratings of 20% and 40% sucrose solutions before and after ingesting 400 ml of 20% sucrose. The authors concluded that contrary to Cabanac's hypothesis, the magnitude of postingestive change in itself cannot be considered a valid indicator of whether an individual is at set point [31].

Anorectic patients who are substantially below normal body weight, and so presumably below "set point," might be expected to show an absence of satiety aversion to sucrose. Garfinkel et al [20] tested 26 anorectic patients and 16 normal-weight volunteers for responsiveness to 20% sucrose solutions presented both before and at three-minute intervals for an hour following a 400 kcal meal. Following repeated sampling, anorectic patients showed slightly higher pleasantness ratings for sucrose compared with normal volunteers and were thought not to show the normal satiety aversion to sucrose. It is worth noting that the failure to develop satiety aversion to sucrose was closely related to disturbances in body image as measured using the anomor-

phic lens technique [20,32].

A follow-up study [33] addressed the issue of the stability of satiety aversion to sucrose following weight regain. Sixteen of 26 original anorectic patients and 13 of 16 original controls were tested one year after the original study. Subjects consumed each of two lunches on two days, one appearing high in calories and one low in calories, although the meals were of equal caloric value (400 kcal). A 20% sucrose solution was tasted before and after the meal. No change was found in the magnitude of satiety aversion to sucrose following the consumption of either meal, and no difference in the development of sucrose aversion was found in either group. The failure of anorectic individuals to show an aversion to sucrose appeared stable from year to year and significant weight gain seemed to have no effect. Clearly, responsiveness to sweet taste, as tracked by the present technique, is not as closely linked to indices of body weight as previously thought. However, other congitive variables may be involved: In both groups of subjects, overestimators of body size, compared with underestimators, failed to show satiety aversion to sucrose [33].

COMPLEX TASTE STIMULI

Because the scientific study of taste preferences has been based largely on model systems that do not resemble real foods, there is growing concern that laboratory results may not be applicable to real-life situations [34-36]. For example, patterns of response to sweetness or saltiness cannot be extrapolated from water solutions to the more complex apricot nectar or tomato juice [36,37]. More investigators now urge that studies on taste perception and preferences be conducted with suprathreshold stimuli that are more representative of foods commonly encountered in the diet. Such studies would examine the potential contributions to food preference of sensory factors other than the four tastes, and would focus on aspects of food texture, consistency, and flavor, which have remained largely unexplored in clinical research [35]. In particular, despite growing evidence that eating disorder patients dislike not carbohydrate but fat, very little is known about the psychophysics or the hedonics of fat perception or about human preferences for fat-containing foods [12,13].

Sensory studies of complex food-like stimuli require the use of procedures that are capable of mapping sensory space and describing taste preference of individual subjects. Such procedures, originally developed for marketing research studies, include multidimensional scaling [38,39], used for product positioning in the marketplace; unfolding techniques, used to examine criteria underlying judgements of preference [40]; and response surface methodology, which maps consumer preferences as a function of perceived stimulus attributes [12,13].

For our sensory stimuli, we used a range of commercially available dairy foods, sweetened with different amounts of sucrose. Preliminary studies on the taste of dairy products reveled that sweetness/sourness and the butter fat content were the key aspects of taste sensation. Normal-weight subjects identified cream cheese and sweetened condensed milk as highly preferred, while buttermilk and skim milk ranked lowest on the list. To determine whether the liking for these high-fat foods was the consequence of stimulus sweetness or its fat content, we developed a simplified system of milk products for further investigations of the sensory space [13].

The subjects were asked to rate the perceived intensity of sweetness, fatness, and creaminess of 20 different mixtures of milk, cream, and sugar, and assigned an overall hedonic rating to each sample. As expected, intensity estimates increased as logarithmic functions of sucrose or fat concentration, and no mixture phenomena were observed. Fat levels did not affect the judgment of sweetness, while estimates of creaminess were independent of sugar levels. In contrast, hedonic responses to sweetness were strongly modulated by the fat content of the samples tested. Sucrose in heavy cream was rated as much more palatable than the same amount of sucrose in skim milk. In fact, of all 20 samples, the favorite was heavy cream mixed with safflower oil (>50% fat w/w) and sweetened with only 10% sucrose [13].

Tests of this procedure with normal-weight, obese, and stabilized reduced-obese subject populations [41] have shown that taste and hedonic responsiveness are not affected by short-term changes in metabolic status, such as overnight fasting. Long-term nutritional status was more important: Obese patients disliked sweetness and preferred high-fat foods, while reduced-obese patients showed enhanced preferences for both sugar and fat.

Additional studies [42] suggest that anorectic patients at below- normal body weight dislike the oral sensation of fat. In contrast to obese patients who greatly preferred fat to sugar, anorectic patients preferred intense sweetness over fat. These changes in taste responsiveness were reliably linked to the measure of body mass index (weight/height^2), and it is tempting to speculate that they are metabolically determined [12]. Among potential metabolic antecedents of this behavioral response are insulin levels, adipose cell size, or lipoprotein lipase activity [41]. The contribution of the

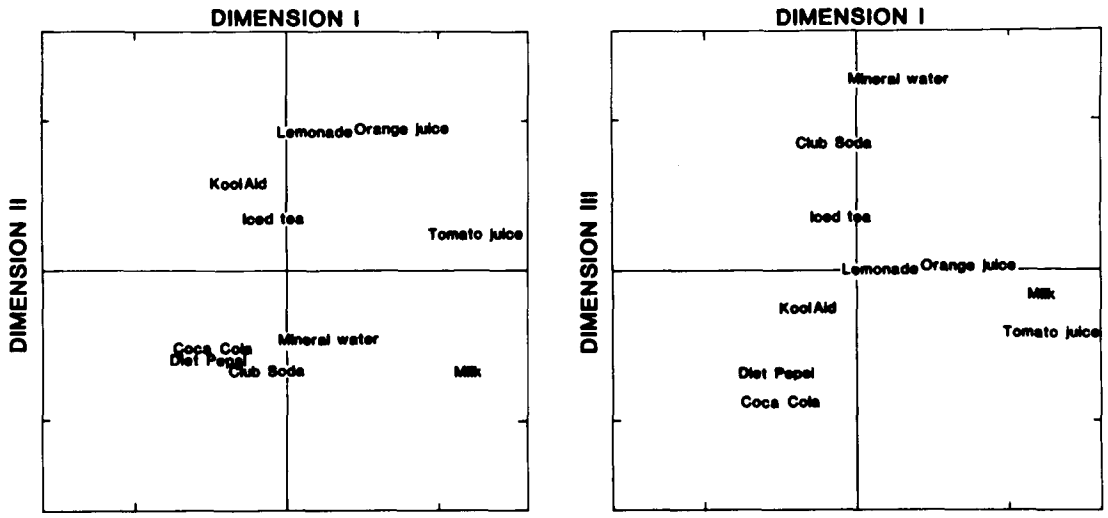

Figure 11.1 A three-dimensional MDS solution of the perceptual space for a variety of soft drinks achieved by the SINDSCAL program. Dimension I: naturalness; Dimension II: taste; Dimension III: sweetness.

endogenous opioid system also deserves further study.

ATTITUDINAL FACTORS AND DEVELOPMENT OF AVERSIONS

Nausea and self-induced vomiting, which are prevalent among anorectic women, may play a role in the development of food aversions. The acquisition of taste aversion among 260 female and 257 male college students was the focus of one study [43] conducted using questionnaires or written interviews. The mean number of aversions reported was 0.8 per person; one or more aversions was reported for 65% of the subjects. Females were more likely to report aversions than males (70.3% versus 60.5%), and aversions were more likely to form with less-familiar, less-preferred foods. One fourth of the aversions were to alcoholic beverages, and 29% of aversions generalized to related foods.

A similar study [44] of three groups of subjects with eating and drinking disorders included anorectic/bulimic women, chronic alcoholics, and college students who were heavy users of alcohol. The anorectic population included seven women diagnosed as anorectic, two diagnosed as bulimic, and ten with both disorders. Among the anorectic women, 29% reported one or more aversions; and among those suffering from both disorders, 80% reported one or more aversions. Overall, 58% reported one or more aversions. While college students reported strength of aversion-forming nausea at 4 on a 5-point scale, subjects with eating disorders reported a 4.7 rating. However, no subject reported that self-induced vomiting was a specific cause of an aversion [44].

COGNITIVE STRUCTURE

Dieting obese and anorectic patients are likely to differ in terms of their attitudes toward food and dieting. Whereas the reduced obese typically overeat calorie-dense foods and eventually regain body weight, the behavior of anorectic patients is greatly influenced by "cognitive structure" that includes self-image and nutritional attitudes and beliefs. Although they are severely emaciated, anorectic patients often report that they are eating enough calories to maintain what they insist is a normal state of health and body weight. Total elimination of starches or fats from the anorectic diet [2] may be the direct result of the patients' distorted beliefs regarding the nutritional or caloric value of different foods. Misperceptions of anorectic individuals regarding their body size or body image have been investigated in some detail [32], but little data are available on perceptual distortions with regard to foods [23].

Quantitative techniques borrowed from marketing research studies permit the mapping of preference space for a variety of food items. The major premise of a mathematical technique known as multidimensional scaling (MDS) is that judgements of psychological similarity between pairs of items reflect the location of the items in perceptual space, whose chief dimensions correspond to the principal features of the scaled items [38,39]. The technique can therefore be used to map mental spaces for food stimuli and to examine potential differences in "point of view" among individuals or

groups of individuals.

One study employing these techniques has been carried out with anorectic patients. The data are preliminary and should be regarded solely as a demonstration of the new methodology. The subjects were 12 normal-weight individuals (mean weight, 145 lb), four obese subjects (mean weight, 393 lb), and a group of seven young women hospitalized for anorexia nervosa (mean weight, 92 lb). The study employed direct proximity judgements to provide an estimate of the degree of perceived similarity among different food items—in this case, soft drinks, presented verbally by brand name. The subjects were also asked to rate each item along a number of adjective scales and to indicate a degree of preference for each item, using the standard nine-point hedonic preference scale [39]. MDS of proximity matrices provided a three-dimensional solution that is summarized in the two panels of Figure 1. It can be seen that the subjects distinguished between the different soft drinks on the basis of three sensory attributes: (1) naturalness, (2) taste, and (3) sweetness and calories. Dimension I ("naturalness") distinguished between fruit juices and carbonated beverages, while Dimension II correlated with the concepts of "taste". Dimension III represented the concepts of sweetness and calories and discriminated between club soda and mineral water and the sweetened and more caloric soft drinks.

Table 11.1 Simple correlations between self-reported preference ratings and bipolar adjective scales as a function of subject group

	Preference ratings		
	Normal-weight (n=12)	Obese (n=4)	Anorectic (n=7)
Artificial - Natural	0.87*	0.56	0.45
Dry - Sweet	-0.03	0.09	-0.55
Low calorie - High calorie	0.09	0.19	-0.31
Tasteless - Intense taste	0.46	0.75*	0.07

NOTE: * denotes $p < .05$

Although the subjects did not differ in their perceptions of the beverages, self-reported preference ratings differed among the groups. As seen in Table 1, the preferences of normal-weight subjects correlated with the concept of naturalness (Dimension I), while obese individuals preferred those soft drinks that were more intensely flavored (Dimension II). Anorectic patients showed no distinct preferences: Their ratings were generally lower throughout, with calorie-free diet soda and mineral water reported as the most preferred items.

The data suggest that the three groups of subjects tend to use different criteria in determining their food preferences. It remains to be seen whether the patterns of food preference are the cause or merely the result of disordered eating and abnormal body weight. Prospective studies of cognitive structure following weight regain should provide information as to whether taste preferences and food choices in eating disorders are affected by physiological varaible and body weight status.

REFERENCES

1. Drewnowski A. Cognitive structure in obesity and dieting. In Greenwood MRC, ed. Obesity: contemporary issues in clinical nutrition. New York: Churchill Livingstone 1983:
2. Russell GFM. The nutritional disorder in anorexia nervosa. J Psychosom Res, 1967; 11:141-9.
3. Brown GM. Neuroendocrine abnormalities in psychosocial dwarfism and anorexia nervosa. In Van Praag HM, Dekker M, eds. Handbook of biological psychiatry, part III: Brain mechanisms and abnormal behavior, genetics, and neuroendocrinology. New York: 1980:
4. Sherman BM, Halmi KA. The effect of nutritional rehabilitation on hypothalamic-pituitary function in anorexia nervosa. In Vigersky RA, ed. Anorexia Nervosa. New York: Raven Press, 1977:
5. Mawson AR. Anorexia nervosa and the regulation of intake: A review. Psychol Med, 1974; 4:289-308.
6. Halmi KA. Anorexia nervosa. In Grahame-Smith DG, Hippius H, Winokur G, eds. Psychopharmacology, vol 1, part 2, clinical psychopharmacology. Amsterdam: Excerpta Medica, 1982:
7. Leibowitz SF. Hypothalamic noradrenergic system: Role in control of appetite and relation to anorexia nervosa. In New directions in anorexia nervosa. Proceedings of the Fourth Ross Conference on Medical Research, 1982:
8. Leibowitz SF. Hypothalamic catecholamine systems in relation to control of eating behavior and to mechanisms of reward. In Hoebel BG, Novin D, eds. The neural basis of feeding and reward. Brunswick, ME: Haer Institute, 1982:
9. Leibowitz SF, Hor L. Endorphinergic and alpha-noradrenergic systems in the paraventricular nucleus: Effects on eating behavior. Peptides, 1982; 3:421-8.
10. Blass EM. Opioids, sugar and the inherent taste of sweet: broad motivational implications. In Dobbing J, ed. Sweetness. Springer-Verlag, Berlin, 1987.
11. Drewnowski A, Gruen R, Grinker JA. Carbohydrates, sweet taste and obesity: Changing consumption patterns and health implications. In Lineback DR, Inglett GE, eds. Basic symposium on food carbohydrates. New York: AVI Publishing, 1982:
12. Drewnowski A, Greenwood MRC, Halmi KA. Carbohydrate or fat phobia: Taste responsiveness in ano-

rexia nervosa. Fed Proc, 1984; 43(3):475.
13. Drewnowski A, Greenwood MRC. Cream and sugar: Human preferences for high-fat foods. Physiol Behav, 1983; 30:629-33.
14. Russell GFM. Bulimia nervosa: An ominous variant of anorexia nervosa. Psychol Med, 1979; 9:429-48.
15. Casper RC, Eckert ED, Halmi KA, et al. Bulimia: Its incidence and clinical importance in patients with anorexia nervosa. Arch Gen Psychiat, 1980; 37:1030-5.
16. Garfinkel PE, Moldofsky H, Garner DM. The heterogeneity of anorexia nervosa: Bulimia as a distinct subgroup. Arch Gen Psychiat, 1980; 37:1036-40.
17. Casper R, Kirschner B, Sandstead H, et al. An evaluation of trace metals, vitamins, and taste function in anorexia nervosa. Am J Clin Nutr, 1980; 33:1801-8.
18. Rolls BJ, Rowe EA, Sweeney K. Sensory specific satiety in man. Physiol Behav, 1981; 271:137-42.
19. Garfinkel PE. Perception of hunger and satiety in anorexia nervosa. Psychol Med, 1974; 4:309-15.
20. Garfinkel PE, Moldofsky H, Garner DM, et al. Body awareness in anorexia nervosa: Disturbances in "body image" and "satiety." Psychosom Med, 1978; 40:487-98.
21. Holt S, Ford MJ, Grant S, et al. Abnormal gastric emptying in primary anorexia nervosa. Br J Psychiatry, 1981; 139:550-2.
22. Russell DM, Freedman ML, Feighlin DHI, et al. Delayed gastric emptying and improvement with domperidone in a patient with anorexia nervosa. AM J Psychiatry, 1983; 140(9):1235-6.
23. Drewnowski A, Pierce B, Halmi KA. Fat aversion in eating disorders. Appetite (in press).
24. Grinker J. Obesity and sweet taste. Am J Clin Nutr, 1978; 31:1078-87.
25. Grinker JA, Hirsch J, Smith D. Taste sensitivity and susceptibility to external influence in obese and normal-weight subjects. J Personal Soc Psychol, 1972; 22:320-5.
26. Lacey JH, Stanley PA, Crutchfield SM. Sucrose sensitivity in anorexia nervosa. J Psychosom Res, 1977; 21:17-21.
27. Cabanac M. Physiological role of pleasure. Science, 1971; 173:1103-7.
28. Cabanac M, Duclaux R. Obesity: Absence of satiety aversion to sucrose. Science, 1970; 168:496-7.
29. Grinker JA, Price J, Greenwood MRC. Studies of taste in childhood obesity. In Novin D, Wyrwicka W, Bray GA, eds. Hunger: Basic mechanisms and clinical implications. New York: Raven Press, 1976:
30. Rodin J, Moskowitz, Bray GA. Relationship between obesity, weight loss, and taste responsiveness. Physiol Behav, 1976; 17:391-7.
31. Gilbert DG, Hagen RL. Taste in underweight, overweight, and normal-weight subjects before, during, and after sucrose ingestion. Addictive Behaviors, 1980; 5:137-42.
32. Garner DM, Garfinkel PE, Moldofsky H. Perceptual experiences in anorexia nervosa and obesity. Can Psychiat Assoc J, 1978; 23:249-63.
33. Garfinkel PE, Moldofsky H, Garner DM. The stability of perceptual disturbances in anorexia nervosa. Psychol Med, 1979; 9:703-8.
34. Moskowitz HR, Kluter RA, Westerling J, et al. Sugar sweetness and pleasantness: Evidence for different psychophysical laws. Science, 1974; 184:583-5.
35. Moskowitz HR. Taste and food technology: Acceptability, aesthetics and preference. In Carterette EL, Friedman MP, eds. Handbook of perception, vol VIA, tasting and smelling. New York: Academic Press, 1978:
36. Pangborn RM, Pecore SD. Taste perception of sodium chloride in relation to dietary intake of salt. Am J Clin Nutr, 1982; 35:510-20.
37. Witherly SA, Pangborn RM, Stern JS. Gustatory responses and eating duration of obese and lean adults. Appetite, 1980; 1:53-63.
38. Schiffman SS, Musante G, Conger J. Application of multidimensional scaling to ratings of foods for obese and normal-weight individuals. Physiol Behav, 1979; 23:1-9.
39. Drewnowski A, Grinker JA, Hirsch J. Obesity and flavor perception: Multidimensional scaling of soft drinks. Appetite, 1982; 3:361-8.
40. Birch LL. Preschool children's food preferences and consumption patterns. J Nutr Ed, 1979; 11:189-92.
41. Drewnowski A, Brunzell JB, Sande K, et al. Sweet tooth reconsidered: taste responsiveness in human obesity. Physiol Behav, 1985; 35:617-622.
42. Drewnowski A, Halmi, KA, Pierce B, Gibbs J, Smith GP. Taste and eating disorders. Am J Clin Nutr, 1987; 46:442-50.
43. Logue AW, Ophir I, Strauss KE. The acquisition of taste aversions in humans. Behav Res Ther, 1981; 19:319-33.
44. Logue AW, Logue KR, Strauss KE. The acquisition of taste aversions in humans with eating and drinking disorders. Behav Res Ther, 1983; 21:275-89.