Obesity and Eating

Internal and external cues differentially affece the eating behavior of obese and normal subjects.

Stanley Schachter

Current conceptions of hunger control mechanisms indicate that food deprivation leads to various peripheral physiological changes such as modification of blood constituents, increase in gastric motility, changes in body temperature, and the like. By means of some still debated mechanism, these changes are detected by a hypothalamic feeding center. Presumably some or all facets of this activated machinery lead the organism to search out and consume food. There appears to be no doubt that peripheral physiological changes and activation of the hypothalamic feeding center are inevitable consequences of food deprivation. On the basis of current knowledge, however, one may ask, when this biological machinery is activated, do we necessarily describe ourselves as hungry, and eat? For most of us raised on the notion that hunger is the most primitive of motives, wired into the animal and unmistakable in its cues, the question may seem far-fetched, but there is increasing reason to suspect that there are major individual differences in the extent to which these physiological changes are associated with the desire to eat.

On the clinical level, the analyst Hilde Bruch (*1*) has observed that her obese patients literally do not know when they are physiologically hungry. To account for this observation she suggests that, during childhood, these patients were not taught to discriminate between hunger and such states as fear, anger, and anxiety. If this is so, these people may be labeling almost any state of arousal "hunger," or, alternatively, labeling no internal state "hunger."

If Bruch's speculations are correct, it should be anticipated that the set of physiological symptoms which are considered characteristic of food deprivation are not labeled "hunger" by the obese. In other words the obese literally may not know when they are physiologically hungry. For at least one of the presumed physiological correlates of food deprivation, this does appear to be the case. In an absorbing study, Stunkard (*2*, *3*) has related gastric motility to self-reports of hunger in 37 obese subjects and 37 subjects of normal size. A subject, who had eaten no breakfast, came to the laboratory at 9 a.m.; he swallowed a gastric balloon, and for 4 hours Stunkard continuously recorded gastric motility. Every 15 minutes the subject was asked if he was hungry. He answered "yes" or "no," and that is all there was to the study. We have, then, a record of the extent to which a subject's self-report of hunger corresponds to his gastric motility. The results show (i) that obese and normal subjects do not differ significantly in degree of gastric motility, and (ii) that, when the stomach is not contracting, the reports of obese and normal subjects are quite similar, both groups reporting hunger roughly 38 percent of the time. When the stomach is contracting, however, the reports of the two groups differ markedly. For normal subjects, self-report of hunger coincides with gastric motility 71 percent of the time. For the obese, the percentage is only 47.6. Stunkard's work seems to indicate that obese and normal subjects do not refer to the same bodily state when they use the term *hunger*.

Effects of Food Deprivation and Fear

If this inference is correct, we should anticipate that, if we were to directly manipulate gastric motility and the other symptoms that we associate with hunger, we would, for normal subjects, be directly manipulating feelings of hunger and eating behavior. For the obese there would be no correspondence between manipulated internal state and eating behavior. To test these expectations, Goldman, Gordon, and I (*4*) performed an experiment in which bodily state was manipulated by two means—(i) by the obvious technique of manipulating food deprivation, so that some subjects had empty stomachs and others had full stomachs before eating; (ii) by manipulating fear, so that some subjects were badly frightened and others were quite calm immediately before eating. Carlson (*5*) has indicated that fear inhibits gastric motility; Cannon (*6*) also has demonstrated that fear inhibits motility, and has shown that it leads to the liberation, from the liver, of sugar into the blood. Hypoglycemia and gastric contractions are generally considered the chief peripheral physiological correlates of food deprivation.

Our experiment was conducted under the guise of a study of taste. A subject came to the laboratory in mid-afternoon or evening. He had been called the previous evening and asked not to eat the meal (lunch or dinner) preceding his appointment at the laboratory. The experiment was introduced as a study of "the interdependence of the basic human senses—of the way in which the stimulation of one sense affects another." Specifically, the subject was told that this study would be concerned with "the effects of tactile stimulation on the way things taste."

It was explained that all subjects had been asked not to eat a meal before coming to the laboratory because "in any scientific experiment it is necessary that the subjects be as similar as possible in all relevant ways. As you probably know from your own experience," the experimenter continued, "an important factor in determining how things taste is what you have recently eaten." The introduction over, the experimenter then proceeded as follows.

For the "full stomach" condition he said to the subject, "In order to guarantee that your recent taste experiences are similar to those of other subjects who have taken part in this experiment, we should now like you to eat exactly the same thing they did. Just help yourself to the roast beef sandwiches on the table. Eat as much as you want—till you're full."

For the "empty stomach" condition, the subjects, of course, were not fed.

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Next, the subject was seated in front of five bowls of crackers and told, "We want you to taste five different kinds of crackers and tell us how they taste to you." The experimenter then gave the subject a long set of rating scales and said, "We want you to judge each cracker on the dimensions (salty, cheesy, garlicky, and so on) listed on this sheet. Taste as many or as few of the crackers of each type as you want in making your judgments; the important thing is that your ratings be as accurate as possible."

Before permitting the subject to eat, the experimenter continued with the next stage of the experiment—the manipulation of fear.

"As I mentioned," he said, "our primary interest in this experiment is the effect of tactile stimulation on taste. Electric stimulation is the means we use to excite your skin receptors. We use this method in order to carefully control the amount of stimulation you receive."

For the "low fear" condition the subject was told, "For the effects in which we are interested, we need to use only the lowest level of stimulation. At most you will feel a slight tingle. Probably you will feel nothing at all. We are only interested in the effect of very weak stimulation."

For the "high fear" condition the experimenter pointed to a large black console loaded with electrical junk and said, "That machine is the one we will be using. I am afraid that these shocks will be painful. For them to have any effect on your taste sensations, the voltage must be rather high. There will, of course, be no permanent damage. Do you have a heart condition?" A large electrode connected to the console was then attached to each of the subject's ankles, and the experimenter concluded, "The best way for us to test the effect of tactile stimulation is to have you rate the crackers now, before the electric shock, and then rate them again, after the shock, to see what changes in your ratings the shock has made."

The subject then proceeded to taste and rate crackers for 15 minutes, under the impression that this was a taste test; meanwhile we were simply counting the number of crackers he ate (*7*). We then had measures of the amounts eaten by subjects who initially had either empty or full stomachs and who were initially either frightened or calm. There were of course, two types of subjects: obese subjects (from 14 percent to 75 percent overweight) and normal subjects (from 8 percent underweight to 9 percent overweight).

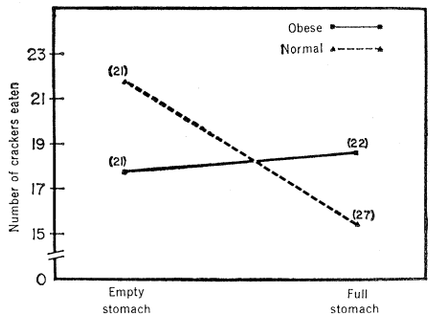

Fig. 1. Effects of preliminary eating on the amounts eaten during the experiment by normal and obese subjects. Numbers in parentheses are numbers of subjects.

To review expectations: If we were correct in thinking that the obese do not label as hunger the bodily states associated with food deprivation, then our several experimental manipulations should have had no effects on the amount eaten by obese subjects; on the other hand, the eating behavior of normal subjects should have directly paralleled the effects of the manipulations on bodily state.

It will be a surprise to no one to learn, from Fig. 1, that the normal subjects ate considerably fewer crackers when their stomachs were full than when their stomachs were empty. The results for obese subjects stand in fascinating contrast. They ate as much —in fact, slightly more—when their stomachs were full as when they were empty (interaction $P < .05$). Obviously the actual state of the stomach has nothing to do with the eating behavior of the obese.

In Fig. 2, pertaining to the effect of fear, we note an analogous picture. Fear markedly decreased the number of crackers the normal subjects ate but had no effect on the number eaten by the obese (interaction $P < .01$). Again, there was a small, though nonsignificant, reversal: the fearful obese ate slightly more than the calm obese.

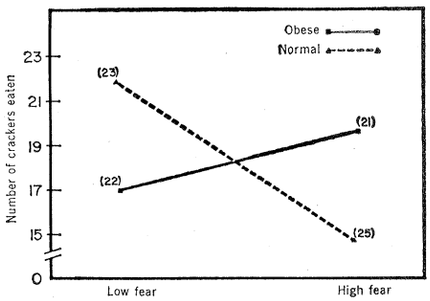

Fig. 2. Effects of fear on the amounts eaten by normal and obese subjects. Numbers in parentheses are numbers of subjects.

It seems clear that the set of bodily symptoms the subject labels "hunger" differs for obese and normal subjects. Whether one measures gastric motility, as Stunkard did, or manipulates it, as I assume my co-workers and I have done, one finds, for normal subjects, a high degree of correspondence between the state of the gut and eating behavior and, for obese subjects, virtually no correspondence. While all of our manipulations have had a major effect on the amounts eaten by normal subjects, nothing that we have done has had a substantial effect on the amounts eaten by obese subjects.

Effects of the Circumstances of Eating

With these facts in mind, let us turn to the work of Hashim and Van Itallie (*8*) of the Nutrition Clinic, St. Luke's Hospital, New York City. Their findings may be summarized as follows: virtually everything these workers do seems to have a major effect on the eating behavior of the obese and almost no effect on the eating behavior of the normal subject.

These researchers have prepared a bland liquid diet similar to commercial preparations such as vanilla-flavored Nutrament or Metrecal. The subjects are restricted to this monotonous diet for periods ranging from a week to several months. They can eat as much or as little of it as they want. Some of the subjects get a pitcher full and pour themselves a meal any time they wish. Other subjects are fed by a machine which delivers a mouthful every time the subject presses a button. With either feeding technique, the eating situation has the following characteristics. (i) The food itself is unappealing. (ii) Eating is entirely self-determined: whether or not the subject eats, how much he eats, and when he eats are matters decided by him and no one else. Absolutely no pressure is brought to bear to limit his consumption. (iii) The eating situation is devoid of any social or domestic trappings. It is basic eating; it will keep the subject alive, but it's not much fun.

To date, six grossly obese and five normal individuals have been subjects in these studies. In Fig. 3 the eating

curves for a typical pair of subjects over a 21-day period are plotted. Both subjects were healthy people who lived in the hospital during the entire study. The obese subject was a 52-year-old woman, 5 feet 3 inches (1.6 meters) tall, who weighed 307 pounds (138 kilograms) on admission. The normal subject was a 30-year-old male, 5 feet 7 inches tall, who weighed 132 pounds.

The subject's estimated daily caloric intake before entering the hospital (as determined from a detailed interview) is plotted at the left in Fig. 3. Each subject, while in the hospital but before entering upon the experimental regime, was fed a general hospital diet. The obese subject was placed on a 2400-calorie diet for 7 days and a 1200-calorie diet for the next 8 days. As may be seen in Fig. 3, she ate everything on her tray throughout this 15-day period. The normal subject was placed on a 2400-calorie diet for 2 days, and he too ate everything.

With the beginning of the experiment proper, the difference in the eating behavior of the two subjects was dramatic and startling. The food consumption of the obese subject dropped precipitately the moment she entered upon the experimental regime, and it remained at an incredibly low level for the duration of the experiment. This effect is so dramatic that the weight of one obese subject who took part in the experiment for 8 months dropped from 410 to 190 pounds. On the other hand, the food consumption of the normal subject of Fig. 3 dropped slightly on the first 2 days, then returned to a fairly steady 2300 grams or so of food a day. The curves for these two subjects are typical. Each of the six obese subjects has manifested this marked and persistent decrease in food consumption during the experiment; each of the normal subjects has steadily consumed about his normal amount of food.

Before suggesting possible interpretations, I should note certain marked differences between these two groups of subjects. Most important, the obese subjects had come to the clinic for help in solving their weight problem and were, of course, motivated to lose weight. The normal subjects were simply volunteers. Doubtless this difference could account for the observed difference in eating behavior during the experiment, and until obese volunteers, unconcerned with their weight, are used as subjects in similar studies, we cannot be sure of the interpretation of this phenomenon.

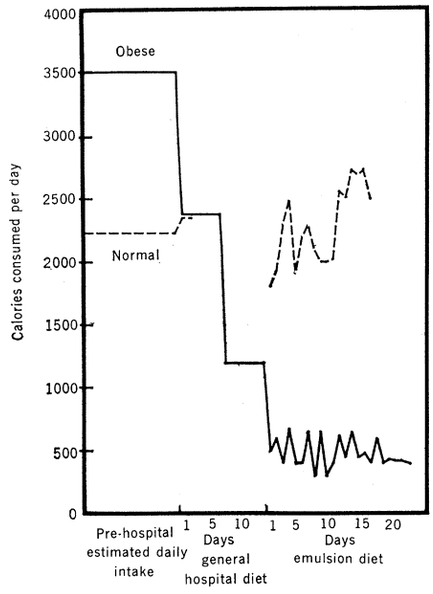

Fig. 3. The effects of an emulsion diet on the amounts eaten by an obese and a normal subject.

However, I think we should not, solely on grounds of methodological fastidiousness, dismiss these findings. It was concern with weight that brought these obese subjects to the clinic. Each of them, before entering the hospital and while in the hospital before being put on the experimental diet, was motivated to lose weight. Yet, despite this motivation, none of these subjects had been capable of restricting his diet at home, and each of them, when fed the general hospital diet, had eaten everything on his tray. Only when the food was dull and the act of eating was self-initiated and devoid of any ritual trappings did the obese subject, motivated or not, severely limit his consumption.

Internal and External Control

On the one hand, then, our experiments indicate virtually no relationship between internal physiological state and the eating behavior of the obese subject; on the other hand, these case studies seem to indicate a close tie between the eating behavior of the obese and what might be called the circumstances of eating. When the food is dull and the eating situation is uninteresting, the obese subject eats virtually nothing. For the normal subject, the situation is just the reverse: his eating behavior seems directly linked to his physiological state but is relatively unaffected by the external circumstances or the ritual associated with eating.

Given this set of facts it seems clear that eating is triggered by different sets of stimuli in obese and normal subjects. Indeed, there is growing reason to suspect that the eating behavior of the obese is relatively unrelated to any internal state but is, in large part, under external control, being initiated and terminated by stimuli external to the organism. Let me give a few examples. A person whose eating behavior is under external control will stroll by a pastry shop, find the food in the window irresistible, and, even if he has recently eaten, go in and buy something. He will pass by a hamburger stand, smell the broiling meat, and, even though he has just eaten, buy a hamburger. Obviously such external factors—smell, sight, taste, other people's actions—to some extent affect anyone's eating. However, in normal individuals such external factors interact with internal state. They may affect what, where, and how much the normal individual eats, but they do so chiefly when he is in a state of physiological hunger. For the obese, I suggest, internal state is irrelevant and eating is determined largely by external factors.

This hypothesis obviously fits the data presented here, as well it should, since it is an *ad hoc* construction designed specifically to fit these data. Let us see, then, what independent support there is for the hypothesis, and where the hypothesis leads.

Effects of Manipulating Time

Among the multitude of external food-relevant cues, one of the most intriguing is the passage of time. Everyone "knows" that 4 to 6 hours after eating his last meal he should eat his next one. Everyone "knows" that, within narrow limits, there are set times for eating regular meals. We should, then, expect that if we manipulate time we should be able to manipulate the eating behavior of the obese subjects. In order to do this, Gross and I (*9*) simply gimmicked two clocks so that one ran at half normal speed and the other, at twice normal speed. A subject arrives at 5:00 p.m., ostensibly to take part in an experiment on the relationship of base levels of autonomic reactivity to personality factors. He is ushered into a windowless room containing nothing but electronic equipment and a clock. Electrodes are put on his wrists, his watch is removed "so that it will not get gummed up with electrode jelly," and

he is connected to a polygraph. All this takes 5 minutes, and at 5:05 he is left alone, with nothing to do for a true 30 minutes, while ostensibly we are getting a record of galvanic skin response and cardiac rate in a subject at rest. There are two experimental conditions. In one, the experimenter returns after a true 30 minutes and the clock reads 5:20. In the other, the clock reads 6:05, which is normal dinner time for most subjects. In both cases the experimenter is carrying a box of crackers and nibbling a cracker as he comes into the room; he puts the box down, invites the subject to help himself, removes the electrodes from the subject's wrists, and proceeds with personality testing for exactly 5 minutes. This done, he gives the subject a personality inventory which he is to complete and leaves him alone with the box of crackers for another true 10 minutes. There are two groups of subjects—normal and obese—and the only datum we collect is the weight of the box of crackers before and after the subject has had a chance at it.

If these ideas on internal and external controls of eating behavior are correct, normal subjects, whose eating behavior is presumably linked to internal state, should be relatively unaffected by the manipulation and should eat roughly the same number of crackers regardless of whether the clock reads 5:20 or 6:05. The obese, on the other hand, whose eating behavior is presumably under external control, should eat very few crackers when the clock reads 5:20 and a great many crackers when it reads 6:05.

The data of Fig. 4 do indeed indicate that the obese subjects eat almost twice as many crackers when they think the time is 6:05 as they do when they believe it to be 5:20. For normal subjects, the trend is just the reverse (interaction $P = .002$)—an unanticipated finding but one which seems embarrassingly simple to explain, as witness the several normal subjects who thought the time was 6:05 and politely refused the crackers, saying, "No thanks, I don't want to spoil my dinner." Obviously cognitive factors affected the eating behavior of both the normal and the obese subjects, but there was a vast difference. While the manipulation of the clock served to trigger or stimulate eating among the obese, it had the opposite effect on normal subjects, most of whom at this hour were, we presume, physiologically hungry, aware that they would eat dinner very shortly, and unwilling to spoil their dinner by filling up on crackers.

Effects of Taste

In another study, Nisbett (*10*) examined the effects of taste on eating behavior. Nisbett reasoned that taste, like the sight or smell of food, is essentially an external stimulus to eating. Nisbett, in his experiment, also extended the range of weight deviation by including a group of underweight subjects as well as obese and normal subjects. His purpose in so doing was to examine the hypothesis that the relative potency of external versus internal controls is a dimension directly related to the degree of overweight. If the hypothesis was correct, he reasoned, the taste of food would have the greatest impact on the amounts eaten by obese subjects and the least impact on the amounts eaten by underweight subjects. To test this, Nisbett had his subjects eat as much as they wanted of one of two kinds of vanilla ice cream; one was a delicious and expensive product, the other an acrid concoction of cheap vanilla and quinine which he called "vanilla bitters." The effects of taste are presented in Fig. 5, in which the subjects ratings of how good or bad the ice cream is are plotted against the amount eaten. As may be seen in Fig. 5, when the ice cream was rated "fairly good" or better, the obese subjects ate considerably more than the normal subjects did; these, in turn, ate more than the underweight subjects did. When the ice cream was rated "not very good" or worse, the ordering tended to reverse: the underweight subjects ate more than either the normal or the obese subjects. This experiment, then, indicates that the external, or at least nonvisceral, cue *taste* does have differential effects on the eating behavior of underweight, normal, and obese subjects.

The indications, from Nisbett's experiment, that the degree of dependence on external cues relative to internal cues varies with deviation from normal weight are intriguing, for, if further work supports this hypothesis, we may have the beginnings of a plausible explanation of why the thin are thin and the fat are fat. We know from Carlson's work (*5*) that gastric contractions cease after a small amount of food has been introduced into the stomach. To the extent that such contractions are directly related to the hunger "experience"—to the extent that a person's eating is under internal control—he should "eat like a bird," eating only enough to stop the contractions. Eating beyond this point should be a function of external cues—the taste, sight, and smell of food. Individuals whose eating is externally controlled, then, should find it hard to stop eating. This hypothesis may account for the notorious "binge" eating of the obese (*11*) or the monumental meals described in loving detail by students (*12*) of the great, fat gastronomic magnificoes.

This rough attempt to explain why the obese are obese in itself raises intriguing questions. For example, does the external control of eating behavior inevitably lead to obesity? It is evident, I believe, that not only is such a linkage logically not inevitable but that the condition of external control of eating may in rare but specifiable circumstances lead to emaciation. A person whose eating is externally controlled should eat and grow fat when food-related cues are abundant and when he is fully aware of them. However, when such cues are lacking or when for some reason, such as withdrawal or depression, the individual is unaware of the cues, the person under external control would, one would expect, not eat, and, if the condition persisted, would grow "concentration-camp" thin. From study of the clinical literature one does get the impression that there is an odd but distinct relationship between obesity and extreme emaciation. For example, 11 of 21 subjects of case studies discussed by Bliss and Branch in *Anorexia Nervosa* (*13*) were, at some time in their lives, obese. In the case of eight of these 11 subjects, anorexia was preceded and accompanied by either marked withdrawal or intense depression. In contrast, intense attacks of anxiety or nervousness [states which our experiment (*4*) suggests would inhibit eating in normal individuals] seem to be associated with the development of anorexia among most of the ten subjects who were originally of normal size.

At this point, these speculations are simply idea-spinning—fun, but ephemeral. Let us return to the results of the studies described so far. These can be quickly summarized as follows.

1) Physiological correlates of food deprivation, such as gastric motility, are directly related to eating behavior and to the reported experience of hunger in normal subjects but unrelated in obese subjects (*3*, *4*).

2) External or nonvisceral cues, such as smell, taste, the sight of other people eating, and the passage of time, affect eating behavior to a greater extent in obese subjects than in normal subjects (*8–10*).

Obesity and Fasting

Given these basic facts, their implications have ramifications in almost any area pertaining to food and eating, and some of our studies have been concerned with the implications of these experimental results for eating behavior in a variety of nonlaboratory settings. Thus, Goldman, Jaffa, and I (*14*) have studied fasting on Yom Kippur, the Jewish Day of Atonement, on which the orthodox Jew is supposed to go without food for 24 hours. Reasoning that, on this occasion, food-relevant external cues are particularly scarce, one would expect obese Jews to be more likely to fast than normal Jews. In a study of 296 religious Jewish college students (defined as Jewish college students who had been to a synagogue at least once during the preceding year on occasions other than a wedding or a bar mitzvah), this proves to be the case, for 83.3 percent of obese Jews fasted, as compared with 68.8 percent of normal Jews ($P < .05$).

Further, this external-internal control schema leads to the prediction that fat, fasting Jews who spend a great deal of time in the synagogue on Yom Kippur will suffer less from fasting than fat, fasting Jews who spend little time in the synagogue. There should be no such relationship for normal fasting Jews. Obviously, there will be far fewer food-related cues in the synagogue than on the street or at home. Therefore, for obese Jews, the likelihood that the impulse to eat will be triggered is greater outside of the synagogue than within it. For normal Jews, this distinction is of less importance. In or out of the synagogue, stomach pangs are stomach pangs. Again, the data support the expectation. When the number of hours in the synagogue is correlated with self-ratings of the unpleasantness of fasting, for obese subjects the correlation is $-.50$, whereas for normal subjects the correlation is only $-.18$. In a test of the difference between correlations, $P = .03$. Obviously, for the obese, the more time the individual spends in the synagogue, the less of an ordeal fasting is. For normals, the number of hours in the synagogue has little to do with the difficulty of the fast.

Obesity and Choice of Eating Place

In another study (*14*) we examined the relationship of obesity to choice of eating places. From Nisbett's findings on taste, it seemed a plausible guess that the obese would be more drawn to good restaurants and more repelled by bad ones than normal subjects would be. At Columbia, students have the option of eating in the university dining halls or in any of the many restaurants that surround the campus. At Columbia, as probably at every similar institution in the United States, students have a low opinion of the institution's food. If a freshman elects to eat in a dormitory dining hall, he may, if he chooses, join a prepayment food plan at the beginning of the school year. Any time after 1 November he may, by paying a penalty of $15, cancel his food contract. If we accept prevailing campus opinion of the institution's food as being at all realistically based, we should anticipate that those for whom taste or food quality is most important will be the most likely to let their food contracts expire. Obese freshmen, then, should be more likely to drop out of the food plan than normal freshmen. Again, the data support the expectation: 86.5 percent of fat freshmen cancel their contracts as compared with 67.1 percent of normal freshmen ($P < .05$). Obesity does to some extent serve as a basis for predicting who will choose to eat institutional food.

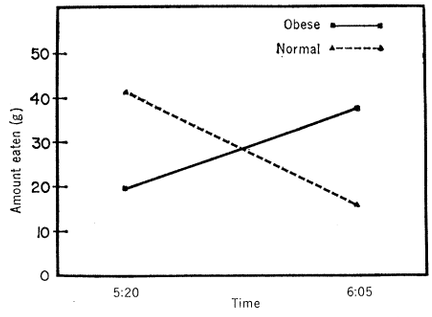

Fig. 4. The effects of manipulation of time on the amounts eaten by obese and normal subjects.

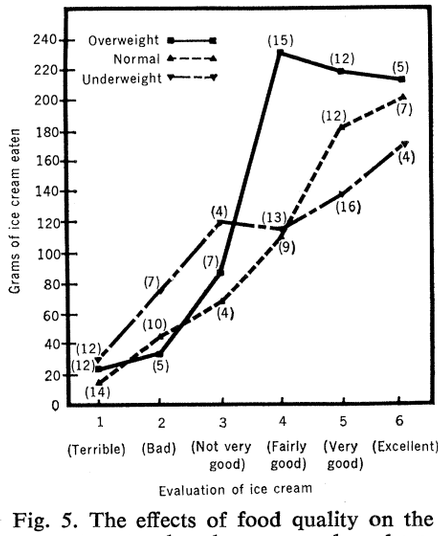

Fig. 5. The effects of food quality on the amounts eaten by obese, normal, and underweight subjects. Numbers in parentheses are numbers of subjects.

Obesity and Adjustment to New Eating Schedules

In the final study in this series (*14*) we examined the relationship of obesity to the difficulty of adjusting to new eating schedules imposed by time-zone changes. This study involved an analysis of data collected by the medical department of Air France in a study of physiological effects of time-zone changes on 236 flight personnel assigned to the Paris–New York and Paris–Montreal flights. Most of these flights leave Paris around noon, French time; fly for approximately 8 hours; and land in North America sometime between 2:00 and 3:00 p.m. Eastern time. Flight-crew members eat lunch shortly after takeoff and, being occupied with landing preparations, are not served another meal during the flight. They land some 7 hours after their last meal, at a time that is later than the local lunch hour and earlier than the local dinner time.

Though this study was not directly concerned with eating behavior, the interviewers systematically noted all individuals who volunteered the information that they "suffered from the discordance between their physiological state and meal time in America" (*15*). One would anticipate that the fatter individuals, being sensitive to external cues (local meal hours) rather than internal ones, would adapt most readily to local eating schedules and be least likely to complain of the discrepancy between American meal times and physiological state.

Given the physical requirements involved in the selection of aircrews, there are, of course, relatively few really obese people in this sample. However, the results of Nisbett's experiment (*10*) indicate that the degree of reliance on external relative to internal cues may well be a dimension which varies with the degree of deviation from normal weight. It seems reasonable, then, to anticipate that, even within a restricted sample, there will be differences in response between the heavier and the lighter members of the sample. This is the case. In comparing the 101 flight personnel who are overweight (0.1 to 29

percent overweight) with the 135 who are not overweight (0 to 25 percent underweight), we find that 11.9 percent of the overweight complain as compared with 25.3 percent of the nonoverweight ($P < .01$). It does appear that the fatter were less troubled by the effects of time changes on eating than the thinner flyers (*16*).

These persistent findings that the obese are relatively insensitive to variations in the physiological correlates of food deprivation but highly sensitive to environmental, food-related cues is, perhaps, one key to understanding the notorious long-run ineffectiveness of virtually all attempts to treat obesity (*17*). The use of anorexigenic drugs such as amphetamine or of bulk-producing, nonnutritive substances such as methyl cellulose is based on the premise that such agents dampen the intensity of the physiological symptoms of food deprivation. Probably they do, but these symptoms appear to have little to do with whether or not a fat person eats. Restricted, low-calorie diets should be effective just so long as the obese dieter is able to blind himself to food-relevant cues or so long as he exists in a world barren of such cues. In the Hashim and Van Itallie study (*8*), the subjects did, in fact, live in such a world. Restricted to a Metrecal-like diet and to a small hospital ward, all the obese subjects lost impressive amounts of weight. However, on their return to normal living, to a man they returned to their original weights.

References and Notes

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15. J. Lavernhe and E. Lafontaine (Air France), personal communication.
16. Obviously, I do not mean to imply that the *only* explanation of the results of these three nonlaboratory studies lies in this formulation of the external-internal control of eating behavior. These studies were deliberately designed to test implications of this general schema in field settings. As with any field research, alternative explanations of the findings are legion, and, within the context of any specific study, impossible to rule out. Alternative formulations of this entire series of studies are considered in the original papers [see Schachter *et al.* (*4* and *9*), Nisbett (*10*), and Goldman *et al.* (*14*)].
17. A. Stunkard and M. McLaren-Hume, *Arch. Internal Med.* **103**, 79 (1959); A. R. Feinstein, *J. Chronic Diseases* **11**, 349 (1960).

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Family and Social Pathology in the Ghetto

Jobs and income are urgently needed to strengthen ghetto families and to reduce civil disorder.

Hyman Rodman

The importance of the family is universally recognized. There are of course many differences in family structure and organization, and in family influence, from society to society and from one period of time to another within the same society. In the changing United States family, for example, certain of the educational, recreational, protective, and economic functions of the family have been taken over by other institutions. The nuclear family is a small and residentially separate unit, and especially in urban areas it is relatively anonymous. Outside agencies—whether neighbors, kinsmen, friends, or police—know less about the families in the community and the individuals within the families. As a result the degree of observation and control of individuals within the United States has lessened as the country has become more urbanized.

The American family has not merely lost functions—it has also added to its functions an increasing responsibility for the emotional welfare and affectional satisfaction of the members of the family (*1*, pp. 249–292). Since individual development and satisfaction have been increasingly emphasized, the strong formal bonds tying together husband and wife regardless of mutual satisfaction, or the ties sanctioning strong authoritarian control over children, have lessened. As a result, divorce and other structural forms of family instability have increased, and the amount of authoritarian control families exert over their children has decreased. The family's responsibility for fostering internalized control on the part of the children has therefore become increasingly important.

All families within the United States are to some degree affected by these historical changes. More anonymity and a greater emphasis upon the individual have also made a considerable difference in the interdependence of family and community life. Instances in which people have ignored others in need of help, often dramatically reported by the mass media, are far outnumbered by situations in which individuals ignore deviant behavior because of the difficulty of distinguishing between help and interference. Will an attempt to curb rowdy behavior on the part of teen-agers be met with abuse from these teen-agers and their parents, or with obedience from the teen-agers

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