Journal of Personality and Social Psychology
1968, Vol. 10, No. 2, 91–97

EFFECTS OF FEAR, FOOD DEPRIVATION, AND OBESITY ON EATING [1]

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This study examines the effects of manipulated fear and food deprivation on the amounts eaten by obese and normal *S*s. Normal *S*s eat more when they are calm than when frightened and eat more when they are food deprived than when they are sated. The manipulations have no effects on the amounts eaten by obese *S*s who eat roughly the same amounts in all experimental conditions.

In a series of studies of the interaction of cognitive and physiological determinants of emotional state (Schachter, 1964; Schachter & Latané, 1964; Schachter & Singer, 1962; Schachter & Wheeler, 1962) it has been demonstrated that precisely the same bodily state may be associated with any of a variety of mood or behavioral states depending upon cognitive and situational factors. In these experiments, bodily state was manipulated by disguised injections of epinephrine or placebo and subjects were placed in situations contrived to produce a variety of emotional states. Subjects in an epinephrine-induced state of arousal prove readily manipulable into states that, by subject self-description and behavior, can reasonably be labeled euphoria, anger, amusement at a movie, and anxiety.

These demonstrations of the "plasticity" of interpretation of bodily state have depended upon the experimental trick of simultaneously and independently manipulating physiological and cognitive variables. Evidence is mounting, however, that the labels and self-descriptions attached to naturally occurring bodily states may be just as modifiable as those attached to exogenously induced bodily states. Nisbett and Schachter (1966), for example, have demonstrated that the experience of pain is readily manipulable by strictly cognitive procedures. Razran's (1962) review of Russian work on interoceptive conditioning indicates that even such presumably nonmalleable feelings as those associated with the volume of urine in the bladder are astonishingly manipulable by what are essentially cognitive techniques.

[1] This study was supported by Grant GS-732 from the National Science Foundation.

The fact that the interpretation and labelling of even naturally occurring bodily states are so readily manipulable opens up questions with almost metaphysical overtones. Obviously, attaching a particular label to any particular internal or visceral syndrome is a learned, cognitively and socially determined act. Though we are inclined to assume that such labels are invariant and universal, it is evident that there is no compelling reason for this to be so. We know, for example, that gastric motility is a direct consequence of food deprivation. Whether or not the label "hunger" is applied to the feelings associated with this physiological symptom has, of course, little to do with the symptom itself, but is in good part determined by the individual's developmental feeding history. If feeding is usually coincidental with these symptoms one can reasonably anticipate that the label "hunger" will be applied to this set of feelings and that the individual will behave in a manner appropriate to this coincidence of label and symptoms. If feeding is chronically inappropriate to physiological conditions, there is probably little reason to anticipate that the label "hunger" will be applied to gastric motility and hypoglycemia.

It is precisely this state of affairs that Bruch (1961) placed at the heart of her theorizing about obesity, for she hypothesized that the obese have not learned to discriminate between the physiological symptoms accompanying food deprivation and the condition of arousal characteristic of emotional states such as fear, anger, and anxiety. Whether or not this developmental line of speculation is correct is an open question, but there is at least evidence that the obese, as a

class, do not label gastric contractions as hunger. Using a gastric balloon to measure contractions, Stunkard and Koch (1964) have demonstrated that for obese subjects there is relatively little correspondence between the state of the stomach and self-reports of hunger. The obese are almost as likely to report hunger when the stomach is not contracting as when it is and, conversely, as likely to deny hunger when the stomach is contracting as when it is not. In distinct contrast there is a relatively close correspondence between the state of the stomach and reports of hunger for normal-size subjects. They are far more likely to report that they feel hungry when the stomach is contracting than when it is not.

Two additional facts are helpful in interpreting these findings. First, during the 4-hour experimental period in this study, gastric contractions were equally frequent for obese and normal subjects. Second, Griggs and Stunkard (1964) have demonstrated that obese subjects can be readily trained to recognize gastric contractions—a fact which they interpret as indicating that the obese do not suffer from a defective visceral sensorium. It would appear, then, that the obese do have gastric contractions, that they are, at least, capable of perceiving them and that, unlike normals, they do not describe themselves as feeling hungry when their stomachs are contracting. Hunger, then, appears to be a label which is not universally applied to an identical set of physiological symptoms.

If all of this is correct, it seems reasonable to assume that the actual eating behavior of obese and normal subjects will correspond to this same pattern; that is, the amount of food eaten should, for normal subjects, relate directly to the state of the viscera while, for obese subjects, there should be no relation. The test of this expectation is certainly simple enough, requiring only the experimental manipulation of visceral states and the measurement of food consumption.

In the present experiment the physiological correlates of food deprivation were manipulated by two means: first, by the obvious method of directly manipulating food deprivation so that some subjects entered an experimental eating situation with empty stomachs and others with full stomachs; second, by manipulating fear so that some subjects entered the eating situation frightened and others calm. Carlson (1915) has presented evidence indicating that fear inhibits gastric motility; Cannon (1916) has demonstrated that the state of fear leads to the suppression of gastric movement and the liberation of sugar from the liver into the blood. Hypoglycemia and gastric contractions are generally considered the chief peripheral physiological correlates of food deprivation. If it is correct that the obese do not label these symptoms as hunger, it should follow that the eating behavior of obese subjects will be unaffected by these manipulations. They should eat just as much when their stomachs are full as when they are empty and just as much when they are frightened as when they are calm. Normal subjects, on the other hand, should be directly affected by the manipulations. They should eat less with full stomachs than with empty ones and should eat more when they are calm than when frightened.

METHOD

The experiment was conducted within the framework of a study of taste. Subjects, all male students at Columbia, came to the laboratory in midafternoon (2:00 or 3:00 P.M.) or evening (7:00 or 8:00 P.M.). Two subjects were always scheduled to run at the same time. If one of the subjects didn't show up, a stooge was run in his place. All subjects had been contacted the previous evening and asked not to eat the meal (lunch or dinner) preceding their experimental appointment. The experimenter's introductory patter was an expanded version of the following:

> A subject of considerable importance in psychology today is the interdependence of the basic human senses, that is, the way the stimulation of one sense affects another. To take a recent example, research has discovered that certain sounds act as very effective pain killers. Some dentists are, in fact, using these sounds, instead of novacaine, to "block out" pain when they work on your teeth. Some psychologists believe that similar relationships exist for all the senses—the experiment we are working on now concerns the effect of tactile stimulation on the way things taste.
>
> The reason we asked you not to eat before coming here is that 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, an important factor in determining how things taste is what you have recently eaten. For example, after eating any

richly spiced food such as pizza, almost everything else tastes pretty bland.

Manipulating Preloading

Following this introduction, the experimenter manipulated preloading as follows: In the full-stomach condition he said,

> In order to guarantee that your recent taste experiences are entirely similar, we should now like you each to eat exactly the same thing. Just help yourself to the roast beef sandwiches on the table. Eat as much as you want—'till you're full.

The subjects spent about 15 minutes eating, and while they ate filled out a long food-preference questionnaire.

In the empty-stomach [2] condition the subjects, of course, were not fed. They simply spent the 15-minute period filling out the questionnaire about food.

Subjects who were fed were presented with two large roast beef sandwiches and a glass of water. Obese subjects consumed an average of 1.88 sandwiches before stopping while normal subjects averaged 1.74 sandwiches.

Setting up the Experimental Eating Situation

Following the 15-minute eating period, the subject was seated in front of five bowls of crackers and told:

> Now that we are through with the preliminaries we can get to the main part of the experiment. What we are going to have each of you do is to taste five different kinds of crackers and tell us how they taste to you. These are very low calorie crackers designed to resemble commercial products.

The experimenter then presented the subject with a long set of rating scales and said,

> We would like you to judge each cracker on each of the dimensions (salty, cheesy, garlicky, etc.) listed on this sheet—taste as many or as few crackers of each type as you want in making your judgments; the important thing is that your ratings be as accurate as possible.

Manipulating Fear

Before permitting the subjects to eat crackers, the experimenter continued with the final stage of the experiment—the manipulation of fear:

> As I mentioned before, our primary interest in this experiment is the effect of tactile stimulation on taste. Electric stimulation is the means we have chosen to excite your skin receptors. We use this method so that we can carefully control the amount of stimulation you receive.

In low-fear conditions the subject was told,

> In order to create the effect in which we are interested we need use only the lowest level possible. At most you will feel a slight tingle in your skin. Probably you will feel nothing at all. We are only interested in the effect of very weak stimulation.

In high-fear conditions the experimenter pointed to an 8-foot high, jet-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, they must be of a rather high voltage. There will, of course, be no permanent damage.

The subject was then connected to the console by attaching a very large electrode to each ankle. While doing this, the experimenter looked up at the subject and asked, "You don't have a heart condition, do you?"

Following the manipulation of high or low fear the experimenter concluded with,

> The best way for us to test the effect of the tactile stimulation is to have you rate the crackers now, before the electric shock, to see how the crackers taste under normal circumstances, and then rate them again, after the shock, to see what changes in your ratings the shock has made.

Before the subjects ate they filled out a very brief questionnaire designed to evaluate the effectiveness of the manipulations. This was explained to the subject as a means of evaluating the effects of his mood and bodily states on his taste discriminations. That this manipulation of fear was effective is demonstrated by responses to the two 5-point rating scales designed to measure degree of fear. These were headed:

> How anxious do you feel at present?
> How nervous or uneasy do you feel about taking part in this experiment and being shocked?

On the first of these scales, high-fear subjects average 1.70 and low-fear subjects average .96 ($t = 3.81$, $p < .001$). To the second question, high-fear subjects score 2.02 and low-fear subjects 1.34 ($t = 3.04$, $p < .01$). The manipulation succeeded in creating differential fear in the two groups of subjects.

Measuring Eating

The questionnaire answered, the experimenter provided each subject with water, pulled a sliding partition in order to completely separate the two subjects, and then left the subjects alone for 15 minutes during which time they tasted and rated crackers. The subjects were under the impression that they were tasting while, through one-way mirrors, observers simply counted the number of crackers eaten. This experimental disguise was designed to cope with the possibility that some subjects, particularly obese ones,

[2] The gaucheness of these condition labels is unfortunate but inevitable. We assume it is self-evident that given the line of reasoning underlying this study we cannot speak of high or low *hunger* conditions.

might be self-conscious and restrained about eating in an experiment. Subjects in this study do not eat, they taste.

The taste rating scales were deliberately designed so that most of the subjects were able to complete them within 10 minutes. When the subject had completed these ratings, he was left with nothing to do for exactly 2 minutes, free to nibble or not at the crackers as he chose.[3] At the end of this interval the observer signaled the experimenter who promptly entered the room with a new set of taste rating scales which kept almost all subjects fully occupied for the remainder of the eating period.[4] This period over, the experimenter returned, and announced that shocking was about to begin, but asked the subjects first to fill out the same questionnaire on mood and bodily state that they had answered immediately before tasting the crackers. This much done, the experiment was over, the deceptions and their rationale were explained in detail, and the subjects were impressed with the necessity for not talking about the experiment to other students. Subjects in the empty-stomach condition were fed roast beef sandwiches, all subjects filled out a weight-history questionnaire, and finally each subject was weighed and measured.

We have then a measure of the eating behavior of subjects who were empty or full and who were frightened or calm. Finally, of course, there were two groups of subjects—obese and normals. Three variables are then being covaried—preloading, fear, and obesity—in an eight-condition factorial experiment.

Subjects

To provide a pool of subjects, data on the weight, height, and age of Columbia students were obtained from university records and classroom surveys. From these data, the percentage of weight deviation was computed using norms published by the Metropolitan Life Insurance Company (1959).

Potential subjects were contacted by telephone and asked to take part in an experiment on taste. Roughly 70% of all those contacted agreed to do so. The decision was made to consider all students who were 15% overweight or more as obese and those who were 10% overweight or less as normal. Inevitably anomalies of classification occurred, for either some of the students were not particularly accurate in reporting their own measurements or their weight or height had changed. In some cases, subjects who were originally classified as obese proved, when weighed after the experiment, to be lighter than subjects originally classified as normal. Even with such subjects eliminated, there still remained such absurdities as a subject originally classified as obese, proving to be 12.3% overweight on weighing, whereas a subject originally classified as normal proved to be 12.1% overweight. In order to cope with such classification problems, the final distribution of subject weight deviations was examined for those cutoff points which permitted roughly a 5% weight differential between the lightest obese and the heaviest normal subject and at the same time involved the absolute minimal loss of subjects. These cutoff points proved to be 9% overweight for the normal group and 13.6% overweight for the obese group. Eight subjects whose weight deviations fell between these points were automatically eliminated from the data analysis. In addition, all athletes (six subjects), members of the varsity squad in some body-contact sport, were automatically eliminated as subjects. Behnke, Osserman, and William (1953) have demonstrated that football players, though overweight by standard weight charts, are not obese, for their excessive weight is due to disproportionately large muscle and bone content, not to high body fat—a fact which is small surprise to experimenters who, anticipating a nice, plump boy as a subject, found themselves with a Greek god.[5] Finally, eight students (three obese and five normal) were eliminated, for though they had gone without the requested meal they had eaten a snack at a non-meal time.

The experimentally relevant characteristics of the final group of subjects are presented in Table 1. On all characteristics except, of course, weight and percentage of weight deviation the various experimental groups are all quite similar. Obese subjects outweigh normals by an average of 31.5 pounds and are on the average 25.1% overweight as compared with the normal group average of 2.3% overweight.

RESULTS

To review the expected results briefly: If it is correct that gastric contractions and hypoglycemia signal "hunger" for normal subjects and not for obese subjects, if these experimental manipulations have succeeded in alter-

[3] This 2-minute period was introduced in order to determine if the anticipated relationships would be affected by a "free" eating situation in contrast with the experimentally required eating of the "tasting" situation. The pattern of results obtained in the two eating situations were precisely the same. For purposes of expositional simplicity, we have combined these two types of data in presenting the results of the experiment.

[4] A few subjects finished this second set of rating scales before the 15-minute eating period was over. When this happened the observer signaled the experimenter to bring the subject a third set of rating scales. Since the number of crackers eaten is to some extent affected by the number of rating scales filled out, possible effects of this artifact were examined. The experimental effects cannot be accounted for by this artifact.

[5] These problems of classification are, in large part, responsible for the clumsily unequal condition *N*s revealed in Table 1. In later experiments (Nisbett, 1968; Schachter & Gross, 1968) we were able to cope with these problems by obtaining more reliable and recent records of height and weight, by establishing initially more extreme cutoff points, and by learning before hand whether or not a potential subject was an athlete.

TABLE 1

PHYSICAL CHARACTERISTICS OF THE SUBJECTS

Condition			N	Age	Height in in.	Weight in lb.	Mean % weight dev.	Range of % weight dev.
Weight	Fear	Preloading						
Obese	High	Empty	10	21.2	69.7	185.9	+23.8	+13.6 to +40.2
Obese	Low	Empty	11	20.5	69.1	186.0	+25.7	+14.6 to +61.4
Obese	High	Full	11	20.2	68.5	180.4	+24.0	+13.7 to +74.5
Obese	Low	Full	11	20.0	68.5	184.0	+26.8	+14.0 to +45.3
All obese *Ss*			43	20.5	68.9	184.1	+25.1	+13.6 to +74.5
Norm	High	Empty	11	19.6	69.4	148.9	+0.2	−8.1 to + 7.0
Norm	Low	Empty	10	19.3	70.6	158.7	+2.9	−2.7 to + 8.7
Norm	High	Full	14	20.5	69.8	154.8	+2.4	−7.6 to + 9.0
Norm	Low	Full	13	19.8	68.2	148.9	+3.4	−5.5 to + 8.4
All normal *Ss*			48	19.9	69.4	152.6	+2.3	−8.1 to + 9.0

ing these physiological states, it should follow that: (*a*) Fear will reduce the amounts eaten by normal subjects and will not do so for obese subjects; (*b*) preloading will reduce the amount eaten by normal subjects and will not do so for obese subjects.

No predictions are made about the relative amounts eaten by obese and normal subjects in the several conditions, for, of course, nothing is known about base-rate eating by these two groups of subjects in this experimental setup.

The data necessary to test these predictions are presented in Table 2. It will be noted immediately that the predictions are supported, for the two basic interactions are significant. Considering first the effects of preloading it is no surprise that normal subjects eat fewer crackers when their stomachs are full of roast beef sandwiches than when they are empty. Normal subjects in the two full conditions consume an average of 15.32 crackers, and in the two empty conditions, an average of 21.89 crackers. The obese stand in fascinating contrast. They eat as much, in fact slightly more, when their stomachs are full (18.65 crackers) as when they are empty (17.89 crackers). Obviously, within this context, the actual state of the stomach has nothing to do with how much the obese eat.

Turning to fear, we note much the same picture. High fear markedly decreases the number of crackers normal subjects eat and has no effect on the amount eaten by the obese. Again there is a small, though nonsignificant reversal, with the fearful obese eating slightly more than the calm obese.

It is interesting to note that the overall amounts eaten by obese and normal subjects are virtually identical. Combining conditions, obese subjects ate an average of 18.3 crackers and normal subjects, 18.1. The Schachter and Gross (1968) experiment also found no difference in the overall amounts eaten by obese and normal subjects. Crackers were the test food in both studies and we believe that this fact accounts for the findings. Crackers are, after all, a neutral sort of food, neither liked nor disliked by most people. Nisbett's (1968) experiment has demonstrated that only when

TABLE 2

EFFECTS OF FEAR AND PRELOADING ON THE EATING BEHAVIOR OF NORMAL AND OBESE SUBJECTS

Condition	A. Normal *S*		B. Obese *S*	
	N	Average no. crackers eaten	*N*	Average no. crackers eaten
High fear, Full	14	13.78	11	19.64
High fear, Empty	11	15.89	10	19.60
Low fear, Full	13	16.98	11	17.66
Low fear, Empty	10	28.28	11	16.34

Analysis of variance

Source	*SS*	*df*	*F*
Preloading (P)	18.21	1	3.09
Fear (F)	13.44	1	2.28
Weight deviation (W)	.34	1	.06
P × W	27.34	1	4.63*
F × W	54.34	1	9.21**
P × F	7.74	1	1.31
P × F × W	13.60	1	2.30
Error	488.89	83	

* $p < .05$.
** $p < .01$.

the food is good do obese out-eat normal subjects.

DISCUSSION

There appears to be little question that the eating of the obese is not related to the same set of bodily symptoms as is the eating of normal subjects. Whether one measures gastric motility, as in Stunkard's studies, or manipulates it, as we assume we have done in this study, there is a high degree of correspondence between the state of the viscera and the amounts eaten by normal subjects and little correspondence for fat subjects.

Clear-cut as these facts appear to be, they do pose a major dilemma. Obviously the obese are fat; obviously they eat. The feelings associated with gastric motility and hypoglycemia appear to trigger eating in normal subjects. What triggers eating for the obese? Given the fact that the bodily circumstances that prompt a self-report of hunger and subsequent eating seem to have little in common for obese and normal subjects one may conjecture that: (*a*) The obese label an entirely different set of physiological symptoms and feeling states as hunger, or that (*b*) the obese label no bodily states as hunger and their eating is unrelated to any physiological condition, in which case, since the obese obviously do eat, we must assume that their eating is triggered either by psychic states such as anxiety, fear, loneliness, feelings of unworthiness, and so on, or by external food-related stimuli such as the smell of food, the sight of other people eating, discussions of food, and so on.

Considering these alternatives, let us turn first to the hypothesis that psychic states precipitate eating in the obese, a hypothesis which unquestionably is the single most pervasive theme in psychosomatic theorizing about obesity (see Kaplan & Kaplan, 1957, for a review of the literature). In essence, such treatments assume that overeating by the obese represents an attempt to cope with anxiety or fear or emotional disturbance of some kind. This assumption is so accepted as a working hypothesis by experienced psychotherapists that we are reluctant to question it without a wealth of relevant data, but the simple fact is that to date we have found no evidence to support it. Certainly the results of the present experiment offer little support, for as Table 2 shows, in the high-fear conditions, obese subjects eat only trivially and nonsignificantly more ($t = 1.28$, $p > .20$) than they do in low-fear conditions. In an internal analysis of the high-fear data, as one would expect, for normal subjects there is a correlation of $-.42$ ($p < .05$) between self-ratings of fear and the number of crackers eaten. For the obese the correlation is .13, in the direction required by the psychosomatic hypothesis, but again a nonsignificant relation.

TABLE 3

REDUCTION IN FEAR PRODUCED BY EATING

Ss	*N*	Mean ratings after eating minus mean ratings before eating	
		How anxious are you?	How nervous about shock?
Obese	43	−.23	−.02
Normals	48	−.30	−.27

The effect of eating on anxiety level is another datum from this study relevant to any psychosomatic formulation which rests on the assumption that, for the obese, eating is anxiety reducing. Table 3 presents indexes derived by subtracting subjects' self-ratings of fear before they had eaten crackers from their ratings after eating. For both groups of subjects there is a slight decline in fear and there are no differences of consequence between obese and normal subjects. If anything, the trends are in a direction opposite to that required by the psychosomatic hypothesis, for on both scales fear is reduced more for normal than for obese subjects.

In an ongoing longitudinal study of the weight of graduate students during the Stürm und Drang of graduate study, Schachter and Nesbitt have as yet found little indication that the fatter students gain weight during examination periods or at other times of personal stress. Given the weight of clinical experience we do not feel free to reject the psychosomatic hypothesis, but obviously our own data compel us to turn to more serious consideration of other alternatives.

Let us turn next to the possibility that the obese label an entirely different set of physio-

logical symptoms as hunger—a logical possibility, certainly—but which symptoms? Schachter (1964) has suggested that the obese label as hunger the state of arousal of the sympathetic nervous system—an elegant hypothesis, for if correct it not only provides a physiological underpinning for the psychosomatic thesis, but allows integration of the psychosomatic and the "labelling" view of matters. Elegant or not, we have been able to muster no particular support for this notion. Certainly the effects of the fear manipulation in this experiment argue as strongly against this hypothesis as they do against the psychosomatic thesis. Second, in a series of case studies (described in more detail in Schachter, 1967) in which we attempted experimentally to separate the "psychic" from the physiological components of the arousal state by use of a disguised injection of adrenalin, we found no evidence that the obese ate more when injected with adrenalin than when injected with placebo.

If not the state of sympathetic activation, then what? We confess a certain feeling of helplessness, for, short of utter absurdities, it is difficult to conceive of a set of alternative physiological symptoms that one can, with any justification, suggest. Nor are the obese themselves of much assistance, for they seem as inept at describing their visceral states as are any of the rest of us when, not sick, we are suddenly asked to describe what is going on beneath the skin. Casual interviews with the obese centered on the theme, "What do you feel when you say, 'I'm hungry?' " more often than not terminate with an exasperated "I don't know, I just feel like I want to eat."

All of which has led to serious consideration of the possibility that internal state is irrelevant to eating by the obese, and that external, food-relevant cues trigger eating for such people. The following three papers (Goldman, Jaffa, & Schachter, 1968; Nisbett, 1968; Schachter & Gross, 1968) explore experimental and theoretical implications of this suggestion.

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(Received December 1, 1967)