



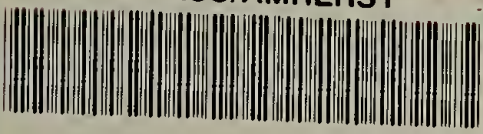
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## **Anxiety and eating in the overweight: the psychosomatic hypothesis revisited.**

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ANXIETY AND EATING IN THE OVERWEIGHT:  
THE PSYCHOSOMATIC HYPOTHESIS REVISITED

A Thesis Presented

By

BRUCE BRITTON KERR

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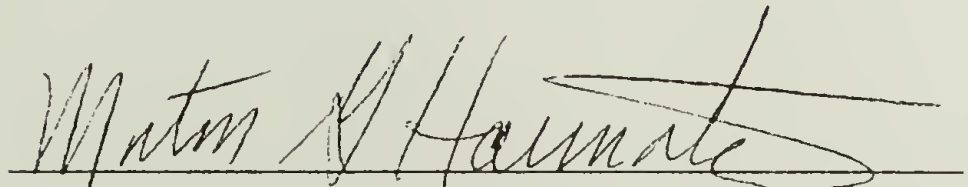
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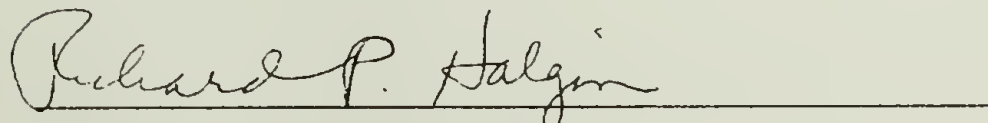
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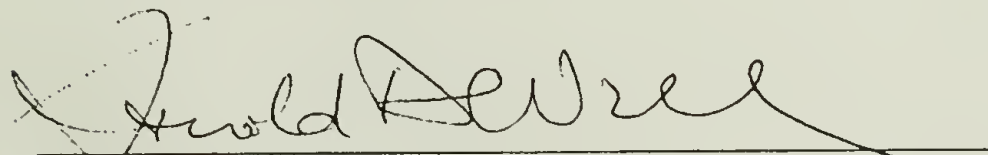
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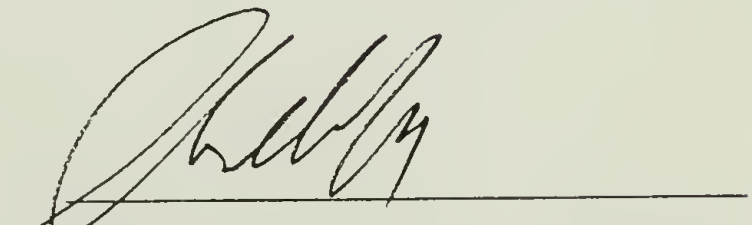
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## C H A P T E R    I

### INTRODUCTION

It has been estimated that as many as 80 million people in this country are overweight (Stuart & Davis, 1972), and excessive weight is certainly a major problem in our society. While there are some unusual physical conditions which can cause a person to be overweight, the most common cause is the intake of food in excess of energy expenditure, or, put more simply, overeating (Stunkard, 1974). The cause(s) of overeating itself, however, is (are) still very much in dispute.

One explanation of overeating which has been advanced by various clinicians working with this problem has been the "psychosomatic hypothesis." This hypothesis makes two claims: (1) that increased anxiety causes increased eating among overweight individuals, and (2) that such anxiety induced eating reduces the levels of felt anxiety. While there is some debate as to the best explanation of this connection between anxiety and increased eating [explanations ranging from conditioned response (Kaplan & Kaplan, 1957) to substitute gratification (Kiell, 1973)], there is a general agreement among clinicians who work in this area that anxiety is frequently an important factor in overeating patterns.

In spite of this clinical agreement, however, the three experimental tests of the psychosomatic hypothesis which have been performed to date have failed to support either of its predictions. Schachter, Goldman, and Gorden (1969) threatened subjects with either very mild or

very painful electric shock, and then measured the amount of food they ate in a decoy tasting task. They found that overweight subjects did not eat significantly more food when threatened with severe shock, nor was there any significant reduction in this group's anxiety scores after they had eaten. McKenna (1972) criticized the Schachter, Goldman, and Gorden study for its use of bland, unappetizing food (crackers) and re-examined the question using a highly appetizing food (cookies). He exposed some of his subjects to various medical equipment (needles, etc.) with the hint that they were for use later in the experiment, and then measured how much of the free food subjects ate while waiting. He found no significant increase in the eating of the overweight subjects who had seen the equipment, and no significant reduction in their levels of anxiety after they had eaten. He argued, however, that there may in fact have been such a decrease which his measures did not detect due to the delay in their administration. Herman and Polivy (1974) looked at the question once again to evaluate this claim. They used a good tasting food (ice cream) and an immediate measure of post consumption anxiety, combined with a manipulation similar to Schacter's: threatening subjects with a painful electric shock and then measuring consumption in a dummy taste rating task. They found no increased eating among the subjects threatened with the painful shock, nor any anxiety reduction effect after eating. Rodan (1977), in her review of these studies, concluded that "thus far experimental attempts to study the psychosomatic hypothesis have been relatively unsuccessful," and that no study to date had produced significant evidence to support either half of the psychosomatic hypothesis.



There are a number of possible reasons that experimental attempts to validate the psychosomatic hypothesis have failed. The most obvious, of course, would be that the psychosomatic hypothesis is indeed false, and this is the conclusion that is often suggested in summaries of the work in this area (Rodan, 1977; Schneider, 1976). The clinical data, however, and the self-reports of many overweight individuals, so often suggest that there is such a relationship that dismissal of this hypothesis on the basis of the failure of a handful of studies to support it seems premature.

A more likely explanation of the failure to experimentally validate the psychosomatic hypothesis lies in the way in which the previous studies have conceptualized and induced anxiety in the laboratory. Each of these studies used the threat of physical pain from some clear object or event, such as the threat of electric shock or the use of needles, to produce their emotional manipulation. Clinical reports in this area (Bruch, 1961, 1973), however, seldom if ever feature the fear of such objective stimuli as the basis for overeating behavior. More often it is interpersonal and intrapersonal types of anxiety that are mentioned in these reports. The crucial assumption that was made in each of these studies was that fear inductions would be adequate analogues to the types of anxieties mentioned in clinical reports. This assumption, however, was not warranted because it was based on an overly simplified conceptualization of anxiety. The position that, once produced, "anxiety" (or fear) is "anxiety" rests on a model of anxiety which suggests that anxiety is an underlying, homogeneous entity, varying in quantity but not in quality, across individuals. Such a homogeneous

conceptualization of anxiety would imply that the use of a single standardized fear stimulus would induce a single, similar state across all subjects ("anxiety") which would be the functional equivalent of other types of anxiety, including those discussed in the clinical observations from which the psychosomatic hypothesis was derived.

A more sophisticated conceptualization of anxiety, on the other hand, would argue that such homogeneity cannot be assumed because (1) the intrapersonal phenomenon that each individual labels as anxiety cannot, a priori, be assumed to be invariant across individuals, (2) the stimuli which elicit that phenomenon cannot be assumed to be invariant across individuals, and (3) within subjects, the response of eating cannot be assumed to be equally associated with different sets of anxiety eliciting stimuli. It would, therefore, argue against the use of a single standardized fear inducing stimulus, because it would not assume that such an induction would produce the same end state in all subjects, or that the end states that it did produce would necessarily be the functional equivalent of the types of phenomena associated with overeating in clinical reports. Instead, such a conceptualization would argue for an anxiety induction technique that could take into account intersubject differences in the experience of anxiety, and which would be an appropriate analogue to the types of anxiety situations referred to in the clinical observations on which the psychosomatic hypothesis was based.

A second major flaw in the experimental tests of the psychosomatic hypothesis to date is their failure to control for possible intersubject differences in the responsiveness of eating to feelings of

anxiety. This reflects the premise in these studies that any connection between anxiety and overeating that does exist would be basically the same across all overweight individuals, and that excess weight is the end result of the same general process in all overweight individuals. Several clinical writers, however, who support the psychosomatic hypothesis, clearly suggest that it may not be functioning equally in all overweight individuals (Bruch, 1961, 1973; Hamberger, 1951).

Instead, they hold that the population of overweight individuals may be broken down into subgroups, only some of which would exhibit this effect. For example, Bruch (1961, 1973) has proposed three subgroups of overweight individuals, only two of which (developmental and reactive) would be expected to show this effect, and Hamberger (1951) proposed four subgroups, only three of which might show eating in response to anxiety, and these in different degrees. While these writers have each arrived at somewhat different subgroups, there is a general agreement among them that overeating and overweight need not always be the product of anxiety, and that eating as a response to anxiety is not uniformly distributed across all overweight individuals.

Some initial empirical evidence which supports and expands such a multiple model of overeating was produced in a study by Harmatz and Kerr (1978). In that study, approximately two hundred subjects completed the "Eating Survey Schedule" (ESS), a self-report instrument designed to assess the responsiveness of an individual's eating behavior to certain internal or external events. Factor analysis of the data resulted in five factors, or dimensions, along which an individual's eating behavior could be considered. These were labelled "anxiety



reduction," "stimulation," "craving-habit," "handling," and "pleasurable relaxation." A cluster analysis of the overweight subjects in the sample indicated the presence of at least two subgroups with significantly different profiles across the five factors, the strongest differences being on the first factor, "anxiety reduction," and the third factor, "craving-habit." These results, although not conclusive, suggest that there is a relationship between anxiety and eating behavior in the overweight, but that that relationship is not uniform across all individuals. Instead, the relationship appears to vary from person to person such that anxiety is an important factor in eating for some overweight individuals, but not for others.

To date, experiments testing the psychosomatic hypothesis have not taken into account the possibility of intersubject differences in the responsiveness of an individual's eating behavior to anxiety, and it may well be that the failure to do so has resulted in increased heterogeneity of the samples used, thus masking any effect that might be present in some individuals. The assumption that all overweight individuals overeat for the same reason, or that the psychosomatic hypothesis either applies equally to all overweight individuals or to none, is not a necessary assumption of the psychosomatic hypothesis, or of a multiple model of overeating behavior, and, therefore, future tests of the psychosomatic hypothesis should control for intersubject differences in the responsiveness of eating behavior to anxiety.

In sum, there is reason to believe that the experimental tests of the psychosomatic hypothesis performed to date have not been adequate tests of that hypothesis due to flaws in their conceptualization of

anxiety, and the possible heterogeneity of the samples used. Taking these issues into account, the present study was designed to provide a test of the relationship between anxiety and eating behavior in overweight individuals which was sensitive to intersubject differences in the experience of anxiety, and in the responsiveness of eating behavior to feelings of anxiety. A group of overweight subjects was screened to determine how responsive their eating behavior was to anxiety, and then half were moved toward a state of increased anxiety, and half toward a state of calm. In each case, the stimuli used in the induction were provided by the subjects themselves. Subjects were then given access to food with a decoy taste rating task, and consumption measured. Pre and post measures of anxiety were recorded. Two hypotheses were generated: (1) that increased anxiety would result in increased eating, but more so in those subjects for whom anxiety was a strong cue for increased eating, and (2) that there would be a significant decrease in levels of anxiety after eating for those subjects for whom anxiety was a strong factor in their overeating.

C H A P T E R    I I  
METHODS AND PROCEDURES

The experiment was in two parts: (1) a mass testing to develop a pool of appropriate subjects, and (2) an experimental session which included (a) a manipulation of the subjects' level of anxiety with pre and post measures, (b) a measure of food consumption, and (c) a post experimental debriefing. These are discussed in detail below.

Subject Screening

An experimental testing session was announced. All subjects were male and female undergraduate psychology student volunteers who participated in the experiment for extra course credit. Subjects were asked to fill out four psychological questionnaires. Of these, three acted as filler, and the other, always presented first, was the Eating Survey Schedule (ESS) described above. Anxiety reduction factor scores were derived from this scale for each subject and represented that subject's estimate of how responsive his or her eating behavior was to cues of anxiety. The ESS also gathered information on height, weight, age, and sex of each subject, and this information was used to derive the subject's relative weight, which was defined as their actual weight divided by the ideal weight for a person of that age, sex, and height as given by the Metropolitan Life Insurance Company actuarial tables. Subjects for the experimental session were selected from this pool.



### The Experimental Session

The experimental session was limited to females for three reasons. First, our culture makes even moderate overweight more of a stigma for females than males. Second, normative data gathered earlier on this same population suggested that the MILC tables overestimated the ideal weight of females as compared with peer norms by about ten percent. In other words, the subjective norms of appropriate weight in this population were about ten percent below those listed in the table. Thus, any bias in the use of these tables as a criterion would be in the direction of excluding any individuals likely to be perceived, by self or others, as being of normal or low weight. Third, the use of only females avoided the possible complication of sex differences in this initial test.

A relative weight of 110 or above was used to place an individual in the overweight category. All females who qualified as overweight were then divided into two groups: "high anxiety responsive eaters" and "low anxiety responsive eaters," on the basis of their ESS anxiety reduction factor scores. The median score (possible range 1 to 5) of 2.705 was used as the dividing line between the two groups. Twenty subjects were then selected at random from each of the high and low groups, and were invited to participate in an experiment on the perception of taste. The average time between the two sessions was about one week.

Subjects were telephoned and an experimental session time was arranged. During that phone call, subjects were instructed to eat a light meal or snack two hours before their scheduled time, and to not

eat after that. Upon arriving at the experiment, the subject was greeted by the experimenter, a third year male clinical graduate student, and seated in a small experimental room equipped with a table and several comfortable chairs. She was then read an introduction to, and description of, the experiment. The major points of the script (see Appendix A for full text) were that (1) the experiment was looking to see if the perception of taste was affected by emotional factors, that (2) subjects would be asked to taste and rate several different foods along a number of standard taste dimensions after experiencing a mood induction, and (3) subjects would be asked to fill out a brief mood check list before and after the induction as an ongoing measure of their mood so that this could be compared with their taste ratings.

The anxiety induction procedure used was a modification of one developed by Strickland (1979) for work on depression. Subjects were asked to relax in a comfortable chair and relate to the experimenter an incident in which they could still vividly recall having been very "calm, relaxed, at ease" or very "tense, anxious, and nervous" depending on their induction condition, anxious or calm (see Appendix A for full text). The experimenter then repeated the story back to the subject, who was instructed to relive the emotions she had just described. This technique allowed each subject to define for herself a stimulus which she identified as anxiety arousing or calming to her, thus allowing for inter-subject variability in the experience of anxiety. Strickland's (1979) use of this technique to elicit feelings of depression, rather than anxiety, produced significant differences on physiological measures of GSR and EMG, and on self-report measures such as the MAACL version of the

### Adjective Check List.

Subjects were assigned at random to one of the two anxiety conditions, and this assignment was not revealed to the subject until after she had completed the pre-induction administration of the mood check list to ensure that that premeasure was not biased by the assignment. The experimenter was aware of the subject's anxiety induction condition, but was not aware of the subject's factor score assignment (high responsive or low responsive).

The state form of the State Trait Anxiety Inventory (STAI) (Spielberger, 1970) was used to assess the subject's level of anxiety during the experiment. It is a carefully validated clinical and research scale which shows very low test-retest effects over periods as brief as one hour ( $r = .16$  for females). This low test-retest effect, while usually not desirable in a test, is desirable in this case as the test is designed to be sensitive to moment to moment changes in anxiety levels, rather than long term consistencies. The value for coefficient alpha, a measure of internal consistency which is a more appropriate measure of the reliability of the scale given the transitory nature of the phenomenon it seeks to measure, for a standardization sample of female college undergraduates was .89, which demonstrates that the scale has a high degree of internal consistency. Thus, the reliability data on this test show it to have the optimal characteristics for a test of state anxiety: low stability across time yet a high degree of internal consistency. The STAI was given three times: pre-induction (m1), post induction (m2), and post food consumption (m3).

After the anxiety induction was completed and the subject had



filled out the STAI for the second time (m2), she was seated at a table on which were three covered bowls of food labelled "1," "2," and "3," and a taste rating form. The foods were always in the same order, since total consumption was the dependent measure of interest, not preference for any specific food. The subject was told that she was now ready to perform the taste rating task (see Appendix A for full text), and was instructed to continue to think about and relive the emotions from her induction story while she tasted and rated each of the foods on the form provided. The experimenter then left the room, so as not to distract the subject, and the subject signaled the experimenter via a push button on the table when she had finished the task. When signaled, the experimenter returned, re-administered the STAI (m3), and then debriefed the subject.

The foods used in the experiment represented three types of snack foods: sweet (sugar wafers), salty (pretzels), and cereal (miniature shredded wheat). Each had the same volume-to-weight ratios when ground up as if chewed, so that equal totals of food consumed across the three trials represented equal volumes in the stomach. Sufficient food was available to each subject to avoid any inhibition which might arise if the subject were able to finish or noticeably decrease the food available. Each bowl of food was weighed on a single arm laboratory gram scale to one one-hundredth of a gram before and after the session, and the amount of food consumed on each trial was calculated by taking the difference between the pre and post weights. The use of different food on each trial was intended to give credibility to the comparative taste rating cover task, and to allow for some variation in individual preferences.

While clearly all possible foods were not used, an attempt was made to cover three major types of easily available snack foods (cookies, pretzels, and cereal) which clinical impression suggests are likely to be involved in overeating patterns.

After the experimental procedure was completed, each subject underwent a post experimental debriefing to assess her perception of the experiment, and the success of the experimental deception. During that time, subjects were asked to answer, in order and in writing, the following questions:

1. Please describe what you did in today's experiment.
2. How effective was the mood induction? Were you able to re-experience the emotions? If so, how realistically? If not, do you know why not?
3. Do you think your mood and your sense of taste are connected? If so, how?
4. More generally, do you think that there are other relationships between your eating and your emotions? What?
5. Please describe what you felt the purpose of today's experiment was.

After answering these questions, subjects were also asked by the experimenter if they were on a diet, and if they had followed the instructions about eating before the session. Subjects then received an explanation of the experiment and had any questions answered.

## C H A P T E R    I I I

### RESULTS

#### Subjects

Three subjects indicated in the verbal debriefing that they were currently on diets and had consciously limited their food intake during the session. They were replaced with three other subjects drawn at random from the same half (high factor group or low factor group) of the subject pool. The relative weights of the final sample had a mean of 118.4 pounds ( $SD = 7.3$ ), and the anxiety reduction factor scores from the ESS, possible range one to five, had a mean of 2.72 ( $SD = 6.92$ ). Examination of the written debriefing forms indicated that none of the subjects had seen through the experimental deception.

Randomization of the subjects on the variables of relative weight and factor score was checked by performing an induction condition (anxious, calm) by factor group (high responsive, low responsive) 2 x 2 analysis of variance on each of the two variables (see Tables 1 and 2). No significant differences in relative weight were found. As expected, the high factor score group had significantly higher factor scores than the low factor score group ( $F = 49.322$ ,  $p = .001$ ), but no significant differences on factor score were found between induction conditions, and there was no significant interaction. A subject's relative weight was not correlated with her factor score ( $r = .0394$ ,  $p = .405$ ), and the time of the subject's session was not correlated with the amount of food she ate ( $r = .01$ ,  $p = .473$ ). The amount of



TABLE 1

ANOVA: Relative Weight by Induction Condition (I.C.)  
and Factor Group (F.G.)

<u>Source</u>	<u>S.S.</u>	<u>df</u>	<u>M.S.</u>	<u>F</u>	<u>p(F)</u>
I.C.	10.00	1	10.00	.180	.674
F.G.	62.5	1	62.5	1.124	.296
I.C. x F.G.	4.90	1	4.90	.088	.768
error	2002.2	36	53.33		

TABLE 2

ANOVA: Factor Score by Induction Condition (I.C.)  
and Factor Group (F.G.)

<u>Source</u>	<u>S.S.</u>	<u>df</u>	<u>M.S.</u>	<u>F</u>	<u>p(F)</u>
I.C.	.486	1	.486	2.285	1.39
F.G.	10.496	1	10.496	49.322	.001
I.C. x F.G.	.038	1	.038	.178	.676
error	7.661	36	.213		

food eaten did show a low but significant negative correlation with relative weight ( $\underline{r} = -.32, p = .02$ ).

Hypothesis #1: Increased Eating under Conditions  
of Heightened Anxiety

The first prediction of the psychosomatic hypothesis is that increased anxiety in an overweight subject will lead to increased eating, although the effect may be stronger in some individuals than in others. The first hypothesis of this study, which was designed to test this assertion, was that if the induction procedure were successful in raising the anxiety level of the subjects in the anxious induction condition, then these subjects should show more eating than the subjects in the calm induction condition, although the effect should be stronger among those people who rate anxiety as an important factor in their own eating pattern (the high factor score group).

In order to see if the induction procedure had been successful, a one between (induction condition), one within (administration) mixed analysis of variance (see Table 3) was performed on the STAI scores. The results showed that there were significant main effects of induction condition ( $\underline{F} = 1896.64, p = .0000$ ) and administration ( $\underline{F} = 28.86, p = .0000$ ), and a significant induction condition by administration interaction ( $\underline{F} = 62.75, p = .0000$ ). Post hoc tests (see Table 5) were conducted using the Bonferroni  $\underline{t}$  procedure outlined by Myers (1972). The critical value for the between group family of tests ( $\underline{df} = 38$ ) was  $\underline{t} = 3.12, p = 0.16$ . The critical value for the two within group families of tests ( $\underline{df} = 19$ ) was  $\underline{t} = 3.32, p = .016$ . The results showed



TABLE 3

ANOVA: Mood Scores by Induction Condition (I.C.)  
and Administration (Ad.)

<u>Source</u>	<u>S.S.</u>	<u>df</u>	<u>M.S.</u>	<u>F</u>	<u>p(F)</u>
I.C.	6840.3	1	6840.3	69.18	.0000
error	3757.567	38	98.88		
Ad.	2380.82	2	1190.41	28.86	.0000
I.C. x Ad.	5176.55	2	2588.28	62.75	.0000
error	3134.63	76	41.24		

TABLE 4  
Mean STAI Scores and Standard Deviations

		<u>Induction Condition</u>	
		<u>anxious</u>	<u>calm</u>
<u>Administration</u>			
	M	39.55	38.50
pre-induction (m1)			
	(SD)	(7.3)	(6.4)
	M	61.55	28.90
<u>post induction</u> (m2)			
	(SD)	(8.0)	(5.5)
	M	40.15	28.55
<u>post consumption</u> (m3)			
	(SD)	(11.5)	(6.5)

TABLE 5

Bonferroni  $t$  Post Hoc Tests on STAI Scores

Bonferroni  $t$ -critical for between group tests ( $df = 38$ ) = 3.12,  $p = .016$ .  
 Bonferroni  $t$ -critical for within group tests ( $df = 19$ ) = 2.32,  $p = .016$ .  
 E.F. = .05, E.C. =  $.05/3 = .016$ .

Between Groups:

<u>contrast</u>	<u>df</u>	<u>t</u>	<u>p(t)</u>
m1 <sup>a</sup>	38	-.48	.633
m2 <sup>b</sup>	38	-15.09	<u>.000</u>
m3 <sup>c</sup>	38	-3.93	<u>.000</u>

Within Groups, Anxious:

<u>contrast</u>	<u>df</u>	<u>t</u>	<u>p(t)</u>
m1-m2	19	-9.75	<u>.000</u>
m2-m3	19	7.54	<u>.000</u>
m1-m3	19	-.22	.798

Within Groups, Calm:

<u>contrast</u>	<u>df</u>	<u>t</u>	<u>p(t)</u>
m1-m2	19	6.34	<u>.000</u>
m2-m3	19	.29	.774
m1-m3	19	6.24	<u>.000</u>

<sup>a</sup>m1 pre-induction administration.

<sup>b</sup>m2 post induction (preconsumption) administration.

<sup>c</sup>m3 post consumption administration.



that there are no significant differences in the pre-induction (m1) STAI scores, but there was a significant difference in the post induction (m2) STAI scores ( $df = 38$ ,  $t = -15.09$ ,  $p = .000$ ). This was the result of a significant increase in the STAI scores of the anxious induction condition group ( $df = 19$ ,  $t = -9.75$ ,  $p = .000$ ), and a significant decrease in the STAI scores of the calm induction group ( $df = 19$ ,  $t = 6.34$ ,  $p = .000$ ). In addition to the analysis of the STAI scores, the effectiveness of the induction was checked via examination of question number 2 on the debriefing form. All subjects reported that they felt the induction had been effective. Thus the condition of heightened anxiety, necessary for the test of the first part of the psychosomatic hypothesis, was achieved.

In order to see if, as predicted by the psychosomatic hypothesis, the anxious group had eaten more food than the calm group, the total amount of food consumed (see Table 6) was analyzed in an induction condition by factor group 2 x 2 analysis of variance. The results, presented in Table 7, show a significant main effect of induction condition ( $F = 6.29$ ,  $p = .017$ ) and indicate that, as predicted, the anxious group ate significantly more than the calm group. There was no significant effect of factor group ( $F < 1$ ), and no significant interaction ( $F < 1$ ). Thus, the results of the first part of this study support the first prediction of the psychosomatic hypothesis: increased eating in overweight individuals under conditions of heightened anxiety. There was no evidence, however, that this effect differed over levels of self-reported responsiveness of eating to anxiety. Instead, the effect was consistent across both the high and low responsiveness groups with

TABLE 6

Cell Means and Standard Deviations for Food Consumption

<u>Factor Group</u>	<u>Induction Condition</u>	
	<u>anxious</u>	<u>calm</u>
M	24.31	15.30
<u>High Responsive</u>		
(SD)	(10.38)	(11.58)
M	20.36	13.92
<u>Low Responsive</u>		
(SD)	(10.24)	(5.71)
M	22.33	14.61
<u>Marginal (averaged over factor group)</u>		
(SD)	(10.24)	(8.91)

TABLE 7

ANOVA: Food Consumed by Induction Condition (I.C.)  
and Factor Group (F.G.)

<u>Source</u>	<u>S.S.</u>	<u>df</u>	<u>M.S.</u>	<u>F</u>	<u>p(F)</u>
I.C.	596.756	1	596.756	6.289	.017
F.G.	71.022	1	71.022	.748	.398
I.C. x F.G.	16.512	1	16.512	.174	.679
error	3416.049	36	94.890		



subjects in the anxious condition eating more than subjects in the calm condition regardless of factor group membership.

Hypothesis #2: Decreased Levels of Anxiety after Eating

The second prediction of the psychosomatic hypothesis is that heightened anxiety in the overweight will be reduced after eating. The Bonferroni  $t$  post hoc tests (Table 5) on the analysis of STAI scores indicated that there was a significant reduction in the anxiety levels of the anxious group after they ate ( $df = 19$ ,  $t = 7.54$ ,  $p = .000$ ). In fact, their anxiety levels returned to their pre-induction level ( $df = 19$ ,  $t = -.22$ ,  $p = .798$ ). The anxiety scores of the calm group, however, were not changed by the food consumption ( $df = 19$ ,  $t = .29$ ,  $p = .774$ ), and remained significantly lower than their pre-induction scores ( $df = 19$ ,  $t = 6.24$ ,  $p = .000$ ). Post hoc tests of this interaction effect were calculated by computing the sum of squares on one degree of freedom for the relevant contrast and dividing it by the error term for the overall interaction effect (Myers, 1972). These showed that the pre-consumption ( $m_2$ ) difference between the group means was significantly larger than the post consumption ( $m_3$ ) difference between group means ( $F = 53.72$ ,  $p = .001$ ), which in turn was significantly larger than the pre-induction ( $m_1$ ) difference between the group means ( $F = 13.5$ ,  $p = .001$ ). Thus, as predicted, there was a significant decrease in the anxiety levels of the anxious group after they ate, and there was no change in the anxiety levels of the calm group after they ate.

Induction Stimuli

Table 8 gives a breakdown of the types of scenes produced by the subjects in each induction condition. Interpersonal anxiety situations were most common in the anxiety condition ( $\underline{n} = 12$ ), followed by school- ( $\underline{n} = 6$ ), and job- ( $\underline{n} = 2$ ) related performance concerns. Vacation scenes were most common in the calm condition ( $\underline{n} = 13$ ).

TABLE 8

Breakdown of Induction Scenes Produced in each Induction Condition

<u>Induction Condition</u>	<u>Theme of Scene Produced</u>	<u>n</u>
<u>Anxious</u>	<u>Interpersonal</u>	<u>12</u>
	involving family	(1)
	involving superior	(2)
	involving friends	(3)
	involving strangers	(3)
	involving leaving home for school	(3)
	<u>School/Job Performance</u>	<u>8</u>
	crucial exam	(6)
	other	(2)
	<u>Calm</u>	<u>Vacation</u>
<u>Post Exam/Work Stress</u>		<u>4</u>
<u>Other</u>		<u>3</u>



## C H A P T E R    I V

### DISCUSSION

The psychosomatic hypothesis makes two general predictions. The first is that heightened anxiety will lead to increased eating in overweight subjects, and the second is that increased eating in overweight subjects will decrease feelings of heightened anxiety. The results of this study provide positive evidence to support both of these predictions. Anxious subjects ate significantly more than calm subjects, thus confirming the first predictions, and anxious subjects showed a significant decrease in their levels of anxiety after they had eaten, thus confirming the second hypothesis.

Previous studies, cited above, which have attempted to demonstrate these effects in the laboratory have failed, and there is one major methodological difference between those studies and the present study which likely accounts for this difference in outcomes: the type of induction procedure which was used in each study to induce heightened levels of anxiety. Each of the previous studies used a single standardized stimulus which was predefined by the experimenters as anxiety arousing. This study, on the other hand, used individualized stimuli which were identified by the subject as anxiety arousing for her. Thus, each subject in the present experiment was allowed to produce an induction stimulus which was sensitive to her own personal experience of anxiety, and this procedural difference had a clear effect on the induction stimuli which were finally used. None of the induction stimuli

produced by the subjects in the present study involved the types of stimuli which were used in the previous studies, that is, the threat of physical pain from some objectively pain producing object or event. None of the subjects in the present study responded to the experimenter's request for a scene in which they had felt "very tense, anxious, and nervous" with a scene in which they had felt fear over some impending physical pain. Instead, the anxiety induction stimuli used in this study sampled the types of inter and intrapersonal anxieties reported in the clinical observations upon which the psychosomatic hypothesis was originally based. They were better analogues to the types of feelings which induce increased eating in the natural environment, and were therefore more likely to induce this effect in the lab. The anxiety induction stimuli used in the previous studies did not sample the types of stimuli commonly associated with the phenomena in vivo, were inaccurate analogues, and were therefore less likely to produce the effect in the laboratory. Thus, the likely reason for the failure of the previous studies to produce results similar to the present study was their collective failure to use adequate and representative induction stimuli.

Given that this study has produced a successful demonstration of the effects predicted by the psychosomatic hypothesis, further research is now required to explain these effects, i.e., further work is needed to explain exactly how anxiety leads to increased eating in overweight individuals, and how such eating operates to produce a reduction in the levels of felt anxiety. There are a number of possible explanations already in contention as to how increased anxiety could lead to

increased eating. The explanation originally proposed to explain this effect was a psychodynamic one which made the general argument that increased eating in the presence of anxiety reflected the unconscious wish for gratification and the substitution of food for more primary, but anxiety arousing, sources of gratification. This has never been proven to be the case, however, and since primacy yields no special status in the search for explanation, it must still be considered only one of several theories until confirmed or disproven. One problem with this type of explanation, as noted by Rodan (1977), is that it views eating as a symptom and implies a degree of underlying psychopathology in the population of overweight individuals as a whole which many feel is inaccurate and not supported by any firm evidence. Rodan (1977) has put forth an alternative explanation which she feels does not imply this underlying psychopathology. She has argued that, while there is no evidence for the psychosomatic effect of increased eating under conditions of anxiety, even if there were, this would not necessarily mean that the anxiety is the direct cause of the increased food consumption. Rather, it may be only the indirect cause via its power to disrupt the self control mechanisms of a person who can be thought of as being in a state of chronic hunger. Thus the psychosomatic effect would not be a sign of any underlying psychopathology, but rather one example of a much larger phenomenon, the disruption of restraint in a deprived organism. Rodan's argument is, *prima facie*, an explanation which could fit the data, although it is not immediately obvious how it would account for the other effect present in the data, the reduction of anxiety which occurred after the subjects ate. A



third explanation for the effect of increased consumption would be that anxiety is a noxious state whose cessation strongly negatively reinforces any response, including eating, which leads to its termination. This explanation, like Rodan's, avoids the assumption of underlying psychopathology, but it also considers the role that the anxiety reducing effect of eating may play in the general pattern. This explanation also, however, begs the question of exactly how food consumption can produce this decrease in anxiety, and that question too could also be answered in a number of ways. Possible explanations to be explored would include distraction, the effects of time, substitute gratification of unconscious wishes, and/or some action at the physiological level. It is not clear from this study which, if any, of these explanations would best account for the phenomena demonstrated here. It is clear that further empirical work should be done to arbitrate among these competing hypotheses.

Finally, further work is also needed to define the limits of the effect demonstrated here. While one could produce a number of theoretical arguments as to why these effects should or should not be limited to certain groups, it is ultimately an empirical question as to if and how strongly the effects produced in this study would be present in other populations such as males, children, older women, and groups with differing degrees of and/or lengths of history of excessive weight. The question should also be investigated using normal weight subjects, as anxiety induced overeating need not lead to increased weight if other variables, such as activity level or self induced vomiting, intervene. The sample used here represents relatively young subjects who



are somewhat closer to the beginning of their potential careers as overweight persons, and the relationship appears rather clearly in their data. However, excessive weight is not without its consequences, and can produce as well as reflect multiple effects on the social, psychological, and physiological levels. It may well be that in older subjects with a longer history of excessive weight, other changes have occurred, both on the socio/psychological and physiological levels, which have linked eating as a response to many different stimuli. While it would seem unlikely that eating would cease to be a response to anxiety (unless it lost its ability to relieve anxiety or had been replaced by a more dominant response), it would also seem quite likely that anxiety might now only be one of a number of different stimuli, on different levels, which would be associated with the response of eating. At that point, the diagnostic picture would become much more complicated. The effects of anxiety would be confounded with many other relationships, and the psychosomatic hypothesis would not fully account for, if indeed it ever did fully account for, an individual's eating pattern. In the long run, the interaction between humans and food is sufficiently complex that no single principle is likely to fully explain the phenomenon. Therefore, it would be better to consider the psychosomatic effects demonstrated in this study as an important set, rather than the only set, of mechanisms which may fuel an overeating pattern.

While the present study is not a treatment study, there are some general implications for treatment which can be drawn. With subjects similar to the sample used in this study which, while it may not represent some of the more extreme cases of excessive weight, certainly

represents some of the most frequent, early intervention which places relatively more emphasis on the development of alternative ways of coping with anxiety could serve both prophylactic as well as curative functions by eliminating the existing response of overeating and thereby preventing some of the future consequences of unchecked consumption. With more advanced cases, however, more comprehensive treatment programs involving a wide range of psychological, behavioral, and physiological considerations would likely be required and these programs, while they would likely contain some of these coping types of treatments, would probably place a different relative emphasis on them.

There is major work left to be done in this area. The present study demonstrates the effects predicted by the psychosomatic hypothesis and thus sets the stage for future efforts to explore and explain the mechanisms by which they work, and the limits within which they exist. This study does not contain the data to arbitrate these questions, but it does provide the evidence that there is indeed something here that is worth attention and explanation. It also, hopefully, provides a much needed piece of common ground for dialogue between clinicians and experimentalists working in this area.

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A P P E N D I X    A  
EXPERIMENTER'S SCRIPT

In this study, we are investigating the relationship between eating and emotions. Specifically, we are interested in seeing if the perception of taste is affected by the mood you are in. We know that other perceptions, such as the perception of color or of sound, can be affected by mood, and we want to see if this is also true about taste. If it is, it might help us to better understand the connection between eating and mood that many people say exists. In order to do this, we are asking people in the study to taste and rate several foods after experiencing a simple mood induction procedure.

The mood induction procedure is basically very simple. In it you will be asked to think of an incident in which you felt a certain emotion, such as happiness, sadness, tension, excitement, etc., which I will specify. You will then be asked to tell this story to me, and I will then tell it back to you as you concentrate on reliving the mood you have just described. After the induction is finished, you will be asked to taste and rate a variety of foods along a number of standard taste dimensions, and fill out a short feedback form. If you are willing to participate in this study, would you please read and sign this consent form. I will be glad to answer any questions which you may have at this time.

(After consent form is signed) O.K., good. Before we start the mood induction, I would like you to take a minute to fill out this mood

check list. I will be asking you to fill this out several times over the course of the session as a check on your mood. It can be filled out quickly; do not spend too much time on any item; your first impression is usually the best. (Hand out and collect the check list.)

Fine. Now we are ready to begin the induction. Please sit back in the chair, relax, and take a few minutes to think of a specific situation in which you found yourself feeling very ("tense, anxious, and nervous," or "calm, at ease, and relaxed," depending on the subject's induction condition). Since you will be trying to recapture and actually relive these emotions just as you did in the situation itself, it is important that you come up with a specific incident which you can still vividly recall. Take as much time as you need to come up with a specific scene, and let me know when you are ready. Any questions? (Answer any questions and wait until subject is ready.)

O.K.? Good. Now, just sit back and tell me the story with as much emphasis on your feelings of ("tension, anxiety, and nervousness" or "being at ease, calm, and relaxed") as you can. Really get into the feelings, focus on them as much as you can, remember how you felt physically, emotionally, and what you were thinking. Listen to yourself as you describe the feelings. Describe the feelings you felt at the time as vividly as you can, and relive the feelings just as they were back then. When you are done, let me know, and I'll tell the story back to you. When I tell it to you, focus in on the emotions in the story and try to actually relive them just as you did in the original situation. Any questions? O.K. Go ahead.

(After induction) Alright. Now just stay seated and continue to

think about the feelings from your story. Try to recall them even more clearly; picture yourself back there again. As you do that, I'd like you to fill out the mood check list and indicate how you feel right now. (When done, move subject to chair in front of food table). O.K. Now we are ready for the taste task. Take your time and sample each of these foods as much as you like and rate each of them on each of the scales on the sheet. Speed is not important; what we are after are accurate ratings of how these foods taste to you. It is important that you continue to focus in on recalling and reliving the emotions from your story so that you will be in that mood as you taste and rate the food, so I will leave so as not to distract you and after I'm gone take a minute to really picture yourself back (some reference to scene such as "back at that party," etc.) and then go ahead and do the task.

(Return when signaled). All done? O.K. I'd like you to fill out the mood check list one more time. (When done, collect.) Thank you, this concludes the experiment. We have a short feedback questionnaire I'd like you to fill out to help us evaluate your understanding of the experiment. Please answer each of these questions, in order, at the bottom of each page. (When done, collect and debrief.)





