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Mood and appetite during minimal-carbohydrate and carbohydrate-supplemented hypocaloric diets[1,2]

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ABSTRACT After a baseline period of free-feeding, 20 obese outpatients alternated between four 2-wk periods of minimal-carbohydrate diet (800 kcal; 58% protein and 42% fat by weight) and of a carbohydrate-supplemented diet (1,000 kcal; 42% protein, 30% fat, and 28% carbohydrate). In a comparison of psychological adjustment during the baseline and low-calorie diets, the initial 2 wk of dieting was associated with a decrease in appetite and elevation of psychological well-being, regardless of the composition of the diet. Thereafter, appetite and mood approached basal levels. Further changes in these psychological reactions to dieting did not vary with the type of diet. There was no support for the idea that a minimal-carbohydrate, protein-supplemented fast decreases appetite and elevates mood more in comparison with a similar diet containing enough carbohydrate to minimize ketosis. *Am J Clin Nutr* 1985;42:371–379.

KEY WORDS Obesity, protein-supplemented diets, appetite, ketosis

Introduction

Very restrictive, low-calorie diets for weight reduction have been popular among dieters and clinicians alike (1). The most obvious justification for a large versus a moderate reduction in caloric intake is that a greater degree of weight loss can be achieved in a shorter period of time. In a review of over 500 studies of weight reduction treatments, including pharmacotherapy, exercise, behavior modification, and various types of diets, Stuart et al (2) found that very restrictive diets produce the largest weight losses next to total fasting. If fasting is supplemented with a small amount of protein and fat, it is possible to minimize loss of muscle tissue, (although the concentration of the more labile proteins of the body is decreased (3)) while still producing a large weight loss (4–7). Compared with other diets, weight loss from the protein-supplemented fast is very impressive in the short run (8). Larger weight loss and better maintenance can result if behavior modification is given in conjunction with the diet to teach the patient how to adhere to the diet and how to develop long-term weight control strategies (5, 9). Many patients who are looking forward to a major weight loss prefer these diets because they feel they are getting a quick, head start toward their goal. An initial large payoff for their efforts is the incentive for many patients to endure the stress of a long-term weight loss program.

Another presumed advantage of the protein-supplemented fast is that the patient is able to tolerate the lack of food. Most patients do not feel hungry or emotionally stressed

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while adhering to this type of diet. Some patients even report that they feel better, *less* hungry, and happier than before going on the diet. Observations of these positive effects on appetite and mood have been reported in several uncontrolled studies of the protein-supplemented fast, chiefly with hospitalized patients (5, 10–12). In addition to what the patient says about his or her appetite, the length of time of adherence to the diet could be another important indication of the psychological effects. In one comparison study in which attrition was reported, fewer patients dropped out of the protein-supplemented fast treatment than dropped out of the low-calorie balanced diet (13).

One reason why the dieter might be less hungry when undergoing the protein-supplemented fast is that there is a drastic reduction of food cues in the environment. Factors such as routine, less palatable food and less contact with food and dining settings would reduce a patient's expectation and drive to eat (14–16). Another possibility is that the composition of the diet, specifically the lack of carbohydrate, has some influence on appetite and emotional processes. In studies of total fasting, similar effects have been observed after the initial 24–72 h of fasting. Since these changes parallel the appearance of ketones in the blood and urine, it is possible that appetite suppression and mood elevation in fasting are caused by ketosis (17–22) and the accompanying changes in brain neurotransmitters which regulate these processes.

Is the ketogenesis resulting from a low-carbohydrate, protein-supplemented fast responsible for the anorectic effects? After an initial period of food restriction, laboratory animals will increase their food intake if they are given additional carbohydrate (23). By contrast, when laboratory animals are given diets with reduced carbohydrate, they eat less in subsequent meals. Only a few investigators have systematically measured appetite in patients during varying degrees of ketosis. In two previous studies comparing inpatients who received carbohydrate-free diets with those receiving carbohydrate-containing diets higher in calories, there was no evidence of a decrease in appetite during the ketogenic diets (24, 25). In our previous study (26), we provided inpatients with one of two 800-calorie diets: a carbohydrate-free diet with extra fat or a carbohydrate-containing diet. The subjective ratings of appetite and measures of emotions were essentially the same for both groups of patients, regardless of diet. The purpose of the present study was to repeat a comparison of these two types of diets on an outpatient basis. It was possible that an inpatient hospital environment, bare of food cues, had facilitated tolerance of a diet. This study was designed to determine whether there would be an effect on appetite and mood in an outpatient, home-based diet program which had fewer external controls and more food stimuli. Another purpose of this study was to measure several psychological variables simultaneously and to see whether changes in appetite corresponded with changes in mood and self-esteem (or whether the psychological responses were independent of each other).

Methods

Subjects

Thirty volunteers were recruited through newspaper advertisements which offered free diet therapy for participants in a diet study. Potential subjects were excluded if they had hypertension or diabetes or any other medical condition which needed treatment. The protocol was approved by the human experimentation committee of the University of Vermont. Twenty subjects completed the study, 19 females and 1 male, 20- to 38-years-old (mean 29.2 yr, SD = 5.12). All of the subjects had been obese since childhood. The average duration of obesity was 19.25 yr (SD = 8.6 yr). The subjects weighed between 70 and 112 kg (mean 84.95 kg) and were between 30 and 113% above ideal weight (mean 54.9%) (based on Metropolitan Life Insurance Company Norms, 27). All subjects had normal oral glucose tolerance tests and a family history negative with respect to diabetes, excessive birth weights, or gestational diabetes. Ten subjects were dropped from the study because they did not adhere to the experimental diet; six dropped out during a carbohydrate-containing phase, four dropped out during a minimal-carbohydrate phase. These subjects were somewhat older (mean 34.5 yr) and weighed somewhat more (mean 88.37 kg) than the subjects who completed the study. In addition, they reported higher levels of depressed, anxious, and hostile moods in the baseline period, although their psychological test scores on these variables were still within the normal range.

Diets and procedure

The 20 subjects were randomly assigned to 1 of 2 within-subject reversal designs. Each group followed a different sequence of experimental diets after a baseline measurement period during which they had continued their usual intake. One sequence for 10 outpatient subjects was 2 wk each of: free-feeding baseline, minimal-carbohydrate diet, carbohydrate-containing diet, minimal-carbohydrate diet, and carbohydrate-containing diet. After a baseline period of 2 wk of free-feeding, 10 different subjects followed the other sequence of experimental diets which was given in the reverse order. In this design, each subject underwent two experimental phases with carbohydrate in the diet and two phases with minimal carbohydrate, and thus each subject could be compared with him or herself for responses to the two diets. It was necessary to have a second group to counterbalance the order of dieting in the other group and to make it possible to control for any effects which sequence or time could have on the subjects' responses to the diets. This also gave us the opportunity to compare the two experimental diets in two separate groups of subjects.

After the medical screening examination, the subjects were instructed to follow their usual diet for 2 wk. The subjects did not keep records of eating during this phase and so the caloric content of their baseline diet was not determined. The minimal-carbohydrate diet consisted of lean meat, fish, or fowl. It contained approximately 800 kcal and was 58% protein (77 g) and 42% fat (55 g). The subjects also drank three glasses each day of a lemon-flavored drink sweetened with aspartame (Searle Nutrasweet). The carbohydrate-containing diet contained approximately 1,000 kcal and was 42% protein, 30% fat, and 28% carbohydrate (50 g). The same daily amount of meat was consumed in both diets. Carbohydrate was given as sucrose in the lemon-flavored liquid supplement. Rather than increase the fat or protein in the minimal-carbohydrate diet, the only modification was that the carbohydrate supplement was omitted. The two diets vary, therefore, not only in carbohydrate but also in caloric content. Potassium intake was supplemented with 25 mEq/day of the chloride and calcium by 200 mg/day of the carbonate. Multivitamins (Squibb Theragran-M) with iron and minerals (12 mg iron, 1.5 mg zinc) were given daily. Beverages which contained less than one calorie per serving (black coffee, tea, diet soda) and club soda and water were allowed ad libitum. Every two weeks, at the end of each diet phase, the subjects met with a research nurse who collected a blood sample and provided the patient with the next supply of liquid supplement. This research assistant was aware of which experimental phase the subject was undergoing. She did not, however, discuss with the subjects their diet plan or their responses to dieting. The subjects knew that the carbohydrate content of their diet was altered from time-to-time, but they did not know when, and they were blind to the experimental hypothesis. On the same day, the subjects met with a dietitian who reviewed their food records to check adherence to the plan. The dietitians were unaware of the subjects' experimental phase with respect to the liquid supplement. Subjects were instructed by the dietitians about the foods and amounts of food to eat and they were given social support for adherence. According to their food records, these subjects did not deviate from the prescribed diet. However, the lesser degree of ketosis in the second period of the diet, as opposed to the first period of either diet, suggests that there was not total conformity (see Discussion). No specific attempts were made to alter psychological adjustment. Finally, the subjects met with a psychologist at the end of each diet phase for the purpose of collecting the psychological measures.

Measures

All measures, except for daily hunger ratings, were taken at 2-wk intervals, at the end of each diet phase. After an overnight fast, venous blood was drawn for measurement of serum beta-hydroxybutyrate.

Emotional states associated with the diets were assessed with the Multiple Affect Adjective Checklist (MAACL, 28). The MAACL consists of a list of 132 adjectives of which the subject is free to endorse as many as are descriptive of their day-to-day moods. Scores are derived for three scales: anxiety, depression, and hostility. Psychological well-being or self-esteem was measured with the Tennessee Self Concept Scale (TSC, 29), which consists of 100 self-descriptive statements regarding body image, social and family relationships, and behavior patterns. The total positive score, which was used in the analysis, is a measure of the subject's self-esteem or how worthwhile and desirable the subject perceives himself or herself to be.

Appetite was assessed in several ways. Each day at home, the subjects rated their hunger just before dinner on a 0 to 100 visual analogue scale, a 100 mm line. Zero was not hungry at all, 100 was extremely hungry. The score for daily hunger ratings for each diet phase was the average of the ratings for 14 days. Also on a daily basis, the subjects kept track of their urges to violate the diet. An urge was defined as any desire to eat some food which was not allowed on the diet (eg, a piece of bread) or desire to eat a greater amount of food than was prescribed (eg, 8 oz of beef instead of 6 oz). They carried 3" × 5" cards with them throughout the day. Whenever they experienced an urge to violate the diet, they indicated so on the card and rated the intensity of the urge from 1 to 10, 1 being slight urge and 10 being extreme urge. This measure was employed during the caloric restriction phases only and not during the baseline, free-feeding phase. The third assessment of appetite was a measurement of the salivary response to the sight of food. The procedure for this method has been described in detail previously (30). Subjects came to the laboratory at noon with a lunch which was consistent with their diet. The food was displayed on the table and the subjects were instructed to relax and to keep their attention on the food. Further, they were guided to focus on the appearance and the aroma of the food and to imagine how it would taste. Three dental cotton rolls were placed for 2 min in standard positions in the mouth to absorb saliva. This was followed by a 2-min rest period. The saliva collection was repeated and followed by another rest period and third saliva collection. Afterwards the subjects were allowed to eat. The tared weight of the dental rolls was used as the measure of salivation. The food stimuli varied from session to session. During the baseline, free-feeding phase subjects brought their typical lunch. However, during the experimental diet phases, the food stimulus was limited to a few ounces of meat and their liquid supplement. The type of meat varied from session to session for some subjects.

Data analysis

One-way within subject analyses of variance (ANOVA) were used to determine the significance of changes across the diet phases for each dependent variable. Separate sets of ANOVAs were performed on each of the two experimental diet sequences. Post hoc comparisons were performed with the Newman-Keuls method.

Results

Overall, the subjects lost an average 11 kg of weight after 8 wk of dieting. The ANOVAs indicated that the weight losses were significant for both experimental sequences across time, with the greatest weight loss occurring during the first 4 wk of dieting. The degree of weight loss was more a function of time than type of diet. See **Table 1** for the mean weights.

The ANOVAs indicated that the fasting serum beta-hydroxybutyrate concentrations or the degree of ketosis associated with the two experimental diets were significantly different, $p < 0.001$ and $p < 0.05$ for sequence 1 and 2, respectively (see Table 1). Within both groups of subjects, the beta-hydroxybutyrate concentrations after minimal-carbohydrate diets were significantly greater than the ketogenic responses of the baseline and carbohydrate-containing phases. The only exception was that the beta-hydroxybutyrate concentration of the second minimal-carbohydrate phase for subjects of sequence 1 was not significantly greater than the ketogenic response of the first carbohydrate-containing phase. (See Table 1 for complete post hoc comparisons between phases.) Comparisons of the ketogenic responses between the two groups or sequences, for example, the first minimal-carbohydrate phase of sequence 1 versus the first minimal-carbohydrate phase of sequence 2, were not significantly different.

Mood and self-esteem

Analysis of the anxiety, depression, and hostility scales of the MAACL showed that there was a slight tendency for these moods to change during the weight-losing phase of the study, compared to basal mood levels. However, the subjects' moods did not change significantly from one experimental diet to another. The ups and downs of moods while dieting were related to the duration of dieting and not to the presence or absence of carbohydrate in the diet. Overall, the subjects showed the greatest improvement in mood after the first 2 wk of dieting, regardless of the sequence of the diets (see **Table 2** for means on the MAACL). There was some inconsistency in the fluctuation of the moods between the two sequences of experimental diets. Subjects in sequence 1 reported less anxiety ($p < 0.001$), depression ($p < 0.01$), and hostility while undergoing the weight loss diets than during baseline, but differences

TABLE 1
Means for weight and beta-hydroxybutyrate

	Baseline diet	Experimental diet (sequence 1, n = 10)			
	Free feeding	No-CHO-1	CHO-1	No-CHO-2	CHO-2
Weight (kg)	83	78	76	73	72
Beta-hydroxybutyrate (mM)/L*	0.09 (0.05)†	3.66 (1.20)	0.83 (0.34)	1.64 (0.54)	0.25 (0.16)
	Baseline diet	**Experimental diet (sequence 2, n = 10)**			
	Free Feeding	**CHO-1**	**No-CHO-1**	**CHO-2**	**No-CHO-2**
Weight (kg)	85	81	78	73	74
Beta-hydroxybutyrate (mM)/L‡	0.08 (0.09)	0.50 (0.34)	2.12 (1.71)	0.25 (0.21)	1.35 (0.64)

Note. Diets: No-CHO = approximately 800 kcal meat
CHO = approximately 800 kcal meat and 200 kcal sucrose supplement

* $p < 0.001$ for 1-way ANOVA. Post hoc tests of significant differences: No-CHO-1 > free feeding, CHO-1, CHO-2; No-CHO-2 > free feeding, CHO-2.

† Standard deviation.

‡ $p < 0.05$ for 1-way ANOVA. Post hoc tests of significant differences: No-CHO-1, No-CHO-2 > free feeding, CHO-1, CHO-2; CHO-1, CHO-2 > free feeding.

TABLE 2
Means for mood and self-esteem

	Baseline diet	Experimental diet (sequence 1, n = 10)			
	Free feeding	No-CHO-1	CHO-1	No-CHO-2	CHO-2
MAACL					
Anxiety*	7.1	4.8	3.8	4.3	3.6
Depression†	12.0	8.8	7.7	8.1	7.7
Hostility	6.5	5.7	5.4	5.4	4.7
TSC (self-esteem)‡	335	349	355	354	361
	Baseline diet	**Experimental diet (sequence 2, n = 10)**			
	Free feeding	**CHO-1**	**No-CHO-1**	**CHO-2**	**No-CHO-2**
MAACL					
Anxiety	6.6	4.5	6.7	7.5	7.1
Depression	13.1	8.8	12.0	12.7	13.5
Hostility	7.6	5.9	7.3	7.3	7.3
TSC (self-esteem)	331	340	339	340	342

Note. MAACL = Multiple Affect Adjective Checklist, TSC = Tennessee Self-Concept scale. Lower scores on MAACL represent more positive moods. Higher scores on TSC represent more positive self-esteem.

* $p < .001$ for 1-way ANOVA. Post hoc tests of significant differences: No-CHO-1, CHO-1, No-CHO-2, CHO-2, < Free feeding.

† $p < .01$ for 1-way ANOVA. Post hoc tests of significant differences: No-CHO-1, CHO-1, No-CHO-2, CHO-2 < Free feeding.

‡ $p < .001$ for 1-way ANOVA. Post hoc tests of significant differences: No-CHO-1, CHO-1, No-CHO-2, CHO-2 > Free feeding; CHO-2 > No-CHO-1.

in mood from one experimental diet to another were not significant. The results on the mood measure were somewhat different for the subjects who underwent sequence 2. They also showed a reduction in negative moods similar in magnitude to that of subjects in sequence 1, from their baseline to the end of the first 2 wk of weight-loss dieting. Unlike subjects in sequence 1 who maintained the mood improvement or further reduced negative emotions, these subjects returned to basal levels of negative moods by the end of the study. There were, however, no differences between the experimental diets. It is noteworthy that the basal levels of negative emotions of these subjects were at the middle of the average range for normal, nonpsychiatric subjects (31). Although some of the changes in the direction of greater well-being were statistically significant, the subjects still fell within the normal range by the end of the study.

The results of the TSC showed that subjects tended to describe themselves in a more positive light as they continued to diet during this study. Self-esteem did not vary as a function of the experimental diets. Subjects did not think any more or less favorably of themselves according to the presence or absence of carbohydrate in the diet. (See Table 2 for the means on the TSC.) Subjects in sequence 1 showed a continuous improvement in self-esteem, $p < 0.001$. Self-esteem during all the experimental diets was greater than during baseline, and self-esteem after the eighth week of dieting was significantly more positive than after the second week of dieting. The improvement in self-esteem for these subjects over the course of the study reflected an increase from the 46th percentile to the 54th percentile based on norms for the TSC in a nonpsychiatric population (29). Self-esteem of subjects in sequence 2 changed from a basal level at the 44th percentile to the 49th percentile. This difference was not statistically significant.

Appetite

There was no difference in appetite between the two experimental diets. In particular, subjects did not experience less hunger when their intake was relatively free of carbohydrate, as compared with their hunger during a carbohydrate-supplemented low-calorie diet. There was, however, a small trend toward a drop in appetite during the 8 wk of caloric restriction, regardless of type of diet, compared with that of the baseline, free-feeding diet. (see **Table 3** for the means of the appetite measures.) Salivation to the sight of food in the laboratory changed significantly for subjects in both sequences, $p < 0.01$ and $p < 0.05$, respectively. In both sequences, the subjects salivated significantly less after the initial 2 wk of caloric restriction than they did during the baseline testing. Afterwards, hunger as estimated by salivation increased slightly, approaching basal levels as the subjects continued to diet. However, the differences in salivation between the first phase of caloric restriction and the 3 subsequent phases were not statistically significant. There were no differences in salivation between experimental diets. The subjects' frequency and intensity of urges to eat food in excess of the prescribed diet did not vary significantly from one experimental phase to another. Regardless of the sequence of diets, subjects reported overall about two episodes each day when they had a definite desire to eat some other food. According to the subjects' daily ratings at home, there were no significant differences in their subjective feelings of hunger from one phase to another.

Discussion

The results of this study must be evaluated in the light of how well the subjects adhered to the protocol. Although some of the subjects had the same or a comparable degree of ketosis as expected when taking a given diet for the second time, 7–8 out of 10 in each group had a decline, with mean values of serum beta-hydroxybutyrate approximately half the initial values (Table 1). The difference was not significant by one-way ANOVA. Yang et al (32) found that an elevation of blood beta-hydroxybutyrate was sustained or continued to increase in 4 subjects given diets of approximately 700 kcal with or without 60 g of carbohydrate over a period of 64 days. In our previous studies involving inpatients (33, 34), the degree of ketosis following 6 wk of a comparable hypocaloric diet was not lower than at 1 wk. Thus, it is unlikely that the decline in serum ketones represented an adaptation. The decrease in our subjects was noted after 42 days of caloric restriction. The subjects of both groups may have gravitated toward foods containing more carbohydrate than planned, although they did not report an increase in hunger or

TABLE 3
Means for appetite measures

	Baseline diet	Experimental diet (sequence 1, n = 10)			
	Free feeding	No-CHO-1	CHO-1	No-CHO-2	CHO-2
Daily hunger rating*	44	41	40	40	39
Number of daily urges to violate diet	—	2.2	2.5	1.5	1.8
Intensity of urges to violate diet†	—	2.9	2.9	2.4	2.8
Salivation (g)‡	1.6	.8	1.3	1.2	1.3
	Baseline diet	**Experimental diet (sequence 2, n = 10)**			
	Free feeding	**CHO-1**	**No-CHO-1**	**CHO-2**	**No-CHO-2**
Daily hunger rating*	42	36	38	41	40
Number of daily urges to violate diet	—	2.0	2.3	2.3	2.4
Intensity of urges to violate diet	—	4.6	4.2	4.3	3.9
Salivation (g)§	1.9	1.1	1.4	1.5	1.6

* 0 = not hungry at all, 100 = extremely hungry.
† 1 = slight urge, 10 = extreme urge.
‡ $p < .01$ for 1-way ANOVA. Post hoc tests of significant differences: No-CHO-1 < Free feeding.
$p < .05$ for 1-way ANOVA. Post hoc tests of significant differences: CHO-1 < Free feeding.

distress in their subjective ratings. It is for this reason that in groups and diet comparisons, we conducted a series of tests correlating the ketogenic response achieved with the eight psychological variables for each of the four experimental phases. Only 3 of the 32 correlations were statistically significant. This is approximately the number which could occur by chance. Therefore, we conclude that the psychological responses to these diets were not related to the amount of carbohydrate and the degree of ketosis. Lack of total conformity is the general experience in outpatient studies, and we must also accept the possibility that some of their other ratings were at variance. The advantages, however, of studying a free-living population are in avoiding the possibility of the inhibition of appetite of obese subjects from withdrawal of cues to eating in an institutional situation, such as that reported by Hashim and Van Itallie (35) in their study of feeding patterns of the obese. With these reservations in mind, we can discuss our findings.

The results of this study indicate that the supposed psychological well-being of individuals undergoing a ketogenic diet is not distinguishable from psychological adjustment during a similar diet with enough carbohydrate to minimize ketosis. Although psychological well-being did not vary with the type of diet employed, subjects felt more favorably about themselves when they were subjected to caloric restriction than during their usual pattern of eating. The greatest improvements in mood and self-esteem came soon, within two weeks, after caloric restriction. Thereafter, the psychological status of the two samples of subjects were slightly different. It is noteworthy that subjects did not become irritable or depressed during dieting. In our previous study of a similar diet regimen, we also found that patients felt better about themselves and did not experience a sudden turn toward negative moods (25). In another recent study which involved systematically measured moods, subjects undergoing a weight-loss diet also showed enhanced psychological adjustment (36). Undoubtedly, the change in body image with weight loss underlies the well-being. Also, patients may feel better about themselves because they felt more in control of their eating behavior. Since a nondieting control group was not employed in the present study, the effect of monitoring psychological adjustment in and of itself cannot be determined. It is also noteworthy that subjects did not exhibit a euphoria or drastic improvement in psychological adjustment as some investigators observed in patients undergoing marked caloric restriction. Although some of the improvements in psychological well-being in the present study were statistically significant, none of the findings was clinically significant. Since these subjects commenced the study in the psychologically normal range, it was not possible for them to show much improvement. Patients with a poorer psychological adjustment who undergo a diet regimen such as this might respond much differently.

The results of this study also indicate that a minimum-carbohydrate hypocaloric diet does not suppress appetite in comparison to appetite during a similar diet with a small carbohydrate supplement. There was no fluctuation in appetite consistent with the presence or absence of carbohydrate in the diet. This finding is consistent with our previous study in which self-reported hunger was not different between two very low calorie diets with and without carbohydrate (26). Although there was no differential effect of the two experimental diets, appetite did seem to be altered slightly by caloric restriction. The largest decreases in hunger came soon during the initial 2 wk of dieting. This was reflected in both subjective and objective measures of hunger. It is interesting that the initial reductions in hunger corresponded with improved psychological well-being. The largest changes over baseline for both variables occurred during this early phase of weight loss. After the initial adaptation to the diet, there was some divergence in appetite states. Some subjects continued to feel less hungry than before starting the diet. Others exhibited about the same degree of hunger as during normal eating. It is noteworthy that subjects did not become *more* hungry, even though they presumably were consuming less food than usual. It is also noteworthy that they did not *lose* their appetite as some investigators report subjects do during diets similar to the protein-supplemented fast of the present study. In fact, subjects reported daily episodes of wanting to eat food in excess of the diet. Appetite, therefore, is not suppressed in the true meaning of the term. Perhaps it appears to be in light of people's expectations that caloric restriction should result in a significant *increase* in hunger.

Some of the reasons why high-protein, low-caloric diets with minimal vs more carbohydrate may not differentially affect appetite have been described earlier in this paper and in our previous paper (26). With respect to the present study, it is possible that subjects were not more hungry during the carbohydrate-containing phases because they were consuming 200 kcal more. Perhaps the extra calories with the glucose supplement offset any psychological advantage the lower caloric, minimal-carbohydrate phase would have. However, there also were no differences in responses to psychological tests between minimal-carbohydrate and carbohydrate-containing diets when eucaloric diets were used in our previous study (26). Another issue which deserves comment concerns the methods we employed to assess appetite. Our appetite measures were subjective ratings and psychophysiological responses in the presence of food. We did not include a measure of eating behavior. It would be possible, as is often done in animal research, to provide subjects with an opportunity to eat freely after the period of caloric restriction is completed. Consumption in the free-feeding situation would be a behavioral measure of appetite. A prior study of high-protein, carbohydrate-restricted hypocaloric diets used such a method to test hunger in laboratory animals (37). Prior carbohydrate deprivation led to a subsequent overconsumption of carbohydrate-containing foods. It is well known that human dieters often rebound and overeat after prolonged caloric restriction (38). No measurement of appetite behaviors during this post-restriction phase has yet been conducted with subjects undergoing the protein-supplemented fast. We felt constrained against putting our subjects into a free-feeding assessment situation because they had entered the study with the expectation of losing weight.

In conclusion, the results of this study give no support to the idea that the type of minimum-carbohydrate, protein-supplemented fast used here for a relatively short period of time specifically decreases appetite or elevates psychological well-being as compared with the effects of one containing enough carbohydrate to minimize ketosis. Any changes in appetite and psychological adjustment which occur while taking carbohydrate-restricted diets persist when extra carbohydrate is added to the diet. Supposed suppression of appetite or some other subjective benefit alone does not appear to be sufficient reason in itself for using minimal-carbohydrate diets of this type. It would appear wise in the future to give individuals restricted diets that have carbohydrate and are not ketogenic, so as not to risk any adverse effects due to ketogenesis.

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References

1. Wadden TA, Stunkard AJ, Brownell KD. Very low calorie diets: their efficacy, safety, and future. Ann Intern Med 1983;99:675–84.
2. Stuart RB, Mitchell C, Jensen JA. In: Prokop CK, Bradley LA, eds. Medical psychology: contributions to behavioral medicine. New York, NY: Academic Press, 1980.
3. Kelleher PC, Phinney SD, Sims EAH, et al. Effects of carbohydrate-containing and carbohydrate-restricted hypocaloric and eucaloric diets on serum concentrations of retinol binding protein, thyroxin binding prealbumin and transferrin. Metabolism 1983;32:96–101.
4. Apfelbaum M. Effects of very restrictive high-protein diets with special reference to nitrogen balance. Int J Obes 1981;5:209–14.
5. Bistrian BR. Clinical use of protein-sparing modified fast. JAMA 1978;21:2299–302.
6. Genuth SM, Castro JH, Vertes V. Weight reduction in obesity by outpatient semistarvation. JAMA 1974;230:987–91.
7. Flatt J-P, Blackburn GL. The metabolic fuel regulating system. Implications for protein sparing therapies during caloric deprivation and disease. Am J Clin Nutr 1974;27:175–87.
8. Brownell KD. Obesity: understanding and treating a serious, prevalent, and refractory disorder. J Consult Clin Psychol 1981;50:280–840.

9. Lindner PG, Blackburn GL. An interdisciplinary approach to obesity utilizing fasting modified by protein-sparing therapy. Obesity/Bar Med 1976;5:198–216.
10. Bistrian BR, Blackburn GL, Stanbury JB. Metabolic aspects of a protein-sparing modified fast in the dietary management of Prader-Willi obesity. N Engl J Med 1977;296:774–9.
11. Bistrian BR, Winterer J, Blackburn GL, Young V, Sherman M. Effect of a protein-sparing diet and brief fast on nitrogen metabolism in mildly obese subjects. J Lab Med 1977;89:1030–5.
12. Apfelbaum M. The effects of very restrictive high protein diets. Clin Endocrinol Med 1975;5:417–30.
13. Iselin HU, Burckhardt P. Balanced hypocaloric diet versus protein-sparing modified fast in the treatment of obesity: a comparative study. Int J Obes 1982;6:175–81.
14. Ferster CB, Nurnberger JL, Levitt EB. The control of eating. J Mathematics 1962;1:87–109.
15. Konorski J. Integrative activity of the brain. Chicago, IL: University of Chicago Press, 1967.
16. Peck JW. Rats defend different body weights depending on palatability and accessibility of their food. J Comp Physiol Psychol 1978;92:555–70.
17. Drenick EJ, Swendseid ME, Blahd WH, Tuttle SG. Prolonged starvation as treatment for severe obesity. JAMA 1964;187:100–5.
18. Duncan CG, Jenson WK, Cristofori FC, Schless GL. Intermittent fasts in the correction and control of intractable obesity. Am J Med Sci 1963;245:515–20.
19. Bloom WL. Fasting as an introduction to the treatment of obesity. Metabolism 1959;8:214–20.
20. Duncan GG, Hunscher MK, Cristofori FC, Duncan TG, Schless GL. Intermittent total fasts and obesity. Postgrad Med 1965;38:523–35.
21. Laszlo J. Changes in the obese patient and his adipose tissue during prolonged starvation. South Med J 1965;58:1099–108.
22. Stetten IW, Viamontes G, Hughes DD, Korol B. Total fasting in psychiatric subjects. Canadian Psychiat Assoc J 1967;12:553–70.
23. Geiselman PJ, Novin D. The role of carbohydrates in appetite, hunger, and obesity. Appetite: Journal for Intake Research 1982;3:203–23.
24. Baird IM, Parsons RL, Howard AN. Clinical and metabolic studies of chemically defined diets in management of obesity. Metabolism 1974;23:645–57.
25. Silverstone JT, Stark JE, Buckle RM. Hunger during total starvation. Lancet 1966;1:1343–4.
26. Rosen JC, Hunt DA, Sims EAHS, Bogardus C. Comparison of carbohydrate-containing and carbohydrate-restricted hypocaloric diets in the treatment of obesity: effects on appetite and mood. Am J Clin Nutr 1982;36:463–9.
27. Society of Actuaries. Build and blood pressure study. 2 Vols. Chicago, IL: Author, 1959.
28. Zuckerman M, Lubin B, Vogel L, Valerius E. Measurement of experimentally induced affects. J Consult Psychol 1964;28:418–25.
29. Fitts WH. Tennessee Self-Concept Scale. Nashville, TN: Counselor Recordings and Tests, 1965.
30. Wooley SC, Wooley OW. Salivation to the sight and thought of food: a new measure of appetite. Psychosom Med 1973;35:136–42.
31. Zuckerman M, Lubin B. Manual for the multiple affect adjective check list. San Diego, CA: Edits Publishers, 1965.
32. Yang MU, Barbosa-Sadaidar JL, Pi-Sunyer FXL. Metabolic effects of substitute carbohydrates for protein in a low caloric diet: a prolonged study in obese patients. Int J Obes 1981;5:231–6.
33. Bogardus C, La Grange BM, Horton ES, Sims EAH. Comparison of carbohydrate-containing and carbohydrate-restricted hypocaloric diets in the treatment of obesity. Endurance and metabolic fuel homeostasis during strenuous exercise. J Clin Invest 1981;68:399–404.
34. Phinney SD, Horton ES, Sims EAH, Hanson JS, Danforth E, La Grange BM. Capacity for moderate exercise in obese subjects after adaptation to a hypocaloric, ketogenic diet. J Clin Invest 1980;1152–61.
35. Hashim SA, Van Itallie TB. Studies in normal and obese subjects with a monitorial food dispensing service. Annals Acad Sci 1965;B1:654–61.
36. Wing RR, Marcus MD, Epstein LH, Kupfer D. Mood and weight loss in a behavioral treatment program. J Consult Clin Psychol 1983;51:153–5.
37. Wurtman JJ, Moses PL, Wurtman RJ. Prior carbohydrate consumption affects the amount of carbohydrate that rats choose to eat. J Nutr 1983;113:70–8.
38. Polivy J, Herman CD, Olmsted MP, Jazwinski C. Restraint and binge eating. In: Hawkins RC, Fremouw WJ, Clement PF, eds. The binge-purge syndrome: Diagnosis, treatment and research. New York: Springer Publishing Company, 1984.