International Journal of Obesity (1997) 21, 14–21

Cognitive effects of a long-term weight reducing diet

MJ Kretsch[1], MW Green[2], AKH Fong[1], NA Elliman[2] and HL Johnson[1]

[1]*U.S. Department of Agricultural, Agriculture Research Service, Western Human Nutrition Research Center, P.O. Box 29997, Presidio of San Francisco, California 94129, USA; and* [2]*Institute of Food Research, Consumer Sciences Department, Earley Gate, Whiteknight's Road, Reading, RG6 2EF, UK*

OBJECTIVE: To investigate if long-term caloric restriction under controlled conditions adversely affects cognitive function in obese women.
SUBJECTS: Healthy, premenopausal women between 23–42 y. Dieting group: $n = 14$. Control group: $n = 11$.
DESIGN: Longitudinal weight loss study (repeated measures within-subject design) with 3 weeks of baseline, 15 weeks of 50% caloric restriction, and 3 weeks of weight stabilization.
MEASUREMENTS: Computerized cognitive function tests (sustained attention, short-term memory, simple reaction time, motor performance and attentional focus), height, body weight, body composition (TOBEC) and behavioral questionnaires (Dutch Eating Behaviour Questionnaire, Eating Attitudes Test, and State-Trait Anxiety Inventory).
RESULTS: Dieting women lost 12.3 ± 5.5 kg (mean ± s.d.) of body weight. Controlled long-term caloric restriction significantly slowed simple reaction time but did not diminish sustained attention, motor performance or immediate memory. Word recall performance significantly improved by 24% at the end of caloric restriction.
CONCLUSIONS: The slowing of simple reaction time is a short-term and long-term consequence of caloric restriction. In contrast to previous short-term dieting studies, sustained attention and immediate memory were not impaired with long-term caloric restriction.

Keywords: obesity; diet; caloric restriction; cognitive function; simple reaction time; women

Introduction

There is a growing body of research that demonstrates associations between food restriction and impairments in cognitive processing efficiency.[1–6] This has implications for the treatment of clinical conditions such as obesity. Therapeutic regimes for the treatment of obesity can include various methods of caloric restriction, such as very low calorie diets, as well as exercise or exercise as an adjunct to caloric restriction. Any effect of these weight loss strategies on the cognitive processing efficiency of obese individuals may have important implications.

Food restriction can be viewed as a continuum, ranging from simply missing a single meal to the severe starvation observed in anorexia nervosa. The state of starvation characteristic of anorexia nervosa is associated with impairments in a number of cognitive parameters. For instance, anorexics have displayed impairments in short term memory,[7] attentional focus and visuo-spatial reasoning.[8] It has also been found that bulimia nervosa patients exhibit impairments of many cognitive functions.[9] However, since neuropsychological deficits are present in individuals with eating disorders, it remains to be determined whether these deficits are the results of starvation *per se* or a combination of starvation and disordered psychopathology.

Non-eating disordered individuals also show associations between caloric restriction and cognitive processing efficiency. There is evidence suggesting that even mild degrees of food restriction such as missing individual meals are associated with cognitive impairments.[10] For instance, mild degrees of food restriction *per se* have been reported to exert either small detrimental effects,[11,12] small beneficial effects,[11] or no effects at all on cognitive processing.[4] Such work reveals a complex interaction between the meal missed (lunch or breakfast), circadian variation, practice and/or fatigue effects and the domain of cognitive function.

There is also evidence that self-imposed dieting regimes undertaken to lose weight are associated with alterations in cognitive processing such as poorer sustained attention,[1,3] diminished immediate recall and longer simple reaction times.[3] Further, it has been found that, within the same individuals performance on a cognitive function task battery was poorer when they reported themselves as currently dieting than when they reported themselves as not currently dieting.[5] This demonstrates that the processing

Mention of a trademark or proprietary product does not constitute endorsement by the U.S. Department of Agriculture and does not imply recommendation over others that may be suitable.

Correspondence: Dr MJ Kretsch.

Received 10 June 1996; revised 2 September 1996; accepted 9 September 1996

impairments were associated with self-imposed dieting *per se*, rather than being due to pre-existing differences between dieters and non-dieters. Since the dieters in this latter study reported no substantial weight loss from their self-imposed diet, it was also tentatively concluded that the underlying nature of the processing impairment was primarily psychological, rather than physiological.

The literature is nearly silent regarding the cognitive effects of long term food restriction *per se* except for the renowned Minnesota study of semi-starvation[13] in which young, normal-weight men were kept in severe caloric restriction for 24 weeks. Apart from the obvious physiological consequences of starvation, subjects reported lapses in concentration, memory deficits and increased incidence of negative mood states such as irritability and depression. Starvation induced deficits in cognitive processing can also be seen from a study investigating vigilance performance amongst bulimia nervosa patients.[14] Vigilance task performance of bulimia patients was poorer than that of a group of non-clinical controls and performance was poorer still amongst a sub-group of bulimia patients exhibiting metabolic signs of starvation.

The purpose of this study was to investigate the effects of controlled, long-term caloric restriction as a weight loss regime on the cognitive function of obese, but otherwise healthy, women. Multiple modalities of cognitive function were investigated, namely, sustained attention, attentional focus, simple reaction time, short-term memory, and motor performance.

Methods

Subjects

Healthy, obese women between the ages of 25–42 y were recruited to participate in this study through local newspaper and poster advertising, local radio public service announcements, and articles in the health sections of newspapers. Approximately 1000 phone inquiries were received in response to the advertising. Telephone screening eliminated those with body weights outside the range of 130 to 150% of ideal body weight (1983 Metropolitan Life Insurance Tables);[15] those unable or unwilling to come to our Center every weekday to consume one meal and to pick up the balance of their food; those unable or unwilling to participate in nine full-day laboratory evaluations during the study; those with chronic illnesses requiring medications; and those who smoked, used recreational drugs or consumed more than 2 alcoholic drinks per week. Eighty-five percent of the prospective volunteers who telephoned our Center did not meet the telephone screening criteria with the majority disqualified because they could not meet either the body weight or time commitment requirements. About 100 eligible volunteers came to our Center where heights and weights were measured; a medical history questionnaire was completed; blood and urine samples were obtained for health, pregnancy and drug evaluations; and a physical examination including electrocardiographic testing was conducted. Psychological screening was not conducted. Women who did not meet the criteria for health or measured height and body weight were excluded as were users of prescription and recreational drugs. Acceptable volunteers were invited to attend a group meeting for a thorough explanation of the study protocol and an opportunity to ask questions of the investigators. Study consent forms approved by Human Use Institutional Review Boards of the University of California at Davis and the United States Department of Agriculture's Agricultural Research Service were then signed by all study volunteers.

Control (non-dieting) and experimental (dieting) subjects were recruited from the same pool of obese women volunteers. Because all volunteers wanted to lose weight, those assigned to the control group were women who called in response to advertising after sufficient volunteers had been obtained for the dieting group. Twelve women were recruited for the control group but only eleven volunteered. Twenty-four women were recruited for the dieting group, twenty-four volunteered, but only fourteen completed the study. Those that left the study either did not comply with the dietary protocol during caloric restriction (50% of the drop-outs) or left for personal reasons including sickness, moving, job problems, etc. Four of the five women who left because of non-compliance with the dietary protocol had a score > 20 on the Eating Attitudes Test (EAT),[16] whereas those who left for personal reasons had EAT scores < 20. An EAT score > 20 is indicative of sub-clinical eating disorders. There were no other distinguishing characteristics between those that completed the study and those that left the study.

Experimental design

Simple reaction time is known to be stable and well suited to repeated measures design studies[17–19] but less is known about the other cognitive performance tests used in this study. Therefore, cognitive performance tests were administered to the control subjects four times across an eight week period to assess stability and reliability of the tests.[17,20,21] (Attrition of control subjects did not allow for repeated-measures testing beyond 4 sessions but all control subjects provided data for all four tests). Body weight and body composition of the control subjects were also measured at each cognitive test point.

The experimental (dieting) subjects participated in a 21 week weight loss study. This was a longitudinal study (repeated-measures within-subject design) comprised of three periods: baseline (3 weeks), 50% caloric restriction (15 weeks) and weight stabilization

(3 weeks). Cognitive function and body height, weight, and composition were measured three times during baseline, three times during caloric restriction (after 5 weeks, 10 weeks, and 15 weeks of caloric restriction), and two times during weight stabilization (weeks 17 and 18). All measurements were conducted on the same day of the week. Based on data from the control group, the first two cognitive test sessions during baseline were used as practice sessions.

Because this study was part of a larger study examining the effects of exercise and dietary carbohydrate level on changes in body weight, body composition and energy metabolism in obese women, Johnson HL *et al*—unpublished data; subjects in the dieting group were randomized to exercise (walking *vs* sedentary) and diet treatment (high carbohydrate *vs* average carbohydrate) groups before the onset of the study. Exercisers were equally distributed between the high carbohydrate and average carbohydrate dietary treatments. Those who exercised walked three miles per weekday (15 miles/week) and exercise compliance was monitored by staff.

Throughout the study, both the control and dieting groups received all their food from the Western Human Nutrition Research Center. Whereas the controls received a diet which allowed *ad libitum* consumption of the provided foods, the dieting group had a precisely formulated diet. The formulated diet provided sufficient calories for body weight maintenance during baseline, 50% of the baseline calories during caloric restriction, and calories sufficient to maintain their new body weight during the weight stabilization period. The women in the dieting group received 47% of their calories from carbohydrate sources during baseline and either 63% of calories (high carbohydrate diet) or 43% of calories (average carbohydrate diet) from carbohydrates during caloric restriction. During the 3 week weight stabilization period following caloric restriction, energy from carbohydrates was continued at the same percentage provided during caloric restriction, that is either 63 or 43%. Protein intake was maintained at 18–20% of calories in all experimental diets.

Diets

An American-type menu consisting of conventional foods was fed throughout the study. Controls received a seven day rotational menu. The experimental group received a seven day rotational menu during baseline and weight stabilization, but a 14 day rotational menu during the 15 weeks of caloric restriction. In addition to the nutrients supplied by the conventional food diets, both the dieting and control groups received a daily multi-vitamin/mineral supplement. The supplement provided 100% of the 1989 Recommended Dietary Allowances (RDA)[22] for all vitamins and minerals except calcium (16% of the RDA), phosphorus (11% of the RDA), and magnesium (25% of the RDA). In addition, the supplement provided those nutrients for which estimated safe and adequate daily dietary intakes have been set.[22] Water, coffee, tea, and diet soft drinks were allowed *ad libitum*. The energy and nutrient composition of the diets were calculated using the 1995 Campbell Soup Company Master Database which includes the United States Department of Agriculture's Nutrient Database for Standard Reference[23] in addition to analyzed values for various Campbell Soup Company frozen entrees, frozen dinners, and canned soups products.

Measurements

Body mass index (kg/m^2) was calculated from measured height and body weight. Body composition was determined by the Total Body Electrical Conductivity (TOBEC) analyzer (Model HA-2, EM-Scan Inc., Springfield, IL).[24] Cognitive function was measured using a computerized task battery that measures multiple modalities of cognitive performance.[4] These included sustained attention (Bakan vigilance task), short-term memory (word recall task), simple reaction time, motor performance (two finger tapping task), and focused attention (Ericksen effect); always administered in this order. Subjects were scheduled for the same time each session, between 8:30 am and 11:30 am, and testing lasted about 45 minutes. Subjects were tested individually in a dimly lit, soundproof room to enhance computer screen visibility and to reduce external noise. All cognitive tasks were programmed and initial data collection and analysis performed using MEL 1.0 (Psychology Software Tools, Inc., Pittsburgh, PA) installed on a DELL 386 personal computer attached to 14" color monitor. Subjects completed self-report questionnaires of eating behavior, food and body shape related concerns, and affective state. These included the Dutch Eating Behaviour Questionnaire,[25] Eating Attitudes Test[16] and State (form Y-1) and Trait (form Y-2) portions of the State-Trait Anxiety Inventory (STAI),[26] respectively. With the exception of the State Anxiety form which was completed at the end of each cognitive test session, the other questionnaires were completed only at the first cognitive test session.

Statistics

To determine the suitability of the cognitive performance tests to a repeated measures study design, the control subjects' cognitive performance scores were analyzed for stability and reliability according to previously published methods.[17,20,21] Stability for each task was evaluated in three ways: session at which the mean score stabilized, session at which the standard error of the mean stabilized, and session at which the inter-session correlation stabilized. Reliability was defined as the inter-session correlation following stabilization, that is if the mean score reached a plateau at session 2, then the correlation between session 3 and 4 was the reliability for the test.

There were no missing data points for the control group for sessions one through four.

Statistical analyses for the dieting group were performed with the personal computer version of the Statistical Analysis System (SAS) for Operating System/2 (OS/2), version 6.08 (SAS Institute, Inc., Cary, NC). Only data from dieting subjects completing the entire study were used ($n = 14$) and there were no missing data points. Variance homogeneity of the cognitive performance data was examined with SAS using the Bartlett's test, Box's modification of the Bartlett's test, and the Hartley's Max F test. Transformations were indicated for the Bakan vigilance task. The Box–Cox power transform method identified the SAS weighting transformation to be the appropriate means for stabilizing the Bakan task variance. Following this transformation, the data were analyzed via repeated measures ANOVA (under the general linear models procedure using the Greenhouse–Geisser adjustment as needed) testing for the effects of diet treatment and exercise group on cognitive function. No significant effects were found for either of these groupings, and therefore, they were dropped from subsequent analyses. Repeated measures ANOVA was next used to test for the effect of test session and significant effects were further examined using SAS Contrast and Profile within the repeated measures ANOVA. Pearson product-moment correlations, linear regressions, and 95 and 99% confidence interval analyses were calculated to examine relationships between the various cognitive and physiological measures. Unless stated, values presented in the text and tables are the mean ± s.d. and the significance criterion used is $P < 0.05$.

Results

Baseline demographic information and behavioral questionnaire scores for the dieting and non-dieting groups are shown in Table 1. There were no significant differences between groups for these measures. The women were obese, ranged in age from 25–42 y, and had a minimum of 12 y of education. The non-dieting control group and the dieting group were balanced for race/ethnicity and included women of Caucasian, African-American, and Hispanic extraction. Mean trait anxiety scores for both groups were slightly higher than the reported norm for working women of the same age range, namely 36.15 ± 9.53 (mean ± s.d.).[26] Average state anxiety scores were about the same for the two groups and did not change significantly across the study for either group. By the end of caloric restriction (week 15), the dieting group had lost 12.3 ± 5.5 kg of body weight, of which 10.4 kg was fat and 1.9 kg was lean body mass (Figure 1). The control subjects maintained body weight throughout their cognitive testing period.

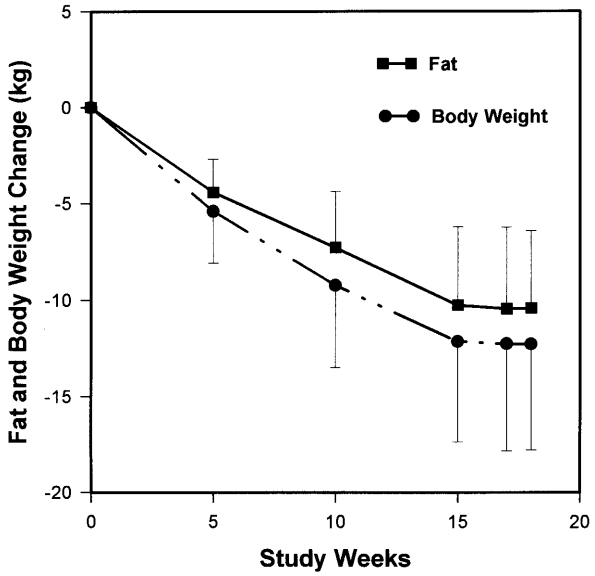

Figure 1 Dieting group ($n = 14$). Body weight and body fat loss (mean ± s.d.) across the 18 week study. Week 0 represents baseline, weeks 1–15 are the 50% caloric restriction period, and weeks 16–18 are the weight stabilization period.

Table 1 Baseline subject information

	Dieting group (n = 14)	*Non-dieting group (n = 11)*
Demographic:		
Age (y)	35.0 ± 5.2[a]	30.1 ± 7.5
Education (y)	15.4 ± 1.8	13.8 ± 1.9
Height (cm)	165.8 ± 8.2	164.7 ± 6.4
Weight (kg)	86.6 ± 11.8	93.2 ± 10.7
BMI[b] (kg/cm^2)	31.5 ± 4.1	34.2 ± 1.9
Behavior Questionnaires:		
Dutch Eating Behaviour:		
Restrained eating	2.8 ± 0.6	3.0 ± 0.8
Emotional eating	3.2 ± 0.9	2.2 ± 0.7
External eating	3.1 ± 0.5	2.8 ± 0.5
Eating Attitudes Test:	12.0 ± 3.7	11.1 ± 7.1
State Trait Anxiety Inventory:		
Trait Anxiety	41.3 ± 11.3	39.9 ± 6.3
State Anxiety	30.4 ± 9.4	32.9 ± 8.5

[a] Mean ± s.d.
[b] BMI = body mass index.

Table 2 Cognitive performance scores for the control group ($n = 11$)[a]

Task	*Test session* 1	2	3	4
Bakan (max = 8 correct hits)	3.0 ± 0.5	3.7 ± 0.5	3.8 ± 0.5	3.9 ± 0.5
Simple reaction time (ms)	334.2 ± 22.7	353.2 ± 25.9	347.5 ± 17.2	350.3 ± 17.5
Word recall (max = 20 words)				
one second presentation	8.3 ± 1.0	7.4 ± 0.7	9.0 ± 0.7	8.0 ± 1.1
two second presentation	10.4 ± 1.5	11.7 ± 1.3	11.8 ± 1.1	11.6 ± 1.2
Tapping (tap/s)	7.5 ± 0.6	7.2 ± 0.4	7.5 ± 0.4	7.7 ± 0.5
Ericksen Effect	− 28.6 ± 15.1	− 10.4 ± 8.2	− 37.1 ± 12.1	− 15.9 ± 14.7

Table 3 Cognitive performance scores for the dieting group ($n = 14$)[a]

Task	*Study period* Baseline	(week 5)	Caloric restriction (week 10)	(week 15)	Stabilization	*Repeated measures ANOVA (significance level) test session*
Bakan (max = 8 correct hits)	3.9 ± 0.5	3.5 ± 0.5	3.9 ± 0.5	4.0 ± 0.5	3.9 ± 0.5	NS
Simple reaction time (ms)	350.0 ± 15.4[b]	357.7 ± 18.7[b]	363.6 ± 19.1[bc]	368.1 ± 16.3[c]	388.3 ± 21.7[d]	0.0001
Word recall (max = 20 words)						
one second presentation	7.3 ± 0.9	6.2 ± 0.6	7.4 ± 0.6	8.1 ± 0.8	7.1 ± 0.6	NS
two second presentation	8.2 ± 0.7[b]	9.1 ± 0.8[b]	9.4 ± 0.9[b]	10.2 ± 1.0[c]	8.3 ± 0.8[b]	0.0014
Tapping (tap/s)	7.3 ± 0.4	6.9 ± 0.5	7.1 ± 0.5	7.1 ± 0.5	7.2 ± 0.4	NS
Ericksen Effect	− 35.4 ± 16.2	− 42.9 ± 31.8	− 16.9 ± 9.3	− 4.8 ± 11.9	− 35.4 ± 15.0	NS

[a] Mean ± s.e.m. NS = not significant. Significant effects for test session were further analyzed using SAS Contrast and Profile within the repeated measures ANOVA.
[b,c,d]Values are significantly different at $P < 0.05$.

The cognitive performance scores of the control group (non-dieting) are shown in Table 2. It was found that the means, standard errors and intertrial correlations stabilized within 2 trials for all cognitive tasks except the word recall (one second presentation) and attentional focus (Ericksen effect) measures. Because these latter tests did not stabilize within four trials, their results are considered unreliable. Reliability (intertrial correlation between the third and fourth trials) was 0.95, 0.93, 0.83, and 0.83 for the Bakan, simple reaction time, two second word recall, and two finger tapping tests, respectively. The stabilized mean Bakan, simple reaction time and tapping scores for the control group were of the same magnitude as those for the dieting subjects at baseline (Table 2). However, the mean two second word recalls cores were slightly higher for the control group than for the dieting group at baseline.

Repeated measures ANOVA revealed a significant effect of test session for the simple reaction time ($P < 0.0001$) and two second word recall ($P < 0.0014$) tasks for the dieting women as shown in Table 3. Compared to baseline, simple reaction time was significantly slower at the end of caloric restriction (−5.2%) and continued to slow further during the weight stabilization period (up to −10.9%). This slowing of simple reaction time occurred in all subjects. Immediate short-term memory at the end of caloric restriction, as reflected by the two second word recall task, was significantly elevated (+24.4%) over both baseline and weight stabilization scores. Although nonsignificant, a performance decrease occurred for the Bakan vigilance task at week five of caloric restriction. No significant change was observed for two finger tapping speed across the study.

Word recall performance during caloric restriction was found to be associated with change in body weight and with state anxiety score. Word recall scores (two second presentation) were significantly correlated with body weight changes ($r = 0.72$,

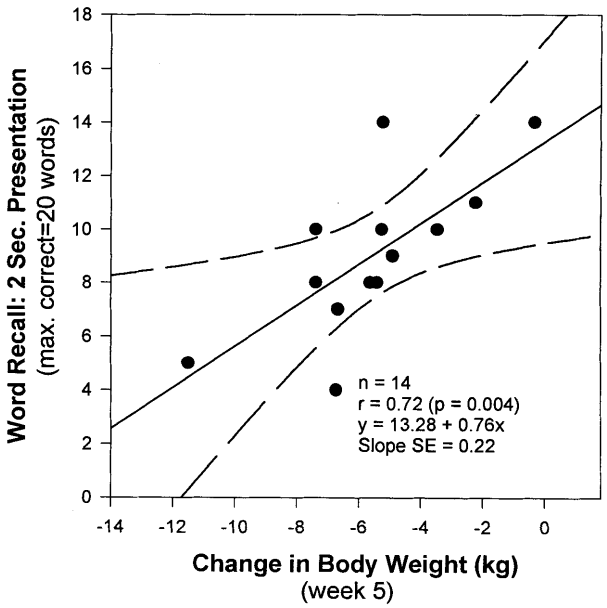

Figure 2 Dieting group ($n = 14$). Scatterplot, regression line and 99% confidence interval for the relationship between the number of correct words recalled (two second presentation) and the change in body weight (kg) at week five of caloric restriction. Pearson correlation coefficient: $r = 0.72$, $P = 0.004$. Linear regression equation: $y = 13.28 + 0.76x$, slope s.e. = 0.22.

$P = 0.004$) at week five of caloric restriction (Figure 2); the subjects with greater body weight loss had lower two second word recall scores. Also at week five of caloric restriction, significant correlations were found between the two second word recall scores and body fat loss ($r = 0.64$, $P = 0.014$) and lean body mass loss ($r = 0.62$, $P = 0.018$). Again, subjects with the greater body fat and lean body mass had lower word recall scores. At week 15 (the same timepoint at which a significant improvement in word recall ability occurred) a significant correlation between state anxiety and two second word recall scores was found ($r = 0.64$, $P = 0.014$), that is the higher the reported state anxiety level the greater the number of correct words recalled. No other significant correlations were found.

Discussion

This study examined the effects of controlled, long-term caloric restriction on various modalities of cognitive function in dieting obese women. The level of caloric restriction was fairly severe with calories set at 50% of the energy required to maintain body weight. Such long-term dieting significantly slowed simple reaction time in the women but did not diminish their sustained attention ability, motor performance, or immediate memory. While the slowing of simple reaction time confirms and extends findings from previous short-term dieting studies conducted with normal-weight women,[3,5] the absence of impairment in sustained attention and immediate memory stands in contrast to those studies.

There is only one other long-term study of caloric restriction in which various aspects of cognitive function were measured, namely the classic Minnesota semi-starvation study.[13] In that study, 32 young normal-weight men received 50% of their energy needs for 24 weeks preceded by 12 weeks of baseline and followed by 12 weeks of refeeding. A significant decline in gross body reaction time was found at the end of caloric restriction. This finding agrees with the study reported herein, in which a significantly longer simple reaction time was found within 15 weeks of caloric restriction. Because slowing of reaction time occurred with caloric restriction in all subjects in both studies this demonstrates a robust finding that is apparent in different subject populations. Further, slowing of reaction time is characteristic of the starvation state accompanying anorexia nervosa.[27]

Another important finding in both studies is that reaction time did not readily reverse upon restoration of sufficient calories to maintain body weight. Reaction time in this study continued to slow reaching a 10.9% decrease three weeks into the weight stabilization period and in the Minnesota study only a 70% recovery in reaction time was found after 12 weeks of refeeding. The biological and practical significance of this central nervous system change is unknown and because it may have important implications for the treatment of obesity, it warrants further investigation.

Of particular importance to the results of this study is the stability of the simple reaction time test. It is known that repeated cognitive test performance may lead to a practice effect. This can be controlled by use of a comparable control group or by pre-experiment practice. While a control group throughout the entire 18 week study reported herein would have been preferable, it is clearly difficult to prevent attrition in a group of obese women who wish to lose weight but for whom there is no weight loss incentive. Thus, in this study each experimental subject served as their own control and the practice effect was controlled by pre-experiment training to a plateau. Further support that the slowing of simple reaction time with long-term caloric restriction is real effect rather than an artifact of repeated task administration can be found in the literature. Other investigators have demonstrated that simple reaction time is not influenced by repeated trials or rehearsals,[17,20] and is therefore stable for use in repeated measures study designs.[17] In fact, it has been found that a few hundred trials are needed to produce a practice effect in simple reaction time,[27] and this is further extended by the use of a variable stimulus onset as was used in this study.

Besides diminished simple reaction time, previous short-term studies have shown that self-imposed dieting impairs performance on sustained attention and immediate memory tasks.[3,5] The sustained attention task employed in those studies was the Bakan vigilance test, a high processing load attention task. Contrary to those studies, no significant change in Bakan vigilance task performance was found in this study in which the Bakan testing was begun five weeks into the dieting period. This lack of change in sustained attention is supported by results from another recent study.[6] In that study, 21 obese women were fed a very low energy, ketogenic or nonketogenic, liquid formula diet for four weeks and cognitive tests were conducted on a weekly basis. They found that subjects on the ketogenic diet exhibited diminished performance on a trail making task, a neuropsychological test that requires higher order mental processing and flexibility. This performance decrement occurred after one week of dieting but was not evident at later time points. Thus, it can be inferred from studies to date that if dieting impairs attention task performance, it appears to occur only at the beginning of the dieting period, it reverses with time, and it occurs only for those attention tasks requiring a high mental processing load.

Whereas poorer immediate memory was found in the short-term, self-imposed dieting studies,[3,5] no such change was found with long-term caloric restriction in either this study or the Minnesota study.[13] Rather, in this study recall performance significantly improved at the end of caloric restriction ($+24\%$) and then

readily reversed to baseline levels upon restoration of sufficient calories to maintain body weight. It could be that if changes in immediate memory occur, as reported in the earlier studies,[3,5] they occur only during the early weeks of dieting. In this study, subjects with the greatest body weight loss after 5 weeks of caloric restriction had the lowest two second word recall scores ($r = 0.72$, $P = 0.004$), however, this relationship was not found after 10 or 15 weeks of caloric restriction.

Another outcome of this study was that state anxiety and word recall scores were significantly and positively correlated at the end of caloric restriction. Why this relationship was only observed at the end of caloric restriction, and not at earlier timepoints, is unknown. The fear of regaining body weight after this timepoint, expressed by some of the women, may have contributed to the observed heightened anxiety. Whatever the cause, this association between state anxiety and word recall score suggests a possible physiological basis for the observed immediate memory improvement. There is considerable evidence in both rodents and humans that epinephrine release is an important contributor to the process by which memory formation is regulated,[28] and epinephrine release is known to occur with heightened anxiety. With the release of epinephrine, a rise in circulating glucose levels occurs which in turn contributes to brain actions responsible for memory formation. In fact, glucose has been found to specifically improve performance on measures of declarative memory (such as retention of word lists) but not to affect performance of nonmemory neuropsychological tasks such as attention, motor speed, or overall IQ.[29–31]

In conclusion, it appears that slowing of simple reaction time is a short-term and long-term consequence of caloric restriction. Although short-term, self-imposed dieting has been shown to cause diminished sustained attention and immediate memory in women; these impairments were not found here with controlled, long-term caloric restriction. However, word recall performance during caloric restriction was found to relate to the amount of body weight lost after five weeks of dieting and to state anxiety level after 15 weeks of dieting. It remains to be determined if the differing patterns of cognitive performance impairments observed among studies to date are a function of the length of caloric restriction or a function of self-imposed diets versus diets fed as part of an experimental regime. Also, further research is needed to examine the physiological and practical significance of simple reaction time slowing with dieting.

Acknowledgements

We express our thanks for the interest and cooperation of the volunteer subjects who made this study possible and to Teresa Barbieri for her dedicated efforts in coordinating this study. We are also appreciative of the statistical advice and guidance provided by Linda Whitehand, Bruce Mackey, and Virginia Gildengorin, and for the service provided by the recruitment, dietetic and nursing staffs. We would also like to acknowledge the Campbell Soup Company, Camden, NJ for providing soup and frozen breakfast/dinner products for use in this study.

References

1 Rogers PJ, Green MW Dieting, dietary restraint and cognitive performance *Br J Clin Psychol* 1993; **32**: 113–116.
2 Rogers PJ, Lloyd HM Nutrition and mental performance *Proc Nutr Soc* 1994; **53**: 443–456.
3 Green MW, Rogers PJ, Elliman, NA, Gatenby SJ Impairment of cognitive performance associated with dieting and high levels of dietary restraint *Physiol and Behavior* 1994; **15**: 447–452.
4 Green MW, Elliman NA, Rogers PJ Lack of effect of short-term fasting on cognitive function *J Psychiat Res* 1995; **29**: 245–253.
5 Green MW, Rogers PJ Impaired cognitive functioning in dieters during dieting *Psychol Med* 1995; **25**: 1003–1010.
6 Wing RR, Vazquez JA, Ryan CM Cognitive effects of ketogenic weight reducing diets *Int J Obes* 1995; **19**: 811–816.
7 Fox CF Neuropsychological correlates of anorexia nervosa *Int J Psychiat Med* 1981; **11**: 285–290.
8 Jones BP, Duncan CC, Brouwers P, Mirsky AF Cognition in eating disorders *J Clin and Exp Neuropsychol* 1991; **13**: 711–728.
9 MacKay SE, Humphries LL, Allen ME, Clawson DR Neuropsychological test performance of bulimia patients *Int J Neuroscience* 1986; **30**: 73–80.
10 Smith AP, Kendrick AM. Meals and performance. In: Smith AP, Jones DM (eds). *Handbook of Human Performance, vol 2: Health and Performance*. Academic Press: London, 1992, pp 2–23.
11 Pollitt E, Leibel RL, Greenfield D Brief fasting, stress, and cognitive function in children *Am J Clin Nutr* 1981; **34**: 1526–1533.
12 Pollitt E, Lewis NL, Garza C, Shulman RJ Fasting and cognitive function *J Psychiat Res* 1983; **17**: 169–174.
13 Keys A *et al*. *The Biology of Human Starvation*. (Vol. 1 & 2). University of Minnesota Press: Minneapolis, 1950.
14 Laessle RG *et al* Cognitive performance in patients with bulimia nervosa: Relationship to intermittent starvation *Biol Psychiatry* 1990; **27**: 549–551.
15 Metropolitan Life Insurance Company 1983 Metropolitan height and weight tables *Stat Bull Metrop Life Insur Co* 1984; **64**: 2–9.
16 Garner DM, Garfinkel PE The Eating Attitudes Test: An index of the symptoms of anorexia nervosa *Psychol Med* 1979; **9**: 273–279.
17 Turnage JJ, Kennedy RS The development and use of a computerized human performance test battery for repeated-measures applications *Human Performance* 1992; **5** (4): 265–301.
18 Krause M, Bittner AC, Jr. *Repeated measures on a choice reaction time task* (Rep. No. NBDL-82R006). New Orleans: Naval Biodynamics Laboratory. (NTIS No. AD A121904), 1982.
19 Davies R, Moray N, Triesman A Imitation responses and the rate of gain of information *Quart J Exp Psychol* 1961; **71**: 321–330.
20 Jones MB. *Stabilization and task definition in a performance test battery* (Final Report, Contract N00203-79-N-5089). New Orleans: U.S. Naval Aerospace Medical Research Laboratory, 1979.
21 Jones MB Sequential precession and diminishing returns in

the acquisition of a motor skill *J Motor Behavior* 1980; **12**: 69–73.
22 National Research Council. *Recommended Dietary Allowances*. 10th ed. National Academy Press: Washington, DC, 1989.
23 US Department of Agriculture. Composition of foods. Agriculture handbook no. 8, revisions 8.1–8.21. Washington, DC: US Government Printing Office, 1976–92.
24 Van Loan MD, Keim NL, Belko AZ. Body composition assessment of a general population using total body electrical conductivity (TOBEC). In: Hermans GPH (ed). *Sports, Medicine and Health*. Pergamon (Elsevier Science): Tarrytown, NY, 1990, pp 665–670.
25 van Strien T, Frijters JER, Bergers GPA, Defares PB The Dutch Eating Behaviour Questionnaire (DEBQ) for assessment of restrained, emotional and external eating behaviour *Int J Eat Dis* 1986; **5**: 295–315.
26 Speilbeger CD *et al Manual for the State-trait Anxiety Inventory*. Palo Alto: Consulting Psychologists Press, Inc., 1983.
27 Maxwell JK, Tucker DM, Townes BD Asymmetric cognitive function in anorexia nervosa *Int J Neuroscience* 1984; **24**: 37–44.
28 Gold PE Role of glucose in regulating the brain and cognition *Am J Clin Nutr* 1995; **61** (suppl): 987S–995S.
29 Hall JL *et al* Glucose enhancement of performance on memory tests in young and aged humans *Neuropsychologia* 1989; **27**: 1129–1138.
30 Manning CA, Hall JL, Gold PE Glucose effects on memory and other neuropsychological tests in elderly humans *Psychol Sci* 1990; **1**: 307–311.
31 Parsons M, Gold PE Glucose enhancement of memory in elderly humans: an inverted-U dose-response curve *Neurobiol Aging* 1992; **13**: 401–404.