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Energy intake highs and lows: how much does consistency matter in weight control?

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Summary

Behavioural weight control programmes recommend adherence to daily energy intake goals, yet also allow for flexibility in intake across days. Evidence is lacking as to whether intake consistency is important for weight control. The current study explored the relation between day-to-day intake consistency and weight loss in the context of behavioural weight loss treatment and examined the relationship between variability in intake and several factors known to be associated with weight control success. Participants (N = 283) enrolled in a 12-month behavioural weight loss programme completed 24-h recalls of dietary intake and psychological measures. At the end of treatment, low intake variability and greater weight loss were associated, but variability was not predictive of weight loss independent of mean intake in continuous analyses. Interestingly, participants who met the programme goal of ≥10% weight loss had less intake variability compared to those who lost <10%, although groups did not differ significantly on mean intake. Results suggest that daily intake consistency may facilitate successful weight loss for some. Additionally, autonomous motivation for weight management and cognitive dietary restraint were inversely related to end-of-treatment intake variability. Additional research is needed to examine whether recommendations to limit intake variability during behavioural weight loss treatment improve long-term weight control.

Keywords

Behavioural weight loss; calories; Intake variability; obesity treatment

Reduced energy intake is recommended for weight loss among overweight and obese adults (body mass index [BMI] ≥ 25.0 kg m^{-2}) (1) and is a key component of behavioural weight management programmes (2). Such programmes commonly use a continuous energy restriction approach in which a daily kilojoule (kJ), or kilocalorie (kcal), goal is set to reduce

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Conflict of Interest Statement

No conflict of interest was declared.

Author contributions

MLB, EMF and MRL designed the main study from which this substudy was formed. MLB and DLR designed the substudy. DLR analysed data. DLR, LMS, KS, MLB, EMF and MRL interpreted the data. LMS, ADP and MEG conducted literature searches. All authors were involved in writing the manuscript, provided critical intellectual contributions to manuscript revision, and approved the final version of the submitted manuscript.

individuals' intake below their energy needs, producing an energy deficit (2–4). Despite the importance of energy intake reduction for successful weight management, many individuals struggle with long-term reduction in intake (2). It is possible that minimizing daily fluctuations in energy intake helps individuals achieve reduced intake over time; however, it is unknown if the intake patterns through which individuals achieve energy deficits affect weight control success. Research has not yet specifically investigated whether consistency in intake across days relates to treatment outcomes. Given the limited research thus far, a descriptive understanding of this potentially important factor (i.e. intake variability) in weight control treatment is needed.

Existing behavioural weight control programmes suggest averaging intake across a period of a week and comparing mean daily intake to goals when determining successful adherence (5). The intake goal that each individual is prescribed is based upon his or her body weight and typically ranges between 5020 and 7531 kJ day^{-1} (1200 and 1800 kcal day^{-1}) (4). Individuals who anticipate difficulty eating within their intake goal on certain days (e.g. due to special occasions) are encouraged to eat below their intake target on one or more preceding days, thereby 'banking' or saving kJs/kcals to balance the anticipated overeating (5). Such recommendations imply that a certain amount of day-to-day variability in intake is acceptable, expected and will not interfere meaningfully with meeting kJ/kcal goals. While it is well known that free-living adults exhibit considerable variability in day-to-day intake (6–8), the degree of variability in intake present among individuals participating in behavioural weight control treatment has yet to be examined. Moreover, the relationship between intake variability and weight loss outcomes in such programmes has not been empirically investigated.

Some evidence supports the allowance of variability in intake in weight-loss efforts. For instance, difficulty remaining adherent to a reduced-energy diet is believed to contribute to weight regain over time (9). In response to challenges associated with consistent energy restriction, several alternative approaches to weight loss and weight loss maintenance have been examined. Preliminary research indicates that intermittent energy restriction (i.e. intermittent periods of complete or partially restricted energy intake and *ad libitum* eating on non-restricted days) produces weight losses comparable to those observed with consistent energy restriction (10,11). Similar weight losses were also observed between individuals prescribed a consistent daily 5020.8 kJ (1200 kcal) diet and those prescribed an alternating-calorie diet of between 2510.4 (600) kcal day^{-1} and 7531.2 (1800) kcal day^{-1} (12). Such evidence suggests that intake variability does not necessarily undermine weight loss success. Although there is currently no research on naturally occurring intake variability (vs. prescribed intake variability) in behavioural weight control programmes, it is possible that greater variability within standardized calorie prescriptions may make weight control efforts more acceptable to individuals by allowing customization of eating behaviours to personal preferences (e.g. greater intake on weekends).

Conversely, it is also possible that limiting intake variability may improve weight control outcomes. Eating a similar number of kJs/kcals each day may, for example, be associated with the formation of stronger healthy eating habits, subsequently promoting the maintenance of reduced energy intake and weight loss over time (13). Greater consistency

could also help individuals reduce their mean intake level (e.g. due to fewer overeating days). Indeed, greater intake variability is associated with greater mean intake among adults not attempting to lose weight (14). Previous examinations suggest that although mean intake level and degree of variability are related, they can be empirically evaluated as separate descriptors of an individual's eating behaviour without redundancy (14). For instance, some may consume a high amount of energy consistently and, therefore, have low variability but high mean intake.

Examination of the relationship between consistent eating patterns and long-term weight loss maintenance suggest limited variability is beneficial. For example, among a study of more than 1400 individuals who had lost at least 13.63 kg (30 lb) and kept the weight off for at least 1 year, self-reported consistency in one's 'strictness' of dieting across both the week (week vs. weekend) and year (holidays vs. rest of the year) was associated with less weight regain over 1 year as well as the greater likelihood of maintaining one's weight within 2.27 kg (5 lb). (15) A later study found similar results when examining risk of weight regain vs. maintenance over 2 years (16). Such findings support the association between dieting consistency and weight control and indicate that greater consistency in intake may improve weight control outcomes. If a relationship between intake variability and weight control exists, it may be important to evaluate the pathways through which this relationship theoretically operates over time; prior to that, however, additional evidence is needed to support the existence of associations between intake variability and outcomes at the conclusion of weight loss treatment.

Research also has yet to investigate psychological factors that may relate to intake variability. Several factors, such as hedonic hunger (i.e. one's appetitive drive to consume palatable food) (17), cognitive dietary restraint (i.e. the degree of conscious control one exhibits over eating behaviours) (18), uncontrolled eating (i.e. one's propensity to eat in response to food cues or emotions) (19) and autonomous motivation for treatment (i.e. the extent to which these behaviours are motivated by personal volition and choice) (20), may also relate to consistency of intake. Examination of whether these factors, which have been associated with weight loss outcomes generally (21–24), are also associated with calorie variability may inform understanding of the expression of calorie variability in weight control. That is, investigation of these potential associations is needed to establish a foundation prior to future evaluations of pathways through which calorie variability may operate in the context of weight control. For instance, it may be that those with greater autonomous motivation for treatment and cognitive restraint may have better control over their eating, yielding lower intake variability and better weight control outcomes. While evaluating such models was outside of the scope of this project, several measures of appetite and eating control were available in the present study and were thus examined to try to gain further insight into possible correlations of intake variability.

At present, the majority of behavioural weight control programmes allow, and even encourage, some day-to-day variability in energy intake. Despite the ubiquity of this recommendation, the relationship of intake variability to weight loss success has not been examined empirically. While it is possible that variability allows for flexibility and is thus a useful weight control tool, it is equally plausible that such variability is problematic.

Given the clinical importance of determining best practices for long-term weight control, the present study examined the associations between intake variability, mean intake, outcome-related psychological factors and weight loss among individuals participating in a year-long behavioural weight control programme. To provide preliminary information on intake consistency in a weight management effort, we examined patterns of variability at two time points: baseline and end-of-treatment. Specifically, to describe the role of intake variability, we aimed (i) to examine the relationship between intake variability as assessed at baseline and end-of-treatment, mean intake and weight loss; (ii) to explore the relationships between intake variability and several psychological factors (i.e. hedonic hunger, cognitive restraint, uncontrolled eating and autonomous motivation) known to be associated with weight control, and (iii) after examining the abovementioned dimensional relationships, to compare intake variability, and the relationships among intake variability and the aforementioned psychological factors, between individuals who did and did not achieve the weight loss goal set by the programme (i.e., ≥10% of initial body weight) to better describe these subgroups.

Method

Participants

Overweight and obese adults (N = 283) were recruited from a large northeastern metropolitan area in the United States for a behavioural weight loss treatment study. The current study was part of a larger trial (R01 DK092374). Eligibility required a BMI of 27–45 kg m^{-2}, age between 18 and 70 years and ability to engage in physical activity (i.e. can walk at least two blocks without stopping for rest). Participants were excluded from the study if they were pregnant or planning to become pregnant over the course of the study; were planning to move from the area or to participate in another weight loss programme; recently began a course of or changed the dosage of medication that could significantly impact weight; lost more than 5% of body weight in the past 6 months; or had a medical or psychiatric condition that could interfere with compliance to the programme's behavioural recommendations. The Institutional Review Board at the supporting university approved the study.

Procedures

Participants received 26 sessions of group-based behavioural weight loss treatment over the course of 1 year. Behavioural and calorie goals reflected standard balanced deficit diet guidelines (i.e. 6276–7531.2 kJ day^{-1} [1500–-1800 kcal day^{-1}] if weight was greater than 113.63 kg [250 lb] or 5021–6276 kJ day^{-1} [1200–1500 kcal day^{-1}] if weight was less than 113.63 kg [250 lb]) (25). Treatment did not include prescribed variability (e.g. fasting), and recommendations regarding daily intake variability were similar to those in other behavioural weight control programmes. For example, participants were instructed to average their energy intake across each week, and to 'bank' kilojoules (kcals) for occasions on which they may eat above their intake goals. Participants were also prescribed a weekly physical activity goal that gradually increased to 250 min $week^{-1}$ of moderate-to-vigorous activity (26). Participants completed research assessments at baseline (T1), mid-treatment (T2; 6 months) and end-of-treatment (T3; 12 months).

Measures

At baseline, participants self-reported their age, gender, race and ethnicity. Body weight was measured in duplicate by study staff at baseline, mid-treatment and end-of-treatment using a Seca® scale accurate to 0.1 kg (measured in light street clothes). Height was measured in duplicate at baseline using a stadiometer. Participants completed dietary recalls at baseline and end-of-treatment. Psychological measures, as described below, were completed at baseline, mid-treatment and end-of-treatment.

Dietary recall—Registered dieticians and experienced support staff from Cincinnati Children's Hospital Medical Center assessed dietary intake using the USDA Automated Multiple-Pass Method (27). Assessors used the latest version of the Nutrition Data System for Research software developed by the University of Minnesota to collect and analyse dietary intake data (28). The food recall included non-consecutive 24-h recalls of two weekdays and one weekend day. The USDA Automated Multiple-Pass Method has been found to provide an accurate measurement of calorie and nutrient intake (29–31).

Power of food scale—The power of food scale (PFS) assesses responsivity to highly palatable foods, also referred to as hedonic hunger. The PFS has adequate internal and test–retest reliability and convergent and discriminant validity (17,32,33). Higher scores indicate greater hedonic hunger.

Three-Factor Eating Questionnaire – Revised 18-item version—Subscales of the Three Factor Eating Questionnaire – Revised 18-item version (TFEQ-R18) assess uncontrolled eating and cognitive restraint (i.e. control over food intake in order to influence body weight and body shape). Higher scores indicate greater uncontrolled eating and greater cognitive restraint, respectively. The TFEQ-R18 has demonstrated strong factor structure and adequate reliability (18).

Treatment Self-Regulation Questionnaire—The version of the Treatment Self-Regulation Questionnaire (TSRQ) completed by participants measures autonomous motivation for changing exercise and diet (34). Higher scores indicate higher autonomous motivation. The TSRQ has adequate construct validity and test–retest reliability (35).

Results

Sample characteristics

Data from eight individuals whose 24-h recall data were determined to be unrealistically low (i.e. ≤3138 kJ or 750 kcals) on at least 1 day were excluded from analyses due to likely reporting error. Therefore, the final sample was comprised of 275 participants at baseline; full data were available for 192 participants at end-of-treatment. Notably, a similar pattern of results was obtained when all participants were included.

The sample was 21% male, ranged in age from 21 to 70 years old ($M = 53.14$, $SD = 9.68$) and had a mean BMI of 35.10 kg m^{-2} ($SD = 4.93$). Approximately 8% of participants identified as Hispanic/Latino. Participants self-identified as White (66%), African–American or Black (29%), American Indian/Alaskan Native (<1%), Asian (<1%) and Native

Hawaiian/Pacific Islander (<1%). Approximately 4% of participants identified as more than one race.

Data analyses

Descriptive statistics—Data were analysed using SPSS version 22 (36). Table 1 displays the means, standard deviations and maximum and minimum values for intake variability and mean intake at baseline and end-of-treatment for (i) the full sample, (ii) those who lost ≥10% of their initial weight at end-of-treatment ($n = 107$) and (iii) those who lost <10% of their initial weight at end-of-treatment ($n = 85$). We operationalized intake variability as the difference (highest intake amount – lowest intake amount) in daily intake reported across the three days of 24-h food recall data per assessment point. We elected to operationalize variability in this manner as (i) it most accurately captures the spread of intake, (ii) has clinical relevance and (iii) the standard deviation, measuring distance from the mean, was suboptimal given that each assessment included only three data points for intake.

A paired sample *t*-test revealed that mean intake significantly decreased from baseline to end-of-treatment for the full sample ($t(178) = 8.53$, $P < .001$, M decrease = 1360 kJ (325 kcal)) and in the subgroup who lost ≥10% ($t(98) = 8.14$, $P < .001$, M decrease = 1649 kJ (394 kcal)). Similarly, calorie variability decreased in the full sample ($t(178) = 5.00$, $P < .001$, M decrease = 1092 kJ (261 kcal)) as well as in the subgroup of those who lost ≥10% ($t(98) = 4.88$, $P < .001$, M decrease = 1184.07 kJ (283 kcal)). Table 2 displays the correlations between calorie variables, psychological variables and weight variables for the full sample along with the ≥10% weight loss subgroup.

Associations with intake variability in the full sample—Intake variability was cross-sectionally associated with mean kJ at both baseline and end-of-treatment (baseline: $r = .52$, $P < .001$; end-of-treatment: $r = .53$, $P < .001$), such that greater variability was associated with greater mean intake. The relationship between intake variability and BMI at baseline was weak and did not reach statistical significance ($r = .07$, $P = .24$).

Intake variability was also associated with psychological measures. Intake variability was specifically cross-sectionally associated with cognitive restraint at both time points (baseline: $r = -.20$, $P = .001$; end-of-treatment: $r = -.19$, $P = .01$), with greater variability being associated with lower cognitive restraint. After controlling for mean intake, the associations between intake variability and cognitive restraint at baseline ($F(2, 264) = 7.93$, $B = -.10$, $P = .16$) and end-of-treatment ($F(2, 162) = 4.25$, $B = -.13$, $P = .15$) were non-significant. At baseline, intake variability was cross-sectionally associated with uncontrolled eating ($r = .15$, $P = .01$), such that greater variability related to greater uncontrolled eating. Autonomous motivation at mid-treatment was predictive of intake variability at end-of-treatment ($r = -.17$, $P = .02$), with greater autonomous motivation relating to less variability. After controlling for mean intake, the cross-sectional association between uncontrolled eating and intake variability at baseline ($F(2, 265) = 9.82$, $B = .004$, $P = .95$) and the predictive association between autonomous motivation at mid-treatment and intake variability at end-of-treatment ($F(2, 175) = 4.23$, $B = -.09$, $P = .29$) were also not significant.

We conducted multiple linear regression analyses to examine the relationship between end-of-treatment intake variability and weight loss. Because individuals with lower baseline weight had lower recommended intake goals, it is possible they would be more limited in the extent to which their kJ could vary below their goal. To evaluate the possibility that low variability and weight loss success was thus an artefact of limited range in potential daily kilojoule intake, we examined the relationships between intake variability in separate models controlling for (i) baseline BMI, (ii) baseline intake variability and (iii) mean intake at end-of-treatment. In the model controlling for baseline BMI, end-of-treatment intake variability was associated with end-of-treatment percent weight loss ($F(2182) = 3.19$, $B = .17$, $P = .02$). The percent weight lost (which is measured on a negative scale) improved by .17 per 1 kJ improvement in variability. End-of-treatment intake variability remained significantly associated with percent weight loss after controlling for baseline intake variability ($F(2, 176) = 2.18$, $B = .15$, $P = .05$). The bivariate relationship between intake variability and weight loss at end-of-treatment was significant ($r = .16$, $P = .03$), although this relationship was no longer statistically significant ($F(2, 180) = 4.04$, $B = .07$, $P = .39$) when controlling for mean kJ at end-of-treatment.

Differences between weight loss subgroups—The *t*-tests revealed no significant differences in intake variability at baseline ($t(269) = .23$, $P = .82$), or in mean intake at baseline ($t(268) = 1.73$, $P = .08$) or end-of-treatment ($t(182) = -1.72$, $P = .09$) between those who lost ≥10% and those who lost <10% of their weight at end-of-treatment. There was a significant difference between groups in intake variability at end-of-treatment, however, such that those with ≥10% weight loss reported lower variability ($t(181) = -2.40$, $P = .02$).

Associations with intake variability in ≥10% weight loss subgroup—As with the full sample, intake variability was cross-sectionally associated with mean kJs at both time points among individuals who achieved ≥10% weight loss (baseline: $r = .48$, $P < .001$; end-of-treatment: $r = .50$, $P < .001$), such that greater variability related to greater mean intake. Intake variability was also cross-sectionally associated with autonomous motivation at end-of-treatment ($r = -.21$, $P = .04$). The only mid-treatment predictor of end-of-treatment intake variability among the psychological variables assessed was autonomous motivation ($r = -.21$, $P = .03$), with greater autonomous motivation at mid-treatment relating to less variability at end-of-treatment. When this relationship was examined in a multiple linear regression model controlling for end-of-treatment mean intake ($B = .49$, $P < .001$), the relationship between mid-treatment autonomous motivation and end-of-treatment intake variability was no longer significant ($B = -.05$, $P = .59$).

Discussion

Reduced energy intake is critical for weight loss (2), but many individuals may experience difficulties reducing or sustaining reduced intake. It is thus important to examine factors that may be related to lowered intake. The current study provides a descriptive account of one such factor – intake variability – at two relevant time points (baseline and end-of-treatment) in the context of behavioural weight control treatment, with several notable findings. First, intake ranges across a 3-day food recall were relatively large at baseline (i.e. 3452 kJ, or 825 kcals, on average). Participants continued to show a considerable amount of variability in

intake at the end of treatment, although it was significantly decreased. Second, consistent with previous research (14), intake variability related to mean intake to a moderate degree at both baseline and end-of-treatment. Although intake variability at baseline was not related to initial BMI or percent weight loss at end-of-treatment, end-of-treatment variability did relate to treatment outcomes, such that participants who exhibited greater variability exhibited less weight loss. The relationship between intake variability and treatment outcomes was not independent of overall intake. These findings suggest that, while overall intake is the most important determinant of weight loss, lower intake variability may function as one method to consume fewer kJ.

When examining the relations of relevant psychological variables to intake variability, cognitive restraint and uncontrolled eating at baseline were both cross-sectionally related to variability, while mid-treatment autonomous motivation and end-of-treatment cognitive restraint were related to end-of-treatment variability. When considering only successful individuals (i.e. those meeting the programme goal, achieving ≥10% weight loss), autonomous motivation at both mid- and end-of-treatment related to variability at end-of-treatment. Together, these results indicate that cognitive restraint and autonomous motivation may help maintain consistency in intake across days during a weight loss effort. Of note, none of the psychological variables were significant predictors of variability after mean intake was also considered as a covariate, which suggests that the relationships between these constructs and variability is not unique from mean intake.

Comparisons of successful and less successful individuals, as defined by the programme goal of ≥10% weight loss, provide additional information regarding the relationship of intake variability to weight control. There were no baseline differences in intake variability or mean intake for those who went on to lose at least 10% of their body weight compared to those who did not reach this threshold. Although the groups did not differ in mean intake at end-of-treatment, those who lost ≥10% of their weight had lower intake variability, highlighting the potential importance of variability in treatment success. However, it is somewhat surprising that those who were successful in meeting the 10% weight loss goal at the conclusion of treatment did not have significantly lower mean intake at end-of-treatment, and conclusions regarding the independent role of intake variability are premature based on these data. It is possible, for instance, that those with reduced variability were also more accurately reporting intake, and thus, differences in overall intake were not reflected.

Overall, these preliminary findings suggest that reduced intake variability in the context of behavioural weight loss treatment is associated with effective weight management, although this relationship is not predictive above the effect of mean intake level. The relationship of variability to weight outcomes at end-of-treatment, but not baseline, suggests that consistency in intake may be one pathway through which overall intake is lowered during the course of treatment and after guidelines for weight loss (e.g. prescription of an intake goal) and self-monitoring skills are established. Higher variability in intake may make it more difficult for individuals to consume below daily intake targets consistent with weight management goals, impeding long-term success. For example, it may be difficult for an individual to maintain a low kJ average across time when routine eating patterns are not well-established. These results are consistent with previous research that has found that

intermittent non-adherence to energy intake goals predict weight loss plateaus occurring after 6 months of treatment using dynamic mathematical models (37).

Limitations

Several limitations of the current study should be noted. First, only 3 days of intake at baseline and end-of-treatment were collected, and examining variability in intake across a broader time frame may improve the validity of variability data. In addition, the current study defined intake variability as the difference in kJ between the highest and lowest days. While this statistical approach improves the clinical utility of the current findings through provision of treatment recommendations, other, more statistically advanced methods of calculating variability exist and may be informative in future investigations. The use of 24-h food recalls to determine kJ intake may have been subjected to reporting error; however, experts trained to probe for under-reporting conducted these recalls, thus providing more accurate data than alternative self-report measures (31). Furthermore, 24-h recall data were likely more accurate at the end-of-treatment assessment as participants received extensive training in accurate self-monitoring during the intervention. Another limitation concerns differing intake goals across individuals. Although individuals presumably had different intake goals at end-of-treatment, we did not assess precise intake goals. Additionally, we assessed baseline intake prior to prescription of intake goals. Consequently, we were unable to examine variability relative to one's desired intake level (e.g. as a percentage of daily goal). Finally, food recalls were not collected at mid-treatment, limiting the extent to which temporal relationships could be examined. Due to the correlational nature of the results, we were thus unable to make inferences related to causality from the current investigation.

Future research and potential implications

Future research should continue to explore the relationship between variability and treatment success. While the current study evaluated intake consistency at the beginning and end-of-treatment, additional information about intake throughout a weight management effort will be vital to further exploring the relationship between variability, overall intake and weight loss. Furthermore, a design that employs random assignment to variability recommendations would aid in exploration of whether reductions in variability enhance treatment efficacy. Longitudinal studies should explore the causal relationships between variability, mean intake and effective weight management, specifically evaluating theoretical models of the relationship between consistency and overall intake. Future studies should also examine whether the effect of intake variability is mediated by overall intake. In addition, future investigations should examine patterns of eating, activity and psychological variables for individuals who are able to lose weight while also having a high degree of variability in intake as such findings could be of use when tailoring recommendations across individuals.

If intake variability does prove to be a pathway towards reduced intake in other samples, future research should evaluate and identify clinical recommendations regarding variability to determine if specific variability ranges impact adherence to intake targets. This investigation, for instance, found that, among those who lost ≥10% of initial body weight, the average difference between their highest and lowest calorie day was just over 2092 kJ (500 kcal). Additionally, if future investigations confirm that consistency is a pathway for

reducing intake, research should examine whether the psychological variables examined in this study impact the development of consistent eating patterns. Finally, future research should investigate whether alternative methods of achieving sustained reductions in overall intake are more or less effective than reduced intake variability.

Conclusion

In conclusion, the current study is the first to investigate the nature of intake variability in the context of behavioural weight loss treatment. Our findings indicate that, on average, intake variability decreases during treatment and provide preliminary support for the relevance of reductions in intake variability to treatment success. Notably, the effect of intake variability was not independent of overall intake, and, as such, lower variability may aid individuals in achieving sustained reductions in overall intake, which subsequently impacts weight. It is important to continue to evaluate and refine clinical recommendations for ways in which lower overall intake may be maintained as these are most likely to promote long-term treatment adherence and improve outcomes in behavioural weight management.

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What is already known about this subject?

- Research on the relation between day-to-day kilojoule/kilocalorie intake variability and weight loss outcomes is limited.

What does this study add?

- Behavioural weight loss participants with lower intake variability at the end of treatment exhibited greater weight losses. Of note, those with lower intake variability also had lower intake overall; therefore, conclusions about the unique importance of intake variability may be inappropriate.
- Autonomous motivation for weight management and cognitive dietary restraint were inversely related to intake variability at the end of treatment.

Table 1

Descriptive statistics

	Mean (kJ)	Mean (kcals)	Standard deviation (kJ)	Standard deviation (kcals)	Max (kJ)	Max (kcals)	Min (kJ)	Min (kcals)
Full sample								
T1 intake variability	3450	825	2385	570	20 054	4793	303	72
T1 mean intake	7875	1882	2212	529	18 762	4484	3474	830
T3 intake variability	2518	602	1747	417	9221	2204	150	36
T3 mean intake	6659	1591	1662	397	12 405	2965	3561	851
≥10% weight loss								
T1 intake variability	3488	834	2058	492	10 194	2436	303	72
T1 mean intake	8141	1946	2073	495	14 373	3435	4409	1054
T3 intake variability	2239	535	1494	357	6943	1659	150	36
T3 mean intake	6472	1547	1555	372	12 161	2906	3887	929
<10% weight loss								
T1 intake variability	3421	818	2612	624	20 054	4793	321	77
T1 mean intake	7671	1833	2298	549	18 762	4484	3474	830
T3 intake variability	2877	688	1978	473	9221	2204	599	143
T3 mean intake	6896	1648	1770	423	12 405	2965	3561	851

kJ, kilojoules; kcals, kilocalories; T1, baseline; T3, end-of-treatment.

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Table 2

Correlation matrix[†]

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1.T1 intake variability		.52***	−.01	−.20**	.15*	.11	.07	−.14*	−.06	.12	.07	.13	.29***	−.02	−.12	−.16*	−.05	−.01
2. T1 mean intake	.48***		−.08	−.22***	.26***	.18**	.07	−.13	−.01	.17*	.14*	.08	.38***	−.10	−.11	−.24***	.04	−.09
3. T1 autonomous	−.08	−.24**		.19**	−.07	−.00	.05	.41***	.17**	−.08	−.04	−.01	−.14	.35***	.14	−.12	−.08	.01
4. T1 cog. Restraint	−.21*	−.23*	.15		−.04	−.07	−.11	.06	.23***	.01	−.03	−.05	−.13	.08	.37***	.03	.00	.05
5. T1 uncontrolled eating	.02	.23*	−.20*	.05		.65***	.06	−.12	.12	.59***	.44***	−.02	.09	−.11	.08	.61***	.12***	−.06
6. T1 power of food	−.08	.03	−.11	−.02	.68***		.07	−.02	−.18**	.37**	.65***	−.03	.10	−.02	.12	.42***	.57***	−.10
7. T1 BMI	−.06	−.10	−.01	−.07	.07	.09		−.07	−.12	.01	.06	−.02	.01	−.06	−.15*	−.01	.02	.12*
8. T2 autonomous	−.20*	−.21*	.50***	.09	−.10	−.00	−.00		.33***	−.34***	−.13*	−.17*	−.20**	.66***	.31***	−.29***	−.23**	−.21**
9. T2 cog. restraint	−.04	.02	.22*	.18*	.12	.14	−.05	.34***		−.09	.08	−.13	−.09	.32**	.58***	−.10	.05	−.33***
10. T2 uncontrolled eating	−.01	.18	−.26**	.10	.54***	.41***	−.03	−.22*	.01		.54***	.05	.11	−.22**	−.08	.75***	.49***	.16*
11. T2 power of food	−.19*	−.02	−.26**	−.09	.44***	.64***	−.03	−.10	.10	.57***		−.02	.08	−.16*	−.00	.48***	.72***	.02
12. T3 intake variability	.11	.02	.07	−.15	−.14	−.08	.00	−.21*	−.11	−.11	−.01		.53***	−.08	−.19*	.12	.05	.16*
13. T3 mean intake	.18	.36***	−.11	−.21*	.09	.10	−.11	−.33**	−.07	.10	.18	.50***		−.11	−.20**	.17*	.12	.18*
14. T3 autonomous	−.06	−.12	.33***	.08	−.04	.04	.01	.70***	.43***	−.10	−.08	−.21*	−.32**		.31***	−.27***	−.14	−.24***
15. T3 cog. restraint	−.07	−.11	.03	.33***	.08	.07	−.10	.22*	.60***	.00	.02	−.18	−.19	.21*		−.13	.03	−.37***
16. T3 uncontrolled Eating	.13	.15	−.26**	.11	.60***	.40***	−.01	−.23*	−.05	.72***	.40***	−.01	.14	−.16	−.06		.56***	.16*
17. T3 power of food	−.20*	−.03	−.20*	−.00	.49***	.59***	−.05	−.13	.12	.49***	.72***	.08	.23*	−.03	.09	.48***		.08
18. T3 weight loss (%)[‡]	.04	.01	.05	−.00	.02	.00	−.02	−.15	−.22*	.11	.02	.04	.21*	−.04	−.25**	.01	.02	

[†]Correlations are presented for full sample (above diagonal) and for the ≥10% weight loss subgroup (below diagonal), respectively.

[‡]Weight loss percent is scored with a negative value

*$P < .05$,

**$P < .01$,

$^{***}P < .001$

T1, baseline; T2, mid-treatment; T3, end-of-treatment.