Portion Control Plate for Weight Loss in Obese Patients With Type 2 Diabetes Mellitus

A Controlled Clinical Trial

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Background: Portion size is an important determinant of energy intake. To our knowledge, no randomized controlled trial has evaluated the efficacy of portion control tools to induce weight loss. In patients with type 2 diabetes mellitus, weight reduction improves glycemic control.

Methods: We randomly assigned 130 obese patients with type 2 diabetes mellitus (including 55 patients taking insulin) to the daily use of a commercially available portion control plate for 6 months (intervention group) vs to usual care in the form of dietary teaching (usual care control group).

Results: Follow-up was 93.8%. Patients in the intervention group lost significantly more weight than control subjects (mean±SD, 1.8%±3.9% vs 0.1%±3.0%, P=.006). Compared with controls, more patients in the intervention group required a decrease in their diabetes medications at 6 months (26.2% vs 10.8%, P=.04).

Conclusions: Compared with usual care, the portion control tool studied was effective in inducing weight loss. The portion control plate also enabled patients with diabetes mellitus to decrease their hypoglycemic medications without sacrificing glycemic control.

Trial Registration: clinicaltrials.gov Identifier: NCT00254124

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Obesity has become a worldwide epidemic, and its prevalence is increasing. Between 1960 and 2000, the prevalence of obesity among American adults increased from 13.4% to 30.9%.1,2 Most cases of type 2 diabetes mellitus (DM) can be attributed directly to obesity. The risk of DM varies with the degree, duration, and distribution of obesity.3,4 Dietary caloric restriction has been shown to improve glycemic control by virtue of weight loss,5 with an additional benefit independent of weight loss.6

The increasing prevalence of obesity is paralleled by increasing portion sizes in the marketplace.7 Portion sizes are an important determinant of energy intake; the number of calories ingested by subjects at a meal has been directly correlated with the serving size offered.7,8 Despite the plethora of diet strategies available, it is primarily the net caloric intake that determines the net change in weight.9,10 The principles of portion control are commonly used by diabetes educators to effect a hypocaloric diet; patients are often taught to use reference sizes relative to their hand size to guide protein, carbohydrate, and fat portions.11 However, to our knowledge, no clinical trial has been published that examined the efficacy of a food portion control tool to control caloric intake and thereby induce weight loss. We conducted a 6-month randomized controlled trial designed to determine the efficacy of a portion control plate to induce weight loss among a population with obesity and type 2 DM.

METHODS

PARTICIPANTS

Patients were recruited from April 1, 2004, through June 30, 2004, from an outpatient diabetes center in Calgary, Alberta. Patients were recruited via fliers posted at the diabetes center or by referral from their diabetes health care providers (ie, physicians, nurses, or dieticians). All included subjects had type 2 DM and a body mass index (calculated as weight in kilograms divided by height in meters squared) of at least 30 and had to be clients of the diabetes center, with at least 6 months’ prestudy.
teaching and management by diabetes nurse educators and dieticians. Exclusion criteria were as follows: presence of cancer, current weight loss medication, history of bulimia or anorexia nervosa, psychiatric illness under the care of a psychiatrist, surgery in the 3 months before enrollment or planned during the study period, weight loss exceeding 10 lb (4.5 kg) in the 2 months preceding study enrollment, and consumption of dinner at a restaurant more than twice weekly or consumption of more than 30% of all meals at restaurants. All participants provided written informed consent, and the local institutional ethical review board approved the protocol. The study was funded by the Stewart Diabetes Education Fund, a non–industry-based trust fund for diabetes research.

STUDY PROTOCOL

A detailed medical and demographic history was recorded at the baseline appointment. Sealed envelopes containing group designations were used to randomly assign subjects to 1 of 2 groups. The intervention group received a calibrated dinner plate and cereal bowl as a means of dietary portion control. A 10- to 15-minute individual counseling session and a booklet outlining the use of these tools were provided. The control group did not receive any treatment or counseling outside of their usual clinical care in the form of dietary assessment and teaching by their dieticians. During the study, patients had access to their dieticians on an as-needed basis as per usual care.

For the 6-month duration of the study, participants agreed not to start any weight loss medication or special diets during the trial period. Participants were contacted by telephone on 4 occasions during the study to remind them of study participation. Study investigators were not involved in any medication adjustments that were made; patients were instructed to consult with their usual physician to make any necessary adjustments.

Patients were seen in follow-up at 6 months, at which time changes in medications and dosages were assessed on an individual basis. Any new hypoglycemic medications or change from one medication to another was assumed to represent an increase in intensity of hypoglycemic medical therapy. If the number of units of insulin was increased in conjunction with a decrease or discontinuation of an oral hypoglycemic agent, the patient was classified as having an increase in intensity of medical therapy. The same principles were applied to any changes made in lipid-lowering or antihypertensive agents.

INTERVENTION

A commercially available calibrated dinner plate and breakfast bowl constituted the intervention (Figure 1). The plate sizes are sex specific; the male plate is calibrated for an approximate 800-cal (3347-J) meal, whereas the female plate is calibrated for a 650-cal (2720-J) meal. This caloric goal is accomplished by dividing the plate area into sections designed to contain predetermined volumes of carbohydrates, proteins, cheese, and sauces, with the remainder of the plate open for vegetables. For consumption of mixed meals that cannot be readily divided into the respective macronutrient sections, food is portioned into the section that represents that meal's dominant macronutrient.

The cereal bowl is calibrated to portion-control breakfast cereal and is not sex specific. This bowl is designed to allow a 200-cal (837-J) meal consisting of cereal and 1/2 cup (118 mL) of milk; this goal is accomplished by calibration at 4 different levels to accommodate cereals of differing caloric density. Subjects were provided with a list of commonly consumed cereals with their corresponding calibration levels in the bowl.

Subjects were instructed to use the calibrated plate for their largest meal of the day and to use the cereal bowl on days when cereal was consumed as their breakfast meal. Patients were counseled not to compensate for portion control at mealtime by eating more at other times of day. To document adherence, participants were asked to keep a daily log documenting their use.

OUTCOME MEASURES

Outcome measures were assessed at baseline and at 6 months. Body weight of the participants, while wearing a hospital gown and no shoes, was measured using a single calibrated scale (Health O Meter; Continental Scale Corporation, Chicago, Ill). Blood pressure was measured using a validated automated machine (BP Tru; VSM Med Tech Ltd, Vancouver, British Columbia). Glycosylated hemoglobin level was measured at baseline and at 6 months by immunoassay (Roche Intra 700; Roche Diagnostics, Indianapolis, Ind), and serum lipid levels were measured following an overnight fast (Roche Hitachi Modular P800 Analyzer; Roche Diagnostics). Low-density lipoprotein levels were calculated according to the Friedewald equation.14

Figure 1. Diet plate and breakfast bowl (The Diet Plate, Glossop, England).13
STATISTICAL ANALYSIS

The primary end point was defined as percentage change from baseline weight. A sample size of 98 individuals would have an 80% power to detect a significant 5% weight loss in the intervention group, assuming a 1% reduction in body weight in the control group and a common SD of 7%. Given an anticipated dropout rate of 25%, the enrollment target was set at 130 patients. A prespecified analysis examining the proportion of patients in each group who achieved at least 5% weight loss was performed. Analyses of change in weight were conducted by intent to treat. For the 8 patients who did not complete the study, we assumed their weight to be unchanged from baseline.

Independent sample t tests were performed to compare continuous variables between the 2 groups. The groups were compared using Pearson χ² test on categorical variables, and the Fisher exact test was used where appropriate. The percentage change in weight was assessed using a 2-sided t test. The Fisher exact test was used to compare proportional weight loss and change in diabetes medication requirements between groups. The level of significance was set at P<.05 for testing the effects of the intervention.

Secondary outcomes were defined as changes in glycosylated hemoglobin level, blood pressure, or cholesterol profile. Changes in these variables were assessed using unpaired t tests, with no adjustment for multiple testing. Subgroup analyses were conducted using linear and logistic regression analyses. Adjusted odds ratios and confidence intervals were calculated using normal approximation with small sample adjustments. Commercially available software (R 2.0.0; http://www.R-project.org) was used for all analyses.

RESULTS

PATIENT CHARACTERISTICS

Two hundred forty-five patients were screened to obtain 130 participants for the study (Figure 2). Sixty-five were excluded because of failure to meet enrollment criteria; 50 declined participation for unspecified reasons. One hundred twenty-two patients were seen at the 6-month visit, for a follow up of 93.8%.

Figure 2. Flow of participants through the trial.

The 2 groups were well matched for baseline characteristics (Table 1). The mean age of participants at baseline was 56 years (age range, 31-77 years), 77 (59.2%) were women, and 55 (42.3%) were taking insulin. Dropouts did not differ from participants who completed the study with respect to any baseline variable.

ADHERENCE TO STUDY PROTOCOL

Two protocol violations were observed during the trial period. Specifically, 1 patient in each group started an unrelated commercial weight loss diet during the study period. Both patients were included in all analyses.

Seven participants in the intervention group were unable to provide logbook documentation of portion control tool use at the 6-month follow-up visit. Among the 53 patients who kept a logbook, the median compliance was 70.8%.

WEIGHT LOSS

Patients in the intervention group lost significantly more weight than control subjects (mean±SD, 1.8±3.9% vs

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Intervention Group (n = 65)</th>
<th>Control Group (n = 65)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>56.8±9.2</td>
<td>55.3±10.8</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>21 (32.3)</td>
<td>32 (49.2)</td>
</tr>
<tr>
<td>Female</td>
<td>44 (67.7)</td>
<td>33 (50.8)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>105.2±20.4</td>
<td>106.0±16.8</td>
</tr>
<tr>
<td>Body mass index</td>
<td>39.1±6.5</td>
<td>38.8±6.4</td>
</tr>
<tr>
<td>Duration of diabetes mellitus, y</td>
<td>8.3±7.2</td>
<td>7.9±8.1</td>
</tr>
<tr>
<td>Heart disease</td>
<td>12 (18.5)</td>
<td>13 (20.0)</td>
</tr>
<tr>
<td>Stroke</td>
<td>5 (7.7)</td>
<td>3 (4.6)</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>128.2±15.3</td>
<td>132.0±14.6</td>
</tr>
<tr>
<td>Diastolic</td>
<td>77.2±10.3</td>
<td>77.9±9.3</td>
</tr>
<tr>
<td>Proportion of each group taking insulin</td>
<td>29 (44.6)</td>
<td>26 (40.0)</td>
</tr>
<tr>
<td>Mean daily insulin, U</td>
<td>75.6±55.0</td>
<td>79.8±57.0</td>
</tr>
<tr>
<td>Proportion of each group using</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oral hypoglycemics</td>
<td>54 (83.1)</td>
<td>51 (78.5)</td>
</tr>
<tr>
<td>Obesigenic medication‡</td>
<td>47 (72.3)</td>
<td>44 (67.7)</td>
</tr>
<tr>
<td>Diet treatment-alone</td>
<td>6 (9.2)</td>
<td>8 (12.3)</td>
</tr>
<tr>
<td>Antihypertensive treatment</td>
<td>57 (87.7)</td>
<td>47 (72.3)</td>
</tr>
<tr>
<td>Lipid-lowering medication</td>
<td>42 (64.6)</td>
<td>37 (56.9)</td>
</tr>
<tr>
<td>Glycosylated hemoglobin level, % (No. of patients)</td>
<td>7.53±1.21 (57)</td>
<td>7.50±1.43 (56)</td>
</tr>
<tr>
<td>Lipid levels, mg/dL (No. of patients)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>185.6±45.6 (56)</td>
<td>187.9±49.5 (56)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>215.2±101.9 (56)</td>
<td>186.0±128.4 (56)</td>
</tr>
<tr>
<td>High-density lipoprotein cholesterol</td>
<td>47.5±11.2 (56)</td>
<td>45.6±9.7 (56)</td>
</tr>
<tr>
<td>Low-density lipoprotein cholesterol</td>
<td>99.0±36.7 (53)</td>
<td>101.7±35.2 (51)</td>
</tr>
</tbody>
</table>

SI conversion factors: To convert cholesterol to millimoles per liter, multiply by 0.0259; triglycerides to millimoles per liter, multiply by 0.0113.

*Data are given as mean±SD or as number (percentage). There were no significant differences between the 2 groups for any baseline variable.

†Calculated as weight in kilograms divided by height in meters squared.

‡Any medication used as treatment for diabetes mellitus (including insulin) except metformin hydrochloride.

Table 1. Baseline Patient Characteristics*
0.1%±3.0%, P =.006; absolute values, 2.1±4.9 kg vs 0.1±3.5 kg, P =.011 (Table 2). Compared with controls, a significantly greater proportion of the intervention group achieved at least 5% weight loss (11/65 [16.9%] vs 3/65 [4.6%], P =.048).

In contrast to patients taking insulin in the control group, patients taking insulin in the intervention group experienced weight loss (−2.6%±4.3% vs 0.1%±3.0%, P =.01) (Table 2). The difference in proportions of patients taking insulin achieving at least 5% weight loss showed a trend toward significance (6/26 [23.1%] in the intervention group vs 1/25 [4.0%] in the control group, P =.10).

In the subgroup of patients not taking insulin, there was no significant difference in change in weight between the intervention group and the control group (−1.3%±3.7% vs −0.2%±3.2%, P =.16) (Table 2). Similarly, there was no significant difference between the 2 groups in the percentage of patients achieving at least 5% weight loss (5/34 [14.7%] in the intervention group vs 2/37 [5.4%] in the control group, P =.25).

**GLYCEMIC CONTROL AND HYPOGLYCEMIC MEDICATION REQUIREMENTS**

Glycosylated hemoglobin levels did not differ between the groups at baseline (Table 1) or at follow-up (Table 3). The intrapatient change in glycosylated hemoglobin level was not different between the groups. The mean within-group change did not differ significantly from zero. However, a significant difference was seen in terms of change in hypoglycemic medications. Compared with controls, a significantly greater proportion of the intervention group experienced a decrease in hypoglycemic medication use (17/65 [26.2%] vs 7/65 [10.8%], P =.04).

Conversely, compared with the intervention group, a greater proportion of the control group required an increase in hypoglycemic medication use at 6 months (22/65 [33.8%] vs 9/65 [13.8%], P =.01) (Table 3). Among insulin users, compared with increased requirements in the control group, patients in the intervention group required a significant decrease in daily insulin (mean ± SD, −8.4±19.3 vs 7.2±24.7 U, P =.02).

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**Table 2. Absolute and Proportional Weight Loss**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intervention Group</th>
<th>Control Group</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage change in weight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entire group</td>
<td>−1.8 ± 3.9 (65)</td>
<td>−0.1 ± 3.0 (65)</td>
<td>.006</td>
</tr>
<tr>
<td>Subgroup taking insulin</td>
<td>−2.6 ± 4.3 (26)</td>
<td>0.1 ± 3.0 (25)</td>
<td>.01</td>
</tr>
<tr>
<td>Subgroup not taking insulin</td>
<td>−1.3 ± 3.7 (34)</td>
<td>−0.2 ± 3.2 (37)</td>
<td>.16</td>
</tr>
<tr>
<td>Absolute change in weight, kg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entire group</td>
<td>−2.1 ± 4.9 (65)</td>
<td>−0.1 ± 3.5 (65)</td>
<td>.01</td>
</tr>
<tr>
<td>Subgroup taking insulin</td>
<td>−3.2 ± 5.9 (26)</td>
<td>0.01 ± 3.40 (25)</td>
<td>.02</td>
</tr>
<tr>
<td>Subgroup not taking insulin</td>
<td>−1.2 ± 3.8 (34)</td>
<td>−0.1 ± 3.8 (37)</td>
<td>.23</td>
</tr>
<tr>
<td>Proportion of each group achieving ≥5% weight loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entire group</td>
<td>11/65 (16.9)</td>
<td>3/65 (4.6)</td>
<td>.048</td>
</tr>
<tr>
<td>Subgroup taking insulin</td>
<td>6/26 (23.1)</td>
<td>1/25 (4.0)</td>
<td>.099</td>
</tr>
<tr>
<td>Subgroup not taking insulin</td>
<td>5/34 (14.7)</td>
<td>2/37 (5.4)</td>
<td>.25</td>
</tr>
</tbody>
</table>

* Data are given as mean ± SD (number of patients) or as number/total number (percentage).
† For differences between the 2 groups.

**Table 3. Changes From Baseline in Glycemic Medication Requirements, Glycosylated Hemoglobin and Serum Lipid Levels, and Blood Pressure**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intervention Group</th>
<th>Control Group</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycosylated hemoglobin level at 6 mo, %</td>
<td>7.79 ± 1.73 (56)</td>
<td>7.54 ± 1.06 (61)</td>
<td>.34</td>
</tr>
<tr>
<td>Intrapatient change in glycosylated hemoglobin level, %</td>
<td>0.22 ± 0.86 (51)</td>
<td>−0.02 ± 1.14 (53)</td>
<td>.23</td>
</tr>
<tr>
<td>Proportion requiring change in hypoglycemic medication</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decrease</td>
<td>17/65 (26.2)</td>
<td>7/65 (10.8)</td>
<td>.04</td>
</tr>
<tr>
<td>Increase</td>
<td>9/65 (13.8)</td>
<td>22/65 (33.8)</td>
<td>.01</td>
</tr>
<tr>
<td>Change in daily insulin among insulin users, U</td>
<td>−8.4 ± 19.3 (26)</td>
<td>7.2 ± 24.7 (25)</td>
<td>.02</td>
</tr>
<tr>
<td>Change, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol level</td>
<td>−9.9 ± 16.4 (50)</td>
<td>−4.1 ± 17.7 (53)</td>
<td>.09</td>
</tr>
<tr>
<td>Triglyceride level</td>
<td>−10.5 ± 27.8 (49)</td>
<td>3.9 ± 48.0 (53)</td>
<td>.07</td>
</tr>
<tr>
<td>High-density lipoprotein cholesterol level</td>
<td>2.0 ± 10.1 (50)</td>
<td>1.2 ± 14.3 (53)</td>
<td>.74</td>
</tr>
<tr>
<td>Low-density lipoprotein cholesterol level</td>
<td>−13.1 ± 24.2 (47)</td>
<td>−5.5 ± 32.2 (47)</td>
<td>.20</td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>5.5 ± 15.4 (59)</td>
<td>−4.2 ± 11.0 (60)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Diastolic</td>
<td>1.4 ± 15.6 (59)</td>
<td>−2.6 ± 9.7 (60)</td>
<td>.10</td>
</tr>
</tbody>
</table>

* Data are given as mean ± SD (number of patients) or as number/total number (percentage).
† For differences between the 2 groups.
SERUM LIPOPROTEINS

Compared with control subjects, patients in the inter-
vvention group had a significantly greater decrease in non-
high-density lipoprotein cholesterol levels (13.6%±20.2% vs 4.8%±23.4%, P = .04). There was no difference be-
tween groups in terms of proportions of each group that experienced an increase in lipid-lowering medication use. No other differences were seen in the cholesterol pro-
files between groups.

BLOOD PRESSURE

Change in systolic blood pressure was significantly higher in the intervention group at 6 months compared with that in the control group (5.5%±15.4% vs –4.2%±11.0%, P<.001) (Table 3). No significant difference was seen between groups with respect to change in diastolic blood pressure. No significant difference was seen between groups in terms of changes in antihypertensive medica-
tion use.

SUBGROUP ANALYSES

Consistent results, as defined by achievement of at least 5% weight loss, were observed regardless of age, sex, education level, baseline body mass index, obesigenic hy-
poglycemic medication use, or insulin use at baseline (Figure 3). Likewise, no variables were found to be pre-
dictive of compliance with the intervention.

ADVERSE EVENTS

One hypoglycemic reaction was reported in the inter-
vention group. It necessitated a visit to the emergency department but had no serious sequelae.

COMMENT

In this first randomized trial (to our knowledge) evalu-
ating the use of a portion control tool as a treatment for obesity, we demonstrated that this simple inexpensive inter-
vention is able to induce weight loss in an obese popu-
lation with type 2 DM. The proportion of patients who achieved at least 5% weight loss in the intervention group was significantly higher than that in the control group receiving usual care. This is important, as a 5% weight loss has been shown to be clinically significant in terms of decreasing morbidity and mortality associated with obesity-linked disorders such as cancer and myocardial infarction.17-19 We demonstrated that this portion control system is effective in decreasing hypoglycemic medica-
tion requirements and in preventing much of the pro-
gressive increase in medication requirements typical of patients with type 2 DM. The decrease in hypoglycemic medication use was made without sacrificing glycemic control.

These results are comparable to results of several stud-
ies using pharmaceutical intervention for weight loss in type 2 DM. In a recent 6-month trial, patients with type 2 DM were randomized to receive sibutramine hydro-
chloride vs placebo.20 The study excluded patients tak-
ing insulin, thus selecting a subgroup in whom it may be easier to achieve weight loss. Although the study found a significantly greater weight loss compared with that in control subjects (4.5 vs 1.7 kg, P<.001), a significant dif-
ference was not seen between groups in the proportions of patients losing at least 5% of body weight. Further-
more, all patients in the sibutramine trial received di-
etary counseling geared toward moderate caloric restric-
tion, which was reinforced at monthly clinic visits. Such counseling was not reinforced during our study, which emphasizes the broader generalizability of our results to routine clinical care. From an economic perspective, the intervention in our study is a 1-time investment that costs approximately one sixth of the cost of a 6-month pre-
scription of sibutramine in Canada.

Similar comparisons can be made with trials of orli-
stat in type 2 DM. In a 1-year randomized trial that ex-
cluded patients taking insulin and placed all subjects on a calculated hypocaloric diet, 39% of patients taking or-
listat lost at least 5% of baseline weight compared with 15.7% receiving placebo (odds ratio, 2.48),21 compared with 11 (16.9%) of 65 patients in our intervention group and 3 (4.6%) of 65 in our control group (odds ratio, 3.67). Although the study was a 1-year trial, the change in weight was stable from 6 months to 1 year. Similar changes in hypoglycemic medication use were seen as were ob-
served in our study. The orlistat trial had a high attri-
tion rate of 40%, which is typical of studies of pharma-
cotherapy for weight loss. The 1-time cost of our portion control intervention is one fourth of the cost of a 6-month prescription of orlistat.
Although we observed no difference between groups in terms of glycosylated hemoglobin levels, this may be because the patients’ primary physicians were free to decrease the number or dosage of hypoglycemic medications in response to lower glycosylated hemoglobin levels. The observed change in hypoglycemic medications may be of great clinical importance by decreasing cost and potential for serious adverse effects. It is plausible that a component of the decrease in hypoglycemic medication use seen in this study was by virtue of decreasing carbohydrate intake alone. The cause of increased blood pressure seen in the intervention group is unclear; this outcome may have been a random play of chance.

It is interesting that the significant weight loss using the intervention in this study was primarily seen in patients taking insulin, based on post hoc analyses, as it is notoriously difficult to achieve weight loss in patients using this anabolic hormone. Further prospective studies are required to explore the success of this tool in combination with various types of hypoglycemic agents.

The successful weight loss seen using this intervention in patients with type 2 DM lends optimism toward its potential success in populations without DM, as investigations of weight loss interventions are typically less successful among obese patients with DM compared with those with obesity without DM.22,23 This portion control intervention was designed to target an overweight adult population and is not specific for use in patients with DM. It should be studied in other obese populations.

This study is subject to some limitations. Overall compliance with the intervention was poor, thereby underestimating the potential success. Having a support system in the form of group meetings or more frequent patient contact may have improved compliance with the portion control tools. Strategies of obesity treatment are more successful when support strategies are in place for patient assistance and for ongoing encouragement.24,25 Also, patients who eat frequent meals outside of the home were excluded from the study, as the use of this intervention would be difficult in restaurants. Proving that this tool induces weight loss among patients who take most of their meals at home may serve as motivation for future patients using this tool to take their meals at home as well, in an environment that is more amenable to control of caloric intake.

As a trial of a meal control tool, this study could not be conducted in double-blind fashion. However, the outcomes in question were objective and were not prone to interpretation bias. This may be a strength of the study in that it describes positive results in a real-life application of this intervention. Finally, data were not collected regarding quantitative dietary composition changes or caloric expenditure; the exact mechanism of the intervention may be more complex than portion control alone.

In conclusion, the portion control tool studied in this trial was effective in inducing weight loss in obese persons with type 2 DM comparable to that seen in investigations of weight loss pharmacotherapy. This simple inexpensive tool also enabled obese patients with DM to decrease their hypoglycemic medication requirements.

This intervention holds promise for use in overweight populations with and without DM.

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Author Contributions: Dr Pedersen had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Pedersen and Kline. Acquisition of data: Pedersen. Analysis and interpretation of data: Pedersen, Kang, and Kline. Drafting of the manuscript: Pedersen. Critical revision of the manuscript for important intellectual content: Pedersen, Kang, and Kline. Statistical analysis: Kang. Obtained funding: Pedersen and Kline. Administrative, technical, and material support: Pedersen. Study supervision: Kline.

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REFERENCES


