

1 **TITLE**

2 **Evaluating the Power of Food Scale in obese subjects and a general sample of individuals:**  
3 **development and measurement properties**

4

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19 **Running head: POWER OF FOOD SCALE**

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23

24 **ABSTRACT**

25 **BACKGROUND:** The Power of Food Scale<sup>®</sup> (PFS) was constructed to assess the psychological  
26 impact of today's food-abundant environments.

27 **OBJECTIVE:** To evaluate the structure of the PFS in diverse populations of obese and  
28 nonobese individuals.

29 **DESIGN:** Data were obtained from obese adults in a clinical trial for a weight-management drug  
30 ( $n = 1741$ ) and overweight, obese, and normal weight adults in a web-based survey ( $n = 1275$ ).

31 Exploratory and confirmatory factor analyses were employed to investigate the PFS structure  
32 using the clinical data. The model developed was then tested using the web-based data.

33 Relationships between PFS domains and body mass index (BMI) were examined. Logistic  
34 regression was used in the web-based survey to evaluate the association between obesity status  
35 and PFS scores.

36 **RESULTS:** Clinical data indicated that the scale was best represented by a 15-item version with  
37 three subscale domains and an aggregate domain (average of three domains); this was confirmed  
38 with data from the web-based survey (Comparative Fit Index: 0.95 and 0.94 for the clinical and  
39 web-based studies, respectively). Cronbach's alpha for both data sets were high, ranging from  
40 0.81–0.91. The relationships between BMI and each domain were weak (and approximately  
41 linear). A full category increase in PFS domain score (range 1–5) increased the odds of being  
42 obese 1.6–2.3 times.

43 **CONCLUSIONS:** The 15-item PFS is best represented by three domains and an aggregate  
44 domain. The PFS may provide a useful tool to evaluate the effects of obesity treatments on  
45 feelings of being controlled by food in an obesogenic food environment.

46 **KEY WORDS:** Power of Food Scale, obese subjects, weight management

## 47 INTRODUCTION

48 Obesity is a risk factor for a number of diseases such as diabetes, heart disease, fatty  
49 liver, sleep apnea, and some cancers, and is associated with substantial direct and indirect health  
50 costs (1-4). Twenty years ago 15% of the adult population in the United States were obese; today  
51 more than 30% are obese and a further 33% are overweight (5-8). This dramatic increase has  
52 driven a need to understand better the regulation of food intake (9, 10).

53 Eating behavior in humans is an extremely complex process modulated by internal  
54 homeostatic processes (11-13) and environmental and social influences (14, 15). As such, energy  
55 homeostasis aims to match energy intake with overall energy needs; however, total food intake is  
56 also influenced by external factors, such as food availability and palatability, which might lead to  
57 excess energy intake during any given meal (16). This tendency to eat beyond immediate  
58 homeostatic needs has presumably evolved because the motivation to seek and consume calories  
59 contributed to survival when food sources were scarce and unpredictable (17). Eating behavior  
60 can be further divided into two linked yet distinct, differentially regulated processes (18, 19): 1)  
61 appetitive responses that precede food intake and 2) consummatory behaviors that determine  
62 what and how much is eaten.

63 Chronic excess energy intake, either via increased meal size or meal number, will  
64 ultimately lead to weight gain and obesity. The reasons why only certain individuals chronically  
65 overeat are unclear (6, 20), but may reflect differences in appetitive and consummatory  
66 responses to food, an enhanced sensitivity to a food-rich environment, and an overpowering of  
67 homeostatic processes by food reward in these individuals (16, 21-23). The motivation to  
68 consume foods beyond homeostatic need has been referred to as “hedonic hunger” (22).

69           There are a number of measures available to study overeating in response to social,  
70 environmental, emotional, and food stimuli, such as the Three-Factor Eating Questionnaire  
71 (TFEQ), Dutch-Eating Behaviour Questionnaire (DEBQ), and the Restraint Scale (24-27), but  
72 none that tap into the appetitive (rather than the consummatory) aspects of eating. To this end,  
73 the Power of Food Scale<sup>®</sup> (PFS) was developed to assess the psychological impact of living in  
74 food-abundant environments, as reflected in feelings of being controlled by food, independent of  
75 food consumption itself (22, 28). PFS items were designed to reflect responsiveness to the food  
76 environment involving three levels of food proximity: 1) food readily available in the  
77 environment but not physically present, 2) food present but not tasted, and 3) food when first  
78 tasted but not consumed.

79           This study examines the factor structure of the PFS using data obtained in a large clinical  
80 population of obese patients recruited into a clinical trial for weight management and from a  
81 diverse group of subjects (including nonobese, overweight, obese subjects) recruited from the  
82 general population.

83

## 84 **METHODS**

### 85 **Development of the PFS**

86           An initial pool of items for the PFS was collected from a group of obese women  
87 participating in a lifestyle-based obesity treatment study ( $n \approx 50$ ). Prior to treatment, participants  
88 were asked to think of ways in which appetizing food affected their thoughts, feelings, or  
89 behavior in situations where eating was not expected or imminent (i.e., not while preparing or  
90 eating a meal or snack). They were also asked to consider the effects of food and the food  
91 environment up to and including the point where they first tasted a food.

92           Any items describing actual food consumption or overeating were eliminated from  
93 consideration to avoid confounding the measure with a behavior it might be used to predict.  
94 Items describing the obesogenic food environment itself, rather than reactions to the  
95 environment, were also excluded, because the PFS presumes that respondents live in an  
96 environment where a variety of palatable foods are readily accessible. Further, items describing  
97 emotional eating were excluded for several reasons: measures of emotional eating already exist  
98 (e.g., DEBQ, Emotional Eating Subscale, revised TFEQ) (24-27), such items suggest a  
99 susceptibility to overeating, and the presumed source of such consumption is negative affect  
100 rather than something about the food environment per se. Items that were redundant or that  
101 described idiosyncratic scenarios that would likely apply to a small percentage of respondents  
102 were also eliminated.

103           In examining the items generated by group members, it became apparent that they could  
104 be categorized according to the proximity of food to the respondent. Because in most developed  
105 countries food is almost always readily available (e.g., in the refrigerator or at a corner store),  
106 people implicitly know that food is constantly attainable. Thus the first level of food availability

107 is abstract, reflecting the impact of the mere accessibility of food (sample item: “I find myself  
108 thinking about food even when I’m not physically hungry”). At a second and more proximal  
109 level, food may not only be available but actually present in a given context. In this case, the  
110 presence of visual and olfactory food cues could potentially influence people differently than if  
111 food is available in the environment but not physically present (sample item: “If I see or smell a  
112 food I like, I get a powerful urge to have some”). The third and most proximal context is when  
113 food is not only present but is, or is about to be, tasted (sample item: “When I eat delicious food I  
114 focus a lot on how good it tastes”).

115 Additional items reflecting the influence of food in these three contexts were developed  
116 so that there were at least six items describing the potential impact of food in each of these three  
117 contexts. In the final version of the PFS evaluated here, items pertaining to the three different  
118 contexts were randomly ordered throughout the 21-item scale. The factor analysis we conducted  
119 on the PFS would determine whether these dimensions emerged as separate factors, but the  
120 purpose of including items reflecting these dimensions was to assess comprehensively the  
121 various ways in which food-abundant environments might influence eating-related beliefs,  
122 thoughts, feelings, and motivations. In order to determine the content validity of the PFS, 14  
123 experts were given a description of the PFS as well as the 21 items and asked to rate each item  
124 with respect to how well it reflected the construct that it aimed to measure. On a 5-point scale  
125 (from 1 = *not at all* to 5 = *very much*), all items received mean scores near or above the mid-  
126 point of the scale, with the lowest item receiving a score of 2.7 (i.e., close to *a fair amount*).

127 It is important to note two assumptions underlying the development and use of the PFS.  
128 The first assumption is that the environmental context in which it is used features relatively  
129 abundant, accessible, palatable, and affordable foods; it is not intended for use in a food-

130 impoverished environment where, for example, the item “I often think about what foods I might  
131 eat later in the day” may be a product not of hedonic anticipation but of hunger and deprivation.  
132 This presumption should help ensure that PFS scores represent psychological temptation rather  
133 than physiological need. The second assumption is that the measure is designed to tap an  
134 individual’s subjective experiences to living in a food-abundant environment and the power of  
135 food over that individual; it is not designed to assess features of the food environment itself. It is  
136 appropriate to use the PFS to compare individuals living in the same or similar food-abundant  
137 environments but not individuals from environments in which the availability of palatable foods  
138 differs significantly.

139

#### 140 **PFS questionnaire**

141 This initially proposed PFS is a 21-item questionnaire presented on a 5-point Likert scale  
142 ranging from 1 (don’t agree at all) to 5 (strongly agree). All items are scored so that a higher item  
143 score indicated a greater responsiveness to the food environment.

144

#### 145 **Study populations**

146 Two distinct study populations were used. The factor structure of the PFS was initially  
147 examined using baseline data, before treatment intervention, from a phase 3 clinical trial of a  
148 candidate weight management compound (which was previously but is no longer in  
149 development).. The dataset included 1741 obese, nondiabetic individuals (obesity defined as  
150 body mass index [BMI]  $>30 \text{ mg/kg}^2$  for subjects without co-morbidities; BMI  $>27 \text{ kg/m}^2$  for  
151 subjects with co-morbidities [treated or untreated hypertension or dyslipidemia]) from the United

152 States and Canada. Questionnaires were administered in the clinic on day one. Data obtained  
153 from these subjects are referred to as “clinical study” data.

154 The structure of the PFS was then further evaluated using data obtained from subjects  
155 recruited from the US arm of the 2006 National Health and Wellness Survey (NHWS). The  
156 dataset included 1275 subjects in total and recruited subjects from the following groups:  
157 nonobese healthy subjects (BMI 18.5–26.9 kg/m<sup>2</sup>, with no diabetes or dyslipidemia), overweight  
158 and obese subjects (BMI ≥ 27.0 kg/m<sup>2</sup>) with diabetes, and overweight and obese subjects (BMI ≥  
159 27.0 kg/m<sup>2</sup>) without diabetes. The NHWS is an annual study of the healthcare attitudes and  
160 behaviors of nationally representative samples of the adult population. In 2006, the NHWS was  
161 fielded to 60 000+ members of the general panel of Lightspeed Research.

162 Through panel identification numbers, respondents were identified and re-contacted for  
163 this study. Any respondent with a BMI ≥ 18.5 kg/m<sup>2</sup> was eligible to be re-contacted to participate  
164 in this study and they were invited to do so randomly. As respondents entered the questionnaire,  
165 they were re-screened for eligibility and categorized into three quota groups: 500 obese subjects  
166 with diabetes, 500 obese subjects without diabetes, and 250 nonobese subjects. Within both the  
167 obese with diabetes and obese without diabetes groups, there were minimum quotas based on  
168 BMI: at least 100 respondents must have a BMI 27–29 kg/m<sup>2</sup>; at least 100 respondents must have  
169 a BMI 30–34 kg/m<sup>2</sup>; at least 100 respondents must have a BMI greater than or equal to 35–39  
170 kg/m<sup>2</sup>; and at least 100 respondents must have a BMI ≥ 40 kg/m<sup>2</sup> (values less than 0.5 were  
171 rounded to the next lower integer and 0.5 or greater to the next higher integer).

172 Once quota groups were filled, only respondents to NHWS who met the criteria for the  
173 open quota groups received subsequent invitations to participate in the study. Members of the  
174 Lightspeed Research panel were recruited through opt-in email, co-registration with Lightspeed

175 Research partners, e-newsletter campaigns, banner placements, and both internal and external  
176 affiliate networks. The questionnaire was self-administered via the internet. The questionnaire  
177 takes approximately 20 minutes to complete. Respondents needed to have internet access to  
178 participate, but that access may or may not have been in their homes. Information about point of  
179 internet access was not collected. The formatting of the screen presentation was as close as  
180 possible to the questionnaire provided in the clinical study. All data were self-reported by the  
181 survey respondents and were not verified against any clinical diagnostics. Data obtained from  
182 these subjects are referred to as “web-based survey” data.

183

#### 184 **Descriptive analyses**

185 Mean, standard deviation, and percentages of floor and ceiling effects were performed at  
186 the item-level.

187

#### 188 **Psychometric analysis**

189 Baseline responses to the PFS questionnaire from subjects in the clinical trial were used  
190 to examine the structure of the PFS and eliminate any unnecessary items. The model developed  
191 was then tested using data from subjects recruited via the web-based survey.

192 Within the clinical trial sample, the number of factors was explored by using a parallel  
193 analysis (29-35). Parallel analysis allows the identification of factors that are beyond chance. In  
194 addition to this factor analysis of real data, a series of simulations were performed involving the  
195 analysis of a matrix of random numbers that represented the same number of cases and variables.  
196 We then used an iterative process of extracting factors from candidate models using exploratory  
197 factor analysis and then tested each model using confirmatory factor analysis. In exploratory

198 factor analysis calculations, for an item to be considered as a part of a particular factor, the  
199 standardized regression coefficient must have a relatively high standardized pattern loadings  
200 ( $\geq 0.40$ ) on this factor and low loadings ( $< 0.40$ ) on the other factors (30).

201 For the confirmatory factor analysis, Bentler's Comparative Fit Index (CFI) was the main  
202 indicator of fit and was obtained for each candidate model; CFI values of  $> 0.9$  indicate  
203 acceptable fit (29). We used two additional indexes to compare models: the Parsimonious  
204 Normed Fit Index (PNFI) (32) and the Expected Cross Validation Index (ECVI) (36). The PNFI  
205 simultaneously reflects both the fit and the parsimony of the model; the model with the largest  
206 PNFI is most parsimonious one. The ECVI gauges the applicability or generalizability of results;  
207 the model with the smallest ECVI value is considered to be the most stable in the population.

208 Confirmatory factor analyses were then undertaken on data obtained from subjects  
209 recruited to the web-based survey to confirm the structure of the PFS model developed using  
210 data from the clinical study. We conducted additional analyses and validity tests to examine the  
211 stability of the measurement model for obese subjects (BMI of  $30+ \text{ kg/m}^2$ ) and nonobese  
212 subjects (BMI of  $< 30 \text{ kg/m}^2$ ), as well as for gender, in the web-based sample. In doing so we  
213 compared and tested two multi-group models: the "no-constraint" model, which does not impose  
214 any constraint on its parameters in the two subgroups of interest (e.g., obese vs. nonobese), and  
215 the "invariant" measurement model, which constrains the corresponding factor loadings in the  
216 two subgroups to be equal. Evidence for the "invariant" measurement model exists if two  
217 conditions are met: the multi-group models exhibits adequate fit [say, Comparative Fit Index  
218 (CFI) of  $0.90+$ ] and that the difference between these two models is negligible ( $0.01$  or less) (37,  
219 38).

220 In the web-based survey, as well as in the clinical study, Cronbach's  $\alpha$  reliability  
221 coefficient (39) was computed for each domain to measure internal consistency. The relationship  
222 of the PFS with BMI was determined using regression (no functional form was imposed). Here  
223 BMI was treated as a continuous outcome (response or dependent) variable and PFS domain as a  
224 discrete predictor (with mean score per item rounded to the nearest discrete category, being one  
225 of the integers from 1 to 5). Moreover, logistic regression was used in the web-based survey to  
226 predict being overweight (yes:  $\text{BMI} \geq 27 \text{ kg/m}^2$ , no:  $\text{BMI} < 27 \text{ kg/m}^2$ ) from the PFS scores, with  
227 age and gender included as covariates.

228 Using both studies, we used Pearson correlation coefficients to examine the association  
229 between PFS and the Three-Eating Eating Questionnaire with 21 items (TFEQ) (26).

230

231

## 232 RESULTS

### 233 Participants

234 There were 1741 patients recruited into the clinical trial. Mean ( $\pm$ SD) BMI was  $38.6 \pm$   
235  $6.7 \text{ kg/m}^2$ ; mean ( $\pm$ SD) age was  $46.3 \pm 11.0 \text{ y}$ ; 1427 (82%) of the patients were female. Of the  
236 1275 subjects recruited to complete the web-based survey (mean [ $\pm$ SD] BMI:  $33.1 \pm 7.6 \text{ kg/m}^2$ ;  
237 mean [ $\pm$ SD] age  $52.5 \pm 12.8 \text{ y}$ ; 39% female), 250 subjects had a BMI of  $18.5\text{--}26.9 \text{ kg/m}^2$  (56%  
238 female), 518 subjects had a BMI of  $27.0\text{--}75.9 \text{ kg/m}^2$  and no diabetes (46% female), 503 subjects  
239 had a BMI of  $27.1\text{--}67.5 \text{ kg/m}^2$  and had a diagnosis of diabetes (25% female), and in four  
240 subjects diabetic status was unavailable.

241 **Table 1** provides descriptive statistics at baseline for the 21 individual PFS items.

242

### 243 Assessment of PFS in the clinical study

#### 244 *Measurement Model Development*

245 Using parallel analysis, we determined that there may be between one and eight factors in  
246 the model. CFI's for 1- and 2-factor models were  $<0.90$ , indicating that these models did not  
247 adequately fit the data. CFI for the 3-, 4-, and 5-factor solutions were  $>0.90$ ; therefore these  
248 models fit the data (**Table 2**). Extracting a solution beyond five factors did not produce a new  
249 structure compared with the 5-factor model. From the 3-, 4-, and 5-factor models, the 3-factor  
250 model (with three weak items removed resulting in an 18-item model) was selected to represent  
251 the PFS measurement model as the most parsimonious solution (based on PNFI).

252

#### 253 *Final Measurement Model*

254 The 3-factor model gave the largest ECVI (**Table 2**). This indicates that, although this  
255 model was the most parsimonious one, it could be less generalizable than the 4- and 5-factor  
256 models. Additional calculations were performed to refine the 3-factor model and achieve better  
257 generalizability and fit of the model. Based on obtaining a CFI and ECVI better than those  
258 obtained for the existing the 3-, 4-, and 5-factor models, a 3-factor model containing 15 items  
259 emerged (see Appendix; permission to use the Power of Food Scale® can be obtained from Dr.  
260 Michael R. Lowe at [lowe@drexel.edu](mailto:lowe@drexel.edu)). Items deleted from the original 21-item PFS are detailed  
261 in (**Table 1**).

262 For the 15-item questionnaire, the relatively high correlations among factors (factor 1 and  
263 factor 2: 0.73; factor 1 and factor 3: 0.72; factor 2 and factor 3: 0.69) suggested that a 3-factor,  
264 second-order model is a well-suited and appropriate model. All of its standardized path  
265 coefficients, including those from the aggregated factor, were statistically significant (all  $t$  values  
266  $>1.96$ ) and exceeded 0.4 (**Figure 1**). We also tested a 1-factor first-order solution (all 15  
267 indicator variables represent one factor) and found that it does not fit the data (CFI = 0.89).

268

### 269 **Confirmation of the 3-factor, second-order model**

270 Using data from all subjects in the web-based study, CFI for the 15-item 3-factor, second-  
271 order solution was 0.94. This confirms that, within a different and more diverse population, a 3-  
272 factor, second-order model of the 15-item PFS continues to fit the data. All standardized path  
273 coefficients, including those from the aggregated factor, were statistically significant (all  $t$  values  
274  $>1.96$ ) and exceeded 0.4 (**Figure 1**). We also tested the 1-factor, first-order model using data  
275 from the web-based survey; the CFI for the 1-factor model was 0.88, indicating that this model  
276 does not adequately fit the data.

277

**278 Characteristics of the 3-factor, second-order model**

279           The 3-factor, second-order structure identified with the clinical population was confirmed  
280 in the relatively more diverse population enrolled in the web-based survey. Each of the three  
281 domain scores was calculated as the mean score per item across items that constitute a given  
282 domain. The aggregate domain score was calculated as the mean of three single domain scores.  
283 Items on the first, second, and third domains relate to “food available”, “food present”, and “food  
284 tasted”, respectively. All factor loadings were clearly statistically significant ( $t > 1.96$ ) and all  
285 standardized factor loadings were large (**Figure 1**).

286           Cronbach’s  $\alpha$  was good for the clinical data (0.87 for factor 1, 0.87 for factor 2, 0.81 for  
287 factor 3, and 0.88 for the aggregate score) and the web-based data (0.91 for factor 1, 0.90 for  
288 factor 2, 0.82 for factor 3, and 0.90 for the aggregate score). In both data sets corrected item-to-  
289 total correlations were in the range 0.58 to 0.84 for factor 1, 0.68 to 0.80 for factor 2, 0.49 to 0.67  
290 for factor 3, and 0.76 to 0.82 for the aggregate score. No evidence of floor or ceiling effects  
291 existed in the clinical sample. However, within the web-based survey there was some evidence  
292 of a floor effect, as half of the items percentages at the low end were responded to by more than  
293 50% of the sample. Studying floor and ceiling effects by obesity and diabetes status showed that  
294 the same items continue to have percentages at the low end that exceeded 50%.

295           In both studies the correlation coefficients between each item and BMI, as well as  
296 between every domain and BMI, were from negligible to small (ranging from 0.003 to 0.24 for  
297 items and from 0.02 to 0.24 for domains). A regression model using a domain score as a  
298 predictor (adjusted for age and gender) was also applied. In the clinical study, a 1-category  
299 increase in factor 1 score was associated with a 0.37 kg/m<sup>2</sup> increase in BMI (95% CI: -0.15,

300 0.89;  $P = 0.17$ ), a 1-category increase in factor 2 score was associated with a  $0.17 \text{ kg/m}^2$  increase  
301 in BMI (95% CI:  $-0.22, 0.57$ ;  $P = 0.39$ ), and a 1-category increase in factor 3 score was  
302 associated with a  $0.22 \text{ kg/m}^2$  increase in BMI (95% CI:  $-0.39, 0.84$ ;  $P = 0.48$ ).

303         Despite the fact that these associations were not statistically significant in the clinical  
304 study, there were some indication of a linear relationship between domain score and BMI in the  
305 web-based survey (**Figure 2**). In the web-based survey, for factor 1 a 1-category difference was  
306 associated with a change in BMI of  $1.81 \text{ kg/m}^2$  (95% CI:  $0.77, 2.85$ ;  $P = 0.0006$ ), for factor 2 a  
307 1-category difference was associated with a change in BMI of  $1.25 \text{ kg/m}^2$  (95% CI:  $0.63, 1.86$ ;  $P$   
308  $< 0.0001$ ), for factor 3 a 1-category difference was associated with a change in BMI of  $0.72$   
309  $\text{kg/m}^2$  (95% CI:  $-1.11, 2.54$ ;  $P = 0.44$ ), and for the aggregated domain a 1-category difference in  
310 score was associated with a change in BMI of  $1.47 \text{ kg/m}^2$  (95% CI:  $-0.61, 3.55$ ;  $P = 0.17$ ).

311 Although the correlation results on PFS and BMI showed at best a modest relationship, where  
312 variability (noise) dominated, the relationships between each factor and BMI were visibly close  
313 to linear (**Figure 2**). This general relationship between PFS domains and BMI was also  
314 supported by comparison of the mean domain scores and mean BMI in studied populations.  
315 Mean scores for each PFS domain were higher in obese subjects in the clinical trial, who had the  
316 greatest mean BMI, and lowest in nonobese participants in the web-based survey, who had the  
317 lowest BMI (**Table 3**).

318         Within the web-based survey, logistic regression in overweight versus non-overweight  
319 participants (adjusted for age and gender) indicated that an increase in a domain score by one  
320 category increased the chance to be overweight by 2.27 times for factor 1 (95% CI:  $1.80, 2.87$ ),  
321 by 1.56 times for factor 2 (95% CI:  $1.32, 1.84$ ), by 1.57 times for factor 3 (95% CI:  $1.28, 1.93$ ),

322 and by 1.97 times for the aggregate score (95% CI: 1.58, 2.45). These results were statistically  
323 significant (the 95% CI did not include the null value of 1).

324         Across both studies the correlations of the PFS subscales and aggregate (overall) score  
325 with the uncontrolled eating and emotional eating on the TFEQ did not exceed 0.70 and hence  
326 were not unduly high (with uncontrolled eating: average = 0.64, range = 0.54 to 0.70; with  
327 emotional eating: average = 0.51, range = 0.40 to 0.63), suggesting that these PFS and TFEQ  
328 subscales were measuring similar but distinct aspect of eating. In addition, across both studies,  
329 the correlations of the PFS subscales and the PFS aggregate score with cognitive restraint on the  
330 TFEQ was small or modest (average: -0.16; range: -0.27 to -0.05), suggesting that these PFS and  
331 TFEQ subscales were measuring different aspects of eating.

332         In the web-based survey, for the obese and non-obese subgroups, the no-constraint model  
333 gave a CFI of 0.9324 and the invariance model gave a CFI of 0.9264 (each of these two models  
334 combined the obese and non-obese subgroups into one model with one CFI). Because these CFI  
335 values exceeded 0.90 and their difference was negligible (0.006), the invariance and stability of  
336 the PFS measurement model relative to obesity status was supported by the data. For gender  
337 subgroups, the no-constraint model gave a CFI of 0.9342 and the invariance model gave a CFI of  
338 0.9323. Because these CFI values exceeded 0.90 and their difference was negligible (0.0019), the  
339 invariance and stability of the PFS measurement model relative to gender was supported by the  
340 web-based data. Data from the clinical sample, which had sufficient numbers of males and  
341 females for meaningful analysis, concurred: the no-constraint model gave a CFI of 0.9449 and  
342 the invariance model gave a CFI of 0.9442, a difference of 0.0007.

343

344

345 **DISCUSSION**

346           We determined that the initially proposed candidate set of 21 items of the PFS could be  
347 reduced to 15, and that the scale was best represented by a 3-factor, second-order model with  
348 three domains and an aggregate domain. For each domain, its score was the mean score per item  
349 in a given domain. Scores for the aggregate domain were calculated as the mean of those 3  
350 domain scores.

351           The three subscale domains and aggregate domain engendered high internal consistency.  
352 Subscale scores from the three domains can be used to provide further description of the  
353 psychological impact of the food environment in each of the three levels of proximity to food:  
354 food readily available in the environment but not physically present, food present but not tasted,  
355 and food when first tasted but not consumed. Corrected-to-total item correlations were, in  
356 general, reasonably strong, supporting the conclusion that items on the same factor represented a  
357 common concept or construct and the adoption of the aggregate score, as a proxy for the  
358 common factor.

359           In the clinical study, there was a weak and not statistically significant relationship  
360 between PFS and BMI. In the web-based survey, the correlation between BMI and PFS  
361 aggregate and domain scores was modest. The PFS may be more strongly related to overeating  
362 tendencies among overweight and obese individuals than to BMI per se. If the PFS measures an  
363 appetitive predisposition for over-consumption of energy, then one would expect a substantial  
364 PFS/BMI relationship; however, a number of factors may mitigate against such a relationship,  
365 such as the energy efficient individuals who gain weight despite minimal overeating and energy  
366 inefficient individuals who do not gain weight despite frequent overeating (40). In addition, the  
367 degree of “pull” obesity-prone individuals feel from the obesogenic environment may be greater

368 when they are gaining weight than when their weight stabilizes at an obese level (41). Studies  
369 have shown that extreme obesity may be associated with a down regulation of dopamine  
370 receptors and a reduction in the rewarding value of food (42). This attenuated PFS/BMI  
371 association suggests the potential usefulness of the PFS to identify those individuals who might  
372 benefit most from particular interventions.

373         The set of correlations between PFS and TFEQ indicated that PFS is measuring aspects  
374 of eating that are distinct or different from TFEQ. The DBEQ was not included as a measure in  
375 the two studies. While we expect that it too would be distinct or different from the PFS, we  
376 encourage further research to examine this. Moreover, the data support the invariance and  
377 stability of the PFS measurement model relative to gender and to obesity status.

378         We also found differences between the clinical and web-based data with regard to floor  
379 effects that were observed only in the web-based survey. Patterns of items with floor effects were  
380 the same among web-based subgroups. The differences between the two studies may reflect the  
381 latter study having a more even distribution and greater range of BMI, and differences between  
382 clinical and nonclinical populations.

383         Eating behavior in humans is a physiological requirement to maintain energy  
384 homeostasis. However, in developed countries, where there is often an abundance of food, it may  
385 be appropriate to differentiate between homeostatic and hedonic hunger (22). Existing measures  
386 assess actual consumption of food associated with various stimuli such as negative mood, social  
387 cues, and hunger (24-27), but do not permit a differentiation of appetitive drive in relation to the  
388 consumption of food. Given the wide diversity of stimuli that have been associated with food  
389 intake, if the PFS can accurately assess individuals' hedonic appetitive drive in food-abundant  
390 environments, it may represent a brief and cost-effective tool in the study of human eating

391 behavior and may potentially be predictive of weight gain or response to weight management  
392 treatment in certain individuals.

393         The PFS could also help in the development of appropriate and effective  
394 pharmacotherapy for obesity. Current anorectic agents affect food intake in different ways – for  
395 example, in obese subjects, the serotonin agent d-fenfluramine reduced perceived hunger but not  
396 the pleasantness of food (43). In contrast, opioid antagonists reduced the pleasantness of food  
397 without affecting hunger ratings (44). In animals, the cannabinoid antagonist rimonabant reduced  
398 the motivational drive for food without influencing palatability (45). If certain obese patients are  
399 especially susceptible to the food environment, an ideal weight management program would  
400 reduce this influence without affecting the pleasure associated with eating. For example an  
401 intervention might reduce the sensitivity of an individual to the food environment, or modify the  
402 nature of foods immediately available to participants (46).

403         The reliability and validity of the 21-item PFS has been explored in two other studies. In  
404 unpublished work, for example, Lowe and colleagues found that the PFS aggregate domain had  
405 high test-retest reliability and was related to several measures of overeating, even after  
406 controlling for scores on the Restraint Scale, a robust predictor of overeating tendencies (47).  
407 Forman et al (28) found that in subjects that kept chocolate with them at all times, but were  
408 instructed not to eat any, PFS aggregate scores were predictive of 1) the strength of chocolate  
409 cravings over 48 h, 2) which participants ate the chocolate, and 3) which coping treatment  
410 worked best with particular individuals. This supports the validity of the PFS in identifying  
411 subjects more sensitive to the constant availability of food and its usefulness in predicting  
412 outcome of an intervention for controlling food cravings. Additional studies to assess further the  
413 psychometric properties of the PFS are required.

414           Our investigation has at least two notable limitations. First, information collected in the  
415 web-based survey relating to BMI and co-morbid conditions were self-reported and were not  
416 verified by physicians or clinical records. Second, there were methodological differences in the  
417 administration of the PFS between the clinical and web-based studies, with the use of pen-and-  
418 paper in the former and electronic screen-based means in the latter; study differences also existed  
419 in the format of the questionnaire itself.

420           In summary, the measurement model of the PFS was evaluated in a large, clinically  
421 relevant population of obese patients in a clinical trial for weight loss management and in a data  
422 set that included a more diverse population of normal weight and overweight and obese subjects.  
423 These data indicate that the 15-item PFS, with three domain scores and an aggregate total score,  
424 may provide a useful tool to evaluate the effects of weight management treatment on the power  
425 of food over an individual and patient susceptibility to the food environment.

426

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445 manuscript writing; J. Cappelleri, A. Bushmakin, R. Gerber, C. Sexton, and N. Kline Leidy; M.  
446 Lowe and J. Karlsson provided significant advice and consultation and participated in the data  
447 interpretation and writing of the manuscript.

448

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- 553

554 **TABLE 1**

555 Descriptive statistics for the initial Power of Food Scale items in the clinical and web-based  
 556 studies<sup>1</sup>

Item no. based on		Item Subject	Clinical study	Web-based study
21-item	15-item			
1	1	I find myself thinking about food even when I am not physically hungry	2.49 ± 1.15	2.07 ± 1.12
2 <sup>2</sup>		When I'm in a situation where delicious foods are present but I have to wait to eat them, it is very difficult for me to wait	2.08 ± 1.13	1.65 ± 0.96
3	2	I get more pleasure from eating than I do from almost anything else	2.05 ± 1.15	1.78 ± 1.04
4 <sup>2</sup>		I feel that food is to me like liquor is to an alcoholic	2.13 ± 1.33	1.6 ± 1.02
5	3	If I see or smell a food I like, I get a powerful urge to have some	2.88 ± 1.19	2.03 ± 1.06
6	4	When I'm around fattening food I love, it's hard to stop myself from at least tasting it	3.23 ± 1.29	2.18 ± 1.22
7 <sup>2</sup>		I often think about what foods I might eat later in the day	2.8 ± 1.28	2.06 ± 1.15
8	5	It's scary to think of the power that food has over me	2.53 ± 1.43	1.64 ± 1.07

9 <sup>2</sup>		When I taste a favorite food, I feel intense pleasure	2.83 ± 1.26	2.14 ± 1.13
10	6	When I know a delicious food is available, I can't help myself from thinking about having some	2.93 ± 1.24	2.09 ± 1.13
11	7	I love the taste of certain foods so much that I can't avoid eating them even if they're bad for me	2.86 ± 1.31	2.15 ± 1.2
12 <sup>2</sup>		When I see delicious foods in advertisements or commercials, it makes me want to eat	2.26 ± 1.13	1.67 ± 0.95
13 <sup>2</sup>		I feel like food controls me rather than the other way round	2.4 ± 1.3	1.54 ± 0.96
14	8	Just before I taste a favorite food, I feel intense anticipation	2.07 ± 1.15	1.54 ± 0.86
15	9	When I eat delicious food I focus a lot on how good it tastes	2.88 ± 1.19	2.51 ± 1.19
16	10	Sometimes, when I'm doing everyday activities, I get an urge to eat "out of the blue" (for no apparent reason)	2.28 ± 1.23	1.69 ± 1
17	11	I think I enjoy eating, a lot more than most other people	2.36 ± 1.28	1.82 ± 1.07
18	12	Hearing someone describe a great meal makes me really want to have something to eat	2.15 ± 1.09	1.66 ± 0.95
19	13	It seems like I have food on my mind a lot	2.28 ± 1.28	1.61 ± 0.99
20	14	It's very important to me that the foods I eat are as delicious as possible	2.98 ± 1.26	2.48 ± 1.26

21	15	Before I eat a favorite food my mouth tends to flood		
		with saliva	1.95 ± 1.17	1.52 ± 0.89

---

557

558 <sup>1</sup>All values are mean ± standard deviation.559 <sup>2</sup>Item has been deleted from questionnaire following psychometric validation.

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563 **TABLE 2**564 Comparative fit indexes of the Power of Food Scale models in the clinical study<sup>1</sup>

Model	CFI	PNFI	ECVI
1-factor (21 items)	0.86	0.77	2.04
2-factor (20 items)	0.90	0.79	1.46
3-factor (18 items)	0.92	0.79	1.04
4-factor (17 items)	0.93	0.77	0.81
5-factor (19 items)	0.94	0.78	0.82
3-factor (15 items)	0.95	0.78	0.48

565

566 <sup>1</sup>CFI: Comparative Fit Index; PNFI: Parsimonious Normed Fit Index; ECVI: Expected Cross

567 Validation Index.

568

569

570

571 **TABLE 3**

572 Power of Food Scale scores (mean  $\pm$  standard error) by group in the clinical and web-based  
 573 studies

	Clinical Study		Web-based Study	
	Obese without diabetes (Group 0)	Obese with diabetes (Group 1)	Obese without diabetes (Group 2)	Nonobese, no diabetes or dyslipidemia (Group 3)
<b>PFS domain<sup>1,2</sup></b>				
Factor 1 (Food Available)	2.33 $\pm$ 0.02	1.82 $\pm$ 0.04	1.86 $\pm$ 0.04	1.48 $\pm$ 0.06
Factor 2 (Food Present)	2.98 $\pm$ 0.02	2.10 $\pm$ 0.05	2.23 $\pm$ 0.05	1.90 $\pm$ 0.07
Factor 3 (Food Tasted)	2.41 $\pm$ 0.02	1.91 $\pm$ 0.04	2.04 $\pm$ 0.04	1.80 $\pm$ 0.05
Aggregate Factor	2.57 $\pm$ 0.02	1.95 $\pm$ 0.04	2.04 $\pm$ 0.04	1.73 $\pm$ 0.05
<b>Body Mass Index</b>				
(kg/m <sup>2</sup> )	38.56	35.99	35.13	23.20

574 <sup>1</sup>PFS: Power of Food Scale

575 <sup>2</sup>Factor 1: difference between group 1 and 2 is not statistically significant ( $P > 0.05$ ); factor 2: all  
 576 differences are statistically significant ( $P < 0.05$ ); factor 3: difference between group 1 and 3 is  
 577 not statistically significant ( $P > 0.05$ ); aggregated factor: difference between group 1 and 2 is not  
 578 statistically significant ( $P > 0.05$ ).

579

580

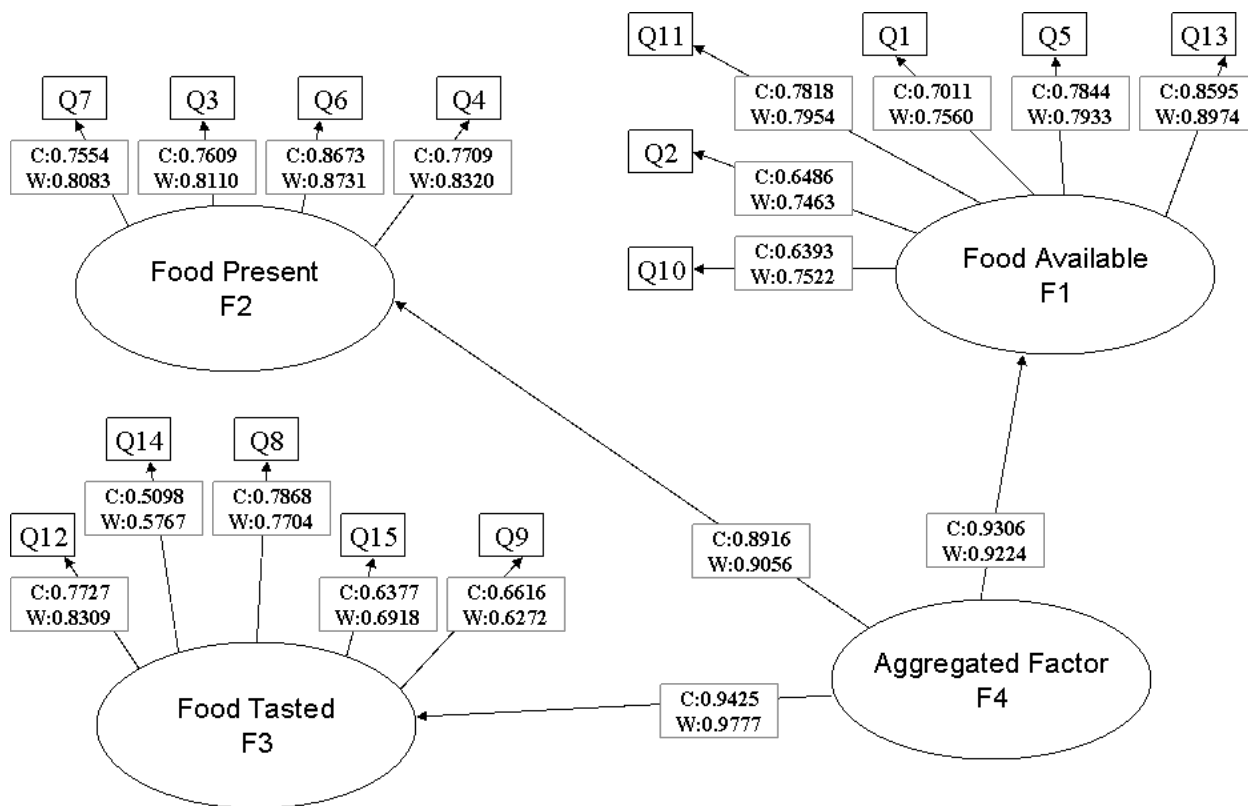
581 **FIGURE 1.** Factor structure of the Power of Food Scale with standardized loadings in the  
582 clinical and web-based studies.

583

584 **FIGURE 2.** Relationship between body mass index (BMI) and Power of Food Scale: Means and  
585 95% confidence intervals for Factors 1, 2 and 3, and Aggregate Factor scores in the web-based  
586 survey.

587

588 Figure 1



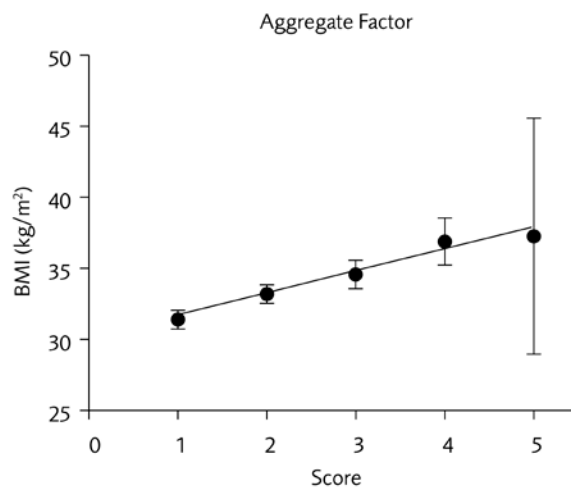
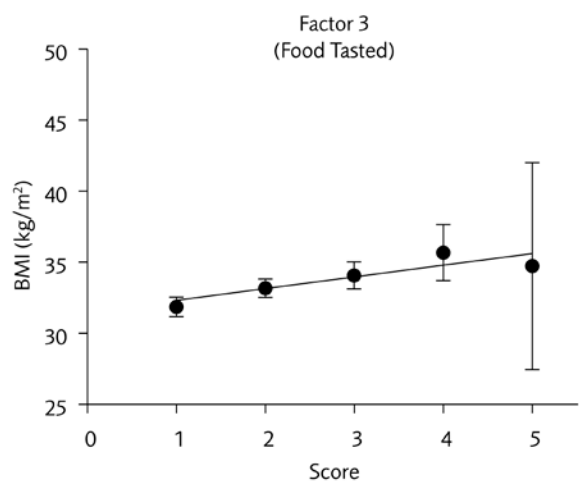
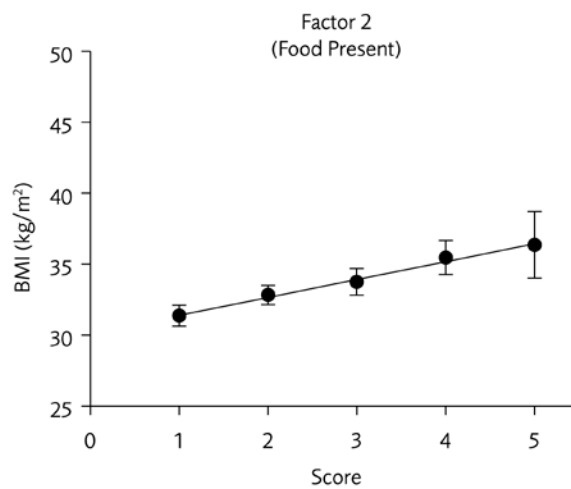
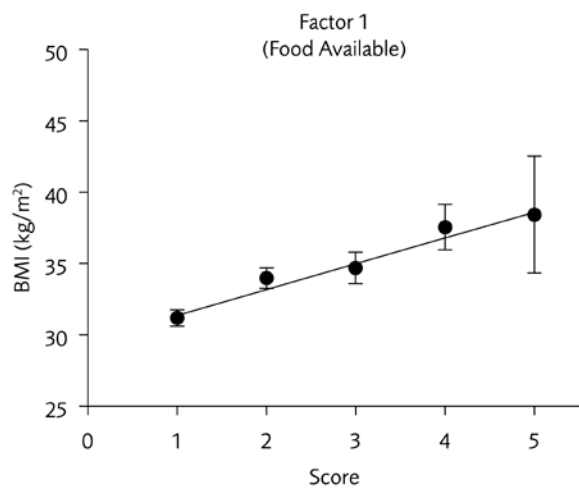
589

590 C: clinical dataset; W: web survey dataset.

591 Item numbers (Q1, Q2, ..., Q15) correspond question numbers in the Appendix.

592

593 Figure 2



594

595

596 Appendix

597 **Power of Food Scale:** Please indicate the extent to which you agree that the following items

598 describe you. Use the following scale from 1–5 for your responses.

		I don't agree (1)	I agree a little (2)	I agree somewh at (3)	I agree quite a bit (4)	I strongly agree (5)
Q1	I find myself thinking about food even when I'm not physically hungry	(1)	(2)	(3)	(4)	(5)
Q2	I get more pleasure from eating than I do from almost anything else	(1)	(2)	(3)	(4)	(5)
Q3	If I see or smell a food I like, I get a powerful urge to have some	(1)	(2)	(3)	(4)	(5)
Q4	When I'm around a fattening food I love, it's hard to stop myself from at least tasting it	(1)	(2)	(3)	(4)	(5)
Q5	It's scary to think of the power that food has over me	(1)	(2)	(3)	(4)	(5)
Q6	When I know a delicious food is available, I can't help myself from thinking about having some	(1)	(2)	(3)	(4)	(5)
Q7	I love the taste of certain foods so much that I can't avoid eating them	(1)	(2)	(3)	(4)	(5)

	even if they're bad for me					
Q8	Just before I taste a favorite food, I feel intense anticipation	(1)	(2)	(3)	(4)	(5)
Q9	When I eat delicious food I focus a lot on how good it tastes	(1)	(2)	(3)	(4)	(5)
Q10	Sometimes, when I'm doing everyday activities, I get an urge to eat "out of the blue" (for no apparent reason)	(1)	(2)	(3)	(4)	(5)
Q11	I think I enjoy eating a lot more than most other people	(1)	(2)	(3)	(4)	(5)
Q12	Hearing someone describe a great meal makes me really want to have something to eat	(1)	(2)	(3)	(4)	(5)
Q13	It seems like I have food on my mind a lot	(1)	(2)	(3)	(4)	(5)
Q14	It's very important to me that the foods I eat are as delicious as possible	(1)	(2)	(3)	(4)	(5)
Q15	Before I eat a favorite food my mouth tends to flood with saliva	(1)	(2)	(3)	(4)	(5)

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