

ORIGINAL ARTICLE

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

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Background: The Power of Food Scale (PFS) was developed to assess the psychological impact of today's food-abundant environments.

Objective: To evaluate the structure of the PFS in diverse populations of obese and nonobese individuals.

Design: Data were obtained from obese adults in a clinical trial for a weight management drug ($n = 1741$), and overweight, obese and normal weight adults in a Web-based survey ($n = 1275$). Exploratory and confirmatory factor analyses were used to investigate the PFS structure using the clinical data. The model developed was then tested using the Web-based data. Relationships between PFS domains and body mass index (BMI) were examined. Logistic regression was used in the Web-based survey to evaluate the association between obesity status and PFS scores.

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

Conclusions: The 15-item PFS is best represented by three domains and an aggregate domain. The PFS may provide a useful tool to evaluate the effects of obesity treatments on feelings of being controlled by food in an obesogenic food environment. *International Journal of Obesity* (2009) 33, 913–922; doi:10.1038/ijo.2009.107; published online 9 June 2009

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Introduction

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

Eating behavior in humans is an extremely complex process modulated by internal homeostatic processes^{11–13} and environmental and social influences.^{14,15} As such,

energy homeostasis aims to match energy intake with overall energy needs; however, total food intake is also influenced by external factors, such as food availability and palatability, which might lead to excess energy intake during any given meal.¹⁶ This tendency to eat beyond immediate homeostatic needs has presumably evolved because the motivation to seek and consume calories contributed to survival when food sources were scarce and unpredictable.¹⁷ Eating behavior can be further divided into two linked yet distinct, differentially regulated processes:^{18,19} (1) appetitive responses that precede food intake and (2) consummatory behaviors that determine what and how much is eaten.

Chronic excess energy intake, either through increased meal size or meal number, will ultimately lead to weight gain and obesity. The reasons why only certain individuals chronically overeat are unclear,^{6,20} but may reflect differences in appetitive and consummatory responses to food, an enhanced sensitivity to a food-rich environment and an

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overpowering of homeostatic processes by food reward in these individuals.^{16,21–23} The motivation to consume foods beyond homeostatic need has been referred to as ‘hedonic hunger’.²²

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

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

Methods

Development of the PFS

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

Any items describing actual food consumption or overeating were eliminated from consideration to avoid confounding the measure with a behavior it might be used to predict. Items describing the obesogenic food environment itself, rather than reactions to the environment, were also excluded, because the PFS presumes that respondents live in an environment where a variety of palatable foods are readily accessible. Further, items describing emotional eating were excluded for several reasons: measures of emotional eating already exist (for example, DEBQ, Emotional Eating Subscale, revised TFEQ),^{24–27} such items suggest a susceptibility to overeating, and the presumed source of such consumption is negative affect rather than something about the food environment *per se*. Items that were redundant or that

described idiosyncratic scenarios that would likely apply to a small percentage of respondents were also eliminated.

In examining the items generated by group members, it became apparent that they could be categorized according to the proximity of food to the respondent. Because in most developed countries food is almost always readily available (for example, in the refrigerator or at a corner store), people implicitly know that food is constantly attainable. Thus the first level of food availability is abstract, reflecting the impact of the mere accessibility of food (sample item: ‘I find myself thinking about food even when I’m not physically hungry’). At a second and more proximal level, food may not only be available but actually present in a given context. In this case, the presence of visual and olfactory food cues could potentially influence people differently than if food is available in the environment but not physically present (sample item: ‘If I see or smell a food I like, I get a powerful urge to have some’). The third and most proximal context is when food is not only present but is, or is about to be, tasted (sample item: ‘When I eat delicious food I focus a lot on how good it tastes’).

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

It is important to note the two assumptions underlying the development and use of the PFS. The first assumption is that the environmental context in which it is used features relatively abundant, accessible, palatable and affordable foods; it is not intended for use in a food-impooverished environment where, for example, the item ‘I often think about what foods I might eat later in the day’ may be a product not of hedonic anticipation but of hunger and deprivation. This presumption should help ensure that PFS scores represent psychological temptation rather than physiological need. The second assumption is that the measure is designed to tap an individual’s subjective experiences to living in a food-abundant environment and the power of food over that individual; it is not designed to assess features of the food environment itself. It is appropriate to use the

PFS to compare individuals living in the same or similar food-abundant environments but not individuals from environments in which the availability of palatable foods differs significantly.

PFS questionnaire

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

Study populations

Two distinct study populations were used. The factor structure of the PFS was initially examined using baseline data, before treatment intervention, from a phase 3 clinical trial of a candidate weight management compound (which was previously but is no longer in development). The data set included 1741 obese, nondiabetic individuals (obesity defined as body mass index (BMI) $>30 \text{ mg kg}^{-2}$ for subjects without comorbidities; BMI $>27 \text{ kg m}^{-2}$ for subjects with comorbidities (treated or untreated hypertension or dyslipidemia)) from the United States and Canada. Questionnaires were administered in the clinic on day 1. Data obtained from these subjects are referred to as 'clinical study' data.

The structure of the PFS was then further evaluated using data obtained from subjects recruited from the US arm of the 2006 National Health and Wellness Survey (NHWS). The data set included 1275 subjects in total and recruited subjects from the following groups: nonobese healthy subjects (BMI $18.5\text{--}26.9 \text{ kg m}^{-2}$, with no diabetes or dyslipidemia), overweight and obese subjects (BMI $\geq 27.0 \text{ kg m}^{-2}$) with diabetes, and overweight and obese subjects (BMI $\geq 27.0 \text{ kg m}^{-2}$) without diabetes. The NHWS is an annual study of the health-care attitudes and behaviors of nationally representative samples of the adult population. In 2006, the NHWS was fielded to more than 60 000 members of the general panel of Lightspeed Research.

Through panel identification numbers, respondents were identified and re-contacted for this study. Any respondent with a BMI $\geq 18.5 \text{ kg m}^{-2}$ was eligible to be re-contacted to participate in this study and they were invited to do so randomly. As respondents entered the questionnaire, they were re-screened for eligibility and categorized into three quota groups: 500 obese subjects with diabetes, 500 obese subjects without diabetes and 250 nonobese subjects. Within both the obese with diabetes and obese without diabetes groups, there were minimum quotas based on BMI: at least 100 respondents must have a BMI 27 to 29 kg m^{-2} ; at least 100 respondents must have a BMI 30 to 34 kg m^{-2} ; at least 100 respondents must have a BMI greater than or equal to 35 to 39 kg m^{-2} and at least 100 respondents must have a BMI $\geq 40 \text{ kg m}^{-2}$ (values less than 0.5 were rounded to the

next lower integer and 0.5 or greater to the next higher integer).

Once quota groups were filled, only respondents to NHWS who met the criteria for the open quota groups received subsequent invitations to participate in the study. Members of the Lightspeed Research panel were recruited through opt-in email, co-registration with Lightspeed Research partners, e-newsletter campaigns, banner placements, and both internal and external affiliate networks. The questionnaire was self-administered through the Internet. The questionnaire takes approximately 20 min to complete. Respondents needed to have the Internet access to participate, but that access may or may not have been in their homes. Information about point of the Internet access was not collected. The formatting of the screen presentation was as close as possible to the questionnaire provided in the clinical study. All data were self-reported by the survey respondents and were not verified against any clinical diagnostics. Data obtained from these subjects are referred to as 'Web-based survey' data.

Descriptive analyses

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

Psychometric analysis

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

Within the clinical trial sample, the number of factors was explored by using a parallel analysis.^{29–35} Parallel analysis allows the identification of factors that are beyond chance. In addition to this factor analysis of real data, a series of simulations were performed involving the analysis of a matrix of random numbers that represented the same number of cases and variables. We then used an iterative process of extracting factors from candidate models using exploratory factor analysis and then tested each model using confirmatory factor analysis. In exploratory factor analysis calculations, for an item to be considered as a part of a particular factor, the standardized regression coefficient must have a relatively high standardized pattern loadings (≥ 0.40) on this factor and low loadings (< 0.40) on the other factors.³⁰

For the confirmatory factor analysis, Bentler's Comparative Fit Index (CFI) was the main indicator of fit and was obtained for each candidate model; CFI values of > 0.9 indicate acceptable fit.²⁹ We used two additional indexes to compare models: the Parsimonious Normed Fit Index (PNFI)³² and the Expected Cross Validation Index (ECVI).³⁶ The PNFI simultaneously reflects both the fit and the parsimony of the model; the model with the largest PNFI is most

parsimonious one. The ECVI gauges the applicability or generalizability of results; the model with the smallest ECVI value is considered to be the most stable in the population.

Confirmatory factor analyses were then undertaken on data obtained from subjects recruited to the Web-based survey to confirm the structure of the PFS model developed using data from the clinical study. We conducted additional analyses and validity tests to examine the stability of the measurement model for obese subjects (BMI of $>30 \text{ kg m}^{-2}$) and nonobese subjects (BMI of $<30 \text{ kg m}^{-2}$), as well as for gender, in the Web-based sample. In doing so we compared and tested two multigroup models: the 'no-constraint' model, which does not impose any constraint on its parameters in the two subgroups of interest (for example, obese vs nonobese), and the 'invariant' measurement model, which constrains the corresponding factor loadings in the two subgroups to be equal. Evidence for the 'invariant' measurement model exists if two conditions are met: the multigroup models exhibit adequate fit (say, CFI of 0.90+) and that the difference between these two models is negligible (0.01 or less).^{37,38}

In the Web-based survey, as well as in the clinical study, Cronbach's α reliability coefficient³⁹ was computed for each domain to measure internal consistency. The relationship of the PFS with BMI was determined using regression (no functional form was imposed). Here BMI was treated as a continuous outcome (response or dependent) variable and PFS domain as a discrete predictor (with mean score per item

rounded to the nearest discrete category, being one of the integers from 1 to 5). Moreover, logistic regression was used in the Web-based survey to 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 age and gender included as covariates.

Using both studies, we used Pearson's correlation coefficients to examine the association between PFS and the TFEQ with 21 items.²⁶

Results

Participants

There were 1741 patients recruited into the clinical trial. Mean \pm s.d. BMI was $38.6 \pm 6.7 \text{ kg m}^{-2}$; mean \pm s.d. age was 46.3 ± 11.0 years; 1427 (82%) of the patients were women. Of the 1275 subjects recruited to complete the Web-based survey (mean \pm s.d. BMI: $33.1 \pm 7.6 \text{ kg m}^{-2}$; mean \pm s.d. age 52.5 ± 12.8 years; 39% women), 250 subjects had a BMI of 18.5 to 26.9 kg m^{-2} (56% women), 518 subjects had a BMI of 27.0 to 75.9 kg m^{-2} and no diabetes (46% women), 503 subjects had a BMI of 27.1 to 67.5 kg m^{-2} and had a diagnosis of diabetes (25% women), and in four subjects diabetic status was unavailable.

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

Table 1 Descriptive statistics for the initial PFS items in the clinical and Web-based studies

Item no. based on questionnaire	15-item	Item subject	Clinical study	Web-based study
1	1	I find myself thinking about food even when I am not physically hungry	2.49 ± 1.15	2.07 ± 1.12
2 ^a		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 ^a		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 ^a		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 ^a		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 ^a		When I see delicious foods in advertisements or commercials, it makes me want to eat	2.26 ± 1.13	1.67 ± 0.95
13 ^a		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

^aItem has been deleted from questionnaire following psychometric validation. All values are mean \pm standard deviation. 2006, Drexel University. All rights reserved. No part of this work may be reproduced or used in any form without the prior written permission of the copyright holder or the author, Michael Lowe.

Assessment of PFS in the clinical study

Measurement model development. Using parallel analysis, we determined that there may be between one and eight factors in the model. CFIs for one- and two-factor models were <0.90, indicating that these models did not adequately fit the data. CFI for the three-, four- and five-factor solutions were >0.90; therefore these models fit the data (Table 2). Extracting a solution beyond five factors did not produce a new structure compared with the five-factor model. From the three-, four- and five-factor models, the three-factor model (with three weak items removed resulting in an 18-item model) was selected to represent the PFS measurement model as the most parsimonious solution (based on PNFI).

Final measurement model. The three-factor model gave the largest ECVI (Table 2). This indicates that although this model was the most parsimonious one, it could be less generalizable than the four- and five-factor models. Additional calculations were performed to refine the three-factor

Table 2 Comparative fit indexes of the PFS models in the clinical study

Model	CFI	PNFI	ECVI
One-factor (21 items)	0.86	0.77	2.04
Two-factor (20 items)	0.90	0.79	1.46
Three-factor (18 items)	0.92	0.79	1.04
Four-factor (17 items)	0.93	0.77	0.81
Five-factor (19 items)	0.94	0.78	0.82
Three-factor (15 items)	0.95	0.78	0.48

Abbreviations: CFI, Comparative Fit Index; PNFI, Parsimonious Normed Fit Index; ECVI, Expected Cross Validation Index.

model and achieve better generalizability and fit of the model. On the basis of obtaining a CFI and ECVI better than those obtained for the existing the three-, four- and five-factor models, a three-factor model containing 15 items emerged (see ; permission to use the PFS can be obtained from Dr MR Lowe at lowe@drexel.edu). Items deleted from the original 21-item PFS are detailed in Table 1.

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

Confirmation of the three-factor, second-order model

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

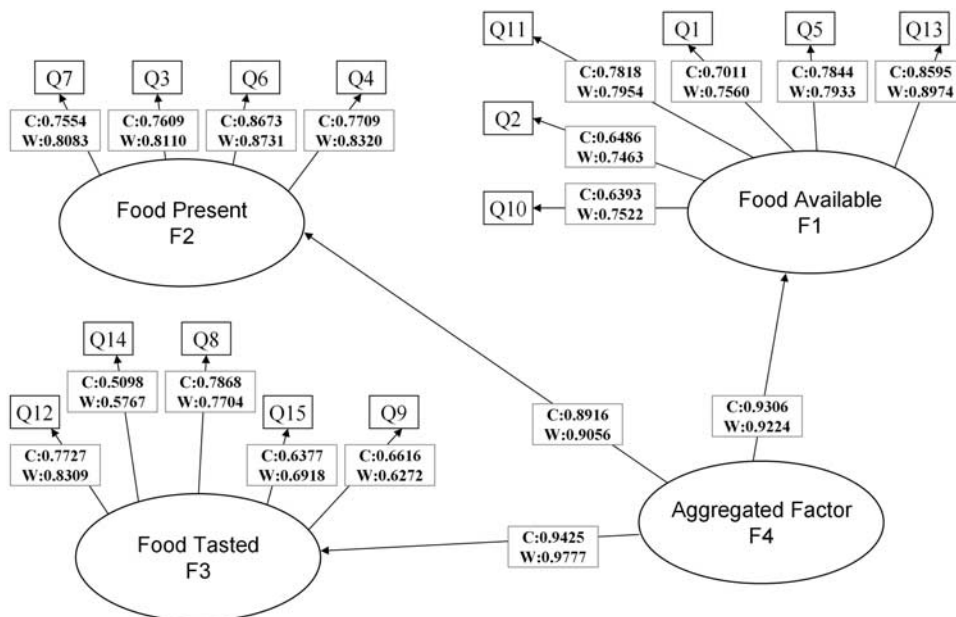


Figure 1 Factor structure of the Power of Food Scale with standardized loadings in the clinical and Web-based studies. C, clinical data set; W, Web survey data set. Item numbers (Q1, Q2, ..., Q15) correspond question numbers in the Appendix.

Characteristics of the three-factor, second-order model

The three-factor, second-order structure identified with the clinical population was confirmed in the relatively more diverse population enrolled in the Web-based survey. Each of the three domain scores was calculated as the mean score per item across items that constitute a given domain. The aggregate domain score was calculated as the mean of three single domain scores. Items on the first, second and third domains relate to 'food available', 'food present' and 'food tasted', respectively. All factor loadings were clearly statistically significant ($t > 1.96$) and all standardized factor loadings were large (Figure 1).

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

In both studies the correlation coefficients between each item and BMI, as well as between every domain and BMI, were from negligible to small (ranging from 0.003 to 0.24 for items and from 0.02 to 0.24 for domains). A regression model using a domain score as a predictor (adjusted for age and gender) was also applied. In the clinical study, a one-category increase in factor 1 score was associated with a 0.37 kg m^{-2} increase in BMI (95% CI: $-0.15, 0.89$; $P = 0.17$) a one-category increase in factor 2 score was associated with a 0.17 kg m^{-2} increase in BMI (95% CI: $-0.22, 0.57$; $P = 0.39$) and a one-category increase in factor 3 score was associated with a 0.22 kg m^{-2} increase in BMI (95% CI: $-0.39, 0.84$; $P = 0.48$).

Despite the fact that these associations were not statistically significant in the clinical study, there were some indication of a linear relationship between domain score and BMI in the Web-based survey (Figure 2). In the Web-based survey, for factor 1 a one-category difference was associated with a change in BMI of 1.81 kg m^{-2} (95% CI: $0.77, 2.85$; $P = 0.0006$), for factor 2 a one-category difference was associated with a change in BMI of 1.25 kg m^{-2} (95% CI: $0.63, 1.86$; $P < 0.0001$), for factor 3 a one-category difference was associated with a change in BMI of 0.72 kg m^{-2} (95% CI: $-1.11, 2.54$; $P = 0.44$) and for the aggregated domain a one-category difference in score was associated with a change in BMI of 1.47 kg m^{-2} (95% CI: $-0.61, 3.55$; $P = 0.17$). Although the correlation results on PFS and BMI showed at best a modest relationship, where variability (noise) dominated, the relationships between each factor and BMI were visibly close to linear (Figure 2). This general relationship between

PFS domains and BMI was also supported by comparison of the mean domain scores and mean BMI in studied populations. Mean scores for each PFS domain were higher in obese subjects in the clinical trial, who had the greatest mean BMI, and lowest in nonobese participants in the Web-based survey, who had the lowest BMI (Table 3).

Within the Web-based survey, logistic regression in overweight vs non-overweight participants (adjusted for age and gender) indicated that an increase in a domain score by one category increased the chance to be overweight by 2.27 times for factor 1 (95% CI: 1.80, 2.87), 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) and by 1.97 times for the aggregate score (95% CI: 1.58, 2.45). These results were statistically significant (the 95% CI did not include the null value of 1).

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

In the Web-based survey, for the obese and nonobese subgroups, the no-constraint model gave a CFI of 0.9324 and the invariance model gave a CFI of 0.9264 (each of these two models combined the obese and nonobese subgroups into one model with one CFI). Because these CFI values exceeded 0.90 and their difference was negligible (0.006), the invariance and stability of the PFS measurement model relative to obesity status was supported by the data. For gender subgroups, the no-constraint model gave a CFI of 0.9342 and the invariance model gave a CFI of 0.9323. Because these CFI values exceeded 0.90 and their difference was negligible (0.0019), the invariance and stability of the PFS measurement model relative to gender was supported by the Web-based data. Data from the clinical sample, which had sufficient numbers of men and women for meaningful analysis, concurred: the no-constraint model gave a CFI of 0.9449 and the invariance model gave a CFI of 0.9442, a difference of 0.0007.

Discussion

We determined that the initially proposed candidate set of 21 items of the PFS could be reduced to 15, and that the scale was best represented by a three-factor, second-order model with three domains and an aggregate domain. For each domain, its score was the mean score per item in a given

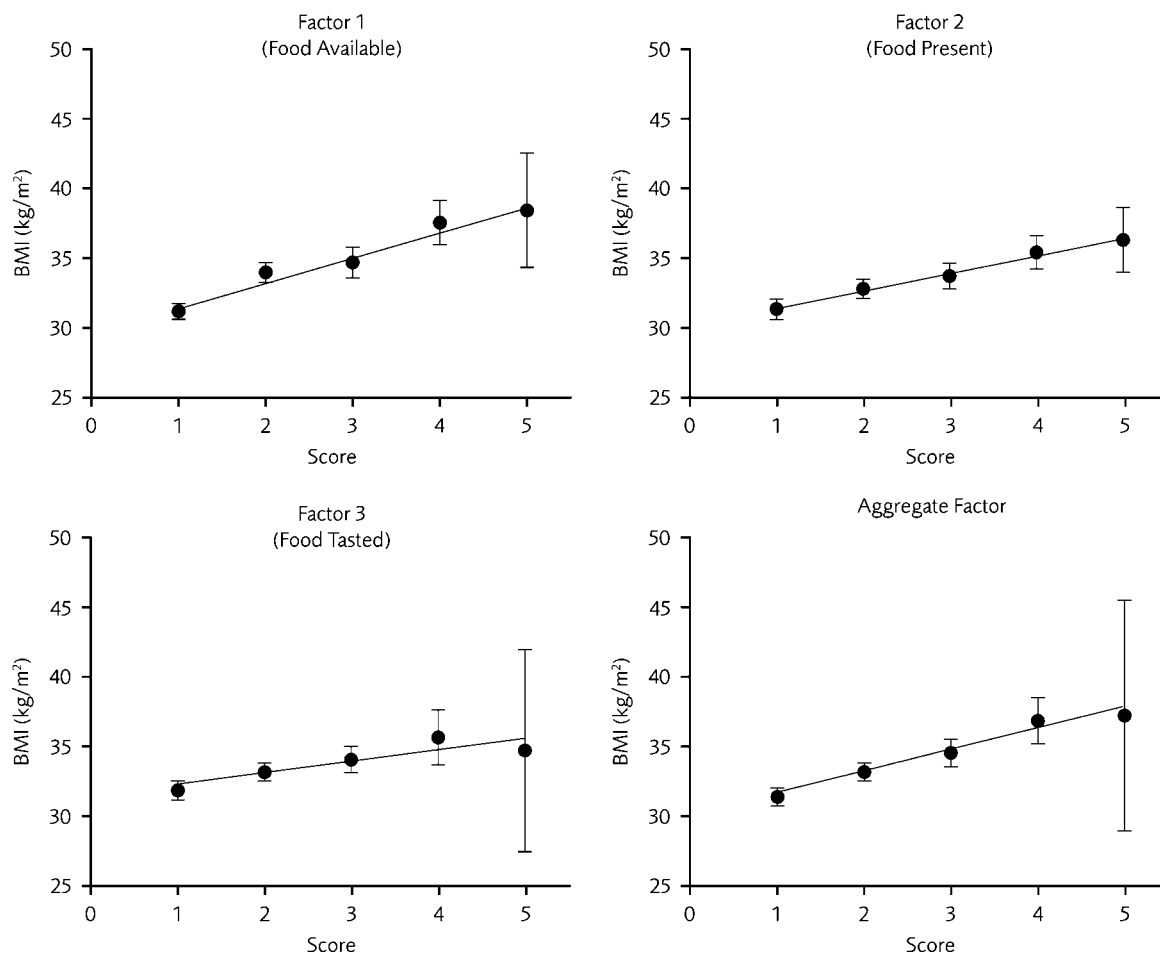


Figure 2 Relationship between body mass index (BMI) and Power of Food Scale: means and 95% confidence intervals for factors 1, 2 and 3, and aggregate factor scores in the Web-based survey.

Table 3 PFS scores (mean \pm standard error) by group in the clinical and Web-based 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)
<i>PFS domain</i> ^a				
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
Body mass index (kg m ⁻²)	38.56	35.99	35.13	23.20

Abbreviation: PFS: Power of Food Scale. ^aFactor 1, difference between groups 1 and 2 is not statistically significant ($P > 0.05$); factor 2, all differences are statistically significant ($P < 0.05$); factor 3, difference between groups 1 and 3 is not statistically significant ($P > 0.05$); aggregated factor, difference between groups 1 and 2 is not statistically significant ($P > 0.05$).

domain. Scores for the aggregate domain were calculated as the mean of those three domain scores.

The three subscale domains and aggregate domain engendered high internal consistency. Subscale scores from the three domains can be used to provide further description of the psychological impact of the food environment in each

of the three levels of proximity to food: food readily available in the environment but not physically present, food present but not tasted and food when first tasted but not consumed. Corrected-to-total item correlations were, in general, reasonably strong, supporting the conclusion that items on the same factor represented a common concept or construct and the

adoption of the aggregate score, as a proxy for the common factor.

In the clinical study, there was a weak and not statistically significant relationship between PFS and BMI. In the Web-based survey, the correlation between BMI and PFS aggregate and domain scores was modest. The PFS may be more strongly related to overeating tendencies among overweight and obese individuals than to BMI *per se*. If the PFS measures an appetitive predisposition for overconsumption of energy, then one would expect a substantial PFS–BMI relationship; however, a number of factors may mitigate against such a relationship, such as the energy-efficient individuals who gain weight despite minimal overeating and energy-inefficient individuals who do not gain weight despite frequent overeating.⁴⁰ In addition, the degree of ‘pull’ obesity-prone individuals feel from the obesogenic environment may be greater when they are gaining weight than when their weight stabilizes at an obese level.⁴¹ Studies have shown that extreme obesity may be associated with a downregulation of dopamine receptors and a reduction in the rewarding value of food.⁴² This attenuated PFS–BMI association suggests the potential usefulness of the PFS to identify those individuals who might benefit most from particular interventions.

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

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

Eating behavior in humans is a physiological requirement to maintain energy homeostasis. However, in developed countries, where there is often an abundance of food, it may be appropriate to differentiate between homeostatic and hedonic hunger.²² Existing measures assess actual consumption of food associated with various stimuli such as negative mood, social cues and hunger,^{24–27} but do not permit a differentiation of appetitive drive in relation to the consumption of food. Given the wide diversity of stimuli that have been associated with food intake, if the PFS can accurately assess individuals’ hedonic appetitive drive in food-abundant environments, it may represent a brief and cost-effective tool in the study of human eating behavior and may potentially be predictive of weight gain or response to weight management treatment in certain individuals.

The PFS could also help in the development of appropriate and effective pharmacotherapy for obesity. Current anorectic

agents affect food intake in different ways—for example, in obese subjects, the serotonin agent d-fenfluramine reduced perceived hunger but not the pleasantness of food.⁴³ In contrast, opioid antagonists reduced the pleasantness of food without affecting hunger ratings.⁴⁴ In animals, the cannabinoid antagonist rimonabant reduced the motivational drive for food without influencing palatability.⁴⁵ If certain obese patients are especially susceptible to the food environment, an ideal weight management program would reduce this influence without affecting the pleasure associated with eating. For example, an intervention might reduce the sensitivity of an individual to the food environment, or modify the nature of foods immediately available to participants.⁴⁶

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

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

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

Conflict of interest

Editorial Support was provided by Joyce A Healey at Pfizer Inc. and Zoë Thornton-Jones at Envision Pharma. JC

Cappelleri, AG Bushmakin and RA Gerber are employees of Pfizer Inc. CC Sexton and NK Leidy are employees of United BioSource Corporation, and were paid consultants to Pfizer Inc. in connection with the development of the paper. J Karlsson and MR Lowe are consultants to Pfizer Inc. in relation to this study.

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Appendix

Power of Food Scale: Please indicate the extent to which you agree that the following items describe you. Use the following scale from 1 to 5 for your responses.

		<i>I don't agree</i>	<i>I agree a little</i>	<i>I agree somewhat</i>	<i>I agree quite a bit</i>	<i>I strongly agree</i>
		(1)	(2)	(3)	(4)	(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 even if they're bad for me	(1)	(2)	(3)	(4)	(5)
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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