Modeling weight-loss maintenance to help prevent body weight regain\textsuperscript{1–3}

Kevin D Hall and Peter N Jordan

ABSTRACT

Background: Lifestyle intervention can successfully induce weight loss in obese persons, at least temporarily. However, there currently is no way to quantitatively estimate the changes of diet or physical activity required to prevent weight regain. Such a tool would be helpful for goal-setting, because obese patients and their physicians could assess at the outset of an intervention whether long-term adherence to the calculated lifestyle change is realistic.

Objective: We aimed to calculate the expected change of steady-state body weight arising from a given change in dietary energy intake and, conversely, to calculate the modification of energy intake required to maintain a particular body-weight change.

Design: We developed a mathematical model using data from 8 longitudinal weight-loss studies representing 157 subjects with initial body weights ranging from 68 to 160 kg and stable weight losses between 7 and 54 kg.

Results: Model calculations closely matched the change data ($R^2 = 0.83 $, $\chi^2 = 2.1, P < 0.01$ for weight changes; $R^2 = 0.91, \chi^2 = 0.87, P < 0.0004$ for energy intake changes). Our model performed significantly better than the previous models for which $\chi^2$ values were 10-fold those of our model. The model also accurately predicted the proportion of weight change resulting from the loss of body fat ($R^2 = 0.90$).

Conclusions: Our model provides realistic calculations of body-weight change and of the dietary modifications required for weight-loss maintenance. Because the model was implemented by using standard spreadsheet software, it can be widely used by physicians and weight-management professionals. Am J Clin Nutr 2008; 88:1495–503.

INTRODUCTION

The increasing prevalence of obesity poses a serious health concern (1). Whereas lifestyle interventions can result in significant weight loss in obese patients, weight regain is very common (2, 3). Therefore, rather than focusing exclusively on weight loss, several investigators have emphasized the importance of maintaining body weight at a lower level and preventing weight regain (4–7). Data from the National Weight Control Registry have been used to glean useful insights regarding the strategies used by persons who have successfully maintained significant weight change over extended periods (6, 7). However, there currently are no available quantitative tools to estimate, at the outset of obesity treatment, the lifestyle changes required to maintain a specific weight-loss goal. In other words, the question remains: If a patient wishes to change his or her body weight by a certain amount ($\Delta$BW), how would his or her diet or physical activity have to change to maintain the goal weight? A quantitative answer to this question would be helpful for goal-setting, because both patient and physician could assess whether long-term adherence to the calculated lifestyle change is a realistic proposition. Such a calculation is not currently possible.

In this report, we propose a mathematical model for calculating the changes in dietary intake and physical activity required to maintain a given body-weight change and to prevent weight regain. To facilitate use of the model by physicians and weight-management professionals, the model was implemented by using standard spreadsheet software that can be downloaded (see Spreadsheet files under “Supplemental data” in the current online issue; the spreadsheet files and an online version of the model are available at http://www2.niddk.nih.gov/NIDDKLabs/LBM/lbmHall.htm). We developed our model by using longitudinal weight-change data from studies that measured energy expenditure (EE) and body composition during periods of weight stability both before and after weight loss (8–15). We compared the performance of our model with that of previous mathematical models (16–18) regarding the capacity to match the steady-state weight-change data, and we found that our model was superior and could provide realistic estimates of both the magnitude of the weight change and the changes in dietary energy intake (EI) required to prevent weight regain.

MATERIALS AND METHODS

Proposed model of steady-state body-weight change

We developed a simple model of the steady-state EE rate of the body as a function of body composition, EI, and physical activity:

$$EE = K + \beta EI + \gamma_{FFM}FFM + \gamma_{FM}FM + \delta (FFM + FM) \quad (1)$$

where $K$ is a constant, $\gamma_{FFM} = 22$ kcal kg$^{-1} \cdot d^{-1}$, and $\gamma_{FM} = 3.2$ kcal kg$^{-1} \cdot d^{-1}$ are the regression coefficients for resting metabolic rate versus fat-free mass (FFM) and fat mass (FM), respectively.

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there is significant debate as to whether such adaptations occur providing the best fit to the empirical Forbes body-composition where FM\textsubscript{init} is the initial body FM, the equation of body-composition change (22) predicts that the change in FFM is given by the following nonlinear function:

\[
\Delta \text{FFM} = \beta \Delta \text{EI} + \left( \gamma_{\text{FFM}} + \delta_{\text{init}} + \Delta \delta \right) \Delta \text{FFM} + \beta FM_{\text{init}} \Delta \delta
\]

where BW\textsubscript{init} is the initial body weight, and ΔFM and ΔFFM are the changes in body fat and FFM, respectively. (Note that the constant, K, no longer appears in equation 2, so we need not specify its value.) Using the fact that ΔBW = ΔFFM + ΔFM, the following equation holds for the change in FFM:

\[
\Delta \text{FFM} = [(1 - \beta) \Delta \text{EI} - BW_{\text{init}} \Delta \delta - (\gamma_{\text{FFM}} + \delta_{\text{init}} + \Delta \delta) \Delta \text{BFM}]
\]

Our previously published modification of the classic Forbes equation of body-composition change (22) predicts that the change in FFM is given by the following nonlinear function:

\[
\Delta \text{FFM} = \Delta \text{BW} + FM_{\text{init}} - C
\]

where FM\textsubscript{init} is the initial body FM, C = 10.4 kg is the constant providing the best fit to the empirical Forbes body-composition curve (23), and is the Lambert W function (24). Equations 3 and 4 can be solved for the expected change in steady-state body weight, as shown in the following equation:

\[
\Delta \text{BW} = (1 - \beta) \Delta \text{EI} - BW_{\text{init}} \Delta \delta - (\gamma_{\text{FFM}} - \gamma_{\text{FM}}) FM_{\text{init}}
\]

\[
+ \frac{C(\gamma_{\text{FFM}} - \gamma_{\text{FM}})}{(\gamma_{\text{FFM}} + \delta_{\text{init}} + \Delta \delta)}
\]

\[
\times \exp \left( \frac{(\gamma_{\text{FFM}} + \delta_{\text{init}} + \Delta \delta) FM_{\text{init}}}{C(\gamma_{\text{FFM}} + \delta_{\text{init}} + \Delta \delta)} \right)
\]

\[
\exp \left( \frac{(1 - \beta) \Delta \text{EI} - BW_{\text{init}} \Delta \delta}{C(\gamma_{\text{FFM}} + \delta_{\text{init}} + \Delta \delta)} \right)
\]

Equation 5 allows us to calculate the change in steady-state body weight, given information about the initial body weight and FM and about the changes in dietary EI and physical activity. We recognize that most readers cannot easily calculate expected weight change results by using equation 5 because of the appearance of the nonelementary Lambert W function. To address this issue, we have provided standard spreadsheet files that will allow readers to examine the predicted changes in steady-state body weight as a function of the model parameters (see Spreadsheet files under “Supplemental data” in the current online issue; the spreadsheet files and an online version of the model are available at http://www2.niddk.nih.gov/NIDDKLabs/LBM/bmHall.htm).

Previous models of steady-state body-weight change

Previous mathematical models have been proposed to calculate ΔBW resulting from a specified change of diet or physical activity (16–18). The first model, by Christiansen et al (16), was given by the following equation:

\[
\Delta \text{BW} = \frac{1}{k} \Delta \left( \frac{\text{EI}}{\text{PAL}} \right)
\]

where \( k = 13.5 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{d}^{-1} \) for men and \( k = 11.6 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{d}^{-1} \) for women, and PAL = the physical activity level, defined as the ratio of total EE rate to the resting metabolic rate.

The second model, by Kozusko (18), expressed the predicted body-weight change according to the following equation:

\[
\Delta \text{BW} = \left( \sqrt{(1 - \alpha)^2 + 4\alpha(1 + \Delta \text{EI/I}_{\text{init}})} \right) + \alpha - 1 \right) \text{BW}_{\text{init}} / 2\alpha
\]

where \( \alpha \) was given by the following equation:

\[
\alpha = \frac{1}{2} \tanh \left( \frac{3/2}{2} \left( \frac{FM_{\text{init}}}{\text{BW}_{\text{init}}} - \left( \frac{FM_{\text{init}}}{\text{BW}_{\text{init}}} - 1 \right) \text{BW}_{\text{init}} \right) \right)
\]

In the third model under consideration, Heymsfield et al (17) used the Institute of Medicine of the National Academies of Science (IOM-NAS) equations to estimate the expected weight change for a given dietary intervention, but those investigators did not allow for changes in physical activity. We accounted for physical activity changes by solving the IOM-NAS equations for the expected body-weight change as follows:

\[
\Delta \text{BW} = \Delta \text{EI}/(K_{1} \text{PA}_{\text{final}}) - (\text{BW}_{\text{init}} + K_{2} h / K_{1}) \text{PA}_{\text{PA}_{\text{final}}}
\]

where \( h \) is height in meters, \( K_{1} = 14.2 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{d}^{-1} \) and \( K_{2} = 503 \text{ kcal} \cdot \text{m}^{-1} \cdot \text{d}^{-1} \) for men, and \( K_{1} = 10.9 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{d}^{-1} \) and \( K_{2} = 660.7 \text{ kcal} \cdot \text{m}^{-1} \cdot \text{d}^{-1} \) for women (25). PA stands for a dimensionless physical activity parameter related to PAL, but it takes on discrete values within a range of PAL values (17, 25).

Proposed model of diet and physical activity changes required to prevent weight regain

Our main goal was to calculate the changes in dietary intake and physical activity required to maintain a specified body-weight change. The following rearrangement of equation 5 expresses the EI change required for a specified body weight change and physical activity change.
\[ \Delta EI = B W_{\text{init}}\Delta \delta/(1 - \beta) + C^2(\gamma_{\text{FFM}} - \gamma_{\text{FM}})F M_{\text{init}}/(1 - \beta) + C^2B W(\gamma_{\text{FFM}} + \delta_{\text{init}} + \Delta \delta)/(1 - \beta) - W[F M_{\text{init}}C \exp \{1 + F M_{\text{init}} + \Delta B W\}] \times C(\gamma_{\text{FFM}} - \gamma_{\text{FM}})/(1 - \beta) \quad (10) \]

We have provided a spreadsheet file for this calculation (see Spreadsheet files under “Supplemental data” in the current online issue; the spreadsheet files and an online version of the model are available at http://www2.niddk.nih.gov/NIDDK labs/LBM/lbmHall.htm). We compared the predicted EI change from equation 10 with the change in the steady-state total EE rate measured in the longitudinal weight-loss studies described below.

Comparison of model results with longitudinal weight-change data

We compared the various model calculations for \( \Delta B W \) with longitudinal weight-change data that were achieved over periods of weight loss ranging from 1 to 14 mo. The whole-body EE rates were accurately measured during periods of weight stability both before and after weight loss either by using the doubly labeled water method (8, 9, 12–15) or by titrating the food intake in an in-patient setting to achieve weight stability for \( \geq 2 \) wk (10, 11). Because the measurements were made during periods of weight stability, we assumed that the EE rate equaled the dietary EI rate. Therefore, the measured changes of EE after weight loss gave an accurate estimate of the dietary EI changes required to maintain the measured body-weight change and prevent regain.

To provide all of the parameter values used in the above calculations, we required measurements of the initial body FM and the PAL. We found 8 longitudinal weight-loss studies, representing 157 patients, that satisfied these criteria, in which a wide range of weight losses were induced either by bariatric surgery (9, 13, 15) or by diet restriction (8, 10–12, 14). The parameters for each study are shown in Table 1.

We evaluated each model in comparison to the weight-change data by using the following measures. First, we calculated the Pearson correlation coefficient \( (r) \) between the model calculations and the data according to the following equation:

\[ r = (N - 1)^{-1} \sum_{i=1}^{N} (y_i - \langle y \rangle)(m_i - \langle m \rangle) \quad (11) \]

where \( y_i \) was the model calculation corresponding to the measured value \( m_i \) for each group and the brackets \( < \) and \( > \) denote the mean value. Second, we calculated the chi-square value, which is a weighted measure of the distance between the model calculations and the data according to the following equation:

\[ \chi^2 = \sum_{i=1}^{N} (y_i - m_i)^2/\sigma_i^2 \quad (12) \]

where \( \sigma_i \) was the SD of the measurement. The chi-square value was our primary measure of model fit to the data. Using the incomplete gamma function, we computed the probability that the chi-square for a correct model should be less than the chi-square calculated for the model (26). Finally, we calculated the coefficient of determination according to the following equation:

\[ r^2 = \frac{\sum_{i=1}^{N} (y_i - \langle y \rangle)(m_i - \langle m \rangle)^2}{\sum_{i=1}^{N} (y_i - \langle y \rangle)^2 \sum_{i=1}^{N} (m_i - \langle m \rangle)^2} \]

### Table 1

<table>
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<th>Data and parameters used for model comparisons in Figures 1 and 2</th>
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<td>van Geemen et al (13)</td>
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<td>Westerterp et al (14)</td>
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<td>Westerterp et al (15)</td>
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**Note:** All such values refer to the change in the steady-state total EE rate measured in the longitudinal weight-loss studies described below.
\[ R^2 = 1 - \frac{\sum(y_i - m_i)^2}{\sum(m_i - \bar{m})^2} \] (13)

Because the models are generally nonlinear, the coefficient of determination \(R^2\) is not identical to the correlation coefficient squared.

The model parameter \(\beta\) was determined by using a weighted least-squares optimization procedure to find the best fit value of \(\beta\) that corresponded to the weight-change data listed in Table 1 (26). To calculate the uncertainty of our estimate of \(\beta\), we performed a Monte-Carlo analysis in which we re-fit \(\beta\) with the use of 5000 sets of synthesized data with the same statistics as the real data. In other words, each synthesized data-point was randomly selected from a normal distribution with a mean and SD corresponding to a real data-point. The uncertainty of the model parameter \(\beta\) was then calculated as the SD of the 5000 best-fit \(\beta\) parameters from the Monte-Carlo simulations. All model calculations and statistical comparisons were performed with the use of MATLAB software (version R2008a; The MathWorks Inc, Natick, MA).

RESULTS

Comparison of results from the proposed model with longitudinal weight-change data

A comparison of results from our model with the measured changes of steady-state body weight given the measured changes of EI and physical activity are plotted in Figure 1. For our proposed model, the best-fit value for the parameter \(\beta\) was 0.24 ± 0.13. Statistical evaluation of our model showed a Pearson correlation coefficient of 0.983 between our model’s body-weight calculations and the measured values. The coefficient of determination was 0.83, which indicated that our model described 83% of the data variability. The chi-square value was 2.08 and, according to an evaluation of the incomplete gamma function, the probability was <0.01 that the chi-square for a correct model should be less than the chi-square calculated for our model (26).

Our model results are compared (Figure 1B) with the measured changes in EI rate required to maintain the average measured weight loss observed in each study \((R^2 = 0.91)\). In this case, the chi-square value was 0.87, and the incomplete gamma function gave a probability of \(3.1 \times 10^{-4}\). Therefore, our model provided an excellent match to the data over a wide range of changes in body weight (Figure 1A) and dietary intake (Figure 1B), which suggests that our model successfully captured the salient physiologic changes.

The comparison of the predicted and the measured fat fraction of the weight loss is plotted in Figure 2; and \((R^2 = 0.90)\) indicates that our model accurately described the observed body-composition changes. There was a wide range of observed body-composition changes, which were due to the different initial body compositions and the range of weight losses in the various studies (22). For example, the initially lean subjects from the Minnesota starvation experiment of Keys et al (10) mostly lost FFM, and only 34% of their weight loss was accounted for by a loss of body fat. In contrast, body fat loss accounted for 84% of the weight loss in the in the obese subjects studied by Leibel et al (11) who reduced their weight by 10%.

Greater steady-state weight change is predicted for persons with higher initial body fat

The fact that our model accounts for variations in body-composition change leads to an interesting result when comparing the calculated steady-state weight changes in people with different initial body weights. The predicted steady-state body-weight changes as a function of the diet change for 2 example subjects are illustrated in Figure 3: the solid line represents the first subject, who corresponds to an average participant from a study by Martin et al (12), and the dashed line represents the second obese subject, who corresponds to an average participant.
can also be used to investigate the sensitivity of the model calculations to changes in parameters, much as were illustrated in Figure 3, where we examined a range of values for the parameter \( \beta \).

### Comparison with previous steady-state weight-change models

The residuals between the various model calculations and the measured body-weight changes for each group of subjects are compared in Figure 4. Our proposed model performed better than the other models because the residual body-weight changes were closer to zero and less spread out than were those in the other models. Whereas the model by Christiansen et al (16) resulted in a respectable correlation coefficient of 0.855 and an \( R^2 \) of 0.64, it had a high chi-square value of 20.9 and a resulting high probability of 0.98 that the chi-square for a correct model would be less than the chi-square calculated for their model. The model by Kozusko (18) resulted in a correlation coefficient of 0.722 but had a poor \( R^2 \) (ie, 0.067) and a high chi-square value of 19.1, which gave a probability of 0.98 that the chi-square for a correct model would be less than the chi-square calculated for his model. The predictions based on the IOM-NAS equations (25) resulted in a correlation coefficient of 0.60, but a very poor \( R^2 \) (ie, −1.79), which indicated that the mean of the data provided a better description of the body weight change than the predictions from the IOM-NAS equations. Correspondingly, the chi-square value of 184 gave a probability of 1 that the chi-square for a correct model would be less than the chi-square calculated for the IOM-NAS equations.

### DISCUSSION

Current methods estimate body weight loss on the basis of simplified rules such as “3500 kcal = 1 pound” (5, 29). Because such rules do not account for changes in EE with weight loss, they do not allow for stabilization of body weight at a new steady state despite continued adherence to a lifestyle intervention. Unfortunately, this crude approximation currently is the only widespread

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![FIGURE 2. Predicted versus measured changes (Δ) in the fat fraction (FM) of body weight (BW) loss (ΔFM/ΔBW) (\( R^2 = 0.90 \)). The dotted lines are lines of identity, and error bars are SEs.](image)

![FIGURE 3. Predicted diet-induced weight losses (ΔBW) as a function of dietary intake changes (ΔEI) for 2 subjects with very different initial body weights corresponding to average data from Martin et al (12) (—, initial BW = 82.3 kg) and Das et al (9) (- - - , initial BW = 139.5 kg). Δ, change; BW, body weight. The dotted lines indicate the sensitivity of the model predictions when the parameter \( \beta \) was changed by ± 1 SD.](image)
TABLE 2
Predicted body weight (BW) changes in 40-y-old sedentary men (PAL = 1.4) of average height (1.77 m) assuming no change of physical activity (Δδ = 0)

<table>
<thead>
<tr>
<th>Change in energy intake (kcal/d) kg</th>
<th>BW&lt;sub&gt;ini&lt;/sub&gt; (kg)</th>
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<sup>1</sup> Blank cells indicate that the prescribed intake changes would result in a BMI (in kg/m<sup>2</sup>) < 18.5. PAL, physical activity level; δ, physical activity parameter; BW<sub>ini</sub>, initial BW.

Clinical tool available for predicting weight change, and any improvement represents a significant step forward.

We addressed this problem by using the available longitudinal weight-loss data to develop a mathematical model that accounts for reduced energy requirements at the lower steady-state body weight. Mathematical modeling of human-body-weight change has been attempted many times and involves solving differential equations that model the rate of body-weight change (16, 18, 30–43). However, these mathematical models typically require specialized software to numerically approximate the equation solutions via computer simulation. In contrast, we solved our nonlinear model equations at the new steady state and provided standard spreadsheet files so that readers can easily perform the calculations for themselves (see Spreadsheet files under “Supplemental data” in the current online issue; the spreadsheet files and an online version of the model are available at http://www2.niddk.nih.gov/NIDDKLabs/LBM/lbmHall.htm).

Previous mathematical models of human-body-weight change have been used to explicitly calculate the steady-state body-weight change for prescribed changes in diet or physical activity (16–18). In the present study, we showed that, whereas 2 of the previous steady-state models provided a reasonably good correlation with the data (16, 18), our model performed significantly better with a chi-square value one-tenth that of the previous models.

The predictions based on the IOM-NAS EE equations were surprisingly poor, and they indicated that these cross-sectional equations should not be used to predict weight change. This conclusion is in contrast to the recent study by Heymsfield et al (17) that reported good agreement between the IOM-NAS equations and the measured EE rate of formerly obese subjects. However, most of the analysis performed in that study involved cross-sectional comparisons. Only 3 longitudinal data sets were used by Heymsfield et al, and their analysis pooled these data and did not report information about variability of the results or model statistics. Our analysis included these same 3 longitudinal data sets, but the data were not pooled, and we included 5 additional longitudinal weight-loss studies. Therefore, we believe that the existing longitudinal weight-loss data do not support the use of the cross-sectional IOM-NAS equations to predict weight change.

Our model was also used to calculate the dietary EI changes required to maintain a given amount of weight loss and to prevent weight regain. This important question has been previously addressed by Heymsfield et al (44) in another study using the IOM-NAS cross-sectional EE equations. However, given the poor performance of these equations in predicting steady-state weight change, we do not recommend their use for predicting changes of EI. Furthermore, the assumed linear dependence of EE on body weight implies that a specified decrement in dietary intake leads to the same weight change regardless of the initial body weight or body composition. This result ignores the facts that body-composition changes are likely to be nonlinear functions of the initial fat mass and the magnitude of weight loss (22, 23) and that FFM contributes to EE to a greater degree than does body fat (19, 45). Our proposed model incorporates nonlinear changes in body composition with weight loss, and Figure 2
TABLE 3
Predicted long-term body weight (BW) changes in 40-y-old sedentary women (PAL = 1.4) of average height (1.63 m) assuming no change in physical activity (Δδ = 0)\(^1\)

<table>
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\(^1\) Blank cells indicate that the prescribed intake changes would result in a BMI (in kg/m\(^2\)) < 18.5. PAL, physical activity level; δ, physical activity parameter; BW\(_{init}\), initial BW.

shows that the resulting model predictions for body-composition change match the data quite well.

Further illustrating the importance of this effect, Figure 3 showed that different initial body compositions can lead to different degrees of weight change for the same reduction in dietary intake. In our model, higher initial body fat leads to greater predicted weight change for an equal decrement in dietary EI.

This can be shown mathematically by considering the simplified case in which the fraction of weight lost as body fat is specified by a parameter (Φ). Equation 3 can then be rearranged to obtain the following equation for the expected weight change:

\[
\Delta BW = \frac{(1 - \beta)ΔEI - BW_{init}Δδ}{\gamma_{FM} + δ_{init} + Δδ - (γ_{FFM} - γ_{FM})Φ} \tag{14}
\]

Assuming that there is no change in physical activity (i.e., Δδ = 0), the predicted steady-state weight change per unit reduction of EI varies with the parameter Φ according to the following equation:

\[
\frac{∂}{∂Φ} \left(\frac{ΔBW}{ΔEI}\right) = \frac{(γ_{FFM} - γ_{FM})(1 - \beta)}{(γ_{FFM} - γ_{FM})Φ + δ_{init}} > 0 \tag{15}
\]

This quantity is greater than zero for γ_{FFM} > γ_{FM}, so the expected weight loss is enhanced as the fraction of weight lost as body fat, Φ, increases. Because the Forbes body-composition theory states that Φ is an increasing function of the initial body fat, persons with higher initial body fat will eventually lose more body weight for a specified decrement in dietary EI.

At first glance, this result appears to be contradictory to our previous study showing that the required energy deficit per unit of weight loss is higher in obese than in lean persons (29). The physiologic explanation derives from the fact that persons with higher initial body fat lose a smaller proportion of their metabolically expensive FFM and are thereby better able to preserve their total EE rate during weight loss. In contrast, an initially lean

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**FIGURE 4.** The difference between the predicted and measured weight losses (ΔBW) are plotted for each mathematical model. Δ, change; BW, body weight. Each data-point compares the model prediction with the mean value for each group of subjects.
individual will lose a greater amount of FFM, which concomitantly decreases the EE rate to a greater degree. Although a greater cumulative energy deficit is required per unit of body weight lost by persons with higher initial body fat (29), this energy deficit will be more readily achieved by persons with higher initial body fat, because the metabolically expensive FFM is spared, whereas the metabolically more inert but energy-dense FM is lost.

Some studies suggested that, after the steady-state body weight has been reached, no adaptation of EE occurs beyond that predicted by body composition change alone (8, 9, 14, 46–52). Others maintained that changes in EE after weight loss cannot be accounted for by body composition changes (11–13, 15, 20, 53–57). Many of these previous studies evaluated resting metabolic rate in the overnight fasted state by using indirect calorimetry, which accounts for only a fraction of the total daily EE, or 24-h EE in a metabolic chamber, which does not represent the free-living situation.

In contrast, the present study used the available doubly labeled water data (8, 9, 12–15)—the gold standard for measuring free-living total EE (58)—to model changes of total free-living EE after a new steady-state body weight was achieved. The best fit value of the unknown parameter $\beta$ (ie, 0.24 ± 0.13) was clearly greater than zero and also $>0.1$, which is the typical value used to represent the thermic effect of feeding (11). This indicates that some degree of adaptation of total EE beyond that expected from body weight change alone was required to explain the experimental observations. We do not propose a physiologic mechanism for such an adaptation. But when using our simplified model of EE rate, with body FM and FFM as the only independent variables, an additional term related to the EI change was required to adequately represent the data.

We do not want to leave the reader with the impression that our model can make precise calculations of weight change or the required lifestyle changes to prevent weight regain by individual subjects. The variability in the weight loss data would suggest that precise calculations would be very difficult with the use of a general model whose parameters are not adjusted for individual subjects. Prospective evaluation of our model using individual subjects, with the possibility of developing personalized models of weight change, will be the subject of future research.

Nevertheless, given the current inability to make any quantitative estimates regarding the expected level of body-weight stabilization or the required lifestyle changes to prevent weight regain, our model represents a significant step forward that we believe is useful for setting goals before an obesity intervention. It is important to emphasize that the present study has not dealt with the time-course of weight loss. If the calculated intervention for maintaining a desired weight change was implemented at the onset of obesity treatment, it would likely take several years for the body weight to reach the steady state (59). Therefore, it may be beneficial to partition an obesity intervention into a weight-loss phase followed by a weight-maintenance phase. The model proposed here would be most useful for helping define the dietary and physical activity changes required for the weight-maintenance phase—a problem for which no clinical tool is currently available.

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