MODELING ALTERNATIVE MOTIVES FOR DIETING

By

Robert S. Goldfarb, Thomas C. Leonard and Steven Suranovic

I. INTRODUCTION

A recent and growing literature considers the economics of weight change and obesity. The leading questions have been "what accounts for the observed rise in obesity over time?" [Chou et al 2002, Lakdawalla and Philipson 2002, Cawley et al 2003, Cutler et al 2003] and, "why do people (especially rational agents) choose to be overweight?" Because body weight can be adjusted by diet and exercise, "obesity is an avoidable state," and "economists expect these adjustments in behavior to take place if the benefits of adjustment exceed the costs" [Philipson [2001, p.1].

But many overweight people prefer not to be overweight, as the existence of a sizable diet industry suggests. Americans pay $40-$100 billion annually to help them lose weight. The Wall Street Journal recently reported that "at any time, 29% of men and 44% of women are on a diet." [Parker-Pope 2003, p. R-1]. Even if these estimates are high, it is hard to gainsay the fact that millions diet, with the aim of losing weight.

This paper asks "*why* do people diet?" The proximate answer is "to lose weight." But because there are different ways by which a person becomes heavier than he wants to be, the ultimate causes of the decision to diet are different. This has theoretical and empirical implications that we explore with a simple graphical model that determines desired weight, and shows how different causes induce dieting.[1]

The paper proceeds as follows. Section I discusses the physiology of weight determination. Section II sets out a plausible list of diet causes. Section III analyzes these alternative causes with a general production function/utility framework. A simple graphical exposition shows how an individual's optimal weight is determined, yielding propositions about "optimal overweightedness." Section IV shows how several of the causes of dieting identified in Section II can be usefully analyzed using this graphical framework. Section V presents conclusions about implications, applications suggested by the analysis, and possible extensions.

I. BACKGROUND: THE PHYSIOLOGY OF WEIGHT DETERMINATION

The production function for weight determination begins with the view that weight gain results when energy (calorie) intake exceeds energy use. Calories are expended in exercise, digestion of food, and "basal metabolic rate"(BMR), the energy the body expends when at rest. Basal metabolism is in fact the largest source of energy expenditure. A standard result in the physiology/nutrition literature is that BMR declines with age.

That metabolism slows with age suggests the following proposition: *if an individual maintains the same level of calorie intake and exercise as he or she ages, that person will gain weight*. This happens because energy (calorie) intake is constant, but energy use declines. Indeed, in Suranovic-Goldfarb-Leonard 2002 [hereafter SGL2002], we harness a widely-used empirical relation from the physiology literature (the"Harris-Benedict equations") to obtain numerical estimates of the decline of BMR– and therefore calorie expenditure – with age.[2] These estimates are then used to generate weight-change scenarios.

The proposition that weight will rise with age even with constant calorie intake is consistent with evidence that weight **does in fact rise with age.** Costa and Steckel [1997, p. 55] examine body-mass index by age (from age 19 to 72) for a number of cross-sections from 1864

through 1991.[3] The 1991 cross-section, for example, shows body mass index rising from between 23 and 24 at age 18-19 to between 26 and 27 at age 50-64, then falling to a little below 26 at age 65-79.[4] Cutler et al [2003] also find that weight increases with age up to an age between 50 and 55.[5]

These empirical findings about weight gain with age, and the underlying contribution of falling BMR, provide important information for our modeling of weight choice and the incentive to diet.

II. A TAXONOMY OF CAUSES OF DIETING

People diet in an attempt to lose weight, but there are varied causes of perceived overweightedness. Understanding possible motives for dieting seems an important step in deepening our ability to analyze dieting phenomena both theoretically and empirically. In this section we provide a provisional taxonomy of diet causes, including: (i) "aging-associated" dieting; (ii) "disease-provoked" dieting; (iii) "physical-life-events-provoked" dieting; (iv) "style-provoked" dieting; (v) "smoking-cessation" dieting; and (vi) "innovation-provoked" dieting. Brief elaborations follow.

(ii)**"aging-associated" dieting.** This kind of dieting stems from the fact, discussed in the previous section, that weight increases with age, given constant calorie intake. The age-associated weight gain may create incentives to diet. This motive is investigated in our previous paper [SGL 2002].

(ii)**"disease-provoked" dieting.** This kind of dieting stems in the most extreme case from what might be called the "diet or die" motive. An individual is diagnosed with a medical condition requiring that he lose weight to reduce threats to health or even life.[6] [7]

(iii) **"physical-life-events-provoked" dieting.** This category refers to episodic physical events that create what the individual may perceive to be weight disequilibria, a departure from the weight that the individual views as desirable. An example is weight gain from pregnancy that remains post-pregnancy. A diet may be undertaken to lose this "extra" weight.

(iv) **"style-provoked" dieting.** There appears, at least anecdotally, to be a pattern whereby some people begin in the spring to shed winter pounds in order to be able to fit into bathing suits. This is stylistic dieting which is on a seasonal/yearly cycle. There is another kind of stylistic dieting which is not cyclical, but episodic: dieting in preparation for such life ceremonies as family weddings or alumni reunions.

(v) **"smoking-cessation" dieting.** Nicotine may suppress appetite, so those who quit smoking sometimes gain weight. This weight gain can induce dieting.[8]

(vi) **"innovation-provoked" dieting**. Knowledge innovations, including new diet drugs, new diet methods, and improvements in knowledge about the costs of overweightedness, may all lead to "innovation-provoked" dieting.

We next develop a quite general production function/utility framework capable of encompassing a variety of these reasons. We note in passing that one of the interesting features of this list of alternative provocations is that the diets identified differ in their likely patterns over time. Some are episodic, others are yearly-cyclical, while still others may well vary secularly as the individual ages.

III. A GRAPHICAL PRODUCTION/UTILITY FRAMEWORK FOR CONCEPTUALIZING WEIGHT EQUILIBRIUM AND DIETING

An analytical framework for considering weight determination and dieting decisions requires two elements: the determinants of the "production of weight," and the determinants of

the utility of different weight levels. We develop a simple graphical analysis embodying these "production/supply" and "utility/demand" features. This graphical analysis generates a mechanism for describing a weight optimum, one with some striking features, and shows how reasons for dieting can be analyzed.

Determinants of the production of weight.

As noted, the physiology literature predicts weight change when calorie intake differs from calorie expenditure. Therefore, the determinants of weight change are those that affect calorie intake, calorie expenditure, or both.

Basal metabolism, the largest component of energy expenditure, is itself affected by physical characteristics: lean muscle mass, weight, height, age and so forth. Moreover, the Harris-Benedict equations indicate that the effect on basal metabolism of weight, height and age is different for men and women, a point also made by Cutler et al [2003].[9] Exercise levels will be affected by the physical strenuousness of work and leisure activities. Calories expended from exercise are also believed to decline with age. [10]

Factors affecting calorie intake include appetite and its determinants, the calorie density of available foods, the physical/psychological ability to maintain a "diet," (that is, a calorie cutback) and the individual's information regarding calorie intake and expenditure. The determinants of appetite are not well understood. [11] Some drugs are known to stimulate or suppress appetite, and the weight gain that often follows smoking cessation is consistent with the idea that nicotine suppresses appetite. [12] Some factors might affect both energy use and energy intake. Exercise expends calories and may influence appetite, thus calorie intake. Pregnancy and some illnesses also can affect calorie intake and expenditure simultaneously.

Some important features of these relationships are summarized in Figure 1. Consider the EH -- for "high energy expenditure"-- curve. It represents the physical ("production") relationship between food and weight, holding energy use constant. For a given level of energy use-- as determined by the individual's lifestyle--the higher the level of food intake each period, the higher the maintainable weight. The low-energy expenditure (EL) curve is drawn lower because a lower calorie expenditure implies, for the same level of calorie intake as on EH, a higher maintainable weight. The curves are linear because the Harris-Benedict equations indicate that BMR rises linearly with weight. Each curve has a positive intercept because the Harris-Benedict equations themselves contain a positive intercept, and in addition indicate that BMR use depends on age and height as well as weight. Thus, individuals of different heights would have different intercepts, and the intercept would shift down as age increases. The EL curve is drawn with a lower slope than the EH curve based on the specification of energy use equations in SGL [2002]. They multiply the Harris-Benedict equations by coefficients of energy use taken from the physiology literature: the coefficient of a "moderate exercise" lifestyle is higher than that for a "low exercise" lifestyle, and so forth.

Determinants of the utility of various weight levels.

Faced with these underlying weight-production relationships embodied in the series of E curves such as EH and EL, where on that production surface does the individual choose to be? This will depend on the utility associated with various weight levels.

Disutility associated with weight different from a "desired" level falls into three categories: negative health effects, negative appearance effects, and increases in "task costs." Negative health effects include the lowered life expectancy associated with excess weight, poorer health while living, and increased costs of health care. Appearance effects can be internal,

involving one's own dislike of one's own body image, or external, generated by the reactions of others to the individual's "nonideal" weight. These external "social interaction" effects would include (but are not limited to) job effects such as lower wages [see for example, Cawley 2003] or difficulty in obtaining employment, and social disapprobation more generally. Increases in "task costs" include daily life annoyances such as increasing difficulty in finding clothing that fits, trying to squeeze into airline seats designed for thinner individuals, the increased difficulty of performing physical activities such as climbing stairs, gardening, and so forth.

Some important features of these relationships between weight and utility are summarized by the three indifference curves U_1 , U_2 , U_3 in Figure 1. [13] To understand why these indifference curves are drawn U-shaped, consider the case of an individual who gets utility from food consumption, and also has a weight level he considers most desirable. This most-desirable weight level W^* depends in turn on the kinds of health, appearance and task cost considerations discussed above; to emphasize this dependence, we call W^* the "desired health-and-appearance weight."

Since this individual views W^* as the most "health and appearance desirable weight," consider the situation if his actual weight W_a is $< W^*$. In this case, an added pound, bringing him closer to W^*, is a good. If, instead, W_a is $> W^*$, added pounds (moving him further away from W^*) are bads. This implies that this individual's indifference curves between food (F) and weight are U-shaped, with the minimum point of each indifference curve being at W^*. The several indifference curves in Figure 1 display this shape.

The indifference curves in Figure 1 include the additional feature that, along any vertical line to the right of W^*, the indifference curves get steeper as F rises. This incorporates the idea that the marginal utility of food is decreasing as F rises.[14]

An additional constraint: income.

Besides the food-weight production relationship embodied in curves like EH and EL, there is also an income constraint. The horizontal lines at F_1 and F_2 in Figure 1 show two possible income constraints, the maximum F allowed by the individual's budget. As we explain below in discussing equilibrium, the income constraint F_1 is drawn in Figure 1 so it is non-binding, while F_2, if it applied, would be binding. [15]

Equilibrium with "optimal overweightedness".

Suppose this individual's lifestyle generates energy use level EH, and that he faces the income constraint at F_1 (so the F_2 constraint does not apply). Then his utility maximizing weight choice will be at point B in figure 1, with weight level W_E. Point B with weight level W_E gives the highest utility level achievable along the (binding) EH constraint. At B the level of food consumption is below F_1, so that income constraint (the horizontal line at F_1) is not binding.

So W_E is the optimal weight choice, given the constraints that this individual faces. But W_E is clearly greater than W^*, this individual's most health-and-appearance desirable weight. That is, this analysis *predicts* that, if the income constraint is not binding, the typical individual will *choose a weight above his health-and-appearance desirable weight* W^*. That is, overweightedness--meaning weight above the weight level that is most desirable based on appearance and health concerns-- will be utility- maximizing! That is, there will be ***"optimal overweightedness,"*** a result also derived by Levy [2002] from a much more mathematically complex model and analysis. [16]

Equilibrium with a binding income constraint.

Now suppose instead that the income constraint at F_2 applies instead of F_1. Then the equilibrium point will be at D in Figure 1, with optimal underweightedness. That is, weight

choices below W^* in this version of our model only arise because of a binding income constraint. [17]

An additional modeling twist: "food becoming a bad" produces circular indifference curves and "optimal underweightedness."

Both modeling logic and empirical concerns suggest the usefulness of considering what happens if food also becomes a "bad" at some high level of consumption. The modeling logic is simply that food can in fact become a bad--just as weight does--above some satiation limit. The empirical concern is that there are people who are not income constrained but nonetheless view themselves as underweight--that is, below their health-and-appearance desirable weight--but cannot manage to gain enough weight to reach that level.

To see how this might happen, consider an actual example. A friend of one of the authors told us that his daughter, an athlete, views herself as underweight (a view the friend agrees with), but is "unable" to eat enough to get her weight up to the level she wants. Because she is an athlete, she has a relatively high EH curve. Cutting back on athletic activity, thereby shifting her EH curve, does not seem to be an option she wants to adopt.

Incorporating the idea that food intake becomes a bad at some satiation consumption level F_s results in the circular indifference curves U_1 and U_2 displayed in Figure 2. Above F_s on the vertical axis food is a bad. To the *left* of W^* and above F_s, weight is a good and food is a bad, so indifference curves are upward sloping. To the *right* of W^* and above F_s both weight and food are bads, so the indifference curves become downward-sloping again. Below F_s the indifference curves are U-shaped, just as they were in Figure 1. The overall result is circular indifference curves. [18]

With circular indifference curves and a nonbinding income constraint, a "steep enough" EH curve such as the one shown in Figure 2 will generate an equilibrium point W_L to the left of W^* and above F_s. This person is "optimally underweight." She is consuming a level of F that is above her satiation level because she would like for health and attractiveness reasons to weigh more than she does, but the marginal disutility of more food exceeds the potential gains from realizing a higher weight. And like our athlete, reducing calorie expenditure from athletic activity is an option she is unwilling to choose.

For the remainder of our analysis, we restrict ourselves to the U-shaped indifference curves of Figure 1. We are concerned with analyzing diet motives, and optimally-underweight individuals are not diet candidates. Instead, as we will see, it is individuals with equilibria on the rising segment of the U-shaped indifference curves who will be candidates for dieting.

IV. GRAPHICAL ANALYSIS OF REASONS FOR DIETING

An advantage of this graphical framework is that it shows how the effects of several of the reasons for dieting can be usefully conceptualized. We consider first disease-provoked dieting, then aging- provoked dieting, then "physical-life-events-provoked" dieting, then "style-provoked" dieting.

Disease-provoked dieting.

Consider the "diet or die" scenario, yielding disease-provoked dieting. In this scenario, the individual discovers that his health is threatened by his current weight situation. In terms of the graphical model, this can usefully be thought of as a shift in the individual's desired health-and-appearance weight level W^* (which will cause his optimal weight W_E to also change). The "diet or die" news represents new information about the relation between weight levels and health, causing W^* to shift down.

How does this affect the indifference curves portrayed in Figure 1? Since the new health information shifts W^* to the left, it shifts the minimum points of all of the U-shaped indifference curves to the left. The new equilibrium weight W_{EN} will be below the old equilibrium weight W_E, at a tangency between an indifference curve and EH, "lower down" on EH, with a new lower level of F. [19] This implies that the individual finds it desirable to reduce weight and food intake. That is, that the individual **will consider dieting**.

We say "will consider dieting," rather than "will diet" because, as analyzed in SGL 2002, there are adjustment costs associated with a diet, so that the individual needs to perform his own cost-benefit analysis of actually engaging in a diet. [20] This point is just one example of a general feature of comparative static analysis as embodied in our graphical framework. The comparison of W_{EN} and W_E is a comparison of static equilibrium positions. The problem of how (and whether) one gets from one comparative static equilibrium position to another is a problem involving dynamics, and cannot be solved by these comparative statics.

This graphical analysis of the disease provocation for dieting illustrates a very important general conceptual result about the economic interpretation of circumstances provoking dieting. *Diet provocations often involve changes in circumstances ("shocks") that shift the individual's equilibrium level of weight and food intake.*

Aging-associated dieting.

Disease-provoked dieting involved shifts in indifference curves. Aging-associated dieting instead involves shifts in the weight production curve. Specifically, this kind of dieting is associated with shifts downward in EH (and in every E curve) as basal metabolic rate falls with age. It can also involve shifts from an EH to an EL curve, should exercise also decline with age.

Consider Figure 3, which shows two EH curves. The higher curve, EH_Y, is the weight production relationship at a younger age. The lower curve, EH_S is the relationship at an older, more "senior" age. The Harris-Benedict equations suggest that the curve's slope does not change, but the intercept of the curve shifts down with age. [21]

The parallel downward shift in the EH equation to EH_S results in a new tangency--a new optimal weight W_{EN} at point C on a lower indifference curve. However, because (as explained in Section II above) the indifference curves have **rising** slopes along a vertical line as *F* rises, the new optimal weight W_{EN} **is higher than the previous optimal weight** $\boldsymbol{W_E}$. Since the individual's weight if he maintains his old *F* level will be at point G, he has an incentive to diet to get to C. If in addition the individual's exercise level falls as he ages, the cut in food consumption *F* needed to attain the newly optimal weight level will be even larger.

A particularly striking aspect of this graphical analysis is its consistency with two results that might seem conflicting at first blush. Aging creates an incentive to diet, at the same time as it **raises** the individual's optimal weight! Put differently, even though the individual's optimal weight rises as he ages, he still must cut his food intake to reach that newly optimal higher weight. This has the important collateral attraction that it **predicts** what the data reveals--that weight rises with age. [22] [23]

Aging is a continuing dynamic process, but we are using comparative static analysis to proxy its effect. Since the individual is constantly aging, would he or she be constantly dieting? Recall our discussion above about the dynamics of moving from one equilibrium to another. Our previous paper focusing on aging-associated dieting [SGL 2002] postulated and modeled the adjustment costs associated with such dieting. That paper shows that, in the face of adjustment costs, diets would be sporadic rather than continuous. Intuitively, one would **only** undertake a

diet once there was a weight increase large enough that the disutility of the added weight was exceeded the adjustment costs of dieting. One reader suggested calling this the "enough is enough" weight gain condition for provoking a diet.

***Physical-life-events-provoked dieting*.**

Consider the case of postpartum weight gain from pregnancy. Consider Figure 3 once again, but ignore the line EH_S . One simple way to conceptualize this weight gain is to view the physical changes accompanying pregnancy as driving the individual's postpartum weight to some point like R along the EH_Y curve. [24] That is, pregnancy has driven the individual's postpartum weight away from the optimum. Nothing has happened to shift the indifference curves, so that after pregnancy the person's optimal weight is still W_E .This implies the incentive to diet to get from R back to point B and weight W_E .

***Style-provoked dieting*.**

We focus on yearly-cyclical dieting to fit into bathing suits, rather than on the episodic dieting associated with life events such as weddings. In terms of our graphical analysis, two complementary factors are likely to cause a shift in the equilibrium weight W_E as bathing suit season approaches. First, the desired health-and-appearance weight level W^* falls because the appearance payoff to a lower weight rises. This shifts the indifference curves to the left. Second, the real cost of energy use declines because it is now possible to exercise out of doors. This can result in a shift to a different E curve for the individual, an E curve representing a higher energy use.

The fall in W^* by itself would lower W_E . The movement to a new higher E curve (to the left of the previous E curve) would further alter W_E but would also allow a given weight cutback to be achieved with a less severe cutback in F. One implication is that the desired loss in weight

would require a larger reduction in food intake *F*, the smaller the shift in E. Indeed, a large enough rise in E might completely eliminate any need to cut *F*. To completely analyze this outcome, one would need to explicitly model the choice of exercise levels, and therefore of E, which our graphical analysis treats as exogenous (but see the comments about modeling exercise effects in footnote 16 above). [25]

One implication from comparing these applications.

The introduction to this paper claimed that differing reasons for dieting exist because there are different ways by which individuals become heavier than they want to be. The four reasons for dieting just analyzed show that different ways of becoming heavier generate different graphical implications. Disease-provoked dieting set off a shift in indifference curves, while aging-associated dieting instead set off a shift in the food-weight "production" constraint. The style provocation involved both a shift in indifference curves and a movement to a different food weight constraint. The pregnancy-related motive did not fit any of these three previous patterns.

We have shown how our graphical framework can be used to analyze four of the six provocations we identified in Section II above. What of the remaining two: innovation-provoked dieting, and smoking-cessation dieting? Innovation-provoked dieting is addressable by our graphical framework if the source of the innovation involves new health information (about, for example, health considerations affecting desirable weight). It is not addressable if the information innovation has to do with the nature of diets themselves, and choice among diets. In these latter cases, the innovation involves the choice of whether and how to move to a comparative static solution, a question of dynamics. It also involves the choice among types of diets, a choice our framework does not address.

How about smoking-cessation dieting? It is not yet clear whether our framework can capture the essential features of this reason for dieting. [26] The existence of this difficulty is a topic for further research.

An appendix available upon request considers the three previous papers [Dockner-Feichtinger 1993, Levy 2002 and SGL 2002] that have analyses of dieting in light of the framework just set forth.

VI. CONCLUSION

This paper has attempted to advance the economic analysis of dieting. Its contribution stems from two sources: the specification of a list of provocations for dieting, and the development of a general graphical framework for analyzing weight determination and dieting. This concluding section considers three topics: (i) empirical and conceptual gains from specifying a list of reasons for dieting; (ii) some useful results of the graphical framework; and (iii) research extensions suggested by the analysis.

Empirical and conceptual gains from specifying a list of diet provocations.

The specification of the list *by itself* brings out interesting issues and implications about over-life patterns of dieting, and about multiple dieting spells.

Over-life patterns of dieting. As previously mentioned, an interesting feature of the list is the differences in the time-patterns of dieting over life that they imply. Some provocations imply cyclical dieting patterns, some imply secular growth in dieting with age, some imply episodic dieting.

Multiple spells. Three previous articles about weight change and dieting [Dockner-Feichtinger 1993, Levy 2002, and Suranovic, Goldfarb and Leonard 2002] had focused on trying

to explain the existence of multiple diet spells. Our list of reasons for dieting allows several new inferences about how to analyze multiple spells.

First, the analysis produces **an entirely different source of multiple spells** from what the previous literature identifies. In that previous literature, multiple spells were generated by "single-cause" models of weight change. The list just offered, however, suggests that multiple spells might instead arise because the individual in question is subject to *more than one* of these dieting causes over time. For example, an individual might have had two dieting spells, the earlier one to diet for a wedding, the latter to lower his blood sugar.

Second, explaining analytically the existence of multiple diets or diet cycling was a major focus of the three previous papers **because cyclical diets were seen as a threat to the rational choice view of human behavior**. The argument is that multiple diets appear to be harmful to the individual, so why would a rational individual ever display such behavior? [27] But if multiple spells are from multiple causes, they may not in fact represent harmful behavior. Our individual above who had two spells, one of them dieting for a wedding, and the other to lower his blood sugar, would hardly be a candidate for the "harmful multiple spells" designation.

Third, suppose one sets out to identify the actual empirical incidence of multiple spells of dieting, in part to measure the extent of harmful dieting behavior. To make the inference that any multiple spells actually observed do in fact represent harmful behavior, one would need to identify the reasons for the multiple spells. If our two-spell individual above was in our data set, he would not fit the "harmful multiple spells" categorization.

Some useful results of the graphical framework .

Our graphical framework provides an intuitively attractive analysis of the determination of the individual's optimal weight. An additional attraction is that it yields the striking

prediction of "optimal overweightedness," a result gotten previously by Levy [2002] only at the cost of much greater mathematical complexity. Moreover, the graphical framework generates implications consistent with the empirical fact that weight rises with age, an implication that neither the Levy [2002] nor the Dockner-Feichtinger [1993] papers focusing on multiple spells were able to (and did not try to) derive. A particularly satisfying aspect of the model's analysis of aging-associated dieting is the consistency of its implications with two results that might seem conflicting at first blush. Even though the individual's optimal weight rises as he ages, he still must cut his food intake to reach that newly optimal higher weight. The framework also provides a method for identifying how different reasons for dieting affect the individual's optimal weight and food intake level. An unusual feature of the framework is its use of U-shaped indifference curves, and its extension to circular indifference curves.

Research extensions suggested by the analysis.

There are numerous additional aspects of weight change and dieting that the current framework does not adequately explore, some of which seem ripe for future modeling research. Here are six examples.

1.**.** Norms about desirable weight levels may change with age or life circumstance. Thus, for example, twenty-something individuals may have a different (more stringent) view of desirable weight than do forty-something individuals. A related idea is that single individuals looking for mates may have different views about desirable and acceptable weight levels than do older married couples. Such changes are relevant to models of aging-associated dieting and style-provoked dieting. Introducing changes in the desirable level of weight as the individual ages could affect the age-pattern of diets the individual might be predicted to undertake [in the SGL 2002 model, for example]. Similarly, if views about desirable weight changed as one's marital

status changed, a number of predictions about differing dieting patterns for those who married versus those who remained single might emerge.

2. In our graphical model above, energy use is treated as exogenous. But, as our analysis of style-provoked dieting suggested, one aspect of energy use, exercise level, is subject to individual choice. This choice needs to be explicitly modeled. We suggest in footnotes 16 and 17 a way of thinking about how to endogenize the exercise choice, and argued that doing so would not alter the kinds of equilibria obtained.

3. We have treated dieting as a matter of reducing caloric intake. However, recent changes in views about particular kinds of diets, such as the Atkins diet, suggest the possibility that changing the nutritional content of what one eats may be a way of reducing weight. The implications of these changing views about the weight implications of nutritional content provide an interesting possible extension of the model in this paper. An additional potential extension arises if foods of different nutritional content come with different prices "per calorie." This might make the budget constraint more complex than the horizontal line in our diagrams.[28] Another possible source of complexity in the budget line is suggested in footnote 15 above.

4.The changing views just mentioned are consistent with a further assertion, that uncertainty and imperfect information may play a sizable part in weight-determination decisions. As mentioned earlier in the paper, the determinants of appetite are not well-understood. This contributes to a situation in which there are competing diets, based on mutually incompatible beliefs about underlying physiological determinants of weight change. Our "innovation-provoked" motive for weight gain only exists because of severe limitations as to what is known about the physiology of weight change.

An interesting issue here is how diet failure and diet learning might affect diet choices. For example, the SGL 2002 model assumes that, having decided to cut calories by a specific percentage, the individual simply goes ahead and does so. Similarly, our graphical analysis assumes that weight equilibria will in fact be attained, even if cutting food intake is required. Three amendments to this "just do it" assumption can be imagined. A first amendment might involve recognizing that there is a nonzero probability of failing to stay on a planned diet. A second, related amendment, might recognize that diet trials may be subject to "learning-by-doing;"the first dieting attempt having failed, the individual learns from this very failure about ways to increase the probability of success. A third amendment would recognize the potential interaction between diet failure and exogenous diet innovations. Multiple diets might arise from this interaction. In particular, information innovations about new kinds of diets, new information about existing kinds of diets, or new diet drugs, might cause individuals to undertake multiple diets. Consider the following scenario. The first type of diet the individual tries, perhaps a commercial diet method, does not work. A new diet comes along, and the individual decides to try it, on the chance that it will work better for him than the previous method. That is, failure of the first diet, combined with the emergence of new diet methods, produces a "trial and error" pattern of multiple diets.[29]

5. There are several modeling literatures not focused primarily on dieting that generate approaches that might fruitfully be applied to dieting. For example, the multiple selves approach, [see, for example, Thaler and Shefrin 1981] and the Orphanides-Zervos [1995] rational addiction model, in which individuals face uncertainty about their propensity to become addicted, both have the potential to provide interesting analyses of disease-provoked dieting. Work stemming from the meta-preference approach, itself brought into economics by Amartya Sen [1977], raises

the issue of why individuals might regret their own eating habits even when indulging in them, and how this regret might provoke dieting. See, for example, David George [1998; 2001, especially pp. 10-11 and 109-113].

6. As mentioned in the introduction, much of the literature on weight change has focused on trying to identify factors contributing to the growth in obesity over time. One can ask how that growth might be explained using our graphical framework. It is easy to see one way in which our graphical framework can replicate one of the major suggestions in the existing literature. Lakdawalla and Philipson 2002 for example stress the effects of technological change in lowering the real cost of food consumption over time. In our Figure 1, this idea can be captured as a movement from the F_2 to the F_1 constraint; as food prices fall, the income constraint on food consumption becomes less constraining. This shift in the constraint results in a movement from equilibrium point D to equilibrium point B, producing an increase in the individual's equilibrium weight. This replicates the empirical observation of rising obesity over time. Additional ways in which the graphical framework might be used to generate secular increases in obesity remains a topic for future research.

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Figure 1

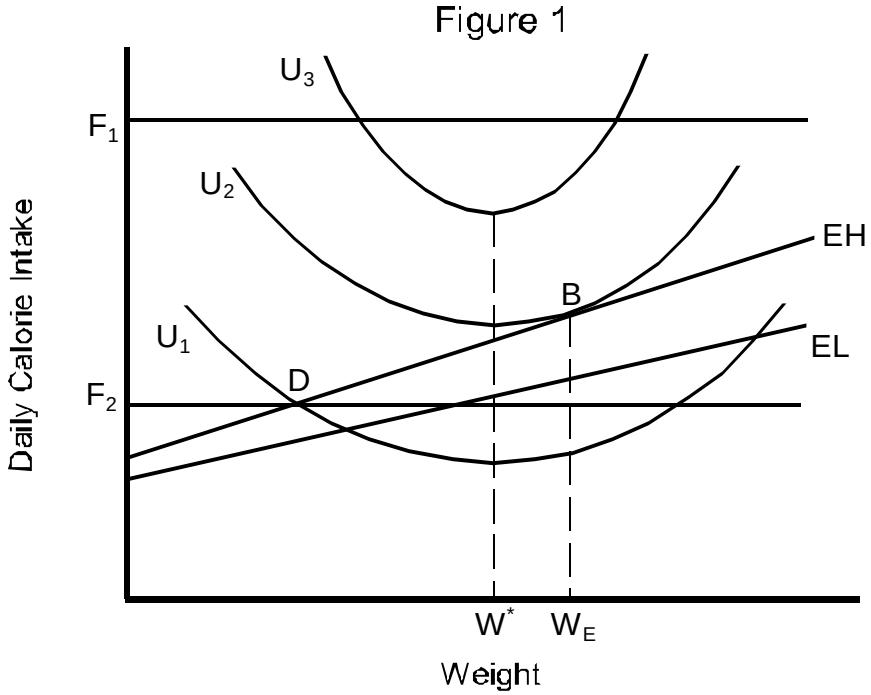


Figure 2

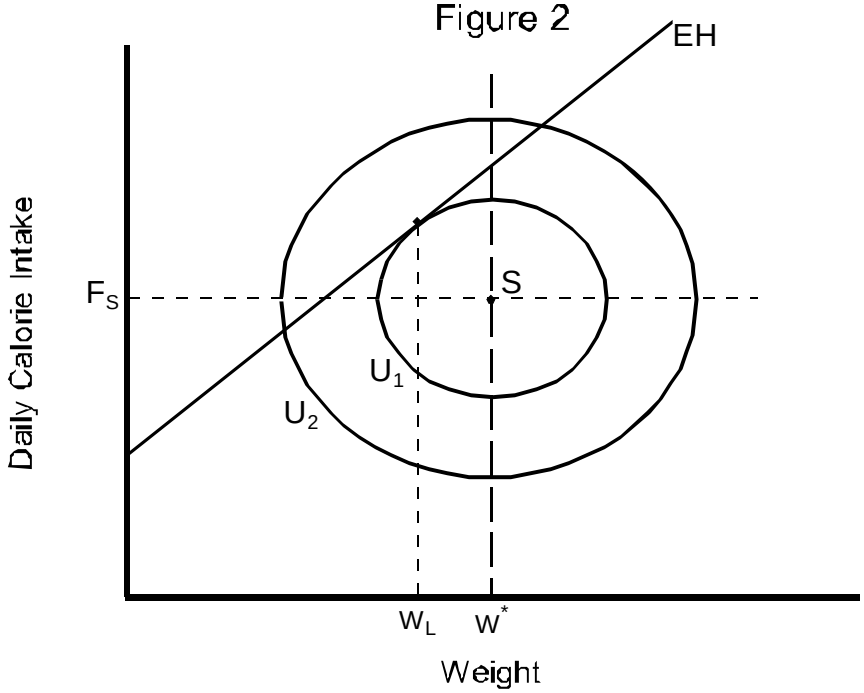
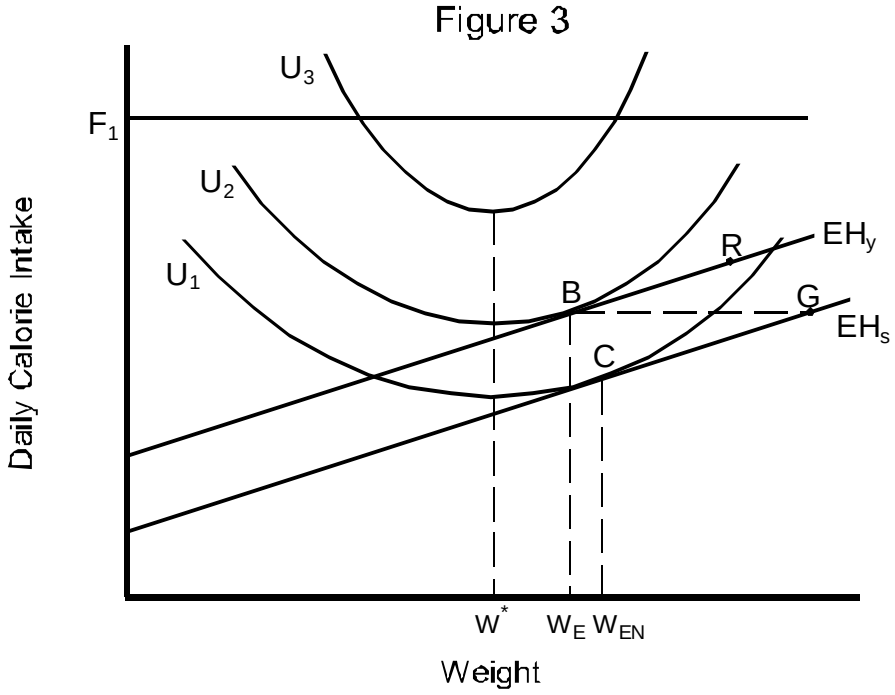


Figure 3



NOTES

The authors benefited from discussions with or comments from Bryan Boulier, Edwin Dean, Sara Markowitz, Don Parsons, Larry Promisel, and Herman Stekler while developing the analysis in this paper. Comments from two anonymous referees led to improvements in the paper.

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[1] There are a few theoretical papers that try to model both the choice of weight, and the decision to diet [see for example, Dockner and Feichtinger 1993, Levy 2002 and Suranovic, Goldfarb and Leonard 2002]. None of these papers consider multiple causes of dieting, and the first two have other serious shortcomings (discussed in passing below and extensively in an appendix available from us) as guides to developing a sound conceptual and empirical understanding of dieting. In addition, several of the papers in the empirical literature attempting to explain the growth in obesity contain modeling frameworks, but they do not focus on, nor do they predict or explain the existence of diets. See Lakdawalla and Philipson 2002, and Cutler et al 2003.

[2] For documentation of the claim that BMR declines with age, and citations to and an exposition of the Harris-Benedict equations, see the discussion and citations in SGL 2002.

[3] Body Mass Index, or BMI, is measured as weight in kilograms divided by height in meters squared. BMI between 25 and 30 is considered medically overweight, and BMI above 30 is considered medically obese.

[4] Costa and Steckel's focus is on showing the secular increase in body-mass-index over time *controlling for age*. That is, age is on the diagram merely as a control variable. The data underlying the 1991 age-weight results are for males, and are from the National Health Interview Survey. The 1961 data on the same graph seems to show a rising BMI from age 18-19 (BMI 23) to age 40-49 (BMI close to 26), then a slight decline to around 25½ at age 50-64, falling to 25 at age 65-79. The 1961 data is also for men, and is based on the National Health Examination Survey.

[5] The sample used in the Cutler et al regression includes data from the 1971-75 and 1988-94. The regression contains a dummy variable which takes the value 1 if the observation is from the later (1988-94) data set. Age and age squared are entered as variables. Both variables are statistically significant; age has a positive and age-squared a negative sign. The linear term dominates until around age 52 for males and age 54 for females.

[6] We became aware of this motive from comments by two colleagues at other universities, both of whom had undertaken serious diets that resulted in sustained weight loss. One of the accounts went as follows. The individual had had a very serious heart attack, and was told by his doctor to lose weight and keep it off "or else." Faced with the "or else," this individual undertook to change his eating habits, and succeeded in considerably reducing his weight, and keeping it down. The second account was quite similar, but the triggering event was a blood test--after a long period without such test information--that showed very high blood sugar. Since diabetes had

been a serious affliction of this individual's older male relatives, he decided to get his weight under control as a way of controlling blood sugar to ward off diabetes. Once again, the individual was quite successful at losing weight and keeping it off.

[7] A related phenomenon involves undertaking a severe diet not to react to a specific disease diagnosis, but as a general strategy to prolong life. Animal studies suggest that extremely restricted calorie intake, which stresses the body, is strongly correlated with greater longevity (Lane et al. 1996).

[8] This type of dieting could be included under category ii, disease-provoked dieting. We list it separately because it is of particular interest, both empirically and (as we will see below) analytically. In particular, it presents analytical puzzles not shared by other entries in category ii. Rather than sweeping these difficulties under the rug, we highlight them by creating this separate category.

[9] Cutler et al [2003] cites epidemiological literature from the mid-1980's as indicating that a simple linear regression of BMR on weight produces different intercept and slope coefficients by gender [Cutler, et al, Appendix, no page number].

[10] Whitney et al [1998] for example, note that "BMR begins to decrease in early adulthood...at a rate of about 2 percent/decade. A reduction in voluntary activity as well brings the total decline in energy expenditure to 5 percent/decade." (P. 263).

[11] Wilmore and Costill [1999] observe that how body weight is regulated is far from completely understood, and "has puzzled scientists for years." (p.666). For example, how does the body generate appetite "signals" that keep calorie intake in rough correspondence to energy

requirements? A New York Times article [Kolata, 2000] on scientific research into weight gain mechanisms noted the following. “Instead of endlessly rejiggering diets...a new wave of scientists is getting at the molecular causes of appetite and satiety, unraveling elaborate chemical pathways that control how much animals, including people, eat. Some of the newly discovered molecules are thought to keep body weight stable by controlling appetite, so that someone who eats a huge meal one evening will not have a voracious appetite the next morning.” (p. F-1)

[12] Evidence in Cawley et al [2003] suggests that people believe that smoking affects appetite. Their empirical work indicates that the probability of beginning to smoke is higher among adolescent females who are overweight, who report themselves as being overweight, or who indicate that they are trying to lose weight.

[13] Edwin Dean suggested the idea of this graphical formulation of the weight production/utility model, and the specific way we draw the indifference curves.

[14] Consider an additively separable utility function in F and W. Additive separability means that MU_F is a function only of F and MU_W is a function only of W. Along a vertical line in Figure 1, W--and therefore MU_W--is constant. Therefore the MRS along a vertical line varies with MU_F only. For $W>W^*$, MU_W is negative. Therefore MRS is the absolute value of MU_W / MU_F. If MU_F declines with higher F due to diminishing MU of food, then MRS (=absolute value of MU_W / MU_F) will *rise* as F rises. Thus the slope of each indifference curve rises as F rises along a vertical line to the right of W^*. To put this result more intuitively, as units of F add less and less utility, *more* units of F are needed to compensate for one extra W, so dF/dW must rise as F rises, holding W constant.

[15] Larry Promisel suggested an interesting elaboration on this simple income constraint involving the idea that weight above some critically high level W_{HI} would provoke costs associated with health problems. This would imply that the budget constraint would change shape at W_{HI} . This shape change might involve a parallel shift down, or a change from a flat to a negative slope, or both.

[16] A referee suggested that an individual may not choose the overweight equilibrium at point B if he could adjust his exercise level costlessly. In the extreme case, the EH line might shift up to the left until a corner solution with the income constraint *F* was reached. While making the choice of exercise endogenous is certainly desirable, it would in general preserve an equilibrium like B, and not result in an income-constrained corner solution. The reason is that changing activity levels away from habitual levels will have a utility cost; it is costly, especially, to raise one's level of exercise. Changing exercise levels would lower utility at all levels of food intake and weight. Thus the indifference curves would shift down if a higher activity level were chosen. Our figure 1 can be interpreted as the equilibrium which results AFTER all utility-costly but net-utility -raising increases in exercise have been adopted.

[17] Notice that point D will move to the right as the F_2 constraint shifts up. It is even possible that, if the F_2 constraint *just happens* to be tangent to an indifference curve at *W**, the income-constrained equilibrium would be at *W**. A referee suggested that an individual would in fact *always* be able to move to an income-constrained equilibrium at *W** by *simply cutting back* on his or her exertion ("exercise") level. This suggestion would be correct only if the individual could *costlessly* lower exertion at each exertion level. However, consider an individual whose only exercise involves essential daily chores, such as his trip to work by bus and subway. He has

already minimized his exertion levels--unless he quits work (which of course would *lower* his income constraint), he simply cannot lower his exertion below what it currently is. Such a person, if currently at point D in figure 1, simply *cannot,* at costs he is willing to incur, shift his EH curve to the right.

[18] This circular indifference curve apparatus was suggested by a referee.

[19] The result that the new equilibrium weight is lower depends on the plausible assumption that the shift in indifference curves is not accompanied by a very considerable increase in the "spread" of each indifference curve to the right of the new *W**. A large enough increase in the "spread" (that is, a sizable decrease in the slope to the right of *W** at each *F* level), could alter the prediction of a falling weight and lower *F*.

[20] Note that the individual might also consider a new exercise regime. Exactly the same cautions apply: the individual needs to perform his own cost-benefit analysis of actually engaging in a new exercise regimen.

[21] This is because the coefficient on age is negative, so that the intercept in weight-food space decreases with age. The coefficient on the weight term itself is not affected by age in the Harris-Benedict equations.

[22] A weakness of two of the previous papers that produced models of weight change and dieting, Dockner-Feichtinger 1993 and Levy 2002, is that neither model generated predictions of weight rising with age. In contrast, SGL 2002 does generate such a prediction.

[23] A referee mentioned the possibility that aging might reduce appetite as it slowed metabolism. The referee further suggested that this might induce a direct movement from B to C, and that

such an “appetite reaction” might not be “dieting” in the “conventional” sense. While the possibility is plausible, we know of no empirical evidence that appetite declines with aging.

[24] Note our phrase “one simple way.” A referee pointed out that pregnancy might instead shift the EH curve.

[25] Our thinking about the stylistic dieting case was aided by seeing an intriguing paper by Smith [2002] containing an evolutionary biology model of weight change. Specifically, his “rodent foraging” model, in which rodents grow fat in the “summer,” when foraging/food availability is cheap, in order to survive the “winter,” when foraging is difficult, suggested to us the notion that costs of energy intake and use might vary systematically by season. The shift in “demand” for thinness by season in our stylistic dieting case has no analogy in the Smith model.

[26] A common view of smoking is that it curbs appetite. If one stops smoking, and appetite increases, this suggests that the shape of the individual’s indifference curves change, though *W** does not move. A plausible hypothesis is that the indifference curves flatten out to the right of *W**. This happens because the increase in appetite *raises* the *MU* of food. If this were the only effect of smoke cessation, then optimal weight would rise and optimal food intake would *rise.* No reason to diet here. To get a prediction of a diet associated with smoking cessation, there would need to be a *decrease* in energy use from smoking cessation which overwhelmed the appetite effect. As of the writing of this paper, we do not yet have a satisfactory answer about the energy use effects of smoking cessation.

[27] Dockner and Feichtinger 1993 for example put the issue as follows: “Much of empirically observable consumption behavior seems to contradict rational choice theory. Here we refer to heavy eating followed by strict dieting; smoking, quitting and starting again...and so on” [p.

256]. The focus of their article is to show that cyclical patterns of weight change can in fact be derived from rational choice behavior, in particular from a rational addiction model.

[28] We owe both these suggestions to an anonymous referee.

[29] Where might it all end, if none of the first n diets proves satisfactory? Sara Markowitz suggested to us the possibility that repeated diet failures might at some point provoke the individual to choose a more radical weight loss alternative, such as surgery, a choice recently made by NBC meteorologist Al Roker. The analytical issue here would be to model the choice of dieting versus more radical alternatives such a surgery.