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AN ECONOMIC ANALYSIS OF WEIGHT CHANGE, OVEREATING AND DIETING

by

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Excess body weight is the second-leading cause of preventable death and disease in the United States; 300,000 Americans die annually from obesity-related causes (McGinnis and Foege 1993; Must et al. 1999).[1] Only smoking exacts a higher toll. The costs of excess weight go beyond health risks; overweight persons tend to suffer low self-esteem, often earn lower wages (Cawley 2000), and spend more on weight-loss programs. U.S. obesity and overweight rates have increased sharply in the last decade, and an upward trend is also found elsewhere in the developed world. Fully 35 percent of Americans are overweight, and an additional 20 percent are obese (Mokdad et al. 2001).[2] The Centers for Disease Control (CDC) now refer to excess weight and obesity as an "epidemic."

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[1] "An excess body weight of 30% is associated with an increase of 25% to 42% in mortality, and mortality increases with increasing body weight." (Kushner 1993). This may be why even small amounts of weight loss can lower the risk of obesity-related illnesses. (Mitchell, 1997 p. 363, citing Goldstein 1992). "Much of the morbidity [disease] associated with obesity is due to an increase in the occurrence of hypertension... and [type 2] diabetes mellitus, all of which contribute to an increased risk of cardiovascular disease (Sjostrom, 1992...) (Mitchell, 1997, p. 359; see also Wilmore and Costill, 1999,pp.671-72). The external costs of eating too much and exercising too little may exceed those from smoking (Manning et al. 1991).

[2] Excess weight and obesity refer to the ratio of body fat to lean body mass. The body mass index (BMI) proxies for this ratio, where BMI = weight (kgs)/height (meters)^2 or BMI = (705)weight (lbs)/height (inches)^2. You are deemed overweight if your BMI is 25 or greater but less than 30, and you are obese with a BMI of 30 or more.

The paper proceeds as follows. Section I of the paper puts the question of "why does a person become overweight or obese" in the context of rational choice versus nonrational choice modeling frameworks. Section II begins by indicating how our paper improves on earlier rational choice attempts to model weight change and weight cycling. It then sets out how the nutrition literature conceptualizes weight change, focusing on the so-called "Harris-Benedict equations." In Section III, this weight change framework is used to create alternative weight-change-and-diet scenarios. In Section IV we analyze diet choice by developing a utility-maximizing model of dieting. Results are developed showing when long and mild diets are superior versus inferior to short and severe diets. Further results indicate that multiple diets are sometimes utility-maximizing. A clear intuition for this possibility is presented. Section V contains conclusions.

I INTRODUCTION: RATIONAL VERSUS NONRATIONAL CHOICE ALTERNATIVES

Why does a person become overweight or obese? The proximate answer is simple: he consumes more calories than he expends. An answer to the ultimate question, however – *why* does someone regularly choose to consume more calories than demanded by energy expenditures? – confronts a deep conceptual problem dating back to the Platonic dialogues. How should we regard apparently self-defeating choices, such as gaining unwanted excess weight, where consumption costs and benefits are separate in time, so that today's choices have consequences for one's future "self"?

There are, broadly speaking, two opposing traditions regarding self-defeating choice.[3] The first "non-rational" approach regards *apparently* self-defeating choices as *truly* self-defeating. Consumers smoke, overindulge in food and drink, pay taxes too soon, and save and exercise too little when they truly would prefer to do otherwise. Agents in the non-rational tradition often do not know or cannot help what they are doing. Their decision making incompetence arises from immaturity, or strong myopia, or irresistible cravings, or systematic decision making errors.

With respect to overeating, the non-rational tradition emphasizes genetic predisposition to excess

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[3] See Goldfarb, Leonard and Suranovic (2001) for a more extensive methodological discussion of rival explanatory approaches to self-defeating behavior, with particular reference to the case of smoking.

weight, poor health and nutrition information, or, even with decent information, the systematic inability to make choices consistent with one's preferences.[4] These non-rational explanations seem insufficient to us. Obesity does run in families, but genes do not change rapidly enough to account for the last decade's increase in obesity. Similarly, it seems likely that today's heavier consumers are better informed than were their thinner predecessors about the calorie content of foods they eat, and about the health risks of excess weight.[5] Nor is it obvious why today's heavier consumers would be more prone to systematic decision errors.

In contrast, the second "rational choice" tradition argues that apparently self-defeating choices may *not* be truly self defeating. A simple rational-choice explanation for obesity is that people actually prefer eating more and being fat to eating less and being thinner. "Ideal" weight is only what medical and public health experts deem ideal for health, and individuals whose well being involves more than just health concerns can rationally prefer the costs of excess weight to the costs of eating less or exercising more. As a recent paper by Levy, squarely in the rational choice tradition, puts it, a "positive difference between the rationally and the physiologically optimal level of weight indicates the individual's rationally optimal level of overweightness." (Levy, 2002, p.888).

In middle age, one might regret the excess pounds from choices earlier in life - just as smokers can regret taking up the habit, drinkers can regret hangovers, and borrowers can regret accumulated debt. But this regret need not imply that earlier choices, such as "overeating", were non-rational (Suranovic, Goldfarb and Leonard 1999). When choice and consequence are separate in time, such that costs come later (and benefits are sunk), regret can be consistent with lifetime rational choice (Goldfarb, Leonard, and Suranovic 2001).

The rational-choice approach explains increasing obesity rates with reference to changing relative

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[4] Some nutritionists argue that today's consumers eat more and they do so because of food industry practices. Fast food firms (especially) make their products irresistible by spiking them with calorie-dense ingredients, and by surreptitiously increasing portion sizes. Consumers are implicitly regarded as unable to resist the temptation of supersized portions. (Young and Nestle 2002).

[5] This is not to deny that some trends can make it more costly to count calories, such as the increase in consumption of restaurant and prepared foods, which tend to be unlabeled.

costs. Because Type 2 diabetes and hypertension are now (arguably) more treatable with medication, the health risks of a given level of obesity have declined.[6] Moreover, the costs of maintaining lower weight have grown: increasingly sedentary work means that physical activity must be funded out of valuable leisure time (Philipson and Posner 1999).

Rational-choice explanations for excess weight are closer in spirit to our approach, but they too are inconsistent with some stylized facts. Consider the fact that millions are dieting recidivists. If people are fat and happy – that is, all things considered, they truly prefer their weight above the public-health ideal – why do so many attempt to lose weight by dieting? And why, when they diet, do they eventually stop dieting, only to start again — an on-and-off-again pattern of multiple diets called cyclical dieting? (For one kind of answer, with its own difficulties, see Dockner and Fechtinger 1993 and Levy 2002. Our analysis is distinctively different from these two papers, and as we discuss below, has distinctive advantages).

This paper offers an account that is rational choice in spirit, and also consistent with widespread consumption behaviors, including cyclical dieting. We envision, for lack of a more felicitous term, a boundedly rational consumer. Our agent is fully rational in the sense that she does what she truly prefers. She purposefully and carefully compares the (discounted) expected costs and benefits of alternative consumption trajectories, and selects the best alternative from among them.. She weighs the pleasure of eating against the health and other risks of excess weight, and she even considers the additional adjustment costs a diet will impose.

Our individual's rationality is bounded in the following sense. She does not consider the space of all conceivable consumption trajectories; she only chooses among alternatives that each specify a constant daily calorie intake, a start date for calorie reduction, and a diet duration. We indicate why this is a usefully plausible "real world" assumption later in the paper. A much more subtle source of boundedness--so subtle that its boundedness is arguable--is the following. Our consumer knows the adjustments costs a diet imposes – the costs of eating less than- habitual consumption. But she does not fully take into account the complex intertemporal dynamics wherein previous choices, though optimal on a period-by-period basis,

[6] A counterargument is that more types of disease susceptibility have been identified with obesity.

affect her level of habitual calorie consumption and thereby the expected adjustment costs of dieting. Our consumer is not myopic, as in the non-rational approach to self-defeating behavior, but neither is she perfectly prescient (nor computationally prodigious), as typically required by time-consistency models in the rational choice approach to self-defeating behavior.

II. THE PHYSIOLOGY OF WEIGHT CHANGE

While obesity is front-page news, and the weight-loss industry has become a multi-billion dollar colossus, the economics literature on diet and exercise remains small. What work there is tends not to be grounded in the nutrition and physiology literature, whereas our work incorporates and builds on that literature's findings about the process and parameters of weight change. Dockner and Feichtinger (1993) modify Becker and Murphy's (1988) rational-addiction model, and, at least for some parameter values, obtain limit cycles. Their result is of interest formally but has important empirical disadvantages. There is, for example, no way to empirically determine whether the particular parameter values required for weight cycling are relevant to actual consumption choices. Levy (2002) also obtains some interesting formal results; he finds, without assuming addiction, that "overweightness" is optimal, and predicts explosive weight cycling under some conditions. It is subject to the same "no way to determine..." criticism as is Dockner and Feichtinger. A quite striking testimonial to the empirical limitations of the Levy model is offered by Levy himself: "However, the model's prediction of diverging spiral trajectories of food consumption and weight is unlikely to be supported by empirical analyses." (Levy, 2002,p.895).

The model we develop below has very large empirical advantages over these previous contributions. First, partly because our work is grounded in findings from the nutrition and physiology literature, we produce an empirical prediction consistent with what seems a widely recognized stylized fact: weight, and weight control problems, seem to increase with age. Put more starkly, even in a world of near-ideally rational agents, an aging person (population) is a fatter person (population). Neither of the two prior papers contains this prediction. Second, while the earlier articles refer to the concept of dieting, neither of them has an explicit concept of what a diet is, so it is impossible to use them to develop concrete results and empirical predictions about actual diets. In Levy (2002), for example, the term "diet" typically appears with the phrase "fluctuations in individual's food consumption and weight." For example, "The model's prediction of fluctuations in individual's food-consumption and weight is consistent with the observed phenomenon of binges followed by strict diets." (p.895). Since the diet is not a focus of the analytics, there is no attention to exactly what the term might mean conceptually or empirically. Below, we provide a precise analytical notion of exactly what a diet is, and the parameters needed to characterize one.

The nutrition literature models weight determination and change by analyzing the balance of energy intake and energy expenditure.[7] Physiologists typically disaggregate energy expenditures into three components: resting or basal metabolism, food digestion, and muscular physical activity. Basal metabolic function accounts for 60 to 75 percent of energy expenditure in most individuals; another ten percent is burned eating and absorbing food. Muscular activity accounts for the remaining 15 to 30 percent, in a moderately active individual (McArdle et al 1996: 151). A continuing excess of energy intake over energy expenditure results in weight gain.

Basal metabolic energy requirements or basal metabolic rate (BMR) can be estimated as a function of age, weight, height and sex. Because they are widely employed in the nutrition literature, we use the venerable Harris-Benedict equations (Harris and Benedict, 1919):[8]

For men: $BMR = 66 + 13.7\,W + 5H - 6.8\,A$

For women: $BMR = 655 + 9.6W + 1.8H - 4.7A$

where BMR = basal metabolic rate, H = height in cm, W = weight in kg

A = age in years.

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[7] See, for example, Willett 1990 pp. 245-46; Melby et al 1998, p.6, Forbes 1999 p. 801, and Wilmore and Costill, 1999, pp.667-68.

[8] While numerous other equations are available, "many researchers use the Harris-Benedict method to determine BMR." (Whitney, Cataldo and Rolfes, 1998, p.267).A literature exists that compares various BMR estimating equations, and examines how well they seem to work for more narrowly defined populations. (See, for example, Cunningham,1980, Vaughan et al 1991, Taaffe et al 1995, Liu et al 1995, Wong et al 1996, Tverskaya et al, 1998).These articles typically obtain direct laboratory measures of BMR for a sample of individuals (for a discussion of the laboratory techniques used to measure BMR, see McArdle et al, 1996, pp.139-141). They then compare the actual BMR laboratory measures to the predicted measures of BMR from the various available predicting equations. The Harris-Benedict equations seem to have survived these explorations.

Energy expenditure on physical activity (and the thermic effect of food) is captured by applying a multiplier to the basal metabolism estimates, as follows:

Sedentary	= BMR X 1.2 (little or no exercise, desk job)
Lightly active	= BMR X 1.375 (light exercise/sports 1-3 days/wk)
Mod. active	= BMR X 1.55 (moderate exercise/sports 3-5 days/wk)
Very active	= BMR X 1.725 (hard exercise/sports 6-7 days/wk)
Extremely active	= BMR X 1.9 (hard daily exercise/sports & physical job)[9]

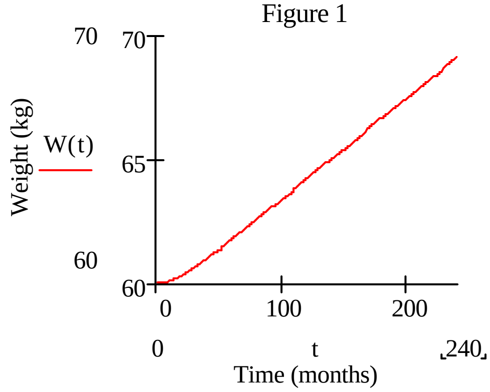
As is clear from the equations, basal metabolism slows with age. This is well-documented in the physiology/nutrition literature (see the cites in McArdle et al 1996, p.152. See also Vaughan et al 1991). There is also evidence, if less extensive, that energy expenditure on physical activity declines later in life.[10]

For our analysis, there are two key implications. First, even with constant calorie intake, age insidiously increases weight, and does so at a faster rate if exercise levels also fall off over time. Second, these facts by themselves suggest a rationale for dieting. An aging person will, *ceteris paribus*, gain excess weight, and must diet (or exercise more) to avoid weight gain.

III. A WEIGHT-CHANGE-AND-DIET SCENARIO

We use this framework to generate weight change scenarios that isolate the underlying conditions likely to lead to dieting behavior. Consider a 20 year old woman, "Sara," who is 170cm tall (5'6") and weighs 60kg (132 lbs.). Because she is moderately active, we multiply her initial BMR of 1443 by an activity factor of 1.55 to obtain her Total Daily Energy Expenditure (TDEE), measured in calories. Given

Figure 1



9 Note that the sedentary multiplier gives a calorie usage that is 20% higher than one's BMR. This is likely to include the 10% thermic effect of food absorption plus some additional amount for low levels of physical activity.

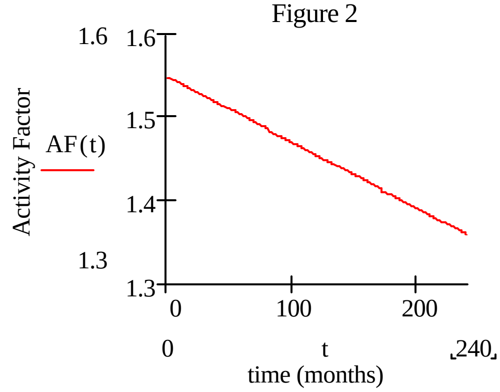
10 McArdle et al, 1996 p. 159, assert that the energy use associated with a specific exercise activity decreases from middle age on because of "the general 'aging effect' on aerobic capacity." See also Rising et al, 1994, a study based on a sample of Pima Indians.

her energy demands, Sara at age 20 requires 2237 daily calories.

If Sara were to consume 2237 calories daily for the next 20 years, a slowing metabolic rate reduces the calories requirements needed to maintain her weight. For every 7700 calories accumulated above the number of calories needed each day, Sara gains one kilogram. Her weight trajectory is shown in Figure 1.[11]

Time periods *t* correspond to months, thus the 240 periods on the horizontal axis is a span of 20 years. Sara's weight, W(t) is measured in kilograms. Simply by aging, Sara gains nearly 10 kilograms (22 pounds) by age 40.

Figure 2

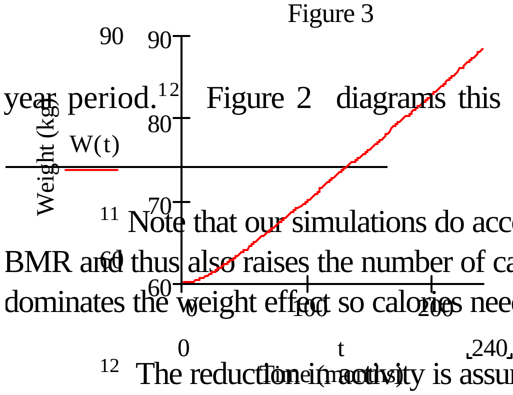


If, as is likely, physical activity also declines in age, Sara gains still more over time. Assume Sara's activity level falls gradually and linearly as in equation 1:

$$AF(t) = AF0 - at \qquad (1)$$

We set AF0 = 1.55 as the initial activity level, and a = .0008 allows for a gradual reduction in activity over the 20 year period.[12] Figure 2 diagrams this pattern, which embodies the assumption that Sara becomes

Figure 3



[11] Note that our simulations do account for the fact that rising weight raises the individual's BMR and thus also raises the number of calories needed to maintain weight. However, the aging effect dominates the weight effect so calories needed to maintain weight falls with age.

[12] The reduction in activity is assumed to lower the individual's total daily energy expenditure (TDEE) by approximately 10 - 15% per decade. This is a larger per-decade decline than the average referred to in the literature, as the following summary statement indicates: "the BMR begins to decrease in early adulthood (after growth and development cease) 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." (Whitney et al, 1998, p. 263) We use a larger estimate of this decline because we

only lightly active to sedentary by age 40.

Even though the 40-year-old Sara eats no more than she did at age 20, a slowing metabolism and less physical activity combine to increase her weight by over 28 kilograms (62 pounds). (See Figure 3) She is obese. The extra 28 kilograms amounts to an daily excess of a mere 30 calories, the equivalent of two surplus potato chips per day. [13]

Effects of a Diet

The only way for Sara to prevent weight from rising inexorably throughout her life is to reduce her calorie intake (i.e., diet) or to increase her physical activity, or both.

Many weight loss strategies might be pursued: diets differ by the degree of caloric restriction, the length of the diet, and the types of calories (fat, carbohydrate, protein) allowed. The proliferation of diet plans attest to this. Diets can also supplemented with exercise. In this paper we ignore the issues of diet composition and exercise to focus on calorie reduction. Calories are calories are calories, we assume; and any change in weight is accomplished through reduced calorie intake alone.

A diet is specified by three parameters: the diet's starting date D_0; its duration or period, p; and the degree of calorie reduction, z, which is defined relative to the individual's habitual level of daily calorie intake. Sara's habitual calorie intake at age 20 is assumed to be 2237 calories per day. The consumer does not consider the entire universe of possible consumption trajectories – she chooses among constant calorie options only, ignoring for example, continuously declining or periodically changing food consumption plans.

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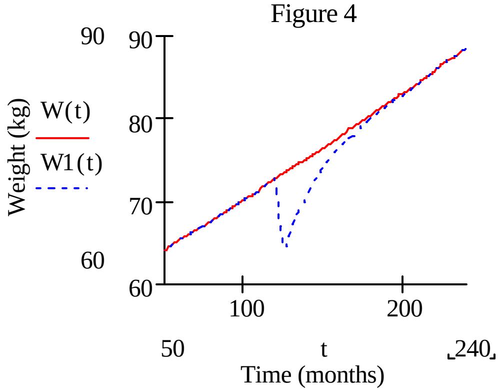
are concerned not with the population average, but with that segment of the population more-likely-than-average to develop weight gain problems.

[13] For the reader skeptical that a few potato chips per day could have such a large effect, we present the following citation. The Washington Post of Feb 10, 2003 contained an article in their "Lean Plate Club" column series. This article, called "The Lean Plate Club: the Single Cookie Theory," contains the following claim. "Just one small cookie a day. That's all that a team of researchers suggests stands between most Americans' piling on an average of two pounds per year and holding the line against weight gain. The team, led by James O. Hill, director of the Human Nutrition Center in Denver, came to that conclusion....To the best of our knowledge, it looks like 100 calories a day less is plenty to hold the line on gaining two pounds a year," Hill says...."Tiny things can make a big difference."

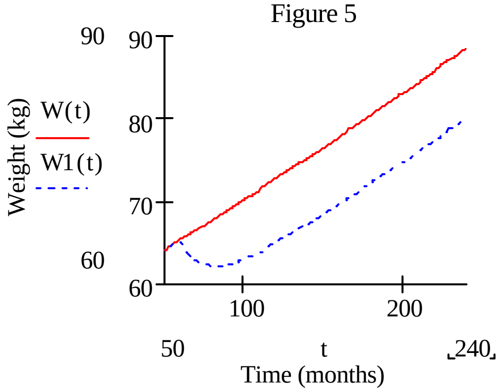
We think that most dieters, in practice, target a given level of consumption. The simplicity of a constant target has several advantages. Unlike more complex calorie plans, it provides the dieter with a kind of motivational concreteness: “forgo only one muffin a day, and the pounds will eventually come off.” A fixed target also helps conserve on the non-trivial transactions costs of determining and counting the calories in everything one eats. Precision matters: the 40-year-old Sara is obese, recall, after an effective daily excess of only 30 calories. The market for prepackaged diet plans – where firms such as Weight Watchers sell prepared meals with calories exactly determined – exists, in part, to economize on the transactions costs of calorie accounting. The strategy of ignoring continuously or periodically changing consumption plans also conserves on decision-making costs, while maintaining a sizable set of options.

Figure 4



To illustrate the effects of a diet, assume that Sara reduces her calorie intake by 20%, for a 6 month period beginning at age 30 (period 120), when age has increased her weight to 72 kg. A twenty percent reduction means giving up, for example, a cup of whole-milk yogurt, and a 20-ounce cola, daily. Assume that in the seventh month and beyond, Sara goes off the diet, and reverts to her age-20 calorie level. Sara’s weight path with the diet is shown by the dotted line, path W1(t) in Figure 4. The solid line, path W(t), shows her weight path with no diet. The diet is initially very effective, as many are. In six months, she loses a full 8.4 kilograms (18.4 pounds).However, its effectiveness is temporary. Once Sara stops dieting – her calorie intake reverts back to its habitual level – a slowing metabolism and reduced exercise cause her weight to increase, eventually returning to the same level it would it be with no diet at all.

Figure 5



Suppose instead that Sara undertakes a longer diet. *Ceteris paribus*, this will prove to be more effective in keeping her weight down. But a slowing metabolism and reduced exercise can thwart even the disciplined, long-term dieter. Consider for example a diet consisting of a 5% reduction in calorie intake (e.g., forego one cup of skim-milk daily) from age 25 to age 40 (period 60 to 240). The effect on Sara's weight is shown in Figure 5. The diet path is given by W1(t) whereas the no diet weight path is W(t). Though weight declines for about 20 periods with the diet, it eventually begins its upward trend again. A modest diet, even a permanent one, will lower weight relative to no diet, but it cannot provide a permanently lower weight.

These simple weight change scenarios are consistent with widespread consumption experience: (1) aging (i.e., slower metabolism and lessened physical activity) adds pounds and by itself creates a common-sense rationale for dieting, and (2) weight is easy to lose, but hard to keep off. Even permanently lower calorie consumption, while improving the outcome, may not buck the upward trend caused by slower metabolism and reduced exercise.

Next, we ask, what choices will a "boundedly" rational consumer make? Given the weight trajectories considered above, would a person like Sara choose to diet? If so, when would she begin and how strict a diet would she choose? If she decides to diet temporarily, would she have an incentive in the future to choose to diet again? To answer these questions, we need to specify the key tradeoff: how much one eats and how much one weighs.

IV. A UTILITY-MAXIMIZING MODEL OF DIETING

The Costs and Benefits of Eating

Here we develop an additively separable, instantaneous utility function with three elements: (1) consumption benefits, which are a positive linear function of calories; (2) weight costs, a quadratic (symmetric) loss function which is minimized at "ideal" weight; and (3) dieting costs, which are asymmetric adjustment costs from reducing calorie intake below a habitual "calorie trend."

Sara makes consumption choices by weighing the costs and benefits of various alternatives, choosing the option with the greatest net benefits (or utility), as discounted at her rate of time preference. Sara's utility is a function of how much she eats, and how much she weighs.

Consumption Benefits

Food consumption is clearly an enjoyable activity which we treat as a standard economic good:

Sara's utility rises with increased food consumption. Because the relative utility effects of food consumption, dieting and weight gain are of central importance in the subsequent analysis, we normalize Sara's utility from her initial food consumption to 100 using the following formula:

$$B(t) = 100\ c(t) / C0$$

where B(t) is the benefit from food consumption at time t, c(t) is the quantity of daily calories consumed at time t and C0 is the initial daily caloric intake at age 20, set to maintain Sara's initial weight. Note that c(0) = C0. Thus, utility benefits will equal 100 if she consumes her age-20 calorie level. Since benefits are linear in calories, a 500-calorie slice of pecan pie is twice as good as a 250-calorie yogurt, and two slices are four times as good.[14]

Weight Costs

A person's weight can affect well being in several ways. Perhaps most obvious is the psychological effect on self-esteem. All societies have valued and rewarded desirable physical attributes. In turn-of-the-millennium North America, a slender appearance is deemed desirable, and those without this physique, especially those farthest from it, can suffer anxiety, depression, and reduced self esteem.

Heath risks are another consequence of excess weight. Excess weight increases the risk of death and disease, which reduces well-being directly. These effects enter the analysis formally as do the psychological effects: the more that weight deviates from an ideal weight, the greater are the utility losses.

The loss function L(t) describes the utility losses created when the consumer deviates from her ideal weight:

$$L(t) = [15*(W(t) - W0) / W0]^2$$

[14] Although it makes sense to assume that the marginal utility of food consumption decreases, perhaps even becoming negative at some point, we exclude this feature from the model to focus more on the relative effects of dieting, weight gain and daily food consumption. Our model also assumes that calories are calories are calories. This runs contrary to some diet theories, like Atkins's, which claim that some calories (from carbohydrates) are worse than others.

Here W(t) is Sara's weight at time t and W0 is her initial ideal weight of 60 kg. Utility losses rise at an increasing rate with deviations from W0. Recall that 7700 calories equals one kilogram. This means, for example, that if you reduce your daily calorie intake by 250 (one cup of jellied yogurt) and increase exercise sufficient to burn another daily 300 calories, you will lose ½ kilogram per week.[15]

Figure 6

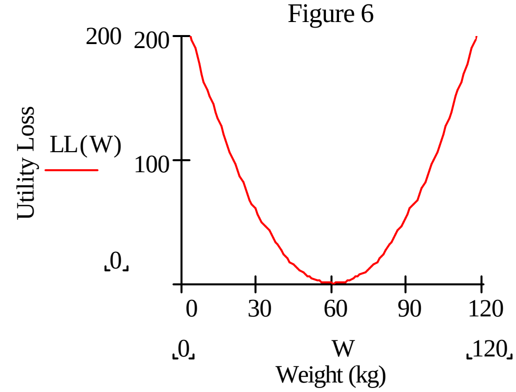


Figure 6 shows how the L function behaves. It shows utility losses (LL) as a function of weight, W, rather than a function of time. The effect of non-ideal weight on well being surely varies from person to person. For Sara we calibrate the loss function relative to the 100 units of utility that she obtains from consuming her habitual calorie level C0. For concreteness, recall that the 40-year-old Sara who has never dieted weighs more than 88 kilos. The utility loss function implies that daily costs in well-being of the excess 28 kilos are fully half of the benefits she obtains from eating.

Dieting adjustment costs

Eating habits are hard to change. It is not merely that eating and food rituals are culturally central, and at the heart of daily life. They are also hard to change because our bodies are evolutionarily programmed to counter significant changes in calorie intake. The effect is homeostatic: when calorie intake falls long enough to deplete bodily fat stores, the body responds by stimulating appetite and slowing the rate at which stored fat calories are burned. When bodily fat is increasing, appetite is suppressed and a higher burn rate occurs.[16] The human body naturally pushes against attempts to affect weight with changes in diet. There can also be psychological costs to dieting – especially for people who find comfort and solace in

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[15] By using a quadratic loss function, we assume that being under ideal weight is just as costly as being over ideal weight. For large deviations below ideal weight, this would be consistent with anorexia or perhaps malnourishment.

[16] The mechanisms by which the body determines energy requirements, and then increases or decreases hunger in response, are both complex and subtle. It "has puzzled scientists for years" (Wilmore and Costill, 1999, p.666).

eating.

We model the difficulty in reducing calorie consumption as an adjustment cost, that is, as a cost above and beyond the foregone pleasure of eating. We have experimented with several adjustment cost functions; it is clear that results depend critically on how one specifies the adjustment costs of eating less. The following formulation is the simplest that is consistent with the adjustment-cost attributes we think are most plausible:

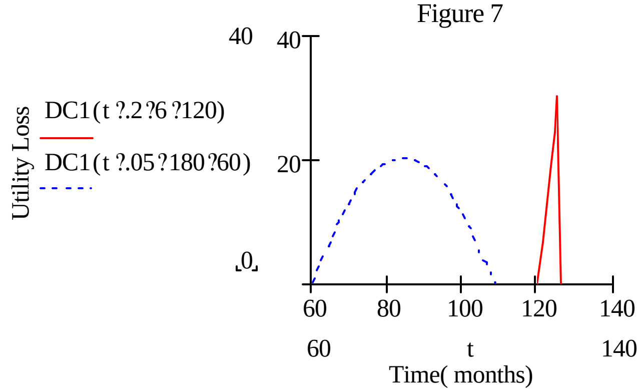
$$DC(t) = .0005\,(t - D0)(|avgc(t) - c(t)|)$$

where DC(t) represents Sara's diet costs at time t, avgc(t) is her average daily calorie consumption during the previous 4 years (48 periods), D0 is the period in which she begins the diet, and the number .0005 is a scale factor.

The adjustment cost function embeds the following claims: (1) changing eating habits is hard and costly but not impossible; (2) *ceteris paribus*, longer diets are harder than shorter diets; (3) more severe diets, as measured by the extent to which calories are cut below habitual levels, are more difficult than milder diets; (4) but, eventually, sufficient dieting persistence is rewarded with a reduction in adjustment costs. Adjustment costs are increasing in both diet duration (t-D0) and in diet severity, $|avgc(t) - c(t)|$. But, the very habitual consumption that makes dieting hard, can also, with sufficient diet persistence, evolve into an asset. Dieting virtue is, eventually, rewarded, because avgc(t) converges to c(t). After enough time, what was once a diet becomes a habit.

For illustration, Figure 7 shows the adjustment costs for the two diets already considered. With Sara's permanent 5% diet (begun in period 60 at age 25), dieting costs, rise, then fall back to zero by period 110. We denote these dieting costs as DC1(t,.05,180,60), where the .05 indicates the 5% cut; the 180 indicates that the diet lasts 180 months; and the 60 indicates

Figure 7



the diet starts in period 60. Dieting is hard, but persistence is rewarded with enough time – it gets easier after you get "over the hump." Because the time it takes to get over the hump is a function of the number of periods over which habitual consumption is determined--what is called in the behavioral economics literature the "reference level of consumption"--, the likelihood of long-term diet success, as we will see, depends critically on this parameter.

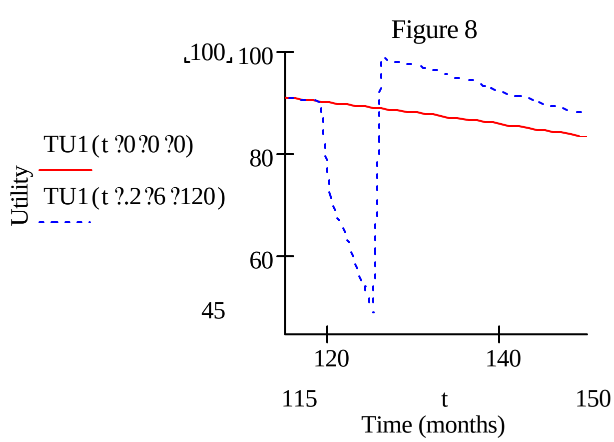
For comparison, the dotted line shows dieting costs,DC1(t,.2,6,120), for a severe diet, the 20% reduction undertaken for 6 months beginning in period 120. In this case the costs rise much more steeply due to the greater calorie reduction. They revert to zero only because the diet is stopped.

Combining All the Utility Effects

Sara's utility at time t is the sum of the utility derived from consumption, weight, and diet, that is,

U(t) = B(t) - L(t) - DC (t)

How does Sara's utility with a diet compare to her utility in the absence of a diet? Consider again the diet where Sara reduces her calorie intake by 20% for six months beginning at age 30 (period 120). The utility that Sara experiences as a result of that diet is shown as the dotted line in Figure 8. The solid line represents the utility Sara would have in each period if she refrains from a diet. Notice that the diet causes a sharp initial drop in utility during the early periods of the diet. However, because of her weight loss during the diet she is able to achieve a higher level of well-being in post-diet periods. Hence Sara's decision to diet will be based on how she trades off utility losses in early periods with later utility gains.



Beginning a Diet

We assume that Sara weighs the costs and benefits of alternative consumption trajectories and diets according to the formulae set forth above. In particular, when deciding whether to diet, she evaluates the present discounted value of the stream of future utility with a diet, comparing it to the present value of utility without a diet. We will imagine that Sara evaluates these paths at time 0, age 20, and calculates the effect through period 240, at age 40.

Recall that Sara does not search the vast space of all possible diet variations, but considers alternatives that differ in percentage reduction, duration, and start date. Consider two ways Sara might decide to diet. First, she might consider a relatively small calorie reduction implemented over a long period of time, a **long and mild diet.** Or, she might consider a relatively large calorie reduction for a short period of time, a **short and severe,** or **crash** diet. **Long and mild** diets have the advantage of a less severe per-period dieting cost, and the disadvantage of a longer duration. Long and mild diets also reduce weight relatively more slowly. Short and severe diets offer rapid weight loss benefits, but at a decidedly higher initial cost.

Both types of diets can make Sara better off than with no dieting. The best **long and mild** diet she can choose (given the specified parameter values) is a 4% calorie reduction begun in period 90 and continuing to the end of planning horizon (period 240). The best **short and severe** diet she can choose works out to a 100% calorie reduction for 3 periods beginning in period 191 – a starvation diet that achieves the fastest weight loss possible in the shortest amount of time. Compared to each other, given the parameter values chosen, the **crash** diet yields greater utility than does the **long and mild** diet, as shown in Table 1.

Table 1

Sara's Diets

% Calorie Reduction	Length of Diet (months)	Period of Diet Start	PDV of Utility (utils)
-	No Diet	-	13,525
4%	150	90	13,742
100%	3	191	13,762

Figure 9

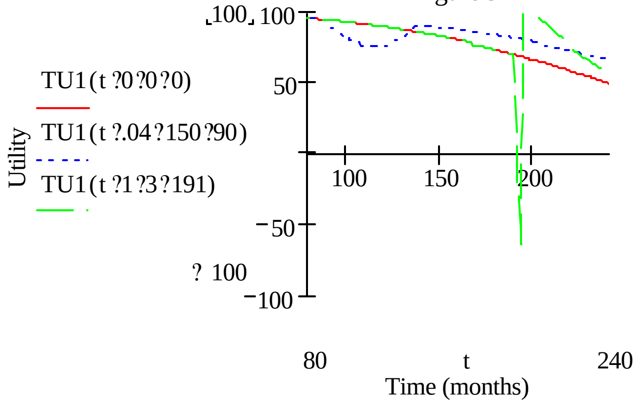
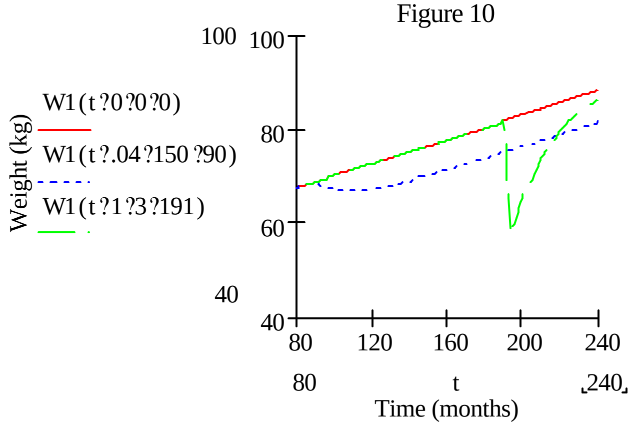


Figure 9 depicts the utility paths for each type of diet compared to the no-diet scenario. The solid, continuously falling line, TU1(t,0,0,0), is the no-diet utility path. The **long and mild** diet, , TU1(t,.04,150,90), depicted by the narrow dashed lines, reveals that the short-term pain of dieting is offset by long term benefit of lower weight. The **optimal short and severe diet,** , TU1(t,1,3,191)**,** yields extreme utility loss during the short starvation period followed by a considerable improvement in utility afterwards.

The weight path Sara would follow under each scenario is shown in Figure 10. Notice that the long and mild diet, depicted by the narrow dashed line, reduces weight moderately at first, but in time Sara's weight continues it's upward trend. For the starvation diet, Sara's weight plunges dramatically until she achieves her ideal weight again in just three short periods. Afterwards her weight increases, reverting to the no-diet trend in a few years.

Figure 10



Clearly, a sustained zero-calorie diet is unrealistic, not to say dangerous. Consumers ordinarily don't fast for long periods, and those that do generally ingest some calories with the fluids they must consume. Nonetheless, the analysis does illustrate some interesting results, consistent with the evidence.[17]

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[17] One should think of this severe diet as the extreme case of a diet with just enough sustenance to preserve life. We could have built in a minimum calorie constraint, but the choice of the calorie level would be both controversial and arbitrary. To avoid arbitrary choice, we let the constraint be 0 calories. It should also be noted, that there are proponents of near-starvation diets, not as a device for weight control per se, but with the idea of increasing longevity. Animal (including primate) studies

Crash diets don't work, in the sense that the large weight loss is regained fairly quickly. But, measured in well being, crash diets have a kind of logic to them (medical complications set to one side). The weight loss, though transient, is profound enough to offer real long-term gains in well-being. Viewed over long periods, even enormous short-term costs can be preferable to small but chronic deprivation. On the other hand, true starvation diets are not viable for long periods, so that reductions are constrained. The more that reductions are constrained, the more preferred will be long and mild diets. Measured not in pounds but in welfare, the choice between long and mild diets and crash diets is not one of principle, but a matter of the details.

As noted previously, results depend critically on dieting adjustment costs, especially the "inertia" embedded in "the reference level of consumption.". A longer period of habit persistence makes it harder for dieters to get over the hump, that is, to "reset" their habitual consumption level, and thereby tends to make long and mild diets relatively less attractive when compared to crash diets. In effect, the long-term pain of adjustment is worse than the short-term pain of intense food deprivation. By the same token, a shorter period of habit persistence reduces the pain of adjustment, and makes long and mild diets relatively more attractive than the short and severe variety.

Since the shape of the diet adjustment cost function is playing a crucial role here, it is worth giving an intuitive explanation of **why** that shape is so important. Consider someone who suffers rapidly increasing (and very large) adjustment costs the longer she stays on the diet. **Such a person may sensibly choose to stay on a diet only a short time, to avoid the rapidly cumulating costs**. In contrast, someone not subject to these exploding adjustment costs, may well find it sensible to stay on the diet.

Table 2 illustrates the point, with a sensitivity analysis. We show the utility level attained under the two diet cases under consideration, with three different periods of time for calculating average consumption. The intermediate case, with 48 month calculation, corresponds to the example above. Average consumption calculated over a 6 month period corresponds to a case in which habits are not very persistent. Within 6 months all dieting costs will revert to zero, since the individual will have become

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suggest that extremely restricted calorie intake, which slows metabolism, is strongly correlated with greater longevity (Lane et al. 1996).

acclimated to the new consumption patterns. In this case the value of a long and mild diet rises considerably. The value of a **short and severe** diet also rises, but not enough to keep up with the **long and mild** diet. Alternatively if consumption habits are very persistent, as when average consumption is calculated over 120 periods, then the effectiveness of a long and mild decline significantly: it takes too long to get over the hump. Crash diets become preferred.

Table 2 Utility with Varying Diet Cost Persistence			
Type of Diet	6 months average	48 months average	120 months average
No Diet	13,525	13,525	13,525
long and mild	14,057	13,742	12,328
short and severe	13,773	13,762	13,761

We can think about variation of habit persistence (in the dieting adjustment cost function) across persons as generating variation in optimal diet types.[18]

Cyclical Dieting

We've seen that slowing metabolism and reduced physical activity can eventually lead to weight increase even for the disciplined dieter, the dieter who maintains a level of consumption below the age-20 ideal level. This insidious weight gain can create the conditions for consideration of a second diet.

In this section we evaluate whether a repeat diet can be welfare-improving. Clearly, a repeat diet would not be possible if a person chooses a continuous fixed percentage reduction for the rest of the evaluation period. Consider a repeat diet for the person who chooses a temporary calorie reduction. In Sara's situation above, using the 48 period average consumption term in the dieting function implies that

[18] There is an analogy to the results in our smoking model (Suranovic, Goldfarb and Leonard 1999). In that analysis, systematic variation in the shape of the withdrawal cost function--a kind of adjustment function-- causes systematic changes in the pattern of quitting. These patterns vary from immediate ("cold-turkey") quitting at one extreme, to gradual quitting (a "slow" reduction in the number of cigarettes smoked per day) at the other.

Sara's optimal one-time diet is a starvation diet for three periods beginning in period 191. In that case, after Sara ends this first diet, a second diet makes her better off. Table 3 shows the best second diets for Sara for different waiting spells. Notice that a second diet becomes welfare enhancing very soon after the first diet is completed.

Table 3

Sara's Repeat Diets

% Calorie Reduction	Length of Diet (months)	Period of Delay	PDV of Utility (utils)
No Diet	0	-	13,762
75%	2	5	13,806
100%	2	10	13,836
100%	2	15	13,857
100%	**2**	**20**	**13,865**
100%	2	25	13,843

As Table 3 indicates, the optimal second diet starts after a 20 period delay. This diet too is a starvation diet that lasts just two periods. The weight path, W1(t,1,1,3,2,191), generated by this two-diet set is shown in Figure 11. Note how rapidly Sara's weight rises back towards her original weight. The second diet effectively brings Sara's weight back to her ideal level, albeit temporarily, since once she reverts back to her original habits, her weight rises rapidly once again. Nonetheless, the lifetime utility Sara gets from the double diet path exceeds her utility from one diet and from no diet. Measured in pounds, *her diets are ultimately unsuccessful*, but measured in well-being, Sara is better off with the multiple diet choice.

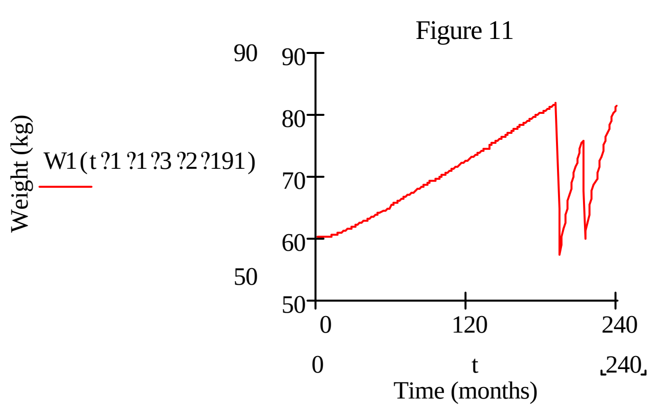
In discussing the choice of long versus short diets in the previous section, we stressed the importance of the shape of the adjustment function, noting specifically that **a person with rapidly cumulating adjustment costs may sensibly choose to stay on a diet only a short time, to avoid these exploding costs.** The result that, for such as person, multiple diets may be an attractive choice, is a simple extension of the "she-will-choose-a -short-diet" result. **If exploding adjustment costs make a**

long diet prohibitively expensive in utility terms, it becomes a more attractive alternative to undertake a series of shorter diets! What makes this alternative possible is that the adjustment cost "ticker" is reset at zero once one has been off the previous diet for at least one period.

It is interesting to note that these choices are being made, by assumption, with Sara's full knowledge about the eventual weight path to be followed. One might instead imagine that an individual's decision to attempt a second diet may be made because she expects that the diet will be successful and will allow her to maintain a lower weight indefinitely. Indeed, if Sara did believe that, it would make a second diet even more likely to occur. However, what this example suggests is that even if Sara knows that she will regain all the weight, it can still make sense for her to suffer through the second diet.

V. CONCLUSION

The obesity "epidemic," unhappy dieters, and cyclical dieting are widespread phenomena that, at first glance, appear anomalous from a rational choice perspective. We offer a rational-choice explanation of these apparently self-defeating behaviors, grounded in a physiological model of calorie income and expenditure. Age insidiously adds excess weight, albeit in tiny increments, and the adjustment costs of dieting make it challenging to offset these natural effects, even when they are recognized.



We do not, as does the non-rational tradition, regard eating choices and weight outcomes as unwanted mistakes. Sara, the hypothetical agent, is rational in that she is purposeful, forward looking, and calculating. But her foresight and dynamic modeling skills are not unbounded, and some weight outcomes are, in part, a by-product of these very human limitations.

In our view (with exceptions for persons with metabolic disorders), unwanted excess weight is not helpfully explained (only) by bad genes, poor information, and involuntary cravings. We have meaningful choices when trading off the pleasures of eating with the pain of excess weight and exercise. But neither is it adequate to suggest that the tens of millions of overweight and obese Americans all sat down and chose to be fat. The boundedly rational dieter knows what she doing, but what she does, nonetheless, is not always what her future self, with the benefit of hindsight, would have had her do.

The modeling framework we have developed in this paper has immense advantages over the earlier papers by Dockner and Feichtinger (1993) and Levy (2002). Our paper contains the strong prediction that weight is likely to rise with age, a prediction not available in either of the two previous papers. Compare our prediction to Levy's " prediction of diverging spiral trajectories of food consumption and weight," which he himself acknowledges "is unlikely to be supported by empirical analyses." (Levy, 2002,p.895). Moreover, we are able to characterize types of diets and analyze when long and mild diets are likely to be more attractive than short and severe ones. More generally, our model suggests the crucial importance of the shape of dieting adjustment costs in determining choice of types of diets.

There are a number of potential extensions of our modeling framework. As previously emphasized, one centrlal feature of the model is that variations in the adjustment cost function can generate variations in optimal diet patterns, and therefore in patterns of weight change with age. At one extreme, individuals whose habits adjust very quickly and at low cost to a new diet regimen may be able to "relatively costlessly" make periodic downward calorie adjustments that actually keep their weight from rising ("change their life-styles," in the language of the recent dieting literature). At the other extreme, individuals may go on "crash" diets repeatedly but inexorably gain weight. Using this modeling framework to simulate a population that varies by adjustment cost function might allow interesting simulation experiments generating weight change characteristics over time for this simulated population, and allow testing "aggregate" weight change propositions about this same population.

A second extension would be to analyze how results about dieting behavior change under differing assumptions about Sara's level of sophistication. Suppose for example, that Sara is much more naive than we have assumed above. This naivete might involve Sara's imagining at some point that even though she has gained weight consistently as she grows older that her future weight path will stabilize at its current level. It might also involve a naive belief about the effects of dieting: that once a diet has allowed her to achieve a lower weight, she expects to be able to continue to maintain that lower weight even after she stops dieting. This set of assumptions is consistent with “status quo bias.” Sara believes that the status quo will be maintained if she does not diet, and that a diet will achieve a new status quo that will also automatically be maintained without additional effort. What would Sara’s diet choices look like under this alternative set of assumptions?

A third possible extension involves the evaluation of alternative specific diets of the type actually offered commercially. If people do make decisions about whether to diet in the way our model posits, then the framework may be useable to test the likely effectiveness of alternative diet strategies. It may be possible to characterize the differences between Atkins, Weight Watchers, and other popular diets in ways the model could fruitfully analyze. Moreover the model might provide insights about the choice between quick and deep diets, which were perhaps more common years ago, and the new gospel which seems to espouse lifetime diet changes. These examples are meant to suggest that there seems to be a rich menu of possibilities for extending and applying the modeling framework in interesting ways.

REFERENCES

Ainslie, George. 1999. "The Dangers of Willpower." In Elster and Skog, pp. 65-92.

Bender, David. 1997. Introduction to Nutrition and Metabolism 2nd edition. London: Taylor and Francis.

Brownell, Kelly and Judith Rodin. 1994. "Medical, Metabolic and Psychological Effects of Weight Cycling," Archives of Internal Medicine, 154 (June 27), pp. 1325-1330.

Brownell, Kelly, Judith Rodin and Jack Wilmore (eds). 1992 Eating, Body Weight and Performance in Athletes. Philadelphia: Lea and Febiger.

Capaldi, Elizabeth and Terry Powley (eds). 1990. Taste, Experience and Feeding. Washington,D.C.: American Psychological Association.

Cawley, John. 2000. "Body Weight and Women's Labor Market Outcomes" NBER Working Paper 7841. NBER: Cambridge.

Cunningham, John. 1980. "A Reanalysis of the Factors Influencing Basal Metabolic Rate in Normal Adults," American Journal of Clinical Nutrition,33 (November) pp. 2372-2374.

Dockner, Engelbert and Feichtinger, Gustav. 1993. "Cyclical Consumption Patterns and Rational Addiction," American Economic Review. 83:1 (March), pp.265-263.

Elster, Jon and Ole-J?rgen Skog (eds.). 1999. *Getting Hooked: Rationality and Addiction*. Cambridge: Cambridge University Press

Forbes, Albert. 1999. "Body Composition: Influence of Nutrition, Physical Activity, Growth and Aging," in Maurice Shills, James Olson, Moshe Shike, and A. Catherine Ross, editors, Modern Nutrition in Health and Disease 9th Edition. Baltimore: Williams and Wilkins, pp.789-807.

Frayn, Keith. 1996. Metabolic Regulation. London: Portland Press.

Garrow, John. 1986. "Exercise, Diet and Thermogenesis," in Myron Winick, Editor Nutririon and Exercise. New York: John Wiley and Sons, pp. 51-65.

Gladwell, Malcolm. 1998. "The Pima Paradox," The New Yorker, (February 2).

Goldfarb, Robert, Thomas Leonard and Steven Suranovic. 2001. "Are Rival Theories of Smoking Underdetermined?" Journal of Economic Methodology, 8 (June), pp. 229-251.

Gruber, Jonathan and Botond K?szegi. 2001. "Is Addiction 'Rational'? Theory and Evidence" NBER Working Paper.

Harris, J. Arthur and Francis Benedict. 1919. A Biometric Study of Basal Metabolism in Man . Washington,D.C. The Carnegie Institution.

Jeffery, Robert. 1996. "Does Weight Cycling Present a Health Risk?," American Journal of Clinical Nutrition 63 (suppl) pp. 452S-455S.

Kolata, Gina. 2000. "How the Body Knows When to Gain or Lose," The New York Times, October 17, 2000 ("NY Times on the Web" download, pp. 1-9.)

Laibson, David. 1997. "Golden Eggs and Hyperbolic Discounting" *QJE* 112: 443-477.

Mark A. Lane, David J. Baer, William V. Rumpler, Richard Weindruch, Donald K. Ingram, Edward M. Tilmont, Richard G. Cutler, George S. Roth 1996. "Calorie Restriction Lowers Body Temperature in Rhesus Monkeys, Consistent with a Postulated Anti-Aging Mechanism in Rodents" *Proceedings of the National Academy of Sciences* 93(9):(Apr. 30, 1996), pp. 4159-4164.

Leonard, Thomas C., Robert Goldfarb and Steven Suranovic. 2000. "New on Paternalism and Public Policy." *Economics and Philosophy* 16(2): 323-331.

Levy, Amnon. 2002. "Rational Eating: Can It Lead To Overweightness or Underweightness?" *Journal of Health Economics*, 21, pp.887-899.

Liu, H.Y., Y. F. Lu, and WJ Chen. 1995. "Predictive Equations for Basal Metabolic Rate in Chinese Adults: a Cross-validation Study," Journal of the American Diet Association 95:12 (Dec) pp.1403-8.

Loewenstein, George. 1992. " The Fall and Rise of Psychological Explanations in the Economics of Intertemporal Choice," in George Loewenstein and Jon Elster (eds.), *Choice over Time*. New York: Russell Sage Foundation, pp. 3-34.

Willard G. Manning, Emmett B. Keeler, Joseph P. Newhouse, Elizabeth M. Sloss, and Jeffrey Wasserman. 1991. *The Costs of Poor Health Habits*, A RAND Study, Cambridge: Harvard University Press

McArdle,William, Frank Katch, and Victor Katch. 1996. Exercise Physiology 4th edition Baltimore: Williams and Watkins

Melby, Christopher, S. Renee Commerford, and James Hill. 1998. "Exercise, Macronutrient Balance, and Weight Control," in David Lamb, and Robert Murray, editors. Exercise, Nutrition and Weight Control. Perspectives in Exercise Science and Sports Medicine, Vol 11. Carmel, Indiana: Cooper Publishing, pp.

1-55.

Mitchell, Mary Kay.1997 .Nutrition Across the Life Span. Philadelphia: Harcourt Brace (Saunders)

Mokdad, AH Bowman BA, Ford ES, et al. The continuing epidemics of obesity and diabetes in the United States. *JAMA*. September 12, 2001; 286(10):1195-1200.

Must, Aviva, Jennifer Spadano, Eugenie H. Coakley, Alison E. Field, Graham Colditz, William H. Dietz, (1999). "The Disease Burden Associated With Over weight and Obesity." *JAMA*. 282: 1530-1538.

Philipson, Tomas J. and Richard A. Posner 1999. "The Long-run Growth in Obesity as a Function of Technological Change" NBER Working Paper 7423. Cambridge, MA: NBER

Poehlman, Eric and Edward Horton. 1992. "Determinants of Body Weight Regulation,"in Kelly Brownell, Judith Rodin and Jack Wilmore, editors, Eating, Body Weight and Performance in Athletes. Philadelphia: Lea and Febiger, pp. 33-48.

Rising, Russell, Ingeborg Harper, Anne Fontvielle, Robert Ferraro, Maximilian Spraul and Eric Ravussin. 1994. "Determinants of Total Daily Energy Expenditure: Variability in Physical Activity," American Journal of Clinical Nutrition 59:4 (April) 800-4.

Sims, Ethan. 1988. "Exercise and Energy Balance in the Control of Obesity and Hypertension," in Edward Horton and Ronald Terjung, Editors, Exercise, Nutrition, and Energy Metabolism. New York: Macmillan, pp. 242-257.

Squires, Sally. 2001. "Most Diets Work, but Not for Long," The Washington Post , (January 10), p. A-1.

Strotz, R.H. (1956), Myopia and Inconsistency in Dynamic Utility Maximization , The Review of Economic Studies 23, 165-180.

Suranovic, Steven, Robert Goldfarb and Thomas Leonard. 1999. "An Economic Theory of Cigarette Addiction," Journal of Health Economics , 18, pp.1-29.

Taaffe,D.R., J. Thompson, G. Butterfield, R. Marcus. "Accuracy of Equations to Pedict Basal Metabolic Rate in Older Women," Journal of the American Diet Association 95:12 (Dec) pp.1387-92.

Tallmadge, Katherine. 2000. "Metabolism Mathematics," The Washington Post. Health Section (August 22), pp. 10-13.

Thaler, Richard. 1981. "Some Empirical Evidence on Dynamic Inconsistency." *Economic Letters* 8: 201-207.

Thaler, Richard. 2000. "From Homo Economicus to Homo Sapiens" *Journal of Economic Perspectives* 14(1) Winter: 133–141

Tverskaya,Rozalia, Russell Riding, Debra Brown, and Fima Lifshitz. 1998. "Comparison of Several Equations and Derivation of a New Equation for Calculating Basal Metabolic Rate in Obese Children," Journal of the American College of Nutrition 17:4 (August) pp. 333-6)

Vaughan, Linda, Francesco Zurlo, and Eric Ravussin. 1991."Aging and Energy Expenditure," American Journal of Clinical Nutrition, 53:4 (April), pp. 821-5.

Weingarten, Harvey. 1992. "Determinants of Food Intake: Hunger and Satiety," in Kelly Brownell, Judith Rodin and Jack Wilmore, editors, Eating, Body Weight and Performance in Athletes. Philadelphia: Lea and Febiger, pp. 17-32.

Whitney, Eleanor, Corinne Cataldo and Sharon Rolfes.1998. Understanding Normal and Clinical Nutrition. 5th edition Belmont, CA: West Wadsworth.

Willett, Walter. 1990 Nutritional Epidemiology. Oxford: Oxford Univ Press.

Wilmore, Jack and David Costill. 1999. Physiology of Sport and Exercise. 2nd Edition.Champaign, Ill.:Human Kinetics

Wolf, Anne and Graham Colditz. 1996. "Social and Economic Effects of Body Weight in the United States," American Journal of Clinical Nutrition, 63 (Suppl) (March) 466S-469S.

Wong, William, Nancy Butte, Albert Hergenroeder, Rebecca Hill, Janice Stuff, and E. Obr'ian Smith.1996. "Are Basal Metabolic Rate Prediction Equations Appropriate for Female Children and Adolescents?" Journal of Applied Physiology 81:6(Dec) pp. 2407-14

Young, Lisa and Marion Nestle. 2002. "The Contribution of Expanding Portion Sizes to the US Obesity Epidemic" *Research and Practice* 92(2): 246-49.