Exploring Price-Independent Mechanisms in the Obesity Epidemic

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ABSTRACT

Obesity is a rapidly growing epidemic in the United States and a major public health challenge worldwide. To counteract this epidemic effectively, better understanding of its *mechanisms* are needed—we must understand not just what factors play a role, but how and why they matter. Most studies to date have focused on prices, technology, and the general availability of food. Less attention has been paid to the roles of social influence and the physiology of energy balance—despite growing evidence that both play important roles. In this paper, we present some initial findings from our analysis of two non-price mechanisms for obesity: the physiology of dieting, and socially influenced weight changes.

We show how the core equations governing the physiology of weight change can generate many of the known facts about diet and weight gain, including: the difficulty of maintaining a diet over a long period, high rates of recidivism after dieting, and substantial individual heterogeneity in the success of different types of diets. Using a new quantitative index of recidivist temptation, we develop a range of novel diets.

The notion that social norms are implicated in the obesity epidemic is not new. However, we show how a simple *conformist* social mechanism alone can drive a sharp increase in average weight. For initial weight distributions satisfying criteria identified here—and met by U.S. obesity data—a simple "Follow the Average" (FTA) weight adjustment rule generates increased mean weight. Indeed, the general FTA process, discussed mathematically below, can generate a rich variety of dynamics beyond obesity, including oscillatory behavior for which no conformist explanation has been considered.

We argue that integrative models adding such social and physiological mechanisms to economic ones will provide deeper explanations of the observed dynamics of obesity and a powerful array of policy interventions tailored to specific communities and individuals within them. The paper concludes with a sketch of one such model.

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Obesity is a rapidly growing epidemic in the United States and many other countries worldwide, and represents a major public health challenge. To counteract this epidemic effectively, better understanding of its *drivers* are needed—we must understand not just what factors play a role but how and why they matter. By modeling the mechanisms at work, research can help identify the most effective allocation of scarce government and public health resources, design novel and counter-intuitive intervention strategies, and guide future empirical work.

Most studies to date have focused on prices, technology, and the general availability of food. Less attention has been paid to the role of social influences and of the physiology of energy balance—despite growing evidence that both the social and the physiological play important roles in individual decision-making about eating and exercise.

In this paper, we present some initial findings from our analysis of two non-price mechanisms for obesity—physiology and social norms—and propose a multi-level synthesis using agent-based modeling.

Background – The Facts of the Obesity Epidemic

We will focus on three sets of important facts about the obesity epidemic: the rapid growth in overall rates of obesity and overweight in the United States over the last three decades, the demographic disparities in incidence and clear evidence of a role for social norms, and finally the well documented problem of recidivism and heterogeneity in dieting success.

Rapid increase in overall incidence

Over the last three decades, the percentage of Americans classified as obese (body mass index, or BMI, of 30 or greater[1]) has more than doubled, to more than 30% (see Figure 1). Fully two thirds of Americans are overweight (BMI of 25 or greater). These increases have significant costs to public health, due to the strong links between unhealthy body weight and diabetes, high blood pressure, and high cholesterol—leading one public health researcher to recently call obesity "the gravest and most poorly controlled public health threat of our time." [2]

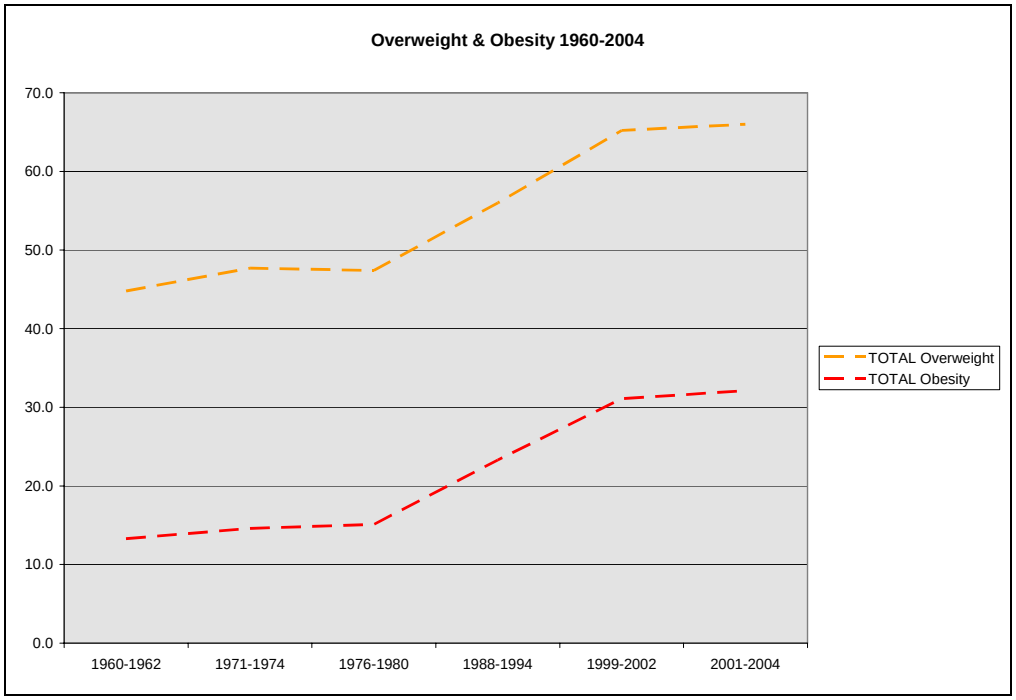

Figure 1 – *Rise in incidence of overweight/obesity in the United States. Source: CDC.*

An especially disturbing problem is the increase in childhood obesity, which has direct health and self-esteem costs and is also strongly linked to obesity in adulthood. In the past three decades, the percentage of children who are considered overweight or

[1] BMI is defined using the ratio of weight to height squared (BMI= 703*lb/in^2). [1]

obese has doubled (from 15% to nearly 30% by most estimates)[2]. Research in the U.S. shows that the odds of adult obesity increase with childhood BMI, that the age of obesity onset in childhood is correlated with risk of obesity in adulthood, and that parental obesity is associated with a higher likelihood that obese children become obese adults.[3] Thus the dramatic rise in obesity among children and adolescents is an ominous portent of even larger increases in adult obesity in the future.

Demographic Disparities and the Role of Social Norms

Not only has the incidence of obesity and overweight risen rapidly, but important disparities in both aggregate incidence and in social norms of obesity between demographic groups persist. For example, obesity incidence in the U.S., and its growth, are not equivalent across socioeconomic cohorts or racial groups.[4] Incidence of obesity remains highest among those near the poverty line [7-8]. Among children, the highest BMI is in families with incomes just over the poverty line, while children from both poorer and richer families are less likely to be overweight [9].

In addition, incidence of overweight and obesity is heavily skewed by race and gender (see Figure 2). Approximately 76% of Black and Mexican-American adults are overweight or obese [10], with incidence among black females nearing 80%--compared to incidence of 58% among white females. Among adolescents, studies also show that minorities engage in consistently higher levels of sedentary behavior than do their white counterparts [11].

[2] Discussion of childhood obesity rates is qualified, as the definition of obesity in children is controversial. However, there is wide agreement that the weight distribution among children is shifting upward, and that the increases are cause for concern [3].
[3] For example, the probability that a child who is obese at age 3-5 will be obese as an adult grows from 24% if neither parent is obese to 62% if one of the parents is obese [4].
[4] Obesity trends in European countries such as France and Germany exhibit similar patterns [5, 6].

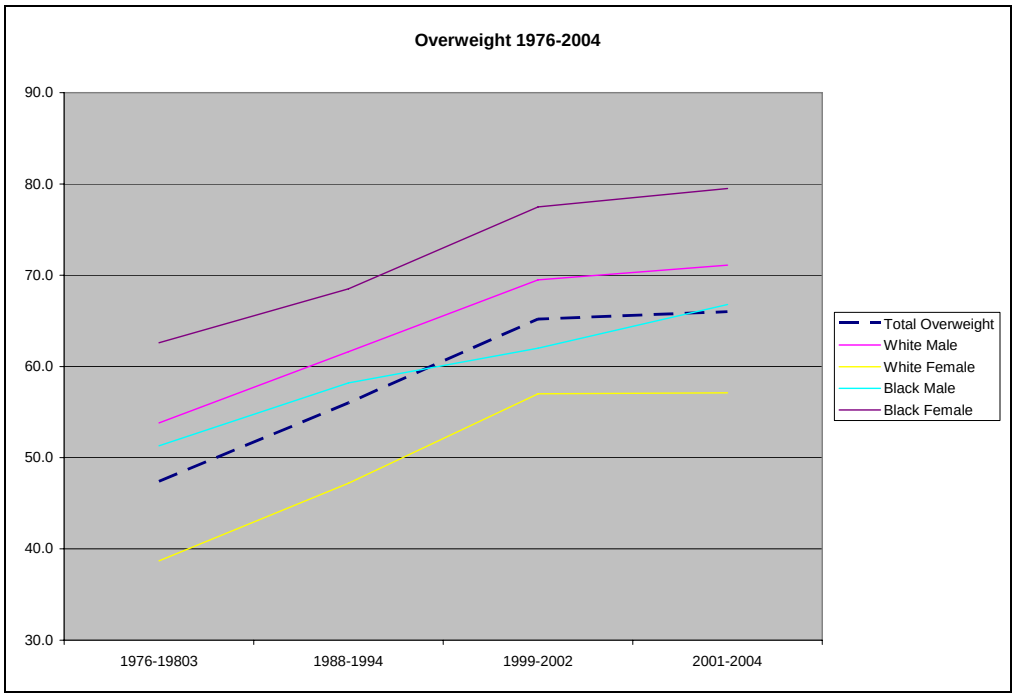

Figure 2 – *Increase in US overweight/obesity by race and gender. Source: CDC.*

There is strong evidence that *social norms* regarding body type play important roles in explaining these disparities among socioeconomic and racial groups--an individual's "ideal weight" often reflects social influences. Several studies find that the cultural ideal for black women, in particular, is heavier than for other groups (see [13-17])—black women who are at least 20 lbs overweight often do not consider weight a problem [18-19]. In general, dissatisfaction with body weight (resulting from discrepancy between current and ideal body type) occurs at a lower BMI in white women than for black and Hispanic women [20]. Dissatisfaction begins among white women at an average BMI of 24.6 (below the BMI=25 cutoff for overweight), but at an average of 29.2 and 28.5, respectively, for black and Hispanic women (well above the cutoff for overweight).

Adolescents' perceptions of size also vary significantly by race and gender—with black females seven times more likely than white to say they were not overweight,

despite the reality of much higher incidence of overweight and obesity among black females [12]. White females reported wanting to be smaller than their current size and felt encouraged to lose weight by significant others, while black females tended to feel happy with their current size and reported that their size was considered positive by significant others. Related research suggests substantial variation in obesity-related discrimination due to differences in ideal weight norms [8, 21].

Recidivism and Heterogeneity in Dieting

Central to efforts to reduce obesity and overweight is the topic of dieting. Although there is much written in the popular media about dieting, there have been relatively few careful longitudinal studies of diet until recently. Recent work has confirmed three "stylized facts" about dieting that are familiar from popular experience. First, gains from dieting are often short-lived because rates of recidivism (a tendency to regain weight) are generally very high. Although evidence of short-term gains from dieting is abundant, there is little evidence of lasting benefit. The few studies that have used long-term follow-ups find that the majority of individuals regain "virtually all of the weight" lost through dieting [39]. Second, the most crucial factor for weight loss and improved health through diet appears to be *adherence* to the diet regimen—not the specific foods eaten—and adherence rates fall to very low levels over periods of time approaching a year [40]. The same studies confirm a third fact: different diet types seem to work best for different people, although no clear categorization of either diets or types of individuals has been derived.

Previous Approaches to Modeling Obesity

Much of the research on obesity has focused on changes in the price and technology of food as the main explanatory factors [22-24]. Such studies attribute the increase in obesity in the U.S. to the declining prices of food and the increasing convenience of preparing home-cooked meals [see 22]. Similar arguments are made throughout the literature on obesity, and prices are almost certainly part of the story. In our view, however, they cannot fully explain either the rise in obesity or (especially) the variance in incidence across cohorts.

Recent empirical research lends weight to the argument that the current obesity epidemic cannot be reduced to a single explanation (such as changes in the price of food), but rather results from a complex interaction of social, physiological, and economic factors. For example, a recent study in the New England Journal of Medicine demonstrates empirically that social networks play an important role in the spread of obesity [41]. Other research has demonstrated that models can do a significantly better job of explaining changes in obesity incidence and distribution by including social norms in individuals' utility functions than by focusing on food price and ease of preparation alone [7]. Studies of motives for dieting also confirm that decision-making about eating cannot be reduced to economic factors alone [25]. And, indeed, the role of social norms in eating behaviors has been explicitly documented in recent experimental work [26].

In addition, the role of physical activity and exercise has often been overlooked in research on obesity, which has a long tradition of focusing on eating behavior and on the prices of *food* [27]. Yet examination of the key physiological processes of energy balance (see below) shows clearly the important role of exercise and physical activity in

achieving and maintaining healthy weight, and there is growing evidence that the effective cost of *exercise* has gone up over the same period as obesity has increased. In fact, the upward trend in obesity and overweight in the United States is mirrored by a *downward* trend in physical activity, and the same socioeconomic and racial disparities observed in obesity incidence can be found in the data on physical activity [28].

In sum, most previous research on obesity has focused on a small number of factors, especially prices and eating. There has been little attention to the role of social influence and norms, to the interaction of physiological realities with decision-making about eating, to the role of exercise, or to the impact of media and public health messages. Yet there is evidence that each of these factors may be important in understanding the rise in obesity and discrepancies in its incidence.

Analysis of Price-Independent Mechanisms

In this paper, we focus on price-independent, individually-based, and simple mechanisms for explaining the key facts of the obesity epidemic. The focus on price-independent mechanisms stems from our belief that prices alone cannot capture the full story; there is clear evidence that both social norms and physiology play important roles. Our focus on individually-based mechanisms comes from a belief that a truly explanatory model must provide a mechanism that can *generate,* or "grow" key phenomena from the bottom up. For a full exposition and defense of this generative explanatory standard, see [42]. Our focus on simple decision mechanisms derives from a large empirical and

psychological literature on bounded rationality [43], and from an interest in model parsimony.

We will begin our analysis with the core equations known to govern the physiology of human energy balance (weight loss and weight gain). We will show through straightforward analysis how these equations can generate many of the known facts about diet and weight gain, including: the difficulty of maintaining a diet over a long period, high rates of recidivism (tendency to regain weight), and substantial individual heterogeneity in the success of different types of diets (one size does not fit all).

We will then explore a simple model of social influence in decision-making about eating and weight, and demonstrate a mechanism through which ideal weight norms alone can drive a sharp increase in incidence of overweight and obesity—even beginning in a population which is predominantly of healthy weight.

I. Physiology

We have thoroughly surveyed the clinical nutrition literature for physiology equations. The Harris-Benedict equations [37] are generally accepted as empirically corroborated and reliable predictors of weight change as a function of daily caloric intake (C); basal metabolic rate [itself a specific function of current weight (w), height (h), and age (A)], and activity level (α). The equation governing female weight gain differs from that governing male weight gain, in respects which will prove directly relevant to policy. With the variables as just defined, and one pound equal to 3500 calories, the equations are:

$$\Delta w_{male} = \frac{C - \alpha[66 + 6.23w + 12.7h - 6.8A]}{3500}$$

$$\Delta w_{female} = \frac{C - \alpha[655 + 4.35w + 4.7h - 4.7A]}{3500}$$

Immediate Implications of Physiology

Virtually everyone acquainted with dieting will agree on two points: first, that the last ten pounds is harder to lose than the first, and second, and that having lost weight, it is difficult to keep it off. And, indeed, careful empirical studies confirm these phenomena [39-40]. It is not widely appreciated, however, that both these well-known patterns are predicted by the Harris-Benedict equations.

While these results can be proved in the general case (see Appendix I), an exposition by numerical example may be more transparent. So, posit a representative 40-year old female, of height 5'4", with a moderate activity level[5], and initial BMI of 30 (i.e., 175 lbs). From the equations, we compute that a daily caloric intake of 2292 calories will exactly maintain this BMI.

Suppose her goal is to lose 1 lb per week until her BMI is 20. Since 1 lb equals 3500 calories, she must eat 500 calories per day *fewer than the maintenance level* at every point in her weight loss program. The problem is that, as her weight falls, her maintenance caloric level also falls (because her BMR does), and with it the daily calories she is permitted if weight loss is to continue.

[5] PAL (Physical Activity Level) for moderate activity level derived from Black et al.'s 2004 analysis of 500+ distinct doubly-labelled water studies [38]

Continuing numerically, 2292 was her day 1 maintenance level. So, for the first week, she must cut 500 per day, to 1792 cal/day. After a week, she'll have lost 1 lb. But, her BMR has fallen, as per the Harris-Benedict equations shown above. So, she must cut her daily caloric intake further to keep losing. And so it goes every week. After 59 weeks, her BMI reaches 20. But to lose that last pound (to drop from 118 to 117) she needed to consume a mere 1405 calories per day for the final week. So, it is indeed much harder to lose the last pound (1405) than the first (1792).

Why is it hard to keep it off? To reach the goal of 117 lbs, our subject reduced her daily caloric intake to 1405. However, from the equations, we calculate that *maintenance* caloric intake for this final weight is actually 1905. Notice that this exceeds the caloric intake from the first day of the diet, which was 1792. And, most importantly, it exceeds by more than 35% the intake (1405 calories) on the final day of the diet.

This means that, having lost her weight, the subject could go back to eating at her pre-diet level of 2292 calories per day for almost a week and a half before gaining even one full pound. This is why it is easy to "fall off the wagon" for weeks with virtually no visible effect on weight. By then old eating habits may be re-established; one overshoots the mark, and regains the weight. Thus, the equations also offer insight into the familiar pattern of so-called "yo-yo" dieting. This is why it is hard to keep weight off.

Temptation and Psychological Heterogeneity

The crux of this problem is that maintenance calories exceed current intake during the diet. So, as one loses weight, one can, in fact "get away with" eating more. Hammond formalizes this by defining "temptation" on any day t as follows:

$$\text{Temptation} = \frac{(\text{Maintenance\_calories} - \text{Current\_caloric\_intake})}{\text{Current\_caloric\_intake}}$$

This is *the percent by which one could increase calories above the current level without gaining weight.* We will shortly demonstrate and exploit the fact that the temptation trajectory over time is diet dependent.

Temptation Trajectories of Selected Diets

We have devised and begun to explore a number of diets.

CLR Diet.

The diet used in the numerical example above is a constant loss rate [CLR] diet (1 lb lost per week). It can be shown that for CLR diets, (a) Temptation to cheat increases (strictly) as weight falls, (b) that it does so at an increasing rate, and (c) that it attains its maximum at the lowest weight. This is shown in Figure 3 below (and demonstrated analytically in Appendix I).

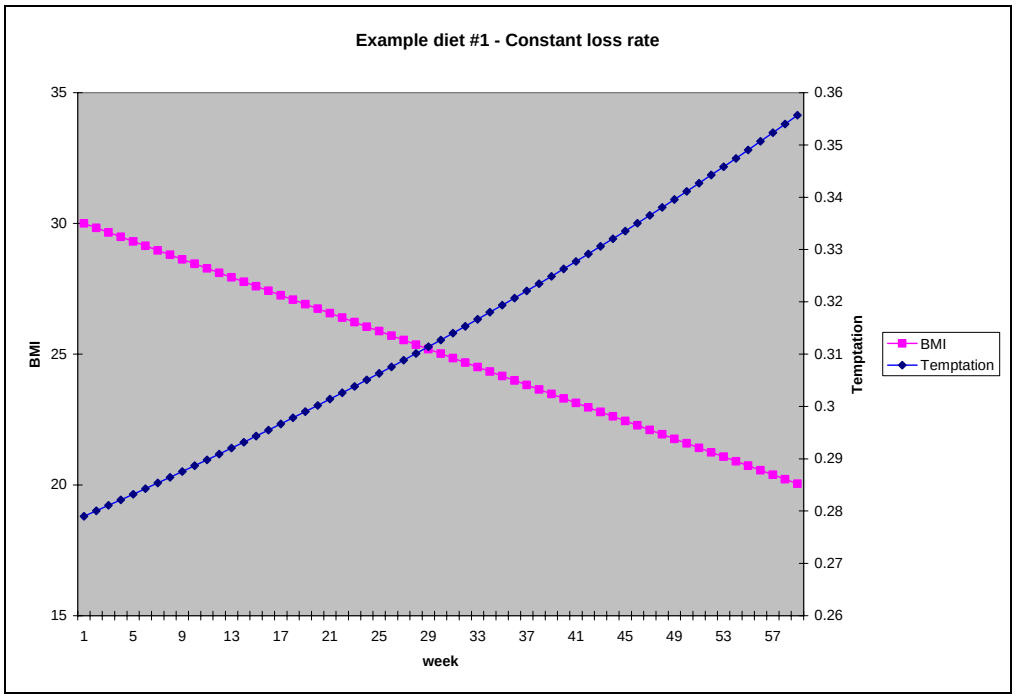

Figure 3. *BMI vs. Temptation for the CLR Diet*

We have said little about individual heterogeneity in this connection. People certainly differ in their capacity to cope with temptation. They also differ in the urgency with which they must lose weight. For some, it can be a serious medical imperative. Modeling permits us to design different diets, and consider how well-suited they are to different individuals, given their medical situation and psychological profile. We just considered the Constant Loss Rate (or CLR) diet, from our numerical example.

CEL Diet

A second type of diet would specify a constant eating level, or CEL. In general, one would pick a target weight and then use the Harris-Benedict equations to compute the maintenance daily calories for that weight. One would simply cut one's eating to that level in one shot, and just stay there until the goal weight is attained. For the numerical example above, this CEL diet will take our subject from her initial BMI of 30 to her goal

of 20 if she cuts her initial daily intake by 500 calories and sticks to it. However, it will take her substantially longer than the CLR diet discussed earlier.

CTL Diet

Yet a third type of diet would sustain a constant temptation level (CTL). Here, one would fix (perhaps based on MRI studies or other evaluations) a sustainable level of Temptation (defined technically as above). Each week, one would reduce daily caloric intake as much as possible without exceeding this constant temptation level. In our numerical example, the diet then ends when the target BMI of 20 is attained.

For our 40 year old female numerical example, the BMI trajectories for these three diets are plotted in Figure 4.

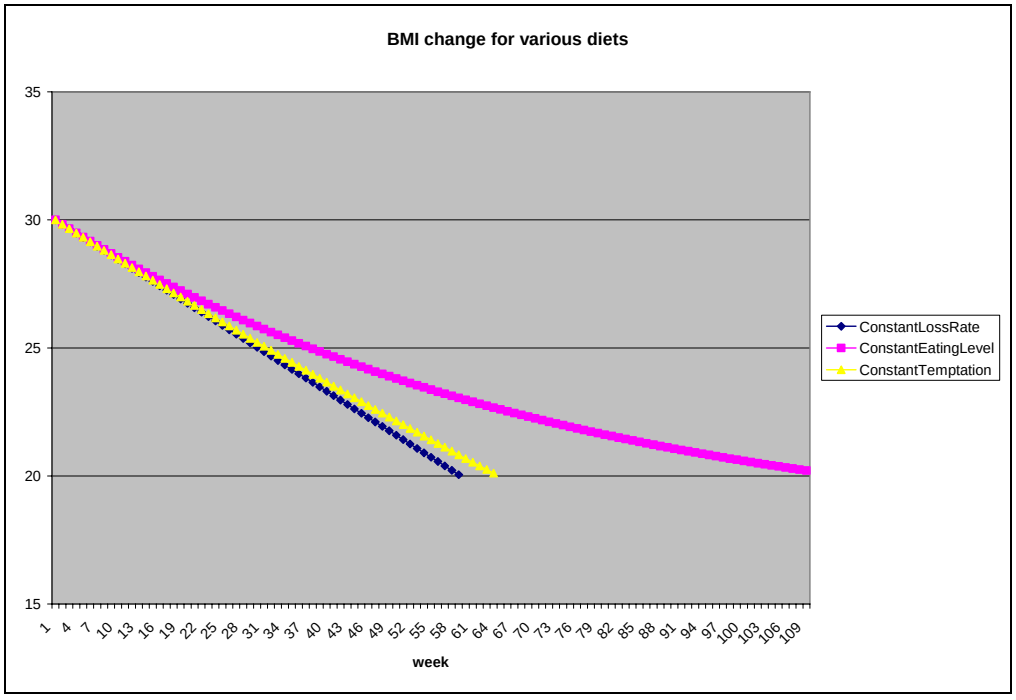

Figure 4. *BMI over time for three diets.*

The main thing to notice is that they can all succeed, but that the CLR diet is fastest, that the CTL diet is slower by roughly two months, and that the CEL diet is by far the slowest, requiring more than two years to complete.

The more surprising, and potentially policy-relevant, result of this exercise is the radically different temptation trajectories associated with these diets (Fig 5).

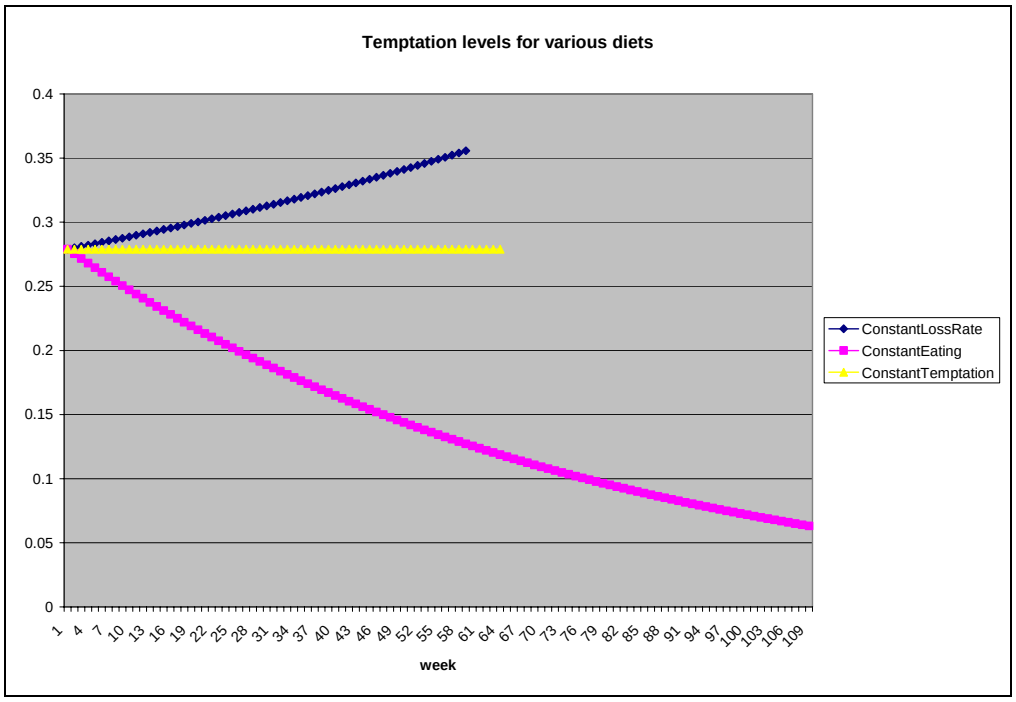

Figure 5. *Temptation Trajectories for three diets.*

By design, the constant temptation diet [CTD] produces a horizontal temptation trajectory. The constant loss rate diet [CLR] by contrast, entails ever-increasing temptation. In this sense, it is the "tougher" of the two. But it is also somewhat faster, as shown in figure 4. Whether the increased temptation is worth incurring would depend on the medical urgency with which one must lose, and the prospects of success given one's psychological capacity to endure the rising temptation curve. Diametrically opposite is the CEL diet. Here, temptation actually falls over time. The diet becomes progressively

easier to stay on. But weight loss is dramatically slower on this diet than the others. Again, if one's psychology precludes success on the rising temptation [CLR] diet, this slower diet may be indicated. However, one's obesity may pose an immediate health threat requiring sharp near-term weight loss, and a different diet. We have also explored further diets, for example, the "punctuated equilibrium" diet in which sustained periods of rigorous caloric restriction are punctuated by short episodes of high caloric intake.

Gender Differences

Individual heterogeneity in the effectiveness of diets is not limited to psychological or health differences. Other demographic differences may also be significant. For example, the calculations above were for a single representative female. However, the Harris-Benedict equations clearly show important differences by sex. For example, the constant multiplier on alpha, the activity level, is 66 for men, but 655 for women, higher by a factor of literally ten. So, let us consider the same three diets treated above, CLR, CEL, CTL, but do each analysis separately for men and women. First we show the BMI trajectories; then the Temptation trajectories, broken out by gender. Numerous differences are evident in Figure 6.

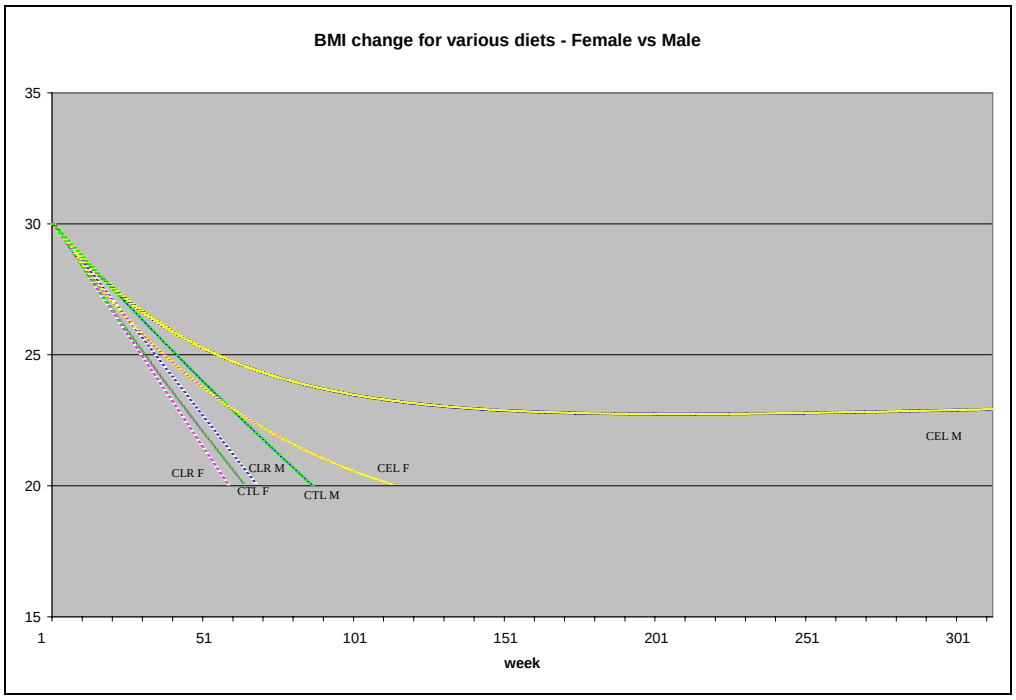

Figure 6. *BMI Trajectories for Selected Diets by Gender*

Perhaps the most startling is the gender difference in effectiveness of the CEL diet: females reach the 20 BMI goal in 110 weeks, while males never attain it. In turn, temptation is consistently higher in women than in men, as shown in Figure 7.

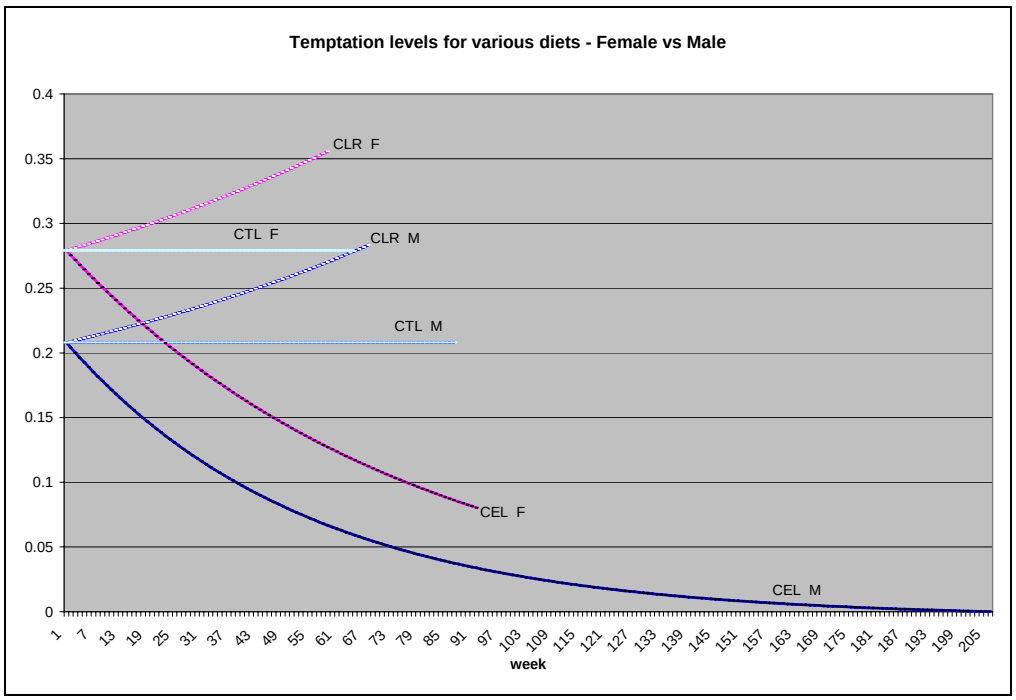

Figure 7. *Temptation Trajectories for Selected Diets by Gender.*

Obviously, these particular results depend on the specific numerical assumptions made. But it is abundantly clear that gender is an important heterogeneity—among others--to include [40]. This emerges clearly from the Harris-Benedict equations, which compactly capture individual physiological aspects of the problem.

II. Social Influences

Growing empirical evidence makes it clear that *social* influence also plays an important role in obesity [7,12-21,26,41]. The mechanisms and dynamics of social influence are not well understood, however, and it is clear that social influence can take many different forms. Social groups (e.g., blacks and whites) may vary widely in their weight target and, in turn, their habits and lifestyle choices regarding diet and exercise [7, 12-16]. Group-specific expectations of economic opportunity can also affect concern with weight, and its impact on happiness, that is, individual appraisals of well-being [8, 12, 17-20, 35, 36]. There can also be vicious cycles between the social and physical, as when overweight children avoid (or are ostracized from) participation in sports, exacerbating their obesity [32-34.] Children and adolescents may be influenced by peer networks, by parents, and by messages received from school and media. These all may play a role in shaping the individual's weight goals and behavior.

While a full treatment of social influences will be important in designing a comprehensive model (see below), an extremely simple example shows the fundamental, and counter-intuitive, role they can play. Our analysis of core *physiological* mechanisms

showed how they can generate one set of key facts about obesity, namely the dynamics of dieting. Recall that another central fact documented in the obesity literature is the crucial upward trend in BMI (Figure 1). Is there a simple plausible *social* mechanism that may be implicated in this trend? We believe so.

Follow the Average (FTA)

People routinely compare themselves to others in their social group. Often, they seek simply to conform. One would not think that the impulse to simply "be average" could produce dynamics of any interest, or increases in the average itself. Intuition would surely suggest rapid convergence to the initial mean. This intuition is correct if the initial distribution is symmetrical about the mean. But it is *not* necessarily correct if the distribution is skewed, as we will show. This is of particular relevance in the context of obesity, since the real weight distribution in the United States *is* highly skewed. In circumstances of this nature, what does happen if everyone seeks simply to "follow the average?"

We will illustrate by numerical example (for a full analytical discussion of the general case, see Appendix II). To begin, let us assume the following initial weights among five people:

Individual weights: 100 100 100 100 150 Mean = 110.

Let us suppose now that every person wants to be like the average. For simplicity, we'll assume that weights can be directly adjusted (instead of invoking more complex

physiology) but, as in the real world, the adjustment rate is limited—they can only adjust their weight up or down by 5 lbs per period. Each individual's weight adjustment rule, then, is:

If my weight > average, lose 5 lbs.
If my weight < average, gain 5 lbs. [6]
If my weight is exactly equal to the average, maintain it.

How do things unfold?

The first four people are below average (at 100 lb), so they increase by 5 lbs, while the 150 lb person is above the mean, so she reduces by five. This produces a new distribution:

Individual weights: 105 105 105 105 145 Mean = 113.

Note that the mean has gone up, even though movement is toward the center (average). The next round of "mean seeking" follows a similar pattern—the first four people are below the average and increase their weight, while the fifth is above the average and decreases. This yields:

Individual weights: 110 110 110 110 140 Mean = 116.

Notice that four out of five have now attained the initial mean of 110. But that is no longer the group average, which has grown to 116. Thus, the process of adjustment will continue.

[6] We need not posit that people actively increase their weight. One might argue that, for those below the average, the forces balancing temptation (as technically defined earlier) are relatively weak, so the normal impulse to overeat prevails and they gain.

Ultimately, there is indeed convergence to an average, but it is *not* the average obtaining at the start (110)—in fact, it is 125 (see Table 1).

Individual Weights					***Mean***
100	100	100	100	150	*110*
105	105	105	105	145	*113*
110	110	110	110	140	*116*
115	115	115	115	135	*119*
120	120	120	120	130	*122*
125	125	125	125	125	*125*

Table 1. Numerical illustration of a rising mean under the FTA mechanism.

When initial distributions are skewed, then, the simplest conformist impulse—match the mean—actually *overshoots* its initial objective, *increasing* the mean.

It is important to note that the linear increase in the average weight is altogether consistent with the abrupt increase in percentage overweight (and obese) observed in the data (in Figure 1)[7].

The result can be proved more generally for any unimodal distribution that meets certain criteria (see Appendix II). This sort of *social* mechanism can compound the physiological ones discussed above to produce dynamics comparable to those of the obesity epidemic. Calibration of the integrated social-physiological model to those data is an important topic for future research.

[7] Imagine that the cutoff for overweight in our example was 120. While the mean grows linearly, the percentage above 120 jumps sharply at a particular stage (in the next to last row).

While the "follow the average" (FTA) process is of clear relevance to obesity dynamics, it may well underlie other forms of social adjustment where the impulse to conform eventuates in unexpected dynamics. As the analysis in Appendix II notes, the process can produce a rich array of dynamics, including cycling as well as convergence. This suggests that FTA may be implicated in phenomena far beyond obesity, including political and economic dynamics.

Toward an Integrative Model of Obesity

In this paper, we have shown how several simple, individually-based, non-price mechanisms (including norms, social influences, and physiology) can provide insight into the obesity epidemic. The Harris-Benedict equations alone explain core facts of individual obesity and dieting (such as the familiar pattern of recidivism), while the simple "follow the average" social adjustment mechanism produces a powerful, and counter-intuitive upward BMI dynamic in the population. These results are notable in their own right, and highlight the importance of non-price mechanisms in the obesity epidemic. A fully integrated model will offer a still deeper explanation of the observed dynamics and a powerful array of policy interventions tailored to specific communities and individuals within them. In carrying this line of work forward, we plan to exploit the technique of agent-based modeling ABM [29,42].

Gedankenmodel

Somewhat more specifically, we imagine an ABM in which individual children/adolescents are embedded in a social structure where they are influenced by parents, by peers, and by media. Different social groups might have different exemplars for ideal weight. Social conformity toward the group ideal weight (as in the FTA mechanism) would be an important driver of individual behavior. Depending on the network structure of contacts and influence, stigmatization of obesity might occur and could lead to divergent behaviors—including dieting, increased overeating (75-80% of subject in one study self-report this response to weight stigmatization [31]), or avoidance of physical activities [32-34]. Heterogeneity in diet choice and diet outcome (as analyzed above) would play an important role. Individual behavioral responses would, in turn, affect changes in individual weight according to detailed individual physiology (captured in the Harris-Benedict equations).

By including physiology, a complex social structure, diverse norms, and heterogeneity of behavioral responses, the model would attempt to "grow" observed empirical patterns and trends. Specific macro-level "targets" for the simulation model would include: the observed overall growth in obesity over time (and the shape of the growth rate, with acceleration in 1980); differing distributions of obesity in different demographic groups (e.g., black/white adolescent females); and different levels of subjective well-being for *equally obese* (same BMI) individuals from different groups (e.g. racial groups).

Having generated these in an agent model, one could more thoroughly and credibly explore the space of policy interventions best tailored to the communities at greatest risk.

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Appendix I: Algebraic Analysis of Diet and Temptation Results

A. CLR diet

Claim: Temptation on this diet is strictly increasing, and at increasing rate.

Proof:

Define "temptation" at any time[8] *t* to be

$$\tau = \frac{(TDEE_t - C_t)}{C_t} \ . \qquad [1]$$

Since 1 lb = 3500 cal, a constant loss rate of 1 lb. / week requires that 7(C-TDEE) = -3500. Thus

$$C_t = TDEE_t - 500 \qquad [2]$$

at each time step *t.* Substituting this into [1], we obtain

$$\tau_t = \frac{TDEE_t - (TDEE_t - 500)}{(TDEE_t - 500)}, \qquad [3]$$

which reduces to:

$$\tau_t = \frac{500}{(TDEE_t - 500)}. \qquad [4]$$

For the female case, this is

$$\tau_t = \frac{500}{\alpha[655 + 4.35w_t + 4.7h - 4.7A] - 500}. \qquad [5]$$

For the specific α, *h* and *A* values used in the numerical example, this simplifies to

$$\tau_t = \frac{1}{0.01w_t + 1.3} \ , \qquad [6]$$

[8] In Appendices I and II, we will sometimes suppress the explicit time dependence where it is obvious, for notational convenience.

which is obviously of the general form

$$\tau_t = \frac{1}{Aw_t + B}. \qquad [7]$$

Thus, as w_t decreases linearly the temptation will increase monotonically at an increasing rate, as was to be shown. Note that the male case is completely parallel.

B. CTL diet

Temptation for this diet is constant by definition.

C. CEL diet

Claim: Temptation on this diet is strictly decreasing, and at a decreasing rate.

Proof:

The proof proceeds in two steps. First, we show that temptation is linear in *w*; then, that *w* is strictly decreasing at a decreasing rate.

To begin, define temptation at any time *t* as in [1] above. The eating level C on this diet is fixed at the maintenance level for target BMI (a constant, *X*), which is to say that for all *t*

$$C = X = \alpha BMR(targetWeight). \qquad [8]$$

Substituting, the equation for temptation reduces to:

$$\tau_t = \frac{\alpha}{X}(BMR_t) - 1\ . \qquad [9]$$

Assuming α, *h*, *A*, and *X* to be constants, the equation is further reducible. For the female numerical example used in the paper, we obtain

$$\tau_t = 0.0043w_t - 0.28\,. \qquad [10]$$

So temptation is linear in *w*. Now we show that w decreases at a decreasing rate. By the female Harris-Benedict equation, during the course of this diet *w* changes as follows:

$$w_{t+1} = w_t + \frac{X - \alpha[655 + 4.35w_t + 4.7h - 4.7A]}{3500} \qquad [11]$$

Assuming α, *h*, *A*, and *X* to be constants, this equation is also further reducible. For the female numerical example used in the paper:

$$w_{t+1} = 0.9979w_t + 0.141262, \qquad [12]$$

which is of the general form

$$w_{t+1} = Aw_t + B, \text{ with } 0 < A < 1, \text{ and B positive.} \qquad [13]$$

Thus *w* decreases at a decreasing rate, and since temptation is linear in *w*, it does as well. The male case is parallel.

Analytical Appendix II: Social norms and "Follow the Average"

Two specific cases

To best elucidate the logic behind the result, we first present an analysis of two specific cases: one for a distribution with an odd number of elements (n=5) and one for a distribution with an even number of elements (n=4). For the general proof, see the section below.

Example 1: n=5

Start with a unimodal distribution comprised of five elements with values: $\{x_1, x_2, x_3, x_4, x_5\}$

Define $\tilde{x}$ = *median* of the set, and $\bar{x}$ = the *mean*. Assume that $\bar{x} > \tilde{x}$.

(1) By definition, half of these numbers are $\leq \tilde{x}$, and half are $\geq \tilde{x}$. As in any set with an odd number of elements, at least one of them must be equal to the median itself.

Without loss of generality, then, assign:

$$x_1, x_2 \leq \tilde{x}$$
$$x_3 = \tilde{x}$$
$$x_4, x_5 \geq \tilde{x}$$

(2) To apply our "follow the average" rule, individual element values change from one period to the next as follows: if below the average, increase by a fixed amount k; if above the average, decrease by k instead.

Since $\bar{x} > \tilde{x}$ for our sample, the *minimum* increase in weight will be as follows[9]:

$x_1 \rightarrow x_1+k$, since $x_1 \leq \tilde{x}$ (and $\tilde{x} < \bar{x}$)
$x_2 \rightarrow x_2+k$, since $x_2 \leq \tilde{x}$ (and $\tilde{x} < \bar{x}$)
$x_3 \rightarrow x_3+k$, since $x_3 = \tilde{x}$ (and $\tilde{x} < \bar{x}$)
$x_4 \rightarrow x_4-k$, since $x_4 \geq \tilde{x}$ (and so x_4 *might* be $> \bar{x}$)
$x_5 \rightarrow x_5-k$, since $x_5 \geq \tilde{x}$ (and so x_5 *might* be $> \bar{x}$)

(3) Even assuming x_4 and x_5 decrease in value, the distribution's mean will go up:

$$\bar{x}_t = \frac{x_1 + x_2 + x_3 + x_4 + x_5}{5}$$

$$\bar{x}_{t+1} = \frac{(x_1 + k) + (x_2 + k) + (x_3 + k) + (x_4 - k) + (x_5 - k)}{5}$$

[9] The arrow symbol means "updates to."

$$= \frac{x_1 + x_2 + x_3 + x_4 + x_5 + k}{5}$$

$$= \bar{x}_t + \frac{k}{5}$$

(5) By updating x_1 thru x_5 and recalculating $\tilde{x}$ and $\bar{x}$ in the new distribution, the same processes will repeat until $\bar{x} \leq \tilde{x}$. We now illustrate the even-numbered case.

Example 2: n=4

For an even number of elements $\{x_1 \ldots x_4\}$, the median is defined as the mean of the middle two elements. Without loss of generality, then, assign $x_1 \leq x_2 \leq x_3 \leq x_4$, so $\tilde{x} = \frac{x_2 + x_3}{2}$.

Under the "follow the average" rule:

$x_1 \rightarrow x_1+n$, since $x_1 \leq \tilde{x}$, and $\tilde{x} < \bar{x}$
$x_2 \rightarrow x_2+n$, since $x_2 \leq \tilde{x}$, and $\tilde{x} < \bar{x}$

What about x_3? Under the same logic as above (in the *n=5* case), x_3 must go up in order for the overall distribution average to go up.

Remember that $x_3 \geq x_2$. Thus there are two possibilities:

a. If $x_3=x_2$
Then $\tilde{x} = x_2 = x_3$
Since $\bar{x} > \tilde{x}$, $\bar{x} > x_3$ and x_3 will go up (and so will the average).

b. If $x_3 > x_2$

Under this condition, x_3 will go up only if $x_3 < \frac{x_4 + x_2 + x_1}{3}$.

Why? Under the rule, x_3 will go up only if $x_3 < \bar{x}$

$$x_3 < \bar{x}$$

$$x_3 < \frac{x_1 + x_2 + x_3 + x_4}{4}$$

$$3x_3 < x_1 + x_2 + x_4$$

$$x_3 < \frac{x_4 + x_2 + x_1}{3}$$

This cannot be proved to be true in all cases[10], so it becomes an additional condition. Its generalization is [A2] below. We now proceed to the general case.

[10] Since $\bar{x} > \tilde{x}$, we can show that $x_4 > x_3+x_2-x_1$. But, $x_3+x_2-x_1 < 3x_3-x_1-x_2$ so we cannot show if $x_4 > 3x_3-x_1-x_2$

General case

To set this up, we define the following[11]:

n = number of elements in a distribution
k = step size of adjustment of individual elements between periods under the FTA rule
$\bar{x}$ = arithmetic mean of a distribution
$\tilde{x}$ = median of a distribution

Proposition: *In a discrete unimodal distribution, the FTA ("follow the average") process will result in a monotonic increase in the mean, as long as two assumptions hold:*

[A1] The arithmetic mean of the distribution is strictly greater than its median (e.g. the distribution has positive skew).

[A2]
$$x_{\frac{n}{2}+1} < \frac{\sum_{i=1}^{\frac{n}{2}} x_i + \sum_{i=\frac{n}{2}+2}^{n} x_i}{(n-1)}$$

In fact, [A2] is needed only for the even-numbered element case, as shown below. For the odd-numbered elements case, [A1] is sufficient.

Proof:

Begin with a discrete unimodal distribution. The definition of the median in any discrete distribution differs depending on whether n is even or odd. Therefore there are two cases:

Case I. An odd number of elements

(1) In a distribution with an odd number of elements $\{x_1, x_2, \ldots, x_n\}$, the median is defined as the ((n+1)/2)th element:

$$\tilde{x} = x_{\frac{n+1}{2}}$$

By definition, half of the elements in the distribution are $\leq \tilde{x}$, and half are $\geq \tilde{x}$.

Without loss of generality, then, order the elements from lowest (x_1) to highest (x_n), so:

$$x_1, x_2, \ldots, x_{\frac{n-1}{2}} \leq \tilde{x}$$
$$x_{\frac{n+1}{2}} = \tilde{x}$$

[11] Again, where obvious, explicit time dependence is suppressed to reduce notational clutter.

$$x_{\frac{n+2}{2}}, ..., x_n \geq \tilde{x}$$

(2) To apply the FTA rule, individual element values change from period *t* to *t+1* as follows:

$$\text{If } x_i(t) < \bar{x}, \text{ then } x_i(t+1) = x_i(t) + k$$
$$\text{If } x_i(t) > \bar{x}, \text{ then } x_i(t+1) = x_i(t) - k$$
$$\text{If } x_i(t) = \bar{x}, \text{ then } x_i(t+1) = x_i(t)$$

(3) Since $\bar{x} > \tilde{x}$ (under [A1]), the following changes occur in the first period:

(i) For *i* =1 to $\frac{n-1}{2}$, $x_i(t+1) = x_i(t) + k$ because $x_i \leq \tilde{x}$ and $\tilde{x} < \bar{x}$

(ii) For *i* = $\frac{n+1}{2}$, $x_i(t+1) = x_i(t) + k$ because $x_{\frac{n+1}{2}} = \tilde{x}$ and $\tilde{x} < \bar{x}$

(iii) For *i*= $\frac{n+3}{2}$ to *n*, we may have $x_i(t+1) = x_i(t) + k$, or $x_i(t+1) = x_i(t) - k$, since $x_i \geq \tilde{x}$ (and so x_i might be > or < $\bar{x}$)

(4) Regardless of the direction of movement in (iii), the mean will increase from period *t* to period *t+1*. A *lower bound* on the increase in mean can be calculated as follows. The mean at time *t* is

$$\bar{x}(t) = \frac{\sum_{i=1}^{n} x_i(t)}{n} = \frac{x_1(t) + x_2(t) + ... + x_n(t)}{n}.$$

Updating,

$$\bar{x}(t+1) = \frac{[\sum_{i=1}^{\frac{n+1}{2}} (x_i(t) + k)] + [\sum_{i=\frac{n+3}{2}}^{n} (x_i(t) - k)]}{n}$$

$$= \frac{(x_1(t) + k) + (x_2(t) + k) + ... + (x_{\frac{n+1}{2}}(t) + k) + (x_{\frac{n+3}{2}}(t) - k) + ...(x_n(t) - k)}{n}$$

$$= \frac{x_1(t) + x_2(t) + ... + x_n(t) + k}{n}$$

$$= \bar{x}\ (t) + \frac{k}{n}.$$

(5) The process will repeat for the new distribution $\{ x_1(t+1), x_2(t+1), ..., x_n(t+1) \}$, new mean $\bar{x}\ (t+1)$, and new median $\tilde{x}\ (t+1)$, and will continue to repeat until $\bar{x} \le \tilde{x}$, in which case [A1] no longer holds. Cycling and other dynamics are possible in such cases. A full analysis is forthcoming.

Case II. An even number of elements

If there is an even number of elements, assumption [A2] is needed:

[A2] $$x_{\frac{n}{2}+1} < \frac{\sum_{i=1}^{\frac{n}{2}} x_i + \sum_{i=\frac{n}{2}+2}^{n} x_i}{(n-1)}$$

(1) In a distribution with an even number of elements $\{x_1, x_2, \ldots, x_n\}$, the median is defined as the average of elements (n/2) and (n/2)+1:

$$\tilde{x} = \frac{x_{\frac{n}{2}} + x_{\frac{n}{2}+1}}{2}$$

As before, half of the elements in the distribution are $\le \tilde{x}$, and half are $\ge \tilde{x}$.

Without loss of generality, then, number elements from lowest (x_1) to highest (x_n), so:

$$x_1, x_2, ..., x_{\frac{n}{2}} \le \tilde{x}$$
$$x_{\frac{n+1}{2}}, ..., x_n \ge \tilde{x}$$

(2) To apply the "follow the average" rule, individual element values change from period t to $t+1$ as follows:

$$\text{If } x_i(t) < \bar{x}, \text{ then } x_i(t+1) = x_i(t) + k$$
$$\text{If } x_i(t) > \bar{x}, \text{ then } x_i(t+1) = x_i(t) - k$$
$$\text{If } x_i(t) = \bar{x}, \text{ then } x_i(t+1) = x_i(t)$$

(3) Since $\bar{x} > \tilde{x}$ (under [A2]), the following changes occur in the first period:

(i) For i =1 to $\frac{n}{2}$, $x_i(t+1) = x_i(t) + k$ because $x_i \le \tilde{x}$ and $\tilde{x} < \bar{x}$

(ii) For i= $(\frac{n}{2}+1)$ to n, we may have $x_i(t+1) = x_i(t) + k$, or $x_i(t+1) = x_i(t) - k$, since $x_i \ge \tilde{x}$ (and so x_i might be > or < $\bar{x}$)

In order for the Proposition to be true, at least element $x_{\frac{n}{2}+1}$ must increase by k (rather than decreasing by k), following the same logic as in I (4) above.

This requires:

$$x_{\frac{n}{2}+1} < \bar{x}$$

$$x_{\frac{n}{2}+1} < \frac{x_1 + x_2 + \ldots + x_n}{n}$$

$$x_{\frac{n}{2}+1} < \frac{[\sum_{i=1}^{\frac{n}{2}} x_i] + x_{\frac{n}{2}+1} + [\sum_{i=\frac{n}{2}+2}^{n} x_i]}{n}$$

$$n(x_{\frac{n}{2}+1}) < [\sum_{i=1}^{\frac{n}{2}} x_i] + x_{\frac{n}{2}+1} + [\sum_{i=\frac{n}{2}+2}^{n} x_i]$$

$$(n-1)(x_{\frac{n}{2}+1}) < [\sum_{i=1}^{\frac{n}{2}} x_i] + [\sum_{i=\frac{n}{2}+2}^{n} x_i]$$

$$x_{\frac{n}{2}+1} < \frac{\sum_{i=1}^{\frac{n}{2}} x_i + \sum_{i=\frac{n}{2}+2}^{n} x_i}{(n-1)},$$

which is precisely condition [A2]. This completes the proof.