A DYNAMIC MODEL FOR UNDERSTANDING EATING DISORDERS

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Abstract. A system dynamics model is presented which integrates current knowledge on the various aspects of normal and abnormal weight control and which provides new insights into the mechanisms underlying certain eating disorders. Anorexia nervosa, in both its purging and non-purging variants, emerges from the model as a behavior pattern tied up with the fear of weight gain which serves to strengthen the individual's drive toward extreme slimness. Policy tests suggest that appetite-suppressing drugs may be helpful in reducing this fear and its negative physical consequences. The encouragement or discouragement of physical activity may also serve the goal of stabilizing the individual, depending on how different therapeutic objectives are weighted for the specific individual. Future research may take the form of model enhancement or of empirical studies guided by the model's structure and behavior.

BACKGROUND

"Our whole society is so preoccupied with slimness that there is a need to draw attention to the fact that many can achieve it only at a great sacrifice of health and competence." (Bruch 1973, p. 384)

The eating disorders known as anorexia nervosa and bulimia have gained much attention in recent years and are currently considered major public health threats in the United States and elsewhere in the developed world. Anorexia is characterized by extreme weight loss, intense fear of gaining weight, and denial of being emaciated or even underweight. Bulimia (or "binge-purge syndrome") is characterized by frequent episodes of binge-eating followed by purging via self-induced vomiting or the use of laxatives, enemas or diuretics. These potentially fatal disorders typically affect adolescent girls and young women from well-to-do families and with no prior history of serious medical problems. Within this population, anorexia and bulimia may have a combined prevalence rate in the U.S. as high as ten percent. Treatment results have, in general, been unsatisfactory over the long term.

Of the two syndromes, anorexia is by far the more studied to date and consequently the better understood. Indeed, bulimia has only recently been recognized as a syndrome distinct from anorexia and is probably of greatest clinical concern when appearing in conjunction with anorexia. It is estimated that 30-50% of all anorexics are also bulimic (Crisp 1980, Halmi 1982, Polivy & Herman 1985). Both syndromes may be considered examples of voluntary dieting gone berserk and are essentially psychological in origin. Where "pure" anorexics tend to be quite lonely and emotionally restrained, bulimics tend to be more outgoing and impulsive, often involved with alcohol or drugs and/or sexually active at an early age. Pure anorexia also appears to have a better prognosis than bulimic anorexia, tending to start earlier in adolescence and to respond with more lasting effect to medical attention.

Typical Pattern of Development

As an infant, the needs of the anorexic-to-be are not met appropriately by her mother, who, for example, may turn to food as a cure-all for the crying child. As a result, her senses of self-identity and self-effectiveness do not develop normally as a child. Instead, she takes her cues almost entirely from the outside world, obeys her parents and teachers to a fault, and is considered by them to be a "model" child with great future promise. Her stable life is disturbed, however, with the onset of puberty, which threatens her with emotional and physical changes as well as the need for self-reliance and acceptance by her peers. She may feel the full impact of these changes in her early teens or perhaps not until she moves away from her parents' home.

Unwilling and unable to deal with the emotional challenge posed by adolescence she focuses instead on her body as the locus of control: She makes slimness her goal and hopes the rest will follow. However, as she loses weight, she finds that her inner turmoil continues. Retreating into herself, she attributes her problems to insufficient weight loss and intensifies her low-carbohydrate diet; she also becomes hyperactive in an attempt to lose weight more surely. Her growing physical hunger may cause her to steal or hide food and occasionally to binge. While she is physically attracted to food, she is also now terrified by the loss of control it represents. She may turn to purging as a means of assuaging the guilt and fear she feels after binging. Ultimately (perhaps within a year or two of initial weight loss), she loses 25-50% of her former body weight--enough to actually reverse pubertal development. In this emaciated condition she deludedly considers herself to be of normal weight.

Unfortunately, her body (not to mention her mind) is far from being in normal condition: Her starvation may lead to any of a number of metabolic, cardio-vascular, renal, or blood disorders. If she is bulimic, she also risks serious

gastric disorders and loss of critical electrolytes. Because she is skilled at hiding her condition from others and from herself, she may not enter treatment until her physical deterioration is well advanced. Hospitalization may follow an episode of physical or emotional collapse.

Approaches to Treatment

Without question, the first task in treating a seriously ill anorexic is to stabilize her acute condition, generally through the administration of needed electrolytes. Beyond the acute phase, however, there is considerable disagreement and confusion surrounding the issue of treatment. An approach that succeeds for one individual may fail for another. An approach that appears effective in the short term may prove ineffective or even detrimental in the long term. Where pure anorexics are often uncooperative, bulimics are easily frustrated in therapy. Often, "recovered" anorexics and bulimics relapse into their former condition when their fragile psychological balance is disturbed.

Treatments may be viewed as physiological, behavioristic, or cognitive in approach. Physiological treatments include refeeding, drugs, hormones, electro-convulsive therapy, and exercise regimens. Behavioristic approaches include deconditioning of food anxiety and providing rewards for weight gain. Cognitive approaches include individual psychotherapy, group therapy, and family therapy. Some researchers advocate a combination of gradual refeeding and some form of cognitive therapy, noting (1) that the body's needs must be met before cognitive therapy can be fully effective and (2) that refeeding alone does not undo the underlying psychological syndrome that leads the individual to pursue emaciation.

Crisp (1980), for example, advocates a two-phase approach: In the first phase, the patient is confined to bed and fed a diet of 3000 calories per day until a normal "target" weight is achieved. In the second phase, still in the hospital, she receives training in personal and social skills and engages in various forms of psycho-therapy as well as regular physical activity. When successful, this approach serves to "rekindle" the patient's adolescence, an experience which may cause her to panic or to become depressed. Control of this depression may then become the final goal of therapy. Crisp reports that his approach speeds the improvement of the 40% of anorexics who recover in any case, makes possible the improvement of an additional 30-40%, and reduces the rate of fatality.

Crisp also points out, however, that the evaluation of alternative treatments has never been systematically undertaken, largely because of the difficulty of long-term follow-up with the eating disordered. Such an evaluation should start with a careful statement of therapeutic objectives. It should also seek to clarify the natural courses or "behavior modes" of anorexia and bulimia and their variants.

Finally, it should attempt to identify those patient characteristics that can affect the optimal approach to treatment.

PURPOSE AND APPROACH

The purpose of this paper is to present a dynamic model that can bolster our understanding of both normal and abnormal eating behaviors. This model integrates current knowledge regarding the physiological, behavioral, and cognitive aspects of weight control. The goal of such an integrative model is to make possible some of the evaluation described above, in the controlled, self-consistent, quick, and inexpensive context of a computerized laboratory. The model is first used to generate various normal and abnormal behavior modes. It is then tested to determine whether appetite-altering drugs or regular exercise--both subjects of some controversy--may have the potential to ameliorate the effects of abnormal dieting.

The next section describes broadly the structure of the system dynamics model used for this analysis, with special attention given to the regulatory feedback loops dominating the picture. This is followed by a description and explanation of model behavior under a variety of scenarios. The paper concludes with a summary of contributions and findings and suggestions for future research.

MODEL STRUCTURE

The scope and boundary of the dynamic model are illustrated in Figure 1. The model's sixty-odd interacting variables cover those aspects of physiology, cognition, and behavior that can affect and be affected by eating and weight changes over the course of weeks and months. Beyond the model's explanatory boundary lie those aspects of the individual's physical and emotional development, as well as her social, cultural, and nutritional surroundings, that form the context for her behavior. This context may be viewed as a set of givens that "calibrate" the individual physiologically, cognitively, and behaviorally, thus predisposing her toward one dynamic pattern or another. For example, such givens determine the individual's natural level of fat (her so-called "set-point"), her relative willpower when dieting, and the body weight she would find acceptable.

The physiology of caloric expenditure and storage--"what happens to the food"-- is central to the model and is pictured, somewhat simplified, in Figure 2. Body weight may be seen as consisting of three parts: skeletal (or "fixed") weight, fat, and muscle. Only fat and muscle are variable in the model and may be lost when caloric expenditure exceeds caloric intake--that is, when the "caloric balance" is

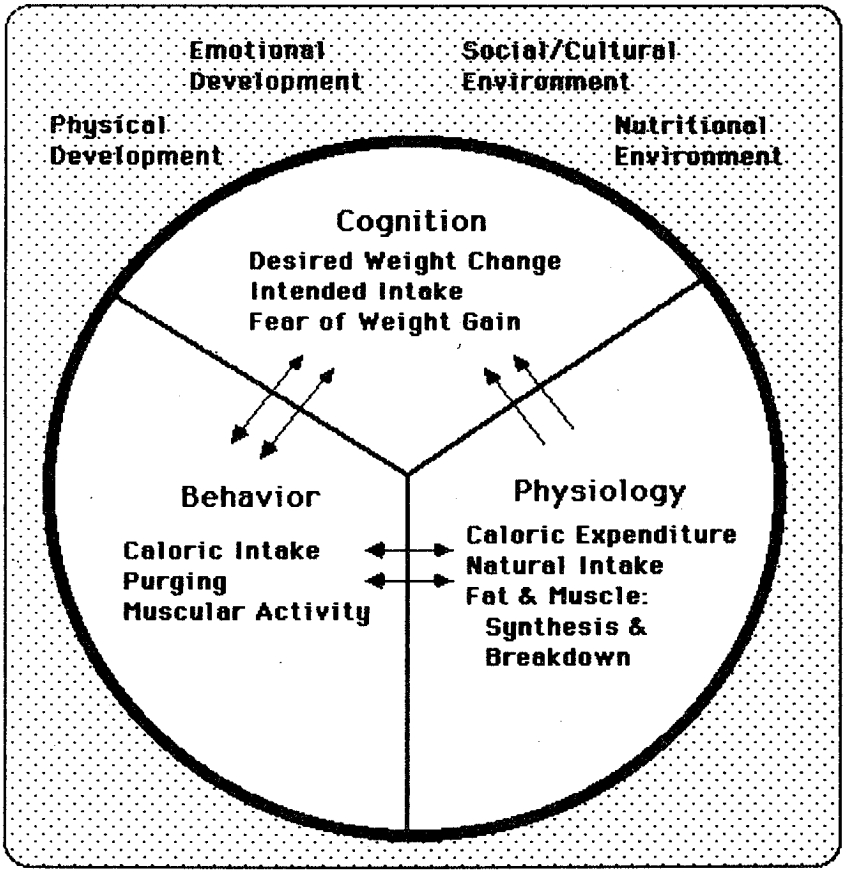

Figure1. Model Scope and Boundary

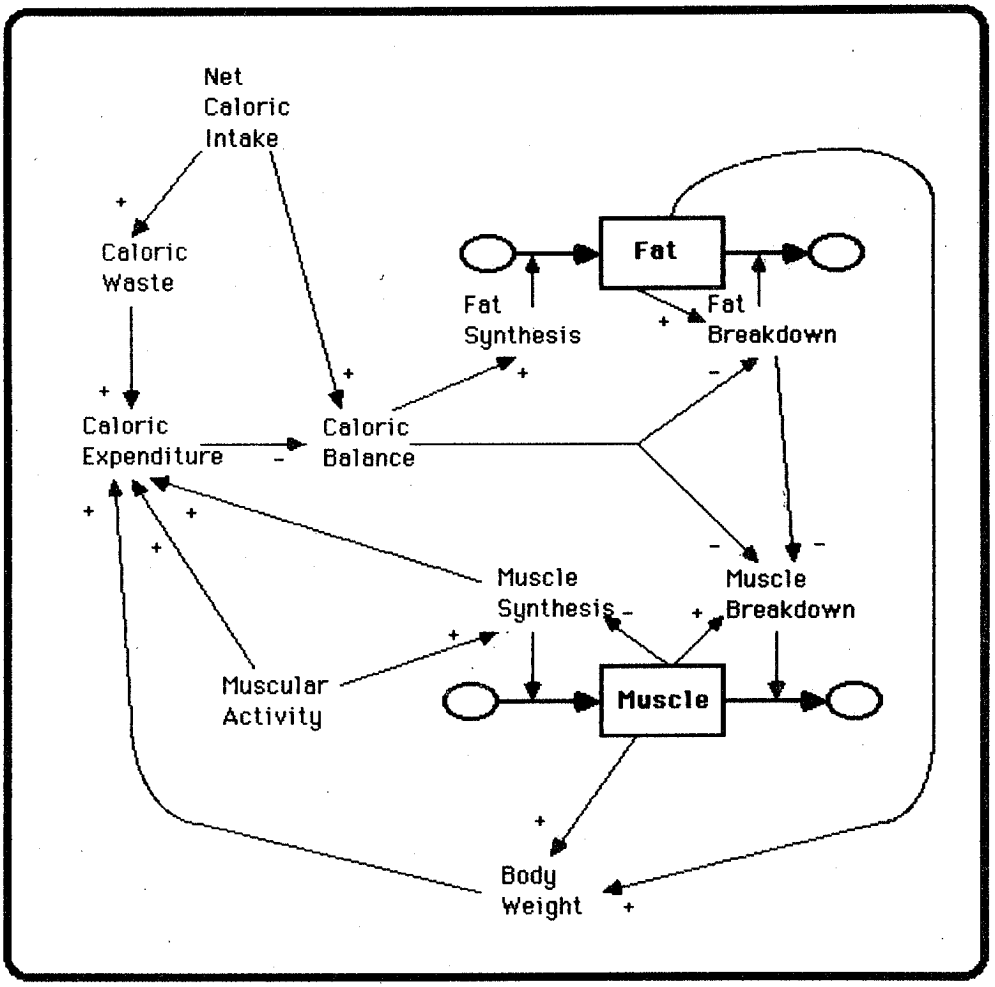

Figure 2. Caloric Expenditure and Storage

negative. Normally, fat is much preferred over muscle for providing energy; but when fat is sufficiently depleted, the rate of muscle breakdown may increase. Fat is synthesized when the caloric balance is positive. Muscle is synthesized to compensate for the normal wear and tear of muscle, and also when an increased level of muscular activity stimulates the building of additional muscle mass.

As shown in Figure 2, the model portrays caloric expenditure as consisting of four components, including three "productive" uses and "waste" (or elimination). The body's basal (resting) metabolic rate may be calculated as a logarithmically increasing function of body weight. Muscular activity increases the use of calories, as does the diversion of calories for muscle synthesis. Caloric waste may be excretory or thermic in nature, with thermogenesis clearly the dominant element; normally, only about 5% of caloric intake is excreted while perhaps 25-30% is eliminated through thermogenesis. Furthermore, the thermogensis fraction is rather flexible, homeostatically increasing when caloric intake exceeds the body's needs and decreasing when intake falls short of those needs (Stock & Rothwell 1982).

Figure 3 portrays the various feedback mechanisms potentially affecting the regulation of caloric intake within the context of intentional dieting. The two loops at the upper right illustrate the way in which intake is adjusted, in a diet free of "cheating", so that the individual's desired weight is achieved. In such an "ideal" self-imposed diet, intake (=intended intake) is reduced until the recent rate of weight loss matches that which is desired. When body weight is sufficiently close to its desired level, the desired loss rate will start to diminish; in response, intake will rise. Continuing in this way, the desired steady-state may be reached smoothly and with little or no fluctuation.

The loop at the upper left in Figure 3 shows how the body's needs tend to interfere with dieting. The body's "natural" caloric intake increases above normal when body weight falls below its natural level. The natural weight, in turn, is primarily determined by the set-point for fat, but may also be affected by muscular activity or by the relative palatability of available food. When natural intake is sufficiently greater than intended intake (that is, when the body is sufficiently hungry to overcome the dieter's willpower), unintentional eating will occur; and the amount of such cheating or binging will tend to grow as the intended intake and body weight drop. Clearly, this loop acts in opposition to the normal dieting loops described above and is responsible for the failure of most strenuous diets.

The two loops at the bottom of Figure 3 show what can happen when the individual is so single-mindedly determined to lose weight that the failure to do so (at the desired rate) threatens her very sense of self-worth. In response to unintended intake, such an individual becomes fearful of the danger such "undesirable" food

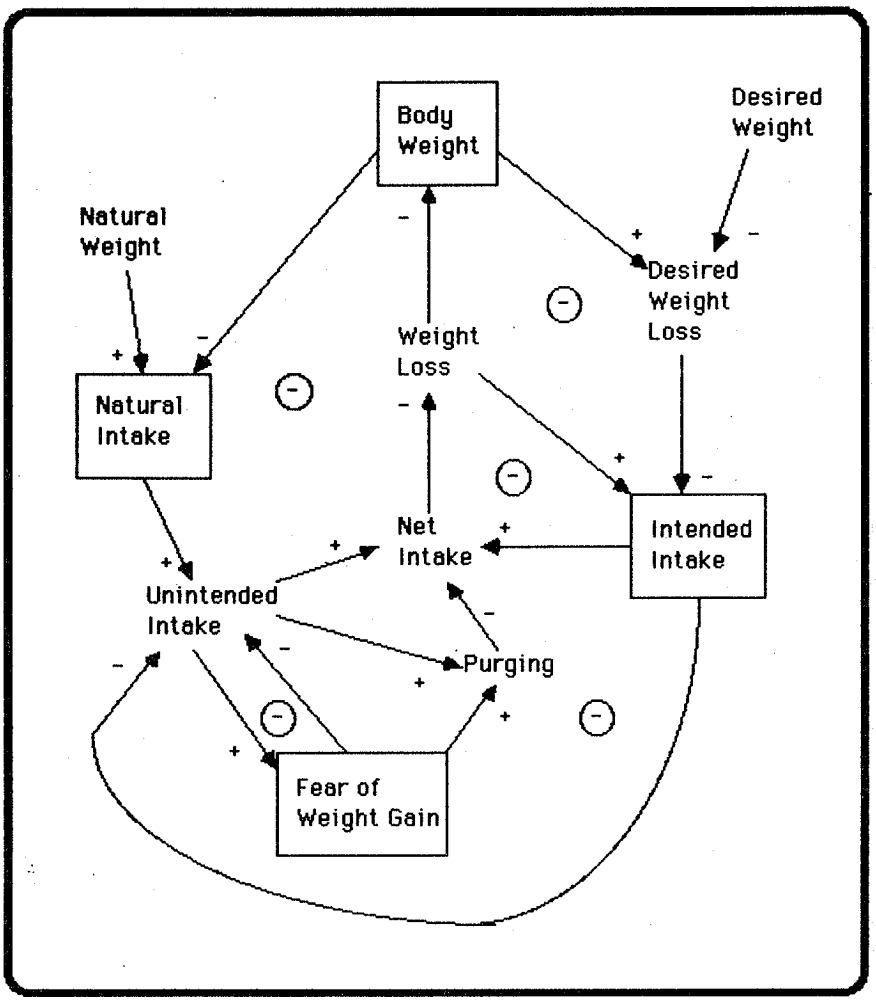

Figure 3. Regulation of Caloric Intake in Normal and Abnormal Dieting

represents. She then enlists this fear in the battle to overcome the body's needs and reduces, perhaps even eliminates, cheating (see the loop at lower left); Crisp refers to this stance as a "phobic avoidance" of food.

If the individual is so inclined, she will also purge after binging, the frequency of such self-abuse increasing the more fearful she becomes. Though it can ultimately be life-threatening, note that purging (like "phobic avoidance") is essentially a means of control--as the minus sign in the loop at lower right suggests. This loop says that purging reduces net intake and so assists in weight loss; by doing so, it actually allows for some relaxation of intended intake and reduces the pressure to binge. Reduced binging, in turn, reduces the fear of weight gain and the amount of purging. Purging may thus be viewed--perhaps unexpectedly--as a self-limiting process that can reduce both weight and the anorexic's fear of weight gain.

The two loops just discussed demonstrate that, whether or not the anorexic purges, her fear of food--as disabling as it can be emotionally--does serve to support her "relentless pursuit of thinness" (Bruch 1973).

Figure 4 illustrates how muscular activity may affect and be affected by dieting. In a normal dieter, activity will tend to decrease when the diet is sufficiently strenuous; lassitude and fatigue are well-known consequences of food deprivation (Bruch 1973, Keys et. al. 1950). Reduced muscular activity, in turn, will tend to reduce the body's need for food, and thus moderate to some extent the dieter's hunger (see the negative loop). While this may seem a desirable effect, it is also true that reduced muscular activity depresses the rate of muscle synthesis (see Figure 2), which unfortunately increases the proportion of total weight loss due to muscle loss as opposed to fat loss.

An anorexic, on the other hand, responds to her fear of weight gain not only by reducing caloric intake but also by becoming more--not less--active than usual. She may thereby avoid some of the muscle loss that is her fate as a strenuous dieter; however, this comes at the cost of reinforced hunger, binging, and fear (see the positive loop).

The degree to which muscular activity and the loops of Figure 4 can alter the basic dynamics suggested by Figure 3 is not self-evident. The model has been calibrated with some care so that this issue may be usefully addressed.

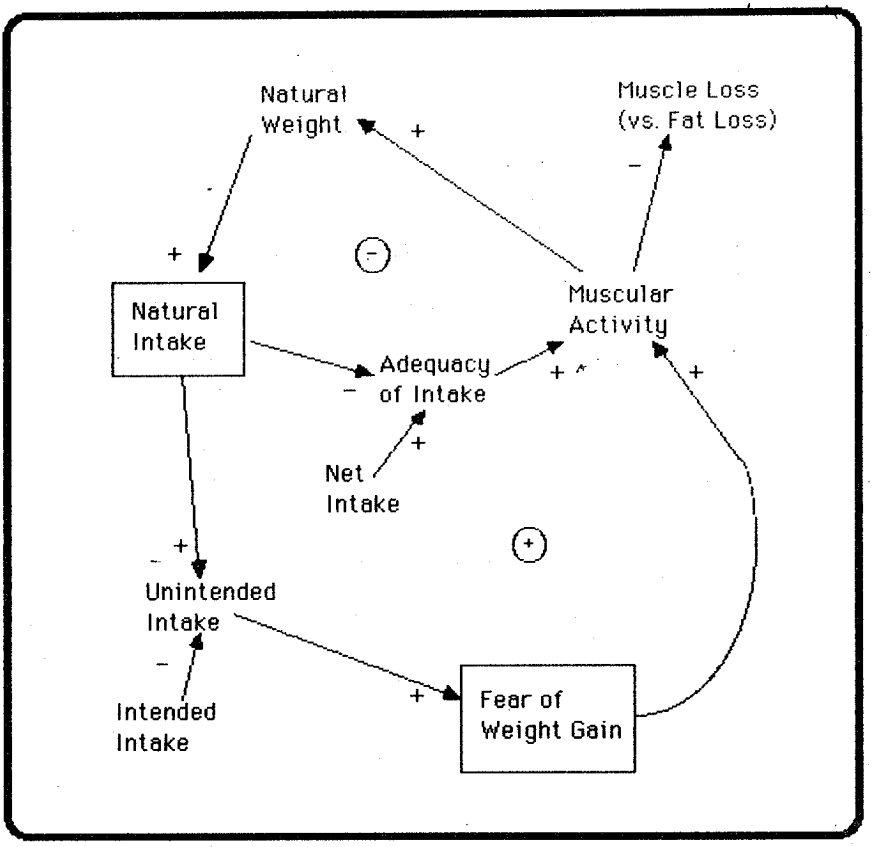

Figure 4. The Role of Muscular Activity in Normal and Abnormal Dieting

MODEL BEHAVIOR

"Normal Behavior"

Three sets of model tests were performed to investigate the effects of restrained caloric intake in the absence of the anorexic's abnormal psychology. These tests provided understanding of normal patterns of eating and weight change and served as a baseline against which eating disorders could be compared.

In the first set of tests, intake is reduced below its initial "natural" level in a single step, without opportunity for cheating. Such a fixed reduction in intake results in a caloric deficit, leading to a loss of weight and, if the reduction is great enough, a decline in muscular activity. The new equilibrium is approached more or less exponentially with an adjustment time of about 100 days; this figure results from the fact that daily caloric expenditure amounts to about 1% of the body's stored energy. Thus, the full adjustment requires on the order of 3 x 100 = 300 days.

In half of these tests, the reduction in intake is assumed to be accompanied by a large increase in the normal level of muscular activity. As expected, increased exercise reduces the share of weight loss borne by muscle; indeed, if the reduction in intake is not too great, muscle may be built while fat is lost. Harder to predict, however, is the effect of increased exercise on total body weight.

The tests indicate that, even when caloric intake is quite low, weight is actually somewhat greater with increased exercise than without it for the first 100-200 days. The primary effect of exercise during this initial period is to "convert" caloric storage away from fat and toward muscle. Since fat is considerably more energy-dense than muscle (9300 Cal./kg. fat vs. 4100 Cal./kg. muscle), this conversion moderates the weight consequences of caloric loss. Following this period of conversion, muscle synthesis ceases and the energy cost of exercise starts to take its toll on total weight; if intake is extremely restricted, increased exercise may even eventually reduce muscle itself due to the unavailability of fat. In summary, increased exercise combined with restricted intake initially slows the loss of body weight but ultimately (after 200 or more days) enhances it.

The second set of tests of "normal" behavior examines the rebound back to normal weight following the end of strict caloric restriction. Empirical experiments with both humans and animals suggest that the greater-than-normal eating characteristic of refeeding tends to persist for a time even after normal body weight is regained (Keys et. al.1950, Polivy & Herman 1985). These experiments also suggest that the longer the period of restriction is, the greater and more prolonged

the overshoot will be. The model does reproduce these results, but only when it is assumed that the level of muscular activity is not significantly diminished in the face of hunger. (It is not entirely clear whether or not this was the case in the empirical experiments; Keys et. al. do seem to suggest, however, that their human subjects, though tired, continued to exercise during the fast.) When hunger-induced lassitude is an important factor, it reduces the body's needs and so reduces the individual's voraciousness during and immediately after refeeding.

The third set of tests consider a normal diet in which the individual sets a desired weight and adjusts her intended intake accordingly, and in which unintended intake may occur, as in the upper loops of Figure 3. When her goal is not too ambitious, the dieter is able to achieve her desired weight smoothly, quickly (within a year), and with a minimum of cheating. When her goal is somewhat more ambitious--or when she adds vigorous exercise to her regimen--she may still reach her goal, but does so with greater difficulty. When her goal is overly ambitious, however, she reaches an equilibrium weight short of her goal. Despite her intention to eat minimally, her willpower is simply not great enough to stick faithfully to such a diet at a weight that her body deems "unacceptable". Indeed, in real life (though beyond the scope of the current model), continued frustration may well cause her to drop the diet altogether and to return to her initial weight.

"Anorexic Behavior"

Extensive model testing was performed portraying the anorexic's characteristic pattern of fear-ridden dieting. In these tests, the hypothetical anorexic may be thought of as a 16-year old girl, 158 cm. (5'2") tall, who, at 54 kg. (119 lbs.) is slightly heavier than the average girl of her age and height (Schottelius & Schottelius 1973). An adolescent trauma of some sort, in combination with her underlying anorexic personality, triggers a wish to lose weight rapidly. Indeed, it is assumed she will fail to find her body acceptable until she has lost 30% of her starting weight, down to a pre-pubescent 38 kgs. (84 lbs.). In one set of tests, she is assumed to be unwilling to purge after unintended intake; in a second set, she is assumed to be a potential purger.

Figures 5 and 6 show the typical course of development when our anorexic is assumed to be non-purging and endowed with considerable ability--when motivated by fear--to adhere to a difficult diet. Figure 5 shows that she does indeed achieve her acceptable weight, about 450 days after the diet is triggered. But as she loses weight, she becomes increasingly fearful of gaining weight until this fear is nearly all-enveloping. The source of this fear is unintended intake, which is seen in Figure 6 as the gap between actual and intended intake. Despite her fear-bolstered willpower, her body's need for food creates increasing pressure to

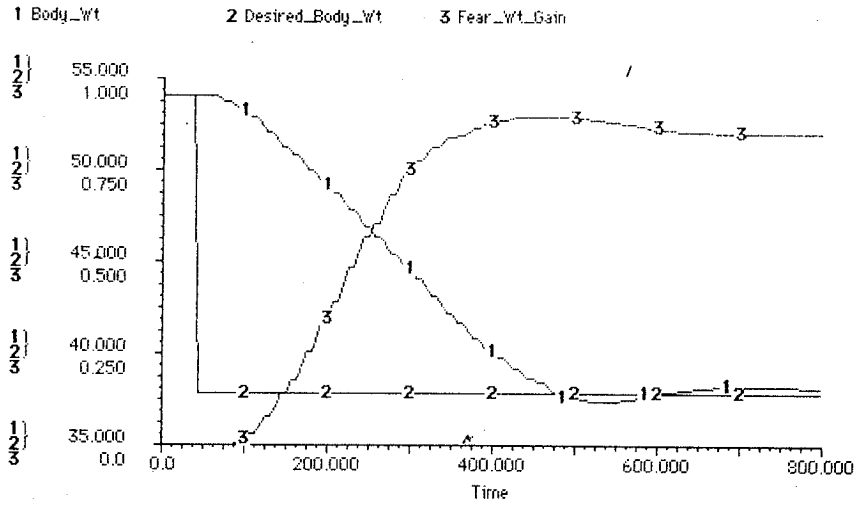

Figure 5. Non-Purging Anorexic: Weight and Fear

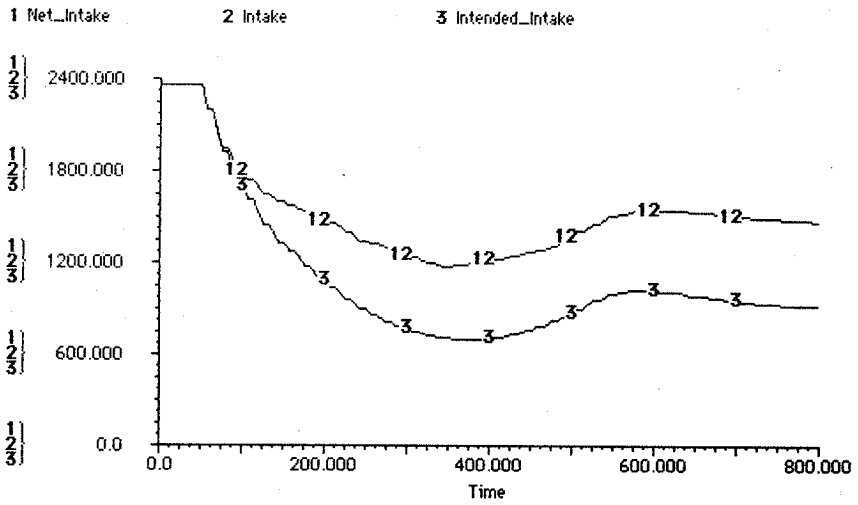

Figure 6. Non-Purging Anorexic: Caloric Intake

eat. She surrenders somewhat to this hunger, but not so much that her goal cannot be reached.

Figures 7 and 8 illustrate the central role of fear-driven adherence in enabling the non-purging anorexic to achieve her acceptable weight. The scenario producing this simulation differs from the preceding one only in the assumption regarding the effect of fear on willpower: Here, this effect has only half of its former strength. Now, not only does our anorexic not reach her goal, but her level of fear becomes even greater than in the previous simulation. This, of course, results from a greater amount of cheating, under a diet that has become unrealistically restrictive given her limited willpower. The state of terror and frustration that is portrayed here is undoubtedly unstable: Either our "weak" anorexic will temporarily give up on her diet, or she will turn to purging.

Figures 9 and 10 show the result of allowing the "weak" anorexic of the previous scenario to purge in response to her binge-created fear of gaining weight. Her initial behavior matches that of the preceding run, but by Day 200 she is fearful enough that she starts to purge after some of her binges. (Purging may be seen in Figure 10 as the gap between "intake" and "net intake".) By Day 300, she is fearful enough that she is purging all of her unintended intake and more (see Crisp 1980). This allows her to lose weight quickly and even to raise her intended intake as her acceptable weight is approached; by Day 400 she has reached her "goal". As a result of her now less-restrictive diet, her fear of gaining weight is lower than in the preceding scenarios. In sum, purging would appear to be an effective way for the impulsive anorexic to lose weight and become less fearful--if it were not for its potentially devastating effect on her health.

Policy Testing

On the subject of policy testing, it is necessary first to understand that the scope of the current model is not sufficiently broad to allow meaningful evaluation of alternative psychotherapeutic approaches to anorexia and bulimia. Such approaches seek, in the model's terms, to increase the individual's desired body weight to a safe and sustainable level. The psychological mechanisms that determine desired body weight are not well understood, however, and do not appear in the model. Although this does imply that the model is not an appropriate tool for evaluations of long-term prognosis, it does <u>not</u> mean that the model may not be useful for evaluating more immediate interventions that may be able to move the individual out of danger on a more permanent basis than can be achieved through forced refeeding.

Two such potential stabilizers are (1) the use of appetite-altering drugs and (2) the modification of activity level. Tricyclic drugs and insulin have been

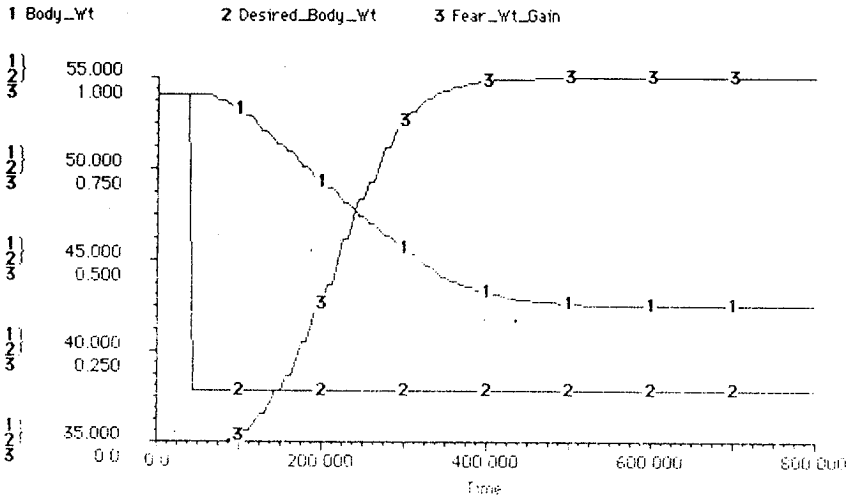

Figure 7. Non-Purging Anorexic with Reduced Willpower: Weight and Fear

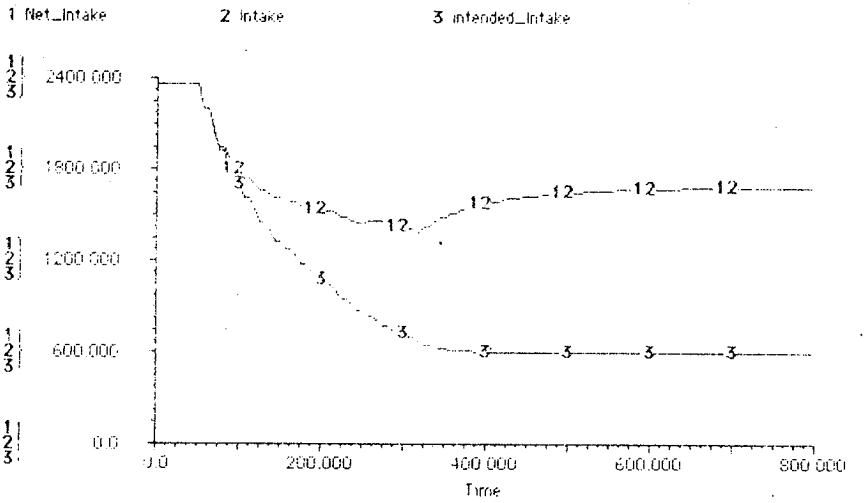

Figure 8. Non-Purging Anorexic with Reduced Willpower: Caloric Intake

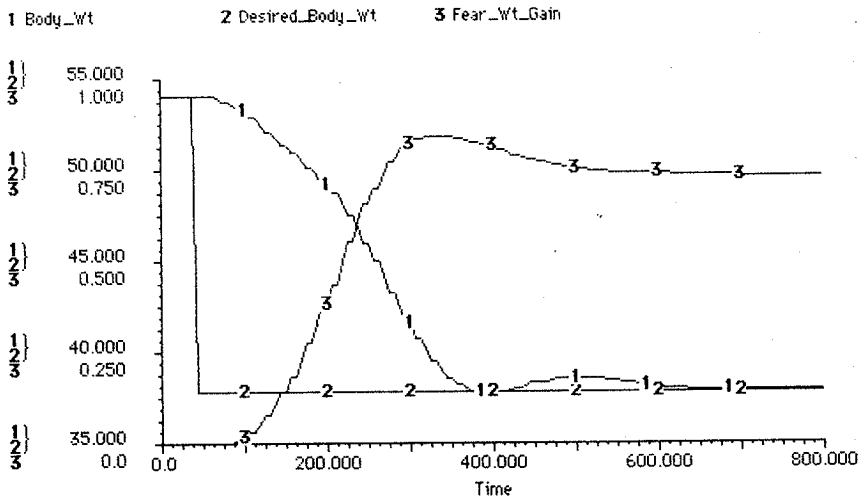

Figure 9. Purging Anorexic: Weight and Fear

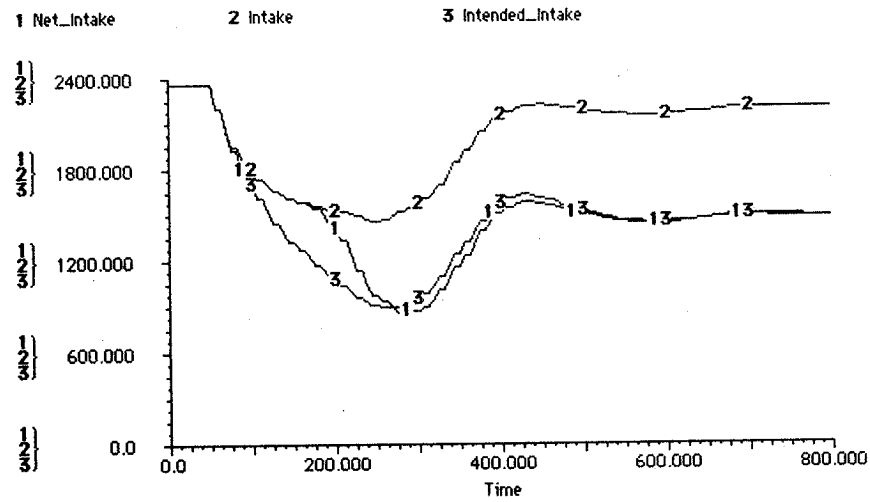

Figure 10. Purging Anorexic: Caloric Intake

prescribed as appetite enhancers to some anorexics. Crisp (1980) claims that this approach is dangerous to the anorexic--particularly if she purges--and suggests that appetite suppressants, if anything, may improve the clinical picture. Exercise regimens have also been advocated by some in connection with anorexia (Crisp 1980), while others have claimed that muscular activity plays too small a role in total caloric expenditure (typically amounting to only about 10%) to be taken seriously (Stock & Rothwell 1982).

These interventions may be evaluated in terms of their ability to stabilize the individual physically and/or psychologically. In the context of the model, physical risk is represented by the loss of muscle and by purging, while psychological risk is represented by the fear of weight gain. These three criteria suggest simple diagnostic measures for comparing simulations of alternative policies.

Two identical batteries of policy tests were performed: one for the non-purging anorexic whose base case corresponds to Figures 5 and 6, and one for the purging anorexic whose base case corresponds to Figures 9 and 10. In all of these tests, interventions (one or two per test) are introduced on Day 400 and the course of development observed through Day 800; this proves to be sufficient time to observe the impact of all interventions into steady-state. The interventions tested include: appetite enhancement (25%), appetite suppression (25%), reduced exercise (25%), and increased exercise (25%, 50%). (In the model, alteration of appetite corresponds to an exogenous multiplier on natural intake, while alteration of exercise corresponds to a similar multiplier on muscular activity.)

As it turns out, the interventions' overall impacts are similar for the two kinds of anorexic. Appetite enhancement neither increases the anorexic's steady-state weight nor her muscle, but instead increases her fear and, if she is a purger, her purging. Appetite suppression has the opposite effect, which is beneficial. Exercise has mixed effects, though it too does not affect the anorexic's total weight in steady-state. Increased exercise does, however, reduce the loss of muscle relative to fat; on the other hand, it tends to increase hunger and binging, and so also increases fear and purging. Decreased exercise has the opposite effects.

Figure 11 provides an example of the magnitudes of impact of selected policies and also suggests the potential benefits of a two-pronged policy. The three diagnostic measures, Fraction of (initial) Muscle Lost (0-1), Fear of Weight Gain (0-1), and Calories Purged per Week, are all measured on Day 800. Clearly, both appetite suppression and exercise can play significantly beneficial roles. Note that when the two interventions are combined, their benefits are combined as well. The optimal policy depends, of course, on how the three criteria in Figure 11 are weighted, but appetite suppression seems generally to be indicated. Beyond

	Fraction of Muscle Lost	Fear of Weight Gain	Calories Purged per Week
A. Non-Purging Anorexic			
1. Base Case	.17	.85	0
2. Suppressed Appetite	.17	.60	0
3. Vigorous Exercise	.12	.86	0
4. Suppressed Appetite + Vigorous Exercise	.12	.65	0
B. Purging Anorexic			
1. Base Case	.18	.73	5000
2. Suppressed Appetite	.18	.55	2100
3. Vigorous Exercise	.13	.75	5700
4. Suppressed Appetite + Vigorous Exercise	.12	.57	2600

Figure 11. Diagnostic Summary of Policy Test Results (Day 800)

that, a concern with muscle loss suggests a substantial increase in exercise; while the lack of any such concern may well suggest a reduction in muscular activity in order to reduce fear and purging a bit further.

CONCLUSIONS

The model presented in this paper represents a first major step toward integrating knowledge, in a self-consistent and testable manner, from the various areas that impinge upon the study of eating disorders. Although the current model is not as detailed as it might be in matters of physiology and psychology, it can reproduce observed modes of behavior, for what seem to be the "right" reasons. The story it tells is not one of "vicious cycles", as eating disorders are sometimes described (Bruch 1973). The model tells instead a story of two competing regulatory mechanisms--one physiological and one cognitive.

From this regulatory perspective, the anorexic may be seen as a disturbed individual so committed to the cognitive control of her weight that her physical and mental health become of only secondary importance to her. Whether through the use of "phobic avoidance" or through the use of purging, she actually puts her fear of food to work in support of the one cause that truly matters to her: the pursuit of pre-pubertal thinness.

Taking this cause as a given, the model suggests that perhaps the best one can hope to do for the anorexic in the intermediate term is to lessen the terrifying pull of her body's needs, through appetite suppression, and if necessary, to reduce the risk posed by her loss of muscle, through increased exercise. Note that anorexics tend toward hyperactivity anyway, so that this second recommendation may be superfluous (or at least not likely to meet with resistance) in many cases. The model suggests that the anorexic's hyperactivity be viewed in a more positive light than it normally is.

The limitations of the current model, particularly its inability to provide an evaluation of psychotherapeutic alternatives, suggest model enhancement as a potentially fruitful avenue for future research. Model enhancement might also focus on issues related to obesity and non-anorexic bulimia, which the current model addresses incompletely. Also, whereas the model has thus far been developed solely on the basis of written material and modeling logic, it may also prove valuable for the sake of model improvement to consult directly with clinicians who treat the eating disordered.

Future research might also involve empirical studies based on relationships contained in or generated by the model. For example, the idea that muscular

activity during caloric restriction can have distinctly different short-term and long-term effects begs for confirmation. More generally, the model's multivariate patterns of behavior would seem to constitute a call for careful longitudinal research, which is still waiting to be done in the area of eating disorders.

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