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Exercise and Diet in Obesity Treatment: An Integrative System Dynamics Perspective

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ABSTRACT

ABDEL-HAMID, T. K. Exercise and Diet in Obesity Treatment: An Integrative System Dynamics Perspective. *Med. Sci. Sports Exerc.*, Vol. 35, No. 3, pp. 400–414, 2003. **Purpose:** Demonstrate the utility of System Dynamics computer modeling to study and gain insight into the impacts of physical activity and diet on weight gain and loss. **Methods:** A holistic System Dynamics computer model is presented that integrates the processes of human metabolism, hormonal regulation, body composition, nutrition, and physical activity. These processes are not independent of one another, and the model captures the complex interdependencies between them in the regulation of body weight and energy metabolism. The article demonstrates how such an integrative simulation model can serve as a viable laboratory tool for controlled experimentation to investigate the impacts of physical activity and diet on body weight and composition. **Results:** In one experiment, weight loss from a moderate level of daily exercise was slightly less than the loss from dieting. Although exercise did have a favorable impact on body composition by protecting against the loss in fat-free mass (FFM), it, however, failed to blunt the drop in resting energy expenditure (REE) that accompanies diet-based weight loss. The smaller loss in FFM did indeed induce a smaller drop in *nominal* REE, however, the preservation of FFM also affected a relatively larger loss in FM, which, in turn, induced a larger adaptive reduction in the metabolic rate. The two adaptations almost totally offset one another, causing minimal differences in REE. In a second experiment, exercise regimens of moderate- to high-level intensity proved counterproductive as weight-reducing strategies. However, when the diet was changed from a balanced composition to one that was highly loaded with carbohydrates, it became possible to sustain the intense exercise regimen over the experimental period and achieve a significant drop in body weight. **Conclusion:** The results underscore the significant interaction effects between physical activity, diet, and body composition and demonstrate the utility of computer-based experimentation to study, gain insight into, and make predictions about their dynamics. **Key Words:** METABOLISM, BODY COMPOSITION, WEIGHT LOSS, SIMULATION, FEEDBACK

Obesity develops when a chronic, quantitative imbalance exists between energy intake and energy expenditure. To unbalance the energy equation in the direction of weight loss requires decreasing food energy intake, increasing physical activity, or altering both simultaneously. To date, the emphasis of treatment has been on the energy intake side of the energy balance equation (22). Dieting, currently the mainstay of obesity treatment for most people, is most often undertaken as a self-directed process with instruction from a book or slimming club within the community or often just by self-induced restraint.

There are at least two reasons for why decreasing energy intake has been favored over increasing energy expenditure through physical activity. The first is economic. In recent years, the cost of burning extra calories has surged as work has become more sedentary. It used to be that people got paid to engage in manual labor. Today, one pays to exercise, not so much in money, but in foregone leisure time (23).

Second, it has been difficult to demonstrate the efficacy of exercise as a treatment strategy for obesity. Although a considerable number of studies have been done to investigate whether exercise contributes to weight loss, the results have been mixed (26). On the exercise side, McArdle et al. (21) cite evidence to support the contention that an increased level of regular physical activity may be more effective than dieting for long-term weight control. And in a meta-analysis study conducted to assess the effects of exercise on changes in body mass, Ballor and Keesey (4) found that increases in physical activity results in body mass reductions (for sedentary males). But, in a subsequent meta-analysis of 46 published studies, Ballor and Poehlman (5) found no significant impact of exercise on weight loss, although a review by Andrews (2) found that exercise had only modest effects on weight loss that were an order of magnitude less than those resulting from energy restriction alone. One possible clue to the variability of results in this area is offered in Saris' recent review (26), who found that only a few of the studies to-date were well controlled.

In 1999, the American College of Sports Medicine sponsored a scientific roundtable to review the current state of knowledge in the field (12). In its final report, the panel lamented the paucity of randomized clinical trials to study the role of physical activity in the prevention and treatment of obesity, and noted that for the limited number of trials

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that have been conducted, a majority failed to demonstrate statistically significant results. The panel concluded that most studies suffered, in part, from short time frames and small sample sizes because of the complexity and expense of performing the intensive measurements needed.

In addition, and perhaps of greater concern, is the major methodological problem of ensuring adherence of experimental subjects to an exercise protocol (12). Ideally, all studies on food intake and energy expenditure would be carried out under natural free-living conditions in which eating and exercise behaviors could occur without hindrance and could be measured precisely and accurately. In practice, this is not possible because methods for measuring total energy intake and expenditure under natural circumstances are often unreliable. On the other hand, laboratory-based studies allow the accurate assessment of food intake and energy expenditure but under highly artificial circumstances (19).

Accordingly, it is useful to seek other methods for testing and experimentation. Simulation-based experimentation provides a viable laboratory tool for such a task. In addition to permitting less costly and less time-consuming experimentation, simulation-type models make "perfectly" controlled experimentation possible. In the model system, unlike the real systems, the effect of changing one factor can be observed while all other factors are held unchanged. Internally, the model provides complete control of the system (27).

This study attempts to demonstrate the utility of System Dynamics modeling as a vehicle for controlled experimentation to study and gain insight into the impacts of physical activity and diet on body weight and composition. Prevention of obesity requires an understanding of the quantitative imbalance between energy intake and energy expenditure and modification of behaviors that predispose to weight gain. Because the problem is quantitative and dynamic, it is well suited to computer simulation.

METHODS

Arguments for a System Dynamics perspective. System Dynamics is the application of feedback control systems principles and techniques to model, analyze, and understand the dynamic behavior of complex feedback systems. Its origins trace back to the pioneering work of Jay W. Forrester, whose seminal book *Industrial Dynamics* (11) is still a significant statement of philosophy and methodology in the field. System Dynamics is aimed at the study and analysis of certain kinds of complex systems, known as dynamic feedback systems. These are systems characterized by a large number of interrelated variables that interact dynamically over time through information-feedback structures. Although the words complex, dynamic, and system have been applied to all sorts of situations, feedback is the differentiating descriptor here. Indeed, feedback processes are seen in System Dynamics to hold the key to structuring and clarifying relationships within such systems and in understanding their dynamic behavior.

Most succinctly, feedback is the transmission and return of information. For example, a feedback system exists whenever an action taker will later be influenced by the consequences of his or her actions. More generally, feedback refers to the situation of X affecting Y and Y in turn affecting X, perhaps through a chain of causes and effects. One cannot study the link between X and Y and, independently, the link between Y and X and predict how the system will behave. Only the study of the whole system as a feedback system will lead to correct results.

The human body is a conglomerate of interrelated and interdependent feedback control processes, which together constitute such a dynamic feedback system, albeit a highly complex one (13). Feedback structures occur at the cellular level as well as the whole-body level. Example feedback mechanisms include the multitude of hormonal-based homeostatic processes that act to maintain the body's internal stability in the face of changes in its external or internal environment. Essentially all of the organs and tissues of the body perform functions that help to maintain these constant conditions. Indeed, If one had to describe, with a single word, what physiology is all about, that word would be homeostasis... it refers to regulatory mechanisms by which biologic systems tend to maintain the internal stability necessary for survival while adjusting to internal or external threats to that stability. If homeostasis is successful, life continues; if it is unsuccessful, disease and, perhaps, death ensue (17).

Such homeostatic mechanisms act by so-called negative feedback. Feedback loops divide naturally into two categories, which are labeled deviation-counteracting feedback or negative loops, and deviation-amplifying feedback or positive loops. Negative loops counter and oppose change, whereas positive loops tend to reinforce or amplify whatever is happening in the system.

Although most control systems of the body act by negative feedback, in rare instances, the body has learned to use positive feedback to its advantage. For example, in progressive training, an initial training overload causes physiological capacity to expand, and as the physiological capacity of the body expands, the initial training stimulus must be increased (progression) to maintain overload, leading to further expansion of physiological capacity (7,15). In other words, the initiating stimulus causes more of the same, which is positive feedback. [In all cases in which positive feedback is useful, the positive feedback itself is part of an overall negative process, which causes the change to eventually plateau (13).]

Ultimately, the purpose in applying System Dynamics is to facilitate understanding of the relationship between the behavior of a complex system over time and its underlying feedback structure. For this, system dynamicists rely on computer simulation. Even though the dynamic implications of isolated loops such as those discussed above may be reasonably obvious, the interconnected feedback structures of real problems are often so complex that the behavior they generate over time can usually be traced only by simulation.

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Computer simulation is particularly suited to the study of continuous systems, in which system variables change not in discrete jumps but continuously over time. This is a characteristic of all living systems, which by definition are in constant flux. Yet, because of the complexity and expense of continuous measurements, most experimental studies of human energy expenditure have relied on discrete, rather than continuous, measurement protocols. This can be a serious limitation, because a negative finding (e.g., finding no association between low energy expenditure and subsequent weight gain) may simply mean that the timing of the measurements did not coincide with the period of reduced/increased energy expenditure (25).

In addition to handling dynamic complexity and permitting continuous measurements, simulation-type models make "perfectly" controlled experimentation possible. In the model system, unlike real systems, the effect of changing one factor can be observed while all other factors are held unchanged. Internally, the model provides complete control of the system (27).

System Dynamics model structure. Much of the research to date on human bioenergetics has deliberately emphasized one aspect or problem area in isolation. For example, the typical study is undertaken to assess the effects of a single variable on another such as studying the effect of one type of diet or of physical activity on weight loss/gain.

Clearly, such micro-oriented research is necessary for the study and understanding of any complex system such as the one governing the processes of human bioenergetics. However, breaking a complex system into its component pieces and studying the pieces separately is often insufficient in understanding the whole. The human body is an amazing feat of system integration, where changes in one of its subsystems can be traced throughout the body's entire system. As a result, the behavior of an individual subsystem in isolation may be different from its behavior when it interacts with other subsystems. For example, the creation of a negative energy balance, which is necessary for all slimming regimens, invokes a wide variety of different physiological outcomes each of which is likely to be affected by a number of influencing variables. The outcomes include: energy-sparing adaptations, alterations in fat and lean body mass, altered energy substrate handling, changes in appetite, health-related outcomes (e.g., glucose tolerance), and altered hormonal status (24).

Thus, before we can say that we have a complete understanding of a complex system such as the one governing the processes of human bioenergetics, it is necessary to show that our knowledge of the individual components can be put together in a total system, i.e., an organization can be synthesized, which allows for the interactions of all the relevant variables and for all the structural components. Thanks to the availability of affordable, high-quality computing capabilities, we can now construct silicon surrogates of complex real-world processes. No longer do we have to break off chunks of the actual system to study in isolation with the hope that we can then reassemble these chunks of partial knowledge into an understanding of the overall system itself (8).

The research effort described in this paper builds upon and extends what has been learned about the micro-components of human bioenergetics to construct a holistic mathematical model integrating the processes of human metabolism, hormonal regulation, body composition, nutrition, and physical activity. These processes are typically fragmented between many different disciplines and conceptual frameworks. This work, thus, seeks to bring these processes together highlighting the interdependence of these various aspects of the complex system that is the human body.

Figure 1 provides an overview of the model's four major subsystems and some of their interrelationships. The model's four interrelated subsystems are body composition, energy intake, energy expenditure, and energy metabolism. The remainder of this section provides an overview of the first three subsystems, followed by a more detailed discussion of the energy metabolism subsystem. The later discussion is intended to serve two purposes. First, it sets the conceptual groundwork for interpreting the results of the simulation experiments presented in the next section. Second, it introduces the reader to the structure and formulation of a System Dynamics model. [A more detailed description of the model's entire structure and its validation is provided in (1).]

Body composition subsystem. In the model, body composition is viewed in terms of two compartments: fat mass (FM) and fat-free mass (FFM). Body fat is of two types: 1) essential fat, which is the fat associated with bone marrow, the central nervous system, viscera (internal organs), and cell membranes; and 2) storage fat, which is fat (triglyceride) stored in adipose tissue. (Differences in regional adipose tissue distribution independent of total body fat are thus below the level of aggregation of this model.) FFM constitutes the skeleton, muscle, connective tissue, organ tissues, skin, bone, and water.

After digestion and absorption, nutrient fat is mainly used to fill the triglyceride stores in adipose tissue (18). In the model, the size of the storage fat compartment reflects both the number and size of the fat cells. Fat cells expand in size as they fill with fat droplets until they reach their biologic upper limit (approximately 1.0 μg of fat) (21). When the cells reach their maximum size, they divide, increasing cell number. Thus, obesity develops when a person's fat cells increase in number, in size, or quite often both. With fat loss, the size of fat cells dwindles but not their number (29).

Whenever significant amounts of body weight are lost/gained, both FFM and FM participate in the weight loss/gain process. The relative contribution of these two body components to weight gain/loss depends, in part, on a person's initial body composition. On the basis of body composition studies of individuals of varying body fat content, Forbes (10) derived an empirical relationship that predicts reasonably well the relative changes in body composition when body weight (W) is gained or lost:

$$d(\mathrm{FFM})/d(\mathrm{FM}) = 10.4/\mathrm{FM}.$$

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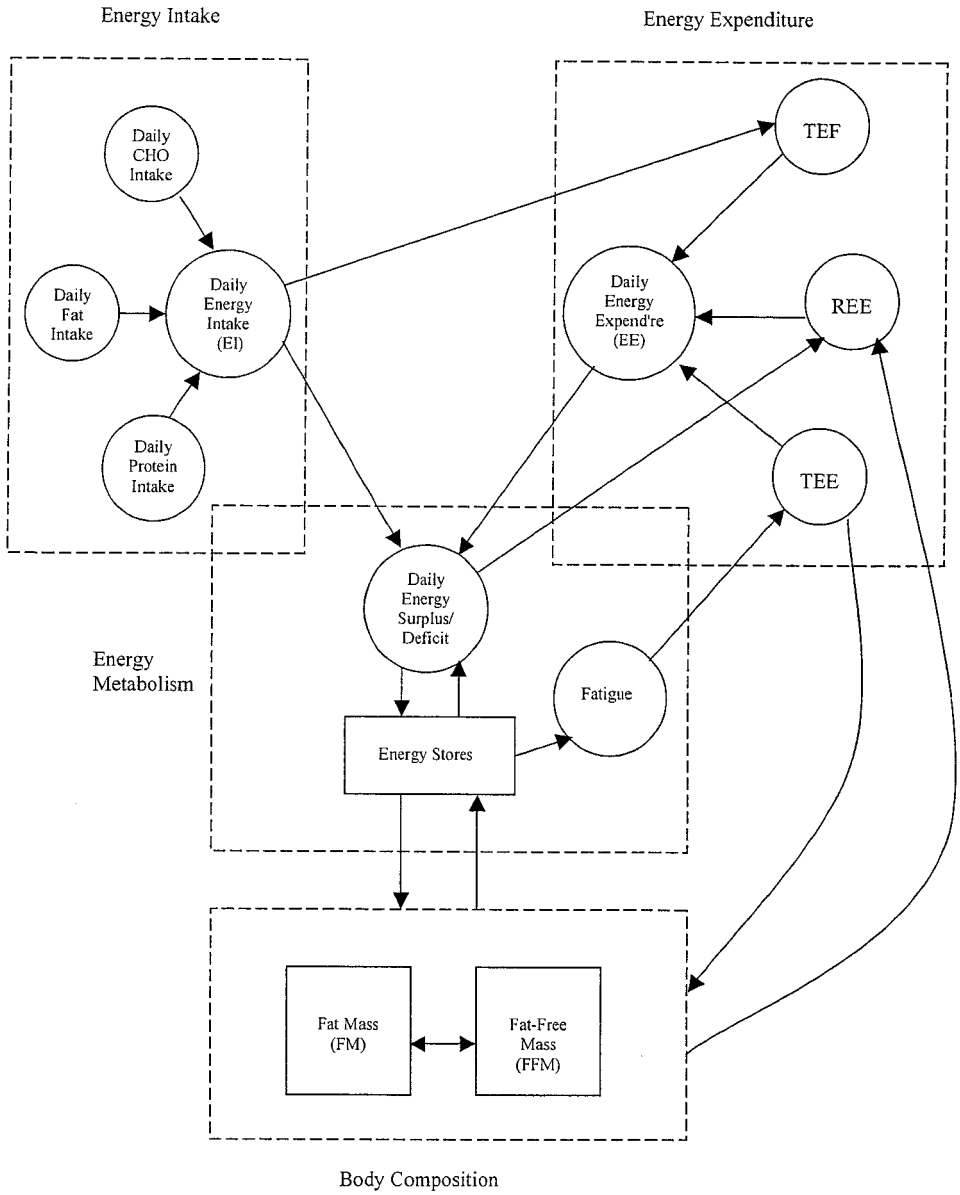

FIGURE 1—Overview of the model's four subsystems.

This empirically derived relationship is implemented in the model to assess the composition of weight loss/gain due to energy restriction/surplus. (In a System Dynamics computer program, such relationships are implemented in the form of difference equations.) However, as will be demonstrated later, there are situations in which this FM/FFM linkage may be violated. Exercising, for example, makes it possible to lose some weight without sacrificing FFM (5). This favorable impact on body composition occurs partly because of increases in muscle size through increased protein synthesis during exercise (21). Such skeletal muscle adaptations are rapid, and they rapidly plateau, which suggests that a ceiling exists with respect to the ability of exercise to increase muscle mass, a ceiling that depends on exercise type (3). This is captured in the model as a "logistic equation," an S-shaped exponential growth that eventually plateaus, with a ceiling parameter (defining the ceiling for muscle enlargement rate) of $0.15\%\cdot d^{-1}$ (5,6,21,27).

Energy intake and expenditure subsystems. Daily energy intake is defined in the model by the daily food energy consumed together with the diet's composition (carbohydrates, fats, and proteins). The energy intake subsystem models the body's use of the energy-yielding nutrients to fuel its metabolic and physical activities, and rearranges any excess into storage compounds to be used between meals and overnight when fresh energy supplies run low.

The body's total energy expenditure is divided in the model into three components. The largest component is the resting energy expenditure (REE). In most sedentary adults, the REE makes up about 60–70% of total energy expenditure. As shown in Figure 1, three factors establish the value of REE in the model. FM and FFM, the two compartments constituting the body's total mass, establish the body's nominal REE. The following empirically derived formulation is used in the model (28),

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$$\text{Nominal REE} = 0.024 \cdot \text{FM} + 0.102 \cdot \text{FFM} + 0.85\ (\text{MJ} \cdot \text{d}^{-1}).$$

The third factor affecting REE is the body's energy balance (16). During caloric deprivation, the body adapts physiologically to decrease the metabolic activity of the tissue mass at the cellular level in order to conserve energy and restrain the rate of weight loss. In the event of over-consumption and weight gain, there is in a converse fashion, an exaggerated increase in daily REE above that expected from the increase in tissue mass (20,25). That is, REE adjusted for metabolic mass increases with positive energy balance and decreases with negative energy balance. Research findings suggest (and it is assumed in the model) that this adjustment in REE increases/decreases linearly (and by as much as 20%) with the changes in the average adipose cell size (9,14,25). This is because the amount of energy stored in the fat cells faithfully reflects changes in the body's energy balance (the difference between caloric intake and expenditure) over time.

The second component of energy expenditure is the amount of energy the body uses to process food, known as the thermic effect of food (TEF). The TEF accounts for approximately 10% of the daily energy expenditure but can vary depending on the amount and the composition of the diet (29).

The third component of daily energy expenditure is the energy expended for muscular work, known as the thermic effect of activity (TEA). The TEA of an individual not engaged in heavy labor accounts for 15–20% of daily energy expenditure but can increase by a factor of 2 with very heavy exercise (29). As per the reference standards of the World Health Organization, the energy cost of routine physical activity is determined in the model as the product of the REE and an appropriate activity factor, namely, the physical activity ratio. Thus, the 24-h energy expenditure is equal to the REE multiplied by an appropriate physical activity factor and added to the energy cost of diet-induced thermogenesis.

Energy metabolism subsystem. Figure 2 depicts the structure for glucose metabolism. The four building blocks for metabolizing glucose are: 1) digestion/absorption, 2) hormonal regulation, 3) utilization, and 4) storage. The metabolic processes for amino acids and free fatty acids (FFA) have similar structures. The schematic conventions used in Figure 2 are the standard conventions used in System Dynamics models and are discussed in the Appendix. The equations, units, and typical initial values of the key stock variables are summarized in Table 1.

Glucose and FFA metabolism. In a normal individual, the blood's glucose concentration is maintained within the narrow range of 70–100 $\text{mg} \cdot \text{dL}^{-1}$ (17). Because maintaining glucose balance is critical, the body uses glucose frugally when the diet provides only small amounts (relying more on fat as a source of energy) and freely when stores are abundant (displacing fat in the fuel mix) (29).

In the model (as in reality), this homeostatic regulation is achieved primarily by two hormones, namely, insulin, which moves glucose from the blood into the cells for use as an energy source, and glucagon, which brings glucose out of storage (primarily in the liver) when necessary. Most of the cells take only the glucose they can use for energy right away, but the liver and muscle cells can assemble the small glucose units into long, branching chains of glycogen for storage, a process called glycogenesis. After the liver and muscle cells have stored as much glycogen as they can—an amount sufficient to supply the energy needs of the body for only 12–24 h—the additional glucose is converted by the liver into fat and is stored in the fat cells. Thus, high blood glucose returns to normal as excess glucose is stored as glycogen (which can be converted back to glucose) and fat (which cannot be) (13).

When carbohydrates are available in short supply, all the fat-sparing effects of carbohydrates are lost and actually reversed. This is achieved through several hormonal changes that take place to promote rapid fatty acid mobilization from adipose tissue and its utilization for energy in place of the absent carbohydrates. Among the most important of these is a marked decrease in pancreatic secretion of insulin caused by the absence of carbohydrates. In the absence of insulin, the enzyme hormone-sensitive lipase in the fat cells becomes strongly activated. This causes hydrolysis of the stored triglycerides, releasing large quantities of fatty acids and glycerol into the circulating blood. Consequently, the plasma concentration of FFA begins to rise within minutes. FFA then become the main energy substrate used by essentially all tissues of the body besides the brain (13).

When the hormonal system senses a drop in the blood glucose concentration level (as may occur between meals), the alpha cells of the pancreas respond by secreting the hormone glucagon into the blood. Glucagon raises blood glucose by signaling the liver to dismantle its glycogen stores and release glucose into the blood, a process called glycogenolysis (29). If a person does not eat carbohydrates to replenish the liver's limited glycogen stores, they will be quickly depleted. If this happens (e.g., due to dietary restriction or prolonged exercise), body proteins are dismantled to make glucose to fuel the brain's special cells. The conversion of protein to glucose, a process called gluconeogenesis, is the body's metabolic pathway to augment glucose availability and maintain plasma glucose levels in the face of depleted glycogen stores (29). The price paid, however, is a temporary reduction in the body's protein stores, particularly muscle protein.

Insulin/glucagon regulation of glucose are examples of negative feedback loops that are integrated into and imbedded within the larger system of glucose metabolism shown in Figure 2. For the reader's convenience, the insulin feedback loop is extracted and presented in isolation in Figure 3.

Protein/amino acid metabolism. Unlike glucose and FFA, whose primary task is to fuel biologic work, the primary task of amino acids is to provide the major building blocks for the synthesis of body tissue. A total of 20–30 g of the body's proteins are degraded daily and used to produce other body chemicals, a process referred to as the daily obligatory loss of proteins. Therefore, all cells must continue to form new proteins to take the place of those that are

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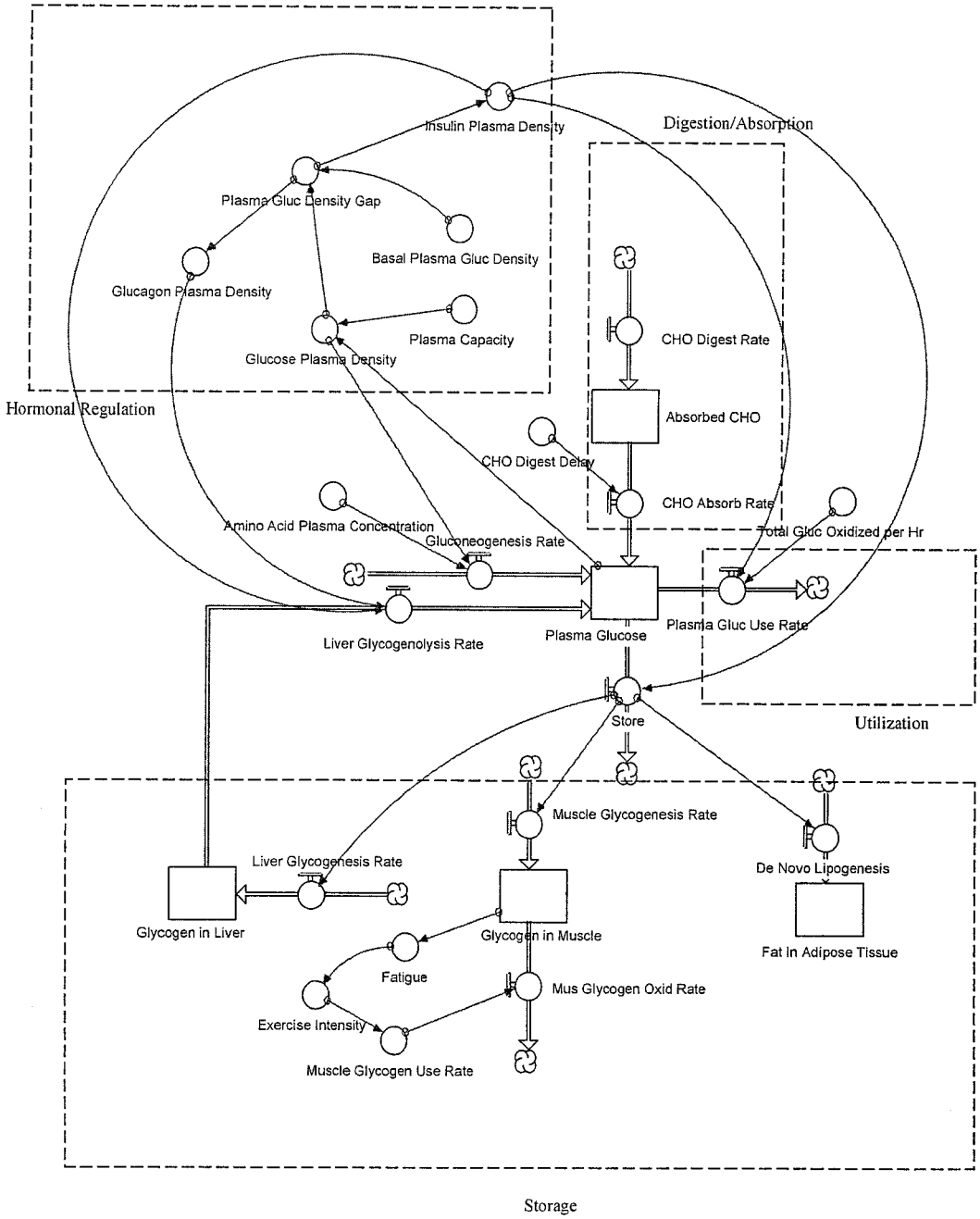

FIGURE 2—Glucose metabolism.

being destroyed. To prevent a net loss of protein from the body, a daily supply of protein is needed in the diet. Once the cells replenish their protein stores, any additional amino acids in the blood are used for energy (displacing fat in the fuel mix) or stored as fat (13).

Even though amino acids are needed to do the work that only they can perform—build vital proteins—they will be sacrificed to provide energy and glucose if need be. When this occurs, the protein is not extracted from specialized storage depots; rather, protein in the body is available only as the working and structural components of the tissues. Thus, to supply the needed energy when supplies of glucose and/or FFA are inadequate, the body in effect dismantles its own tissue and uses them for energy. Although this may appear like a heavy price to pay, it is a life-saving adaptation, for without energy, cells die and without glucose the brain and nervous system falter.

Exercise metabolism. During physical activity, the muscles' requirements for fuel are met by mobilization of reserves within muscle cells and from extra-muscular fuel depots. How much of which fuels they use depends on an interplay among the fuels available from the diet, the intensity and duration of the activity, and the degree to which the body is conditioned to perform that activity (29).

During low- to moderate-intensity physical activity, the lungs and circulatory system have no trouble keeping up

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TABLE 1. Stock variables in Figure 2.

Variable Name	Units	Typical Initial Values	Diff. Equation
Absorbed CHO	g	50 g	Absorbed_CHO(t) = Absorbed_CHO(t − dt) + (CHO_Digest_Rate − CHO_Absorb_Rate) * dt
Plasma glucose	g	5 g	Plasma_Glucose(t) = Plasma_Glucose(t − dt) + (CHO_Absorb_Rate + Gluconeogenesis Rate + Liver Glycogenolysis Rate − Plasma_Glucose_Use_Rate − Plasma_Glucose Store_Rate) * dt
Glycogen in liver	g	100 g	Glycogen_in_Liver(t) = Glycogen_in_Liver(t − dt) + (Liver_Glycogenesis_Rate − Liver_Glycogenolysis_Rate) * dt
Glycogen in muscle	g	1–3% of muscle mass	Glycogen_in_Muscle(t) = Glycogen_in_Muscle(t − dt) + (Muscle_Glycogenesis_Rate − Muscle_Glycogen_Oxid_Rate) * dt
Fat mass	kg	15–25% of initial weight	Fat_Mass(t) = Fat_Mass(t − dt) + (Lipogenesis_Rate − Lipolysis) * dt
Fat-free mass	kg	75–85% of initial weight	Fat_Free_Mass(t) = Fat_Free_Mass(t − dt) + (FFM_Gain_Rate − FFM_Loss_Rate) * dt

with the muscles' need for oxygen—the activity is aerobic. With the availability of oxygen, the muscles can derive their energy from both glucose and fatty acids because both can be oxidized to provide energy (29).

Early in a session of moderate exercise, approximately half the energy expended is derived from glucose and half from FFA. To maintain the supply of glucose in circulation, liver glycogen is converted into glucose and released into the bloodstream. The muscles use both this glucose and their own glycogen stores to fuel their work. But as exercise continues for an hour or more, two things happen. First, the liver's limited store of glycogen is depleted. As this happens, glucose output by the liver fails to keep pace with muscle use and so blood glucose concentration drops (21).

Second, the hormone epinephrine is released by the adrenal medullae as a result of sympathetic stimulation. In response, the fat cells begin to rapidly break down their stored triglycerides, liberating fatty acids into the blood. The FFA concentration in the blood can rise as much as eightfold, and the use of these fatty acids by the muscles for energy is correspondingly increased (13). This causes a steady decline in the combustion of glucose for energy with a concomitant increase in FFA utilization. Toward the end of prolonged exercise, FFA may supply as much as 80% of the total energy required.

Intense activity presents a different metabolic situation. Whenever a person exercises at a rate that exceeds the capacity of the heart and lungs to supply oxygen to the muscles, aerobic metabolism cannot meet energy needs. Instead, the muscles must draw more heavily on glucose, which is the only fuel that can be used anaerobically (29). In addition, a selective dependence on glucose metabolism during intense physical activity has an additional advantage, namely, its rapidity for energy transfer compared to fats (about twice as fast) (21).

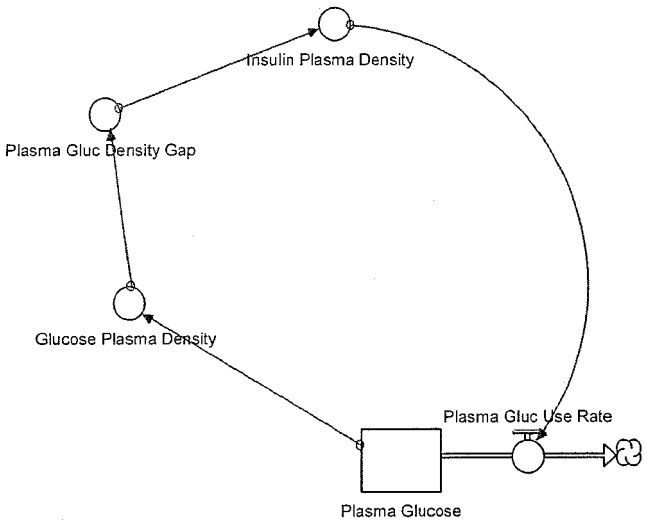

FIGURE 3—Insulin feedback loop.

At the start of an intense exercise session, glucose energy is supplied from the glycogen stored in the active muscles. As exercise continues and the muscles' glycogen stores decline, blood glucose becomes the major source of glucose energy. [Glycogen depletion usually occurs within 1–2 h from the onset of intense activity (21).] Muscle glycogen depletion causes muscle fatigue, which in turn greatly diminishes exercise capacity, making continued exertion more and more difficult. With the depletion of liver and muscle glycogen and a continued large use of blood glucose by active muscle, blood glucose eventually falls to hypoglycemic levels (less than 45 mg of glucose per 100 mL of blood) (21). The symptoms of a modest reduction in blood glucose (hypoglycemia) include feelings of weakness, hunger, and dizziness. Endurance athletes commonly refer to this sensation of fatigue as "bonking" or "hitting the wall."

RESULTS

As mentioned earlier, the experimental results to date with respect to the relationship between exercise behavior and obesity treatment have been mixed. As a result, there is a lack of consensus among both researchers and practitioners with respect to the effects of exercise training on body weight. Yet, the argument for favoring exercise in treating obesity remains compelling. It goes something like this: Exercise training can protect against the loss in FFM usually observed when weight loss is achieved through dietary restriction. By conserving and even increasing the fat-free body mass (the principal metabolically active component of total body mass), exercise may blunt the drop in resting metabolism that frequently accompanies diet-based weight loss. Thus, using exercise as a focus of treatment contributes to total energy expenditure both through the cost of the exercise itself and through its possible effect of reversing or offsetting a diet-induced depression in metabolic energy (5,21).

In this section, the results of two simulation experiments to investigate the impacts of physical activity on body weight and composition will be discussed. The experiments' results replicate the "mix" of results reported in the literature, as well as provide causal explanation for their variability. The first experiment compares the impacts of food restriction and exercise on the amount and composition of

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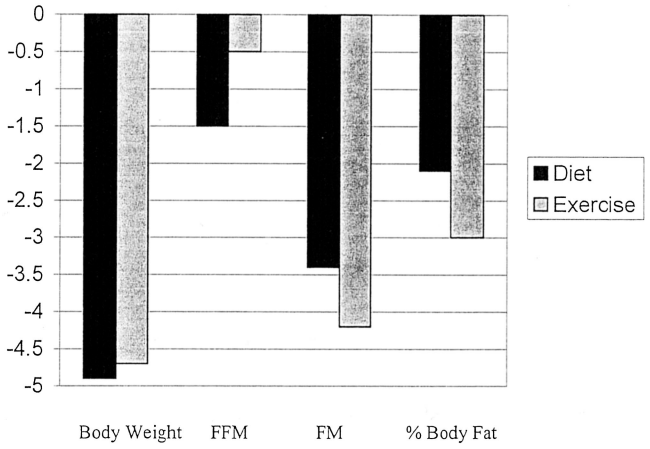

FIGURE 4—Diet vs exercise.

weight loss. In the second experiment, the potential interaction effects between diet composition and physical activity is investigated.

Diet versus exercise. The objective of the first experiment was to compare the impacts of diet- and exercise-induced energy deficits on the amount and composition of weight loss. The subject of the simulation is an overweight sedentary male subject with an initial total weight of 100 kg and 25% body fat, maintained on a daily dietary input of 14.25 MJ composed of 50% carbohydrate, 35% fat, and 15% protein.

Following recommendations for desirable daily caloric reductions in the obesity-treatment literature, a 2 $MJ \cdot d^{-1}$ energy deficit is induced, first by food restriction and then in a second simulation run by a light daily exercise. A 2 $MJ \cdot d^{-1}$ exercise regimen is equivalent to a 2-h-long leisurely walk. The duration for both experimental treatments was 2000 h (12 wk).

A comparison of the changes in body weight and composition (FM, FFM, and percent body fat) for the dieting and exercising interventions are shown in Figure 4. The first thing to notice is that the difference in weight loss between dieting and exercising is not significant. After 12 wk of dieting, body weight dropped by 4.9 kg (to 95.1 kg), whereas a daily exercise regimen for the same period caused weight to drop a comparable 4.7 kg (to 95.3 kg).

The composition of the weight lost was quite different, however. As Figure 4 shows, in the exercising treatment, FM dropped by 4.2 kg (from 25 kg to 20.8), whereas FFM dropped by only 0.5 kg (from 75 to 74.5). That is, 90% of the weight loss was FM and only 10% was FFM. This contrasts with the dieting treatment in which 70% of the weight loss was FM and 30% was FFM (FM dropped by 3.4 kg, whereas the FFM loss was 1.5 kg).

The results, thus, demonstrate that although a program of moderate exercise coupled with a balanced diet results in approximately the same weight loss as an energy restricted diet, exercise can favorably modify the composition of the weight lost, resulting in more weight being lost in the form of fat. Of equal importance is the fact that conservation of FFM occurred with exercise training that was, for the most part, fairly moderate.

The interesting question now is why, despite the favorable impact that exercising did have in protecting against the loss in FFM, it failed to induce the argued-for larger losses in body weight. Recall that the argument for exercise as the preferred treatment strategy is that by conserving or increasing FFM, exercise would sufficiently blunt the drop in resting metabolism that accompanies diet-based weight loss to affect a larger energy deficit and, in turn, induce a larger loss in body weight. The answer is twofold.

First, consider how the FM and FFM compartments adjusted over time to the diet and exercise interventions (shown in Fig. 5, A and B). Notice that at the start of the exercise scenario, there is an initial rise in FFM (curve 2 in Fig. 5B). This reflects the increase in muscle size for our sedentary obese subject as he embarks on the 2 $MJ \cdot d^{-1}$ exercise regimen (a relatively high overload for him). Such exercise-induced physiologic changes, however, depend primarily on the relative intensity of the overload. Thus, as exercise continues at the same intensity level, the initial adaptive responses eventually level off as fitness improves and the initial training stimulus is rendered subthreshold (21). Ultimately, FFM declines (as does FM), because of the cumulative effects of sustaining a daily 2-MJ energy deficit.

In both treatments, the body relies on its FM reserves to compensate for the daily caloric deficit. However, in the exercise treatment, additional FM energy is called upon to compensate for the initial buildup and then the smaller decline in FFM. Although this obviously induces larger losses in FM in the exercise treatment, it also (perhaps less obviously) precipitates a net gain in body mass. This is because in an energy-deficit situation such as experienced here, gaining a kg of FFM consumes considerably less than the metabolic energy stored in a kg of FM. Using the most commonly used hydration constant for FFM of 0.73 (i.e., assuming FFM to being 73% water and 27% protein), one arrives at the energy densities of FFM and FM to be 6 and 38 $MJ \cdot kg^{-1}$, respectively (28). And so, one reason for the smaller net drop in total body weight in the exercise intervention is that smaller losses in FM are used to fuel larger gains in FFM.

Next, the impacts of the dieting and exercising treatments on the resting metabolic rate were compared. Figure 6 shows that initially a gap does develop between the two REE curves, but as the experiment progresses REE progressively declines in both experimental treatments and the gap between them eventually disappears. To understand why, remember that FM and FFM establish the body's nominal REE. The actual value of REE is obtained after the body adjusts this nominal value for any energy imbalances—increasing REE when in positive energy balance and decreasing it when in negative energy balance (20).

By protecting against the loss in FFM, which as explained earlier is the more metabolically active component of total body mass, exercising does indeed induce a consistently higher level of nominal REE, as clearly demonstrated in Figure 7. However, the very same dynamic (the preservation

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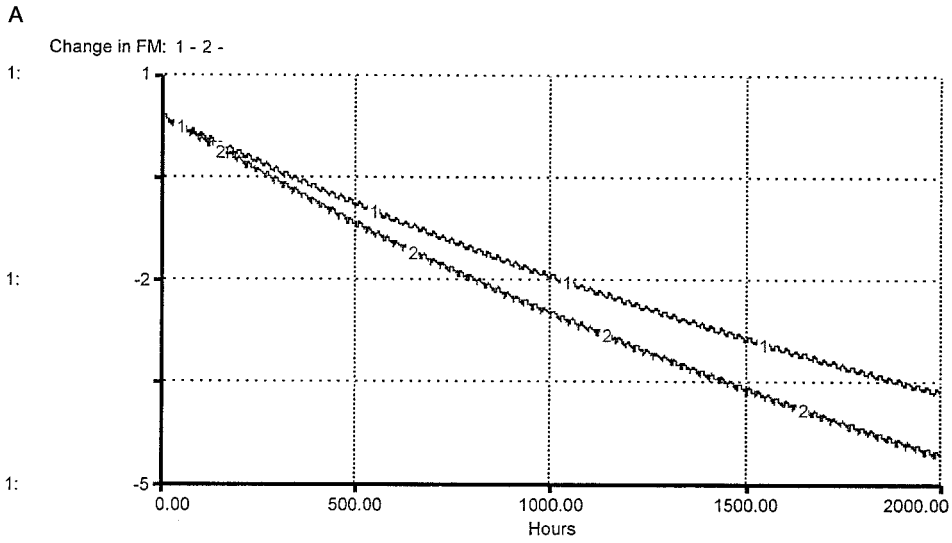

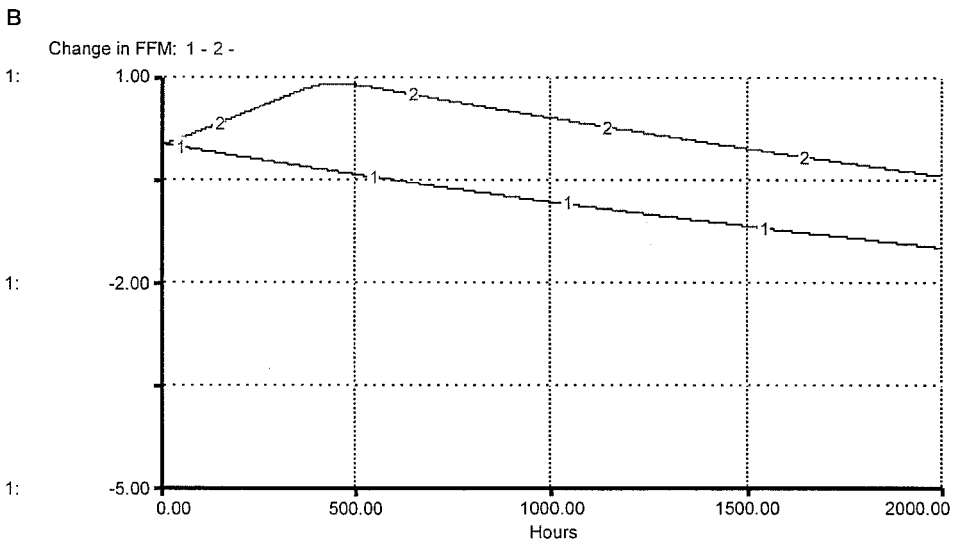

FIGURE 5—(A) Change in FM (kg) in response to dieting (1) and exercise (2). (B) Change in FFM (kg) in response to dieting (1) and exercise (2).

of FFM) precipitates, as explained above, relatively larger losses in FM that, in turn, induce a larger adaptive reduction in the metabolic rate. This is because the amount of energy stored in the form of fat faithfully reflects changes in the body's energy balance (the difference between caloric intake and expenditure) over time. And so, a larger drop in FM, as would be affected by a larger caloric deprivation, induces a larger suppression in REE. Research findings have

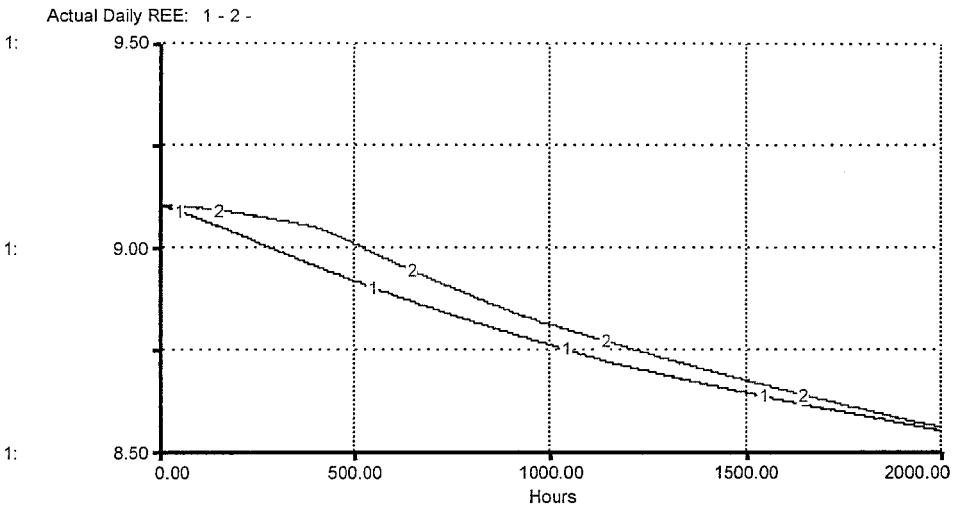

FIGURE 6—Actual daily REE ($MJ \cdot d^{-1}$) in response to dieting (1) and exercise (2).

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demonstrated that after adjustment for decreased body mass the amount of reduction in REE during the dynamic phase of weight loss is largely predicted by the degree of body fat depletion, i.e., the greater the degree of fat depletion, the greater the suppression of REE. Furthermore, such adaptive reductions in REE can be significantly greater than the predicted nominal changes in REE that would be accounted for by the changes in body mass (9). Dullo and Jacquet (9) concluded that the demonstrated specificity of the relation between suppressed REE and fat depletion ". . . may reflect the operation of a control system with a negative feedback loop between a component of regulatory thermogenesis and the state of depletion of the fat stores."

In summary, despite favorably protecting against the loss in FFM, an exercise treatment can fail to induce the argued-for larger losses in body weight for at least two reasons. First, exercise can cause a net increase in total body weight when smaller losses in FM are used to fuel larger gains in FFM (primarily in the form of muscle). Second, although the conservation/increase in FFM does indeed partially blunt the drop in nominal REE (as seen in Fig. 7), the very same dynamic (the preservation of FFM) affects relatively larger losses in FM, which in turn induce a larger adaptive reduction in the metabolic rate. The two adaptations almost totally offset one another, causing minimal differences in actual REE between the dieting and exercising scenarios (as seen in Fig. 6).

Exercise intensity × diet composition interaction. Because for most people the most costly aspect of exercise is the time spent doing it, there is an understandable inclination to aim at exercising at the highest possible level of intensity in order to induce the largest possible energy deficit per exercise session. A second experiment was conducted to assess just how much would be gained by aiming to exercise at higher levels of intensity in a person's available time window.

Three exercise intensity targets were evaluated: exercising at a level of 1 $MJ \cdot h^{-1}$, 2 $MJ \cdot h^{-1}$, and a high intensity level of 3 $MJ \cdot h^{-1}$. In all cases, the exercise duration was set at 2 h every day. (Note that the lowest exercise level of 1 $MJ \cdot h^{-1}$ is the same as that used in the first experiment.) As with the first experiment, the initial weight of the simulated subject was 100 kg with 25% body fat, maintained on a dietary energy input of 14.25 $MJ \cdot d^{-1}$ composed of 50% carbohydrate, 35% fat, and 15% protein. The simulated duration was also the same, 2000 h (12 wk).

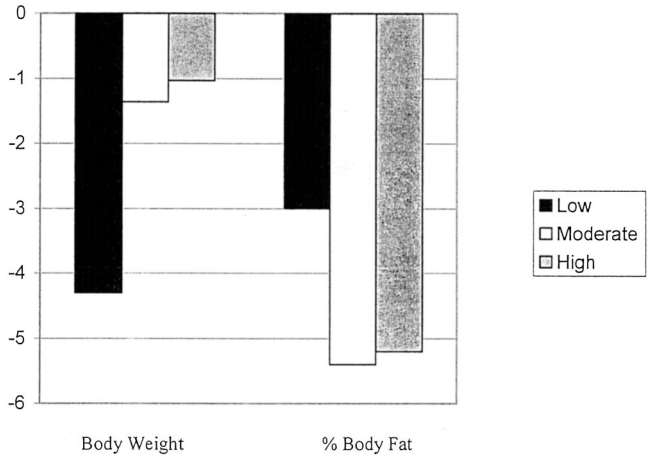

FIGURE 8—Impact of different exercise intensity levels.

The resulting losses in body weight and changes in body fatness at the three exercise intensity levels are presented in Figure 8. Notice that although the percentage of body fat decreased as the subject exercised more intensely, surprisingly, less weight was lost.

Two factors contributed to the smaller losses in body weight at the higher intensity levels. First is the increase in FFM (mainly muscle mass) that accompanied the increase in the level of exercise intensity. This is clearly depicted in Figure 9, which compares the changes in weight and body composition between the 1 and 2 $MJ \cdot h^{-1}$ exercise intensity levels. Exercising at the low intensity level caused total body weight to drop by 4.7 kg (from 100 kg to 95.3) as a combination of a modest 0.5-kg drop in FFM and a more substantial 4.2-kg drop in FM. Compare this to exercising at the medium level of 2 $MJ \cdot h^{-1}$ where total body weight

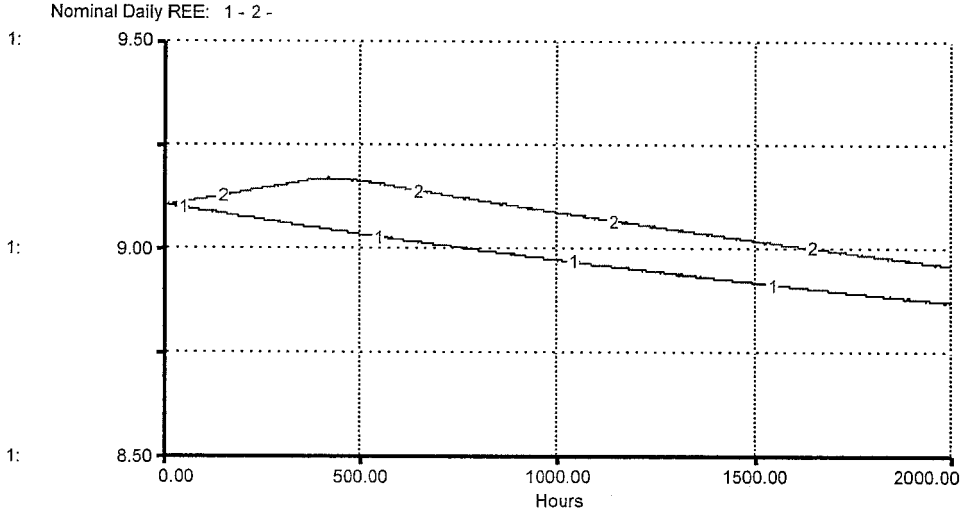

FIGURE 7—Nominal daily REE ($MJ \cdot d^{-1}$) in response to dieting (1) and exercise (2).

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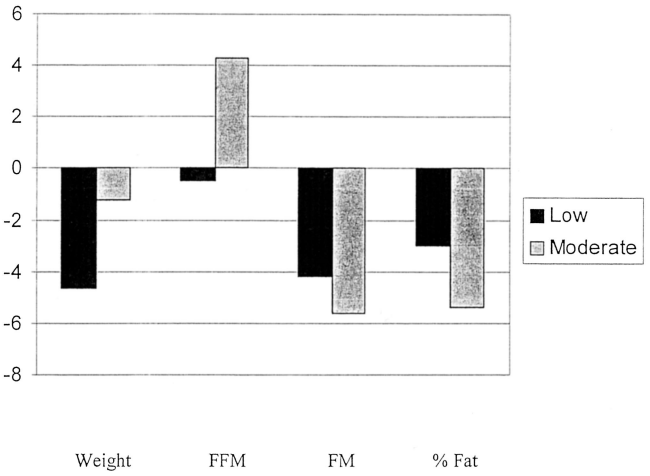

FIGURE 9—Low vs moderate exercise.

dropped by only 1.3 kg (from 100 to 98.7 kg). Although FM decreased by 5.6 kg, the loss in total body weight was minimal because FFM actually increased by 4.3 kg. This, in turn, caused a favorable drop in percent body fat, lowering it from 25% to 19.6%.

The second factor contributing to the smaller than expected loss in body weight at higher exercise intensity levels is perhaps less obvious. It is an example of "policy resistance," a phenomenon often encountered in complex feedback systems whereby interventions are delayed, diluted, or defeated because of compensating (and often unanticipated) feedback in the system (27). Here, the strategy of aiming to increase exercise intensity in order to induce a larger energy deficit per exercise session can, and in the case of our sedentary 100-kg obese subject does, collide head on with biological realities. The results are seen in Figure 10. At the low intensity level, the subject's actual energy expenditure matched the target level of 14 $MJ \cdot wk^{-1}$. In the moderate intensity scenario, he only expends 63% of the weekly target of 28 MJ. And, when a high intensity type of exercise is selected, he ends up expending less exercise energy in absolute terms (16 vs 17 $MJ \cdot wk^{-1}$ in the moderate intensity case).

In this case, the resistance to the high intensity strategy lies in the body's selective dependence on different energy sources to fuel differing levels of exercise activity and a person's capacity to meet the body's specific energy needs. As was explained earlier, whenever a person exercises at a rate that exceeds the capacity of the heart and lungs to supply oxygen to the muscles, the muscles must draw more heavily on glucose (primarily in the form of muscle glycogen), because it is the only fuel that can be metabolized to produce energy without the simultaneous utilization of oxygen. During intense exercise, a selective dependence on glucose metabolism has an additional advantage, namely, its rapidity for energy transfer compared with fats (about twice as fast).

How much exercising would a particular person's glycogen reserves sustain depends on the intensity and duration of effort, as well as the fitness and nutritional status of the exerciser. An overweight sedentary person embarking on a new exercise regimen to treat obesity (like our 100-kg experimental subject) will not only start with more limited glycogen stores but will also tend to use his reserves at a faster rate than, for example, a trained athlete. This is because muscle cells that repeatedly deplete their glycogen through exercise adapt to store greater amounts of glycogen to support that work. Conditioned muscles also rely less on glycogen and more on fat for energy, so glycogen utilization occurs more slowly in trained than in untrained individuals at a given work intensity (29).

Generally, glycogen depletion usually occurs within 1–2 h from the onset of intense activity. As glycogen is depleted, the muscles become fatigued, which in turn greatly diminishes the capacity to continue exercising at a high intensity level. (This dynamic is what causes energy expenditure level to drop at the higher intensity level in the experiment). Recovery from exhaustive muscle glycogen depletion often requires days. But because comparatively little glycogen is stored in the body, its recovery time can be influenced considerably by the composition of the diet. For example, on a high-carbohydrate diet, full recovery occurs in about 2 d. On the other hand, people on a high-fat/high-protein diet or on no food at all show extremely little recovery even after as long as 5 d (13).

Thus, how much carbohydrate a person eats should influence the capacity to maintain a daily exercise regimen. To quantitatively assess this influence, the relatively balanced composition of the daily diet was changed from 50% carbohydrate, 35% fat, and 15% protein to a high-carbohydrate diet of 75% carbohydrate, 10% fat, and 15% protein, and a new set of simulations run for the three exercise intensity levels.

Figure 11 compares the results of the high-carbohydrate diet together with the earlier results from the balanced diet. The differences are particularly dramatic at the medium exercise intensity level (of 2 $MJ \cdot h^{-1}$). With the new high-carbohydrate diet, total body weight dropped by 10.2 kg (from 100 to 89.8 kg), an amount that is almost 8 times as large as the weight lost with the balanced diet at the same exercise intensity level. (The composition of the 10.2-kg loss was as follows: a 7.8-kg drop in FFM combined with a 2.4-kg loss of FM, inducing a drop in percent body fat

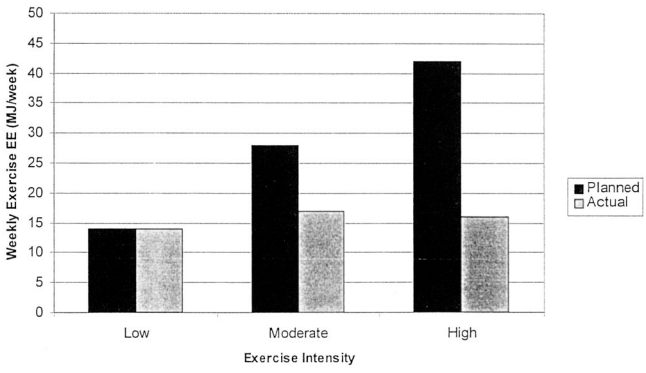

FIGURE 10—Actual vs planned weekly exercise energy expenditures at different exercise intensity levels.

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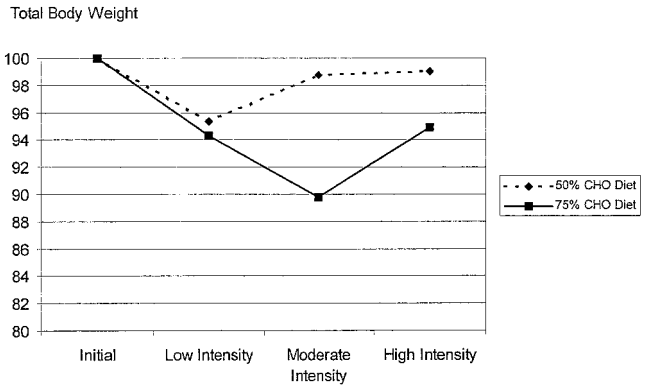

FIGURE 11—High-carbohydrate vs balanced diet.

dropped to 19.2.) The drop in body weight is also almost double the 5.7-kg loss (from 100 to 94.3 kg) achieved when exercising at the low intensity level of 1 MJ·h^{-1} on the new high-carbohydrate diet.

By replenishing the glycogen stores more effectively, the daily dose of high-carbohydrate diet maintained the body's capacity to exercise daily at a higher intensity level. As a result, the cumulative exercise energy expended per exercise session and consequently over the 12-wk period increased (Fig. 12). On the other hand, the increased level of carbohydrate in the new diet was still not large enough to replenish the more severe level of glycogen depletion suffered when exercising at the highest intensity level (3 MJ·h^{-1}). As a result, less cumulative energy was expended at the highest exercise level as compared with the moderate intensity level, which in turn resulted in a smaller weight loss (5.0 kg).

In toto, the results underscore the significant interaction effects between diet composition and physical activity, and emphasize the critical role that diet composition can have in exercise-based treatment interventions.

DISCUSSION

Summary of study findings. The current study was conducted within the context of a much broader research effort to study, gain insight into, and make predictions about the dynamics of human bioenergetics, metabolism, body composition, and physical performance. A major part of this effort was devoted to the development of a System Dynamics model that integrates the processes of human metabolism, hormonal regulation, body composition, nutrition, and physical activity. These processes are typically fragmented between many different disciplines and conceptual frameworks. This work, thus, seeks to bring these ideas together, highlighting the interdependence of these various aspects of the complex system that is the human body.

What is gained in understanding through the use of such an integrative mathematical model is achieved by comprehending the laws built into the model. Because a System Dynamics model is a causal model, with clear cause-and-effect connections between modeled system variables, it is particularly suited to serve as a framework to explain/understand experimental observations. This complements well the preponderance of statistical-type studies of human bioenergetics because even when high correlations are found, it is necessary to establish a likely reason for what is happening before any useful conclusions can be drawn (30).

To many, however, the practical outcome of a theoretical model is prediction. Through computer simulation, the System Dynamics model presented in this article is intended to predict the dynamic interactions of the body's interrelated physiologic/metabolic subsystems to gain insight into the consequences for human performance in health and disease.

The experimental results in this study replicate the "mix" of results reported in the literature on the relationship between exercise behavior and obesity development, as well as provide causal explanation for their variability. The results of the first experiment demonstrate that an active program of exercise can have at least two potential benefits pertinent to the treatment of obesity. First, a sustained exercise regimen, even at a moderate level, provides an important contribution to total energy expenditure through the cost of the exercise itself. Over a 12-wk period, weight loss from a moderate level of daily exercise (equivalent to a 2-h-long leisurely walk) was comparable to the loss from food restriction (when both produced equivalent energy deficits). This may provide a more palatable option to many patients who may view exercise as a strategy to help maintain weight loss while allowing the consumption of enough calories to supply the body with adequate nutrients as well as energy.

Perhaps of greater significance, exercise protects against the loss of FFM that occurs when weight loss is achieved through diet alone and thus promotes favorable changes in body composition. The results show a drop in percent body fat from 25% to 19.2% with a moderate level of exercise and a high-carbohydrate diet. This is potentially quite significant, because maximizing fat loss yields the greatest reduction in coronary heart risk (5). In addition, the conservation of muscle mass during exercise-induced weight loss maintains a person's ability to perform behavior of daily living requiring strength and/or muscular staying power.

Exercise failed to induce the argued-for larger losses in total body weight despite the favorable impact it did have in protecting against the loss in FFM for two reasons. First, exercise caused a net increase in total body weight when

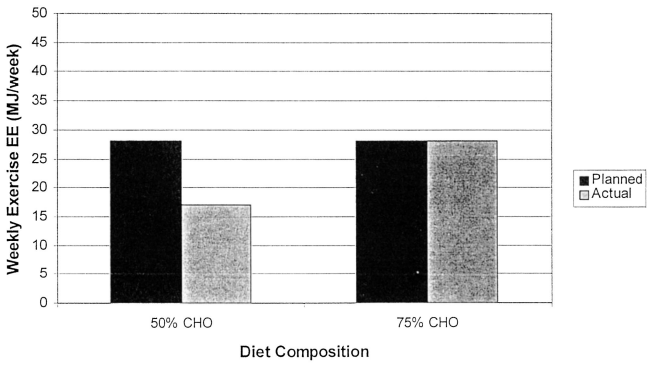

FIGURE 12—Planned vs actual weekly exercise energy expenditures at moderate exercise intensity, different diet compositions.

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smaller losses in FM were used to fuel larger gains in FFM (primarily in the form of muscle). Second, although the conservation in FFM did indeed partially blunt the drop in nominal REE, the very same dynamic (the preservation of FFM) affected relatively larger losses in FM, which, in turn, induced a larger adaptive reduction in the metabolic rate. The two adaptations almost totally offset one another, causing minimal differences in actual REE between the dieting and exercising scenarios.

In the second experiment, an exercise regimen of moderate to high level intensity proved counter-productive as a weight-reducing strategy in the case of an obese sedentary subject. This was due to the limited energy reserves (specifically, muscle glycogen) available to such individuals. However, when the diet was changed from a balanced composition to one that was highly loaded with carbohydrates, it became possible to sustain the high exercise effort over the prolonged experimental period, and achieve a significant drop in body weight.

The potential significance of these results is twofold. First, the results underscore the importance of considering the interaction of diet composition and physical activity in the treatment of obesity. Most previous research examined diet composition and physical activity independently.

Second, the results highlight the risks in many quick-loss diets promoting low carbohydrate intake. The results emphasize the important role diet composition can have in establishing the appropriate energy reserves for long-term exercise and training. Specifically, a diet deficient in carbohydrates results in dehydration as muscle and liver glycogen stores are depleted diminishing exercise capacity, which almost eliminates physical activity as a source of caloric expenditure.

Study limitations. Simulation-based experimentation entails giving up the richness of context to obtain control. A potentially limiting factor, therefore, concerns the nature of the experimental task. The experimental design limited the subject's decision task to a single dimension, for example, setting a target intensity level. In reality, the decision task can be richer involving decisions about exercise type, duration, time of day to exercise, etc. It should be noted, however, that this limitation could be somewhat relaxed because it is technically feasible to extend the System Dynamics model presented in this paper to explore (and optimize) multidimensional decision functions (a research direction that the author is currently pursuing.)

The generalizability of the current results is also tempered by the set of exercise parameters selected (e.g., an exercise regimen of 2 $MJ \cdot d^{-1}$, 7 $d \cdot wk^{-1}$). In real life, subjects have an almost infinite set of treatment options. No experiment, including this one, can therefore claim to generalize to all types of exercise treatments/regimens.

A third potentially limiting factor concerns the model's boundary. Although the model presented greatly expands on the previous research's capacity to include several simultaneous determinants of weight/body composition changes, like all models, it is limited by design (and necessity) in its boundary. For instance, (partially) genetic factors such as cardiovascular fitness and regional adipose tissue distribution are not captured in the current model.

Extending the current model to capture body fat topography would be a particularly promising research direction. Most of the research exploring this area has focused on the associated health risks of body fat distribution, in particular cardiovascular disease, diabetes, and hypertension. Using an extended version of the model to study the effects of weight-reduction interventions (diet or exercise) on preferential regional mobilization of fat, and how this may be affected by exercise type and intensity would be a worthwhile contribution to the extant literature.

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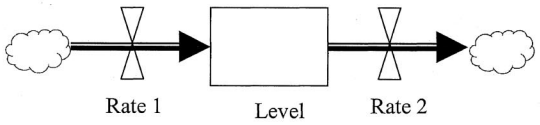
APPENDIX

System Dynamics Variables and their Notation

The schematic conventions used in Figures 2 and 3 are the standard conventions used in System Dynamics models. Model variables are one of three types: level, rate, and auxiliary. A *level* is an accumulation, or an integration, over time of flows or changes that come into and go out of the level. They characterize the state of the system and generate the information upon which decisions and actions are based. Levels give systems inertia and provide them with memory. The flows increasing and decreasing a level are called *rates*. Thus, *Glycogen in Liver* (in Figure 2) is a level that is increased by the *Liver Glycogenesis* Rate and decreased by the *Liver Glycogenolysis Rate*.

System Dynamics uses a particular diagramming notation for levels, rates and flows. As shown in the schematic below, levels are represented by rectangles (suggesting a container holding the contents of the level). Rates are represented by valves, inflows are represented by a pipe (arrow) pointing into (adding to) the level, and outflows are represented by pipes pointing out of (subtracting from) the level.



Mathematically, levels *integrate* or accumulate their flows; the net flow into the level is the rate of change of the level. Hence the structure represented above corresponds exactly to the following integral equation:

$$\text{Level}(t) = \int [\text{Rate1} - \text{Rate2}]dt + \text{Level}(t_0)$$

Where Rate1 and Rate2 represent the value of the inflow at any time t between the initial time t_0 and the current time t. Equivalently, the net rate of change of any level, its derivative, is the inflow less the outflow, defining the differential equation

$$d(\text{level})/dt = \text{Rate1}(t) - \text{Rate2}(t).$$

The cloud-like symbol represent *sources* and *sinks* for the "stuff" that flows into and out of levels. The flows that are controlled by the rates are either information flows or physical flows. The two types of arrow designators are:

Physical Flows

Information Flows

In principal, mathematical description of a system requires only the levels and their rates of change. Usually, however, it is very difficult to write a rate equation without first doing some (often complex) algebraic computations. For ease of communication and clarity it is often helpful to define intermediate or *auxiliary variables*. Thus, auxiliary variables, as their name implies, aid in the formulation of rate equations. Auxiliary variables are represented

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