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# The non-regulation of food intake in humans: Hope for reversing the epidemic of obesity

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**Abstract**

Few doubt that human feeding behavior is part of larger biology regulatory system of energy stores, but the extent to which eating behavior is controlled by these biology systems and how much is due to responses to environmental stimuli is presently under debate. The results of a series of studies are presented which have attempted to determine the responsiveness of human feeding behavior to some of the "classic" biological variables that have conventionally been used to argue the biological basis of eating behavior. When humans are challenged with either overfeeding, underfeeding, or alterations of the caloric density of the diet, they fail to demonstrate precise caloric compensation. When challenged with changes in environmental stimuli, on the other hand, humans appear to be very sensitive to changes in portion size, the number of people with whom they eat, the amount that others eat and the variety of foods available. Other more chronic influences demonstrate that body weight appears to change when people move from one area of the world to another, when they enter the college environment, or when they either marry or break up. It is argued that because humans appear to be more responsive to the external environment than internal biological cues, it should be possible to curb or even reverse the epidemic of obesity by changing aspects of the external environment or human interactions with environmental variables rather than changing their internal environment through pharmacology.


*Keywords:* Control of food intake; Determinants of body weight; Energy imbalance; Epidemic of obesity

Ever since the classic publications of Gordon Kennedy [1], the idea that body weight is well regulated through the biological control of food intake has been firmly implanted in the literature. Indeed, the homeostatic model upon which Kennedy's model is based has a long and rich history in psychology [2]. More importantly, the regulation model of food intake fits uncannily well with contemporary views of the brain as a monitor of peripheral body fat depots via such messengers as leptin [3], insulin [4], acetylation-stimulating protein [5], and adiponectin [5], initiating a set of peptide and neurohumoral events that constitute the neural substrates of eating behavior and energy expenditure [6]. As the majority of the presentations at this symposium attest, most contemporary research on the controls of energy intake and expenditure is directed to the investigation of the various biological components involved in the regulation of body weight.

The way we conceptualize body weight within this regulatory model has a profound effect on the way we think about possible treatments for overweight and obesity. When the control of body weight is viewed as a biological problem, then the putative solutions to the problem of overweight and obesity will also be biological; i.e., drugs, surgery, etc. From this perspective any attempt to alter environmental variables such as to reduce portion size or introduce low-fat foods or even lose weight by dietary restriction would appear futile because the biological controls would detect the error and make the necessary adjustments in energy intake and expenditure to insure that body tissue is not gained or lost.

However, the average weight of people everywhere is increasing to such an extent that experts have been referring to the increase in body weight as an epidemic [7–17]. If body weight of humans is biologically regulated, it must not be regulated too precisely, otherwise why are we gaining weight? Several years ago, I suggested that biology, rather than determining a "Set-Point" for body weight, sets a range of body weights (Settling zone) that is regulated [18]. As depicted in Fig. 1, within this zone of body weight, however, those behaviors responsible for the determination of body weight,

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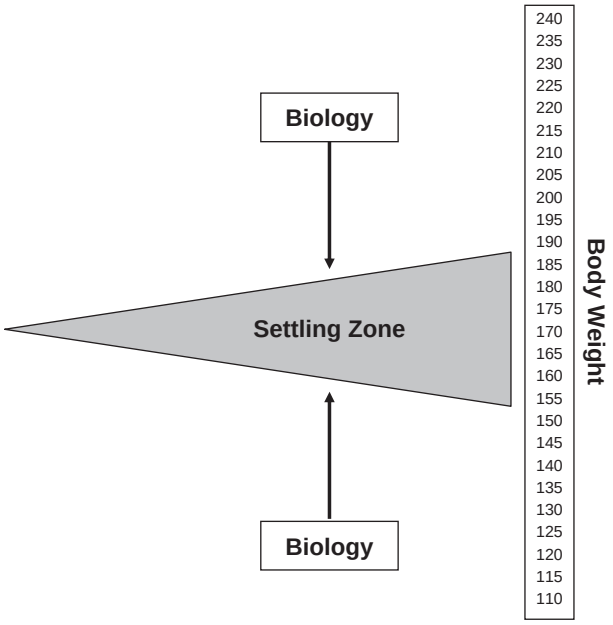


Fig. 1. Model of settling zone concept of the regulation of body weight. According to this model biology sets a range of values for an individual's body weight, but within the zone, body weight is determined by environmental variables that modulate behaviors involved in the control of energy intake and/or expenditure.

feeding behavior and physical activity, are not part of the regulatory system but rather are controlled to a large extent by external, environmental factors. Popkin also presents data from a very different perspective of the power of environmental factors determining body weight and perhaps the development of overweight and obesity [19].

The purpose of this paper is to review the evidence to determine how sensitive humans are to biological and environmental factors in order to determine the kind of approach we may use to curb, or even reverse, this epidemic of obesity.

## 1. Basic tests of the regulation of food intake

### *1.1. Energy deficit*

One of the most fundamental, and intuitive, demonstrations of the biological regulation of food intake is the food deprivation paradigm. Depriving an animal (or human) of food is almost always associated with an increase in behaviors associated with recovering that food. The implication of such thinking is that food deprivation is necessary for eating behavior to occur. George Collier put a major dent in this conceptual framework by demonstrating that for animals, food deprivation was not necessary to demonstrate an increase in behaviors associated with feeding. Indeed, animals would alter their feeding patterns to avoid deprivation [20,21].

Fortunately, most people in the industrialized world rarely endure more than 24 h without food. In fact, the longest period of food deprivation most people suffer each day usually occurs during the period when we sleep. When we awake we "break the fast" by consuming the first meal of the day, breakfast. Omitting breakfast is an easy way to extend the overnight food deprivation. If precise biological regulation occurs, then omitting breakfast should cause an increase in food consumed later in the day. We have examined this question in two separate studies of the effects of eating or not eating breakfast [18]. Both studies provided basically the same results. Only the second study will be presented.

The participants were young and healthy students and staff members. For the three test days, the participants ate all their food prepared by the Cornell Metabolic Unit. The food was served from a buffet and could be eaten either in or outside the unit, but all uneaten food had to be returned in the original containers. All food was weighed before and after consumption. During the first week of testing, participants ate all three meals and were given mid-morning, mid-afternoon and evening snacks to eat when they were not in the unit, if they desired. On the second week, half of the participants were not given breakfast or mid-morning snacks while the other group consumed their normal breakfast. These conditions were reversed for the third week of testing. The participants were instructed to eat as much or as little as they desired at the meals and return to the buffet as many times as they wanted.

The results can be seen in Fig. 2. Consistent with the regulation model, participants consumed more food at lunch (approximately 150 kcal) when they skipped breakfast than when they ate it ($p=0.04$). However, the average intake at breakfast was about 600 kcal whereas the increase in the energy consumed at the lunch was only about 150 kcal. Because there was no difference in energy intake at any other meal through the day, withdrawing breakfast resulted in a daily deficit of about 450 kcal. In our first study omitting breakfast resulted in no increase in the intake at lunch, but the breakfast was only about 300 kcal. These data are consistent with other experimental [22,23] and cross-sectional data [24–26] that demonstrate that removing breakfast is not compensated sufficiently by an increase in subsequent energy intake resulting in deficit in a daily energy intake.

Another technique to impose a small food deprivation in humans is to withhold eating snacks between meals. In a design similar to that mentioned above, participants obtained their meals and mid-morning and mid-afternoon snack for two days separated by one week from a Metabolic Unit. Fig. 3 illustrates the results of this within-subject study when participants consumed or were denied the 250 kcal snacks. The snacks were consumed 2 h before the subsequent meal. The results were very clear: eating a snack at least 2 h prior to eating a meal had no effect on the amount consumed at the subsequent meals. Consequently, consuming snacks simply adds calories, not eating them causes a reduction in daily intake. These data are consistent with the work on preloading which shows that feeding people food at least an hour and a half prior to testing has no effect on the amount consumed at the following meal [27]. It appears, therefore, that imposing small energy deficits within a day does not evoke a precise regulatory mechanism to maintain energy balance.

It is possible that accurate energy regulation does not occur within a day, but acts over longer time intervals. The following study explored the effect of depriving humans of food on