

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

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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).

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 Figure 1, within this zone of body weight, however, those behaviors responsible for the determination of body weight, feeding behavior and physical activity, are not part of the regulatory system but rather are controlled to a large extent by external, environmental factors.

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.

Basic Tests of the Regulation of Food Intake

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 (19, 20).

Fortunately, most people in the industrialized world rarely endure more than 24 hours 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 their original containers. All food was weighed before and after consumption. During the first week of testing, participants ate all three meals and were given midmorning, 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 Figure 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 of omitting breakfast resulted no increase in the intake at lunch, but the breakfast was only about 300 kcal. These data are consistent with other experimental (21, 22) and cross-sectional data (23-25) that demonstrate that removing breakfast is not compensated sufficiently by an increase in subsequent in energy intake resulting in deficit in 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 those mentioned above, participants obtained their meals and mid-morning and mid-afternoon snack for two days separated by one week from a Metabolic Unit. Figure 3 illustrates the results of this within-subject study when participants consumed or were denied the 250 kcal snacks. The snacks were consumed two hours before the subsequent meal. The results were very clear: eating a snack at least two hours 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 daily intake. These data are consistent with the work on preloading which show 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 (26). 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 measured intake over longer periods of time. Participants ate all their meals

from our Metabolic Unit from Monday through Friday on three separate weeks. On Monday, the participants either (a) could eat as much or little as they wanted, (b) had their food intake limited to 1200 kcal, or (c) underwent a 24 hour fast. Food intake was measured at each meal beginning at breakfast on Tuesday until Friday night. Each participant served as their own control. Figure 4 shows the results. Depriving the participants of food on Monday had no significant effect on intake on Tuesday, or any other day of the week. As in the other two cases, depriving humans of food, for at least brief periods of time, does not produce accurate energetic compensation.

Energy surfeit

The overfeeding paradigm is also cited as a classic demonstration of the regulation of body weight in animals. After being overfed, animals reduce their intake and lose weight (27). We examined this paradigm in humans (28). Young adults ate ad libitum from foods prepared in our Metabolic Unit for two weeks, where all food intake was measured. For the next two weeks they were then given 133 percent of the amount of food they ate during the first two weeks. They were then allowed to eat as much or as little as they desired for the next three weeks of recovery. Figure 5 shows the effect of the overfeeding on energy intake.

Despite complaining bitterly about being unable to eat another bite of food, total energy intake of the participants returned to pre-overfeeding levels on the very first day following the termination of the overfeeding and remained at normal levels throughout the three week recovery period. Body weight decreased during recovery indicating that the mechanism responsible for the return of body weight was not accomplished through the control of energy intake, but rather through the control of energy expenditure.

Thus, humans appear not to be able to respond to either energy deficits or energy surfeits by demonstrating accurate adjustments of their food intake. If they don't regulate caloric intake and there is not change in energy expenditure then their body weight must change.

Changing Energy Density of Food

There is a considerable literature on the effects of changing energy density of food on human food intake (for a comprehensive review see (29)). Energy density is most commonly altered by changing the dietary composition or by using fat or sugar substitutes. Although the results are not unanimous, there is considerable consensus that humans do not respond to changes in the energy density by making accurate changes in their energy intake. A particularly striking example of this lack of accurate adjustment of energy intake to changes in energy density can be seen in Figure 6.

In this study (30), participants received a seven day rotation diet for 11 weeks consisting of foods containing either 35 or 25 percent calories as fat. The foods in the two diets were identical except for the fat content. Each participant served as their own control in a counterbalanced design. What is clear from this figure is that even after 11 weeks, participants receiving the less energy dense diet consumed fewer calories and lost more weight than when they consumed the higher energy dense diet. There was little evidence of caloric compensation. The same conclusion is true of studies of fat and sugar

substitutes. It is important to know that reducing the fat content from 35 to 25 percent of calories produced about a 200 kcal reduction in daily energy intake, not a trivial amount.

Portion Size

What has been described so far are studies of energetic manipulations that should have elicited a compensatory response if eating behavior were tightly coupled to biological regulatory mechanism involved in maintaining body weight constant. However, as presented above and in the vast literature on human feeding behavior, there is very little evidence eating behavior is tightly coupled to biological mechanisms involved in energy balance. But how “unregulated” is human eating behavior? The extent to which these energetic errors continue before biological regulation is initiated defines how much of the “epidemic of obesity” is due to changes in our environment.

Marian Nestle has been arguing for some time that one of causes of the increase in body weight of the American population is the introduction of larger size portions both in market (17) as well as at home (31). An increasing number of studies have begun to appear in the literature demonstrating the effect of serving portion on the amount humans consume. (32-38). Figure 7 illustrates the findings of one of these studies. Participants were asked to eat as much or as little as they wanted from a buffet style lunch. When they returned the following week they were served “restaurant” style with either 100 percent, 125 percent, or 150 percent of the amount they had previously taken from the buffet. As can be seen in figure 7, participants increased the amount they consumed in proportion to the amount they were served. The difference between what the 100 percent and the 150 percent condition amounted was about 200 kcal, again not a trivial number. Not only is this an impressive amount of energy consumed resulting from merely altering the amount of food placed in front of adults, but portion size appears to be the most important factor determining the amount consumed by children (37).

Social Facilitation

Another variable that appears to greatly affect the amount of food people consume is the number of people with whom they eat. The more people who eat together, they more food they consume (39-44). The effect is not small. Figure 8 is redrawn from a study by de Castro (45) and demonstrates that the difference between eating alone and eating in groups of 17 amounts to about 400 kcal.

It has been suggested that one of the reasons for the “epidemic of obesity” is the fact that we are eating outside the home more frequently where the number of people present is usually much larger than the number in the home. Recently we performed (unpublished) a study which demonstrated that not only is the number of people an important determinant of the amount of food people eat, but so is the amount of food those people around you eat. The study was performed in the Metabolic Unit and consisted of both participants and confederates. On the first day, participants and confederates ate their lunch from a buffet where they could eat as much or as little as they desired. In a counterbalanced order, for the second and third lunch, the confederates ate either more or less than they had at the initial lunch. Figure 9 shows the amount of food the participants consumed as a function of what the confederates ate. When the confederates ate less food than they ate previously, the intake of the participants did not change. However, when the confederates ate more than they ate previously, the

participants consumed significantly more food than they did on the initial condition. The energetic effect of eating with others who eat more food is about 40 kcal.

Variety

The number of different foods consumed has been found to correlate with the amount consumed (46, 47). Recently, we completed an experimental study (unpublished) of the effect of variety on energy consumption. We fed participants two different meals for lunch, buffet style, in our metabolic unit. The meal was either the typical American “tripartite” plate consisting of (a) a food that was most protein (chicken), (b) a food that was primarily starch (potato, rice), and (c) a vegetable. This was called the Composite condition. On other occasions, the participants received only the protein food and the vegetable (Low Carb condition) or only the starch and vegetable (Vegetarian condition). Participants were told to eat as much or as little as they desired. Figure 10 shows the results. Removing the carbohydrate food caused a significant reduction in energy consumed. Even more impressive was the fact that when the participants were given the Vegetarian meal, they consumed even fewer calories than on either the Low Carb or the Composite meal. The difference between intake of the Composite meal and the Vegetarian meal was about 200 Kcal.

Other Environmental Conditions that Affect Energy Intake and Body Weight.

Although the experimental conditions described above demonstrate that eating behavior of humans is not tightly coupled to a precise biological regulatory system, most of these studies use relatively short observation periods. There is another body of literature that also demonstrates imprecise energetic regulation, but measured over considerably longer periods of time. This literature lacks the precision of laboratory studies, but provides a reasonable estimate of magnitude of environmental effects on body weight.

Perhaps the clearest example of the plasticity of body weight is the well known observation that we are getting fatter. Figure 11 shows this increase in body weight in the US adjusted for age as analyzed by Zhang and Wang (48). Similar increases in body weight have been observed in American adults (14), American children (7), American minorities (12), Canada (10), Latin America (49), Asia (50), Europe (15) and Africa (51, 52). Such changes in weight cannot be caused by changes in genetics or even physiological regulatory systems, but rather by changes in the recent environment that cause small, yet persistent, changes culminating in the “globesity” problem we face today.

Analysis of the slope of this function represented in Figure 11 yields some indication of the degree of energetic error necessary to cause this increase in body weight. From about 1973 to about 2000, males gained an average of 7.3 kg and females 10.6 kg. If we use the commonly accepted value of 7700 kcal of energy intake is required to increase body weight by 1 kg, then the amount of excessive intake required to produce this degree of weight intake is on the order of 7.3 kcal/day for males and 5.7 kcal/day for females. This means that a change in the environment that can cause an average increase of 5 to 8 kcal a day, is sufficient to produce the “epidemic of obesity.” This calculation is important for two reasons. First, it demonstrates that the effects observed in the laboratory, such as the variety or the portion size effect, are more than sufficient to cause

the obesity epidemic. Secondly, if this small amount of daily energy is sufficient to cause the increase in body weight, then a reduction of about 5 to 8 kcal a day will be sufficient to reverse it.

Another example of the plasticity of body weight is the study of human migration. When people move to the US from places where the incidence of obesity is less than the United States, they tend to gain body weight (53-59). Some estimate of the magnitude of the effect can be seen in Figure 12 from a study by Goel et al. (54). Two aspects of this study are particularly important for this discussion. First, when immigrants come to this country, a greater percent are of “normal weight” (BMI > 18 and <25) than those who were born here. Second, the longer they remain in the United States, the lower the prevalence of normal weight individual is reduced to the level of native born Americans. A similar phenomenon can be seen using obesity as the measure. In this case, there is a lower prevalence of obesity (BMI > 30) among recent immigrants, but after remaining in this country for 15 years, their prevalence increases to the about the same amount level as native born Americans. Obviously changes in energy intake and expenditure are insufficient to prevent changes in body weight.

The literature on the effect of socioeconomics on body weight is another area where the control of food intake and expenditure appears insufficient to maintain body weight constant. In general, the lower the socioeconomic class, measured either as education level, income, or occupation, the greater the body weight as indicated by the prevalence of obesity (60-64). The etiology of relationship is quite complex with few suggestions as to why people of lower socioeconomic status, particularly women, have greater BMI's than those of higher status. But the fact that the relationship between socioeconomic class and body weight (obesity) is not static but appears to be changing over time (48) argues for environmental causes for the error in energy balance.

Marriage also demonstrates the powerful effect that environment has on body weight. It has been known for a long time that the body weight of one spouse correlates significantly with the body weight of the other spouse (65-70). Of course, this could mean that people of similar weights marry each other. However, a number of studies have demonstrated that weight difference between spouses diminishes with time (67, 71, 72) suggesting that it is the common environment (eating, exercise) that is responsible for the correlation. Moreover, the gain in weight of one spouse is significantly correlated with the weight gain of the other spouse (73). Finally, getting married results in a significant increase in body weight whereas getting a divorce results in a loss in weight (73, 74). A common environment shared by the two people plays a significant role in determining those behaviors that affect energy balance.

One final example of the plasticity of body weight can be seen in the rather powerful effect of attending college on weight gain. We have observed that freshmen who enter college, at least at Cornell, gain approximately 2 kg over their first 12 weeks of college (75). The two best predictors of the weight gain were eating snacks, particularly late night snacks, and eating in the “all-you-can-eat” dining halls. A calculation of the degree of energetic error that is responsible for this freshman weight gain reveals that an average consumption of an additional 9 kcal daily is sufficient to cause this weight gain, a value close to the figure estimated to cause an increase in body weight in the US population as mentioned above.

Weight Monitoring as a means of Controlling Weight Gain

The conceptual framework for the arguments and data presented is that a zone of body weights exist (Settling Zone) within which eating behavior (and energy expenditure) is controlled more by external environmental stimuli than internal physiological mechanisms involved in regulating body weight. Fluctuations in daily body weight, therefore, represent the degree to which people respond to those environmental stimuli that affect energy balance.

Theoretically, if people monitored their daily weight, a small increase in weight would provide warning that their energy balance was positive. They then could make small changes in their subsequent behaviors (eating, exercise) thereby correcting the energetic error. Unfortunately, daily weight fluctuations represent not only increases and decreases in real tissue mass (energy balance), but also reflect changes in body water content, gastrointestinal contents, and body glycogen stores. Fortunately many statistical methods exist that enable us “to see through the noise” of daily weight fluctuation in order to obtain a better indication of changes in tissue mass. One such technique that we have been exploring to use weight as an indicator of energetic error is an algorithm based on linear regression called the Tissue Monitoring System (TMS).

We have tested the TMS in two independent studies as a method to prevent the freshman weight gain during their first semester at college (76). In both studies, freshman women were provided with bathroom scales and were instructed to email their weight to us every day. After collecting seven days of data, we emailed back to the students the slope of the linear regression of the seven points against time. We provided the students with fundamental instruction in energy balance and how to interpret the slope. The controls were simply weighed at the beginning and the end of the semester. The results can be seen in Figure 13. In both studies the group that used the TMS gain no weight (not significantly different from 0), whereas the weight gain of the controls was between 2 and 4 kg ($p < 0.001$). When we asked the experimental group what they did to prevent the weight gain, some students replied that they skipped snacks, others said they skipped dessert, still others skipped a meal. As indicated above, such techniques are affective in reducing daily energy intake. Other students increased their exercise to maintain energy balance, another activity that does not result in energetic compensation and thus will cause a negative energy balance (77-81).

These TMS studies support our contention that when people are provided with information as to their state of energy balance, they can make the necessary changes to maintain their weight constant. The kinds of changes people make depend upon what activities fit into their life style. What appears clear, however, is that people know what to do to achieve energy balance, but what they need is information about the status of their energy balance.

Non-Regulation of Food Intake and Hope for Reversing Obesity

To control our weight in an environment that constantly pressures use to consume more calories than we need requires some technological feedback. Daily monitoring of body weight may be necessary to allow us to control our eating behavior energy expenditure similar to the way that people have learned to control their blood pressure though biofeedback or their glucose levels by monitoring blood glucose concentrations.

With daily monitoring of body weight, it is easier to correct positive energetic errors than wait until we have gained a noticeable amount of weight and undertake a more traditional, though ineffective means of losing weight. The reason it is easier is because the energetic errors are smaller and would require only small changes in behavior or lifestyle. We can acquire control over our weight because food intake is not regulated enabling us to use small corrective measures such as reducing portion size or skipping a meal to correct the small energetic error in a short period of time. However, constant surveillance of our weight and some behavioral restraint may be necessary to counter the pervasive stimuli in our environment that cause us to eat more than we need to maintain energy balance and to help us stop, or even reverse, the global trend towards increasing obesity.

Figure Captions

- Figure 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.
- Figure 2. The effect of eating or omitting breakfast on subsequent intake. The only statistically significant effect of eating breakfast was at lunch. (unpublished data)
- Figure 3. The effect of eating or omitting midmorning and midafternoon snacks on the consumption of meals. The consumption of snacks and meals was significantly greater than the consumption of meals without snacks ($p < 0.02$). (unpublished data)
- Figure 4. The effect of either eating ad libitum, eating 1200 kcals of food, or total fasting on Monday on the total daily energy consumed on Tuesday through Friday. Intake was not affected by how much food was consumed on Monday. (unpublished data)
- Figure 5. The effect of overfeeding on daily energy intake. Energy intake during overfeeding was significantly greater than baseline ($p < 0.001$). However, intake during recovery was not statistically different than baseline. (adapted from (28))
- Figure 6. Daily energy intake of participants spontaneously consuming foods that contain either 25 percent calories as fat, or 35 percent. The two functions are statistically different ($p < 0.01$). (adapted from (30))
- Figure 7. Amount consumed when participants were served either 100, 125 or 150 percent of the amount they ate when they selected the same food themselves. Each portion size was significantly different from each other ($p < 0.01$). (adapted from (38)).
- Figure 8. Amount consumed at a meal by participants as a function of the number of people they ate with. (adapted from (45))
- Figure 9. Amount of food consumed by participants as a function of how much "confederates" ate. When "confederates" ate more (High Intake) than in control condition, participants also consumed more food ($p < 0.05$). (unpublished data)
- Figure 10. Amount consumed when either the high carbohydrate food or the high protein food was eliminated from a meal consisting of a carbohydrate food, a protein food, and a vegetable. All groups were statistically different from each other ($p < 0.02$). (unpublished data)
- Figure 11. Average body weight for males and females as estimated from the four HANES surveys. (adapted from (48)).
- Figure 12. Percent US immigrants of normal weight (BMI $18.5 \leq < 25$) and obese (BMI > 30) as a function of the length of time they have lived in US. (adapted from Goel et al. (54))
- Figure 13. Change in body weight over first 12 weeks of attending college of freshman who used TMS as a means of controlling their weight and untreated controls in two independent studies. (Adapted from (76)).

Figure 1

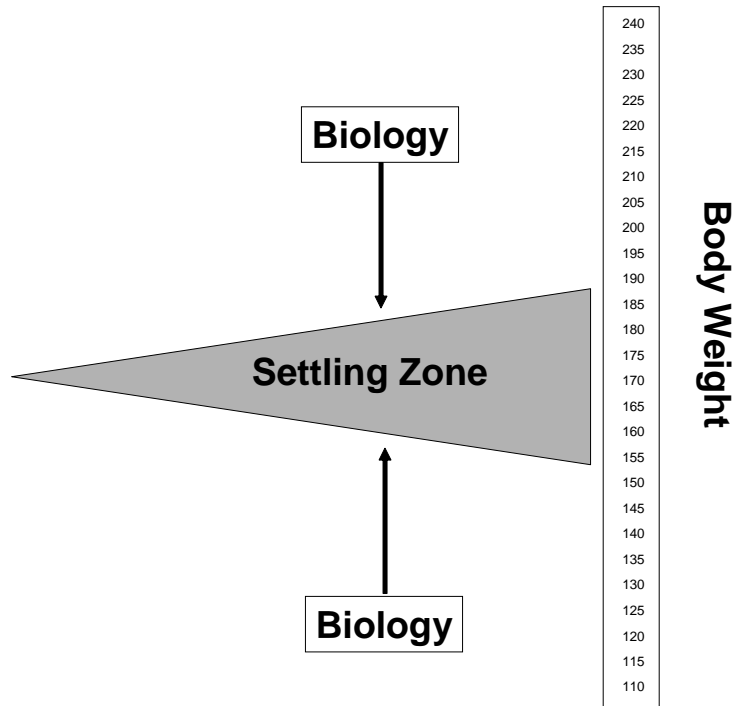


Figure 2

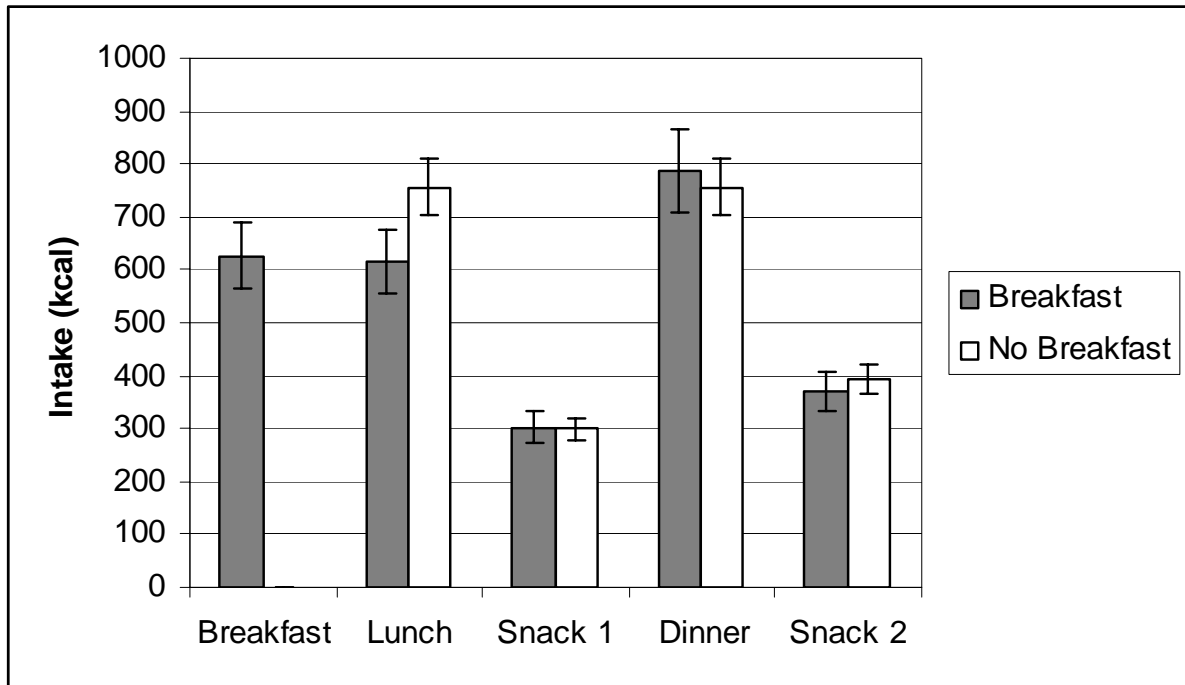


Figure 3

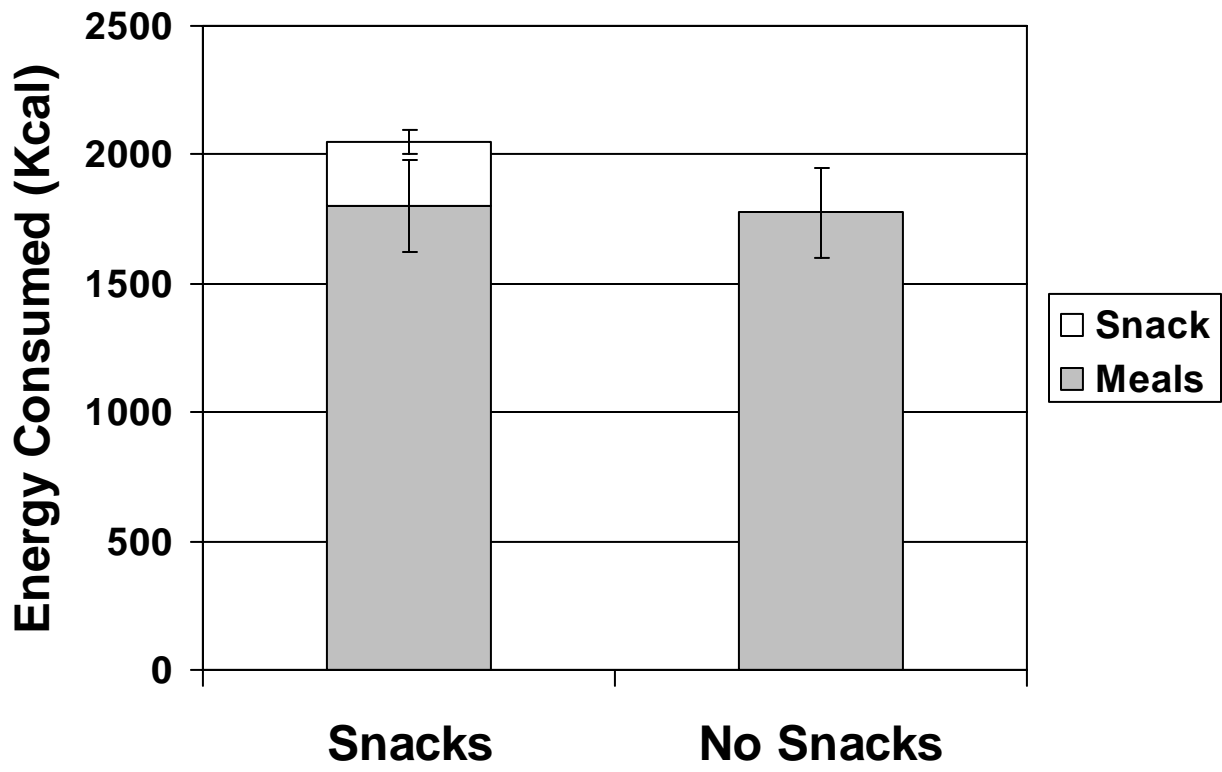


Figure 4

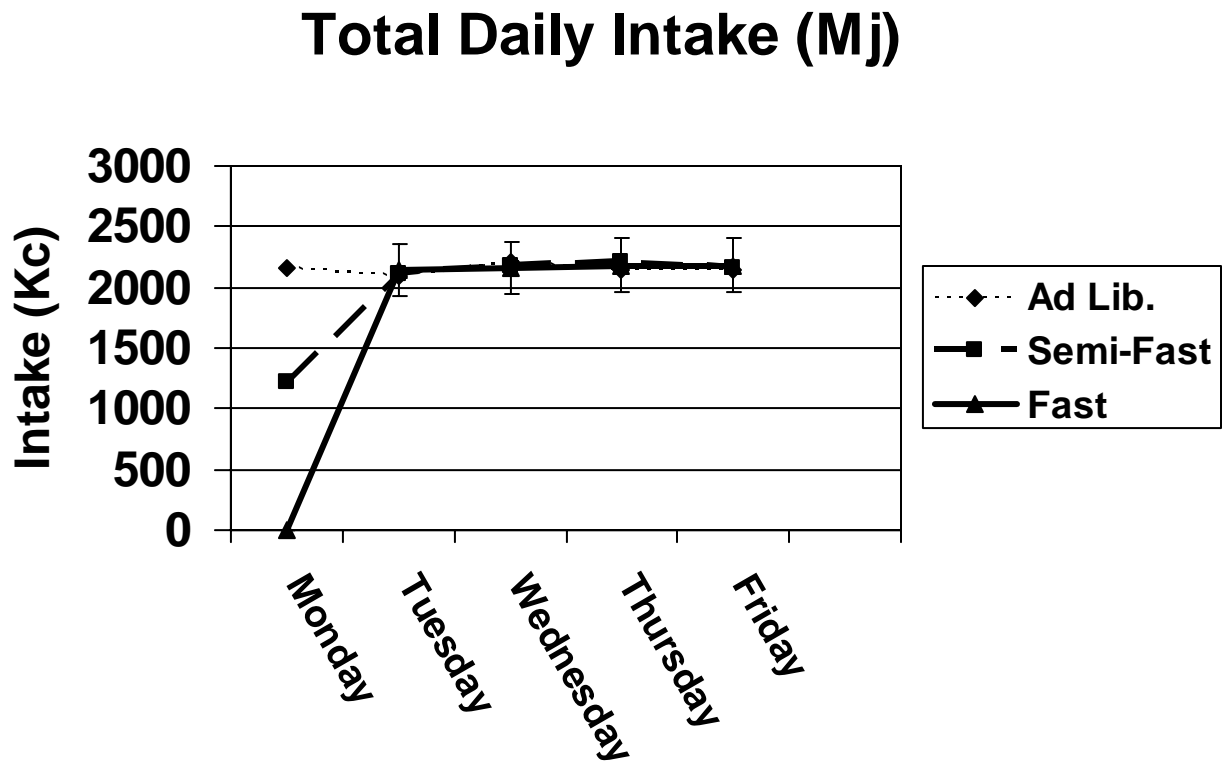


Figure 5

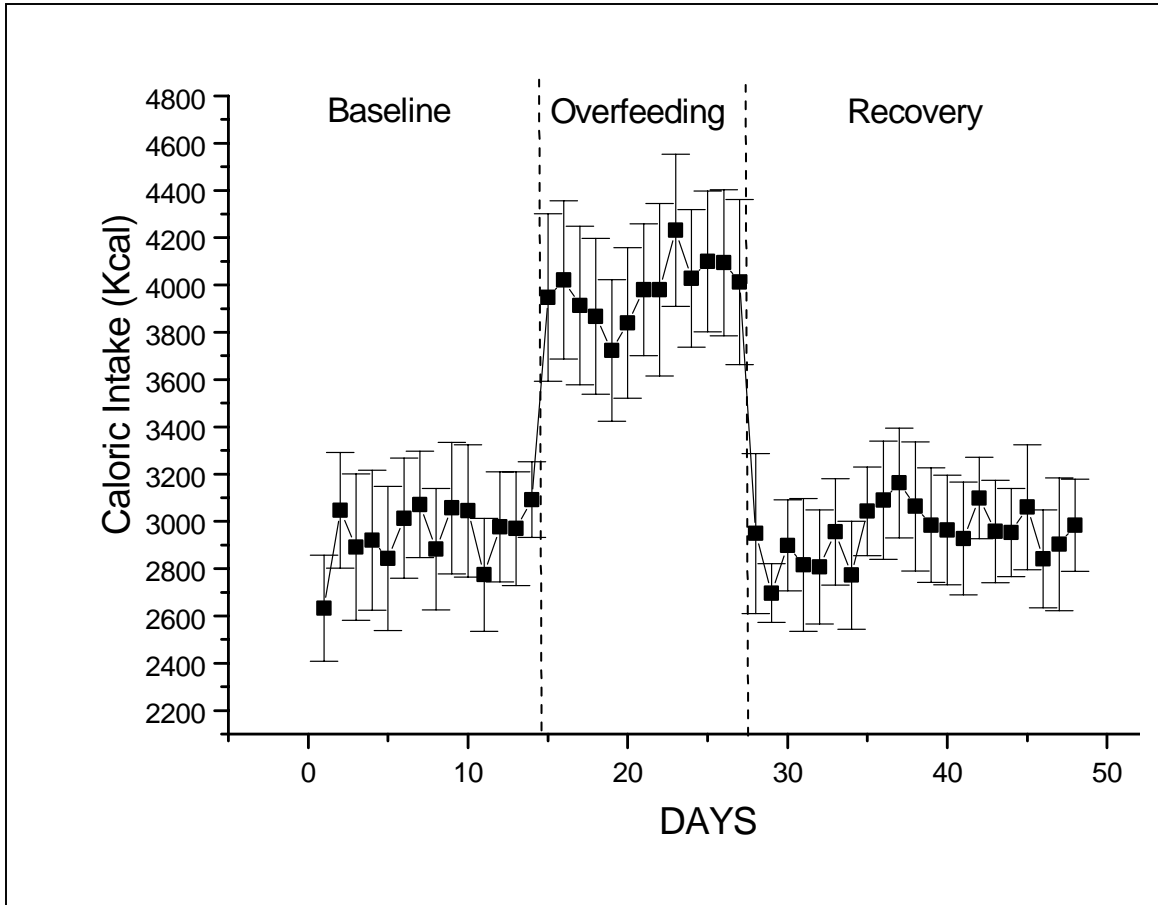


Figure 6

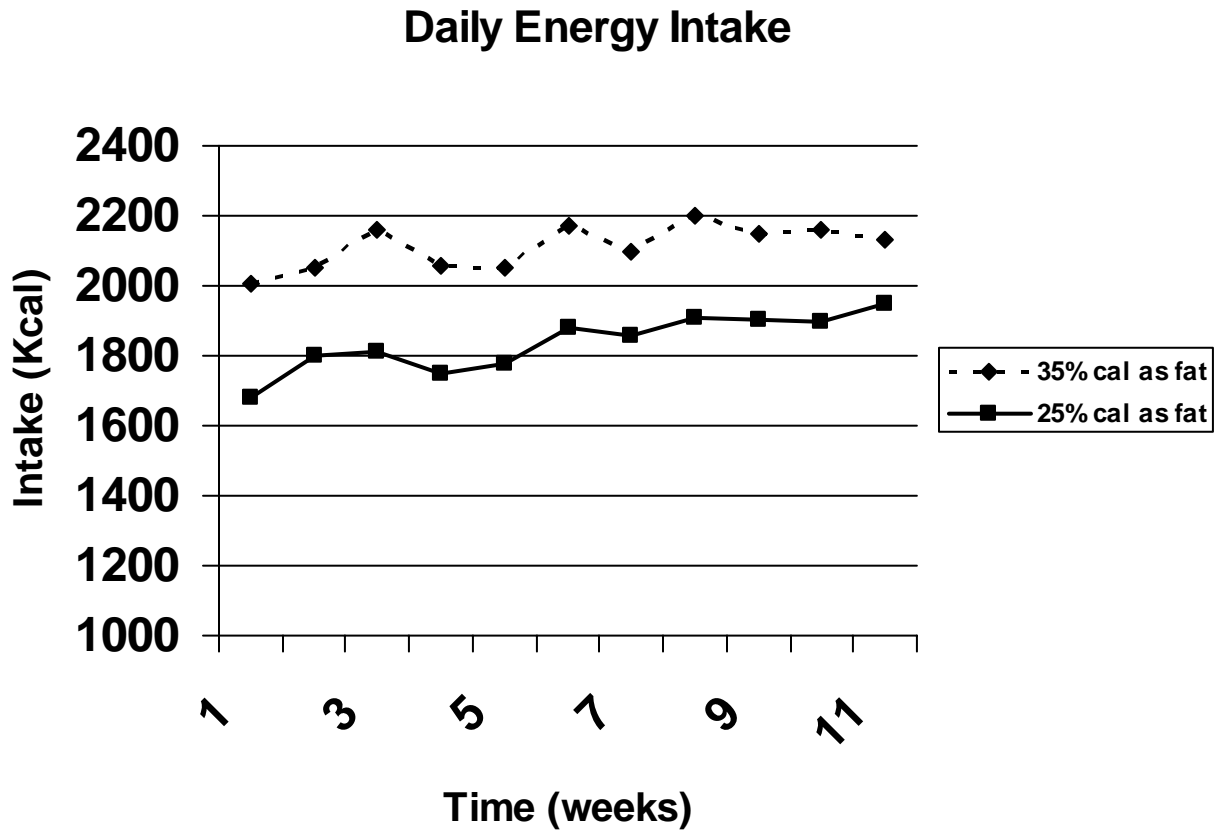


Figure 7

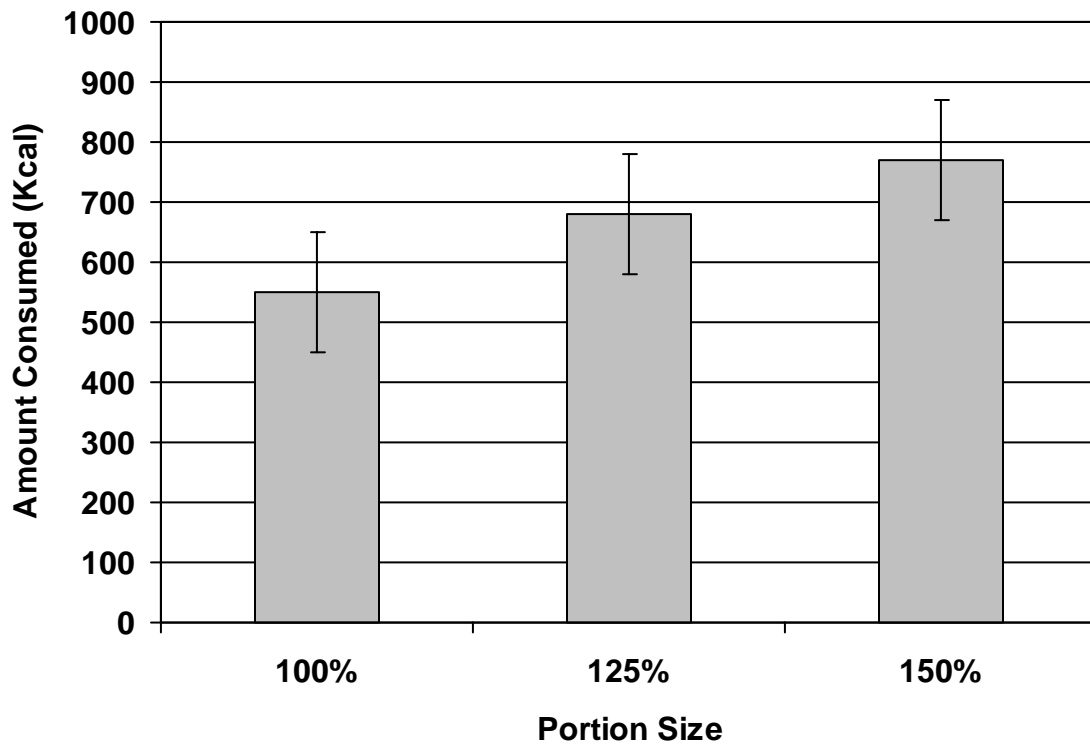


Figure 8

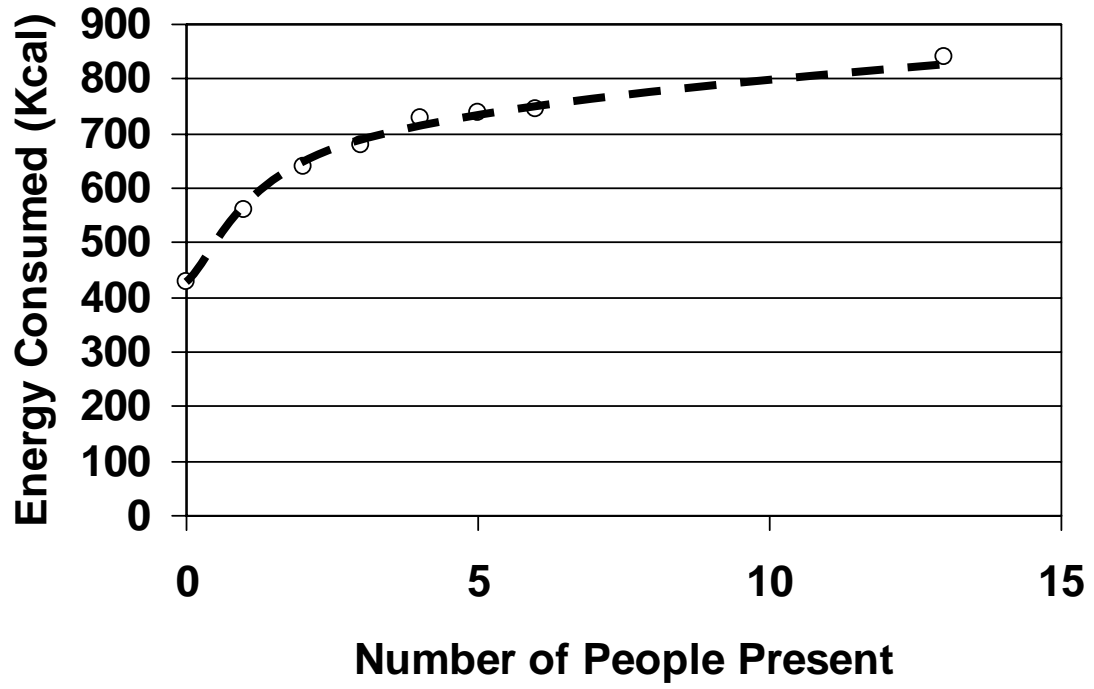


Figure 9

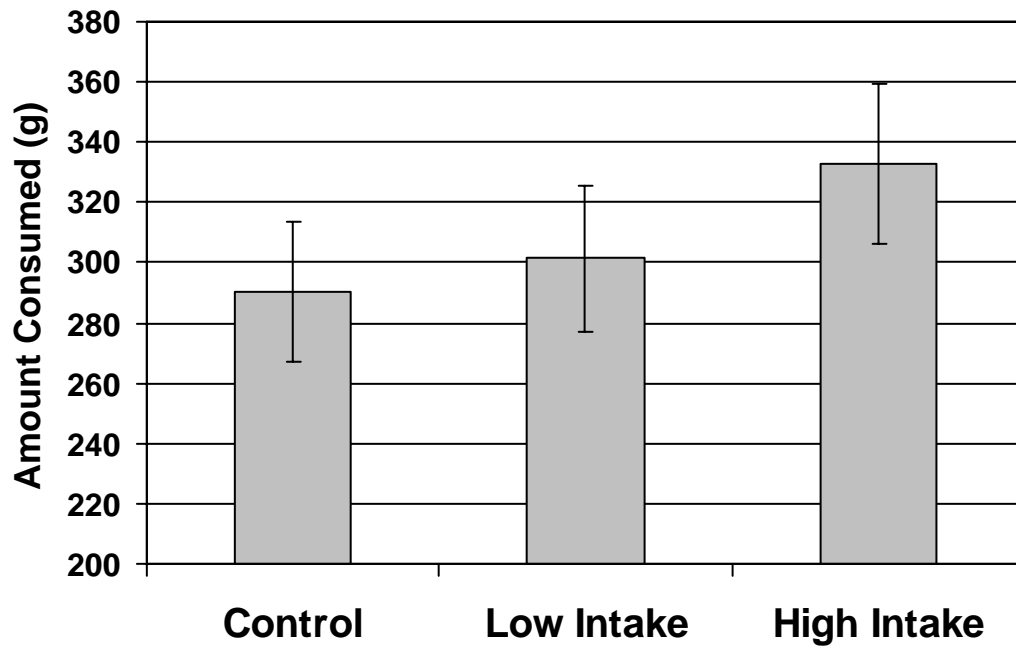


Figure 10

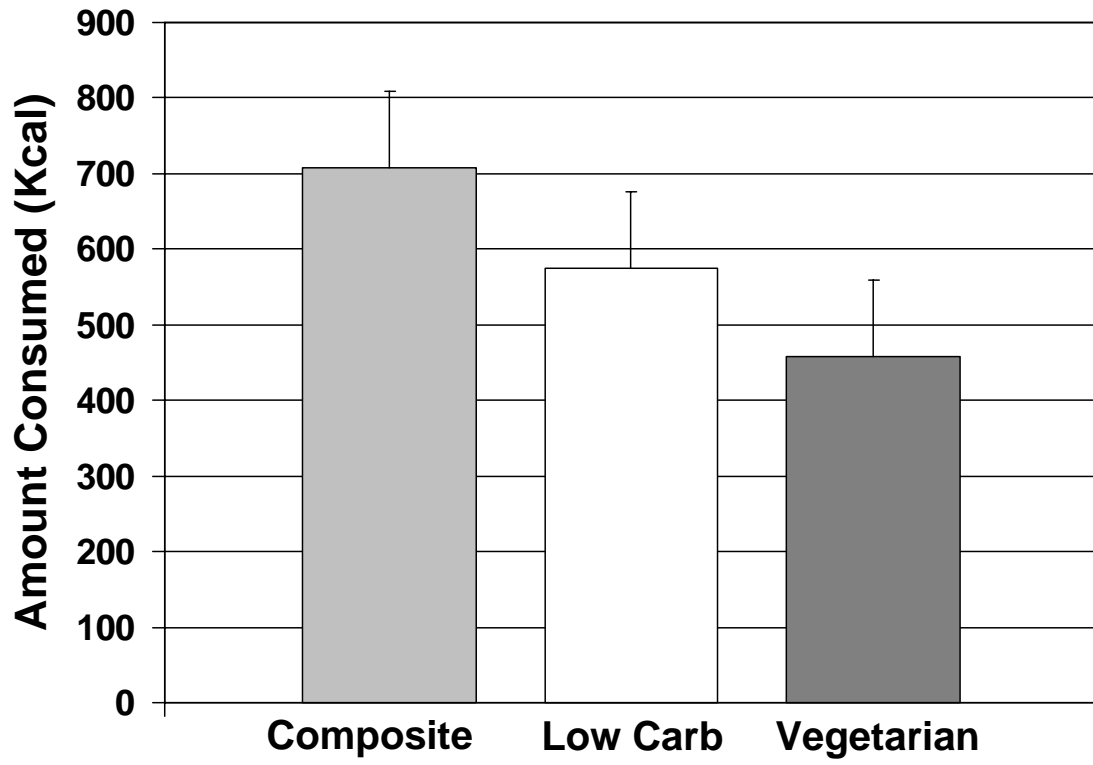


Figure 11

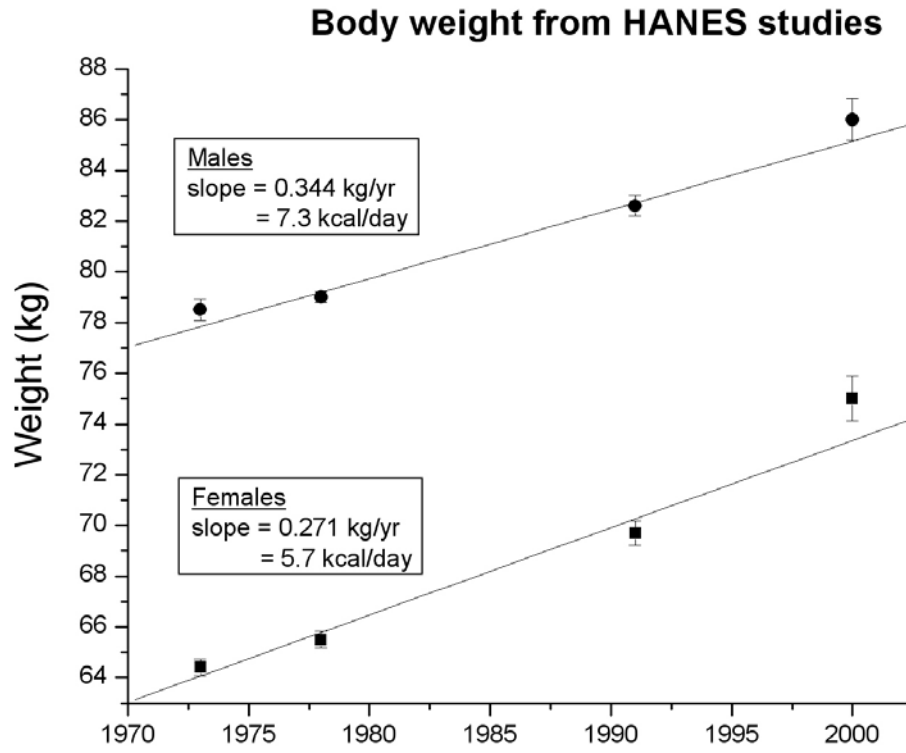


Figure 12

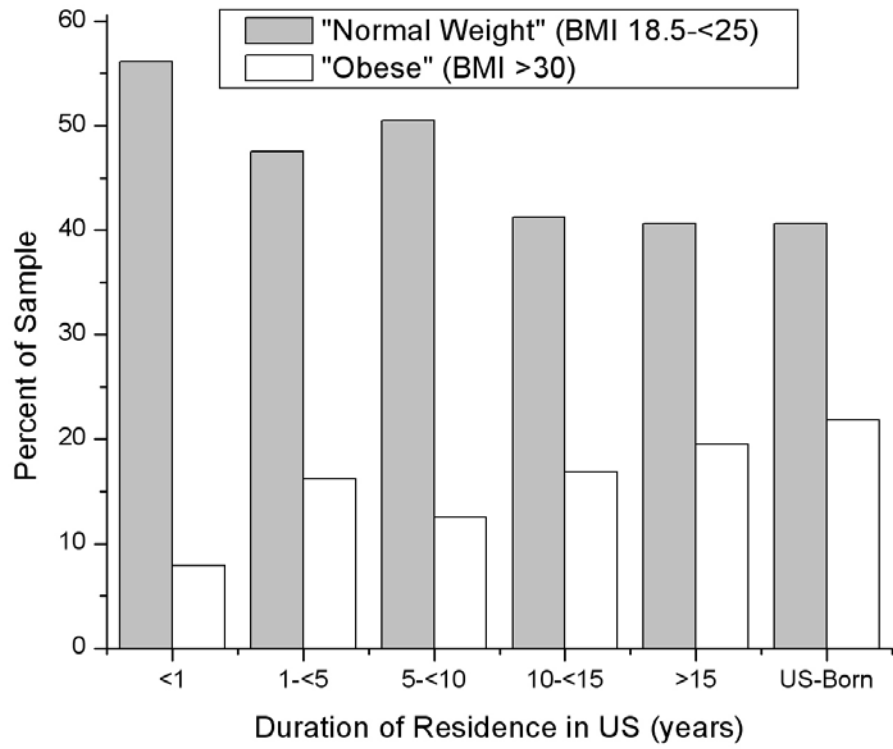
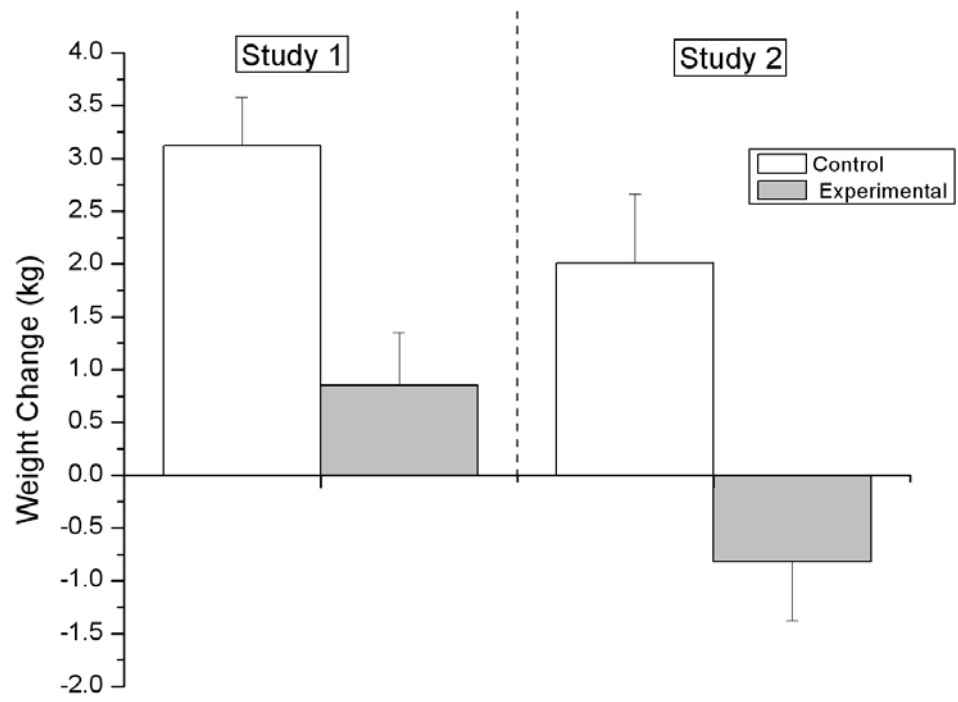


Figure 13



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