What Makes Food Fattening? A Pavlovian Theory of Weight Control

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Abstract

The theory described in this article assumes that the body-fat set point – how much body fat the brain tries to maintain – is controlled by flavor-calorie associations. Calorie-associated flavors raise the set point – the stronger the association, the greater the increase. In the absence of calorie-associated flavors, the set point declines. Given some plausible assumptions, the mechanism regulates body fat according to the availability of food, increasing body fat when food is abundant, decreasing body fat when food is scarce. The theory explains a wide range of human and animal data, including effects and correlations involving pre-exposure, pureeing, moistening food, bland food, glycemic index, supermarket food, junk food, fasting, intragastric feeding, and income. It also helped find a new way to lose weight.

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If two foods, A and B, have the same number of calories, will eating A have the same effect on your weight as eating B? A government report says yes: “A calorie is a calorie is a calorie” (National Heart, Lung, and Blood Institute, 1998, p. 150) – meaning that the source of the calories does not matter. But many disagree. A food with a low glycemic index may “decrease hunger” (Ludwig, 2000, p. 212S) more than a equicaloric food with a high glycemic index, reducing consumption of other foods. Popular books about weight control often stress the fat or carbohydrate content of food (e.g., Atkins, 1992; Sears, 1995; Steward, Bethea, Andrews & Balart, 1995).

This article describes a theory of weight control that emphasizes a food’s effect on regulation – on the body fat *set point*, the amount of body fat the weight-control system tries to maintain. According to this theory, two equicaloric foods may affect the set point quite differently. But it is not fat or carbohydrate content that matters; it is how strongly the flavor of the food is associated with calories, which depends on many factors, including experience. The theory proposes that a food whose flavor is strongly associated with calories raises the set point much more (and thus is much more fattening) than a food whose flavor is weakly associated with calories.

The idea that body weight is regulated by a system with a set point (reviewed below) is old and well-known. That the set point depends on diet is also an old idea. It was first proposed by Corbit and Stellar (1964), who found that rats fed ad libitum stabilized at a higher weight when given what the researchers considered more palatable food. Other studies had similar results (Keesey & Boyle, 1973; Peck, 1978). Sclafani and Springer (1976) found that rats

regulated their weight at a higher level when they were allowed to eat supermarket foods (e.g., salami, cookies) in addition to lab chow. Supermarket food caused a 260% increase in weight gain.

Michel Cabanac and his colleagues brilliantly extended this line of research. Cabanac, Duclaux, and Spector (1971), using human subjects, found that a glucose solution ingested via a nasogastric tube reduced the pleasantness ratings of sucrose solutions tasted one hour later, a change they called *allesthesia*. Cabanac et al. (1971) also tested persons who had lost 3 kg over the previous two weeks by eating less of their usual diet. For these subjects, the nasogastric glucose did not cause allesthesia. Cabanac et al. explained the results by assuming that allesthesia is part of the body-fat regulatory system: When food is no longer pleasant, eating stops. When body fat is at or above its set point, allesthesia occurs sooner in a meal than when body fat is below its set point. The body fat levels of the subjects who had just lost weight, unlike the earlier subjects, were below set point, so allesthesia was delayed.

A later study (Cabanac & Rabe, 1976) measured the effect of another way of losing weight: drinking a nutritionally-complete liquid food. For two weeks, subjects consumed only the liquid food, as much as desired. They lost about 3 kg. With these subjects, the nasogastric glucose produced as much allesthesia as it did with non-dieting persons. Cabanac and Rabe proposed that drinking the liquid diet, unlike eating less of the same food, lowered the set point so that body-fat level and set point remained close. They noted that persons on the liquid diet "were always in good spirits" (p. 678) while persons who lost weight by eating less "had to continually fight off their hunger and would spend the night dreaming of food" (p. 678). Cabanac and Rabe suggested that the liquid diet lowered the set point because of its "monotonous and

insipid taste" (p. 676).

Cabanac and his colleagues asked what controlled the set point when many researchers did not accept that a set point existed, much less that it could change (e.g., Bolles, 1990). Eventually these ideas – a set point exists, it can change, and diet affects it – became more popular. Mrosovsky (1990) described twenty-odd examples of change in the levels of regulated variables, making it clear that set points could change. Keesey and Hirvonen (1997) argued that human obesity is due to an elevated set point and that "obesity research should [identify] the factors responsible for setting the level at which such individuals regulate body weight" (p. 1880S). They believed that diet was one of those factors.

This article takes this line of thought one step further. It proposes a general theory of how diet controls the set point.

BACKGROUND

Set Point Regulation of Body Fat

Kennedy (1950) proposed that body fat is regulated by a system with a set point. When the caloric density of their food was cut in half, after a few days rats doubled the volume of food that they ate. Other labs found similar results. In spite of changes that had a big effect on eating behavior (e.g., meal size), rats kept their body weight nearly constant (Collier, Hirsch & Hamlin, 1972; Levitsky, 1974). After rats were food-deprived for a few weeks, they ate more than usual until their weight reached what it would have been without the deprivation (e.g., Mitchel & Keesey, 1977). Participants in the Minnesota Semi-Starvation Study lost a lot of weight due to limited rations and became very hungry. After the restriction ended, they remained more hungry than usual and ate more than usual until they reached their initial weight (Keys et al., 1950).

One objection to the idea of a regulatory system with a set point was that a stable weight can be achieved in other ways (Bolles, 1990; Garrow & Stalley, 1975; Harris, 1990; Wirtshafter & Davis, 1977). Any negative feedback loop will do, they argued. Bolles (1990) made this point vividly: When a cat gets too fat, it can no longer catch rats, so it loses weight. That a fat cat is slow is not due to a regulatory system. Weight constancy might be due to one or more negative feedback loops that exist for other reasons.

Is weight constancy accidental? Set point regulation of body fat by the brain requires that the brain "know" the amount of body fat, just as set point regulation of room temperature (by a thermostat) requires that the thermostat "know" the room temperature. The set point idea implies, in other words, that something reaching the brain must vary with the amount of body fat. The Bolles et al. view – that weight constancy is an accident – does not require such a signal. For this reason, the discovery of leptin, a hormone whose blood concentration varies with amount of body fat (Woods, Schwartz, Baskin & Seeley, 2000), strongly favored the set-point view. Leptin has just the properties needed for regulation.

When body fat is below set point, research suggests that two changes take place. First, hunger increases. Humans report more hunger than usual (Keys et al., 1950); rats eat more often (Levitsky & Collier, 1968). Second, allesthesia decreases. Food stays pleasant longer. In humans, this is revealed by pleasantness ratings and meal length (Cabanac, Duclaux & Spector, 1971; Cabanac & Frankham, 2002); in rats, by meal length (Levitsky & Collier, 1968). Both changes tend to increase body fat.

Pavlovian Conditioning

Pavlov found that following the sound of a bell with food caused the sound of the bell to

produce salivation. Following a neutral event – the *conditioned stimulus* (CS) – with an important event – the *unconditioned stimulus* (US) – caused the first event (the CS) to control behavior (reviewed by Domjan, 1996). The new effect of the CS (e.g., salivation) is the *conditioned response* (CR).

Flavor-calorie learning, also called *flavor preference learning*, is an example of Pavlovian conditioning. Experience of a flavor (CS) followed by digestion of a source of energy (US), such as fat, carbohydrate, or protein, increases preference for the flavor (CR). Pairing a flavor with calories makes the flavor taste better, in other words. First demonstrated by Holman (1969), it has been studied extensively by Sclafani and others (Capaldi, 1996; Mehiel, 1991; Sclafani, 1991). The work of Bolles, Hayward, and Crandall (1981) provides an example. One flavor (CS+) was mixed with flour, another flavor (CS-) with chalk. Rats were allowed to eat both mixtures for three or four days, then tested with the two flavors added to identical mixtures of half flour, half chalk. The rats ate more of the CS+ mixture than the CS- mixture. Flavor-calorie learning has been demonstrated several times with human subjects (Birch, McPhee, Steinberg & Sullivan, 1990; Booth, Mather & Fuller, 1982; Gibson, Wainwright & Booth, 1995; Johnson, McPhee & Birch, 1991).

THE THEORY

The theory consists of two assumptions:

1. Calorie-associated flavors raise the set point. The stronger the association, the greater the increase.

2. Meanwhile the set point steadily decreases. The higher it is, the faster the decrease.

The overall change is the sum of these two changes – increases caused by food added to a steady

decline. Assuming the flavor-generated increases are relatively fast, the set-point-versus-time function resembles a sawtooth wave: up fast, down slow, up fast, down slow, and so on. Although the set point is never constant, the level around which it oscillates may be constant. The level of oscillation will be constant when

increase generated by one day's food = steady decrease during one day.

The increase generated by food depends on what is eaten (Assumption 1), that is,

rate of increase = f(diet).

The steady decrease depends on the value of the set point (Assumption 2), that is,

rate of decrease = f(set point).

The set point shifts up or down until the rate of decrease equals the rate of increase. As it moves up, the rate of decrease increases; as it moves down, the rate of decrease goes down. Iin this way, diet controls the set point. Figure 1 shows what happens after a increase in how much food raises the set point. The set point gradually rises, increasing the steady decrease, until equality is restored.

SUPPORTING EVIDENCE

Most Persuasive

Similarity To Other Storage Systems

Many commodities, such as butter, feed grain, soybeans, wheat, and frozen chickens, are stored by distributors. The amount stored usually depends on the current price: The lower the price, the more stored (Williams & Wright, 1991). This dependence is profitable if the cost of storage is low compared to the variation in price over time (Williams & Wright, 1991). Most of us act the same way: When something we often buy is on sale and is easy to store (paper towels,

toothpaste, canned goods), we stock up.

Fat stores energy. The same principle applied to body fat implies that amount of fat the brain tries to store (the set point) should depend on the price of food: the lower the price, the higher the set point. We cannot do a calculation to show that this would have been beneficial when body-fat storage systems were shaped by evolution but it is certainly plausible. Fat has benefits (insulation, protection, and cushioning) as well as costs (metabolic, the energy needed to carry it) so the net storage cost of small and moderate amounts of fat may have been very low. There is evidence that exposure to cold increases body fat (Endo, Omoe & Ishikawa, 1993; Ruff, 2002), implying that fat provides significant insulation. The cost of food obviously varied a great deal over time due to seasonal differences (less food in winter). Neel (1962) explained high rates of obesity and Type 2 diabetes among groups such as the Pima Indians of Arizona by assuming that their genes were shaped by long periods of scarcity mixed with relatively short periods of plenty. Under these conditions, Neel assumed, it was beneficial to increase fat stores quickly during the periods of plenty.

The proposed mechanism tends to increase body fat when food is cheap and reduce body fat when food is expensive. It does so given two plausible assumptions. *First*, when food becomes more abundant (cheaper), the number of available foods increases. That is how ecosystems work: Prosperity and diversity go together. During famines, the number of available foods goes down (Aykroyd, 1975). The term *famine food* refers to a food that is usually available but is eaten only when other foods disappear. *Second*, the strength of flavor-calorie associations guides choice: When choosing between foods, we tend to choose the one with the strongest flavor-calorie association. Rat experiments support this assumption (Sclafani, 1991). When the

number of available foods increases, the strength of the strongest available flavor-calorie association will also increase. This is a basic principle of sampling: The larger a group of people, the taller will be the tallest person in the group. Thus as food becomes more abundant, the average strength of the flavor-calorie associations of what is eaten will increase. This will raise the set point, according to the theory.

This argument also supports one aspect of the theory: the use of flavor-calorie associations to regulate storage. In a properly-run fat-storage system, the amount of fat should depend on the availability of food and not on the availability of other things (e.g., plants not eaten). Animals that learn what to eat must react to the availability of what they have learned to eat. Because flavor-calorie learning probably plays a large role in determining what we eat (Reisbick, 1973; Sclafani, 1995), it should be involved in control of the set point.

Other theories. This principle (amount of body fat should vary with the price of food) can be used to judge other weight-control ideas. Some pass the test – regulate fat appropriately – and some do not. Cabanac and Rabe's (1976) idea that a "monotonous" (p. 676) diet lowers the set point passes this test, because, as mentioned earlier, when food became scarcer it probably became less diverse. A theory that assumes a constant set point (e.g., Hervey, 1969) fails this test. The idea that a high-fat diet raises the set point may not pass this test because the percentage of calories from fat may not have correlated with overall abundance of food. Game animals (available in winter) have a larger percentage of calories from fat than most food plants (not available in winter). For fruit-eating animals such as humans, greater availability of fruit would have reduced the percentage of calories from fat.

Ramirez (1990a)

Ramirez (1990a) found that adding saccharin to a liquid diet caused rats to gain weight. The effect had some puzzling characteristics:

1. *Prior experience with saccharin eliminated it*. The effect did not appear when rats drank saccharin-flavored water for several days before being given the liquid diet. Ramirez observed this twice.

2. *Prior experience with the liquid diet eliminated it*. When rats ate the liquid diet for 1-3 weeks before the addition of saccharin, the effect did not occur. Ramirez observed this twice.

3. *It took about one week to reach full strength*. The effect was weaker during the first week of saccharin than during later weeks.

Explanation. The flavor of saccharin (CS) became associated with the calories in the liquid diet (US), causing the flavor to raise the set point. Preexposure to the saccharin or the liquid diet reduced the effect because they reduced the strength of this association. Such pre-exposure effects are a well-established feature of Pavlovian conditioning. Many experiments have found that exposure to the CS without the US reduced the effect of later CS-US pairings (e.g., Lubow, 1973). Likewise, many experiments have found that exposure to the US without the CS has the same effect (e.g., Randich & LoLordo, 1979). Both effects have been found with flavor aversion learning, where pairing a flavor with illness or nausea creates an aversion to the flavor (De Brugada, Hall & Symonds, 2004; Dibattista, Hollis-Walker & Hague, 2003; Salvy, Pierce, Heth & Russell, 2002) and with flavor acceptance learning, where pairing a flavor with calories increases ingestion of foods with that flavor (Ramirez, 1996). Ramirez (1996) observed flavor acceptance learning using saccharin flavor.

The effect grew with time because the association took time to learn. Flavor-calorie associations take several days to reach full strength (e.g., Bolles, Hayward & Crandall, 1981). Effects that change with time are discussed in detail below ("Delayed Effects").

Other explanations. Ramirez (1990a) concluded that learning was involved. He considered two other explanations (palatability changes, and osmotic-pressure changes) but decided both were unlikely.

The preexposure effects make implausible a non-associative explanation of saccharine's effect on weight (Ramirez, 1996). When preexposure to Event X reduces later learning with Event X, it is hard to avoid concluding that the later learning required more than Event X. Several types of learning, such as habituation, decrease of neophobia, and learning how to eat a new food, involve only one event. To explain preexposure results such as these in terms of single-event (non-associative) learning, one has to assume that during preexposure learning went backwards – for instance, each experience of Event X *reduced* habituation. There is no empirical support for such an assumption.

Labouré, Saux & Nicolaidis (2001)

Labouré, Saux & Nicolaidis (2001) measured the effect of the texture of food on the weight of rats. "A [nutritionally] complete diet was prepared with cooked pieces of meat, beans, cream, starch, and water and presented to the rats in two different textures: a blended puree and a rough mixture that required a lot of chewing" (p. R780). They tried to make the two foods equal in every way except texture: "Great care was taken to avoid modifying caloric density and nutrient composition" (p. R780-781).

During a short experiment, with eight one-hour sessions spread over eight days, the

mixture was eaten more than the puree. When the two foods were presented separately, the rats ate more of the mixture. When the two foods were presented together, allowing a choice between them, the rats ate three times as much mixture as puree.

Based on these results, one would expect the mixture to be, if anything, more fattening than the puree. In fact, the opposite happened. During a longer experiment, the mixture caused *less* weight gain than the puree. There were two groups of rats, one fed only the mixture, the other only the puree. Their food intake was equal at first but after three weeks the puree rats were eating more than the mixture rats. By the end of the experiment (six weeks), the puree rats had gained one-quarter more weight than the mixture rats.

Another six-week experiment, with different rats, gave continuous exposure to both foods. Initially the rats ate more mixture than puree but by the fourth week their preference reversed.

Explanation. Pavlovian conditioning, including flavor-calorie learning, requires that the CS and the US meet in the brain – that is, the brain changes caused by CS and the brain changes caused by the US must overlap. It follows that the strength of the CR (not just its existence) depends on how much (not just whether) the two signals overlap (Roberts, 2004). Figure 2 shows this idea graphically. The eventual strength of the CR is proportional to the area of overlap. This idea correctly predicts that reducing CS intensity, reducing US intensity, and increasing the time between CS offset and US onset all reduce asymptotic CR strength (Mackintosh, 1974).

To this general idea is added an assumption about this experiment: Pureeing the mixture speeded up digestion. Pureeing increased the food's surface area. For the same reason that a sheet dissolves much faster than a sphere, the increase in surface area increased the rate of

dissolution (separation of food molecules from each other) and digestion via enzymes (chemical reactions). This assumption is supported by the fact that the puree caused an earlier peak in plasma insulin than the mixture. The researchers themselves wrote that "blending the ingredients may make them more readily digestible" (Labouré, Saux & Nicolaidis, 2001, p. R787).

Faster digestion led to faster calorie detection – a US signal with an earlier peak. This increased the asymptotic strength of the flavor-calorie association because it increased CS-US overlap (Figure 3). The preference reversal, with the right time course to be due to flavor-calorie learning, supports the assumption that the puree came to have a stronger flavor-calorie association than the mixture. Labouré, Saux & Nicolaidis (2001, p. R787) concluded that the preference reversal was due to associative learning ("a Garcia-type phenomenon").

Other explanations. Labouré, Saux & Nicolaidis (2001) do not explain the weight difference. The results contradict Cabanac and Rabe's (1976, p. 676) idea that a "monotonous" taste will lower the set point because the flavor of the puree was more constant from one bite to the next than the taste of the mixture.

A New Way to Lose Weight

The theory helped find a powerful new way of losing weight – drinking unflavored fructose water between meals (Roberts, 2001, 2004). The theory did not predict this effect, but it led me to it. During a visit to Paris, my appetite vanished. I wanted to eat three meals per day, but had to force myself to eat even one. The lack of hunger had no obvious source. I felt fine and had been physically active. The theory described here suggested an answer. It implies that a food with a new flavor (a flavor not yet associated with calories) will not raise the set point. As it happened, I *had* eaten foods with flavors new to me. Because of the heat, I had drunk two or

three sucrose-sweetened soft drinks each day for several days. I had chosen drinks new to me, not available at home, with flavors with new to me.

To find out if the new soft drinks had eliminated my appetite, after coming home I tried drinking unflavored fructose water (fructose dissolved in water). The French soft drinks were probably sweetened with sucrose, but I used fructose because of its much lower glycemic index (Foster-Powell, Holt & Brand-Miller, 2002). I drank it between meals (at least an hour away from a meal) to prevent association of its calories with the flavors of other foods.

Fructose water dramatically reduced my appetite. For weeks, I was not hungry at all. Lack of hunger made it easy to eat much less than usual. Figure 4 shows my fructose consumption and my weight. In about three months, I lost 40 lb (18 kg). The daily amount of fructose it took to keep me comfortably at this (much) lower weight was about 140 kcal (0.6 mJ) per day. After being told several times that I was too thin, I chose to gain 10 lb (4.5 kg). A brief test of sucrose water suggested that it had similar effects (Roberts, 2004).

Explanation. Calorie-containing food that does not raise the set point will lower the level around which the set point oscillates, the theory implies. The weight loss caused by fructose water can be explained by assuming that it did not raise the set point. This is quite plausible. The sweetness-calorie association of fructose water – the strength of the association between its sweetness and its calories – is likely to be weak at best for two reasons. First, sweetness is inconsistently paired with calories for those who consume artificial sweeteners. Diet sodas, for instance, taste sweet but have almost no calories. I had drunk many diet sodas; except for the Paris experience, I never drank sugar-sweetened sodas. Many Pavlovian-conditioning experiments have found that reducing the accuracy with which a CS predicts a US reduces the

effect of CS-US pairings (Domjan, 1996). Warwick and Schiffman (1991) found that inconsistent flavor-calorie relationships reduced the flavor-calorie learning of rats. Second, fructose is a weak US. Fructose generates relatively weak flavor-calorie associations in rat experiments; some experiments have failed to find a reliable preference for a flavor paired with fructose over a flavor paired with water (Ackroff, Touzani, Peets & Sclafani, 2001; Sclafani & Ackroff, 1994).

Figure 4 does not contradict previous research. In spite of the usual view that sugar is fattening (e. g., Yudkin, 1972), the effects of sugar on weight are not simple. Based on the Dietary and Nutritional Survey of British Adults, Gibson (1996, p. 405) concluded that "sugars appear to have a weak *negative* [italics added] association with BMI that is not totally explained by confounders such as dieting, under-reporting or the inverse correlation between energy from sugars and fat."

Likewise, rat experiments "do not support the idea that sucrose causes obesity in humans" (Ramirez, 1987c, p. 1). When rats are given sucrose solution to drink, in addition to lab chow, they usually gain weight. However, other carbohydrates in solution are just as fattening (Ramirez, 1987c; Sclafani, 1987). When sugar is given in dry form (replacing other carbohydrate), its power to cause weight gain is much less (Ramirez, 1987c). Kratz and Levitsky (1979) found that giving rats sugar in granular form was less fattening when the rest of the diet consisted of protein, fat, and carbohydrate in separate cups from which the rats could choose than when it consisted of a composite food, a mixture of protein, fat, and carbohydrate. This implies that when sugar is fattening, the rest of the diet is involved in producing the effect. In the rat experiments in which sugar water has caused weight gain, the sugar water could have been

drunk soon after eating lab chow. Thus it may have strengthened the association of the flavor of lab chow with calories.

The widespread belief that soft drinks are fattening (see below) makes these results especially impressive. Tordoff and Alleva (1990) gave human subjects cola-flavored soda sweetened with high-fructose corn syrup. It caused an increase of 1 lb (0.5 kg) over three weeks. But they used *flavored* sugar water – a difference the theory implies is crucial.

Other explanations. Was my weight loss due to expectations? When I began drinking fructose water, I thought it was possible it would cause weight loss, but it turned out to be far more powerful than I expected (Roberts, 2004). (Note the diminishing amounts of fructose/day in Figure 4.) I had previously lost weight via four different dietary changes, such as drinking large amounts of water and eating a low-glycemic-index diet (Roberts, 2004). None of them produced anything close to the results of Figure 4. The maximum weight loss was 13 ld (6 kg). In one case the weight loss lasted only four weeks, implying that expectations have at most a short-lived effect.

CS-US Overlap

This section shows how the idea of Figure 2 – that the strength of flavor-calorie associations depends on CS-US overlap – explains several facts about weight control in addition to the texture results.

Bland Food

In several cases bland food has apparently caused easy weight loss, suggesting that it lowered the set point:

1. Kempner (1944) used a “rice diet” (p. 125) to treat the kidney disease and high blood

pressure of two patients. One of them started at 69 kg (BMI 25) and lost 10 kg in 15 days; eight months later his weight was even lower (58 kg). Another started at 74 kg (BMI 26) and lost 10 kg over less than eight weeks. In addition to rice, the diet included “sugar, fruit and fruit juices” (p. 125).

2. Herbert (1962) had his food finely chopped and boiled three times to remove all folate. The treatment also removed a lot of flavor molecules. Starting at 77 kg, Herbert lost 12 kg over 19 weeks without trying. There is no reason to think folate deficiency causes weight loss.

3. As mentioned earlier, Cabanac and Rabe (1976)’s subjects got all of their calories from Renutril (a bland nutritionally-balanced liquid food) for three weeks. They could consume as much Renutril as they wanted. Starting at 60-70 kg, they lost an average of 3 kg in three weeks.

4. Eating a diet consisting mainly of sushi (without wasabi), starting at 83 kg I lost six kg over three weeks (Roberts, 2004). I ate as much as I wanted, but I ate less than usual and lost weight because I was less hungry than usual. After losing 6 kg my appetite returned but I did not regain the weight as long as I continued the diet.

Explanation. Bland food lowered the set point because it produced weaker flavor-calorie associations than the person’s previous food. Figure 5 illustrates why. Many Pavlovian-conditioning experiments have found that reductions in CS intensity reduce asymptotic CR strength (Mackintosh, 1974).

Other explanations. Cabanac and Rabe (1976) attributed their subjects’ weight loss partly to the “monotonous” (p. 676) nature of their diet. This might be true, but the description does not apply well to the other three examples. Herbert’s diet was as varied as usual, except for its texture. Monotony per se does not appear to cause weight loss. I once ate a rather monotonous

diet (pasta and salad at every meal) for several weeks. At first I lost weight but then gained it all back (Roberts, 2004). In the Labouré, Saux and Nicolaidis (2001) experiments, the puree (one flavor) was more monotonous than the mixture (several flavors) yet caused more weight gain.

Low-Glycemic-Index Food

A food's *glycemic index* (GI) indicates how fast its consumption increases blood glucose. High-GI foods include bread and potatoes; low-GI foods include beans and lentils. In several long-term studies, a lower-GI diet has been associated with lower body weight:

1. Ludwig et al. (1999) found a negative correlation between fiber intake and both weight and weight gain over 10 years. Fiber content and GI are negatively correlated (Wolever, 1990).

2. Liu, Willett, Manson, Hu, Rosner, and Colditz (2003) found a strong negative association between fiber intake and weight gain over 12 years.

3. Kromhout, Bloemberg, Seidell, Nissinen, and Menotti (2001) found a negative correlation between population fiber intake and subscapular skinfold thickness.

4. In Rio de Janeiro, consumption of a diet high in rice and beans (a low-GI food) was associated with a lower rate of overweight and obesity (Sichieri, 2002).

Several experiments support the same conclusion:

1. Obese women lost more weight eating a low-GI diet for 12 weeks than a conventionally-balanced diet. Both diets lasted 12 weeks (Slabber et al., 1994).

2. Rats were given starch with either a high or low GI. After five weeks, rats fed the high-GI starch had more body fat (Lerer-Metzger, Rizkalla, Luo, Champ, Kabir, Bruzzo, Bornet & Slama, 1996).

3. Pawlak, Denyer, and Brand-Miller (2000) did a similar experiment for 32 weeks. At

the end of the experiment, rats in the high-GI group had 40% more body fat than rats in the low-GI group.

4. Obese children were given either a low-GI diet or a low-fat diet for four months. The low-GI diet produced more weight loss (Spieth et al., 2000).

5. For three weeks, middle-aged men with cardiac risk factors ate a low-GI diet or a diet high in sucrose. The low-GI diet produced more weight loss (Brynes et al., 2003).

6. Obese adolescents were advised to eat either a low-glycemic-load diet or a low-fat diet. After one year, the low-glycemic-load diet produced more weight loss (Ebbeling, Leidig, Sinclair, Hangen, & Ludwig, 2003).

Explanation. Low-GI foods are digested more slowly than other foods and thus produce a later calorie signal. This reduces CS-US overlap, and thus produces weaker CS-US associations. Figure 2 provides a graphical illustration, with puree = higher GI and mixture = lower GI.

More evidence that the GI of food affects the setpoint comes from a study by Pereira, Swain, Goldfine, Rifai and Ludwig (2004). Obese and overweight young adults were given one of two restricted-calorie diets, either low-glycemic-load or low-fat, for 16 weeks. The two diets had the same number of calories, so it was to be expected that both groups lost the same amount of weight. The subjects eating the low-glycemic-load diet reported less hunger, suggesting that their set points were lower than the set points of the subjects eating the other diet.

Other explanations. According to Brand-Miller, Holt, Pawlak, and McMillan (2002, p. 281S), “low-GI foods may benefit weight control in 2 ways: 1) by promoting satiety and 2) by promoting fat oxidation at the expense of carbohydrate oxidation.” But these explanations are

incomplete because they ignore evidence for regulation. Brand-Miller et al. do not explain why either effect should produce lasting weight loss. If more satiety causes less to be eaten at a given meal or a longer wait until the next meal, then regulation – the existence of a mechanism that tries to keep weight constant -- implies that more calories will be consumed at later meal. Rat experiments find that the fewer calories consumed at a meal, the shorter the wait until the next meal (Strubbe & Woods, 2004), consistent with regulation. According to any version of the set point theory of weight control, the only way to produce lasting weight loss is to lower the set point. It is unclear why the two effects mentioned by Brand-Miller et al. would do so.

Low-Glycemic-Index Food and Later Consumption

Six studies have found that after a meal of low-GI food, subjects eat less than after a meal of high-GI food (reviewed by Ludwig, 2000). For example, Ludwig, Majzoub, Al-Zahrani, Dallal, Blanco and Roberts (1999) gave subjects breakfast and lunch of either instant oatmeal (high GI) or a vegetable omelet with fruit (low GI). Between breakfast and lunch, the low-GI subjects were less hungry than the high-GI subjects. During the five hours after lunch, when subjects could eat what they wanted, the low-GI subjects ate about half as many calories as the high-GI subjects. Ludwig (2000) reviewed nine other tudies that found similar effects on satiety and hunger and one study that did not find a reliable difference.

Explanation. Because the low-GI food had a later calorie signal, its flavor was more weakly associated with calories, as explained earlier. Thus the low-GI meal raised the set point less than the high-GI meal, which reduced future consumption.

Other explanations. The paradoxical nature of this result has not been noticed. No one would deny that if high- and low-GI foods differ in their effect on weight it is the high-GI food

that is more fattening. It makes perfect sense for Ludwig (2000, p. 281S) to state that "the functional hyperinsulinemia associated with high GI diets may promote weight gain by preferentially directing nutrients away from oxidation in muscle and toward storage in fat." Yet if high-GI foods cause *more* fat storage than low-GI foods they should *reduce* consumption at later meals because of set-point regulation. For high-GI foods to produce *more* consumption at later meals requires – if you believe in set-point regulation – that hyperinsulinemia produces *less* fat storage than usual, which makes no sense given current understanding of what insulin does. In short, if you take set-point regulation for granted, and standard ideas about the effects of insulin for granted, and assume a *constant* set point, this result is very hard to explain.

Moist Food

Many experiments with rats and farm animals have found that adding water to dry food causes weight gain, even though water is available separately (Adkins, Wertz, Boffman, Hove, 1967; Bernadis & Bellinger, 1981, 1982; Keane, Smutko, Krieger & Denton, 1963; King, 1982; Ozelci, Romsos & Leveille, 1978; Ramirez, 1987a, 1987b; Sclafani, 1987; Sclafani & Xenakis, 1984). The weight increase reflected an increase in body fat (Adkins, Wertz, Boffman, Hove, 1967; Ramirez, 1987b; Sclafani, 1987). In some cases, adding water doubled the rate of weight gain. Ramirez (1987b) found that a moist-food diet increased the body fat of rats as much as a high-fat diet.

Explanation. Addition of water to dry food strengthens flavor-calorie associations in two ways: (a) increasing the flavor signal, thus increasing CS-US overlap and (b) speeding up digestion, thereby increasing CS-US overlap. Supporting the assumption that learning is involved, Ramirez (1987b) found that when wet food was introduced, rats initially consumed

fewer calories than other rats fed dry food. The wet/dry difference took about two weeks to reach full strength, roughly the same speed as flavor-calorie learning.

It is easy to notice that moist food is more flavorful than the same food dry. Ramirez (1991) found that a bitter substance was more effective in reducing intake when added to moist food than when added to dry food and concluded that "adding water to a food makes it easier for an animal to taste its food" (p. 387). That moisture speeds up digestion is harder to notice, but Lepovsky, Chari-Bitron, Lyman and Dimick (1960) found that "the rate of digestion is slower in chickens fed without water" (p. 394). Sclafani, Vigorito and Pfeiffer (1988) found that wet food produced more of an increase in plasma glucose than dry food.

Assuming that our understanding of the molecular events involved in taste and digestion is correct, then stronger flavor and faster digestion are obvious results of adding water. Taste and digestion require individual (free-floating) molecules. A taste signal is generated when a molecule binds to a taste receptor (a large protein). Digestion involves enzymes acting on single molecules. Adding water to food causes the water-soluble portions of the food, such as sucrose, fructose, and other carbohydrates, to be broken down into individual molecules. Adding water to food speeds up digestion because a necessary first step (generation of free-floating molecules) partially occurs before the food is eaten.

Once the water-soluble portions of a food are fully dissolved, however, more water should hurt rather than help, because it will dilute the flavor or energy-containing molecules. Dilution of the critical molecules will reduce flavor or slow digestion; for example, adding water to orange juice reduces the intensity of the flavor. Experiments have found the predicted reduction in weight gain with too much water (Keane, Smutko, Krieger & Denton, 1962;

Ramirez, 1987a). For example, Ramirez (1987a) found that food that was three-quarters water increased body fat less than food that was half water. More support for this analysis is the failure of Ramirez (1987b) to find an effect of adding water to a high-fat low-starch diet. Water does not dissolve fat.

Other explanations. To test the idea that wet food is more fattening because it is more palatable, Ramirez (1988) compared plain dry food with food that was both wet and bitter. Were palatability important, a large reduction in palatability should eliminate the effect. Yet even when the wet food was so bitter that the dry food was preferred in two-choice tests, the wet food still caused more weight gain than the dry food. Sclafani (1987) did a similar experiment. He added sucrose octa acetate (SOA), a bitter-tasting substance, to Polycose dissolved in water. He found that "rats do not initially prefer SOA-Polycose solution to Polycose powder or sucrose powder, yet in the present experiment the SOA-Polycose group consumed more saccharide and gained more weight than the Polycose-powder or sucrose-powder groups" (p. 160). He also found that changes in the taste of a wet diet, large enough to have a big effect on preference tests, did not reliably affect weight gain.

The change in texture produced by water is unlikely to matter. Large changes in viscosity produced in other ways had little effect on growth (Ramirez, 1987a; Sclafani, 1987).

According to King (1982), "the soaking of hard grains, such as maize, wheat, and barley, improves digestibility largely by ensuring more complete mastication" (p. 129), but he gives no support for this view. In rat experiments, the comparison is usually between wet and dry powder (e.g., Bernadis & Bellinger, 1981), which do not need to be chewed.

That wet food is more fattening than dry food contradicts the popular idea that water

promotes weight loss (e.g., Irons, 1998; Sears, 1995; Tennesen, 2000). As Drewnowski and Specter (2004, p. 8) put it, "bulky foods with a high water content are said to promote a feeling of fullness, which leads to reduced energy intakes both at the test meal and throughout the day." Obesity is often blamed on energy-dense foods (e. g., Institute of Medicine, 1995) but wet food is less energy-dense than the same food dry.

Fast Food/Junk Food

Research supports the widespread belief that fast food and junk food are fattening. Several surveys have found positive associations between fast food consumption and weight gain or junk food consumption and weight gain. Binkley, Eales, and Jekanowski (2000) found that persons who ate at fast food restaurants weighed more than those that did not, controlling for other factors. A survey of middle-aged American women found that more frequent eating at fast food restaurants was associated with greater BMI (French, Harnack, & Jeffery, 2000). The average BMI of persons in the highest third (who averaged 3 fast-food meals per week) was 2.8 more than the BMI of persons in the lowest third (who averaged zero fast-food meals per week). Over three years, persons who ate more fast food gained more weight. An increase in the frequency of fast-food meals was associated with an increase in weight. Jeffery and French (1998) found that the frequency of fast-food meals was positively correlated with BMI in women; the results with men were not reliable. Bowman, Gortmaker, Ebbeling, Pereira and Ludwig (2004) give other reasons to believe fast food and junk food cause obesity.

Other studies have found similar correlations with single foods. Two studies have found that an increase in the consumption of sugar-sweetened drinks was associated with weight increase in schoolchildren (Berkey, Rockett, Field, Gillman & Colditz, 2004;

Ludwig, Peterson & Gortmaker, 2001). Liebman, Pelican, Moore, Holmes, Wardlaw, Melcher, Liddil, Paul, Dunnagan, and Haynes (2003), surveying adults in Wyoming, Montana, and Idaho, found that consumption of soft drinks and other sweetened drinks was strongly associated with greater likelihood of being overweight or obese. Other studies have found positive correlations between eating French fries (French, Jeffery, Forster, McGovern, Kelder, & Baxter, 1994) or hot dogs (Harris, French, Jeffery, McGovern, & Wing, 1994) and obesity and/or weight gain.

James, Thomas, Cavan and Kerr(2004) did an experiment in which some primary-school children received an education program designed to discourage soft-drink consumption and others did not. A year late, students given the program had a smaller increase in the incidence of obesity than the other students.

Explanation. Figure 2 shows how the strength of a food's flavor-calorie association depends on both its flavor and its calorie source in a kind of multiplicative way. To produce a strong association (large overlap), the flavor must be strong *and* the calories quickly detected. Figure 2 does not show the importance of two other factors:

1. *Repetition.* Rat experiments show that flavor-calorie associations require repeated flavor-calorie pairings to reach full strength (Sclafani, 1991). Therefore a food must be eaten several times to produce the strongest possible association.

2. *Uniform flavor*. Because repetition is necessary, uniformity matters: The less variation in flavor, the better. The more variation in flavor from one instance to the next, the more of what is called *stimulus-generalization decrement*. Pavlov trained dogs to salivate when shown a certain color. They salivated less when shown a slightly different color.

To raise the set point the largest possible amount, then, a food should have (a) a strong

flavor signal and (b) quickly-detected calories and (c) be eaten many times with (d) exactly the same flavor each time. Fast foods and junk foods meet these four requirements much better than most foods (Table 1). Raw foods, such as apples, are widely available but have weaker tastes than the foods of Table 1, have a lower glycemic index (Foster-Powell, Holt & Brand-Miller, 2002), and, probably, vary more in flavor. Homemade foods, such as soups, have stronger flavors than raw foods because spices and other sources of flavor are added but probably vary more than manufactured foods. The packaged foods one buys in supermarkets are intermediate between fast foods and homemade foods – perhaps as uniform and strongly flavored as fast foods but not as convenient. A fast food that does not have a strong flavor is french fries. But french fries are not usually eaten alone. Because cooked potatoes have a high glycemic index, french fries should make the strongly-flavored foods they are eaten with more fattening.

Not only do fast foods and junk foods have strong flavors, quickly-digested calories, and low variation, they also benefit from what in other contexts is called a *network effect* – the larger the network, the greater the value of each new node. A phone connected to a network of 1000 phones is worth more than one connected to 10 phones. According to the analysis here, people learn to like the flavor of fast foods. As a food's flavor-calorie association becomes stronger, it tastes better, so the price a person is willing to pay for it increases and the profit to be made selling it increases. Consider the first McDonald's. Its only source of customers who had learned to like the exact flavors of its foods were those customers who had previously eaten there. In contrast, the ten-thousandth McDonald's can draw on customers who have learned to like the flavors of McDonald's food at other McDonald's. Thus a chain restaurant, if the chain is large enough, has an enormous advantage over other restaurants, in the sense of being able to deliver

more pleasure for the same price. The same argument applies to products such as Coke. If sold from just one store, the only persons who have learned to like it will be those who bought it at that store. After it achieves wide distribution, however, new locations benefit from the many people who learned to like Coke by drinking Cokes bought elsewhere. The management of McDonald's and Coca-Cola appreciate this point, of course, so they pay great attention to uniformity of product, doing their best to ensure that every McDonald's product and every Coke tastes the same no matter where you buy them (Schlosser, 2001). Of course, McDonald's and similar restaurants and Coke and similar drinks are great economic successes (French, Story & Jeffery, 2001; Schlosser, 2001).

Modern fast foods and junk foods, according to this analysis, are something new in the world of food. Not only are they perfectly suited to produce very strong flavor-calorie associations, they are widely available at low prices. As a result, a large number of people are regularly experiencing very strong flavor-calorie associations. Nothing like this has happened before. We can assume that the stronger its flavor-calorie association, the more pleasure a food provides. Estimates of expected pleasure are used to choose between different activities (Cabanac, 2002). An activity expected to provide a great deal of pleasure will be chosen over activities expected to provide less pleasure. So it makes sense that people sometimes treat fast foods and junk foods like addictive drugs (Pelchat, 2002). Sheehan (1995) described an Iowa family living near poverty and seemingly headed toward bankruptcy. To save the price of a stamp, they paid bills in person. Nevertheless, the husband and wife both drank a lot of Pepsi every day, calling themselves "Pepsiholics" (p. 84). Almost every week, they ate dinner at McDonald's. "Going out to dinner is as necessary to me as paying water bills," said the husband

(p. 89). Kirk (2002) describes a similar example. Ciment (1996) wrote that as a high-school student she deeply wanted to save money to go to New York. She took on extra jobs, but couldn't manage to save anything. "I wasn't wanton with my money. . . I just felt the dire need to reward myself for all my dogged hard work, to splurge on the extra candy bar, the jumbo Coke" (p. 131). When In-N-Out Burger, a California chain, opened a new store in 1996, one of the first customers was a college student majoring in health education. He told a reporter, "I was such a fan, I [had been] driving to Atascadero whenever I could convince somebody it was worth a three-hour drive. . . Now we have one here, and I'm in heaven" (Pope, 1996, p. 7A).

Other explanations. The usual explanation is that fast foods and junk foods are high in fat or sucrose and/or are energy-dense (e.g., Brownell & Horgen, 2004) and/or contain many calories (Young & Nestle, 2002). Research does not support these explanations. Willett (2002) made clear the lack of evidence that high-fat foods cause obesity (see also Ludwig et al., 1999). As mentioned earlier, Ramirez (1987c) explains why "animal studies do not support the idea that sucrose intake causes obesity in humans" (p. 1). The effects of moistening food, described above, shows that greater energy density can cause weight loss rather than weight gain. The idea that meals with many calories (large portion sizes) cause obesity ignores the evidence for regulation, discussed earlier. If one could gain weight simply by eating more calories, then one should be able to lose weight simply by eating fewer calories, which notoriously doesn't work. In rat experiments, as mentioned earlier, a smaller meal is associated with a shorter wait until the next meal (Strubbe & Woods, 2004).

Dietary Variety

Raynor and Epstein (2001) reviewed many rat experiments in which "greater dietary

variety [was] associated with increased body weight and fat" (p. 325). By dietary variety they meant the number of foods available at one time. Two popular weight-loss books (millions of copies sold) say that foods eaten separately will be less fattening than the same foods eaten together (Diamond & Diamond, 1985; Montignac, 1991, English translation 1999).

Explanation. Two foods eaten at the same time can be more fattening than the same foods eaten at different times because of *cross-conditioning*, where the flavor of one food becomes associated with the calories of the other food. Consider two foods, one with a strong CS and weak US, the other with a weak CS and strong US. Eaten separately, neither will generate a strong flavor-calorie association. If eaten at the same time, however, a strong flavor-calorie association will be formed by the strong CS of one food and the strong US of the other food. Many rat experiments about flavor-calorie learning have used separate sources of flavors and calories (Capaldi, 1996). Dietary-variety experiments are also affected by the tendency of rats to choose the food with the stronger flavor-calorie association (Sclafani, 1991). If rats are given Food A and Food B separately (e.g., on separate days), they will consume roughly equal amounts of the two foods in order to get the right amount of calories. But if the two foods are available at the same time, the food with the stronger flavor-calorie association can be eaten in greater amounts, raising the average flavor-calorie association of the diet.

Other explanations. Raynor and Epstein (2001) argue that "sensory-specific satiety" (p. 325) causes a rat to eat less of a meal with just one food than of a meal with more than one food. This makes sense. The problem is the implicit assumption, not discussed by Raynor and Epstein, that anything that reduces consumption at one meal will cause lasting weight loss. The problem with this assumption is that it ignores the evidence for regulation. In rat experiments, as

mentioned earlier, a smaller meal is associated with a short wait until the next meal (Strubbe & Woods, 2004).

Sensory-specific satiety may be why Labouré, Saux and Nicolaidis (2001) found that rats initially ate more of a mixture (composed of several different foods) than of a puree of the mixture (which had of course just one flavor). Recall that the puree eventually caused more weight gain than the mixture.

Economic Correlations

4.3.1 Rich and Poor Countries

Persons in rich countries weigh more than persons in poor countries. Figure 6 shows the correlation between BMI and per capita income in 1966, the date of the most recent many-country study that measured BMI.

Explanation. As income increased over the range shown in Figure 6, food made by hand from raw materials was replaced by the sort of machine-processed and packaged food that fills the supermarkets of rich countries. With an income of $10,000/year, you can afford supermarket orange juice; with an income of $100/year, you cannot. Persons living in rich countries could in theory eat as people in poor countries do (the ingredients are available), but choose not to. There are two things to explain: why supermarket food is chosen over (cheaper) homemade food; and why supermarket food is more fattening than homemade food.

Why is supermarket food chosen over homemade food? This is a choice that many readers of this article make daily, no doubt, so suffice it to say that supermarket food saves a great deal of time. And some of the tastiest supermarket foods, such as cheese, prepared meat

(ham, sausage), sweets (e.g., ice cream, candy, pastries), and soft drinks, are difficult or impossible to make at home.

Why is supermarket food more fattening than homemade food? Supermarket food is just a milder version of fast food and junk food and is relatively fattening for the same reasons. Shelf space is limited. Better-selling foods replace worse-selling ones. One way to sell more is to taste better; and one way to taste better is to have a stronger flavor-calorie association. Thus strong flavors, quickly-digested calories, exact repetition of flavor, and wide availability are encouraged or selected for. Homemade foods can have strong flavors easily enough; but it is not easy to repeat the same flavor each time – home cooking is less standardized than factory production. Supermarket foods benefit from network effects just as fast foods do, so it is easy to understand why some food companies are very large.

In the United States and other rich countries, the relationship between income and weight is the opposite of Figure 6 – it is poverty, not wealth, that is associated with greater weight (Darmon, Ferguson & Briend, 2003; Drewnowski & Specter, 2004). This can be reconciled with the explanation given above if it is assumed that the relationship between cost per calorie and strength of flavor-calorie associations follows the function shown in Figure 7 – that it reaches a maximum and then declines. In the American marketplace, the foods that produce the strongest flavor-calorie associations (and are therefore the most fattening) are not the most expensive but are fast foods and junk foods, for reasons explained earlier. In the United States, the poor are relatively well-off, compared to other countries, and consumption varies between foods that are as cheap as fast foods and more expensive foods. In Berkeley, California, for example, a large container of Coke costs about $2.00/1000 kcal; a McDonald’s double cheeseburger, about

$2.20/1000 kcal; an apple, at least $6.00/1000 kcal (in February 2004). Drewnowski and Specter (2004) make a detailed connection between poverty in the United States and consumption of more fattening foods. In poor countries, by contrast, fast foods are among the most expensive foods. A poor person in the United States can afford a Coke; a poor person in China cannot.

The view that the relationship between poverty and obesity in the United States is due to the United States' relative affluence is supported by data from poorer countries such as India and Brazil, in which rich persons weigh more than poor persons (De Vasconcellos, 1994; Naidu & Rao, 1994; Sobal & Stunkard, 1989).

The two outliers in Figure 6 (Zimbabwe and Japan) support the idea that food can have a big effect. It is unlikely that they are due to exercise – less exercise in Zimbabwe compared to other poor countries or more exercise than usual in Japan compared to other rich countries. Persons in Zimbabwe cannot have had access to the many devices that reduce physical activity in rich countries; they could not afford them. Nor can persons in Japan failed to have access to those devices. Why Japan is exceptional is discussed below. As for Zimbabwe, the theory implies that persons in a poor country can be relatively fat if their main calorie source has a high glycemic index. Strong complex flavors are relatively cheap. Persons in poor countries who eat a lot of potatoes, which have a high glycemic index, should be relatively fat, too.

Other explanations. Some of the weight difference between poor countries and rich ones may be due to differences in physical activity, but it is unlikely that most of it is. Ordinary amounts of exercise produce little weight loss. Only extreme amounts of exercise, such as training for a marathon, would make someone from the United States as thin as someone from Kenya or India (Williams, 1997). Garrow and Summerbell (1995, p. 1), after a review of the

literature on exercise and weight loss, concluded that "aerobic exercise causes modest weight loss without dieting." Fogelholm and Kukkonen-Harjula (2000, p. 106), after a similar review, concluded that "the effects of a prescribed exercise programme remain very limited." A panel report about obesity treatment sponsored by the National Institutes of Health (National Heart, Lung, and Blood Institute, 1998) summarized clinical trials of exercise (mainly aerobic exercise) by stating that "10 of the 12 RCT [randomized clinical trial] articles reported a mean weight loss of 2.4 kg (5.3 lb) . . . or a mean reduction in BMI of 0.7 kg/m^2" (p. 45) and even this small difference was an over-estimate. In the two omitted RCTs, the exercise group weighed slightly *more* than the control group. A recent experiment on the effects of exercise on weight loss, involving 201 overweight sedentary women, failed to find significant differences between the effects of different amounts of exercise (Jakicic, Marcus, Gallagher, Napolitano & Lang, 2003). The range of the effects of exercise was about 2% of the women's average body weight. The range of BMI shown in Figure 5 is about 20%.

Gutiérrez-Fisac, Guallar-Castillón, Diez-Gañán, Garcia, Banegas, and Artalejo (2002), studying a large sample of Spaniards, found no reliable association between body mass index and physical activity at work. This was consistent with other studies of the effect of work activity, which they reviewed. They did find that more intense leisure-time physical activity was associated with lower weight–but the BMI difference between "inactive" and "intense" was only 1.4.

Willett (2002) convincingly argued that differences in fat intake are unlikely to cause weight differences of this size (see also Ludwig et al., 1999). For a different view, which I find less persuasive, see Astrup (2002).

Japanese Thinness

Figure 6 shows that in 1966 the Japanese were much thinner than persons of other countries with similar incomes.

Explanation. Japanese food has weaker flavors than other cuisines. As one cookbook says, “Most Japanese cuisine is seasoned only lightly; strong spices are never used” (Suzuki, 1994, p. 8). Weaker flavors lead to weaker flavor-calorie associations, as discussed earlier (Figure 2). In addition, at the time of the data shown in Figure 7, Japanese cuisine was also low in high-GI foods, such as bread and potatoes. According to Barer-Stein (1980, p. 335), “the staples of the traditional Japanese diet are rice, fish and seafood, vegetables and tea.” This too should have reduced the strength of flavor-calorie associations, as discussed earlier.

When Japanese emigrate to the United States and adopt an American diet, they eventually weigh as much as other Americans (Curb & Marcus, 1991), which supports this explanation.

Other explanations. A study comparing Japanese and American telephone executives found a weight difference close to the American-Japanese difference shown in Figure 7 (Sakai, Comstock, Stone & Suzuki, 1977). They also found that the Americans exercised *more* than the Japanese, implying that a difference in exercise is unlikely to explain Japanese thinness. The Japanese executives smoked more (64% were present smokers) than the Americans (42% were present smokers). Smoking certainly causes weight loss but its effect is too small to explain the 20-kg difference between the two groups. A study of monozygotic twins found that heavy smokers were 4 kg lighter than nonsmokers (Eisen, Lyons, Goldberg, & True, 1993). Thus smoking explains no more than 0.8 kg (4 kg times the 22% difference in smoking) of the 20-kg

difference in weight.

Sclafani and Springer (1976)

To make rats fat quickly, Sclafani and Springer (1976) placed supermarket food, namely "chocolate chip cookies, salami, cheese, bananas, marshmallow, milk chocolate, and peanut butter" (Sclafani & Springer, 1976, p. 462), in their home cages. Lab chow remained available. The rats could eat as much as desired. After eight weeks, they had gained over three times as much weight than rats not given supermarket food – far more weight gain than a high-fat diet produced under the same conditions (Sclafani & Springer, 1976). Similar results have been reported many times (Raynor & Epstein, 2001).

Explanation. Why was supermarket food much more fattening than lab chow? As stated earlier, supermarket food competes for shelf space. Foods that produce a strong flavor-calorie association will be preferred, other things equal (Sclafani, 1991). Supermarket foods are selected for this property, in the sense that foods with more of it are more likely to be bought; and foods that are not bought are no longer stocked. Lab chow does not undergo the same selection process. Rats are not given a choice between different versions of lab chow and how much tasty rats find a particular version has little effect on what is bought or made.

The supermarket food took one to two weeks to cause weight gain, which argues that learning was involved (Ramirez, 1990a). This is roughly the length of time required for flavor-calorie learning to become strong (e. g., Bolles, Hayward & Crandall, 1981).

Other explanations. Perhaps the supermarket food was more palatable than the lab chow. However, the delay in the start of this effect argues against this explanation (Ramirez, 1990a). Ramirez, Tordoff and Friedman (1989, p. 163) noted that "evidence for this hypothesis [that

palatable food causes obesity] is particularly weak."

Second Assumption

The theory's second assumption is that the set point steadily decreases. The evidence in this section supports this assumption by showing that the set point seems to decrease if no flavor is experienced.

Less-Than-Compensatory Eating After a Fast

Johnstone, Faber, Gibney, Elia, Horgan, Golden & Stubbs (2002) studied the effects of a 36-hr fast (one full day) on 24 men and women. While fasting, they were hungrier than usual. On the day after the fast, they could eat as much as they wanted but "consumed much less energy than required [only 25% more than usual] to compensate for the energy deficit induced by the fast" (p. 1626). Yet after the first post-fast meal (breakfast), they were no hungrier than usual.

Explanation. The set point declined during the fast. Because of the decline, the amount of food needed to bring body fat to the set point level was less than the energy deficit produced by the fast.

Other explanations. None, so far. Johnstone et al. (2002) do not give a theoretical explanation of their results.

Fantino (1976)

Fantino (1976)'s human subjects ingested a balanced liquid diet via a nasogastric tube. Thus their food had no flavor. They could ingest as much as desired. In about three weeks, they lost 10% of their body weight, a very large amount to lose so fast. Fantino was a student of Cabanac's and this experiment was probably motivated by something like Cabanac and Rabe's

(1976) idea that the set point is controlled by flavor. It illuminates what happens in the absence of flavor.

Explanation. Without flavor experience, the set point falls, regardless of caloric intake.

Other explanations. Perhaps the nasogastric tube caused loss of appetite. However, there is no indication of this in other studies with such tubes (Stratton, 2001).

Related research. Other tube-feeding experiments are also relevant. Although Fantino's subjects could infuse as many calories as they wanted, they probably felt deprived in other ways. Stratton (2001, p. 149) noted that patients fed a complete diet by tube for long periods of time nevertheless "tasted, chewed, and then spat out foodstuffs in order to satisfy their appetite." This suggests that food intake is controlled by more than the fat storage system – at least a desire to taste and chew. Studying rats, Nicolaidis and Rowland (1976) found that intravenous infusion of glucose suppressed food intake less than infusion of a more nutritionally-complete solution with the same caloric value. The idea that nutritional needs other than energy control food intake was supported by the cafeteria experiments of Richter (1942), in which rats chose a healthy diet from among many alternatives.

Flavorless feeding does not eliminate weight regulation. Rats that pressed a bar for food they could not taste or smell (delivered via an intragastric tube) kept their weight constant in spite of changes in the calorie density of the infused food and the bar press requirement (Epstein & Teitelbaum, 1962). So there is a body-fat regulatory system that depends on something besides flavor. However, flavorless feeding may eliminate regulation at a high level (i.e., obesity). McGinty, Epstein and Teitelbaum (1965) gave rats ventromedial hypothalamic lesions, which usually produce extreme obesity. When these rats were put in the Epstein and Teitelbaum (1962)

situation – so that they fed themselves by pressing a bar for intragastric food – they gained weight slowly, if at all. If already obese, they lost weight. “Our rats did not reach high levels of obesity when they could not taste or smell their food” (McGinty, Epstein & Teitelbaum, 1965, p. 417). When the intragastric food was accompanied by a small amount of flavored solution, however, extreme overeating resumed. Full resumption took a few days, a reasonable length of time to learn to asymptote an association between the flavor of the solution and the intragastric calories. The McGinty, Epstein and Teitelbaum (1965) results are entirely consistent with a set point raised by calorie-associated flavors.

Miscellaneous

Delayed Weight Gain

A new food’s eventual effect on weight is often different from its initial effect. In every case I know of, new food becomes more fattening – the weight loss decreases or the weight gain increases. Here are examples:

1. Willett (1998) reviewed five studies of one year or longer on the effect of low-fat diets. In eight of nine cases (five intervention groups, four control groups) where it could be assessed, lost weight was at least partly regained.

2. Swinburn, Metcalf, and Ley (2001) measured the effect of a low-fat diet on persons with glucose intolerance. The difference between control and treatment groups was greatest after one year, when the treatment group weighed an average of 3.3 kg less. The difference between the groups shrank year by year and after five years was gone.

3. Sclafani and Springer (1976), as mentioned earlier, gave rats a selection of

supermarket food in addition to their lab chow. The treatment and control groups were equal in weight for about two weeks; after that, the treatment group gained more weight.

4. Ramirez (1990a) – the study with pre-exposure effects, described earlier – found that the weight-gain effect of saccharin took about one week to reach full strength. It was weaker during the first week of saccharin than during later weeks.

5. Ramirez (1987b), as mentioned earlier, found that rats given wet food introduced, rats initially ate less of it than other rats given dry food. After two weeks, however, they ate more of it, and gained more weight.

6. Warwick, Synowski, and Bell (2002, Experiment 1) shifted rats from a diet of ordinary lab chow to a liquid high-carbohydrate diet. The rats lost weight for about a day, stayed at roughly the same weight for seven days, and then started to gain weight.

7. Warwick (2003) switched rats from a solid diet to a liquid one. After the change, their weights decreased for two days, were constant for about seven days, then increased.

Other studies show almost the same thing. Phelan, Hill, Lang, Dibello, and Wing (2003) studied persons in the National Weight Control Registry, which consists of persons “who have lost # 13.6 kg (30 lb) and kept it off # 1 yr” (Phelan, Hill, Lang, Dibello & Wing, 2003, p. 1079) in any way. In almost every case, the method of weight loss included dietary change. Phelan et al. found that after two years in the registry, the members had gained an average of 1.9 kg/yr, far more than the average weight gain of similar unselected adults, about 0.7 kg/yr. They also found that “recovery from even minor weight gain was uncommon” (p. 1079). A study of female nurses found that participants who lost at least 10% of their initial weight over 2 years gained an average of 2.3 kg/yr over the next four years (Field, Wing, Manson, Spiegelman & Willett,

2001).

Not all means of weight loss show this pattern. Persons who lost weight by taking orlistat, a drug that reduces fat absorption, showed no signs of weight regain during the first year (Finer, James, Kopelman, Lean, & Williams, 2000).

Explanation. The new diets included new flavors, not yet associated with calories. Experience with the new flavors created and strengthened flavor-calorie associations. This took time. As the new flavors became better associated with calories, they raised the set point more.

Other explanations. To explain the disappearing weight loss caused by low-fat diets, Willett (1998) considered the possibility that "compliance may deteriorate with time" (p. 559S). Because some changes caused by the low-fat diet persisted, Willett concluded that the weight regain was not due to increasing noncompliance but rather to "compensatory mechanisms" (p. 556S). Compensatory mechanisms would not explain cases of delayed weight gain (e.g., Sclafani & Springer, 1976). Swinburn, Metcalf, and Ley (2001) did not offer an explanation of the disappearance of the weight difference. However, they divided subjects in the intervention group into two groups, more and less complaint. By the end of the experiment, weights of the two groups did not differ–both were the same as the control group–which supports Willett's conclusion that noncompliance was not the cause of weight regain.

Flavorings

Hirsch and Gallant-Shean (2004) gave human subjects flavorings to sprinkle on their food – two flavors each month for six months. Cheddar cheese and cocoa were the first two, onion and spearmint the next two, horseradish and banana after that, and so on. Initially, subjects had an average weight of 90 kg (197 lb) and an average BMI of 31. During the six-month

experiment, their BMI decreased 13%. They lost 16 kg (35 lb) compared to control subjects who did a traditional diet program (and gained a small amount of weight),.

Explanation. The flavorings reduced recognition of familiar flavors. Over years, the subjects had learned to associate the flavors of their usual food with calories; now this learning was lost, or partially lost, because the flavors changed. The importance of uniformity of flavor was mentioned earlier in the discussion of junk food. The combination Familiar Flavor X plus cocoa flavoring (new) will raise the set point less than Familiar Flavor X alone. Several features of the treatment – use of more than one flavoring per month, new flavors every month, and addition of flavoring via sprinkles (quite variable) – made it more difficult for the new flavor combinations (such as Familiar Flavor X plus cocoa) to become strongly associated with calories.

Hirsch and Gallant-Shean were not aware of the theory described here but their work supports one of its most interesting predictions: If the flavors of your food are always changing (never the same flavor twice) you will have a low set point. Hirsch and Gallant-Shean came close to creating such a world.

Other explanations. These results are hard for most theories to explain because a tiny percentage change (close to zero) in the chemical composition of the diet caused a large percentage change in weight.

CONCLUSIONS

Summary of Theory

The theory takes a familiar idea -- body fat is regulated by a system with a set point – and adds two rules about how the set point changes. One rule is that calorie-associated flavors raise

the set point – the stronger the association, the greater the increase. The other rule is that these increases are superimposed on a steady decline – the greater the set point, the faster the decline. A steady state is reached when the rate of flavor-generated increases equals the rate of decline.

A food is fattening (raises the set point) to the extent its flavor is associated with calories. The strongest flavor-calorie associations will occur, learning research implies, when four things are true: (a) the flavor is strong and complex flavor; (b) the food is digested quickly; (c) the food is eaten repeatedly; and (d) the flavor is exactly the same from one instance to the next. These four traits combine in a multiplicative way in the sense that if one is entirely absent, the food will not raise the set point at all.

Relation to Previous Work

This work has several precursors. It is an elaboration of the Cabanac and Rabe (1976) proposal that flavor controls the set point. Cabanac knew that the body-temperature set point depends on ambient temperature (Hammel, 1968), which made it easier for him to think that the body-*fat* set point is controlled by outside conditions. Corbit and Stellar's (1964) results had suggested the importance of palatability. A more recent precursor is Keesey and Hirvonen's (1997) conclusion that obesity is due to an elevated set point, elevated at least partly by diet. To the importance of flavor the theory adds the importance of learning. Ramirez (1990a) reached the same conclusion. The work of Sclafani and others on flavor-calorie learning clarified what that learning would be.

Pavlovian conditioning has been used to explain a wide range of phenomena. J. B. Watson used it to explain Little Albert's fear of rabbits. Recent examples include Siegel's (1999) theory of drug tolerance and craving and Siegel and Allen's (1992) theory of the McCullough effect, a

visual aftereffect. But this is the first time it has been put at the center of a theory of weight control.

Previous ideas about what makes food fattening have emphasized single *nutritional* dimensions, such as calorie content or density, fat content or density, sugar content or density, carbohydrate content or density, and glycemic index or glycemic load (Foster-Powell, Holt & Brand-Miller, 2002). In contrast, this theory emphasizes a single *psychological* dimension, the strength of flavor-calorie associations.

Strength of Evidence

How plausible is the theory? It has several attractive features:

1. The mechanism does something useful: Adjusts the amount of stored energy (fat) according to the cost of obtaining energy ("Similarity to other storage systems"). It can be argued a priori that a fat-storage mechanism should do this, and man-made storage systems often work this way (Williams & Wright, 1991). Because flavor-calorie learning presumably controls what humans and rats eat, such learning should be involved in the adjustment.

2. It explains strong and well-established effects or correlations, including the BMI/income correlation (Figure 7), the Sclafani and Springer (1976) results and similar results (Raynor & Epstein, 2001), and the effect of moistening food.

3. It explains several hard-to-explain effects, including the effects of pre-exposure (Ramirez, 1990a), pureeing food (Labouré, Saux & Nicolaidis, 2001), moistening food, and sprinkling flavoring on food (Hirsch & Gallant-Sheen, 2004).

4. It explains several effects or correlations that seem contradictory: (a) Bland food (e.g., Herbert, 1962) and food with no flavor at all (Fantino, 1976) cause weight loss; so does food with

more flavor than usual (Hirsch & Gallant-Sheen, 2004). (b) Increasing the number of flavors available can cause both weight gain (Raynor & Epstein, 2001; Sclafani & Springer, 1976) and weight loss (Hirsch & Gallant-Sheen, 2004). (c) An increase in income can be correlated with both weight gain and weight loss.

5. The theory helped find a powerful and surprising way of losing weight (drinking unflavored fructose water, Figure 4).

6. The evidence is diverse. Four different ways of varying the strength of flavor-calorie associations are involved: changes in CS strength (bland food), changes in the CS-US interval (texture, low-GI food), CS preexposure (Ramirez, 1990a), and US preexposure (Ramirez, 1990a). It includes two different studies that support the idea that the set point declines in the absence of flavor: with (Fantino, 1976) and without (Johnstone et al., 2002) caloric intake during the absence of flavor.

Research Implications

The supporting evidence was gathered for other purposes, an important weakness. The theory would gain plausibility if it made correct predictions or, more realistically, given the complexity of food, led to the discovery of new effects.

The theory's most basic predictions are:

1. *Novelty*. New food (with new flavors) will lower the set point because new flavors will not be associated with calories. As the new flavors become associated with calories, the set point should increase. The results described in "Delayed Effects" support these predictions.

2. *CS variation*. Variation in the flavor of familiar foods will reduce the set point. The results of Hirsch and Gallant-Sheen (2004), whose subjects sprinkled flavorings on their food,

support this prediction.

3. *CS strength*. Increasing the strength and/or complexity of a weak flavor will increase the strength of the flavor-calorie association and thus make the food more fattening. The results described under "Bland Food" support this prediction.

4. *CS-US interval*. Treatments that speed up detection of a food's calories will increase the strength of the flavor-calorie association and thus make the food more fattening. The results of Labouré, Saux & Nicolaidis (2001), that pureeing a mixture made it more fattening, support this prediction.

5. *Separation*. A strongly-flavored food with few calories (e.g., thin slices of pepperoni) and a weakly-flavored food with quickly-digested calories (e. g., mashed potatoes) will be more fattening when eaten at the same time than when eaten hours apart. When eaten together, the strong CS provided by one can become associated with the strong US provided by the other.

6. *Correlation between flavor-calorie strength and weight gain*. The stronger the flavor-calorie association, the greater the weight gain. For example, intragastic glucose produces stronger flavor-calorie associations than intragastic fructose (Ackroff, Touzani, Peets & Sclafani, 2001). So cherry-flavored glucose water should be more fattening than cherry-flavored fructose water.

7. *Unreinforced exposure.* Treatments that weaken flavor-calorie associations will reduce the set point. A good way to weaken a Pavlovian association is to present the CS without the US. For example, giving rats cherry-flavored glucose water in addition to lab chow should cause weight gain. Experience with cherry-flavored water without calories should reduce this effect. Siegel (1999) used similar predictions to test a Pavlovian theory of drug tolerance.

The theory also suggests changes in how weight control experiments are done. The implication that novelty matters (supported by the examples described in "Delayed Effects") means that novelty should be measured or controlled. Otherwise the intended difference between conditions may be confounded with a difference in novelty. Mattes (1996), for example, found that solid, semisolid, and liquid foods reduced later intake by different amounts. Because the three types of food may have differed in novelty (maybe the liquid foods were less familiar), the outcome differences may have been due to novelty differences rather than differences in water content.

A broader implication is that more attention should be paid to measuring the set point (e.g., Cabanac & Frankham, 2002). With rats fed ad libitum, it is reasonable to assume their actual weight is close to their set point weight (Cabanac & Gosselin, 1996). With humans, this assumption is less certain. Whether or not a treatment changes the difference between actual weight and set point weight will often affect interpretation of the results. For example, Leibel, Rosenbaum, and Hirsch (1995) caused human subjects to lose weight by feeding them restricted amounts of a liquid diet. As they lost weight, their metabolism slowed down. Leibel et al. took the reduced metabolic rate to be part of a system that resists weight loss. But the liquid diet may have substantially lowered the set point (Cabanac & Rabe, 1976). If the set-point weight remained close to the actual weight, that would suggest a different interpretation of the metabolic changes.

Practical Implications

Brownell and Hagen (2004) blamed recent increases in American obesity on "a toxic environment" (Brownell & Hagen, 2004, p. 7) of fast food, junk food, and ads for fast food and junk food. The theory supports this conclusion (see "Junk Food/Fast Food") but casts a wider net:

It suggests that a large fraction of packaged food is to blame because much of it has a strong flavor, quickly-digested calories, very similar taste from one instance to the next, and is eaten repeatedly.

The weight-control system, according to the theory, is designed to make us fat when food is cheap – and food is getting cheaper every year as incomes rise. The better something tastes, the more can be charged for it; and one way to make food taste better is to give it a stronger flavor-calorie association. This would explain why the food industry makes many products with strong flavors and high-glycemic-index components (such as bread and sucrose), uses low-variation mass production, and relies on repetition (also called repeat business). The food companies that most fully incorporated those elements outperformed their competition.

The theory supports the common recommendation to avoid foods with a high glycemic index (e.g., Atkins, 1992; Montignac, 1999; Steward, Bethea, Andrews & Balart,1995) but also provides some unusual advice:

1. *Eat new foods.* No food with a new flavor is fattening, the theory implies.

2. *Vary the flavor of foods eaten repeatedly.* If products came with optional flavoring packets and consumers added varying amounts of the flavorings, this would produce variation in flavor. The results of Hirsch and Gallant-Sheen (2004) suggest the power of this advice.

3. *Consume calories with no flavor associations.* Ingestion of calories with no flavor should lower the set point, the theory implies. The fructose-water results suggest that ingestion of a small fraction of one's daily calorie intake this way may substantially reduce the set point. Flavorless vegetable oils (vegetable oils, such as olive oil, from which all flavor molecules have been removed) are a possible source of calories without taste.

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AUTHOR NOTE

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Table 1

Why Fast Food and Junk Food Produce Strong Flavor-Calorie Associations

	Source of			
food	**strong/complex flavor**	**quickly-detected calories**	**uniformity**	**repetition**
Coke	Flavoring	sucrose, glucose in high-fructose corn syrup	mass production	widely available
McDonald's hamburger	ketchup, mustard, pickles, onions, pepper	hamburger bun, French fries eaten at the same time	standardized preparation, mass-produced ingredients	widely available
Pizza Hut pizza	sauce, toppings	bread	standardized preparation, mass-produced ingredients	widely available
Krispy Kreme doughnut	fillings and/or toppings	Sucrose, bread	mass production	widely available
hot dog	spices in meat, mustard	hot dog bun	mass-produced ingredients	widely available
chocolate bar (Snickers, Mars, etc.)	chocolate, flavorings	sucrose	mass production	widely available

Note. In most cases, fat and/or water in the food increase the flavor signal by dissolving fat-soluble or water-soluble flavor molecules. Whether the fat in hamburgers, pizza, etc., substantially increases the calorie signal is unclear. *Mass production* means large-scale production with great attention to reducing variation (i.e., quality control).

FIGURE CAPTIONS

Figure 1. How the set point changes in response to more fattening food. The new food raises the set point more than the old food.

Figure 2. The importance of CS-US overlap. The area of the shaded portion is proportional to the eventual strength of the resulting flavor-calorie association. The more area, the stronger the association. CS = conditioned stimulus. US = unconditioned stimulus. CR = conditioned response. *Asymptotic* CR strength = CR strength after the CS and US have been paired many times.

Figure 3. Why the puree of Labouré, Saux and Nicolaidis (2001) produced a stronger flavor-calorie association than their mixture.

Figure 4. My experience with fructose water.

Figure 5. Bland food generates a weaker flavor signal than ordinary food and thus generates a weaker flavor-calorie association.

Figure 6. Correlation between BMI and per capita income. Each point is a different country. BMI's: Intersalt Cooperative Research Group (1988). Incomes: 1986 data from an economic data base called the Penn World Tables 5.6. The tables are described in Summers and Heston (1991). The data are at http://datacentre2.chass.utoronto.ca/pwt/.

Figure 7. Hypothesized strength of flavor-calorie associations versus cost.

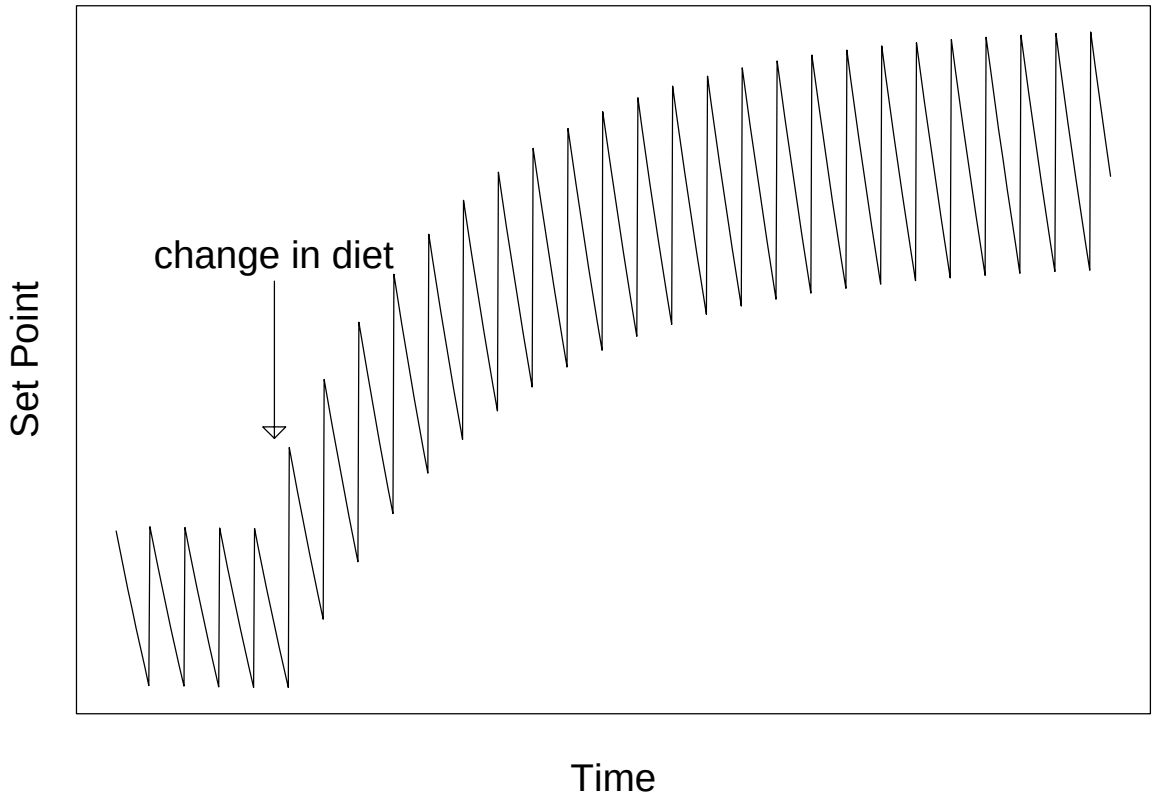


Figure 1

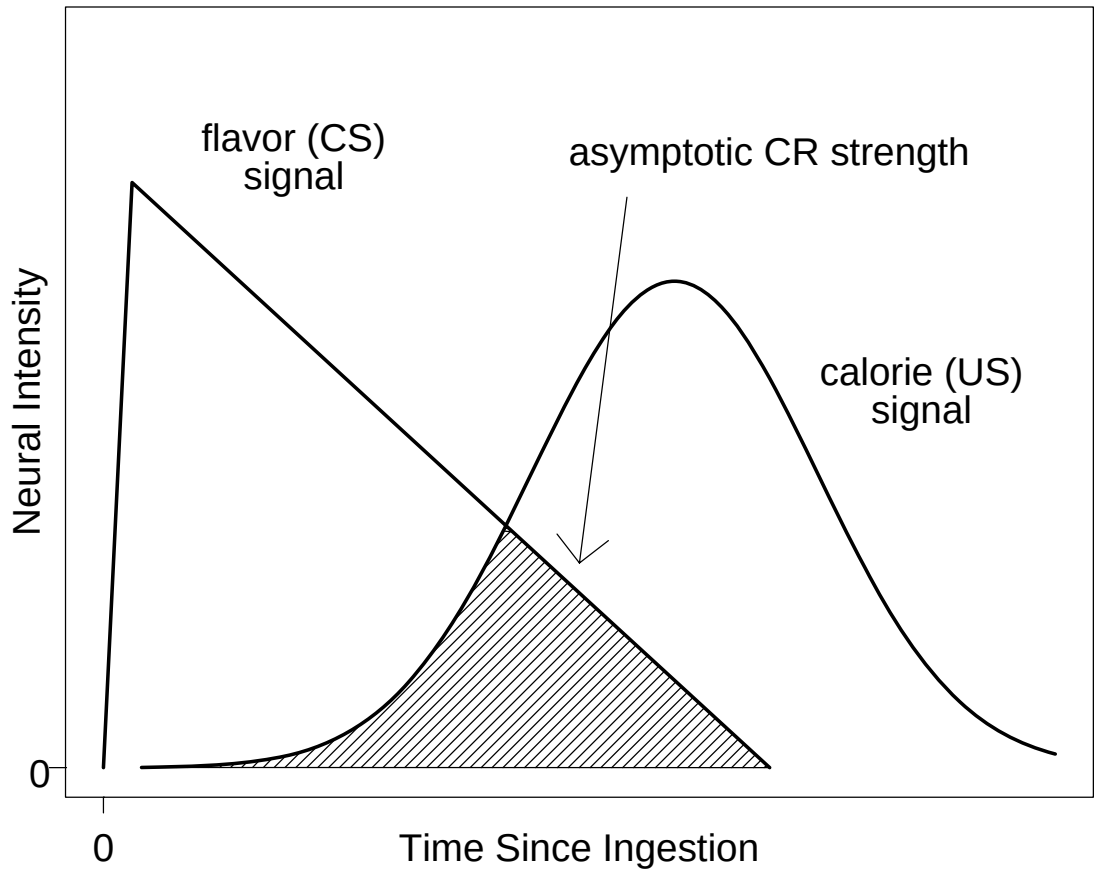
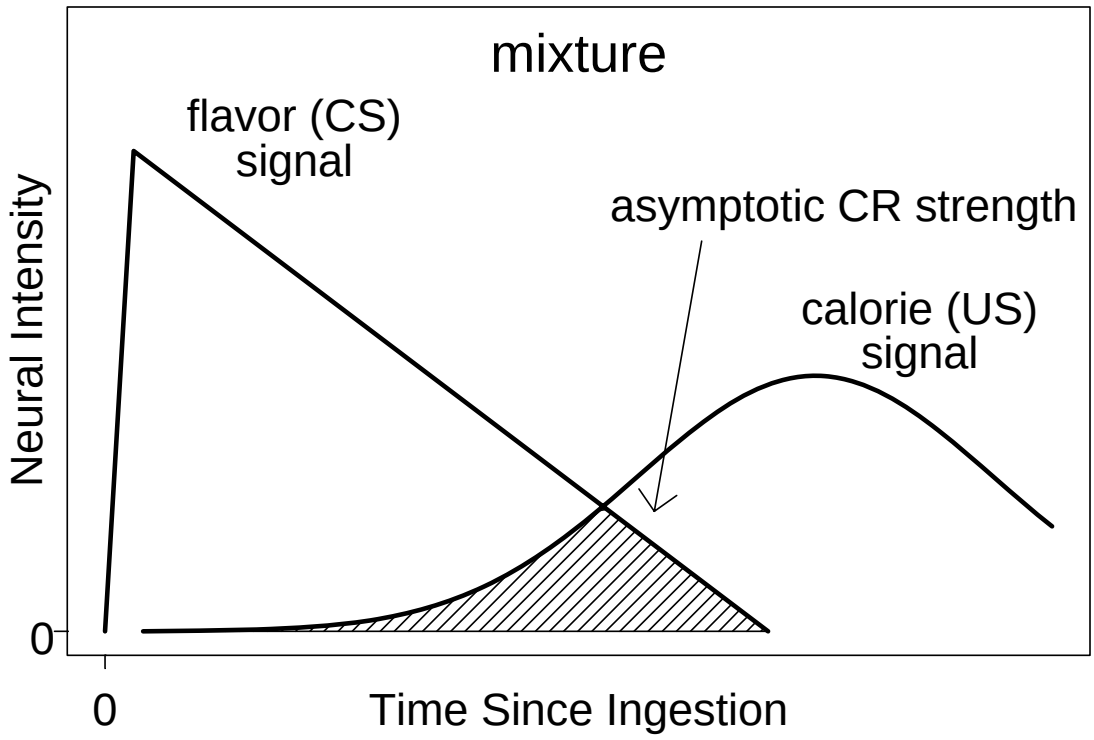

flavor (CS)
signal
asymptotic CR strength
calorie (US)
signal
Neural Intensity
0
0
Time Since Ingestion

Figure 2


mixture
flavor (CS)
signal
asymptotic CR strength
calorie (US)
signal
Neural Intensity
0
0
Time Since Ingestion

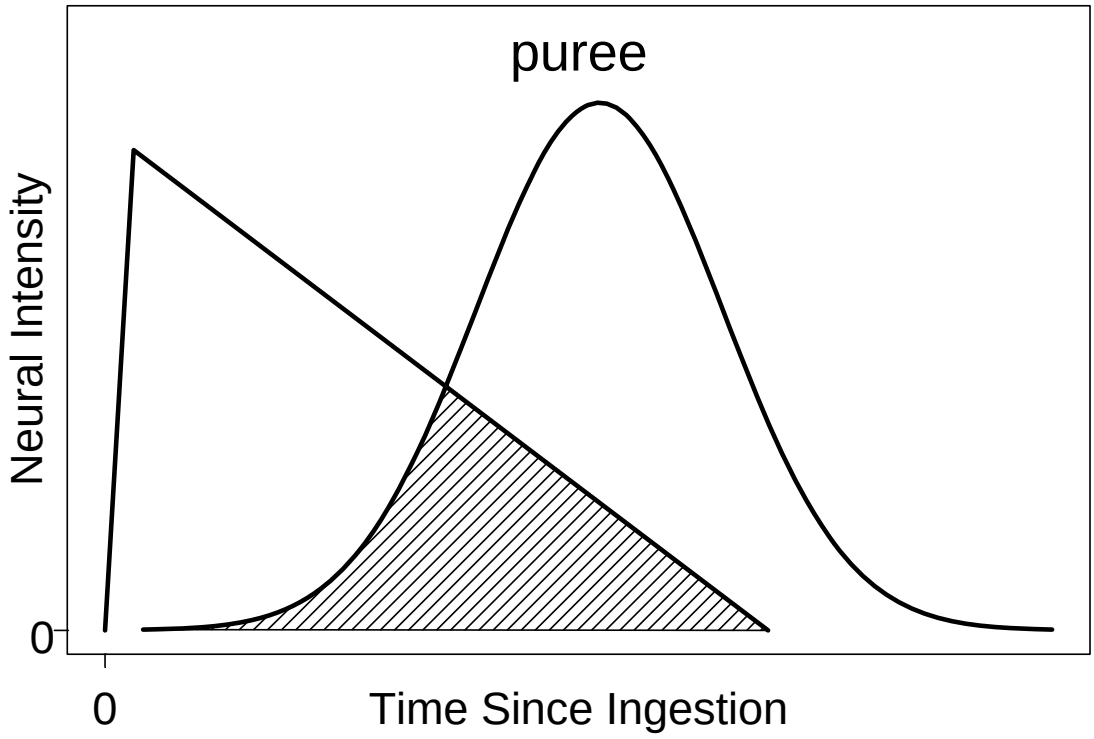

puree
Neural Intensity
0
0
Time Since Ingestion

Figure 3

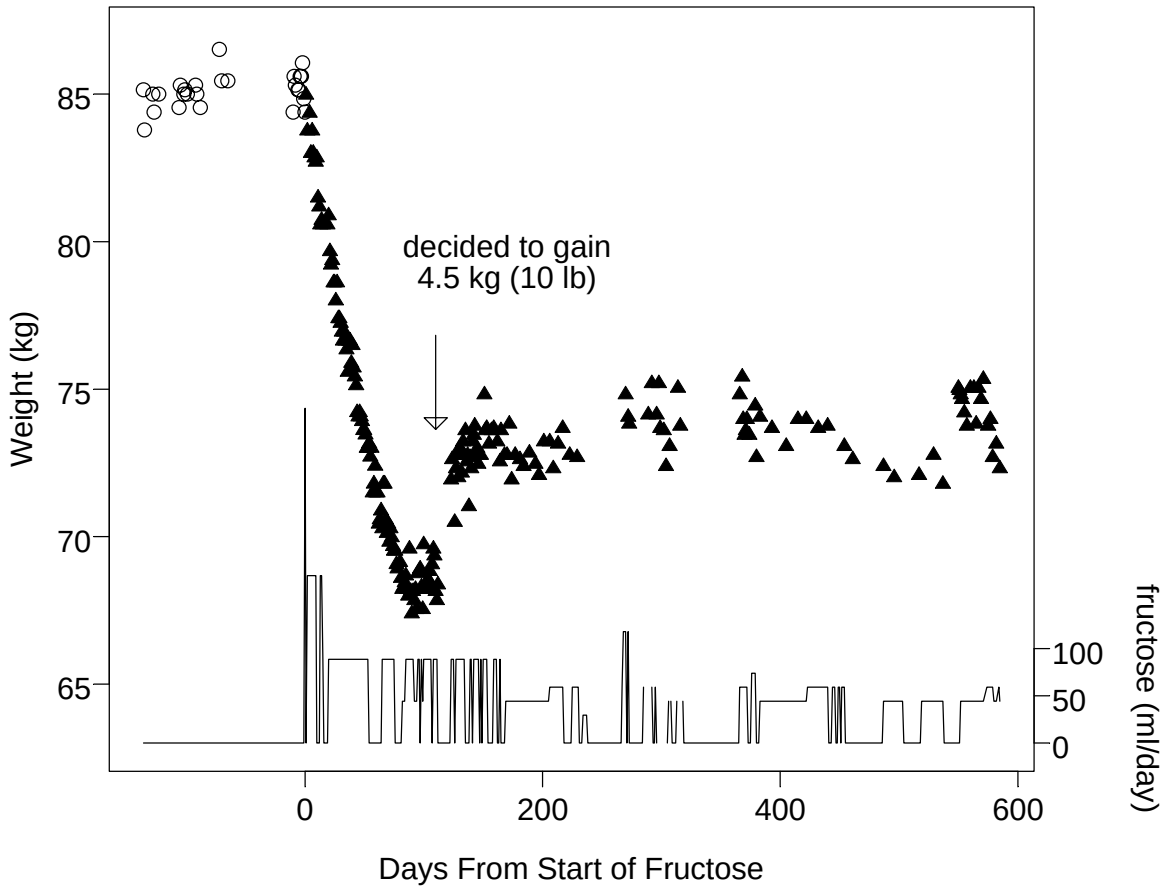

85
80
75
70
65
Weight (kg)
decided to gain
4.5 kg (10 lb)
100
50
0
fructose (ml/day)
0
200
400
600
Days From Start of Fructose

Figure 4

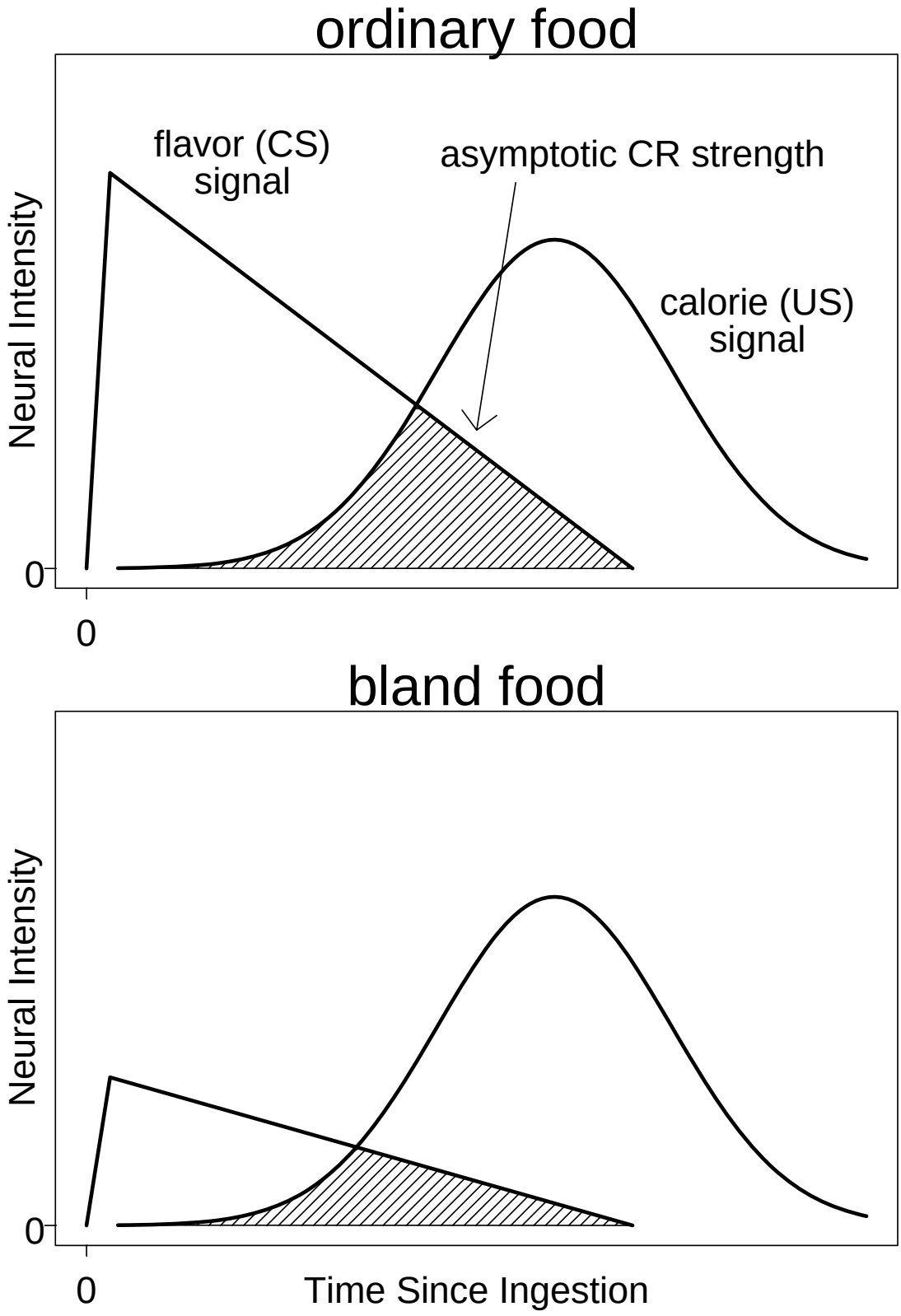
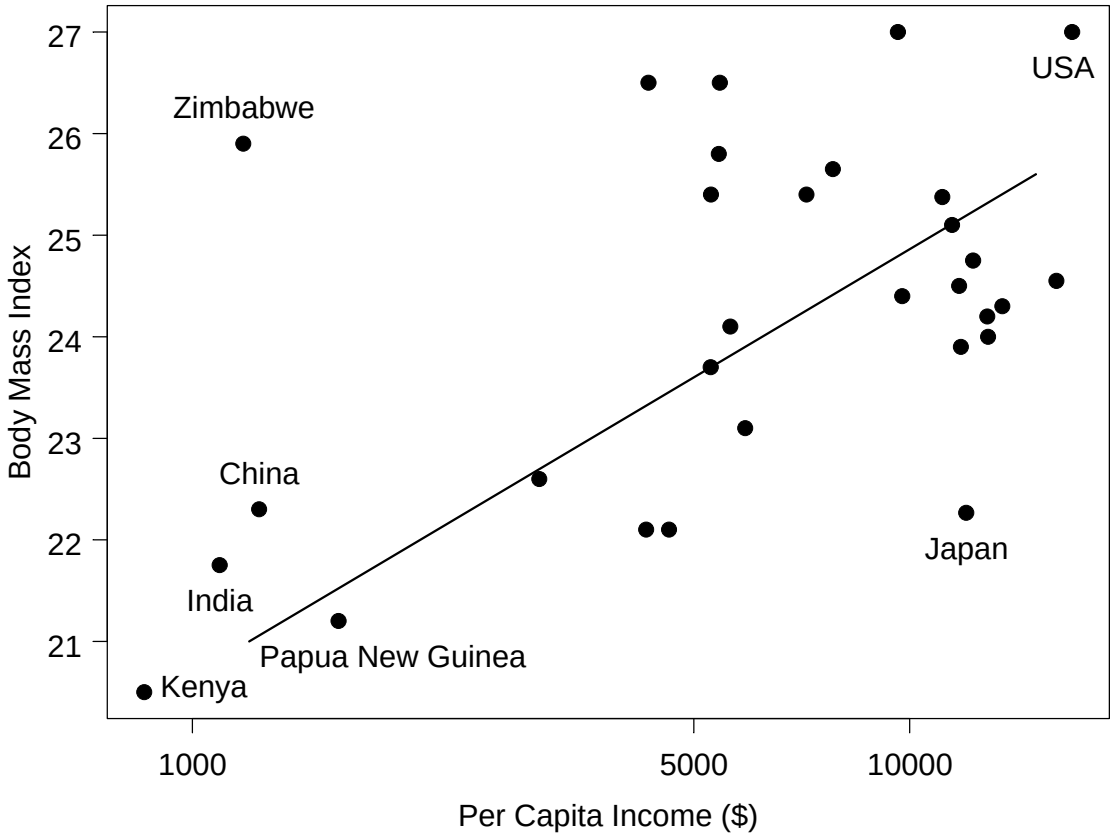

ordinary food
flavor (CS) signal
asymptotic CR strength
calorie (US) signal
Neural Intensity
0
0
bland food
Neural Intensity
0
0
Time Since Ingestion

Figure 5


27
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22
21
Body Mass Index
USA
Zimbabwe
China
India
Japan
Papua New Guinea
Kenya
1000
5000
10000
Per Capita Income ($)

Figure 6

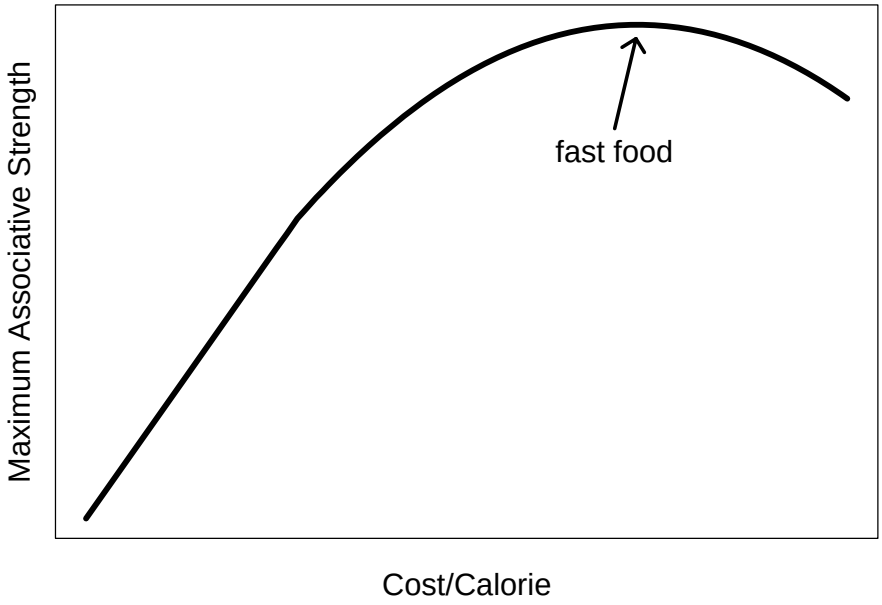

Figure 7