

An investigation of the Satiety Mechanism: A Research Initiative



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Abstract

The regulation of satiety is a complex subject beyond the reader's wildest imaginations. Multitudes of hormones such as leptin and insulin play intricate roles in its regulation. The lateral and ventromedial hypothalamic areas of the brain are but a few anatomical aspects which relay information of hunger and satiation to the body. This entry strives to take the most comprehensive analysis ever of the mechanisms which regulate satiety and how they can be manipulated for the benefit of the athlete under various scenarios.

Introduction

Meals

Meals are the basic measurement for caloric consumption due to the fact that the amount of food you consume is determined by the size and frequency of your meals. Three basic phases which affect your meals are hunger, satiation, and satiety. These aspects will be broken down subsequently.

Dual Center Hypothesis

Hunger may be defined as the sensation which drives an individual to find and consume food [1]. In 1940, Hetherington and Ranson postulated that feeding involved activation of the lateral hypothalamus (LHA). They found two areas of the hypothalamus that monitor feeding and body fat [2]. Shortly thereafter, the dual center hypothesis was invented [3]. This states that the LHA is responsible for hunger and subsequent feeding, while the ventromedial hypothalamus (VMH) is responsible for satiety and satiation. To clarify, after the ingestion of a certain amount of food, a suppression of hunger occurs that will lead to the termination of feeding. This is referred to as satiation. The feeling of fullness between meals is referred to as satiety [1].

Recent experiments have brought support to this hypothesis. For instance, studies show electrical impulses on the lateral hypothalamic area of animals results in feeding, while an impulse on the ventromedial aspect promotes satiation and eventual termination of feeding [4, 5, 6]. Moreover, it has been shown that harming the VMH leads to insulin resistance and hyperinsulinemia in animals. Further, subjects with an injured VMH eat an immense amount of excess calories, resulting in obesity [7, 8].

While the roles of the lateral and ventromedial hypothalamic areas are well established, the dual center hypothesis is simplistic to say the least. You see, there are many more mechanisms which control feeding and satiation. For instance, nuclei throughout the inferior brain stem collaborate and deliver messages to endocrine organs and forebrain structures. The midbrain and thalamus further interpret this information in accordance to the sensory properties of food, and the forebrain nuclei communicate the positive aspects of feeding. These feeding centers are informed by multitudes of hormonal and neural outputs on the metabolic stasis of the body [1, 9].

A variety of other factors such as insulin, neuropeptide Y, leptin, catecholamines, and ghrelin must be taken into account. Today we are going to discuss these factors and how you, the athlete, can properly manipulate them to achieve your goals.

Feeding Regulation

A multitude of elements can influence your caloric intake, but these can be simplified into two categories: anorexigenic and orexigenic factors. The former promotes a lack or loss of appetite for food, while the latter results in a desire for food. Some topics discussed will be a combination of both. We begin with the protein hormone leptin.

Leptin

Leptin is a protein hormone secreted from adipose tissue [10]. Since its discovery, leptin has been considered a contributor to satiety [11]. Multitudes of studies confirm this hypothesis. For instance, leptin reduced food consumption and body weight when given to rats intraperitoneally (administered by entering the peritoneum, which is the membrane that lines the cavity of the abdomen), intravenously (through veins), or intracerebrally (in the cerebrum) [12, 13, 14, 15, 16]. Furthermore, Chapelot et al. demonstrated that plasma leptin concentrations increase during spontaneous intermeal intervals, and decline before the onset of a meal, showing leptin may contribute to meal patterns [40].

Body fat is a huge factor in leptin concentrations. The amount of leptin secreted into the blood stream is largely dictated by how much fat one has. For example, leptin was measured in seventy-one obese individuals and 108 normal weighted ones [17]. Leptin in the latter group was measured at 8 ng/ml. Results showed the largest discrepancy occurred when body fat reached over 25%—increasing anywhere from three-tenfold.

Carbohydrates are also a potent leptin regulator. Insulin given to rats increases leptin gene expression, and inhibits the reduction in leptin mRNA caused by calorie restriction and fasting (i.e. 36 hours) [19, 20]. Similar effects have also been

reported in humans [21, 22]. Glucose metabolism has been shown to be an important factor in leptin utilization; this may be why insulin stimulates leptin secretion [42].



To add to the comment on calorie restriction, a decrease of 10% in body weight has been shown to decrease leptin by 53% in some cases [29]. Moreover, a decrease in leptin during starvation diets will promote energy conservation by decreasing thyroid hormone-induced thermogenesis (one way is by decreasing NPY, discussed later) and increasing glucocorticoids that mobilize energy stores [1]. Brent et al. tested the interactions between energy intake and fat loss on plasma leptin during prolonged, moderate and severe energy restriction [43]. Mean leptin decreased markedly by up to 66% ($P < 0.001$) at week one of energy restriction, and then gradually thereafter. Additionally, Keim, Stern, and Havel tested the effect on women of a chronic energy deficit on plasma leptin concentrations and self-reported appetite to explore possible relations between leptin and appetite sensations [44]. Twelve healthy women participated in a three-week study of neutral energy balance, followed by 12 weeks of energy deficit (feeding reduced by 2 MJ/d and energy expenditure increased by 0.8 MJ/d). Leptin diminished by 54% after 1 week of a moderate energy deficit and remained low after 6 and 12 weeks. Leptin was associated with self-reported hunger, desire to eat, and prospective consumption; the largest increase in hunger was associated with the sharpest decline in leptin. Moreover, leptin and hunger were not influenced by the amount of weight and body fat lost. Thus, fasting and very low-calorie diets lead to a decline in leptin production.

Romon et al. sought out to test whether the macronutrient content of the meal could influence postprandial (after eating) leptin response, and if leptin levels were associated with postprandial satiety, hunger, and subsequent food intake [31]. They used 22 healthy subjects (half male and half female) and had them eat a diet consisting of carbohydrates (81%) or fat (79%), with about the same amount of

protein in each, while the third group fasted. In both genders, leptin response was higher after the carbohydrate meal than after the fat meal or while fasting. Leptin response was significantly correlated to insulin response ($r = 0.51$, $P < 0.0001$). Leptin started to rapidly rise 4-5 hours after a meal; however, leptin had no acute effect on hunger. This was expected, though. Results show higher leptin concentrations results in long-term satiety rather than short. For example, in rodents, food intake was reduced after 4 hours of leptin administration [32]. While in monkeys, leptin elicited its anorexic effects on the second day of injection [33].

An important mechanism by which leptin works is inhibiting neuropeptide Y (NPY, which will be discussed in-depth further on; just understand now that it is a major orexigenic peptide) synthesis and release [23]. Cusin et al. demonstrated that injecting leptin in lean rats diminished NPY synthesis in sites of production in the hypothalamus [24]. Many other studies also testify to this [25, 26].

However, this clearly is not the only means by which leptin works. Erickson, Clegg, and Palmiter demonstrated this by injecting leptin into mutant mice deficient in NPY. Five days of leptin administration significantly reduced their food intake, body weight, and adipose tissue mass, showing that leptin can increase satiety by actions independent of NPY [27].

Further research shows that glucocorticoids negatively affect leptin. For instance, Zakrzewska et al. injected 3 μg of leptin into three groups: normal rats, adrenalectomized rats (surgical removal of an adrenal gland, this gland secretes glucocorticoids, the most abundant in the body being cortisol), and adrenalectomized rats with the added supplementation of glucocorticoids [28]. The first group showed a small reduction in body weight and food consumption, the second group had very strong and long-lasting reduction in food and body weight, while supplementation with glucocorticoids in the third group inhibited leptin's effects. This may explain why people with Addison's disease—a rare endocrine disease that results from the underproduction of aldosterone and cortisol by the adrenal glands—are usually hypophagic (under eat, anorexic). Leptin may also exert its effects via the central nervous system [41]. For more on glucocorticoids, refer to [Endocrine Insanity Part I](#).

Leptin also acts synergistically with cholecystokinin (CCK, discussed in-depth further on) to promote satiety [35, 36, 37, 38]. To investigate the physiological relevance of this observation, Julie et al. injected saline intraperitoneally (IP, near abdominal organs) or CCK into 48-h-fasted or fed rats [39]. They hypothesized that leptin deficiency, induced by fasting, weakened the satiety response to CCK. Fasting blunted the satiety response to 3.0 $\mu\text{g}/\text{kg}$ of CCK, such that 30-min food intake was suppressed by 65.1% (relative to saline-treated controls) in fasted rats vs. 85.9% in the fed state ($P < 0.05$). They also tested how NPY injections would affect CCK, and found that this further attenuated its effects, further supporting the hypothesis leptin deficiency, weakens the satiety response of CCK, as NPY is higher when leptin is lower. There results were consistent with other experiments that leptin does assist CCK.

A very fascinating study reported that leptin in humans is secreted by circadian rhythms [30]. Madhur et al. tested the 24-h profiles of circulating leptin levels in three groups: obese, obese people with non-insulin-dependent diabetes mellitus (NIDDM), and lean individuals. In all the three groups, serum leptin levels were highest between midnight and early morning hours, and lowest around noon to

mid-afternoon. They postulate that this inherent mechanism could be to suppress appetite during the night when fasting. Now, while your night and early morning leptin levels are higher, this can be manipulated according to your diet. Havel, Townsend, and Teff portrayed that the mean daily levels as well as the nocturnal (night) concentrations rise higher after high-carbohydrate/low-fat meals than after low-carbohydrate/high-fat meals [34]. For an in-depth analysis on the circadian rhythm, see Knowlden's (2002, 2003) sleeping articles in the anatomy section under 'hormones'.

The study of leptin is quite fascinating. Look for a full article on its many actions in the future. If you want to learn more about leptin, refer to [Metabolic Primer Part II](#).

CCK

CCK is the most investigated satiety signal. In 1973, Gibbs et al. gave purified or synthetic CCK to rats before a meal and observed that it dose-dependently reduced the size of the meal [48]. Since then, hundreds of experiments have demonstrated CCK's ability to reduce meal size, including in humans [48, 49, 50, 51]. Moreover, further evidence to this was given when many scientists observed that injecting CCK-1 receptor antagonists before a meal causes hyperphagia (over eating) in animals and humans [52, 53]. Additionally, older people commonly eat less than younger individuals. To test what mechanism(s) produce(s) this anorexic effect, MacIntosh et al. examined eight healthy, old (65–80 y) and seven young (20–34 y) men after administering lipids and glucose for 120 min on separate days [78]. Plasma CCK, glucagon-like peptide 1 (GLP-1), and peptide YY (PYY) concentrations were measured. CCK concentrations were significantly higher in older than in younger subjects, while Plasma GLP-1 and PYY concentrations were not significantly different between groups.

A popular suggestion on how CCK works is that when stimulated, it acts in a local paracrine manner to stimulate CCK1 receptors on the sensory fibers of the vagus, making the nerves sensitive to CCK and other stimuli such as gastric distension, and further promotes slow gastric emptying [55]. This is significant because stomach sensory information is brought to the brain by these nerves, so making them more sensitive would promote satiation. Results show that cutting vagus nerves makes CCK worthless at reducing meal size, strongly supporting this hypothesis [54]. Additionally, gastric distension is a potent signal in satiety. For example, Geliebter, Westreich, and Gage inserted a balloon into four lean and obese individuals [113]. They then filled the balloons with 0-800 ml of water. The results showed that as volume increased, food intake decreased.

Therefore, CCK relaxes the stomach and makes you very sensitive to gastric pressure, promoting satiation [65]. Furthermore, CCK stimulates pancreatic enzyme secretion [74], promotes gall bladder contractions [75], constricts the pylorus [76], and by inhibiting gastric emptying, promotes gastric distension [77].

CCK may also act as a neurotransmitter or neuromodulator within the brain to inhibit gastric emptying. For example, injection of CCK into the hypothalamic and nucleus of the solitary tract have been observed to significantly inhibit gastric emptying [66, 67, 68]. Further, Roger et al. demonstrated that endogenous CCK may enter the bloodstream to inhibit gastric emptying by an endocrine mechanism.

Moran McHugh tested for a mechanism by which CCK promotes satiety [46]. They injected CCK into their participants and tested for the rate of gastric emptying. Gastric emptying was inhibited by this hormone. The onset of the inhibition is rapid, and its effect brief. They concluded that CCK can be thought of as a link in a chain of physiological elements—the satiety effect depends upon inhibition of gastric emptying, which then leads to gastric distention with increased food consumption. Additionally, CCK has a short half-life (1-2 minutes). Because of this, injecting CCK 15 minutes before a meal is ineffective at promoting satiation [56].

Scientists have studied extensively whether CCK would be an effective supplement for reducing meal size in humans. The results are disappointing. When CCK is given continuously, it quickly becomes ineffective [58]. Moreover, when CCK is given before every meal in rats for a short while, it is effective at reducing meal size, but the animals compensate by eating more food over the next several days [57]. Thus far, CCK does not appear to be an effective supplement, but rather must be manipulated through diet. However, this also has its problems.

Studies show that humans adapt very quickly to high fat diets (HFD), which subsequently decreases the satiating response to nutrients. For instance, Cunningham et al. showed that consumption of a high-fat diet for two weeks led to an acceleration in the gastric emptying rate of high-fat test meals [59]. However, CCK is still raised very high during HFD, making these results rather strange [60]. To test the mechanisms by which this adaptation occurs, Covasa and Ritter injected CCK into rats on low fat diets and high fat diets [61]. The former group had a much slower rate of gastric emptying (26.2-55.1%) than the later group (10.0-31.7%). This shows that HFD may cause subjects to be insensitive to CCK's satiating effects.

To further investigate this, French et al. had 12 male subjects consume a high-fat diet (58% energy) for two weeks, testing levels of cholecystokinin (CCK), food intake, and subjective feelings of hunger and fullness [62]. The results showed a significant enlargement in the average daily food consumption, increasing feelings of hunger and declining fullness. And again, CCK was substantially higher, further supporting the hypothesis that high-fat diets reduce the body's sensitivity to this hormone.

Castiglione, Read, and French sought to test whether this effect on gastric emptying was nutrient-specific [63]. Studies were carried out on eight healthy, free-living male volunteers between the ages of 19 and 26. Their original fat intake was between 30-40%. In the test they increased this to 55% for 14 days. They then gave them high-fat and high-carbohydrate meals. The high carbohydrate meals had nearly the same rate of gastric emptying before and after the experiment. However, the rate of lipid emptying was much faster, consistent with the previous experiments. This shows HFD adaptations are nutrient-specific to fats, and not to carbohydrates.

Now, most athletes never would have this much fat in their diet. However, this does show the folly in excessively high-fat/low-carb diets, which not surprisingly often advocate the wholly ignorant, logically invalid, and completely unscientific protocol of fat and fiber post-workout. Moreover, you can see that people who eat junk food constantly (i.e. pizza) are going to be much more susceptible to continued binges than someone on a lower fat diet. To exemplify the harm in this adaptation, rats fed the same amount of fat at a slow rate consumed less energy per day, had longer between-meal intervals, and gained less weight over a two-week period than after

infusion of fat at a more rapid rate [125]. So, those reading need to take a close look at their diet, and be sure they are not consuming excessive amounts of fat right now.

As displayed above, the greatest stimulator of CCK is fat [47]. Carbohydrates are very poor CCK producers, while proteins, on the other hand, may be potent CCK manufacturers. For example, Forster and Dockray gave rats a liquid test meal of peptone. The results showed delayed gastric emptying and a short elevated response in CCK, lasting ten min or less [69]. Further, Sharara et al. administered proteins, protein hydrolysates, amino acids, glucose, and starch to several subjects [70]. The results showed intact proteins were the only nutrients to stimulate CCK release. It is postulated that protein stimulates CCK by reducing trypsin degradation of CCK releasing factors in the intestinal lumen [71]. This is significant because administration of trypsin antagonists elevates plasma CCK levels [72]. To test this hypothesis, Woltman and Reidelberger composed a rather genius experiment [73]. Using non-fasted rats, they gave peptone alone, and peptone with Devazepide, which is a CCK antagonist. If CCK does assist proteins satiating effects, then devazepide should significantly attenuate peptone-induced anorexia. The results showed peptone by itself decreased food intake by 18-96%, while devazepide with peptone decreased these effects by 29-65%. This strongly suggests that CCK plays a major role in the satiety response to duodenal delivery of protein.

Glucostatic Theory

In the 1950s, Jean Mayer composed various experiments on rats and mice that lead to the glucostatic theory [87]. Studies in the '60s and '70s by Steffens et al. showed that glucose is low at the onset of a meal, and rises at meal termination [88, 89]. And further experiments have given great establishment to this theory.

The theory states that glucose is monitored by the central nervous system [82]. Evidence shows that glucoreceptors and glucosensitive neurons in the hypothalamus do indeed perform this task [83]. For example, Himmi, Boyer, and Orsini simultaneously monitored blood glucose level and forebrain unit activity in rats [84]. They injected glucose or phlorizin into them and observed transient fluctuations in glycemia, occurring either spontaneously or after the injections. They noted spiked frequencies of more than one-third of the neurons tested in the lateral hypothalamic area in response to the fluctuations. Most of the cells were activated during hypoglycemia and depressed during hyperglycemia. They concluded that, "These neurons might mediate the effects of a drop in blood glucose on either meal initiation or neuroendocrine or autonomic events related to nutritional functions."

Several studies have also shown that the onset of a meal is preceded by low blood sugar. Sylvestre and Magnen monitored blood glucose levels in free-feeding rats for several hours and found that there was a 6 to 8% fall in blood sugar, starting 5-6 min prior to meal onset, before every meal from day to night [85]. Furthermore, Campfield, Smith, Rosenbaum, and Hirsch sought to test whether changes of hunger ratings in humans were related to spontaneous changes in blood glucose concentration [86]. In 83% of the subjects, both the perception and behavioral expression of hunger were preceded by and correlated with brief, transient declines in blood glucose (10% at 27 min). They also are performing an ongoing study by injecting insulin into subjects (which is a hypoglycemic producing hormone). Early results in five subjects showed that hunger increased after insulin-induced transient

declines in blood glucose, but no change in hunger occurs when blood glucose concentration are stable.

With this in mind, several factors must be taken into account. First, we must discuss insulin, which helps regulate blood glucose. Studies demonstrate that it has both orexigenic and anorexic effects.

Insulin has contradicting results. Some studies show it can increase hunger in animals and humans [93, 94]. But this is likely because of its hypoglycemic qualities. Others show it promotes satiety. Brief and Davis examined the effect of chronic infusions of insulin in one of three doses (5, 7.5 or 10 mU/day) on food and water intake in rats. All groups treated with insulin decreased food intake during the day and night, and 10 mU/day produced a significantly greater reduction in water intake than each of the other solutions [79]. Similar results were also found in baboons [80, 81]. Additionally, if insulin or leptin levels are increased within the brain, animals eat less food and lose weight; on the other hand, if the normal leptin or insulin signals within the brain are reduced, animals overeat and become obese [101]. Overall, if used properly, insulin acts as a major anorexic hormone. Additionally, insulin augments the satiating effects of CCK [143]. Lastly, one of its most potent actions is inhibition of NPY/AgRP and stimulation of a-MSH/CART neurons [148]. This will be discussed further on.

Having a high-GI carb such as glucose has also been shown to have a greater short-term satiety than having a slow-burning carb such as oatmeal. This will be explained later.

It is worthy to note that high blood sugar promotes satiety. Studies show hyperglycemia slows gastric emptying [96], increases proximal gastric compliance [97], attenuates gallbladder contraction [98], and prolongs small intestinal transit time [99], among other things [100]. This would contribute to the above results.

The problem with having high-GI carbs (concerning hunger) is that you will promote hyperglycemia, and high blood sugar induces an elevated concentration of insulin. The trouble with having high bursts of insulin is that it acts as a hypoglycemic hormone (one that decreases blood sugar). As displayed above, hypoglycemia induces hunger. So for short term satiety, having pure glucose, such as a post-workout shake, will have a more potent short-term satiety compared to complex carb. However, in the long run, you want normal blood glucose levels. How to manipulate slow-and fast-burning carbs for bulking and cutting cycles will be discussed further on under practical applications.



NPY

The most potent bodily orexigenic substance known to man is Neuropeptide Y (NPY) [1]. NPY is a major brain peptide located in the hypothalamic arcuate nucleus (ARC) that projects to the paraventricular nuclei (PVN) and dorsomedial nuclei (DMH), and is postulated to control energy balance by stimulating feeding and inhibiting thermogenesis, especially under conditions of energy deficit [1].

NPY levels rise in almost every situation of hunger. This includes fasting and hypoglycemia. For example, Vettor et al. administered NPY to normal rats for 7 days [103]. The result was a sustained threefold increase in food intake and an increase in body weight over 40 g.

In order to find out if central injection NPY would alter brown fat thermogenesis and white fat lipoprotein lipase activity, Billington et al. injected NPY into three groups of rats: 1) NPY (5 micrograms/injection) and ad libitum food; 2) NPY (5 micrograms/injection) and food restricted to control intake; 3) saline injection and ad libitum food [102]. The first group ate much more food than the latter two, and there was an increase in white fat lipid storage and a decrease in brown fat thermogenesis in both NPY groups.

Now, as stated above, by acting in the brain, insulin suppresses food intake, whereas NPY has the opposite effect. Fasting increases NPY levels, while lowering insulin levels. Therefore, Schwartz et al. hypothesized that the anorexic effect of insulin could result from an insulinogenic inhibition of NPY gene transcription [104]. To test this, they injected insulin into rats after they fasted for 48 hours. The results showed insulin significantly suppresses the expression of mRNA for NPY in lean rats, strongly supporting their hypothesis and showing that fasting increases NPY synthesis dependent on low insulin levels.

In another experiment, Zarjevski et al. chronically administered 10 micrograms of NPY per day to female rats [105]. This resulted in hyperphagia, increased basal insulinemia, as well as liver and adipose tissue lipogenic (fat-building) activity.

To further demonstrate NPY's effects, Sainsbury et al. injected NPY into normal rats for 6 days. This resulted in hyperphagia, increased body weight gain, hyperinsulinemia, hypercorticonemia, and hypertriglyceridemia (elevated triglyceride levels), compared to control rats [106]. NPY infusion also resulted in an insulin-resistant state in muscles and in a state of insulin hyperresponsiveness in white adipose tissue, as assessed by the measurement of the in vivo (within a living body) glucose utilization index of these tissues.

What is interesting to note is that the above side-effects were entirely prevented when rats were adrenalectomized (surgical removal of an adrenal gland, which secretes glucocorticoids, the most abundant in the body being cortisol) before NPY administration. Also, levels of mRNA for leptin were increased in white adipose tissue after 6 days of NPY infusion in normal rats (due to hyperphagia), and white adipose tissue weight was also increased (also due to hyperphagia). They concluded that, "intact adrenal glands, and probably circulating corticosterone (a glucocorticoid), in particular, are necessary for the establishment of most of the hormonal and metabolic effects induced by chronic...infusion of NPY in normal rats."

You should now further understand the importance of leptin in hunger regulation. Leptin's ability to suppress NPY is a powerful satiety weapon.

Further, you can see why people on low-carb diets are always hungry. Without carbs to facilitate a steady flow of insulin, not to mention blood sugar, the subjects that participate in this are bound to be hungry and have a multitude of carbohydrate cravings.

Feed Back Loop

The feed back loop involves leptin, NPY, and insulin in relation to satiety. First, fasting or strict dieting results in a decrease in leptin. Low concentrations of leptin then result in an increase in NPY synthesis, which promotes feeding. After feeding, if the food contains carbohydrates, insulin rises, which subsequently decreases NPY production, resulting in satiation and eventual termination of feeding. Moreover, as stated above, insulin also increases leptin secretion from adipose tissue, further promoting long-term satiety and decreased NPY concentrations. [18].

Ghrelin

Ghrelin (named after Proto-Indo-European roots "ghre" for grow and "relin" for release) is one of the few, if not the only, signal(s) in the gastrointestinal tract (GI tract) that stimulates hunger. It is a 28-amino acid peptide that rises during prolonged fasts and promotes feeding [108]. Ghrelin is also produced in the brain, and there is some evidence that ghrelin signals are carried by vagal afferent nerves to the brain [111]. Additionally, ghrelin is a strong growth hormone producer [112].

A logical hypothesis is that ghrelin may contribute to the onset of a meal. To test this, David et al. monitored 10 healthy subjects for 24 hours, taking samples of Ghrelin 38 times throughout the day [107]. Plasma ghrelin increased nearly twofold before the onset of a meal, and fell back to normal levels 1 hour afterward. These results bring strong evidence to ghrelin's physiological role in meal initiation for humans.

Wren et al. investigated the effects of ghrelin (5.0 pmol/kg/min) or saline infusion on hunger and food intake in a randomized double-blind cross-over study in nine healthy volunteers [109]. The results showed a statistically significant increase in energy consumed by the participants. Wren et al. again composed an experiment on ghrelin and its orexigenic effects, this time on rats [110]. He injected ghrelin at various concentrations to rats, and observed hyperphagia and subsequent obesity in the tested rats.

Ghrelin partially exerts its effects by speeding gastric emptying [111]. Additionally, in rodents injected with ghrelin, there is an increase in NPY and AgRP mRNA expression [111].

This peptide is very new, however, and much research still needs to be done. Nevertheless, ghrelin seems to be a key player in hunger regulation.

Posture

This next section is quite novel, to say the least. It appears that posture can have a profound effect on digestion. For example, Theresa et al. composed an experiment to test posture and meal structure on gastric emptying and satiety [114]. Nine women ingested tomato soup and then immediately or 20 min later an egg sandwich, when seated or when supine (lying on their backs). The results showed the half-emptying rate of the sandwich was 32% longer and the emptying rate after lag phase was almost 39% slower for the subjects who were supine compared to those who were seated upright. In those who consumed the soup immediately before the sandwich, the half-emptying time of the soup was 50% longer. In subjects who waited 20 minutes to consume the sandwich, however, the after-eating satiety lasted a bit longer, but in those that ate both foods immediately, perception of fullness immediately after the meal was higher. Either way, supine is better than seated upright for slowing gastric emptying and enhancing satiety.

Additionally, Murdoch, Fisher, and Hunt tested subjects drinking a saline (salty water) drink sitting, lying on the left side, or lying on the right side [115]. At 10 minutes after ingestion of 750 ml., there was 215 ml. left from subjects lying on the right side, 431 ml. left from subjects lying on the left side ($P < 0.005$), and 308 ml. left from the subjects sitting erect. So lying on the left side is much more effective than sitting erect, and sitting erect is more effect than lying on the right side. However, in a similar experiment with 750 ml. of water with glucose monohydrate, there was no statistical difference between groups. Another study with a low-nutrient soup ingested with olive oil showed emptying occurs more slowly, and hunger was reduced when subjects were lying on the left side than when they are seated [116]. These authors propose that that gravity slows emptying when subjects are on the left side or supine because of the anterior position of the antrum and pylorus relative to the body of the stomach. They also suggested that nutrients such as glucose, which strongly activate intestinal receptors, offset any slowing of gastric emptying by gravity.

Posture has little effect on digestion when solid foods are digested without liquids [117].

Fiber

Dietary fiber is poorly digested; this results in an accumulation of material in the small intestine and subsequent delay in nutrient absorption [119]. Howarth, Saltzman, and Roberts demonstrated that an additional 14 grams of fiber results in a 10% decrease in caloric intake [120].

Fiber also enhances CCK. Having fat and fiber slows the disappearance of lipids from the small intestine, increasing the release of CCK [122]. The addition of barley to a low-fat meal has been shown to have similar effects [123], and having beans as a source of fiber doubles the body's response to cholecystokinin [124].

Many other studies show fiber will enhance insulin sensitivity, increase adsorption, delay small intestine transit time, slow gastric emptying, promote viscosity, and much more [126]. The subject of fiber has been covered in previous issues of JHR. For more on this subject, read [Fiber Dynamics Part I](#) [Fiber Dynamics Part II](#). Also, if you are just starting to supplement with fiber, increase it slowly to avoid gastrointestinal distress [121].

Energy (Calorie) Density

Here is a quote from the current writer to introduce this term [127]:

“Q. What is calorie density?

A. Calorie density (CD) is the number of calories per weight of food. A perfect illustration is found when comparing proteins, carbohydrates, and fats. Typically, proteins and carbs contain 4 calories per gram, while fats have 9 calories per gram. Fats would be said to have a higher calorie density than the former two.”

Many scientists would claim that energy density is the most potent weapon one has for manipulating satiety [121]. This topic has been highly investigated by nutritionists, and results support the claims made. For example, Duncan, Bacon, and Weinsier, over 5 days, gave 20 obese and non-obese subjects a diet low in energy-dense foods and one high in energy-dense foods and then allowed each group to eat until satisfied [128]. The subjects which ate as many low-energy dense foods as they wanted had 1,570 calories, while the subjects which ate high-energy dense foods had 3000. Furthermore, the former group ate 33% longer throughout the day than the latter group.

Water content also effects energy density and can influence hunger. For example, Bell et al. gave subjects a milk-based drink or no drink (control), followed 30 min later by a self-selected lunch, and 4 hours later by a self-selected dinner [129]. The milk drinks were equal in energy and macronutrient content; the only difference was they added water to increase the density to 300, 450, and 600 ml. The participants significantly reduced subsequent intake after the 600 ml milk drink compared to the 300 and 450 ml drinks, showing that adding water to food can be of great value to a dieting individual.

Now, the question remains of whether having water on the side with solid food effects hunger equally to mixing it with a solid food or energy-dense liquid. To test this, Barbara and company gave their subjects 1 of 3 isoenergetic (1128 kJ) preloads 17 min before lunch on 3 days and no preload on 1 day [131]. The preloads consisted of 1) chicken rice casserole, 2) chicken rice casserole served with a glass of water, and 3) chicken rice soup. The soup contained the same ingredients (type and amount) as the casserole that was served with water. Results showed that decreasing the energy density and increasing the volume of the preload by adding water to it significantly increased fullness, reduced hunger, and subsequent energy intake at lunch (26% less kcals were consumed). The equivalent amount of water served as a beverage with a food did not affect satiety. So according to this study, mixing food with water, such as a protein shake, will affect satiety, but having a glass of water and some steak, for example, will have very little additional benefit to hunger than having the steak by itself. Adding water on the side with food has conflicting results, however. For instance, women served breakfast with or without two glasses of water showed that consumption of the water decreased hunger and increased satiety during breakfast, but this effect did not extend beyond the meal [132]. In conclusion, mixing water with food is very well-established for decreasing hunger. Having it on the side is not, but it certainly would not hurt to have some, not to mention the anabolic effects of staying hydrated. The authors postulated that the results could be that water in food increases the weight or volume of the food and

changes the dispersion of nutrients consumed, probably activating mechanisms involved with hunger. On the other hand, water consumed on the side would be processed by thirst mechanisms, which are distinct from those for hunger [131].

Additionally, adding fiber to meals (low-calorie, lowering energy-density) significantly decreased caloric intake in lean women [130].

It has been suggested that density could affect satiety through mechanoreceptors (relay mechanical stimuli information in the nervous system, such as hair cells, which help hearing) or chemoreceptors (detect chemicals and relay information in the nervous system, such as taste) in the oropharyngeal (around the throat) or gastrointestinal tracts. Additionally, the volume of food could influence satiety by affecting the perception of how much has been consumed. People may equate portion size with energy content and adjust subsequent intake accordingly [133].

Because of these results, it has been postulated that carbs and proteins have higher satiating effects than fats, and the results do indeed support this. We will be discussing this further on in the article.

Volume

As discussed previously, energy density (kJ/g) of foods strongly affects satiety. However, the question still remains whether increasing the volume or size of a food, independent of weight, affects hunger. To test this, Rolls, Bell, Bethany, and Waugh fed 28 lean men breakfast, lunch, and dinner in a laboratory 1 d/wk for 4 weeks [118]. They gave them isoenergetic (equal amount of energy), yogurt-based milk shakes that varied only in volume (300, 450, and 600 ml) as a result of the incorporation of different amounts of air. The food contained identical ingredients and weighed the same. The high volume drink significantly affected energy consumption at lunch. Energy intake was approximately 12% lower after the 600 ml drink than after the 300 ml liquid. Subjects also reported greater reductions in hunger and increases in fullness after consumption of both the 450 and 600 ml drinks than after the 300 ml ones. Therefore, varying the volume (irrelevant to weight) does affect satiety and digestion.

This experiment should have profound effects on the nutrition industry. This means that foods such as popcorn, which are very light but puffy because of air volume, have higher satiety than foods with the same weight and energy but less volume. Designing foods with high air volumes would therefore assist a great many of dieting customers; likewise, when dieting, consuming high-volume foods would be of benefit to the athlete.

Exercise

Training can have varying results on hunger. Initially, exercise produces an anorexic effect. Moreover, the higher the intensity, the more pronounced this effect is. In male rats, intense exercise suppressed more food and caused more weight loss than less-intense exercise [134]. In humans, results are very similar [135]. For example, King, Burley, and Blundell collected 23 healthy, lean, male college student/staff members and randomly assigned them to a control, low-intensity and high-intensity exercise treatment in the first study, and to a control, short-duration and long-

duration exercise treatment (high intensity) in the second [136]. Hunger was significantly suppressed during and after intense exercise sessions, and more so than low-intensity workouts. This was a very short-term influence, however. Exercise had no effect on the total amount of food consumed, but it did delay the start of eating during the first meal. In studies in which a test meal was offered 50-75 min after exercise, appetite is not suppressed [137]. So results are consistent that exercise promotes an initial anorexic effect, but this is short-lived, and appetite resumes as normal thereafter.

Mechanisms for this short-term anorexic effect are not completely understood; however, some authors postulate that hormones such as cortisol, catecholamines, and the adrenocorticotrophic hormone (ACTH, which stimulates the release of glucocorticoids) could cause this [138, 139]. Perhaps the best known mechanism is CCK. Cholecystokinin levels quickly rise during intense exercise, and since its anorexic effects are very short-lived, this would fit rather nicely with the above observations [140]. Additionally, hypoxia (reduction of oxygen supply to tissue below physiological levels) promotes a potent anorexic effect [141].

Training's long-term influence on hunger is a different subject. One theory—the glycogenostatic hypothesis—states that individuals consume food to a level that maintains glycogen levels in the body. Results show, however, that glycogen stores themselves only have a minor impact on hunger [142]. Conversely, glycogen depletion may indirectly promote hunger, causing disruptions to occur in the relationship to patterns of blood glucose and spontaneous meal initiation. For example, Kathleen et al. had 10 men (age 20-31 yr) perform glycogen-depleting exercise in the evening, eat a low-carbohydrate dinner, and stay overnight in the laboratory. The next day, blood glucose was monitored continuously. Subjects had access to high-fat and high-carbohydrate foods after baseline glucose and respiratory quotient were determined. Lastly, in the afternoon, 1 h of moderate-intensity exercise was performed. What was interesting is that, in a state of glycogen depletion, the subjects had blood glucose stability for two meals. They consumed four high-carbohydrate sandwiches and 350 ml of a high carbohydrate beverage. After this, the normal fall in blood glucose before the onset of a meal and elevation afterwards returned to normal. In total, 8 of 10 meals were initiated during instability in blood glucose, which is very statistically significant. It is postulated that the reason blood glucose did not decline before meal initiation in a glycogen-depleted state was because the liver switched from retaining glucose to releasing glucose, preserving blood levels. This decline in liver glycogen, however, may be detected by peripheral and central nervous system glucoreceptive elements, and mapped into meal initiation as stated earlier. Initially low rates of glucose utilization could have been due to high free fatty acid concentrations, and low insulin concentrations.

Lastly, a negative energy balance caused by exercise, a decreased energy intake, or a combination of both, suppresses nocturnal leptin secretion. While a positive energy balance, enhances leptin. This would certainly effect satiety.

Cravings

Some unconditioned, innate senses of taste humans have are preferences for sweets, avoidance of bitter foods, and a salt appetite [144]. Furthermore, people tend to over eat sweet and calorie-dense foods, while under eating unpalatable foods [147]. Other foods are learned and selected according to experience. Four factors

which affect food intake are: olfactory (of, relating to or connected with the sense of smell), orosensory (relating to or associated with eating or the sense of taste, texture), sight, and postingestive stimuli (the effects of foods after ingested). If something smells good, looks good, and tastes good, you are more likely to eat it. From this, you can learn to crave certain foods, and likewise learn to avoid certain foods which do not taste, smell, or look good.

Moreover, hunger for certain macronutrients may be mediated by neurotransmitters/modulators. For example, Barton, York, and Bray injected galanin into the lateral cerebral ventricle of rats, and saline into a control group. The galanin group consumed a very high fat diet in response to this. Similar experiments show NPY promotes carb consumption, and serotonin both proteins and carbs [145]. This theory still needs more research, however, and results vary. Nevertheless, there is a strong possibility that being deficient in any macro will promote cravings in that particular macro. Moreover, being deficient in a macro, such as carbs, will induce NPY synthesis, which will further promote strong urges to eat. The craving for carbs in this case may be a learned one, as eating them will decrease NPY mRNA, and subsequently relieve your hunger pangs. Another example would be blood sugar; this is most likely a learned desire from experience, to ingest carbs in response to low blood sugar.



Postingestive stimuli are powerful factors. If you eat a certain food and it gives you food poisoning for a night, you may avoid that food for a long time, even though it may have been a freak accident. Perhaps the most important postingestive factor for bodybuilders though, is an analysis of their ABC (antecedent, behavioral, consequences). For an explanation on this term, read the introduction to [Glutamine: the Conditionally Essential Amino Acid](#). The antecedent for a bowl of ice cream is mixed: it tastes good, but it will also destroy my gains. The behavior can vary because of this. If on a diet, an athlete may break down and go on an ice cream binge; the consequence would be gaining fat, and depression because he/she cheated, etc. For the most part, a hardcore athlete will stick with the latter antecedent, and realize that ice cream is an enemy to his/her body. In fact, many athletes claim to hate greasy, cheat foods after avoiding

them for years, so this makes it easy for them. With this in mind, the athlete will avoid eating junk food, and reap the gains of a strict diet.

Another example may be oatmeal. To most normal people, this food tastes disgusting, but it is a staple in any bodybuilder's diet. Many athletes now actually enjoy the taste of a nice bowl of oatmeal, and even prepare special dishes with it, such as oatmeal mixed with cottage cheese. The athlete knows that this food is great, and may learn to like it (antecedent); furthermore, they will behave by ingesting it frequently, and the results will be very rewarding.

Focus

Cognitive (thinking) distractions have the ability to increase meal intake; likewise, eliminating distractions can cause the meal to be more satiating. Bellisle and Dalix demonstrated this on a group of healthy women [149]. They separated them into several conditions: condition 1, subjects ate alone (baseline); condition 2, subjects ate alone while listening to recorded instructions focusing on the sensory characteristics of the foods (attention); condition 3, subjects ate alone while listening to a recorded detective story (distraction); and condition 4, a group of 4 subjects had lunch together. The same food was presented to all parties. Meal size was significantly higher in the distraction condition than at baseline (mean difference from baseline: 301 ± 26 kJ). The group eating together and attention group had only a few increases in meal size. Listening to something distracting or watching something that catches your attention—such as pumping iron—can therefore help you finish meals on a bulk by waving your attention away from the food. On the other hand, when cutting, you should turn off the T.V and focus on your meal, really enjoying every bite of it.

Artificial Sweeteners

Rogers and Blundell showed that a food mixed with saccharin (an artificial sweetener) had little satiating capacity in comparison to a meal sweetened with glucose or sucrose [150]. Cauty and Chan further compared the effects of aspartame, saccharin, and sucrose on hunger and food intake [151]. They had a group of 20 normal adults consume a standard breakfast, followed 3 h later by 200 ml of either water or a sweetened drink. After this, the subjects recorded every half hour hunger ratings. Hunger wise, the highest rating was given for water; the non-calorie sweetened drink was a bit lower, but not statistically significant; while the sugar was much lower, and had a high statistical significance.

Overall, most the studies have shown that artificial sweeteners either very slightly decrease hunger or do not affect it at all [153]. So supplementing with them will have little benefit when dieting. The only advantage would be to relieve a quick sweet tooth, but nothing more. When bulking, however, adding artificial sweeteners to a plain food such as oatmeal can enhance its palatability, and increase appetite [154]. In addition to this, artificial sweeteners have not been shown to increase weight or fat gain in humans.

For instance, in order to test whether artificial sweeteners increased fat gain in a long term study, Raben et al. fed overweight men and women for 10 weeks with either sucrose or artificial sweeteners everyday [152]. The sucrose group had increased energy intake, body weight, and fat mass, while these effects were not

observed in a similar group of subjects who consumed artificial sweeteners. Therefore artificial sweeteners do appear safe as far as weight gain is concerned.

Sensory-Specific Satiety

The sensory-specific satiety theory states that one is more likely to consume a higher amount of food if their diet has variety, than one who consumes the same foods day in and day out because of an adaptation in the senses to foods [160]. There are hundreds of experiments which support this theory.

When animals are given a diet with a variety of foods to consume, and a diet in which they can either consume one food or the same type of diet consistently, the former group consistently is hyperphagic compared to the latter group [161, 162, 163]. Furthermore, animals given several types of high-fat foods eat more grams and calories than animals given one type of high-fat food [164, 165]. Having the same food, but varying its composition also has the same effect. For example, several experiments demonstrated that serving a group one food, compared to serving a group the same food but with vanilla, lemon, and a variety of other flavors, lead to the latter group having more calories and consuming more grams than the other group [166, 167, 168].

Additionally, having more variety within one meal promotes hyperphagia. For instance, Rolls et al. fed participants four courses, one each of sausages, bread and butter, chocolate dessert, and bananas, or four courses of one of these foods [169]. Those who had variety each meal consumed 44% more food and 60% more energy than the other group. Moreover, people given three flavors of yogurt over several meals, compared to people with one choice, consumed a significantly greater amount of calories [170]. In another experiment by Roll et al., he gave his participants cream cheese sandwiches, and the only difference was that he gave one group several of the same sandwiches with different toppings: salt, lemon essence and saccharin, or curry [172]. The variety group consumed 15% more calories than the plain one. There are hundreds of other testimonies to substantiate this; however, there is a limit. If the difference is too minor, over eating will not occur. For example, giving people strawberry, raspberry, and cherry, with the same color and texture, did not result in increased caloric intake [171].

The authors attribute this to an adaptation in the senses. Having the same food over and over again decreases the palatability and excitement during ingestion. Smelling, and tasting the same food basically becomes a bore; however, when mixing it up, your senses stay sharp and foods are easily consumed.

This has profound effects on bulking and cutting cycles. When cutting it would be best to stick with the same foods, the same texture, and makeup, etc. Only have a couple of carb sources, a couple of protein sources, etc. But when bulking, the more variety the better. When you have oatmeal, make oatmeal cakes, oatmeal shakes, oatmeal cooked, oatmeal pancakes, etc. You want a variety of foods as well; have yams, wheat bread, beans, steak, chicken, etc. Mix it up as well; have burgers, chicken and oatmeal, beans and steak, etc. Now, you can still have some variety during a cut, and you can stay basic on a bulk; this is just another weapon placed at your disposal—use it wisely.

Fats, Carbs, & Proteins

Now its time to tie it all together and show which macronutrient is the most satiating of them all. There is no straight forward answer to this. All three are vital to one's diet, and deficiency in any, as displayed above under cravings, can and will promote hunger. With this in mind, the results show proteins are most satiating, although carbs often come a close second, and some studies (such as the satiety index, discussed further on) show carbs to be number one. Fats are last in just about every case.

First, a major reason fats have less satiety is because they are so energy dense (discussed above). One gram of fat has nine calories, while protein and carbohydrates only have four calories per gram. Therefore, fats are often overeaten. Because of this, the majority of journals recommend a low-fat, high-carb/protein diet for obese individuals [174]. In one experiment, obese people were allowed to gorge themselves on either high-fat or high-carbohydrate foods [175]. Obese subjects voluntarily consumed twice as much energy from the fat, displaying the weak satiating action of lipids.

Johnstone, Stubbs, and Harbron compared all three macros in a study [177]. They gave subjects a high-protein (HP), fat (HF), or carb (HC) diet, and compared hunger and satiety throughout the day. Participants on the high-protein diet felt less hungry and fuller than the other two diets, and the high-carb diet had better results than the high-fat diet. Moreover, the HC diet was more satiating than the HF diet after each meal.

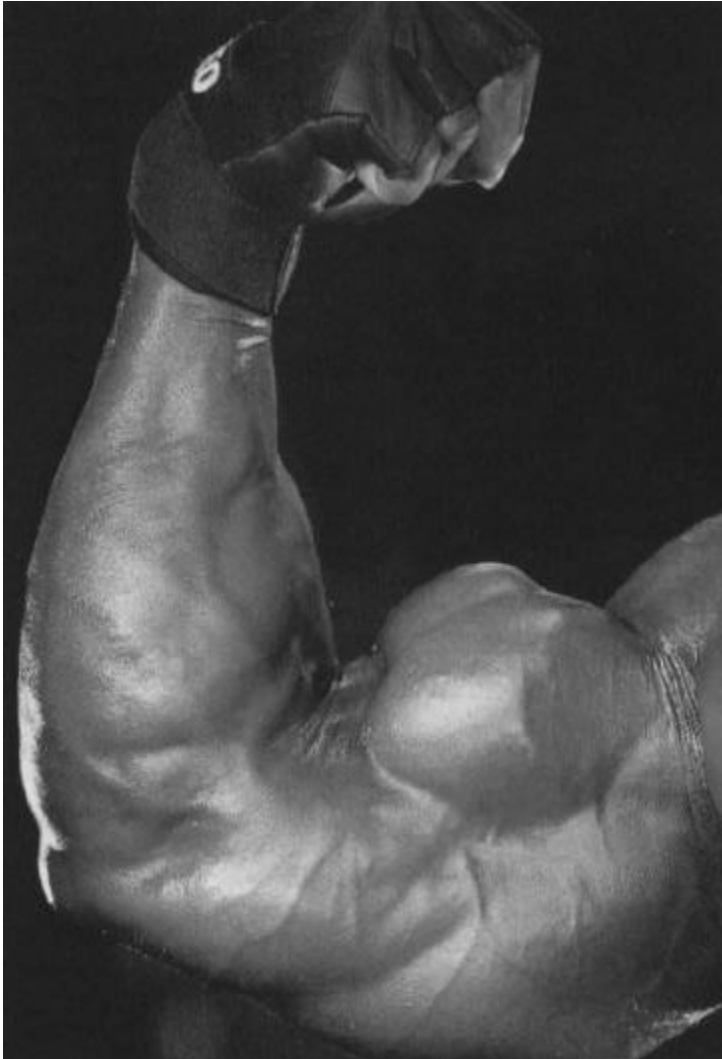
Plantenga et al. composed an experiment to assess a possible relationship between perception of satiety and diet-induced thermogenesis (DIT; this is a whole other subject, which will be talked about in the future, but fats give very little DIT, while proteins and carbs do) [178]. The subjects were eight females, ages 23-33. Subjects were fed a PCF of 29/61/10 and 9/30/61 with all other conditions being equal. Thermogenesis was higher on the high protein/carbohydrate diet, satiety was higher during meals ($P < 0.001$; $P < 0.05$), as well as over 24 h ($P < 0.001$), than with the high fat diet. These results showed a high carb/protein diet will elicit s much higher satiety and DIT than a high fat diet.

In another study, after a lunch, Marmonier, Chapelot, and Sylvestre gave subjects a high-fat (58% of energy from fat), high-protein (77%) or high-carbohydrate (84%) snack, and than viewed how long they waited until dinner [179]. Consumption of a high-protein snack delayed the request for dinner by 60 min. In contrast, a high-fat snack delayed dinner request by 25 min, whereas a high-carbohydrate snack delayed dinner request by 34 min. Snack composition had no impact on energy or macronutrient intakes during dinner. Another experiment on snacking showed similar results, except the delay in meal request was less. Furthermore, the calories consumed at dinner were not suppressed [180]. The authors concluded that snacking has poor satiating efficiency, and may play a role in gaining excess weight. So one should be careful with excessive snaking, and instead opt for full, balanced meals.

There are many other studies like this. In conclusion, proteins appear to have the highest satiety point, then carbs, then fats. You can certainly see yet another logical reason to follow a high-protein diet, as most athletes do.

Fat Chain Length

In order for a bodybuilder to get a proper amount of fat in their diet, they must add specific fatty foods such as peanut butter, flax, safflower, etc. There are a variety of fats, including saturated fats, polyunsaturated fats, and monounsaturated fats. These fats vary in level of satiety though.



In order to see which fat was most satiating, Lawton et al. composed an experiment on two different groups of 20 subjects each (half male and female) [173]. Group one tested the effects of fat A (oleic blend, high in monounsaturated fatty acids (MUFA)) with those of fat B (linoleic blend, high in polyunsaturated fatty acids (PUFA)) and fat C (stearic-oleic blend, high in saturated fatty acids (SFA)). Energy and nutrient intakes were monitored for the rest of the day and for the following day. Profiles of hunger, fullness, and other sensations were monitored by continuous tracking and end-of-the-day questionnaires were filled out. Subjects consumed significantly more energy after consumption of the lunch containing fat A (MUFA), and were hungrier than after the lunches containing fats B (PUFA) or C (SFA), and there was a trend for these effects to continue into the second day. Lastly, fats B had a higher satiety than fats C.

A second study was designed to confirm and extend the findings of Study 1. It compared the effects of fats A, B, C, and additionally, fat D (a linoleic-oleic blend). In Study 2, fat C produced similar effects on appetite to fat A, and there was a tendency for subjects to consume more over the whole test day when they had consumed the lunch containing fat A than when they had consumed the lunch containing any other fat. Additionally, they prolonged this study for 40 days, and the results of test one were confirmed.

Therefore, when bulking you would want to consume a lot of monounsaturated fats such as peanut butter, and when cutting you would want to lower your intake of peanut butter (among other monos) and opt for polyunsaturated fats, such as flax and safflower

Now, while you can vary your fats a bit, you still need to maintain a proper essential fatty acid ratio. Read the following article for instructions on that, [Essential Fatty Acids - An In Depth Analysis](#)

High- vs. Low-GI Carbs

Carbs are often measured by the glycemic index (GI). The higher the GI, the simpler and fast-burning the carbohydrate is; the lower, the slower burning it is. In terms of maintaining a steady flow of blood glucose, you definitely want to opt for low-GI carbs throughout the day (save post-workout); however, in the short-term, high-GI carbs are more satiating than low-GI carbs.

For example, Anderson et al. gave subjects high-glycemic-index preloads (glucose, polylose, and sucrose) and low-glycemic-index preloads (amylose, amylopectin), then measured hunger ratings and how much they ate thereafter [176]. Carbohydrates with a high GI were more satiating, and suppressed hunger much more than low-GI carbs. Other studies also testify to glucose's anorexic effects within 1 hour of consumption [91, 92].

Some reasons for these effects are that insulin is very satiating, NPY would be reduced, blood sugar would be initially higher, nutrients would be delivered quicker, etc. So in the short-term, having a post-workout shake, for example, would be more satiating than oatmeal. High bursts of insulin (induced by high blood sugar) promote hypoglycemia, however. Soon you would get very hungry because of low blood sugar; therefore, you want to have slow-burning carbs throughout the day. On a bulk, however, high-GI carbs may be of benefit. This is discussed further on under practical applications.

Starvation

Some people are hungry because they basically starve themselves. This is why it is important to understand how LBM, height, weight, etc. affect your metabolic rate. For one athlete, 2000 calories may be more than adequate for hunger, health, etc. For another athlete who has twice as much muscle mass, for example, that would be pure starvation and result in constant hunger. Further, as stated above, leptin, CCK, etc., are much less effective and concentrated in the body on a starvation diet. Furthermore, NPY, among other orexigenic factors, will be skyrocketed. I recommend you read [13 Weeks To Hardcore Fat Burning - "The Diet"](#) to understand how to

calculate how many calories you need, and follow the many safe tips for dieting given within.

Other Factors

There are many other factors than an actual physiological need for food that must be considered. People may not consume food because of cultural background, social situations, pressures to look thin, or to fit in with the crowd, which leads to ailments such as anorexia. You could simply have a bad habit, or you could lack any self-discipline and be a lazy, fat, slob.

People tend to eat more in cold weather and less in hot weather [155]. This has given credence to the thermostatic hypothesis, which states that the hypothalamus regulates body temperature and food intake, and proper interaction of temperature and eating behavior [156].

External factors such as smell can influence appetite. Appetite is the physiological desire to eat in relation to sensations for foods, while hunger is a physiological need to eat. So while you may have appetite, your body might not actually need that food. A good example would be if you have a huge bowl of oatmeal, steak, a salad, and some essential fatty acids. At the end of this meal, you would feel stuffed and no longer desire to eat anymore oatmeal or steak. However, you may have an urge to down a nice, palatable bowl of ice cream. That is appetite—not hunger.

Environment is vital to one's success. If you wake up every morning to a box of Krispy Kremes or the smell of freshly baked cookies, you are much more likely to cheat. If, however, you only allow clean foods in your household, such as oatmeal, cottage cheese, peanut butter, whey protein, etc., appetite will be greatly suppressed and, even if you do cheat, a couple bowls of oatmeal will not hurt your diet—in fact, it may help. A Krispy Kreme, on the other hand, is absolutely worthless and will only hurt your body. So I would encourage you to surround yourself with less-tempting foods. Whomever you live with, or work with, try to get them involved in healthy eating. Having people around that support your goals is vital to any bodybuilder's success.

Several studies have shown that stress can promote anorexia [157]. It is postulated that corticotropin-releasing hormone (CRH) and/or serotonin (5-hydroxytryptamine, 5-HT) contribute to this [158]. Both are anorexigenics, and elevate in response to stress throughout the brain, including feeding centers. Additionally, CRH inhibits NPY release [159].

In other situations, such as depression, emotional breakdowns, lowliness, etc., people may react by overeating.

Should You Ignore the Hunger Mechanism?

No, you should not ignore it. If you feel excessively hungry, or anorexic, you need to take a close look at your diet and analyze your weak points. You may have low blood sugar, or NPY may be high due to low-carb dieting. You may be low in fat or protein. You may not be having enough fiber or low energy-dense foods, etc. There are many tricks to manipulating hunger. After reading this article, there is little excuse left not

to get the job done and eat the amount of calories necessary to get you optimal results, without feeling excessively uncomfortable when cutting or bulking.

Other Bodily Factors Which Regulate Hunger

There are literally hundreds of other hormones, peptides, and endo/neurological signals to be discussed, but this can be written about for years on end and through hundreds of papers. In fact, millions of scientific journals are composed on this very subject, and scientists still do not fully comprehend the marvels behind hunger regulation. So to close this out, I am going to give you a quick wrap up on a few more peptides and hormones which regulate hunger.

Among hunger stimulators is agouti-related peptide (AgRP), which is co expressed by NPY. This peptide binds appetite-suppressing hormones melanocortin 3 and 4. Galanin is another hunger peptide increased in the anterior PVN during decreases in calories, when fat utilization is enhanced. Serotonin increases satiety, as well as glucagon, glucagons peptide 1+2, Amylin, Peptide YY, Cocaine Amphetamine Related Transcript (CART), Corticotropin releasing factor (CRF), and prooppiomelanocortin, among others. Last, melanin concentrating hormones increase appetite [181].

The Satiety Index

The following tables are rated according to how much food people ate after consuming each edible. The foods are all compared to white bread at 100%. The higher the percent a food has, the better its satiety [184].

Junk Food		Snacks		Fruits	
Cheese	146%	Crackers	127%	Oranges	202%
Cookies	120%	Peanuts	84%	Apples	197%
Jellybeans	118%			Grapes	162%
Ice cream	96%			Bananas	118%
Crisps	91%				
Mars candy bar	70%				
Doughnuts	68%				
Cake	65%				
Croissant	47%				

Protein Dense		Carb Dense		Cereals + Milk	
Ling fish	225%	Potatoes, boiled	323%	Oatmeal	209%
Beef	176%	Brown pasta	188%	All-Bran	151%
Baked beans	168%	Wholemeal bread	157%	Honey smacks	132%
Eggs	150%	Grain bread	154%	Cornflakes	118%
Lentils	133%	White rice	138%	Special K	116%
		Brown Rice	132%	Sustain	112%
		White pasta	119%	Muesli	100%
		French fries	116%		
		White bread	100%		

Practical Applications

There are multitudes of dieting and bulking strategies to learn from this article. Below I will give a few tips to get you started.

Bulking: The following is applicable if you are bulking and having a difficult time eating enough calories to promote hypertrophy/hyperplasia:

- Have a high variety of meals. People that have variety in their diets may eat 70% more calories than plain diets. Have different varieties of oatmeal such as pancakes or shakes, have different foods such as yams and wheat bread, and have mixtures of food such as beans and steak.
- Listen to something distracting while you eat. I enjoy hearing James Earl Jones read the New Testament on my Bible CDs. Sometimes I will listen to audio clips of creationists destroying the pagan religion of evolution for a good laugh.
- Don't add much water to your foods, as this enhances satiety. Make small protein shakes and bowls of oatmeal.
- Be careful with your fiber intake. I would recommend only one tossed salad so you get all types of fiber and maintain insulin sensitivity, among other benefits. Besides that, you should be getting enough from carbs.
- Choose calorie-dense foods.
- Go high on monounsaturated fats; they have a very low satiety ranking.
- Adding artificial sweeteners to your diet can make food such as oatmeal more palatable and promote eating.

- As a last resort, if you feel you cannot consume any more calories, and are doing everything possible in your power to do so, you may consider going on a little more of a dirty bulk and having higher glycemic, calorie-dense carbs (i.e. raisins). This will promote hypoglycemia via insulin, and induce hunger. Additionally, these types of carbs are easily digested, and will provide more variety. Furthermore, you will reap the anabolic rewards of insulin. Moreover, high-GI carbs have been shown to be superior to low-GI carbs in the area of glycogen replenishment [183]. The problem with this is that you are more susceptible to gaining fat, and insulin sensitivity will decrease. So I would recommend you pay close attention to your body fat, and only go on a short dirty bulk cycle (i.e. 6-8 weeks), then work on improving your insulin sensitivity on a cut or maintenance diet for a few weeks. Then hit back up on your bulk.
- Opt for less-filling foods. Often on a bulk I will have protein shakes instead of whole meats such as steak and chicken. Eating 400 grams of protein and 6000 calories from steak is practically impossible. Having a protein shake is definitely a smart move here. Other palatable foods in the carbohydrate and fat departments should be chosen as well.

Cutting: Below is a list of tips for those cutting and having a difficult time with hunger:

- Often people on low-carb diets will have a post-workout shake, and go low fat the remainder of the day. This will greatly promote hunger. You see, blood sugar levels are already low on this type of diet, and having a high burst of glucose will only augment this. Insulin promotes hypoglycemia, and subsequent urges of hunger will occur. I therefore recommend saving at least one complex carb meal for after your post-workout shake to help stabilize blood sugar levels.
- Eat at least 6 meals a day. Eating more frequently will help satiety and promote stable blood glucose levels
- Enjoy your meals; when you eat, do not listen to something distracting, but rather focus on your food.
- Make sure you have enough protein, as it is the most satiating macronutrient.
- Don't go on an excessively high fat diet (50% or more), as this will speed gastric emptying by making your body less sensitive to CCK.
- Have slow-burning carbs to help maintain blood sugar and decrease NPY mRNA.
- Adding an artificial sweetener may help relive a sweet tooth, but that is about it. You may add this to your regimen if needed.
- Lie down on your back or your left side when you eat and while digesting your food. This will promote satiety, and may slow gastric emptying by 50%! It would be a good idea to do this at night as well after your last meal.
- Adding water to your foods is a great way to promote satiety. Results show mixing a casserole, for example, with just one glass of water to form a soup can decrease subsequent energy intake by 26%. So if you wake up in the morning and have a protein shake and oatmeal, you should put as much water in each as you can. Make a huge, diluted protein shake, as well as puffy oats. Having water separately may assist as well, perhaps by promoting gastric distension, but for the most part it is detected through the drinking mechanism and not through the feeding mechanism. It is definitely much less potent than mixing water with foods; however, you should always be drinking water anyway to stay properly hydrated.

- Make sure you have enough fiber in your diet. Results show that having 14 more grams can decrease daily calories by 10%.
- Stick with low energy-dense foods. People at an all-you-can-eat buffet for a day have half the amount of calories, and eat 33% longer when given low energy-dense foods to eat, compared to those given high energy-dense foods.
- Stick with basic foods; don't change it up too much. Variety causes one to eat more, which is in accordance with the sensory specific satiety theory.
- Make sure you are getting all your macros. Absence of any one can cause cravings.
- Do not starve yourself. Read the 13-week diet to see how many calories you need.
- Surround yourself with a healthy environment. Appetite is much higher when you are constantly around boxes of Krispy Kremes than containers of oatmeal.
- Go high on polyunsaturated fats; they have the highest satiety ranking among fats. Likewise, be careful with your consumption of monounsaturated fats, as they have a very low satiety ranking.
- Choose big bulky foods. Even if they do not weigh a lot, volume is a strong mediator for satiety.
- Keep a steady mind. Depression and stress can negatively impact your diet. The best way to do this is to develop a relationship with the Lord Jesus Christ. Put your pressures on him, and he will comfort you. Jesus said [182], *"Come unto me, all ye that labour and are heavy laden, and I will give you rest. Take my yoke upon you, and learn of me; for I am meek and lowly in heart: and ye shall find rest unto your souls. For my yoke is easy, and my burden is light."*

These are but a few tips. Study this article several times for much more.

Conclusion

Hunger has great spiritual applications. Just as you should not ignore your fleshly hunger, you should not ignore your spiritual hunger. The only way to quench this sensation is by accepting Christ as your savior. I encourage all reading to do so, and cease from spiritual anorexia now, and in the life to come [182].

Revelation 7:13-17

13 And one of the elders answered, saying unto me, What are these which are arrayed in white robes? and whence came they? **14** And I said unto him, Sir, thou knowest. And he said to me, These are they which came out of great tribulation, and have washed their robes, and made them white in the blood of the Lamb. **15** Therefore are they before the throne of God, and serve him day and night in his temple: and he that sitteth on the throne shall dwell among them. **16** *They shall hunger no more, neither thirst any more; neither shall the sun light on them, nor any heat.* **17** For the Lamb which is in the midst of the throne shall feed them, and shall lead them unto living fountains of waters: and God shall wipe away all tears from their eyes.

John 6:35

And Jesus said unto them, I am the bread of life: he that cometh to me shall never **hunger**; and he that believeth on me shall never thirst.

Luke 6:21

Blessed are ye that **hunger** now: for ye shall be filled.

Matthew 5:6

Blessed are they which do **hunger** and thirst after righteousness: for they shall be filled.

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