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The Role of Hormones in Weight Management

by Karon R. LoCicero, M.D.

Obesity Overview

With approximately two-thirds of adults in the United States characterized as overweight or obese, the condition of obesity is widely considered a public health epidemic. Obesity increases the risk of the development of conditions such as hypertension, Type 2 diabetes mellitus, osteoarthritis, coronary artery disease, stroke, and respiratory complications, as well as cancers of the breast, prostate, and colon (The Practical Guide, 2000).

The etiology of obesity is multifactorial but can be abridged to accommodate two dominant categories: physiological and environmental elements. The physiological aspects of obesity include body metabolism, hormones, and the neurological components of appetite regulation. Environmental causes include the abundance of high calorie foods in the Western diet, as well as the prevalence of increasingly sedentary lifestyles due to technological advances.

Neurological Aspects of Weight Regulation

The significance of appetite control in weight regulation, especially in cases of obesity, cannot be overemphasized; appetite control plays a vital role in the competition between energy consumption and energy expenditure. Food consumption behavior is administered by various hormonal, psychological, and neural signals, each characterized as short-term, and each derived from the gastrointestinal tract (Arora, 2006).

Hormones involved in the brain-gut axis of eating behavior come from various sources. Some hormones, such as leptin, arise from fat stores; others, including cholecystikinin (CCK), glucagon-like peptide (GLP), ghrelin, peptide YY, and neuropeptide Y, arise from the brain or gastrointestinal tract. Some of these hormones suppress eating behavior, while others stimulate it.

Recent research has concentrated on the neurological aspects of weight regulation, with a primary focus on the

Continued on page 2

Weight Loss 101: Glycemic Index and Beyond

by Melina Jampolis, M.D.

As a physician nutrition specialist who focuses predominantly on weight loss, I spend a lot of time both seeing patients and scouring the nutrition and medical literature to optimize my dietary recommendations. Nutrition as a sub-specialty in medicine is relatively new despite the fact that Hippocrates wisely said "let food be thy medicine" thousands of years ago. It is also a very difficult field in which to conduct evidence based research as there are so many confounding variables and reporting errors when it comes to diet. Attempts to control for these variables often lead to artificial eating environments which do not necessarily translate into "real life" solutions or results. For this reason, my approach to weight loss is based on a combination of my interpretation and analysis of the best available research combined with my clinical experience seeing patients on a regular basis who are trying to lose weight or naturally lower cholesterol or blood sugar.

My approach focuses on three main objectives: Stabilization of blood sugar, hunger control, and preservation of lean body

Continued on page 6

INSIDE THIS ISSUE

The Role of Hormones in Weight Management

Obesity Overview	page 1
Neurological Aspects of Weight Regulation	page 1
Appetite Regulation and the Hypothalamus	page 2
Neuropeptide Y	page 2
Ghrelin and NPY	page 2
Cholecystikinin and Satiety	page 2
Leptin and CCK	page 3
Recent Studies of CCK	page 3
Norepinephrine and Serotonin	page 4
Endocannabinoid System	page 4
A Personal Approach to Weight Loss	page 5

Weight Loss 101: Glycemic Index and Beyond	page 1
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neuro-gut interaction. The discovery of neuropeptides, including neuropeptide Y (NPY) and cholecystokinin (CCK), has contributed to our understanding of the neurological components of weight control. These neuropeptides play significant roles in the manner in which brain, gut, and fat stores interact to control food intake; multiple feedback loops regulate hunger and satiety.

Appetite Regulation and the Hypothalamus

According to a classic model of the role of the brain-gut axis, eating behavior remains under the control of a satiety center located in the ventromedial hypothalamic area, as well as under a feeding or appetite center located in the lateral hypothalamic area. The satiety center inhibits the feeding center, which is always active but can be suppressed by gastric distention or by hormones, such as CCK (Bray, 2000).

Still, a more sophisticated model of the role of the brain-gut axis has emerged. The Arcuate (infundibular) Nucleus is situated at the base of the hypothalamus (Neary, 2003). The two main populations of Arcuate Nucleus neurons that control food intake are appetite-inhibiting neurons and appetite-stimulating neurons. Pro-opiomelanocortin (POMC) neurons

inhibit food intake, whereas neuropeptide Y (NPY) and agouti-related peptides (AGRP) stimulate appetite (Murphy, 2004).

POMC is the precursor of melanocyte-stimulating hormone, which inhibits eating behavior. AGRP antagonizes the melanocortin receptors, thereby increasing food intake. NPY is one of the most potent appetite-stimulating peptides. Both the AGRP and NPY are inhibited by leptin and insulin, whereas ghrelin activates them (Neary, 2003).

Neuropeptide Y

The most impressive effect NPY has on behavior and assorted functions is its post-central administration arousal of feeding. Chronic administration of NPY encourages hyperphagia and obesity (Arora, 2006).

Y5 receptors, responsible for the mediation of the stimulating effects of NPY, are exhibited at comparatively high levels in the LHA (a hypothalamus site), in close proximity to the location of heightened NPY stimulative activity. When starvation occurs, NPY receptor density dwindles in this location; the release of NPY is enhanced when the body is deprived of food (Arora, 2006).

Ghrelin and NPY

Ghrelin, which tends to increase appetite, is secreted primarily by the stomach (Graaf, 2004). Ghrelin accomplishes appetite stimulation by encouraging the production of NPY (Arora, 2006). Ghrelin decreases after food consumption, but returns to its baseline before the next meal. (Graaf, 2004). Moreover, ghrelin remains a vital component in acute and long-term control of energy stability (Arora, 2006).

Subjects characterized as healthy and lean have generally higher levels of ghrelin in their bodies. Additionally, patients diagnosed with bulimia nervosa have higher levels of ghrelin than control subjects with identical weights. A substantial decrease in ghrelin levels has been noted in patients diagnosed with anorexia nervosa, surprisingly before any substantial changes to their body mass indices have occurred (Arora, 2006).

Cholecystokinin and Satiety

Cholecystokinin has been one of the most widely studied hormones involved in the control of satiety. It is secreted by the mucosal cells in the duodenum, as well as by neurons in the brain. CCK stimulates contraction of the gallbladder to release bile. This response occurs primarily in the presence of fat, such as long chain free fatty acids or protein (amino acids). CCK also stimulates the release of pancreatic enzymes which, along with bile, promote digestion within the small intestine (Bloom, 2005).

CCK plays an active role in the stimulation of the vagus nerve; the vagus nerve sends a signal to the brainstem, and then to the satiety center of the brain. Studies have demonstrated that this effect is reduced by vagotomy. CCK also acts on the dorsomedial nucleus in the hypothalamus by reducing

NUTRI NEWS

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600 Boyce Road • Pittsburgh, PA 15205
Phone: (412) 494-0122 • Fax: (412) 278-6804
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neuropeptide Y, a potent orexigenic peptide (Bloom, 2005).

Additionally, CCK remains a vital component of meal size control processes; CCK endocrine actions in the intestine, as well as its neurocrine actions elsewhere in the body are instrumental in this control. This effect is amplified by gastric distension (Arora, 2006).

While the effects of CCK on food consumption are swift, they fail to endure at length. The half-life of CCK is one to two minutes. Moreover, its effects are mitigated if administered fifteen minutes prior to food intake. However, it has been suggested that CCK is able to positively affect long-term control of meal size and body weight when leptin levels are demonstrably elevated (Arora, 2006).

Leptin and CCK

Leptin, discovered in 1994, is synthesized by adipose tissue. It has been shown to reduce food intake and body weight in certain animal studies (Graaf, 2004). The amount of leptin in the body is proportional to the amount of fat in the body. Leptin inhibits NPY/AgRP neurons and stimulates POMR neurons, thereby decreasing appetite (Murphy, 2004). Low caloric intake causes a decrease in leptin levels and an increase in appetite, whereas high caloric intake causes an increase in leptin levels; it would be reasonable to expect a decrease in appetite. While this has proven true in certain animal studies, it does not hold true for obese humans who already have high circulating leptin levels. It is postulated that leptin resistance plays a significant role in obese individuals, but the etiology remains unclear (Neary, 2003).

When leptin levels rise, a signal that excess energy is being stored is sent to the brain; in turn, appetite decreases and energy expenditure increases. It has also been demonstrated that food intake restrictions – over the course of several days – suppresses levels of leptin in the body. These effects can be reversed by re-feeding (Arora, 2006).

Approximately one-twentieth of obese populations may be regarded as comparatively leptin deficient. This group may benefit from exogenous administration of leptin (Arora, 2006). Interestingly, a study by Matson, et al has demonstrated that the administration of leptin, along with CCK, significantly decreases daily total caloric intake compared to the administration of leptin alone. More research, however, needs to be completed regarding the use of leptin as a weight loss drug in light of the limited results in human studies (Matson, 1997).

Recent Studies of CCK

CCK inhibits gastric emptying, resulting in appetite reduction. A study by Straathof, et al demonstrates that CCK, at postprandial plasma concentrations, decreases basal gastric tone, thereby slowing gastric emptying. It has been hypothesized that

fatty foods, which relax the stomach, exert this effect through stimulation of endogenous CCK (Straathof, 1998). Another study by Matzinger, et al demonstrates that inhibition of food intake in response to intestinal lipid is mediated by CCK (Matzinger, 1999).

A study by Burton-Freeman, et al demonstrates that the feeling of satiety produced by CCK is enhanced by increasing the fat or fiber content of the diet. Whereas fat increases the release of CCK, fiber prolongs its release, perhaps by slowing the rate of fat absorption (Burton-Freeman, 2002). Loxiglumide, a CCK receptor antagonist, stimulates hunger in humans (Beglinger, et al). Other studies have shown that the inhibition of CCK metabolism decreases caloric intake.

Administration of butabindide, which inhibits the CCK-inactivating peptidase, tripeptidyl peptidase, has been shown to elicit CCK-like satiation in rats. Another study illustrates that the oral administration of a proteinase inhibitor extracted from potatoes (POT II), which binds to both trypsin and chymotrypsin, thereby preventing the degradation of endogenous CCK, has been shown to stimulate CCK release and reduce caloric intake by 20% following a fat or protein preload (Little, 2005). Additionally, supplementation with a potato protein extract in humans has been shown to modify the glycemic response to a meal (Spreadbury, 2003).

Kissileff, et al demonstrates that the administration of IV CCK in humans reduced food intake by 19%. An additional study by Kissileff, et al demonstrates that gastric distension enhances the effect of CCK on the reduction of food intake (de Graaf, 2004).

Peptide YY, another protein secreted by the distal ileum and colon, stimulates the Y2 receptor in the hypothalamus; this inhibits the release of neuropeptide Y, the strongest central nervous system stimulant of appetite (de Graaf, 2004). It also slows gastric emptying (Bloom, 2005).

Chronic administration of PYY to rodents resulted in reduced weight gain. Obese patients have suppressed PYY levels. Gastric bypass patients have a greater PYY response to food intake than patients who have not undergone bypass. This response may contribute to the weight loss associated with the surgery (Bloom, 2005). Interestingly, CCK stimulates the release of PYY (Matzinger, 1999).

GLP-1 (glucagon-like peptide-1) is secreted by the ileum in response to fat and carbohydrate intake. GLP-1 stimulates the pancreas to secrete insulin and inhibits glucagon release. It also diminishes gut motility, thus suppressing appetite (de Graaf, 2004; Bloom, 2005). Studies have demonstrated that a dose-dependent reduction in food intake and increase in satiety occur after an IV infusion of GLP-1 (Bray, 2000). Another study demonstrates that intravenous infusion of physiological doses of CCK and GLP-1 produce an additive reduction in food intake

(Matzinger, 1999).

Exenatide, the first in a new class of diabetes medications known as incretin mimetics, shares many of the same characteristics as GLP-1, with the exception of half-life.

GLP-1 is metabolized rapidly (less than two minutes), whereas exenatide has a half-life of several hours. Weight loss tends to occur with exenatide.

Norepinephrine and Serotonin

Other more commonly recognized discoveries in this area are the neurotransmitters norepinephrine and serotonin, both present within the hypothalamus. The prescription drug phentermine causes the release of norepinephrine, which in turn reduces food intake. Another prescription drug, sibutramine, increases both norepinephrine and serotonin by blocking their reuptake; it also stimulates certain 5 HT receptors, which in turn reduces food intake.

Indeed, many prescription weight control drugs aid weight loss by decreasing appetite, increasing metabolic rate, or decreasing fat absorption. However, each has potential side effects, and each remains only somewhat effective. Both phentermine and sibutramine can increase blood pressure and cause dry mouth, insomnia, and constipation. Neither should be used in patients with uncontrolled hypertension, cardiac disease, or a history of stroke.

Recent Advances in Weight Management

Exciting research continues in the field of obesity management. The presently available drugs, such as sibutramine, phentermine, and orlistat, have potentially significant adverse effects. Gut hormones, if able to be used as drugs for appetite control, would be expected to have potential for low side effects, as they would act only on the specific appetite center in the brain (Murphy, 2004).

Unfortunately, gut hormones must be given parenterally as they have an exceedingly short half-life. Otherwise, other forms of administration (e.g., intranasal), or the development of an analogue that is resistant to breakdown, would need to be developed. (Murphy, 2004).

Drugs that mimic or enhance productions of appetite-regulating proteins or neuropeptides are studied extensively at present. For example, trypsin inhibitors, which block the inactivation for CCK, have been shown to suppress food intake in animals and humans (Murphy, 2004).

Exenatide, as mentioned earlier, is an incretin mimetic which is now being used for the treatment of Type 2 diabetes mellitus. This drug is an excellent example of one that mimics natural hormones, the incretins, such as GLP-1. Since exenatide promotes weight loss, it is anticipated that a similar drug, specifically for weight loss, will be available in the near future.

Endocannabinoid System

Another exciting area of interest is the endocannabinoid system; this system plays a vital role in control over appetite and satiety. Stimulation of the endocannabinoid system leads to increased food intake, whereas inhibition of that system decreases food intake (Woods article).

Endocannabinoid receptors (called CB1 and CB2) are located on cell membranes in the brain, gastrointestinal tract, white adipose tissue, and skeletal muscle. Endocannabinoids are fatty acids that are synthesized from phospholipids that are a part of cell membranes. In the brain, they inhibit the release of GABA, which is a neurotransmitter that diminishes food intake (Woods, 2006).

Endocannabinoid receptors in fat cells (adipocytes) increase the activity of lipoprotein lipase, which leads to fat accumulation (Woods, 2006). They also enhance the production of adiponectin from adipocytes (Van Gaal, 2005). Adiponectin improves insulin sensitivity and fatty acid oxidation in muscle thereby improving blood sugar and decreasing triglycerides (Greenway, 2004).

Endocannabinoids within the small intestine have an appetite-stimulating effect. CCK diminishes this effect by suppressing the cannabinoid receptors on the vagal nerves which transmit to the brain.

In skeletal muscle, endocannabinoids reduce energy expenditure. Blockade of the endocannabinoid receptors in skeletal muscle increases glucose uptake and basal oxygen consumption (Woods, 2006).

Rimonabant, which is awaiting FDA approval in the United States, is an endocannabinoid receptor blocker that has been shown, in the RIO (Rimonabant in Obesity) studies, to produce weight loss and improve blood sugar and lipids (Woods, 2006), which tend to be elevated in patients with Metabolic Syndrome (Abdominal obesity, elevated triglycerides, hyperglycemia or diabetes, and low HDL cholesterol).

Presently, I am anticipating approval of this drug in the United States as many of my patients have some or all the features of the metabolic syndrome, including hypertension, excessive intraabdominal fat, hyperlipidemia, and diabetes mellitus or glucose intolerance. I expect that the use of rimonabant in conjunction with a low glycemic diet and the use of "healthy" fats, such as monounsaturated or polyunsaturated fats, would produce significant health benefits in terms of weight loss and metabolic parameters.

A Personal Approach to Weight Loss

With a new fad diet for every season, the selection of and commitment to a weight management plan often proves difficult for patients. Frequently, fad diets fail to provide adequate nutrition; once the diet is complete, many patients experience

weight gain. Moreover, weight lost under a nutritionally unbalanced scheme is not necessarily fat lost (Kozak, et al 2000).

It is advisable for bariatric patients to take an inventory of their weight loss desires, motivations, physical activities, daily food intake, emotional attachments to eating, and outside sources of support (Kozak, et al 2000). After careful consideration of these factors, patients are often better-equipped to design a weight management program tailored to their individual lifestyles.

These programs, which may or may not include prescription weight loss drugs, prove most successful when they maintain cultural sensitivity; a patient accustomed to meals of black beans and yellow rice, or another whose dietary habits include steak and potatoes, will do best with weight management plans that incorporate their tastes, but are adjusted for portion and calorie control.

Additionally, patients should balance calories throughout the day; skipping meals and starvation should be avoided. Successful weight loss begins with healthy eating, and more frequent meals. Divide daily caloric intake into quarters; allot one-fourth each to the three traditional mealtimes, saving the remaining quarter for snacks (Kozak, et al 2000).

It is also advisable to focus on simple, unprocessed foods. These foods contain more vitamins, minerals, and fiber, and have less undesirable additives such as sodium and sugar. Exchange sugary sodas and fruit drinks for water, consume whole grains with significant amounts of fiber, and choose high-protein beans and other legumes to stave off cravings (Kozak, et al 2000).

Still, patients are advised to remain mindful of their caloric intake. To more easily manage this task, watch portion sizes and always eat meals on a plate or out of a bowl. Eating out of containers or other packages makes it difficult to gauge serving sizes. Because precisely measuring foods can prove cumbersome, patients often find shortcuts such as adjusting plate or cup size easier methods to control portions (Kozak, et al 2000).

A personally tailored food plan should be coupled with a personally tailored physical activity plan. No two bodies are the same. Most patients are able to walk at a moderate pace for at least thirty minutes; increasing steps walked per day remains a straightforward and easily adhered to physical activity plan (Kozak, et al 2000).

Formulate an aerobic exercise plan for a greater impact on cardiovascular health, strength, and flexibility. Consider the health benefits, risks, total calories burned, frequency, and convenience of each activity prescribed before adopting a plan. Patients should slot new exercise plans around their current daily routines; a major readjustment of an oft-followed schedule is more consistently followed if the change is not drastic (Kozak, et al 2000).

Resistance training is also advised to build strength, boost metabolism, and increase bone density (Johnson, et al 2000). Patients are advised to develop plans that accommodate their weight-lifting abilities at present, gradually building up the ability to lift more weight. In addition to the visible physical benefits, resistance training also reduces overall levels of body pain, relieves stress, balances moods, and provides energy spikes (Kozak, et al 2000).

In the field of weight management, emphasis must be placed on the development of new, healthful food and activity habits. Patients will find success in balance.

Hopefully, newer developments in the field of neurophysiology, such as CCK research, incretin-mimetics and the endocannabinoid system, will help promote further weight loss beyond that expected from diet alone.

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About the Author:

Dr. Karon R. LoCicero, M.D.

Karon R. LoCicero, M.D. is a native of Tampa, where she attended local schools. She graduated summa cum laude and Phi Beta Kappa from Duke University, where she completed her pre-medical studies and earned a bachelor of arts degree in Cultural Anthropology. She received her medical degree from the University of South Florida College of Medicine. Dr. LoCicero is Board Certified in Internal Medicine and is a Diplomate of the American Board of Bariatric Medicine. She is also a member of the American Obesity Association and the American Society of Bariatric Physicians. Dr. LoCicero serves as Medical Director of LoCicero Medical Group in Tampa, FL, an internal medicine and bariatric practice established in 1988. She enjoys exercising, spending time with her husband and three children, and supporting her patients' commitments to health and wellness.

Weight Loss 101: Glycemic Index and Beyond

continued from page 1

mass and metabolism. By meeting these three objectives, I am able to help many patients lose weight and keep the weight off in an increasingly "obesigenic" environment. I accomplish these objectives with two fundamental recommendations. First, I suggest that patients eat protein based meals every 3-4 hours. Protein helps with both satiety and maintaining lean body mass. Research shows that people who eat even 3% more protein after weight loss maintain fat loss and increase lean body mass. In addition, fascinating new research on the phenotype/diet interaction suggest that people with metabolic syndrome, a group of symptoms which include central adiposity, atherogenic dyslipidemia, and high normal blood sugar may do particularly well on a higher protein diet.

But just eating protein at every meal and snack is not enough. Although research has been somewhat inconsistent on the role of

carbohydrates and weight loss, I feel that both the quantity and the quality of carbohydrates is relevant to both weight loss and optimal health. Let me explain briefly why both are important.

In the '80s and early '90s, fat was seen as the nutrition villain. As a result, a lot of the big food companies cut the fat in many of their products, including cookies, crackers, ice cream, and frozen dinners. Unfortunately, they had to replace the fat with something, and advances in food processing allowed inexpensive new sweeteners, like high-fructose corn syrup; refined sugar; and highly refined, processed carbohydrates to cheaply fill the gap. The problem is that the processing removes most of the vitamins, minerals, and fiber, leaving only rapidly digested, nutrient-depleted carbohydrates, salt, and—in many cases—dangerous fats.

In the past thirty years, these processed snacks, convenience foods, and soft drinks have become a staple in the American diet. Not only are many of them low fat or nonfat, they are also cheap, fast, convenient, and tasty. With less and less time for home-cooked meals, consumption of these foods, along with fast food made from similar products, has grown exponentially. Unfortunately, the cost-savings benefits and ease made possible through modern food processing have a major downside: obesity.

Many nutritionists and obesity experts (including myself) believe that the tremendous increase in highly processed and/or sugary carbohydrates, including sugary snack foods, fast food, baked goods made with refined white flour, chips, and soft drinks has contributed to the current obesity crisis in this country.

In the late '90s, with our nation's obesity and obesity-related diseases, like diabetes and heart disease, hitting record levels, while we were supposedly following a low-fat, high-carbohydrate diet, attention turned to cutting carbohydrates instead of fat. Low-carb diets emerged into the public spotlight, and suddenly double bacon cheeseburgers without the bun were considered health food. Low-carbohydrate diets can work well in the short term for some, but most people have a hard time sticking to them for the long haul. In addition, omitting whole grains and fruit for extended periods undoubtedly has negative health consequences.

So what are the right kinds of carbohydrates? One of the best ways to evaluate carbohydrate quality in a particular food is to consider the effect of that food on blood sugar levels, a value known as the glycemic index, or GI. Basically, the GI ranks foods according to how much a standard amount (50 grams) of a particular food raises blood sugar over a two-hour period. Most vegetables and dairy have a low glycemic index, while some fruits and many grains have a higher glycemic index. Why does the glycemic index of food matter? High GI foods are broken down very quickly into simple sugars. This often leads to an exaggerated release of insulin which can lead to post-

prandial hypoglycemia, hunger, and fatigue. In addition, long term hyperglycemia and hyperinsulinemia can lead to insulin resistance and metabolic syndrome, which is associated with a markedly increased risk of heart disease, stroke, and overall mortality.

Research on the utility of the glycemic index and its role in weight loss has been mixed. Most studies showed increased satiety with a low glycemic index diet and a decrease in subsequent caloric consumption however this has not necessarily translated into improved weight loss. Research has shown that eating a lower-glycemic diet helps decrease a patient’s risk of heart disease and diabetes even without weight loss. In population studies, a low GI diet has been linked with lower BMI and is also associated with improvements in postprandial hyperglycemia in both type-1 and type-2 diabetics. In addition, research shows better cognitive performance following a low GI meal in type-2 diabetics and a decrease in markers of oxidative stress. When it comes to weight loss, however, my opinion is that the glycemic load is more relevant for most people.

The glycemic load (GL) takes into account the actual amount of carbohydrates in an average serving of a given food. By factoring in the quantity and the quality of carbohydrates per serving, you get a better idea of the impact on blood sugar and insulin. In the following box you will find the equation used for calculating the glycemic load. The significance is more easily understood through the examples shown there. These examples also clarify why carrots should not be off-limits for dieters, as most low-carb-diet supporters might lead you to believe.

Figure 1

Glycemic Load = (Glycemic Index x # Carbohydrates per serving) / 100
GL less than or equal to 10 = Low
GL 11–19 = Medium
GL greater than or equal to 20 = High

Figure 2

Glycemic Index of Carrots = 49 # Carbohydrates per ½ cup serving (cooked) = 5 g. Glycemic Load = (49 x 5) / 100 = 2.5
Glycemic Index of Pretzels = 83 # Carbohydrates per 1 ounce serving = 20 g. Glycemic Load = (83 x 20) / 100 = 16
Glycemic Index of Corn Flakes cereal = 92 # Carbohydrates per 1 cup serving = 26 g. Glycemic Load = (92 x 26) / 100 = 24
Glycemic Index of Instant White Rice = 87 # Carbohydrate per ¾ cup serving = 42 g. Glycemic Load = (87 x 42) / 100 = 36

As you can see from these examples, the quantity of carbohydrate is just as important as the quality in determining the impact of a food on blood sugar. I want to be clear, however, that I don’t believe that either value can be used alone for weight loss. I encourage patients to focus more on the “big picture” and eat mainly whole-grain, unprocessed carbohydrates with fewer added sugars, in controlled quantities. Since grains, sugars, and starchy carbohydrates are the most concentrated forms of carbohydrates and contribute the greatest number of calories to the typical American diet, simply cutting back on these foods can improve a person’s diet significantly. Sugar alone makes up about 25 percent of the average American’s daily caloric intake, so by simply reducing sugar (without replacing it with something else), most people will lose weight and improve their health.

Besides diet, there are several other things that can help with stabilization of blood sugar, which is one of the keys to successful weight loss for most. The first is exercise which has been shown to improve glucose control and decrease the risk of metabolic syndrome even without concomitant weight loss. In addition, I often use chromium containing supplements in my practice. Although the FDA has not endorsed chromium’s role in insulin resistance, I have found it quite valuable in patients with metabolic syndrome, insulin resistance and significant carbohydrate cravings, likely due to the essential role chromium plays in the function of insulin. On its own and in combination with biotin (2 mg), it helps considerably reduce blood sugar in type-2 diabetics. In addition, a daily dose of 600 mcg/day has been associated with significant reductions in carbohydrate cravings, and may even help with fatigue and mood swings in some. One observational study showed a trend toward weight loss and, at the very least, less weight gain among obese people taking a chromium supplement, although again I do not believe it plays a direct role in weight loss, but rather helps with blood sugar control and possibly carbohydrate cravings which can benefit weight loss.

I also recommend that my patients with high normal blood sugar or diabetes increase intake of cinnamon. While cinnamon is not typically thought of as a supplement (although it can now be found in numerous dietary supplements), I thought it was worth mentioning here for its remarkable health benefits. It has been shown to improve fasting blood sugar by up to 29 percent, decrease bad cholesterol by up to 27 percent, lower triglycerides by up to 30 percent, and reduce total cholesterol by up to 16 percent. I recommend that patients add it to their diet whenever they can. Ideas include sprinkling it in yogurt, oatmeal, coffee, soups, chili, or any sauce that could use a bit of spice.

In conclusion, while many of the recommendations discussed above including more frequent protein based meals, eating a low glycemic diet, and exercising regularly, can help with weight loss, the best thing that you can do as a health care practitioner

is to give your patients support and encouragement as well as offer some guidance in making changes that they can live with permanently.

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About the Author:

Dr. Melina Jampolis, M.D.

Dr. Melina Jampolis is double board certified in Internal Medicine and as a physician nutrition specialist, one of approximately 200 in the country. She is the host of Fit TV's Diet Doctor and her new book, "The No-Time-to-Lose Diet", was published in January 2007. She maintains a small private practice focusing mainly on weight loss in beautiful San Francisco.