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Leptin: What 'Fat Rats' Have Taught Us

By Michael D. Myers, MD • Los Alamitos, California

We now know that the brain and adipose tissue form an integrated communications network.

Scientists have postulated for many years that adipose tissue must have some function beyond just "hanging out" awaiting the next famine, and that it must be regulated by the central nervous system. In the late 1950s Hervey's parabiosis experiments (surgically connecting two animals to understand physiologic functions) showed that a humoral factor was involved in regulating weight. However, exactly what this factor was and how it functioned was unknown.

One such factor, subsequently named leptin (from the Greek *leptos*, or thin), was not discovered until almost three and a half decades later. Leptin is the only protein product of the "ob" gene that is composed of 146 amino acids. Leptin also is a hormone found only in mammals.

When leptin was first discovered, researchers thought that the "magic bullet" for obesity treatment was at hand. After all, mice bred with severe obesity (the ob/ob mouse) that did not produce any leptin had low metabolic rates, were infertile, and were hyperphagic, which led to severe obesity. When these mice were given leptin, their food intake decreased, their metabolic rate increased, and they lost a significant amount of weight.

Thus, researchers assumed that obese individuals must be like the ob/ob mice and either lacked leptin or had such low levels as to not

protect against obesity. As a result, "leptimania," with its wildly optimistic expectations of a cure for obesity, was born. However, like most manias, it ended in disappointment. When researchers measured the leptin levels in obese individuals, they were surprised to find high levels instead of the low levels they had anticipated. What was going on?

Humans and Leptin

To understand the function of leptin in humans, it is helpful to understand some of leptin's basic physiology. Leptin is produced primarily by white adipose tissue ("fat"). There are many factors involved in the regulation of its production, with significant variations between individuals. Some of the more significant factors are as follows:

Hormonal factors. Several hormones increase leptin production, including insulin (which is generally elevated in obesity) and cortisol. Cortisol is frequently elevated with stress (physiologic or psychologic) and in many mood disorders. Additionally, there is sexual dimorphism, and women generally have much higher leptin levels than men of the same weight (corrected for body fatness). Estrogen appears to stimulate

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Update

Dental Diagnosis and Dilemma

The dentist may be the first health-care professional to suspect a person has an eating disorder. When 123 male and female dentists and dental hygienists in North Dakota were surveyed, 79% reported that they currently had at least 1 or more patients in their practices whom they suspected or knew had bulimia nervosa. Their suspicions arose from the patients' dental profiles, including appearance of abnormal dental erosion. Ninety-two percent of the dental professionals reported they might share their suspicions of an underlying eating disorder with the patient or parents; 42% note that they always do so. The two pieces of advice the dental professionals give to patients, before referring them for further care, are to rinse their mouths with water immediately after vomiting (52%) and to brush their teeth immediately after vomiting (46%). The vast majority of people with suspected eating disorders are referred to primary care physicians; only 3% of the dentists and dental hygienists refer such patients to a psychologist or psychiatrist. The survey results were reported at the Academy for Eating Disorders annual meeting in Boston last April.

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leptin production.

Obesity factors: Obese individuals produce much more leptin than non-obese individuals. This results from the following mechanisms:

1. Obese individuals have larger fat cells than those of nonobese individuals, and larger fat cells produce disproportionately more leptin than smaller cells.

2. Significantly obese individuals frequently have hyperplasia (an increased number of fat cells) compared to their leaner counterparts. Thus they have an increase in the number of leptin-producing cells.

Since leptin is produced primarily by fat cells, higher serum leptin levels are indicative of larger fat stores. Leptin reflects body stores in a steady state condition but it also reflects recent nutritional status. For example, in 1996 Kolacynski noted a 40% increase in leptin levels within 5 hours of overfeeding. Other studies show a greater than 50% decrease in leptin after a 24-hour fast. Once produced, leptin enters the circulation and exhibits its greatest effects in the central nervous system, specifically acting on neurons in the hypothalamus. Leptin binds to leptin receptors that affect the secretion of two other neuropeptides, neuropeptide Y (NPY) and pro-opiomelanocortin (POMC). Leptin causes *increased* synthesis and secretion of POMC and *decreased* synthesis and secretion of NPY in the hypothalamus.

NPY has been identified as one of the most potent stimulators of food intake. In mice, NPY leads to increased food-seeking behavior and reduces metabolic rate. Within increasing (physiologic) leptin concentrations, the secretion of NPY is diminished. POMC has the opposite effect by increasing metabolic rate and decreasing feeding behavior through its effects on other neurotransmitters. Researchers are still studying the exact actions of POMC in humans.

In the ob/ob mouse, a genetic defect prevents leptin production. When leptin fails to signal the

hypothalamus, the brain cannot sense the more-than-adequate nutritional status (fat stores) of the mouse. Thus, the mouse continues to conserve energy by minimizing its metabolic rate and continues to eat in an effort to compensate for what it believes are inadequate energy stores.

Human Obesity

Human obesity (with extremely rare exceptions) doesn't result from the lack of leptin. In fact, obese individuals have leptin levels that are sometimes as much as two to seven times higher than those of normal-weight persons. Unfortunately, leptin levels above a "normal" amount appear to have minimal effects on reducing food intake or increasing metabolism. This is not to say that leptin is not important in metabolic disorders or dysregulated appetite. Although leptin's role in metabolic disorders is fascinating, it is far beyond the scope of this brief review.

Leptin and Appetite

Appetite regulation in humans is extremely complicated and requires integration at three levels. The first level is socially or psychologically based, such as hunger perception and the behavioral effects that follow (such as consuming a meal). The second level comes from the periphery and involves metabolic factors (such as glucose levels, amino acid levels, and hormones produced in the periphery). The third level is the central nervous system, which involves the integration of the psychological and metabolic factors with various neurotransmitters, including leptin.

It appears that in many obese individuals, this third level of integration is overwhelmed by dysregulation of psychological and/or peripheral physiological factors. The higher levels of leptin found in obesity fail to increase metabolic rate or decrease appetite enough to prevent or reverse weight gain.

Leptin and Bulimia Nervosa

Bulimia nervosa is characterized by recurrent episodes of binge

eating combined with inappropriate compensatory mechanisms (such as vomiting postprandially or the use of laxatives) in an attempt to prevent weight gain. Some studies suggest this is caused by impaired post-ingestive satiation with diminished responsiveness of serotonin-mediated pathways. One group of women with bulimia nervosa showed decreased leptin levels as compared to age- and weight-matched controls, with a tendency toward an inverse relationship between frequency of binge-eating episodes with leptin levels. The degree to which leptin contributes to the abnormalities in eating and satiation in bulimia nervosa is not yet fully understood, but there is significant evidence to suggest a major direct or indirect effect.

Anorexia Nervosa and Leptin

Anorexia nervosa is the most serious of the eating disorders, and is characterized by a body mass index (BMI) of less than 17.5, compared with a "normal" BMI of 18.5 to 25. Since adipose tissue is depleted in anorexia nervosa, the levels of leptin are low, as would be expected. However, in some studies, weight regain in individuals with anorexia nervosa increased leptin concentrations much more rapidly than in weight-matched controls, which may contribute to early satiation and a possibly increased metabolic rate. If this is confirmed in future studies, it may be one of the factors that frustrate attempts to achieve and maintain a more normal weight in individuals afflicted with anorexia nervosa.

Conclusion

Although leptin has not been the long-anticipated silver bullet for obesity treatment as initially hoped, it has helped clarify the biological basis of not only obesity, but also of eating disorders and other metabolic disorders. Although this is far from certain, leptin may play a role in helping obese individuals maintain a reduced obese state, and may be helpful in the dysregulated appetite and satiation seen in the major eating disorders.

Suggest Reading

- Blundell JE, Goodson S, Halford JC. Regulation of appetite: role of leptin in signaling systems for drive and satiety. *J Obes Relat Metab Disord* 2001;May; 25; S29.
- Fried SK, Ricci MR, Russell CD, et al. Regulation of leptin production in humans. *J Nutr* 2000;130:3127S.
- Kim S, Moustaid-Moussa N. Secretory, endocrine and autocrine/paracrine function of the adipocyte. *J Nutr* 2000; 130:3110S.

Anorexia Nervosa: Questioning the Effectiveness of Estrogen Replacement

Osteopenia, or loss of bone density, is one of the most serious effects of anorexia nervosa. Estrogen replacement therapy has been one treatment option; however, after a recent study, some researchers are questioning this common practice (*J Pediatr Adolesc Gynecol* 2002;15:135).

In a prospective observational study of 50 teenagers with anorexia nervosa who were taking either calcium supplements or estrogen, Dr. N.H. Golden and colleagues found that estrogen-progestin therapy was no more effective for bone mineral loss than standard calcium supplements and nutritional intervention. They concluded this based on dual-energy x-ray absorptiometric measurements of bone mineral density (BMD) of the lumbar spine and left hip at baseline and one year later. When the young women were first seen, they were malnourished (79.5% of ideal body weight), hypoestrogenemic (the mean estradiol level was 24 pg/ml), and had reduced bone mass (the mean lumbar spine BMD was -2.01 SD below the young adult reference mean).

Estrogen therapy didn't increase BMD

Twenty-two adolescents took estrogen-progestin (20-35 mcg of

ethinyl estradiol) daily as an oral contraceptive and 28 had standard treatment alone. All were given calcium supplements and the same medical, psychological, and nutritional intervention.

At one-year follow-up, there were no significant differences in absolute values or net change in the lumbar spine or femoral neck BMD density between those who received estrogen-progestin and those who received standard treatment. Two and three years later, bone loss persisted and in some cases was progressive.

Are Some Vital Organs Spared in Anorexia Nervosa?

As women with anorexia nervosa lose weight, presumably all parts of the body are affected. However, in a pilot study at Columbia University, researchers have learned that certain vital organs, including the brain, liver, and heart, seem to be protected during extreme weight loss.

As Dr. Laurel Mayer reported at the Academy for Eating Disorders annual meeting in Boston, total body magnetic resonance imaging scans of 6 women with anorexia nervosa hospitalized on the eating disorders unit at Columbia University and 10 healthy controls showed no significant differences in weight of vital organs between the two groups. Skeletal muscle, total fat, spleen and kidney mass were lower in low-weight patients versus normal-weight controls. No significant changes in brain, liver, or heart mass were noted after the anorexic women and controls regained weight.

Other studies have shown a very different pattern. For example, Deborah Katzman and colleagues found that brain mass was affected in adolescent anorexics (*Semin Clin Neuropsychiatry* 2001;6:146). White and gray matter changes and reversible brain atrophy have been reported among adolescents (*Neuroradiology* 2001;43:838). Further studies are needed to show whether adolescents may be more vulnerable to the effects of the disease or whether a different pattern of changes occurs in teens.

'Student Bodies': An Online Self-Help Program

Computer-assisted health education programs have been effective for treating a number of problems, including anxiety, depression, and phobias. More recently eating disorders researchers have investigated how they might put the computer to work to help students at risk of developing an eating disorder.

"Student Bodies," a multimedia psychoeducational program, is currently being tested at San Diego State University and Stanford

University (*J Psychiatric Prac*

2002;8:14).

Since 1995,

Student Bodies has been used

by hundreds of

high school and college students,

and it is currently being evaluated

in a National Institute of Mental

Health-sponsored multi-site, long-

term trial to determine if the

program can reduce risk in students

likely to develop eating disorders.

Components of the program

The main parts of the program are psychoeducational readings on body image, nutrition, exercise, and eating disorders, a body image journal used to track thoughts and feelings, and an asynchronous newsgroup. Female students from the two universities were recruited through campus newspaper ads, flyers on bulletin boards and presentations given in dormitories and sorority houses. Women with a history of bulimia or anorexia nervosa, currently purging or body mass index <18 were excluded from the study.

The design of the program evolved through several stages: first it was delivered via CD-ROM, and discussion messages were sent by e-mail to an e-mail distribution list. A clinical psychologist acted as moderator for the discussion group. Anonymity was assured. However, participants complained that they had to locate a public computer

with a CD drive and load the program each time they wanted to use it, and felt the sound called attention to them in public places. Self-reported compliance was 53%.

The second and third models of the program became more structured, and compliance improved though the course content was unchanged.

Structure improved compliance

Currently, participants are given clearly stated assignments online,

and receive a reminder call from research assistants when an assignment is missed. A set number of group postings

with predetermined topics are also required each week, and participants are encouraged to post additional messages. The participants' perception of their progress is tracked with an online questionnaire that appears each time a student logs on to the program. The moderator for the group is a clinical psychology graduate student close in age to the participants.

Students read an article each week from a course reader, and are encouraged to write a one-page critical reflection paper each week. These are sent via e-mail to the moderator, who also serves as the course instructor. In an important change, anonymity within the group was eliminated through implementation of face-to-face meetings three times during the program for orientation and discussion; furthermore, photographs and personal statements from participants are used to introduce participants. Compliance has risen to 84%.

Suggestions for online programs

The authors offered several suggestions for using such an online system:

1. Tracking systems should be implemented in online interven-

tions. These can be used to determine what screens are not being accessed and what levels of compliance are needed to improve participation.

2. Electronic reminders can be programmed to be sent to participants when certain screens haven't been accessed. Weekly reminders, whether by e-mail or telephone, seem to be helpful.

3. Feedback from participants is essential.

4. Online programs are helped by a clear structure, including explicit expectations about participation and guided assignments.

5. A reward in the form of a grade or monetary payment seems to improve compliance.

Young Girls Who Eat When They're Not Hungry

The ever-increasing incidence of obesity among children has led concerned researchers on a serious mission to find underlying environmental or behavioral causes.

Results of a recent study provide the first evidence that eating when not hungry may represent a stable phenotypic behavior of young overweight girls. Parents' restrictive feeding practices may also contribute to this behavior (*Am J Clin Nutr* 2002;76:226).

Environmental cues count

Dr. Barbara Rolls and her colleagues have demonstrated that with time children become increasingly responsive to environmental cues, such as extra-large portions of energy-dense foods (*J Am Diet Assoc* 2000;100:232). In that study, 2-to 3-year olds ate roughly the same amount of a main course whether they were given a large or small portion. However, 4- to 6-year-olds ate 60% more when the portion size was doubled.

Exposure to tempting foods after lunch

In the current study, Jennifer

A definite structure, heeding feedback from participants, and rewards all seem to help online compliance.

Orlet Fisher and Leann L. Birch evaluated 192 non-Hispanic white girls and their parents, when the girls were 5 and 7 years of age. The girls' eating patterns were recorded over a two-year period when they were exposed to palatable foods after they ate a standard lunch and reported they were no longer hungry. After lunch, each girl was asked to rate 2 bite-sized samples of 10 sweet and savory snack foods, including popcorn, potato chips, pretzels, and ice cream. The experimenter then left the room for 10 minutes. When the experimenter returned, the girl was interviewed about whether her parents let her have the snacks and how she felt about her eating.

Eating without hunger increased risk

Eating without hunger was moderately stable during the 2-year period for most girls. Girls who ate large amounts of snack foods in the absence of hunger at 5 and 7 years of age were more than 4 times more likely to be overweight at both ages.

Food restriction predicted overeating. Parents' reports of restricting their daughter's access to foods at age 5 predicted girls eating in the absence of hunger at age 7, even after controlling for their weight and eating in the absence of hunger at age 5. The extent to which this may represent parental attempts to limit intake among children they already perceive as having a tendency toward obesity is unclear.

Most parents expressed a wish to help their children avoid eating "high-fat" foods. One-half of the mothers and three-fourths of the fathers were overweight (body mass index above 25).

An environment of high-fat foods, in large portions

The study showed evidence for

the environmental effects on overweight among children that result from what the authors term an "obesigenic" food environment.

This is a setting where a wide variety of energy-dense foods are available in "super-size" portions, yet

one in which parents may be restrictive about food.

The authors suggest that a healthier eating atmosphere for

A child with parental restrictions is much more likely to eat restricted foods, with or without being hungry.

BOOK REVIEW

Eating Disorders: Journey to Recovery Workbook

(Laura J. Goodman and Mona Villapiano, Philadelphia, Brunner/Routledge, 2001; 220 pp)

Eating Disorders: Time for Change

(Mona Villapiano and Laura J. Goodman; Philadelphia, Brunner/Routledge, 2001; 174 pp)

This paired set of workbooks for clients and therapists adds to the growing literature of similarly paired workbooks already available. These contributions, written by two experienced psychotherapists, come from a welcome perspective, focusing on the client's motivation and stages of change, using the now-familiar model that has been extensively applied to alcohol and substance abuse.

The client's book begins with images consisting of a tree of life, in which the eating disorder stems from roots and affects all the branches and the web of life. Next are discussions of set-point theory, dieting and emotions, and physical changes associated with weight. This presentation is thoughtful, but I wondered if some clients might be too easily put off by technical terms such as "thermogenesis" and "cognitions," which are introduced without literal explanations. The therapists with whom these workbooks are working may need to explain these terms.

The workbook nicely progresses through nutrition, assessing stage of change, relationships between spirituality and eating; body image; exercise;

children doesn't imply lack of structure, but instead suggests setting limits and offering children an array of healthy foods and portions sizes appropriate to their needs.

This study underscores the fact that children should be encouraged to focus on their own feelings of hunger and fullness as a guide to determining when eating begins and ends. Parents' use of restrictive feeding practices is not effective for eliminating a child's intake and can lead a child to eat restricted foods, with or without being hungry.

women's issues; men's issues for male patients; substance abuse; trauma and abuse. It also addresses special circumstances such as obsessive-compulsive disorder, depression and diabetes; the treatment team; "media madness," a section for family and friends; and a final reprise, followed by resources.

Most chapters are filled with open-ended structured questions, closed-ended questionnaires, tables, stories, poems, illustrations and affirmations. I particularly liked the way the chapters on substance abuse and trauma and abuse dealt with their material. The psychopharmacology chapter includes questions assured to make clients better consumers—with intelligent questions to ask prescribing physicians about medications and what to expect.

The therapist workbook doesn't exactly parallel the client workbook, which may prove confusing. To my mind, a book that more closely coincided with the client's version would have better served therapists. It could then be supplemented with additional material and discussion, as a sort of teacher's guide. Nevertheless, this volume does cover more or less the same ground as the client's workbook, focuses on motivation and stages of change, and contains much useful information and some worthwhile assessment tools.

Here's a suggestion: we need a consumers' guide to workbooks. Although a great deal of their value depends on the coaching of therapists, their intrinsic differences may make some workbooks more suitable for certain audiences than others.

—J.Y.

Taking the Fear Out of Dietary Fat

Fears about dietary fat are rampant among patients in the eating disorders community. For example, to boost flavor but avoid fat, clients sometimes use “culinary creations” such as ketchup instead of dressing on salad.

Beliefs about the perils of fat come from many sources. The most influential source, the media, has been a long-time and harsh critic of fat. It is not surprising that we live in a fat-phobic society, given the constant messages we receive about reducing our fat intake. Individuals with eating disorders may take these messages too far by believing if low-fat eating is good, no-fat eating must be even better.

For this reason, dietitians working with clients with eating disorders may choose not to use the nutrition education tools designed for the general public. The American Food Pyramid and the Canada Food Guide recommend a low-fat intake because a large segment of the North American population deal with chronic diseases associated with high-fat diets. Clients struggling with eating disorders are usually at the other extreme. They present with physical symptoms related to not enough fat in their diets. To promote moderation and balanced eating, dietitians may create their own nutrition guides.

Nutrition Education Ideas

Helping patients improve their fat intake requires an exploration of their beliefs. Providing them with accurate nutrition information can then allow the patient to determine whether their beliefs are true or not. Nutrition education alone will not necessarily change the client’s attitude or behavior about eating fat.

A common barrier to change is the belief that fat calories are stored more efficiently than calories from protein or carbohydrates. Another popular belief is that dietary fat is automatically converted to body fat. I challenge these beliefs by referring to relevant research findings. Although fats are a more concentrated source of food energy (9 kcal/gram) as opposed to protein or carbohydrates (both are 4 kcal/gm), a calorie is essentially a calorie. Too many calories from *any* source results in weight gain.

Another common belief among patients is that fat has no nutritional value. Clients are often surprised to learn that they can develop

to the aging process such as cancer, heart disease, and cataracts. Another important function of dietary fat is facilitating the absorption of all the fat-soluble vitamins (e.g., vitamins A, D, E, and K).

A sense of satiety. Having a moderate amount of fat in a meal or snack also helps to create a feeling of satiety after eating. Fatty acids produced in the digestion of fat stimulate the release of cholecystokinin. This hormone slows the gastric emptying rate and may play a role in limiting food intake.

Feeling more satisfied after a meal may help patients reduce their preoccupation with food, a symptom related to semi-starvation. Because clients are usually frustrated by the amount of time they devote to thinking about food and eating, this can be an attractive function of fat. I often ask patients to test this function by assessing their level of food preoccupation and hunger after having meals that contain a moderate amount of fat versus meals that contain hardly any hidden or added fat.

Helping Patients Add Fat to Their Diets

A number of my restricting patients have a nearly no-fat diet and are afraid they will be “corrupted” if they eat fat. They’re usually constipated and worried about it. I tell them they need about 3 teaspoons of fat in the diet for the bowels to function. This does work and is self-reinforcing.

I also stress how important the intake of certain fats, specifically omega-3 DHA, is for cognitive function. These fats help improve concentration, stabilize mood, and enhance serotonin. I use any of the patient’s unexpected poor performance on mental status tests to support this position. Later I use the improvement on mental status tests to reinforce continued fat intake.

—Alayne Yates, MD

a dietary deficiency from extreme avoidance of fat. Essential fatty acid deficiency is often seen in patients who are severely protein- and calorie-malnourished. The body cannot manufacture adequate quantities of linoleic acid and linolenic acid; therefore, we depend on food sources such as vegetable oils to get these nutrients. Physical manifestations of essential fatty acid deficiency are: dry, cracked, scaling skin, coarsening of the hair, hair loss, impaired wound healing, and possibly diarrhea.

Antioxidants. Some foods in the fats and oils group (e.g., avocados, nuts and seeds, vegetable oils, and wheat germ) are also excellent sources of the antioxidant nutrient, vitamin E. Antioxidants may play a role in preventing diseases that are related

What is Moderate Fat Intake?

A moderate intake of fat can be defined as 30% of the patient’s daily energy intake; both Dietitians of Canada and the American Dietetic Association endorse this recommendation. Often, patients balk at this suggestion and are even more surprised when they learn that the recommended intake for protein is even lower than this—approximately 20% of their daily energy consumption.

An example of a moderate fat intake is that a client with an 1800-kcal meal plan needs a total of 65 gm of fat from both hidden and added sources. This is equivalent to about 12 to 13 teaspoons of oil. I am cautious about discussing the recommended number of fat grams with patients. Many do not find this information helpful, especially if

they are struggling with counting calories and/or fat grams.

When I think the client is eating too little fat, I occasionally estimate her fat intake by reviewing her typical eating pattern. Then, I give her feedback about her intake by comparing it to the recommendation of 30% of total calories per day. Informing the client that she is eating only half the amount of fat advised for moderation may be a good reality check.

Strategies for Improving Dietary Fat Intake

Monounsaturated fats may be easier to incorporate into a patient's eating pattern than other types of fats. These fatty acids are found mostly in nuts, such as almonds, cashews, hazelnuts, pecans, and peanuts. Other good sources are olives, olive oil, peanut oil, canola oil, and avocados. Appropriate servings of these foods are: 1 tablespoon of chopped nuts (with no shells), 1 teaspoon of oil, 10 small or 5 large olives, and 1/8th of a medium avocado.

Many patients are aware that, unlike saturated fats (fatty acids that come from land animal sources), monounsaturated fats do not raise serum cholesterol levels. Another bonus is that they do not lower serum levels of the "good cholesterol," high-density lipoprotein (HDL).

Nut butters, like peanut or almond butter, may be more attractive options for patients who cope better with less-visible fats. These foods will also be excellent sources of monounsaturated fats if they have not been hydrogenated. Natural nut butters are often marketed as "old-fashioned style," and need to have the liquid oil, which is usually floating on the top, mixed into the butter before it is eaten. Two teaspoons of nut butter is an appropriate serving of fat.

A Healthful Trio: ALA, EPA, and DHA

Flaxseed oil seems to be a popular choice for clients who regularly visit health food stores. Flaxseed is the richest source of alpha-linolenic acid (ALA), an

essential omega-3 fatty acid. A 1-tablespoon serving of flaxseed is appropriate. Canola oil, soybean oil, and walnuts also contain ALA. Cold-water marine animals such as salmon, tuna, herring, and mackerel are excellent sources of two other kinds of omega-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These fatty acids down-regulate inflammatory prostaglandins, and may have a beneficial effect against autoimmune diseases like arthritis, lupus, and other inflammatory conditions. Research has also demonstrated that ocean fish, fish oils, and fish oil capsules containing EPA and DHA may lower cholesterol and triglycerides, which could lower the rates of sudden cardiac deaths.

Fish oil capsules should not be used as a way of improving a client's intake of dietary fat. In high doses, the capsules can cause harm, especially if the individual regularly takes aspirin or blood-thinning medications such as warfarin.

A Step-By-Step Process

The best approach for incorporating fat into the diets of extremely resistant patients is to start them out with low-fat food items (e.g., light cream cheese, diet margarine, or low-fat salad dressing). However, I do not recommend using ultra-low-fat foods as an option. After the client has been able to master this addition, I ask her to double the amount of the low-fat item, and use this as a "baby step" toward eating the regular fat version of the food.

—Linda M. Watts, MA, RD

Young Vegans May Lack Some Essential Nutrients

Teens who choose a vegan lifestyle (a vegetarian diet without dairy products) may not get enough riboflavin, vitamin B-12, vitamin D, calcium and selenium in their daily diets without taking dietary supplements, according to Swedish nutritionists.

Since about 5% of Swedish

students aged 16 to 20 eat a vegetarian school lunch, this group of teens posed an ideal testing ground for the nutritionists, Drs. Christel L. Larsson and Gunnar K. Johansson, of Umea University (*Am J Clin Nutr* 2002; 76:100). The two researchers studied the dietary intakes of 30 vegans (15 males and 15 females) and 30 sex-, age- and height-matched omnivores. The mean age was 17.5 years. All 30 vegans had stopped eating meat primarily for ethical reasons; 4 also added health reasons. Nutritional intake was established through diet histories taken during interviews, use of the doubly labeled water method, and measurements of nitrogen, sodium, and potassium excretion in urine. Iron status and serum vitamin B-12 and folate concentrations were measured through blood samples.

Results

First, male vegans weighed less than, and had a lower body mass index (BMI) than, male omnivores (22.0 and 20.5, respectively). There was no difference in BMI and body weight between female omnivores and female vegans.

Next, both vegans and omnivores got a large portion of their daily energy intake from between-meal snacks (33% and 40%, respectively). The vegans had dietary intakes (excluding supplements) that were lower than the average daily requirements for riboflavin (males only), vitamin B12 and vitamin D (females only), calcium, and selenium. Calcium and selenium intake remained low for vegans even with dietary supplements. Six (20%) of female vegans and 7 (23%) of omnivores had low iron levels; low iron was as common among omnivores as vegans. As a group, the vegans had lower vitamin B12 levels and higher folate concentrations than did omnivores.

Finally, the authors note that it is important for adolescents in general, and teenaged vegetarians in particular, to be given practical information about how to combine and prepare a healthy daily diet.

Questions & Answers

The Unselfish Anorexic

Q: Some of my anorexia nervosa patients seem particularly “self-less.” They’ll take on major projects to help other people without really seeming to care what happens to themselves. Sometimes they seem to be pretty self-destructive, almost suicidal in their self-neglect. Is this behavior a suicide equivalent? (E.R., Atlanta, GA)

A: That’s an interesting observation. Many clinicians encounter patients with anorexia nervosa who appear to be extremely altruistic, passionate about “doing good” for others, and acting as people-pleasers, as if they are trying to make others think highly of them and not to become angry with them. Recently, some researchers have distinguished what they call “rejection of life,” or not caring too much about living, from “death preoccupation,” i.e., contemplating or being attracted to death, in some patients with anorexia nervosa and bulimia nervosa. The notion of “rejection of life” reflects the fact that these individuals are attuned to the needs of others and refrain from promoting their own needs or interests. In these researchers’ terms, this way of being in the world often reflects “...a struggle to exist, but to exist in the narrowest possible parameters.” This mode of existence may relate to the fact that

many of these patients feel extraordinary guilt at anything that would seem to be promoting their own self-interests, as if they have no right to exist or to want anything for themselves (*Int J Eat Disord* 2002; 31: 43). It remains to be discovered just how pervasive this phenomenon is among women with eating disorders (or others for that matter) and what correlates with these findings.

Bulimia Nervosa: Infertility Is Uncommon

Patients with bulimia nervosa who are concerned about their future ability to conceive will be reassured by the results of a recent long-term study. Menstrual irregularities are common in bulimia nervosa, but infertility is not, according to a study that followed bulimic women for 10 to 15 years (*Am J Psychiatry* 2002;159:1048).

Scott J. Crow, MD and his co-workers studied 173 women with bulimia nervosa an average of 11.5 years after their bulimia nervosa was discovered. At baseline, the mean body mass index was 21.2, and 38.2% of the patients reported regular menses. Irregular menses were reported by 29.5% of the women and very irregular menses were reported by 27.7%. Eight women (4.6%) had amenorrhea.

At follow-up, 83.2% of the subjects were menstruating; 2% were pregnant (5) and 13.9% had amenorrhea unrelated to pregnancy. Amenorrhea outside of pregnancy

was associated with a lower body mass index at follow-up (21.4) than was amenorrhea with pregnancy. At follow-up, 74.6% of the women had been pregnant at least once. Of 44 subjects who had never been pregnant (25.4%), only 3 (1%) of the total group had tried to conceive but been unable to do so.

Nibbles, by Hunter



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