

EATING DISORDERS REVIEW®

PATIENT INFORMATION HANDOUT:
Herbal Products: Balancing the Good and Bad



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Herbal Agents Used by Eating Disorder Patients

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Individuals with eating disorders no longer have to obtain a prescription or use over-the-counter drugs to lose weight or suppress their appetite. Instead, the whole arena of “dietary supplements” is now open for them to explore.

They may come upon herbal preparations that appear to help in weight loss

or appetite suppression, or that promise to raise mood or decrease anxiety. Unfortunately, they may also encounter agents that cause significant side effects and/or drug interactions. Use of such products can also delay appropriate treatment.

Often these agents are thought to be safe because they are “natural.” Extrapolation to over-the-counter and prescription medications may lead consumers to expect that these products must have demonstrated effectiveness because, after all, they are on the market.

Background

In 1994, Congress passed the Dietary Supplement and Education Act (DSHEA),¹ which relegated herbal agents to the category of “dietary supplements.” In effect, this removed these products from the purview of the Food and Drug Administration (FDA). Currently there are no standard requirements that herbal products be either safe

or effective. In addition, no government agency inspects the manufacture or degree of purity or accuracy of contents of these products. Thus, there is no way to know how much of the ingredients listed are really present in any sample. As a result,

herbal products are a completely unregulated source of potentially dangerous

products readily available to anyone who wants to lose weight.

A recent review demonstrated how widespread use of these agents is in the general population.² Between 1990 and 1997, use of herbal therapies increased by 380%, and in 1997 alone consumers spent \$5.5 billion on herbal therapies.

Advertising for these agents is also ubiquitous. Magazines, newspapers, TV, and health food stores, grocery stores, and pharmacy outlets are prominent sources that extol the advantages of these preparations. In addition, direct consumer mailings, many targeting teenage girls, encourage patients with eating disorders to read fantastic claims for

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Update

Chewing and Spitting Out Food

Purging and restricting behaviors are commonly reported among patients with eating disorders. However, one type of behavior—chewing food and spitting it out without swallowing—hasn't really received very much attention. According to Dr. Leslie Heinberg and colleagues at Johns Hopkins University School of Medicine, Baltimore, and the University of California, San Francisco, chewing and spitting out food without swallowing may indicate a more severe eating disorder. The researchers, who reported the results of their study at the recent Academy for Eating Disorders annual meeting in Boston in April, evaluated the frequency of this behavior among 197 consecutively admitted inpatients; 95% were female, and the average age was 25. Nearly 20% of patients in the study reported chewing and spitting out food at least several times a week. This subgroup had greater drive for thinness, more body dissatisfaction, and reported greater ineffectiveness and poorer physical functioning. Restrictive eating behaviors, purging by vomiting, and excessive exercise were also more common among those who spat out food.

60% of patients do not disclose the use of herbal medicines to their physician.

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these agents and how to acquire them. The Internet has exploded with an abundance of claims for various herbal preparations. The question may not be “if” your patient is taking one of these agents, but “when” and “how many” of the preparations are being consumed. Remember that 60% of patients do not tell their physicians that they are using herbal products.²

Types of Agents

We recently published an overview of alternative medications used by eating disorder patients, including a review of locally available preparations.³ A partial list of active ingredients found in the various types of preparations is shown in *Table 1*.

Weight loss agents. This category includes a number of different types of products. The only type of treatment that has actually produced short-term weight loss is the combination of ephedrine and caffeine.⁴⁻⁷ This type of product may be marketed as a drug combination or as the herbal sources, which include ma huang (ephedrine) and a caffeine source such as guarana seed or Kola (cola) nut. However, extrapolating results from clinical trials that employ pharmaceutical-grade ephedrine and caffeine or research studies using standardized herbal preparations to the use of botanical sources available to a patient is not without difficulties. The botanical sources chosen by the patient may not have the same amounts of either ingredient in the preparation despite the labeling (there could be more or less). Attempts to identify empirical data concerning the effectiveness of other putative weight loss products or ingredients have proven unsuccessful. Unfortunately, the available studies are few and often methodologically flawed.^{8, 9}

Laxatives. Laxative preparations are often combinations of multiple ingredients, many of which have little to do with producing the laxative effect. Such products may contain either herbal or pharmaco-

logical sources of stimulant cathartics, such as bisacodyl, cascara sagrada, or senna. Other potent cathartics, such as magnesium hydroxide or citrate, may be included. Methylcellulose or other bulk laxatives may also be added. At first, these products are quite effective at producing diarrhea. However, over time stimulant cathartics tend to lose their efficacy and can result in a hypoactive bowel.

Diuretics. Diuretic agents are mostly represented by sources of caffeine (guarana seed, cola nut) or the mild diuretic pamabrom. Mild diuresis can be obtained with these products but they are not as effective as prescription agents such as furosemide (Lasix®) or hydrochlorothiazide (HydroDIURIL®).

Adverse Effects

The magnitude of the risk of side effects associated with herbal therapies is well illustrated by a recent case. An herb, *Aristolochia fangchi*, was inadvertently substituted for another, *Stephania tetrandra*, in a weight-loss preparation compounded in Belgium. Seventy individuals who took the herb developed complete renal failure, and 50 more had kidney damage. Cancerous or precancerous lesions were found in 37 of 39 who had a kidney removed.¹⁰ This unfortunate incident is a dramatic illustration that “natural” agents can have very undesirable effects.

Caffeine and ephedrine. Caffeine, found in many beverages, can cause mild agitation, insomnia, tremor, and diuresis. However, ephedrine, which is often combined with caffeine in products, generates a larger concern. Sudden death (myocardial infarction), hemorrhagic and ischemic stroke, acute hepatitis, nephrolithiasis, dizziness, tachycardia, headache, tremor, nervousness, and insomnia have all been reported with this combination.^{4, 11-17} The most common adverse events identified in a recent report included: hypertension (17 reports), palpitations, tachycardia or both (13), stroke (10), and seizures (7). Ten events

resulted in death and 13 events produced permanent disability. Fifty-nine percent of the users were taking the supplements in order to lose weight.¹¹

Yohimbine. Occasionally appetite suppressants will contain yohimbine, an indirect adrenergic agonist. Anxiety, elevated blood pressure, queasiness, sleeplessness, tachycardia, tremor, and vomiting are potential side effects related to this agent.¹⁸

St. John's

Wort. St. John's Wort has been included in many preparations.

Photosensitivity, gastrointestinal irritation, allergic reactions, tiredness, and restlessness have been reported, along with drug interactions. Recently St

John's Wort has been reported to stimulate the CYP450 3A4 enzyme, which is involved in the metabolism of many important medications. Stimulation of this enzyme's activity may lead to a reduction of the blood levels of certain concomitant medications, including cyclosporine, indinavir or possibly oral contraceptives.¹⁹⁻²¹

Chromium picolinate. This product has been reported to be associated with cases of hypoglycemia, nephrotoxicity (at high doses), dissolution of muscle tissue, acute generalized pustules, as well as cognitive and personality disturbances.²²⁻²⁹

Laxatives. As mentioned earlier, laxative products often contain a form of a stimulant cathartic (bisacodyl, cascara sagrada or senna). Problems associated with these agents³ include:

Bisacodyl—GI irritation, fluid and electrolyte loss, cramping, development of tolerance.³⁰

Cascara sagrada—severe vomiting with fresh bark, electrolyte imbalance with misuse, hypokalemia potentiates the toxicity of cardiac glycosides and thiazide

diuretics.³¹

Senna—hepatitis, abdominal cramping, nausea, electrolyte disturbance (e.g., hypokalemia, hypocalcemia, metabolic alkalosis or acidosis). Increased mucus secretion, reduced spontaneous bowel function, and melanotic pigmentation of the colonic mucosa (melanosis coli) may confirm laxative abuse.^{32,33}

ingredients that are very potent and pharmacologically active. Along these lines it should not be surprising that adverse reactions and drug interactions may follow. Uncertainty about these issues stems from the lack of data regarding effects of various herbs, concerns as to what the active ingredients may be, the lack of the practice and enforcement of good manufacturing

standards and a lack of a mechanism for adverse reaction reporting.

Recently Congress created the National Center for Complementary and Alternative Medicine (NCCAM) at the National Institutes of Health (NIH), which may help in addressing some of these issues. The mission of this organization is "to explore

complementary and alternative healing practices in the context of rigorous science; to educate and training CAM researchers; and to disseminate authoritative information to the public and professionals."⁴⁶ The Center has set out to investigate certain herbs for efficacy, currently St. John's Wort, ginkgo biloba, saw palmetto, and glucosamine/chondroitin, which has shown some evidence of efficacy. This process is long overdue but may not produce clinically applicable data soon.

Thus, clinicians treating patients with eating disorders should attempt to identify any alternative medicine treatments the patient is currently using or has used. Then, consulting the literature and/or individuals knowledgeable about herbs will help in determining if the individual has adverse reactions or drug interactions associated with these therapies.

TABLE 1: ACTIVE INGREDIENTS IN SOME ALTERNATIVE DRUGS

WEIGHT LOSS AGENTS	LAXATIVES	DIURETICS
Caffeine (guarana seed, kola nut)	Bisacodyl	Guarana seed (caffeine)
Ephedrine (Ma huang)	Bladder wrack kelp (iodine)	Kola nut (caffeine)
Chromium picolinate	Cascara sagrada	Uva ursi extract (bearberry)
Ginkgo biloba	Ginkgo biloba	
Stimulant laxatives (Cascara sagrada, Senna)	Magnesium hydroxide or citrate	
St. John's Wort	Rhubarb	
Yohimbine	Senna	
	Skullcap	
	Wahoo bark	
	Wild yam root	

Another ingredient in laxatives, bladder wrack kelp, is a source of iodine, which may potentiate hyperthyroidism, worsen preexisting acne, or lead to new acneiform eruptions.^{34, 35}

Other herbs. Skullcap has been associated with hepatic toxicity.^{41, 42} The berries of wahoo bark are toxic. Reactions may include GI symptoms (colic, bloody diarrhea), elevation of body temperature, circulatory disorders, elevated cerebrospinal fluid pressure, stupor progressing to unconsciousness, or tonic clonic spasms.⁴³ Wild yam root has produced a picrotoxin-like effect (tonic clonic spasms headache, dizziness, nausea) on overdose.⁴⁴ The mild diuretic uva ursi has been reported to cause nausea and vomiting.⁴⁵ Other agents include ginkgo biloba, which has been associated with headache, GI upset, allergic skin reactions, and several cases of cerebral hemorrhage, possibly related to its interaction with platelet aggregation and thrombolytic therapy.⁴²⁻⁴⁶

Conclusion

Herbal therapies may include

Note: For a copy of the references to this article, please send a self-addressed stamped envelope to: Eating Disorders Review, 302 S. Pinto Place, Tucson, AZ 85748-6902.

Tantalizing Neurobiological and Genetic Clues to the Development of Eating Disorders

Mary K. Stein, Managing Editor

Genetics, neurobiology, and environment each contribute to the development and perpetuation of eating disorders, according to four experts who participated in a special plenary session on neurobiology and genetics at the 2002 Academy for Eating Disorders meeting in Boston in April. As Moderator Dr. B. Timothy Walsh told the audience, “The nature versus nurture debate of 25 years ago has been succeeded by the clear recognition of an interaction between genes, biology, and the environment.”

The genetics of anorexia nervosa

Twin and family studies suggest substantial heritability for anorexia nervosa, according to Wade Berrettini, MD, PhD. Dr. Berrettini also reported that investigators from the Price Foundation study of anorexia nervosa have identified a probable susceptibility gene for anorexia nervosa located at chromosome 1p34.

Noting that the general public believes that what is “genetic is written in stone,” Dr. Berrettini added that health professionals need to educate the public about the concept of a susceptibility gene. “A single susceptibility gene is neither necessary nor sufficient for the development of AN,” he said, and added that an affected person must inherit several susceptibility genes, each of which produces a small increase in risk for the disorder. Even then, the appropriate environmental influences must also be present.

In family studies of AN, there is a substantial relative risk among first-degree relatives of AN probands, compared with first-degree relatives of control probands, Dr. Berrettini said. He also pointed to three characteristics of partially inherited susceptibility: First, partially inher-

ited disorders tend to aggregate in families, and all types of behavior disorders show familial aggregation. A second characteristic is that the monozygotic (identical) twin concordance is substantially greater than the dizygotic (nonidentical) twin concordance in a host of behavioral disorders, including AN. This information can be used to estimate the fraction of the total risk for AN that can be attributed to genetic influences, he said. A third characteristic of partially inherited disorders comes from adoption studies showing that increased risk for the biological relatives of adoptees with the disorder. Any two pairs of siblings, by virtue of the fact that they have the same mother and father, share 50% of their DNA sequences, he said.

Biocultural influences on the development of eating disorders

Anthropologist Carol Worthman, PhD, outlined biocultural influences on the development of eating disorders. Although she doesn’t directly study eating disorders, she does study the interface between biology and culture, she said.

One of the measures of sociocultural change is age at menarche, and researchers have learned that humans are “very plastic” with respect with to growth and development,” she said. Changes in the timing at menarche can be affected by illness, nutrition, maternal background (maternal well-being in childhood and then in pregnancy), and cultural practices are now playing an enormous role. For example, among the Bundi tribe of the central highlands of New Guinea, the median age at menarche in the mid-1960s was 18 years. Tribal members who moved into more urban areas had more access to better nutrition, including high-protein foods. Over a short

time, there was a decline of 0.8 years in menarche among the rural tribal members but a decline of more than 2 years among those in the urban setting.

Dr. Worthman noted that dehydroepiandrosterone sulfate (DHEAS), an adrenal androgen that shows a flex point at puberty and proceeds to climb linearly across the second decade and into the middle of the third decade, is a good marker of progression into puberty during a period when hormones are rising and falling. Urban children have higher levels of this hormone, presumably because they are at more advanced stages of development. The rural kids lag behind, she said, but the difference between the two groups diminishes with age. DHEAS peaks at age about age 25, so in many settings the rural children never catch up with the urban kids. This example of delayed development may be due to differences in the neuroendocrine system, and may have long-term consequences for neuroendocrine regulation. Typically, male and female Americans have very high levels of DHEAS. Lifetime patterns of steroid exposure can affect the amount of ovarian activity over the life span, Dr. Worthman said.

A study of depression in teens. Dr. Worthman also described some of the findings from the Great Smoky Mountain Study, which has been conducted over the last 8 years in 11 counties of western North Carolina. This population-based study includes about 1500 preteens and teens, 1100 of whom are population samples drawn from those counties and the other 450 of whom are a total sample of age-appropriate Cherokee. Researchers are trying to discover why girls develop depression later in life than boys, and

have twice the rate of depression of boys by age 15. Boys are more likely than girls to be depressed at an early age, said Dr. Worthman. Another area of study is the significant increase in depression reported in girls as they become teens. She noted there is a linear relationship between estradiol levels and the prevalence of depression in girls.

The study has shown an environmentally interactive effect between depression and cortisol response. In low-risk environments, depression was associated with a lowered cortisol response. In high-risk environments, depression was associated with a high cortisol response.

“Because the ecologies in which kids grow up affect their gonadal function, on all kinds of levels, it is interesting to consider the relationship between gonadal steroids and the risk for mood disorders such as depression and the risk for mood disorders at adolescence in girls. Both estradiol and testosterone are implicated suggests that it may not just be the gonads but the adrenal glands as well,” said Dr. Worthman. She added, “I am more and more impressed by the lack of power of our models for dealing with mood and behavior disorders in boys. We have made enormous headway with respect to girls...but not boys. Our models for males are not yet as good as those for girls.” Males are also at risk for all sorts of risk-taking behaviors, including distortions in body image, she said.

The serotonin connection

“In light of recent findings, we have to find a place for serotonin mechanisms in a biopsychosocial framework on eating disorders development,” said Howard Steiger, PHD. He added that the real challenge is finding ways to specify how psychological factors, biological factors, and social imperatives favoring dieting and pursuit of thinness come together to explain why a person develops an eating disorder. Many useful clues come from comorbidity patterns, he said.

People with restricting patterns

have a propensity to affective disturbances, anxiety disorders, and trait pathology characterized by perfectionism, preference for sameness, order, and control, and overly controlled, rigid, brittle behavior. In contrast, the person who binge-eats and purges is prone to anxiety and depression, but has a different spectrum of trait compo-

nents, including impulsivity, mood lability, substance abuse, and dissociation. This is a far more dysregulated kind of profile, he said.

Gender-based effects of serotonin. Dr. Steiger reported that there is also evidence implicating serotonin in the development of

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BOOK REVIEW

Body Image, Eating Disorders and Obesity in Youth: Assessment, Prevention and Treatment

(J. Kevin Thompson and Linda Smolak, eds. Washington, DC: American Psychological Association Inc, 2001;403 pp: \$39.95; ISBN: 1-55798-758-0)

This well-edited book, compiled by two authorities in their respective fields, covers a broad array of subjects of interest to those working with weight-related issues in children and adolescents.

Its four substantial sections, in turn, consider “Foundations,” i.e., developmental aspects of eating and body image in children and adolescents; and “Risk Factors,” including family functioning (and dysfunctioning), protective factors, and issues of sexual abuse (why not psychological abuse and physical abuse too??). Two more sections include “Assessment,” with separate chapters on physical status, body image and eating disturbances in eating disorders and obesity; and “Prevention and Treatment,” dealing with body image disturbances, obesity and eating disorders in children and adolescents.

All these sections are written by well-known authorities; some of this work has appeared in other recent edited volumes that have focused on developmental issues and prevention. But other work described here has not appeared in the mainstream.

Several chapters contain excellent evidence tables outlining major findings of the individual studies reviewed in the text. Although the 14 chapters in this volume include many superb ones, several, out of my usual range of reading, particularly caught my eye. Fischer and Birch’s chapter on early development reviews what is known about the role of maternal diet in early taste and flavor experiences,

influences of breast and formula feeding, food acceptance patterns in infancy and toddlerhood, and very early parental influences and modeling in shaping and controlling food choices, preferences and dislikes. Douchis, Hayden and Wilfley’s review of obesity, body image and eating disorders in ethnically diverse children and adolescents draws attention to the tremendous epidemiological health problems associated with high rates of obesity in children and adolescents of particular ethnic groups (the “New World Syndrome”). It discusses what is currently known of the social and cultural forces contributing to this emergence. Empirical data on effective prevention and intervention in these areas is, unfortunately, extremely limited.

Levine and Smolak review primary prevention, providing an excellent synthesis and literature review of body-image disturbance and eating disturbance, and Robinson and Killen do the same for obesity prevention. A final chapter by Sarwer on plastic surgery in children and adolescents takes us a little bit afield, considering the lengths to which children—and their parents—go to contend with body image dysphorias and body dysmorphic disorder symptoms, in addition to dealing with socially and sometimes physically impairing disfigurements. National statistics on cosmetic surgery for adolescents reveal that nearly 25,000 procedures were performed in 1998, about a third of which were for rhinoplasty. Here’s a bit of trivia for you: In 1998 surgeons performed 1840 breast augmentations for adolescent females and 1862 breast reduction surgeries for gynecomastia in adolescent males.

All in all, this worthwhile collection will be most useful for individuals involved in school and community-based programs concerned with early prevention of body image disturbances, obesity and eating disorders.

—J.Y.

Determining Energy Needs for Patients with Eating Disorders

“Eat when you are hungry and stop when you are full” is an expression I wish I could use with clients. After all, this is how many individuals meet their daily energy needs. But, for most clients with eating disorders, this mantra is not appropriate. For one thing, patients typically do not experience accurate hunger and fullness cues in the early stages of their nutritional recovery. Secondly, even if they do have accurate internal cues, they usually struggle with the ability to give themselves permission to eat. That is the nature of an eating disorder.

A Need for Structure

Generally, patients need a considerable amount of structure when normalizing their eating pattern during recovery. They require guidance about when, what, and how much to eat because the eating disorder has distorted their perception of normal, healthy eating behavior.

Mechanical eating and meal plans are two tools dietitians use to get patients back on track. Mechanical eating determines when the patient should have meals and snacks by suggesting she eat every three to four hours. Meal plans, which are designed with the client's input, determine the content of meals and snacks in terms of the food groups and recommended number of servings.

Determining Energy Needs

Unlike the internal cue approach to eating, however, meal plans entail providing clients with a specific caloric prescription. Dietitians determine the amount of food energy necessary to improve the client's nutritional status and restore their weight, but not make them vulnerable to refeeding

complications. The assessment of energy needs in this population can be challenging because many questions remain unanswered regarding the effects of an eating disorder on energy expenditure.

In the May 2002 edition of the *International Journal of Eating Disorders*, authors de Zwaan, Aslam, and Mitchell summarized what we currently know about this topic. De Zwaan and her col-

In the short term, changes in energy intake are more important than body weight per se in determining energy expenditure for patients with anorexia nervosa.

leagues reviewed the available literature concerning the energy expenditure of individuals with anorexia nervosa, bulimia nervosa, and binge eating disorder (*In J Eat Dis* 2002; 31:361). While the majority of studies measured resting energy expenditure (REE), several measured total energy expenditure using the doubly-labeled water method in both anorexia nervosa and bulimia. A few studies looked at components of total energy expenditure in anorexia nervosa, specifically, dietary induced thermogenesis and activity-induced thermogenesis. It is important to note that REE is the amount of energy used when the body is at complete rest (but the client is not asleep), not controlling for the level of stress and body temperature. REE is one of four components of total energy expenditure.

What the Studies Showed

The authors discovered that the data regarding the impact of binge-eating and purging behavior on REE are inconsistent. To date, it is still not clear whether clients with bulimia nervosa experience the energy-conserving metabolic adaptations that are characteristic of semi-starvation. But for individuals with anorexia nervosa, this is well known. In fact, the most consistent finding noted by the reviewers is a marked reduction in REE when anorexic patients are initially seen

at a low weight. Energy expenditure increases rapidly, however, as they progress with refeeding. In other words, in the short term, changes in energy intake are more important than body weight *per se* in determining energy expenditure for patients with anorexia nervosa.

The authors report that REE is measured most often in clinical settings because it can be done relatively simply using indirect calorimetry. This procedure measures an individual's consumption of oxygen and production of carbon dioxide using either a respiratory chamber or a ventilated hood. Unfortunately,

indirect calorimetry is not an option for most clinicians because of the high cost of the equipment. Instead, they may use predictive equations to estimate the resting energy expenditure of their patients. However, de Zwaan and her team advise clinicians to be cautious about using predictive equations to calculate REE for low-weight patients. Formulas such as the Harris-Benedict Equation or even equations that have been derived specifically for female patients with anorexia nervosa may yield inaccurate estimates of REE.

De Zwaan and her colleagues concluded their review article by recommending that more research be devoted to the subject. They also suggest that future research efforts be designed to further study energy expenditure of individuals with bulimia nervosa and binge eating disorder. As well, the authors believe that future research could lead to reliable and cost-effective ways of measuring REE for patients with anorexia nervosa. Accurate predictions of the energy intake necessary to promote weight restoration for these clients would be clinically useful.

Impact on Clinical Practice

While many in the eating disorders treatment community may not completely understand the effects of an eating disorder on energy expenditure, this review

article can give clinicians some reassurance. Several of the observations made by de Zwaan and associates support current treatment practices. Dietitians in this field are already predicting energy levels that achieve weight goals for patients without putting them at risk of refeeding complications.

Most dietitians have their own method of determining the energy needs of their patients. These methods tend not to rely on predictive equations or indirect calorimetry, but on lessons learned from their clinical experience, making it more of an art than a science.

A colleague, Jadine Cairns, a master's candidate and dietitian with the British Columbia Children's Hospital Eating Disorders Program, recently shared her method of determining energy needs. Cairns reports that she does not use a predictive equation. In the past, she has tried a formula published in 1995 by Schebendach and associates (*Int J Eat Disord* 1995;17:59) but found the equation estimated energy needs that she felt were too high for her patients.

Cairns' approach begins with determining the patient's current daily energy intake. For patients who are considered at low risk of refeeding syndrome, their current food intake determines the caloric content of their initial meal plan. However, she designs the initial meal plan with at least 1200 kcal/day. Typically, patients begin treatment with caloric prescriptions that range from 1200 to 1800 kcal/day. Clients who struggle with bulimia nervosa and are somewhat close to a healthy weight are prescribed at least 1600 to 1800 kcal/day even if they report eating very little. To promote weight gain, Cairns adds 350 to 500 kcal until the client's weight goal is reached. For patients considered at a high risk of refeeding complications, she designs the initial meal plan with a caloric prescription of about 800 kcal/day until their serum values for electrolytes, magnesium, and phosphorus are within normal ranges.

—Linda M. Watts, MA, RD

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eating disorders. Women have higher degrees of basal serotonin metabolism and apparently greater sensitivity to the serotonin system. He cited a recent study by Cowan showing that 3 weeks of modest dieting in women substantially alters serotonin levels. Thus, women are much more susceptible to the potential serotonergic implications of dieting than are men.

According to Dr. Steiger, in active cases of bulimia there is clear evidence of reduction of serotonin tone, blunted prolactin response to serotonin agonists, and reduced platelet binding of serotonin uptake inhibitors. In addition, dietary manipulations that reduce tryptophan also reduce availability of brain serotonin, and this exacerbates binge-eating among active bulimics. Active bulimia goes with reduced serotonin activity, and this is true during recovery as well. Kaye and colleagues have found various forms of normalization of serotonin activity in recovery, but evidence shows ongoing abnormality on the 5-HT_{2A} receptor, and ongoing sensitivity to tryptophan depletion. There is also some evidence of persistence of serotonin anomaly in recovered bulimics.

In anorexia, one should see elevated serotonin tone, but in the active state there is a blunted prolactin response to serotonin agonists, decreased binding of serotonin uptake inhibitors, and reduced serotonin metabolism in bingeing-purging. Perhaps this is explained by phenomena during recovery where various serotonin indices normalize. If so, this would suggest that the reduction in serotonin activity seen in active anorexics has a lot to do with secondary effects of malnutrition.

Dr. Steiger noted that serotonin status can be related to psychopathological profiles among eating disordered patients. One subgroup seems to have relatively intact serotonin function, even though they have severe eating disorders; a second group has greater serotonergic dysregulation.

For patients with more intact serotonin systems, stabilizing eating and stopping dieting behaviors will improve their outcome. In the comorbid group, where developmental experiences have led to more fundamental dysregulation, pharmacologic and special psychotherapeutic adjuncts will need to be aimed at impulsivity and posttraumatic sequelae, Dr. Steiger said.

Genetic and environmental interactions: an animal model

Genetic and environmental influences team to produce modifications in behavior, according to Stephen J. Soumi, PhD. Traumatic experiences early in life may also play an important role, he said.

In his long-term studies with rhesus monkeys, Dr. Soumi found that from 5% to 10% of the monkeys seem to be unusually impulsive and inappropriately aggressive in response to mildly stressful conditions. This modification can take place not only at the level of behavior but or of physiology, or of neurotransmitter level or metabolites, but possibly even at the level of gene expression as well.

Genetic and environmental factors appear to interact throughout development to shape individual differences, he said. He added that the early environment is particularly important.

The Panel

Dr. Walsh is Ruane Professor of Pediatric Psychopharmacology at Columbia University and Director of the Eating Disorders Research Unit at The New York State Psychiatric Institute, New York, NY. **Dr. Berrettini** is Karl E. Rickels Professor of Psychiatry and Director of the Center for Neurobiology and Behavior at the University of Pennsylvania, Philadelphia. **Dr. Worthman** is Director of the Department of Anthropology, and Director of the Laboratory for Comparative Human Biology, both at Emory University, Atlanta. **Dr. Steiger** is Director of the Eating Disorders Program at Douglas Hospital, and professor of psychiatry at McGill University, Montreal. **Dr. Soumi** is Chief, Laboratory of Comparative Etiology, National Institutes of Child Health and Human Development, National Institutes of Health.

Questions & Answers

Spontaneous Pneumothorax

Q: One of my patients had a past medical history of a collapsed lung, which her physician called spontaneous pneumothorax. Is this a recognized complication of anorexia nervosa? (AL, Tallahassee, FL)

A. Spontaneous pneumothorax occurs when a subpleural pulmonary bleb (basically a structural bubble) bursts, and air rushes into the pleural space between the lung and chest wall. The lung then collapses in response to this sudden buildup in air pressure where no air previously existed. Spontaneous pneumothorax most often occurs in young adult individuals with a lean body build, most typically males. These young men usually present with acute chest pain and progressive shortness of breath. In instances where small amounts of air have leaked into the chest wall cavity, treatment can be conservative, and the air may be reabsorbed spontaneously. However, in more serious cases, chest tubes are inserted to help the air escape and the lung to re-inflate.

One case has been reported in a patient with anorexia nervosa, and the authors assume that others have occurred as well (Adson et al, *Psychosomatics* 1998, 39:162). However, a larger number of patients with anorexia nervosa have developed "spontaneous pneumo-

mediastinum." In this situation, high intrathoracic pressure, sometimes associated with vomiting, leads to high intra-alveolar pressure, rupture of perivascular alveoli and escape of air into connective tissues and dissection of air into the mediastinum. Symptoms include acute pleuritic chest pain, shortness of breath and neck pain, and several specific signs on physical examination of the chest.

The important point here is that patients who are vomiters who develop this syndrome require an immediate contrast x-ray of the esophagus to rule out perforation (Boerhaave's syndrome). If untreated, this condition may lead to peritonitis and result in death, and thus must be surgically corrected as quickly as possible.

—J.Y.

Low Serum Phosphorus: A Marker for Severe Malnutrition

Hypophosphatemia is a hallmark of the refeeding syndrome, and is frequently reported in patients with anorexia nervosa. Because of this, Dr. Rollyn M. Ornstein and colleagues at Schneider Children's Hospital, New Hyde Park, NY, recommend that all anorexic inpatients be monitored daily for serum phosphorus levels and given supplementation as needed during the first week of hospitalization. This is especially appropriate for patients who are severely malnourished, according to the authors.

Dr. Ornstein and his group reviewed the charts of 69 anorexia nervosa patients (66 females, 3 males) admitted to an inpatient adolescent medical unit between July 1998 and July 2001. They reported the results of their study at the Academy for Eating Disorders meeting in Boston

in April. Mean age was 15.5 years and the mean percent of ideal body weight (IBW) was 72.7%. Serum phosphorus levels were measured daily for one week and then biweekly to weekly. Patients were started on 1200 to 1400 kcal/day, and calories were gradually increased by 200 kcal every 24 to 48 hours.

Greatest risk at very low IBW

More than a fourth of the group needed phosphorus supplementation, and the authors found that those at greatest risk weighed <70% of IBW. Overall, 27.5% of patients were treated with phosphorus supplements. Four (5%) developed moderate hypophosphatemia (<2.5 mg/dl), and 15 (21%) had mild hypophosphatemia (range: from <3.0 mg/dl to ≥2.5 mg/dl). Those who had moderate hypophosphatemia were significantly more malnourished than those who did not. The patient with the lowest phosphorus levels had short runs of ventricular tachycardias. According to the authors, the lowest levels of serum phosphorus generally occurred on the fourth day of hospitalization.

Nibbles, by Hunter

ANIMAL LABORATORY



"I know they're manipulating our genes, but how come I always wind up in the fat group?"

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Herbal Products: Balancing the Good and Bad

We've all seen the ads for "natural" products that can help you lose weight fast or help suppress appetite. But, do any of these really work? It's a real gamble because there are no standards for these products; the manufacturer doesn't have to prove that the product is safe or effective or even that the product contains the ingredients listed on the label.

To help remedy this problem, Congress recently created the National Center for Complementary and Alternative Medicine (NCCAM) at the National Institutes of Health (NIH), Bethesda, MD. Currently researchers at the Center are studying some herbs that have been helpful for some persons, including St. John's Wort (used for depression), ginkgo biloba (energy and memory), saw palmetto (prostate health), and glucosamine/chondroitin (bones and joints).

Several herbal products of particular interest for persons with eating disorders are found in products that claim to aid weight loss, as well as appetite suppressants and laxatives.

Herbs for Weight Loss

So far the only herbs that have shown any promise as weight-loss drugs are ephedrine and caffeine. These are marketed as a combination of ephedrine and caffeine or as the herbal sources, which include the drug ma huang (ephedrine) and a source of caffeine such as guarana seed or kola nut. However, even if they result in slight weight loss, there are substantial risks. The big problem is that the botanical sources may contain too much or too little of either ingredient; there is no way to know since there are no standards for this.

Caffeine and Ephedra—What's the Harm?

Caffeine, which is found in varying doses in beverages and medications, can cause insomnia and tremors, and acts as a diuretic. It is the most widely used "drug" in the world, thanks to tea and coffee lovers. By itself, caffeine isn't harmful for most people. However, when caffeine is teamed with ephedrine, it can speed up the heart (tachycardia), and cases of heart attacks and stroke have been reported. Other side effects include acute hepatitis, headache, tremor, nervousness, and insomnia.

Ephedra (Ma Huang): One to Avoid

The Chinese have used ephedra for more than 5,000 years. An extract of this powerful herb is ephedrine, one of the most effective treatments known for asthma, allergies, and sinus problems. Ephedra is a central nervous system stimulator that can increase pulse rate and blood pressure. Reputable manufacturers will include a warning about this on the label.

The main problem with ma huang is that many herbal manufacturers spike their ephedra-containing weight loss products with caffeine, usually by adding herbs such as guarana seed or kola nut. By itself caffeine can raise blood pressure and cause heart palpitations. When it is teamed with ephedra, the effects are magnified.

We strongly recommend that you not use ephedra-containing products.

However, if you do, please note the following:

1. Don't use ephedra products that also contain caffeine; this includes those that contain guarana and cola (or kola) nuts.
2. Choose an ephedra product that uses the whole herb, not just the extracted ingredient ephedrine.
3. Reduce the amount as soon as any side effects occur and stop the drug immediately if any of the following symptoms occur: headache, nervousness, sleeplessness, anxiety, nausea, and urinary problems.

Yohimbine: No Proven Benefits

Yohimbine is made from the bark of a West African evergreen tree and was originally used as an aphrodisiac and stimulant for warriors preparing for battle. Some appetite suppressants contain yohimbine. Promoters also claim that it helps decrease body fat. Potential side effects include anxiety, elevated blood pressure, a feeling of queasiness, insomnia, rapid heartbeat, tremors and vomiting. **The FDA has declared yohimbine unsafe and ineffective as an over-the-counter drug.**

Laxatives: Dulling Down the GI Tract

First, and most importantly, laxatives do not really cause the loss of significant amounts of food or help in weight loss. They do cause dehydration and reflex fluid retention. Therefore, laxative use is an ineffective weight-control technique and can be dangerous.

A number of these herbs contain ingredients that act by irritating the lining of the intestines or by directly stimulating the nerves; over time and with overstimulation, the bowel becomes nonresponsive. Laxatives often contain stimulants such as bisacodyl, cascara sagrada, or senna. Bisacodyl can lead to stomach irritation, cramping, and loss of fluids and electrolytes. Cascara sagrada can cause severe vomiting, electrolyte imbalance when abused, and loss of potassium which can make certain diuretics more toxic. Finally, senna can cause abdominal cramps, nausea, increase mucus secretion, and eventually help lead to reduced bowel function.

What Can You Do?

If you buy herbs, how can you tell which have the highest quality and which products really contain what the label claims? There is no guarantee that the ingredients listed will match the actual contents, so remember that contamination, mislabeling, and misidentification can be a problem.

Experts at the Mayo Clinic offer 5 tips for choosing the best brands of herbs:

1. Look for herbal extracts that are standardized. The USP (United States Pharmacopoeia) or NF (National Formulary) mark on the label or package is a clear sign that the manufacturer is following USP standards.
2. Choose a brand that adheres to higher manufacturing standards than are required. All herbal product manufacturers are supposed to follow standards established for processing foods; these are known as Good Manufacturing Practices (GMPs). Some companies set even higher standards for themselves, and usually will advertise this fact. Look for it in their ads.
3. Buy only single-herb products that clearly show how much is in each dose.
4. Beware of claims that sound too good to be true. Use your own common sense. No single herbal product can possibly take care of a wide number of claims.
5. The FDA advises avoiding 5 dangerous herbs: belladonna, comfrey, broom, lobelia, and pennyroyal.

What To Do If You Have a Bad Reaction to a Herbal Product

If you become ill from taking an herbal remedy, call your doctor. You or your doctor should also report the problem to the FDA. Call 800-FDA-1088 (800-332-1088) or go to the FDA's MedWatch Web site: www.fda.gov/medwatch.

When you call, they will ask for certain information:

- a. The name, address, and telephone number of the person who became ill.
- b. The name and address of your doctor or hospital where you were treated.
- c. A description of the problem.
- d. The name of the herbal product and the store where you bought it.

It is also a good idea to contact the manufacturer or distributor listed on the product label, as well as the store where you bought the product; if possible include the lot number listed on the bottle or box.

Resources

Finally, a little knowledge goes a long way—there are many resources available through your local library and the Internet. Some helpful Internet sources are:

www.herc.org/contrib/herbs.html

www.fda.gov/medwatch

www.MayoClinic.com (Food and Nutrition Center)

<http://nccam.nih.gov/> (National Center for Complementary and Alternative Medicine)

www.webMD.com (click on "Health Tools" and then on "Drugs and Herbs")