effects of endogenous opioid peptides in the brain. Naloxone has been shown to retard weight gain in experimental animals (Reid 1985). Although it also appears to reduce food intake in humans (Cohen et al. 1985), studies exploring its clinical use in weight reduction have been disappointing (Levine et al. 1985).

Digestion and Absorption

A variety of commonly used drugs impair the digestion and absorption of certain nutrients.

Laxatives decrease gastrointestinal transit time and reduce the absorption of glucose, calcium, protein, sodium, and potassium (Frier and Scott 1977). It has been suggested that mineral oil solubilizes and sequesters fat-soluble vitamins and prevents their absorption (Morgan 1941; Mahle and Patton 1947), but more recent data suggest this effect may be minimal. Cellulose can decrease absorption of calcium and magnesium (Berstad et al. 1975; Pak 1973).

Excessive consumption of aluminum-containing antacids can induce a phosphate depletion syndrome when dietary phosphate combines with aluminum hydroxide to form aluminum phosphate, which is insoluble and hence is completely excreted (Cooke, Teitelbaum, and Avioli 1978; Insogna et al. 1980). The risk of acute phosphate depletion is greatest when the diet is low in phosphate (Roe 1984). Antacids have been reported to have induced a severe copper deficiency in a patient with decreased gastric emptying time (Anonymous 1984a). Prolonged use of aluminum-containing antacids by patients with impaired renal or biliary excretion should be avoided, because the absorption and accumulation of aluminum under these conditions may impair calcium metabolism and lead to bone disease as is seen in kidney dialysis patients (Lemboke et al. 1982; Herzog et al. 1982).

Cholestyramine and colestipol prevent intestinal reabsorption of bile acids; they lower blood cholesterol levels by enhancing the conversion of cholesterol to bile acids. These drugs, however, also alter bile acid activity and may result in malabsorption of fat, vitamins A, D, K, and B_{12}, and folacin (Whiteside et al. 1965; West and Lloyd 1975). However, use of cholestyramine for 7 to 9 years in the Coronary Primary Prevention Trial by men with high blood cholesterol did not appear to cause nutrient deficiencies. Fat-soluble vitamins are frequently prescribed along with cholestyramine to eliminate any risk of deficiency.

Certain antibiotics, such as neomycin, may damage the intestinal mucosa and also precipitate bile acids, thus decreasing the absorption of vitamin K, carotene, and vitamin A, which are dependent on bile acid action for absorption (Levine 1967; Thompson et al. 1971; Barrowman, D'Mello, and Herxheimer 1973). Excessive amounts of broad-spectrum antibiotics can also destroy the natural vitamin K-producing bacterial population of the intestine, thus inducing bleeding conditions that can be reversed, if necessary, by vitamin K administration (Ansell, Kumar, and Deykin 1977; Anonymous 1984b).

The anti-inflammatory agent colchicine used to treat gout may alter intestinal transport mechanisms, leading to sodium, potassium, lipid, and nitrogen fecal loss (Race, Paes, and Faloon 1970; Webb et al. 1968).

Metabolism and Utilization

Oral Contraceptive Agents. Oral contraceptive agents are formulated from synthetic estrogens and progesterones that can affect metabolic processes involving essential vitamins and minerals. They are used by an estimated 10 million women in the United States (Ory, Forrest, and Lincoln 1983). Although numerous studies have reported laboratory evidence of marginal nutritional deficiencies among women taking these drugs, such evidence has been inconsistent and has only rarely been confirmed by clinical evidence. For example, blood levels of vitamin B_6 were found in some studies to be reduced among 20 percent or more of women using oral contraceptives, but other studies have failed to demonstrate such effects (Thorp 1980). The clinical significance of these observations is uncertain (Leklem 1986; Miller 1986). Circulating levels of zinc and release of zinc from tissues have also been reported to be reduced among users of oral contraceptives, but there is no evidence that such changes alter the dietary requirement for this mineral (King 1987). As with other interactions yielding minimal to moderate decreases in serum levels of micronutrients, clinical manifestations of deficiencies are likely to be detected only when nutritional status is below optimal levels before taking the drug.

Anticonvulsants. Various anticonvulsants accelerate the metabolism and elimination of vitamin D (Harvey 1985) and have been associated with clinical signs of rickets in children and osteomalacia in adults (Matheson et al. 1976). Also, the anticonvulsants phenytoin, phenobarbital, and primidone are capable of inducing a biochemical or clinical folate deficiency state (Chanarin 1979; Lambie and Johnson 1985; Edeh and Toone 1985). These drugs also interfere with metabolism of thiamin (Klein et al. 1977).

Newborn infants of mothers taking barbiturates or phenytoin are more likely to have coagulation defects because of reduced availability of vitamin K (Keith, Gundberg, and Gallop 1980; Keith et al. 1983). Osteomalacia secondary to phenytoin may require replenishment of vitamin K as well as vitamin D to restore the vitamin K-dependent proteins that are important for normal calcium metabolism in bone. Carnitine may be depleted by therapy with the anticonvulsant valproic acid, resulting in hepatic injury and a Reyes-like syndrome (Bohles et al. 1982; Coulter 1984; Murphy, Marquardt, and Shug 1985).

Vitamin Antagonists. Several drugs used as cancer chemotherapeutic agents (e.g., methotrexate), as diuretics (triamterene), or as antimalarial or antibacterial drugs (pyrimethamine) are antagonists of folic acid (Lambie and Johnson 1985). These drugs bind to the enzyme dihydrofolate reductase, preventing the conversion of folic acid to tetrahydrofolate, the vitamin form that is required for synthesis of purines. As a result, DNA synthesis is arrested and the cells die (Anderson, Smith, and Hutchinson 1966; Kahn, Fein, and Brodsky 1968; Lieberman and Bateman 1968; Myatt, Hernandez, and Coatney 1953). The drug sulfasalazine, used to treat ulcerative colitis, inhibits intestinal transport of folic acid and has been associated with the clinical signs of deficiency of this vitamin often seen in persons with inflammatory bowel disease (Halsted, Gandhi, and Tamura 1981).

The vitamin K antagonists—dicumarol, phenprocoumon, and warfarin—are used as anticoagulants; they block a specific carboxylation step in the activation of six vitamin K-dependent clotting factors (Wessler and Gitel 1984; O'Reilly 1985). Parkinsonian patients, whose tremors are controlled by L-dopa, may experience a precipitation of their symptoms if they ingest large doses of vitamin B_6 because of an interaction between L-dopa and pyridoxal-5 phosphate, the active form of this vitamin (Evered 1971; Mars 1974).

The antitubercular drug isoniazid binds and inactivates vitamin B_6, inducing pyridoxine deficiency and its resulting neuropathy (Biehl and Vilter 1954). Consequently, vitamin B_6 is often given to individuals receiving this therapy. One benefit of this interaction is that overdoses of isoniazid can be treated successfully by administration of vitamin B_6 (Wason, Lacouture, and Lovejoy 1981). A similar vitamin B_6-responsive neuropathy occurs in patients taking hydralazine, a hypotensive agent (Raskin and Fishman 1965). Cycloserine, another antitubercular drug, impairs niacin synthesis and absorption and may lead to a niacin deficiency (Heinivaara and Plava 1964).

Vitamin B_{12} metabolism is also affected by a variety of drugs. For example, the antitubercular drug paraminosalicyclic acid affects intestinal transport mechanisms for vitamin B_{12} (Toskes and Deren 1972), and prolonged exposure to nitrous oxide has been reported to produce a megaloblastosis (Chanarin 1982) and a myeloneuropathy similar to symptoms typical of vitamin B_{12} deficiency (Layzer 1978), perhaps because of binding and inactivation of the cobalt present in that vitamin.

Antihypertensive Drugs. These drugs produce disturbances of macronutrient metabolism, resulting in glucose intolerance and hyperlipidemia. Beta blockers increase blood levels of triglycerides but decrease levels of high density lipoprotein cholesterol (Helgeland 1984; Weinberger 1985). Thiazide diuretics decrease glucose tolerance (Amery et al. 1978; Murphy et al. 1982; Perez-Stable and Caralis 1983; Helderman et al. 1983; Ames 1984), increase serum levels of low density lipoprotein cholesterol (Goldman et al. 1980; Grimm et al. 1981; Weinberger 1985), and decrease losses of calcium in urine (McCarron 1985; Stier and Itskovitz 1986). Spironolactone lowers serum levels of high density lipoprotein cholesterol and increases serum levels of insulin (Falch and Schreiner 1983).

Excretion

Diuretics. Diuretics, such as the thiazides and furosemide, decrease the resorption of potassium and other minerals by the kidneys, thereby increasing their excretion (Dyckner and Webster 1979; Morgan, Murkinshaw, and Davidson 1978). Patients on long-term diuretic therapy are usually advised to consume foods rich in potassium.

In sum, drugs cause little nutrient-related difficulty for the great majority of individuals taking medications. Adverse effects are usually limited to persons who consume relatively high doses over prolonged periods, or to persons who have clinical conditions that interfere with normal drug metabolism and excretion or that make them unusually susceptible to drug-induced nutritional deficiencies (Smith and Bidlack 1982).

Effects of Diet on Drug Metabolism

Nutritional factors affect the activity of drugs by altering their rates of absorption, metabolism, or excretion.

Absorption

Dietary factors can decrease, delay, or enhance the absorption of drugs, primarily by altering their availability, their solubility, or the amount of time

they spend in the stomach or intestine (Hathcock 1985). Calcium, for example, can bind tetracycline antibiotics and form a complex that renders both the drug and nutrient unavailable (Roe 1985). Dietary fat enhances the absorption of fat-soluble drugs, but the absorption of drugs that bind to fiber is reduced by high-fiber diets. Drugs such as L-dopa and penicillin G that are metabolized or degraded in the stomach may be partially destroyed when gastric emptying is delayed. The bioavailability of certain drugs, such as the antibacterial nitrofurantoin, the cardiovascular drug propranolol, and the hypotensive drug hydralazine, is enhanced when gastric emptying is delayed and more of the agent can be dissolved in the gastric juice (Melander 1978; Toothaker and Welling 1980). Digitalis absorption is slowed by the presence of food in the gastrointestinal tract. Instructions to take drugs with or between meals, or the coating of drugs to prevent dissolution, attempt to take advantage of these gastric properties, although it is uncertain how well patients adhere to such instructions. The acidity of the gastrointestinal tract also affects drug disposition. A more acidic environment reduces the bioavailability of penicillin and isoniazid but increases the absorption of tetracyclines (Roe 1985).

Food decreases, delays, or enhances the absorption of certain antibiotics (Hathcock 1985). The complexity of the diet and of drug responses makes the clinical implications of such interactions difficult to predict.

Metabolism and Utilization

The effects of drugs are modulated by their rates of metabolism by the liver and other tissues. Drugs that are metabolized slowly do not need to be taken as frequently or in as high doses as those that are rapidly eliminated by the body. Drugs are metabolized by two basic processes. The first (Phase I) metabolic step is usually an oxidation reaction that alters a functional group in the drug. This alteration may either activate the drug or deactivate it. The most common example of this process involves the same mixed-function oxidase enzyme systems that metabolize many other endogenous or foreign (xenobiotic) compounds. These systems contain cytochrome P-450 and other cytochromes and employ reduced nicotinamide adenine dinucleotide phosphate (NADPH) and molecular oxygen in their reactions. A second step (Phase II) conjugates the oxidized drug to an inactive, water-soluble form that can be readily excreted (Bidlack 1982; Meydani 1987; Hathcock 1987a). Most effects of diet on drug metabolism affect the oxidation reactions, whereas relatively little is known about the ways in which nutritional factors affect conjugation reactions (Hathcock 1986).

The rate of drug metabolism by mixed-function oxidase systems can be accelerated (induced) by the drugs themselves as well as by a variety of dietary factors. Such factors include protein, cruciferous vegetables such as broccoli or cabbage, and charcoal-broiled meats (Bidlack 1982). Thiamin deficiency also increases drug metabolism—it has been shown to increase the demethylation of mestranol, for example (Hoyumpa and Schenker 1982).

On the other hand, low-protein, high-carbohydrate diets and deficiencies of several vitamins and minerals reduce levels of drug-metabolizing enzymes and, consequently, the rate of drug metabolism, so that drug concentrations may decline slowly. Thus, in many cases, the net effect of nutritional deficiency is to increase drug potency (Hathcock 1987a). In starved individuals, for example, the activities of the mixed-function oxidase enzymes are reduced and the clearance of drugs such as chloramphenicol and sulfadiazine is impaired. The metabolism of other drugs is unaffected by starvation (Bidlack 1982).

Excretion

High-fiber diets interfere with the enterohepatic circulation of drugs excreted in bile (Hathcock 1985). Urinary acidity, which can be affected by diet, also influences drug elimination. Aspirin, for example, is resorbed in acidic urine, whereas amphetamines are resorbed under more alkaline conditions (Welling and Tse 1983).

Effects of Drug-Food Incompatibilities

Certain drugs can interact with specific nutrients or non-nutrient components in foods to cause acute adverse reactions. Such reactions can be prevented by avoiding the foods when taking the medication. Examples include interactions between monoamine oxidase inhibitors and foods containing tyramine, and between alcohol and disulfiram, hypoglycemic agents, and many other drugs (Taylor 1987).

Monoamine Oxidase Inhibitors

Monoamine oxidase (MAO) inhibitors are mood-elevating agents that were formerly used with great frequency to treat severe depression. Their nutrition-related problems stem from metabolism of the potentially toxic amine tyramine. Tyramine is formed when intestinal bacteria degrade the amino acid tyrosine; it is also found in fermented foods such as cheese, wine,

yogurt, some kinds of sausages, and beer. Under ordinary circumstances, the enzyme MAO converts tyramine to soluble products that can be excreted in urine. Inhibition of MAO in the presence of foods containing tyramine permits this toxic amine to increase in blood to the point where it causes severe—and occasionally fatal—hypertension (Asatoor, Levi, and Milne 1963). Because the use of MAO inhibitors by people who ingest foods containing tyramine can induce a severe hypertensive crisis, they are now prescribed less frequently (Roe 1984).

Alcohol

Acetaldehyde-induced nausea and vomiting can occur within 15 minutes after the consumption of alcohol by persons taking the drugs disulfiram (Antabuse), metronidazole (Flagyl), and chlorpropamide (Diabinese). Disulfiram inhibits the enzyme acetaldehyde dehydrogenase, which oxidizes acetaldehyde, a product derived from the metabolism of alcohol (Hald and Johnson 1948). This interaction, which is so unpleasant as to deter the use of alcohol, is the basis for the use of Antabuse in detoxification programs (for reviews see Roe 1984; Seitz and Simanowski 1987).

Alcohol consumption can cause hypoglycemia in persons with diabetes who are using agents such as the sulfonylureas to lower blood sugar levels (Harris 1971; Carulli, Manenti, and Gallo 1971). It also potentiates the prolongation of bleeding time induced by aspirin (Deykin, Janson, and McMahon 1982), an interaction that can have serious consequences in alcoholic patients who suffer from gastritis, esophageal varices, or peptic ulcers.

Acutely, alcohol inhibits the activity of drug-metabolizing enzymes by displacing other drugs bound to cytochrome P-450, by decreasing the availability of NADPH, or by disturbing the lipid bilayer membrane that provides the microenvironment for drug-metabolizing enzymes (Hoyumpa and Schenker 1982). Chronic alcohol consumption enhances oxidation and toxicity of many substances, for example, acetominophen (Seitz and Simanowski 1987). It increases the toxic side effects of analgesics, anesthetics, anticoagulants, anticonvulsants, antihistamines, antimicrobials, tranquilizers, and narcotics (Anonymous 1979). Meprobamate, chloral hydrate, and barbiturates are also metabolized and excreted more slowly when alcohol is taken simultaneously (Hoyumpa and Schenker 1982). Ingestion of benzodiazepines and alcohol together results in higher plasma concentration of the drugs and enhanced sedative effects due to decreased hepatic clearance. The postural hypotension produced by organic nitrates for angina is accentuated by alcohol (Needleman, Corr, and Johnson 1985).

As a general rule, any toxic side effect of a medication will be potentiated if alcohol is consumed along with it (Anonymous 1979; Seitz and Simanowski 1987).

Effects of Drugs Used in Food Production

Antibiotics such as tetracycline and penicillin are fed to livestock to prevent infections and to promote growth. Because the genetic information that controls some kinds of resistance to antibiotics is carried on transposable genetic elements (plasmids) that can be transferred from one organism to another, questions have been raised as to whether subtherapeutic doses of livestock with antibiotics (a procedure that enhances growth and feed utilization) might encourage the proliferation of antibiotic-resistant microorganisms that are human pathogens or are capable of transferring antibiotic resistance to human pathogens (Stallones 1982).

In 1980, a National Academy of Sciences (NAS) report concluded that the research necessary to establish and to measure potential risks of antibiotic use in animals had not yet been conducted (NAS 1980). Since then, human infections with drug-resistant *Salmonella* have been documented, both as a result of eating hamburger originating from cattle that had been fed subtherapeutic doses of tetracyclines (Holmberg et al. 1984) and from drinking raw cow milk infected with *Salmonella* resistant to chloramphenicol and several other antimicrobial agents (Tacket et al. 1985). As a result of these studies, the Center for Veterinary Medicine of the Food and Drug Administration, with the counsel of a new NAS committee, is reviewing the available research findings on the hazards and benefits of this use of drugs. At issue is whether to institute procedures to suspend low-level uses of penicillins and tetracyclines in animal feeds (Goodman-Malamuth 1986).

Effects of Pharmacologic Doses of Nutrients

Nutrients are sometimes used in unusually high doses for their pharmacologic effect. Niacin, for example, is used pharmacologically to reduce blood cholesterol levels (Grundy et al. 1981; Blankenhorn et al. 1987). Retinoid derivatives of vitamin A have been used successfully to treat severe acne and other conditions (Bollag 1983). All pharmacologic therapies induce side effects (pharmacology has been described as "applied toxicology" because even the desirable effects of drugs are obtained by altering—poisoning—normal metabolic function), and high-dose nutritional therapies are no exception. Although excess water-soluble vitamins are excreted and usually cause little difficulty, side effects have been

reported in cases of excessively high doses (Miller and Hayes 1982). High-dose niacin induces flushing, and neurologic symptoms have been reported from excessive intake of vitamin B_6 (Schaumburg et al. 1983). Excessive intake of fat-soluble vitamins or their derivatives is well known to induce toxic symptoms. Excess vitamin A, for example, causes birth defects in animals, and, possibly, in humans; caution has been urged in its use for women who are pregnant or likely to become pregnant (Teratology Society 1987).

Individuals born without the genes to produce key functional enzymes may require amounts of certain nutrients greatly in excess of those required by most people. Such inborn metabolic errors have been identified for enzymes necessary for absorption, metabolism, or storage of nearly all of the vitamins (Stanbury et al. 1985). In some cases, higher-than-normal intake of the vitamin will restore activity. A classic example of such a vitamin-responsive syndrome is pernicious anemia, a condition of impaired absorption of vitamin B_{12}. Patients with this condition must have exceedingly high doses of the vitamin from food or supplements, or lower doses by injection. Another example is homocystinuria, a condition that results from a deficiency of the enzyme cystathione beta synthetase. Lack of this enzyme leads to the accumulation of methionine, the appearance of homocystine in urine, and clinical symptoms ranging from thromboses and osteoporosis to mental retardation. About 40 percent of individuals with this condition respond favorably to doses of vitamin B_6 that are 50 to 500 times higher than levels defined by Recommended Dietary Allowances (Mudd and Levy 1983).

Acrodermatitis enteropathica, a severe skin and gastrointestinal disorder, is caused by a deficiency in zinc absorption, but it can be overcome by zinc administration at levels greatly in excess of those normally required (Prasad 1983). An inborn error of metabolism resulting in systemic carnitine deficiency, with clinical manifestations of excessive ketone production, has been treated successfully with supplemental carnitine (Wolff et al. 1986).

In other conditions, certain metabolic products cannot be degraded and, therefore, accumulate to toxic levels. In some cases, such disorders can be treated with carefully designed dietary preparations having a very low content of the poorly metabolized nutrient. An example of this type of condition is phenylketonuria, a genetic lack of the enzyme that converts the amino acid phenylalanine to tyrosine. Patients with phenylketonuria accumulate phenylalanine and other metabolites that, at high levels, are toxic and cause mental retardation and other neurologic damage (Anony-

mous 1986d). Dietary treatment is designed to reduce the phenylalanine content of the diet to levels below those that cause symptoms. The special infant formula product Lofenelac, for example, contains sufficient quantities of all of the essential amino acids with the exception of phenylalanine (Tourian and Sidbury 1983).

Implications for Public Health Policy

Dietary Guidance

General Public

Although drugs interact with dietary factors in many ways that impair nutrient availability, evidence about the public health significance of such interactions is insufficient to recommend general shifts in the pattern of use of any particular drug on the basis of its adverse effects on nutritional status. Nor may any implications be drawn at this time for the general public on intake of specific nutrients with relation to nonprescription drug interactions.

Special Populations

Studies of patients consuming multiple drugs for prolonged time periods, especially those patients who are older, suggest that dietary intakes may need to be adjusted to compensate for adverse interactions of specific nutrients with medications and that information should be provided to such patients by qualified health professionals on appropriate use of such diets. Patients taking drugs that induce acute reactions in the presence of dietary factors such as tyramines or alcohol should be instructed on appropriate means to avoid those factors.

Persons with inborn metabolic errors that respond to pharmacologic doses of nutrients or to special products designed to minimize toxic symptoms should be advised on the safe and effective use of such therapies. Health professionals should receive instruction about drug-nutrient interactions to understand how best to maximize drug efficacy and minimize adverse reactions.

Nutrition Programs and Services

Food Labels

Evidence related to the role of diet in drug interactions currently holds no special implications for change in policy related to food labeling.

Drug Labels

Evidence related to the role of diet in drug interactions suggests that drug manufacturers should provide information in the package insert on the potential effects of the medication on nutritional status, and vice versa.

Food Services

Evidence related to the role of diet in drug interactions currently holds no special implications for change in policy related to food service programs.

Food Products

Evidence related to the role of diet in drug interactions currently holds no special implications for change in policy related to packaged food products. Preliminary evidence relating human infections to antibiotic-resistant micro-organisms derived from animals treated with subtherapeutic doses of antibiotics suggests the need for close scrutiny of this practice.

Special Populations

Persons—especially older persons—who consume drugs should be provided with counseling and assistance on dietary methods to avoid adverse drug-nutrient interactions. Persons with inborn metabolic errors requiring therapy with pharmacologic doses of nutrients should be provided with counseling and assistance on appropriate and safe use of such supplements.

Research and Surveillance

Research and surveillance issues of special priority related to the role of diet in drug interactions should include investigations into:

- The extent of drug taking (prescription, over the counter, and illegal) among the population.
- The extent of adverse drug-nutrient interactions in the population and their clinical significance.
- Age-related changes in nutrient metabolism with special implications for pharmaceutical use.
- Effects of medications on the nutritional status of older persons.
- Drug effects on nutrient intake, absorption, metabolism, and excretion.
- Effects of diet, including alcohol, on drug absorption, metabolism, and excretion.

- The effects of antibiotics, hormones, or other drugs in animal feeds on human health.
- The levels of intake of essential nutrients that induce toxic symptoms.
- The most effective means to educate health professionals and the general public about drug-nutrient interactions.

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Chapter 19

Dietary Fads and Frauds

The advertising quack . . . is the black wolf, aye, the Bengal tiger of the profession. . . . He is full of shrewdness and cunning, and knows the poor weak human nature like a book.
Dr. Willis P. King, 1882
Quacks and Quackery in Missouri

Introduction

Historical Perspective

Food, an indispensable ingredient of life, has long been endowed with metaphysical, moral, and theological meanings. The folklore and superstitions of cultures throughout history have attributed healing or harmful properties to certain foods. This tendency has not disappeared with the advent of the sciences of nutrition and medicine. Food folklore continues today, although in many instances it is inconsistent with scientific evidence (Young 1978).

Contemporary food fads often make one or more of the following claims, none of which are substantiated by available scientific evidence (Bitensky 1973; Darby 1974; Shifflett 1976; Deutsch 1977; Stare 1980; Miller 1980; Jarvis 1983, 1984b):

- Some foods have magical, life-promoting properties.
- Modern foods are grown on depleted soil, are overprocessed, and, therefore, cannot provide good nutrition.
- Food supplements are always necessary to ensure good nutrition, and megadoses of nutrients provide "supernutrition."

Definitions

Nutrition fraud is a comprehensive term used by the U.S. Food and Drug Administration (FDA) to describe the abuses that occur as the result of the misleading claims for traditional foods, dietary supplements, and dietary

products and of the deceptive promotion of other food substances, processes, and devices (Nightingale 1984). Nutrition fraud has long been recognized as the leading example of health fraud (Larrick 1963). Food faddism and quackery describe two types of nutrition fraud commonly purveyed to the public (Huenemann 1956; Todhunter 1973; Jarvis 1984a).

Food faddism is a dietary practice based upon an exaggerated belief in the effects of food or nutrition on health and disease. Food fads derive from three beliefs: (1) that special attributes of a particular food may cure disease, (2) that certain foods should be eliminated from the diet because they are harmful, and (3) that certain foods convey special health benefits (McBean and Speckmann 1974). Unlike more transitory fads, many key concepts associated with food faddism persist or reappear periodically. Food faddists are those who follow a particular nutritional practice with excessive zeal and whose claims for its benefits are substantially more than science has substantiated. In most instances, foods praised as beneficial, such as special products or vitamin supplements, are not as good as faddists claim, and those foods condemned as harmful, such as white flour or sugar, are not as bad (Jarvis 1983).

Food quackery, which involves the exploitive, entrepreneurial aspects of food faddism, is the promotion for profit of special foods, products, processes, or appliances with false or misleading health or therapeutic claims. A food quack is one who pretends to have medical or nutritional knowledge and who promotes special foods, products, processes, or appliances with false or misleading claims, usually for personal financial gain.

Factors Contributing to Nutrition Fraud

Nutrition fraud flourishes in the United States today because of the diversity of cultures, the historical tradition of concern for health and the use of natural remedies, and the introduction of advanced communication technologies. Some frauds derive from traditional folklore inherited from the many cultures populating this country, while others are uniquely American (Young 1978; Deutsch 1977).

Food faddism in America had its roots in Great Britain, where patent medicines were advertised and sold by everyone from hairdressers to goldsmiths. In the colonies, legal protection of consumers against fraudulent claims was first recorded in Massachusetts Bay in 1630. A citizen, Nicholas Knopp, was whipped and fined five pounds for selling a cure for scurvy that had "no worth nor value" and was "solde att a very deare rate" (Young 1961).

Food faddism was very common in 19th-century America, perhaps as a result of the high literacy rate and the proliferation of newspapers that provided a medium for advertising. Claims for health benefits were an integral part of the promotion of foods and food components sold by patent medicine men and women and popular health reformers (Whorton 1982). One of the earliest nutrition faddists was Sylvester Graham, a "back to nature" reformer who was suspicious of any food, such as white flour, altered from its "natural" condition (Young 1978; Whorton 1982). His legacy continues among those who question whether processed food of any type can provide adequate nutrition (Deutsch 1977; Stare 1980; Miller 1980).

Popular interest in nutrition, coupled with concern about food shortages during World War I, was fostered by the increasing promotion of the health properties of foods in the early 20th century (Young 1967). Vitamins, by the very nature of their discovery, became associated with the prevention or cure of disease (Todhunter 1973) and were soon promoted as curative agents (Young 1967). George P. Larrick (1959), then Commissioner of Food and Drugs, explained this trend:

> In the wake of scientific advances there often follows a host of persons who misinterpret them and exploit them for private gain. That has been true in the field of nutrition. The nutritionist studies the long-range benefits to the public health from new scientific findings, withholds premature endorsement, and has confidence that future research holds great promise. . . . The promoter . . . does not wait for the facts or the possibility of different findings in the future. He hastens to cash in before all the facts are known. Those interested only in profit employ clever copywriters to promote products by pseudoscientific statements, using half-truths and gross exaggeration to build up a scare psychology. . . .

Today, the traveling patent medicine man has been largely replaced by the highly skilled and organized use of electronic means to promote fraudulent marketing—computers, customized mailing lists, national advertisements, WATS banks of telephone lines, and other mass media. The medium and the details have changed, but the message and the goals remain. It is difficult for consumers to evaluate the validity of the health claims perpetrated by quacks and faddists (Tierney 1984).

Background

Regulation of Nutrition Fraud

The need for public protection from fraud was recognized over a century ago, when Congress enacted statutes to combat mail fraud (Nelson 1984).

The first Federal legislation, the Pure Food and Drug Act of 1906, made it unlawful to manufacture or introduce into interstate commerce adulterated or misbranded food or drug products (Anderson 1964). This Act was passed in response to an intensive campaign against patent medicines and food abuses stimulated by the work of Dr. Harvey Washington Wiley, chief chemist at the U.S. Department of Agriculture. Because of this, it soon became known as "Dr. Wiley's Law."

The Federal Trade Commission Act of 1914 established Federal authority for regulation of false advertising in interstate commerce, including advertising of foods for human consumption. The 1938 Federal Food, Drug, and Cosmetic (FD&C) Act gave the FDA new and more effective authority over health food claims. Under the FD&C Act, a food is considered a drug if therapeutic claims are made for it, and the burden of proving claims fraudulent is less demanding than under the earlier law (Young 1967).

Currently, numerous Government, medical, and consumer-oriented organizations are responsible for preventing and controlling fraud (U.S. Congress 1984b). At the Federal level, the FDA, the U.S. Postal Service (USPS), and the Federal Trade Commission (FTC) have authority to act against various kinds of illicit food and health-related practices. These agencies work cooperatively, and their antifraud activities have become more visible in recent years (Barrett 1985). The regulatory roles of these various Federal agencies are reviewed below.

State enforcement activities against fraud are largely the responsibility of the State attorneys general and offices of consumer affairs and aging. County consumer affairs offices and metropolitan police may also become involved in regulation.

Private agencies and organizations such as the Better Business Bureau, the American Dietetic Association, the American Heart Association, the Arthritis Foundation, the American Cancer Society, the American Medical Association, the National Council Against Health Fraud, and other health professional groups are also active against food fraud. These organizations often maintain informal liaison with each other and actively cooperate with Federal regulatory agencies.

Despite these efforts, misleading claims about foods and nutrients are difficult to regulate. As noted by the 1969 White House Conference on Food, Nutrition, and Health, "No other area of the national health probably is as abused by deception and misinformation as nutrition. Many travesties

cheat the public of enormous sums of money, and of good health as well. Yet the American people falsely believe they are well-protected, both by Government and by the ethics of commerce" (White House Conference 1969).

Not only is regulation difficult because of the complexity of the science base, the ease of exploiting the mails, and the special vulnerabilities of people with health concerns, but in the United States, governments at all levels are limited in what they can do about fraudulent nutrition practices by an obligation to observe the constitutional rights to free speech and a free press. Nutrition information, whether supported scientifically or not, is guaranteed the same protection under the first amendment as any other information (Stephenson 1978; Young 1967). The right to say, write, or publish anything one chooses is protected, as long as it is done without intentional malice (Barrett 1977; Pennington 1984).

Regulatory Roles of Federal Agencies

Food and Drug Administration Authority. The FD&C Act empowers the FDA to prohibit the introduction of any food, drug, device, or cosmetic that is adulterated or misbranded. The Act does not include explicit authority to address health fraud, but many of its general provisions enable actions in these areas. Sections 702–704 authorize the gathering of information about health fraud products and practices through inspections, collection of samples and records of interstate shipments, and gathering of evidence such as photographs and copies of labeling. Many fraud cases are based on misbranding charges resulting from false or misleading labeling of products. Only factual and nonmisleading information is allowed on food labels. Most false promotional claims, therefore, are not made on labels. Instead, they appear in books, lectures, and mass media that are protected by constitutional rights.

The FDA has the authority to use its food additive and drug approval processes to control food products allowed on the market and to remove fraudulent products. Although food products do not need to receive premarket approval as safe for human consumption, some food components need either to be classified as "Generally Recognized As Safe" (GRAS) or to have undergone the required FDA approval process for a food additive. If one or more of a food product's ingredients are subject to these provisions but is neither GRAS nor an approved additive, the product is considered adulterated and therefore illegal.

The FD&C Act defines drugs as articles that are intended for use in the diagnosis, cure, mitigation, treatment, or prevention of diseases in hu-

mans. Most fraudulent food products are classified as foods, but when therapeutic claims are made for them, they are also considered to be drugs. The Act's provisions require drug products to have (1) an approved New Drug Application that demonstrates both safety and efficacy for its intended uses and (2) adequate directions for use. If a food product is also classified as a drug and is considered by the FDA to be ineffective for its claimed use, it will not have an approved New Drug Application. For example, if it is promoted for treating a disease that is not amenable to lay diagnosis, it cannot have adequate directions for use and will not be approved. Recently, the FDA proposed a new policy on the appropriate use of health-related messages on food labels (FDA 1987).

Educating the public on health fraud is complementary to the FDA's regulatory activities. The Agency's magazine, *FDA Consumer*, devotes editorial space to health fraud and includes major articles on the subject. In addition, FDA consumer affairs officials across the country conduct a health fraud consumer education program covering foods as well as medical devices and drugs. The objectives of this program are to increase public knowledge and understanding of the FDA's statutory responsibilities and the limitations of the FDA's authority in protecting consumers from misinformation about foods, dietary supplements, nutrition, and other FDA concerns. The program provides guidance to enable consumers to recognize fraud, evaluate product claims, make informed decisions, and register complaints and concerns about fraudulent products.

U.S. Postal Service Authority. The authority of the USPS to control health fraud is based on a general congressional mandate to protect the public from marketing schemes conducted by mail. The purchaser is particularly vulnerable to fraudulent inducements by mail because of the inability to observe the product before payment (U.S. Congress 1984b). Specific authority to protect the mail order consumer is vested in the criminal fraud statute (18 U.S.C. 1341), the administrative false representation statute (39 U.S.C. 3005), and the supporting injunctive statute (39 U.S.C. 3007).

The mail fraud statute provides that any use of the mails in furtherance of an intentional scheme to defraud is punishable by a fine of $1,000 and imprisonment of up to 5 years. Many nutrition-related schemes are prosecuted under the mail fraud statute; they typically involve cure-alls, instant weight reduction techniques, and a variety of substances falsely described as having the ability to cure diseases or disabilities.

The false representation statute mandates that persons offering goods or services for sale through the mail refrain from misrepresenting their prod-