

## Chapter 18

# Nutrition and Stress

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This text has emphasized the body's adaptive mechanisms that attempt to maintain homeostasis in response to physical and psychosocial stressors (see Chap. 2 for a detailed explanation). However, as we have noted, intense stress can deplete and weaken the body, including, for example, the SNS response to inhibit digestion. As Whitney, Hamilton, and Rolfes (1990) note, "Much of the disability imposed by prolonged stress is nutritional" (p. 13). The purpose of this chapter is to briefly review how some of the basics of nutrition are involved in the stress response, including fatty acids, antioxidants, and serotonin, and will conclude with an introduction of future directions for dietary intervention.

It has been suggested that the mental health of entire nations may be linked to diet, and that the deleterious effects of excessive caloric intake in wealthy countries rivals those from malnutrition in poor ones (Gómez-Pinilla, 2008). The importance of nutrition is apparent when examining the impact of specific foods or diets on disease. For example, the Mediterranean diet, characterized by breads, pastas, legumes, vegetables, fruits, olive oil, ocean fish, and the moderate consumption of red wine, has been associated with lower incidence of Alzheimer's disease and Parkinson's disease (Sofi, Cesari, Abbate, Gensini, & Casini, 2008). Similarly, it has been suggested that a lower incidence of Alzheimer's disease in India may be attributed to the antioxidant-rich curcumin, a medicinal herb found in curry (Frautschy et al., 2001).

### Nutrients and Energy

Nutrients are diverse and complex substances that provide energy for body functions such as growth, metabolism, respiration, and circulation. The type of nutrients and their proportion to others we consume appears to be nearly as important as having

enough food. When responding to stress, our bodies rely on the energy-yielding macronutrients of carbohydrates, proteins, and fats (Charrondiere, Chevassus-Agnes, Marroni, & Burlingame, 2004). Alcohol is the only other source of energy in the diet, but unlike the other macronutrients it is not needed for survival. Both carbohydrates and proteins contain 4 calories per gram, whereas alcohol contains 7 calories per gram, and fat contains 9 calories per gram. To maintain health and regulate weight, energy expenditure should match energy (caloric) intake, or should exceed it to reduce weight (Institute of Medicine [IOM], 2005).

## *Carbohydrates*

It is recommended that 45–65% of the human diet consist of carbohydrates (United States Department of Agriculture; (USDA), 2010). Carbohydrates, the body's main source of energy, exist in substantial amounts in grains, potatoes, fruits, and milk, and in lesser concentration in vegetables and legumes. There are two general types of carbohydrates: simple and complex. Simple carbohydrates or sugars (monosaccharides and disaccharides) come from fruits and vegetables (e.g., glucose, fructose, maltose, and sucrose) and from milk or milk products (lactose and galactose). Simple carbohydrates are especially concentrated in sweeteners such as table sugar, confectioner's sugar, brown sugar, honey, and molasses. Complex carbohydrates, which are mainly starches, include oligosaccharides and polysaccharides and are found in grains such as rice, wheat, rye, barley, and oats. Another important source of starch is legumes such as peanuts, beans, peas, and soybeans. Potatoes, yams, and cassava, a tropical American plant with a starchy root from which tapioca is derived, are other sources of starches. Complex carbohydrates are metabolized more slowly than simple carbohydrates and serve as our major source of vitamins (except B12) and minerals.

Fiber is the indigestible portion of complex carbohydrates (Greenberg, Dintiman, & Myers-Oakes, 1998). Although it is not considered a nutrient because it is not used in metabolism, fiber facilitates digestion and elimination, and certain viscous fibers may delay the movement of food from the stomach to the intestine, creating a sensation of fullness. Fiber can also impact the absorption of fat and cholesterol (IOM, 2005; USDA, 2010). Increased intake of dietary fiber has been associated with a decreased risk for heart disease, stroke, diabetes, hypertension, obesity, hemorrhoids, acid reflux, constipation, and other gastrointestinal conditions (Anderson et al., 2009).

Dietary sugar intake has been a source of considerable debate for years. Despite popular opinion that sugar is related to hyperactivity in children, a meta-analysis of 23 studies indicated that sugar intake does not affect behavior or cognitive performance (Wolraich, Wilson, & White, 1995). A more recent review of a decade of evidence suggests that high-quality studies do not reveal a positive correlation between sugar intake and body mass index (BMI), and no consistent evidence links sugar consumption with attention deficit/hyperactivity disorder (ADHD), dementia, or depression (Ruxton, Gardner, & McNulty, 2010).

## ***Proteins***

The USDA (2010) recommends that 10–35% of the human diet consist of proteins. Proteins, which are used to rebuild, repair, and replace the body's cells, are found in meats, poultry, fish, dairy products, nuts, and legumes, and in smaller amounts in starchy foods and vegetables. Proteins are necessary for tissue growth, retaining lean muscle, generating hormones and enzymes, immune functions, and also as a source of energy in the absence of carbohydrates. Amino acids are the building blocks of proteins. Isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, valine, and histidine are considered essential dietary amino acids because the body cannot synthesize them. Recently, other amino acids such as tyrosine have been classified as indispensable under certain conditions (IOM, 2005; USDA, 2010). Serotonin, which is formed from the essential amino acid tryptophan, will be reviewed later.

## ***Fats***

Despite continuing to receive considerable, albeit understandable, “bad press” in current dieting literature, it is recommended that 20–35% of the human diet consist of fats (USDA, 2010). The body's fat (adipose) cells contain large stores of triglycerides that meet ongoing energy needs. When we are resting, our muscles expend little energy; therefore, it is not necessary to produce adenosine triphosphate (ATP) rapidly. Instead, the body's oxygen system, which uses carbohydrates, fats, and proteins as energy sources, provides required ATP for the resting physiological processes (Williams, 2007). More specifically, carbohydrates and fats combine with oxygen in the cells to provide major sources of energy during rest (Williams, 2007). In fact, in a normal diet fat supplies about 60% of the body's resting energy requirement (Eschelmann, 1996; Groff, Gropper, & Hunt, 1995). However, during periods of potential food deprivation, such as those occurring during prolonged stress, fat stores are likely to contribute an even higher percentage of energy needs (Whitney & Rolfes, 2011). Red meats, whole milk, cheeses, peanut butter, ice cream, butter, bacon, avocados, chocolate, and nuts are examples of foods containing an appreciable quantity of fat. Although fat provides the body with a concentrated source of energy, unused fat from the food we eat is also easily converted to body fat, or adipose tissue, which has an essentially unrestricted storage capacity (Whitney & Rolfes, 2011). Consider, for example, that “the energy liberated from each gram of carbohydrate as it is oxidized to carbon dioxide and water is 4.1 Calories... and that liberated from fat is 9.3 Calories” (Hall, 2011, p. 843). Therefore, it is worth noting that even though fat may be helpful during periods of prolonged stress, a small amount of fat in the body typically goes a long way, particularly during periods of low stress or limited energy expenditure.

Fat is necessary for growth, cushioning of the organs, maintenance of cell membranes, and the absorption of carotenoids and the fat-soluble vitamins A, D, E, and

K. Saturated fat, monounsaturated fat, and cholesterol (a fat steroid) are adequately synthesized by the body and thus unnecessary in the diet. Saturated fat and trans fat, an unsaturated fat with an E-isomer, have been associated with heart disease. Polyunsaturated fats are essential fats that our body needs yet cannot produce. Examples of polyunsaturated fats include omega-6 and omega-3, which play a crucial role in brain function and normal growth and development. Therefore, despite the popular view that consumption of fat should be minimized, recent research suggests that the type of fat consumed is nearly as important as the amount in affecting the body's functions (IOM, 2005; USDA, 2010). Excessive fat intake is a well-known health risk, but fat deficiencies are also associated with poor outcomes including the dysregulation of chronobiological activity and cognitive function (Yehuda, Rabinovitz, & Mostofsky, 1997). We will now explore the relation between fatty acids and stress.

## Fatty Acids and Stress

As noted, essential fatty acids (EFA) must be provided by diet, and are involved in many vital processes including energy production, air-to-blood oxygen transfer, hemoglobin manufacturing, and the transmission of nerve impulses. There are two types of EFAs. The omega-6 EFAs consist of linoleic and arachidonic acids, and the omega-3 EFAs consist of linolenic, eicosapentanoic, and docosahexanoic acids (Wertz, 2009; Yehuda, Rabinovitz, & Mostofsky, 2006). Vegetables, vegetable oils, and ocean fish are important sources of EFAs, whereas wild fresh water fish are not. Simopoulos (2006) suggested that, based on a study of diets in early humans, the ratio of omega-6 to omega-3 EFAs for optimal functioning would be 1:1. The ratio in the current Western diet is as high as 20:1, and Simopoulos credits this imbalance to pathologies such as cardiovascular disease, cancer, depression, and autoimmune diseases.

Simopoulos (2001) compared consumers of the Mediterranean diet in France and Greece to examine the effects of the EFA ratio variants. The EFA ratio in the Greeks was 1:1, while it was 6:1 for the French. The difference in EFAs was accounted for by the Greek diet which consists mostly of ocean fish, as compared to the French diet where they eat more fresh water fish, higher vegetable consumption in Greece, and differences in methods of egg production. It is notable in this study that the Greeks exhibit lower rates of disease and higher life expectancy. The balance of EFAs appears important, but it is also important to avoid excessive EFA consumption as it is associated with health risks such as degenerative diseases, cardiovascular disease, cancer, and diabetes (Yehuda et al., 2006).

Omega-3 diet supplementation has been suggested as a treatment for many disorders associated with the EFA imbalance. Of particular relevance are studies noted by Yehuda et al. (2006) that reported EFA supplementation resulting in improved cardiac response to stress (Rosetti, Seiler, DeLuca, Laposata, & Zurier, 1997), and other studies noting that EFAs can decrease the stress response (Hamazaki et al., 1999;

Sawazaki, Hamazaki, Yazawa, & Kobayashi, 1999). Benefits of supplementation have been reported for many groups including individuals following heart attack (GISSI Prevenzione Investigators, 1999), those diagnosed with bipolar disorder (Stoll et al., 1999), elderly depression (Tajalizadekhoob et al., 2011), and animal models of Parkinson's disease (Bousquet et al., 2011). Moreover, omega-3 supplementation has been associated with a decrease in cognitive deficits in children with developmental disorders (Richardson & Montgomery, 2005). However, other studies in individuals with bipolar disorder (Keck et al., 2006), Alzheimer's disease (Boston, Bennett, Horrobin, & Bennett, 2004), and ADHD (Hirayama, Hamazaki, & Terasawa, 2004; Voigt et al., 2001) reported no effect. In a recent systematic review of the role of fatty acids in health and disease, Riediger, Othman, Suh, and Moghadasian (2009) concluded that the beneficial effects of omega-3s have been demonstrated to the degree that their consumption should be increased.

## Energy Sources and Stress

Previously, we addressed the percentage of fat used as an energy source within the body. However, an additional consideration is the energy potential or energy value of food itself. According to Eschelmann (1996), in a typical American diet, "the energy value of food is 48% from carbohydrate, 35% from fat, and 14% to 18% from protein" (p. 123). Particularly relevant for the purposes of this chapter are that under low or mildly stressful conditions, the body gives preference to carbohydrates first and then fats as an energy source. In fact, carbohydrates and fats are often referred to as "protein spacers." However, during periods of severe, excessive stress, when hunger may be suppressed and digestion may be inefficient, the body may begin to consume protein stores rapidly for energy, often at six to seven times the typical rate, once carbohydrates and fats have been depleted. When this process occurs, dietary protein, along with lean muscle tissue throughout the body (including major organs such as the heart), is converted to glucose to supply energy for the nervous system. Obviously, if this process, known as ketosis, occurs for prolonged periods, then it may seriously jeopardize health.

Therefore, if you are able to eat during a period of stress, do so, of course. You may discover, however, that it is prudent to limit your caloric intake per meal, although you may try to eat more frequently. Also, the body, which conserves fluids during stress, will excrete what it does not need. Therefore, drink fluids, especially water (our most essential nutrient), which will enable your kidneys to help reestablish homeostasis. Although we do not obtain energy directly from water, vitamins, such as A, B, C, D, E, and K, which are organic chemicals, or minerals, such as calcium, iron, phosphorous, potassium, zinc, copper, iodine, magnesium, and fluoride, which are inorganic substances, "provide important components for the metabolic processes that produce the energy required for growth, development, and all life functions" (Brehm, 1998, p. 186). In converting nutrients to energy, vitamins may also produce hormones and break down waste products and toxins. It is also

worth noting that the best way to obtain necessary vitamins and minerals is from the diet, not from supplements.

It also is important to note that when excessive stress begins to subside and homeostasis is being reestablished, we should take the opportunity to replenish ourselves with a healthy balance of foods and exercise (see Chap. 15) to restore both lean and fat tissue. Also, it may be helpful during times of recuperation to recognize the difference between hunger and appetite. When we are physically hungry, our bodies crave the nutrients found in the calories, vitamins, and minerals of food. Appetite, which is often described as “psychological hunger,” is associated with a desire for specific foods. Fortunately, for most of us, the episodes of sustained, intense, debilitating, stress described earlier occur infrequently. Instead, we are generally accustomed to coping with recurrent episodes of mild to moderate psychosocial stressors on a regular basis.

As most of us are aware, mild to moderate and sometimes even high psychosocial stressors can trigger appetites. When this occurs, we may crave and then consume energy dense, sweet and starchy foods, as both our stress level and coping abilities dictate when and how much food we eat (Macht, 1996; Torres & Nowson, 2007). Polivy and Herman (1999) suggest that people disguise their life-related distress by overeating in order to associate their stress with binge eating rather than with the more uncontrollable areas of their lives. When we overeat, we blame ourselves for being weak and lacking self-control, but research suggests that overeating is not necessarily an issue of willpower. According to Wurtman and Suffes (1997), the hunger we experience when under notable levels of psychosocial stressors is biologically rooted, and this is why our cravings generally do not subside until sated and intensify when denied.

Macht (1996) discusses the possible relation between stress and the consumption of low-energy foods heavy in fat, salt, and sugar. He concludes that a person’s emotional stress level heightens when he or she eats food that provide little energy, and this increase in anxiety can then lead to an increase in his or her desire to eat. Low-energy foods such as cheese, potato chips, and cookies are generally deficient in the natural chemicals that moderate stress, whereas a diet that includes high-energy foods such as cereals, beans, raw vegetables, and fruit is helpful in mitigating the stress and cravings we experience.

## **Serotonin and Stress**

Stress and serotonin levels are thought to play an integral role in mood disorders (Maes & Meltzer, 1995), but the direct impact of diet on mood is difficult to quantify because so many complex systems are involved. Serotonin is a monoamine neurotransmitter that is found in carbohydrates and involved in the regulation of mood, sleep, and appetite. Increased serotonin activity in the brain improves the body’s ability to cope with acute stress (Anisman & Zacharko, 1992; Chaouloff, Berton, & Mormède, 1999). However, chronic exposure to

stress may lead to the continuous increase of serotonin activity resulting in an overloaded system and serotonin deficiency. Thus, one's vulnerability and reaction to stressors are important factors in determining serotonin activity (Markus et al., 1998, 2000).

When serotonin levels are low, the brain sends signals to notify the body that serotonin is needed, and this is when we have an impulse to eat. When the food we eat, particularly carbohydrates, are digested, insulin is secreted from the pancreas into the bloodstream, which delivers glucose to the cells to be used as an energy source. Because serotonin from carbohydrates is not activated until about 30 min after digestion, we may tend to overeat because we generally consume food quickly and do not stop until we feel full. If our bodies lack adequate amounts of serotonin, then the food cravings we experience are intense. We may then binge on fatty, salty and sweet food until we feel comfortable. A diet that includes carbohydrates with every meal should maintain our serotonin levels, decreasing impulses to eat unhealthy food (see Wurtman & Suffes, 1997, for additional details).

A diet rich in carbohydrates and low in proteins increases the brain's uptake of the large neutral amino acid (LNAA) tryptophan, the precursor to serotonin. Carbohydrates do not contain tryptophan, but they elevate glucose levels causing insulin secretion, which modifies the ratio of tryptophan to other LNAAs. Insulin has little impact on tryptophan levels, but it significantly decreases the level of other LNAAs such as valine, tyrosine, and leucine (Tadeka et al., 2004). Though proteins contain tryptophan, a diet high in proteins and low in carbohydrates decreases tryptophan availability because proteins possess higher levels of the other amino acids that are competing for access across the blood-brain barrier.

Increasing the uptake of carbohydrates may increase the ratio of tryptophan to other LNAAs by as much as 25%, but the effects on altering mood and coping are limited (Markus, Panhuysen, Jonkman, & Bachman, 1999). However, supplementing food with alpha-lactalbumin (ALAC), a whey-derived protein with high levels of tryptophan, has been shown to increase the plasma concentration of tryptophan (measured as a ratio of tryptophan to the sum of other neutral amino acids) by 50–130% and has been related to improved brain serotonin function, mood, and concentration (Booij, Merens, Markus, & Van der Does, 2006; Markus et al., 2000, 2005). More recently, the administration of hydrolyzed protein has yielded greater increases in plasma concentration of tryptophan and longer-lasting effects on improved mood when compared to the administration of alpha-lactalbumin and pure tryptophan in healthy participants (Markus, Firk, Gerhardt, Klock, & Smolders, 2008).

Though it has been established that tryptophan availability increases serotonin and that serotonin is involved in adaptation to stress, the mechanisms by which this occurs remains poorly understood. According to Soh and Walter (2011), there is little evidence to suggest that diet modification can elevate mood as is frequently reported in the popular media. For example, studies in humans often measure tryptophan levels in blood plasma which does not translate to a corresponding level in the brain. There remains great potential to find further dietary implications for serotonin activity and coping, and we await further research.

## Antioxidants and Stress

Antioxidants, which play a prominent role in maintaining physiological well-being, are another group of neurochemicals associated with diet and stress, and continue to be popular as supplements. Substances that act as antioxidants defend our bodies from an imbalance in the production of free radicals, which are capable of damaging cells and genetic material. Free radicals, which come in various sizes, shapes and chemical configurations, occur from many sources. The body produces free radicals as a byproduct of active aerobic metabolism (turning food into energy), but they are also in the food we eat, the air we breathe, and result from exposure to sunlight (Harvard School of Public Health, (n.d.), <http://www.hsph.harvard.edu/nutritionsource/what-should-you-eat/antioxidants/>). For the purpose of this chapter, since all cells in the human body use O<sub>2</sub> to break down the energy sources of carbohydrates, fats, and proteins, free radicals are formed when O<sub>2</sub> molecules lose an electron during this metabolic process. The free radicals, technically O<sub>2</sub> molecules now with a missing electron, will then attempt to stabilize themselves by indiscreetly stealing, or scavenging, an electron from any nearby molecule, whether the molecule is a protein, fat, or another chemical, such as nucleic acids. Unfortunately, free radicals may attach to and subsequently damage these chemicals in their stabilization quest.

An excess of free radicals, along with its damaging effects, has been referred to as oxidative stress (Sies, 1993), and is enhanced with aging (Knight, 2000). To defend against free radicals and to repair the damage caused by them, the body's cells produce antioxidants, which "donate" electrons to free radicals without turning into free radicals, or electron scavengers, themselves (Lobo, Patil, Phatak, & Chandra, 2010). This in essence may delay or halt the oxidation of other substances. There are likely thousands of substances that can act as antioxidants, but the most familiar ones are vitamin E, vitamin C, selenium, beta-carotene, lycopene, and other carotenoids (Valko et al., 2007).

The use of antioxidants as a means to counter the deleterious effects of free radicals or oxidative stress flourished in the 1990s, when data began to accumulate about the association among free radical damage and degenerative diseases (Harvard School of Public Health, n. d.). Some researchers noted that activation of the sympathetic nervous system increases the number of free radicals and is associated with contributing to heart failure (Belch, Bridges, Scott, & Chopra, 1991; McMurray, Chopra, Abdullah, Smith, & Dargie, 1993). Other studies have suggested the involvement of free radicals in other degenerative diseases, such as cancer, Alzheimer's disease, cognitive impairment, cataracts, and macular degeneration (Asian & Ozben, 2004; Meyer & Sekundo, 2005; Ryan-Harshman & Aldoori, 2005; Valko, Izakovic, Mazur, Rhodes, & Telser, 2004). Moreover, psychiatric illnesses, including autism, dementia, and schizophrenia, as well as anxiety, mood, sleeping and eating disorders have been associated with oxidative stress (Tsaluchidu, Cocchi, Tonello, & Puri, 2008).

In response to these, and other similar findings regarding the effects of oxidative stress, there has been a tremendous push in the past 15 years to increase the consumption of foods rich in antioxidants, and to assess their health benefits empirically. The rationale behind this surge is that if free radicals are contributing to chronic degenerative diseases, then substances with antioxidant properties should ameliorate the problem. More than 250 selected foods were evaluated for their Oxygen Radical Absorbance Capacity (ORAC), a measure of antioxidant success, and results revealed that fruits such as blueberries, acai berries, apples, plums, cranberries, and raspberries, vegetables such as artichokes, spinach, broccoli and sweet potatoes, beans such as kidney and pinto, nuts such as pecans, walnuts, pistachios, hazelnuts, and almonds, herbs such as cinnamon, oregano, cloves, and ginger, beverages such as green tea, red wine, coffee and fruit juices (e.g., pomegranate), and finally dark chocolate for dessert, are among the best sources of antioxidants (Bliss, 2007; Hensrud, 2009).

In 2000, the Food and Nutrition Board of the Institute of Medicine provided recommendations for antioxidant intake for healthy people. The recommended Daily Allowance (RDA) for vitamin E was 15 mg/day, for vitamin C it was 90 mg/day for adult men and 75 mg/day for adult women, with a recommended additional 35 mg/day for cigarette smokers because smoking increases oxidative stress and metabolic turnover of vitamin C, for selenium it was 55 µg/day, and for beta-carotene it was 3–6 mg/day. As part of its 2010 dietary guidelines, the USDA recommends that the average American eat approximately 4–5 cups of produce per day (2.5 cups of vegetables and 2 cups of fruit), or more simply, for every meal or eating occasion, make sure half of your plate contains fruits and vegetables. Obviously, many of the fruits and vegetables chosen are likely to have antioxidant properties. This rationale of fruit and vegetable consumption at every meal seems particularly prudent since the more antioxidants one has circulating in the body, the more free radicals are debilitated. Moreover, since the life cycle of most antioxidants in the diet is relatively brief (<http://www.organic-center.org>), replenishing them every 4–6 h provides the more continual effects (Breakstone, n.d.). However, antioxidants at higher concentrations may adversely impact lipids, proteins, and DNA (Valko et al., 2007).

In addition to the antioxidants found in natural food sources, there is a \$500 million dollar and growing antioxidant supplement industry (Harvard School of Public Health) that has proliferated in response to the touted benefits of antioxidants. In fact, a 2009 study assessing the use of antioxidant supplements reported that they accounted for 54% of vitamin C, 64% of vitamin E, 14% of carotenes, 11% of selenium, and 2% of flavonoid use for US adults (Chun et al., 2009). Despite these relatively high percentages for vitamins C and E, the use of antioxidant supplements may not be as effective as food in providing your body with maximum antioxidant benefits. Unlike supplements that often contain a single or maybe several types of antioxidants, foods contain thousands of types of antioxidants, and it may be this combination of substances in foods that interact with our body's cellular and genetic constituencies to produce a more-noted effect (Hensrud, 2009).

The benefits of antioxidants, whether in dietary or supplement form, have garnered almost panacea-like impact in the media and in consumer products for promoting health or preventing disease. Despite these rather lofty claims, the current empirical evidence suggests that the mechanisms of antioxidant action are reasonably complex, and randomized controlled trials offer little support that taking antioxidants provides substantial protection against chronic conditions such as heart disease or cancer (Cook et al., 2007; Hennekens et al., 1996; Lee et al., 2005; Lonn et al., 2005).

Success in the treatment of neurological disorders through the administration of antioxidants has been demonstrated in animal models, but not in humans, possibly due to human research being mostly observational. Moreover, studies often examine the association of reduced incidence of disease to consumption of foods rich in antioxidants, but fail to demonstrate that it is the antioxidants that are actually responsible for lowered risk (Krauss et al., 2000). It has been suggested that there is a need to more clearly define antioxidants for research purposes because many substances exhibit antioxidant qualities *in vitro*, but fail to do so *in vivo* (Kamat et al., 2008).

The current evidence in support of taking antioxidant supplements is less promising than its use in dietary form. A Cochrane Review, consisting of a comprehensive meta-analytic review of 67 randomized clinical trials of more than 230,000 participants assessing antioxidant supplements (beta-carotene, vitamin A, vitamin C, vitamin E and selenium) versus placebo concluded overall that the antioxidant supplements had no significant effect on mortality or on primary or secondary prevention and that beta-carotene, vitamin E and vitamin A are potentially harmful (Bjelakovic, Nikolova, Gluud, Simonetti, & Gluud, 2008). Although many experts have questioned the Cochrane Review's findings, an updated review of 78 randomized trials of close to 300,000 participants once again reached similar conclusions (Bjelakovic, Nikolova, Gluud, Simonetti, & Gluud, 2012). In light of the current inability to confirm direct links between antioxidants, stress, and ensuing pathologies, future intervention trials are needed to determine further the putative mechanisms involved and the recommended dietary intakes and benefits or adverse effects of supplemental intakes.

## **Stress and Appetite**

By the turn of this century in America, one-third of adults and one-sixth of children and adolescents were obese (Baskin, Ard, Franklin, & Allison, 2005). Obesity, defined as meeting or exceeding a body mass index (BMI) of 30 (National Institutes of Health, 2011), has been associated with daily stressors including job strain (Brunner, Chandola, & Marmot, 2007) and lower socioeconomic status (Kanjilal et al., 2006). From a neurochemical perspective, corticotrophin-releasing hormone has an appetite-suppressing effect during acute stress, whereas the residual cortisol

following stress has an appetite-stimulating effect. In addition, disturbances in HPA-axis functioning have been related to visceral fat accumulation (Rutters, Nieuwenhuizen, Lemmens, Born, & Westerterp-Plantenga, 2010), and cortisol levels have been associated with eating disorders (Lawson et al., 2011).

Stress in daily living has been associated with a preference for high calorie foods (Zellner et al., 2006). In fact, Rutters, Nieuwenhuizen, Lemmens, Born and Westerterp-Plantenga (2008) studied the effects of psychological stress on food intake and food preference in normal and overweight men and women, and reported that stress conditions were associated with food intake even in the absence of hunger and with a preference for sweet fatty foods. In a review of 22 studies, Adams, Minogue, and Lucock (2010) concluded that mental health problems are associated with poor dietary practices, including frequent consumption of saturated fat and refined sugar, and lower consumption of omega-3.

## Caffeinated Energy Drinks

The USA leads the world in the total volume sales of energy drinks, with worldwide consumption of these beverages reaching 906 million gallons in 2006 (Reissig, Strain, & Griffiths, 2009). Self-reports indicate that energy drinks are consumed by 30–50% of adolescents and young adults (Seifert, Schachter, Hershorin & Lipshultz, 2011), and in a sample of college students, those who used energy drinks reported they consumed them for insufficient sleep (67%) to increase energy (65%) and to use in conjunction with alcohol (54%) (Malinauskas, Aeby, Overton, Carpenter-Aeby, & Barber-Heidal, 2007). Although energy drinks contain natural products such as ginseng, taruine, and guarana, their quantity is typically well below what would be expected to produce either therapeutic benefits or adverse effects (Clauson, Shields, McQueen, & Persad, 2008). Caffeine, on the other hand, is the primary active ingredient in energy drinks, and while its content in a six ounce cup of coffee typically ranges between 77 and 150 mg, caffeinated energy drinks can reach 505 mg (Griffiths, Juliano, & Chausmer, 2003). Caffeine, which is a central nervous system stimulant, is considered safe in healthy adults at intake levels of less than 400 mg/day (Cannon, Cooke & McCarthy, 2001; Seifert et al.). Potential performance-enhancing effects of energy drink consumption have been suggested such as their beneficial use when given to sleepy drivers (Horne & Reyner, 2001) or their significant effects on task performance and self-rated mood (Smit & Rogers, 2002).

However, the frequently higher levels of caffeine intake in energy drinks may result in caffeine intoxication, which is recognized as a clinical disorder in both the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2004) and the International Classification of Diseases (World Health Organization, 2008). Symptoms of caffeine intoxication include insomnia, anxiety,

headache, tachycardia, psychomotor agitation, gastrointestinal irritation, increased blood pressure tremors, diabetes, mood disturbance, behavioral disorder, and in rare cases death (Clauson et al., 2008; O'Brien, McCoy, Rhodes, Wagoner, & Wolfson, 2008; Rogers, 2007; Scholey & Kennedy, 2004; Seifert et al., 2011; Whelton et al., 2002). These numerous adverse effects have in part led Seifert and her colleagues (2011) to conclude that, "energy drinks have no therapeutic benefit" (p. 522). Despite Seifert and colleagues claim, the consumer market for and use of energy drinks does not appear to be diminishing anytime soon. Therefore, it seems that additional controlled studies are needed to further assess the use and effects of energy drinks.

## Future Directions

In a recent article in the *American Psychologist*, Walsh (2011) suggested that health professionals have significantly underestimated the importance of nutrition and other lifestyle factors for mental health as well as the potential therapeutic benefits of proper nutrition in the treatment of psychopathology. He addressed the efficacy of various therapeutic lifestyle changes including diet modification, and included an evolutionary perspective. For example, he noted that just as ADHD may be a result of modern children developing in environments far different than those of our ancestors (Bjorklund & Pelligrini, 2002), the shift to a diet radically different than our ancestors likely contributes to many current pathologies. Moreover, some studies suggest that the impact of diet on mental health can be epigenetically transmitted across generations (Gómez-Pinilla, 2008).

Another of the recent trends in the past decade has been the surge of organic foods. In fact, organic food sales in the USA were 26.7 billion in 2010 (Organic Trade Association's 2011 Organic Industry Survey). Organic foods are grown and produced without the use of synthetic pesticides, artificial fertilizers, genetic engineering or ionizing radiation (used to kill bacteria), and the USDA has established requirements that all foods labeled as organic meet stringent government standards (Health & Nutrition, 2006). The absence of pesticides and fertilizers is thought to provide higher nutritional value than conventional food, and there are some data linking the use of pesticides with increasing the stress response and producing oxidative stress in bacterial cells and fish (Miller, Rasmussen, Palace, & Hontela, 2009; Özcan Oruc & Usta, 2007; Pham, Min, & Gu, 2004). However, a critical review of the literature on the safety of organic foods concluded that there are currently limited empirical data "to support or refute claims that organic food is safer and thus, healthier, than conventional food, or vice versa" (Magkos, Arvaniti, & Zampelsa, 2006, p. 47). Regardless, organic foods are infused in our mainstream culture and their popularity continues to grow dramatically. Additional longitudinal research will be helpful in determining their safety, and if they do, indeed, possess superior nutritional composition compared to conventional food production.

There also has been a recent increase in the production of designer foods, which have been commonly referred to in the literature as nutraceuticals or functional foods. Nutraceutical has been defined as any food, part of a food, supplement, genetically engineered food or diet that provides health benefits (DeFelice, 1992). Tadeka et al. (2004) described a functional food as having a tertiary function beyond nutritional necessities for survival and sensory satisfaction. Specifically, they described some tertiary functions as biorhythm regulation, immune and body defense, and as contributions to psychological life components. Many of these nutraceuticals or functional foods, such as omega-3 or antioxidant supplements were discussed in this chapter. As stated earlier, direct effects of supplemental interventions have not been reliably demonstrated.

One of the most promising fields of study to address these difficulties is that of nutrigenomics. Nutrigenomics is the study of the effects of nutrition at the genetic level (Ardekani & Jabbari, 2009). More specifically, it is the study of how nutrition influences homeostasis, and attempts to identify genes that are associated with risk of nutrition-related pathology. It is thought that with new developments in the genome map that optimal diets can be identified and individualized.

The potential for nutrigenomics is apparent in recent studies. For example, Bakker et al. (2010) demonstrated the modulation of inflammation, oxidative stress, and metabolism in overweight subjects utilizing an analysis of gene expression, proteins, and metabolites following the supplementation of diet with products including vitamin C and omega-3s. Sun, Morris, and Zemel (2008) utilized nutrigenomic methods to demonstrate that calcitrol regulates fat cell gene expression and cortisol production in fat cells, and that dietary calcium intake inhibits visceral fatty tissue gene expression. In studies of mice and humans, they identified a calcitrol/cortisol interaction in obesity suggesting that there is potential for dietary interventions to reduce fatty tissue through calcitrol suppression.

## Conclusion

The nutrients we consume, their proportion to other nutrients, and genetic predispositions influence how our body copes with stress. Many nutrients have been identified as particularly relevant to the regulatory systems of the stress response. These include antioxidants which exist in high levels in many plant-based foods, tryptophan which synthesizes into serotonin, and essential fatty acids such as omega-3s. Because our systems and pathways of metabolizing these nutrients are interactive and complex, as well as methodological challenging when conducting human research, it has been difficult to identify causal links of nutrients to specific pathologies. However, the associations between nutrition and physical and mental health are convincing. Pescovegetarian diets, such as the Mediterranean diet, are

frequently associated with better health outcomes and are recommended throughout the literature. The recent research advances in the developing field of nutrigenomics shows promise in identifying effective dietary interventions.

## Summary

This chapter presents a brief overview of how diet and nutrition are associated with the human stress response. The main points are:

1. Many health outcomes including physical and psychological pathologies have been associated with diet.
2. According to the USDA (2010), it is recommended that 45–65% of the human diet consists of carbohydrates, 10–35% consists of proteins, and 20–35% consists of fats. Our bodies rely on these energy sources when responding to stress.
3. Of the two general types of carbohydrates, simple and complex, simple carbohydrates (monosaccharides and disaccharides), or sugars come primarily from fruits, vegetables, and milk (or milk products), and are found in many different sweeteners (e.g., table sugar, honey, molasses). Complex carbohydrates (polysaccharides) are found in grains (e.g., rice, wheat, rye, barley, and oats), legumes (e.g., peanuts, peas, beans), and potatoes.
4. Proteins, composed of amino acids, are used to repair, rebuild, and replace cells. Eggs, milk, beef, fish, and soybeans are foods high in protein.
5. Fat provides the body with a concentrated energy source that may be especially relevant during periods of intense stress. Red meats, whole milk, cheeses, butter, bacon, chocolate, and nuts provide a large quantity of fat.
6. Excessive and deficient fat consumption are both associated with health risk. Omega-6 and omega-3 are two types of essential fatty acids, and an even consumption has been suggested for optimal functioning. Western diets significantly favor omega-6 EFAs, and this imbalance facilitates disease. Omega-3s are found in vegetables, vegetable oils, and saltwater fish.
7. During periods of excessive stress, the body, which usually conserves protein as its energy source, may begin to consume protein stores rapidly when carbohydrates and fats are depleted.
8. Serotonin influences sleep, mood, and appetite. Tryptophan, the precursor to serotonin, is found in proteins among many other large neutral amino acids. Though carbohydrates do not contain tryptophan, a diet rich in carbohydrates and low in protein enhances tryptophan uptake in the brain by modifying the ratio of tryptophan to competing amino acids.
9. Oxidative stress is the imbalance of the oxidant–antioxidant ratio in favor of the oxidants, or free radicals. Antioxidants inhibit or delay oxidation. Oxidative stress has been associated with many physical and mental disorders, and, even though the data are equivocal, there has been a proliferation in the use of dietary antioxidants and supplement antioxidants to try and mitigate the impact of

- oxidative stress. Appetite is an important trigger during psychosocial stressors. Stress has been associated with eating in the absence of hunger and a preference for sweet, calorie-dense foods.
10. Nutrition has been associated with pathology known to have a stress component, but direct causal links among nutrients and disease have not been conclusively demonstrated.
  11. Caffeinated energy drinks have been associated to date with adverse effects including tachycardia, anxiety, seizures, mood disturbance, and tremors, and more controlled studies are needed to further assess these claims.
  12. The surge in organic food consumption in the USA is expected to continue, and more empirical efficacy studies should be conducted.
  13. Nutrigenomics holds promise to identify and individualize optimum diets in light of one's genetic predisposition to disease. In the meantime, a pescovegetarian diet such as the Mediterranean diet has been consistently associated with positive health outcomes.

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## References

- Adams, K., Minogue, V., & Lucock, M. (2010). Nutrition and mental health recovery. *Mental Health and Learning Disabilities Research and Practice*, 7(1), 43–57.
- American Psychiatric Association. (2004). *Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder*. Arlington, VA: American Psychiatric Association. Available online at: <http://psychiatryonline.org/guidelines.aspx>.
- Anderson, J. W., Baird, P., Davis, R. H., Ferreri, S., Knudtson, M., Koraym, A., & Williams, C. L. (2009). Health benefits of dietary fiber. *Nutrition Reviews*, 67, 188–205.
- Anisman, H., & Zacharko, R. M. (1992). Depression as a consequence of inadequate neurochemical adaptation in response to stressors. *The British Journal of Psychiatry*, 160, 36–43.
- Ardekani, A. M., & Jabbari, M. (2009). Nutrigenomics and cancer. *Avicenna Journal of Medical Biotechnology*, 1(1), 9–17.
- Asian, M., & Ozben, T. (2004). Reactive oxygen and nitrogen species in Alzheimer's disease. *Current Alzheimer Research*, 1, 111–119.
- Bakker, G. C. M., van Erk, M. J., Pellis, L., Wopereis, S., Rubingh, C. M., Cnubben, N. H. P., & Hendriks, H. F. J. (2010). An antiinflammatory dietary mix modulates inflammation and oxidative and metabolic stress in overweight men: A nutrigenomics approach. *The American Journal of Clinical Nutrition*, 91, 1044–1059.
- Baskin, M. L., Ard, J., Franklin, F., & Allison, D. B. (2005). Prevalence of obesity in the United States. *Obesity Reviews*, 6, 5–7.
- Belch, J. J., Bridges, A. B., Scott, N., & Chopra, M. (1991). Oxygen free radicals and congestive heart failure. *British Heart Journal*, 65, 245–248.
- Bjelakovic, G., Nikolova, D., Gluud, L. L., Simonetti, R. G., & Gluud, C. (2008). Antioxidant supplements for prevention of mortality in healthy participants and patients with various diseases. *Cochrane Database of Systematic Reviews*, 2, CD007176.

- Bjelakovic, G., Nikolova, D., Gluud, L. L., Simonetti, R. G., & Gluud, C. (2012). Antioxidant supplements for prevention of mortality in healthy participants and patients with various diseases. *Cochrane Database of Systematic Reviews*, 3, CD007176.
- Bjorklund, D. F., & Pelligrini, A. D. (2002). *The origins of human nature*. Washington, DC: American Psychological Association.
- Bliss, R. M. (2007). *Data on food antioxidants aid research*. United States Department of Agriculture. Retrieved June 16, 2012, from <http://www.ars.usda.gov/is/pr/2007/071106.htm>
- Booij, L., Merens, W., Markus, C. R., & Van der Does, A. J. (2006). Diet rich in {alpha}-lactalbumin improves memory in unmedicated recovered depressed patients and matched controls. *Journal of Psychopharmacology*, 20, 526–535.
- Boston, P. F., Bennett, A., Horrobin, D. F., & Bennett, C. N. (2004). Ethyl-EPA in Alzheimer's disease – A pilot study. *Prostaglandins, Leukotrienes, and Essential Fatty Acids*, 71, 341–346.
- Bousquet, M., Gue, K., Emond, V., Julien, P., Kang, J. X., Cicchetti, F., & Calon, F. (2011). Transgenic conversion of omega-6 into omega-3 fatty acids in a mouse model of Parkinson's Disease. *Journal of Lipid Research*, 52, 263–271.
- Breakstone, S. (n.d.). Eat to live longer. Shed unwanted pounds and slow aging with 6 tops diet tips. *Prevention*. Available online: <http://www.prevention.com/eattolivelonger/index.shtml>
- Brehm, B. A. (1998). *Stress management: Increasing your stress resistance*. New York, NY: Longman.
- Brunner, E. J., Chandola, T., & Marmot, M. G. (2007). Prospective effect of job strain on general and central obesity in the Whitehall II study. *American Journal of Epidemiology*, 165, 828–837.
- Cannon, M. E., Cooke, C. T., & McCarthy, J. S. (2001). Caffeine-induced cardiac arrhythmia: An unrecognised danger of healthfood products. *Medical Journal of Australia*, 174(10), 520–521.
- Carlsen, M. H., Halvorsen, B. L., Holte, K., Bohn, S. K., Dragland, S., Sampson, L., ... Blomhoff, R. (2010). The total antioxidant content of more than 3100 foods beverages, spices, herbs and supplements worldwide. *Nutrition Journal*, 9. Retrieved from <http://www.nutritionj.com/content/9/1/3>
- Chaouloff, F., Berton, O., & Mormède, P. (1999). Serotonin and stress. *Neuropsychopharmacology*, 21, 28S–32S.
- Charrondiere, U. R., Chevassus-Agnes, S., Marroni, S., & Burlingame, B. (2004). Impact of different macronutrient definitions and energy conversion factors on energy supply estimations. *Journal of Food Composition and Analysis*, 17, 339–360.
- Chun, O. K., Floegel, A., Chung, S.-J., Chung, C. E., Song, W. O., & Koo, S. I. (2009). Estimation of antioxidant intakes from diet and supplements in U.S. adults. *The Journal of Nutrition*, 140, 317–324.
- Clauson, K. A., Shields, K. M., McQueen, C. E., & Persad, N. (2008). Safety issues associated with commercially available energy drinks. *Pharmacy Today*, 14(5), 52–64.
- Cook, N. R., Albert, C. M., Gaziano, J. M., Zaharris, E., MacFadyen, J., Danielson, E., Manson, J. E. (2007). A randomized factorial trial of vitamins C and E and beta carotene in the secondary prevention of cardiovascular events in women: Results from the Women's Antioxidant Cardiovascular Study. *Archives of Internal Medicine*, 167(15), 1610–1618.
- DeFlice, S. L. (1992). The nutraceutical initiative: a recommendation for U.S. economic and regulatory reforms. *Genetic Engineering News*, 12, 13–15.
- Eschelmann, M. M. (1996). *Introductory nutrition and nutrition therapy* (3rd ed.). Philadelphia, PA: Lippincott.
- Food and Nutrition Board Institute of Medicine. (2000). *Dietary reference intakes for vitamin C, vitamin E, selenium, and carotenoids (A Report of the Panel on Dietary Antioxidants and Related Compounds, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes)*. Washington, DC: National Academy Press.
- Frautschy, S. A., Hu, W., Kim, P., Miller, S. A., Chu, T., Harris-White, M. E., & Cole, G. M. (2001). Phenolic anti-inflammatory antioxidant reversal of A $\beta$  induced cognitive deficits and neuropathology. *Neurobiology of Aging*, 22, 993–1005.

- GISSI Prevenzione Investigators. (1999). Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: Results of the GISSI-Prevenzione trial. Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico. *Lancet*, *354*, 447–455.
- Gómez-Pinilla, F. (2008). Brain foods: The effects of nutrients on brain function. *Nature Reviews Neuroscience*, *9*, 568–578.
- Greenberg, J. S., Dintiman, G. B., & Myers-Oakes, B. (1998). *Physical fitness and wellness* (2nd ed.). Boston, MA: Allyn & Bacon.
- Griffiths, R. R., Juliano, L. M., & Chausmer, A. L. (2003). Caffeine: Pharmacology and clinical effects. In A. W. Graham, T. K. Schultz, M. F. Mayo-Smith, R. K. Ries, & B. B. Wilford (Eds.), *Principles of addiction medicine* (3rd ed., pp. 193–224). Chevy Chase, MD: American Society of Addiction.
- Groff, J., Cropper, S., & Hunt, S. M. (1995). *Advanced nutrition and human metabolism* (2nd ed.). Minneapolis, St. Paul: West.
- Hall, J. E. (2011). *Guyton and Hall Textbook of Medical Physiology* (12th ed.). Philadelphia, PA: Saunders Elsevier.
- Hamazaki, T., Sawazaki, S., Nagasawa, T., Nagao, Y., Kanagawa, Y., & Yazawa, K. (1999). Administration of docosahexaenoic acid influence behavior and plasma catecholamine levels at times of psychological stress. *Lipids*, *34*, S33–S37.
- Harvard School of Public Health (n.d.). The nutrition source. Antioxidants Beyond the hype. Retrieved from: <http://www.hsph.harvard.edu/nutritionsource/what-should-you-eat/antioxidants/>
- Health & Nutrition (2006). *Get the facts about organic foods*. <http://cleveland.ces.ncsu.edu/index.php?page=news&ci=HEAL+4>. Retrieved June 19, 2012.
- Hennekens, C. H., Buring, J. E., Manson, J. E., Stampfer, M., Rosner, B., Cook, N. R., & Peto, R. (1996). Lack of effect of long-term supplementation with beta carotene on the incidence of malignant neoplasms and cardiovascular disease. *New England Journal of Medicine*, *334*(18), 1145–1149.
- Hensrud, D. (2009). Food sources the best choice for antioxidants. Medical Edge Newspaper Column. Mayo Clinic. [www.mayoclinic.org/medical-edge-newspaper-2009/jun-05b.htm](http://www.mayoclinic.org/medical-edge-newspaper-2009/jun-05b.htm). Retrieved June 16, 2012
- Hirayama, S., Hamazaki, T., & Terasawa, K. (2004). Effect of docosahexaenoic acid-containing food administration on symptoms of attention deficit/hyperactivity disorder – a placebo-controlled double-blind study. *European Journal of Clinical Nutrition*, *58*, 467–473.
- Horne, J. A., & Reyner, L. A. (2001). Beneficial effects of an “energy drink” given to sleepy drivers. *Amino Acids*, *20*, 83–89.
- Institute of Medicine. (2005). *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients)*. Washington DC: National Academies Press.
- Kamat, C. D., Gadal, S., Mhatre, M., Williamson, K. S., Pye, Q. N., & Hensley, K. (2008). Antioxidants in central nervous system diseases: Preclinical promise and translational challenges. *Journal of Alzheimer's Disease*, *15*, 473–493.
- Kanjilal, S., Gregg, E. W., Cheng, Y. J., Zhang, P., Nelson, D. E., & Mensah, G., et al. (2006). Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971–2002. *Archives of Internal Medicine*, *166*, 2348–2355.
- Keck, P. E., Mintz, J., McElroy, S. L., Freeman, M. P., Suppes, T., Frye, M. A., & Post, R. M. (2006). Double-blind, randomized, placebo-controlled trials of ethyl-eicosapentanoate in the treatment of bipolar depression and rapid cycling bipolar disorder. *Biological Psychiatry*, *60*, 1020–1022
- Knight, J. A. (2000). The biochemistry of aging. *Advances in Clinical Chemistry*, *35*, 1–62.
- Krauss, R. M., Eckel, R. H., Howard, B., Appel, L. J., Daniels, S. R., Deckelbaum, R. J., ... Bazzarre, T. L. (2000). Revision 2000: A statement for healthcare professionals from the nutrition committee of the American Heart Association. *Circulation*, *102*, 2284–2289
- Lawson, E. A., Eddy, K. T., Donoho, D., Misra, M., Miller, K. K., Meenaghan, E., ... Klibanski, A. (2011). Appetite-regulating hormones cortisol and peptide YY are associated with disordered

- eating psychopathology, independent of body mass index. *European Journal of Endocrinology*, *164*, 253–261.
- Lee, I. M., Cook, N. R., Gaziano, J. M., Gordon, D., Ridker, P. M., Manson, J. E., ... Buring, J. E. (2005). Vitamin E in the primary prevention of cardiovascular disease and cancer: The Women's Health Study: A randomized controlled trial. *Journal of the American Medical Association*, *294*(1), 56–65
- Lobo, V., Patil, A., Phatak, A., & Chandra, N. (2010). Free radicals, antioxidants, and functional foods: Impact on human health. *Pharmacognosy Review*, *4*, 118–126.
- Lonn, E., Bosch, J., Yusuf, S., Sheridan, P., Pogue, J., Arnold, J. M. ... HOPE and HOPE-TOO Trial Investigators (2005). Effects of long-term vitamin E supplementation on cardiovascular events and cancer: A randomized controlled trial. *Journal of the American Medical Association*, *293*(11), 1338–1347
- Maes, M., & Meltzer, H. (1995). The serotonin hypothesis of major depression. In F. E. Bloom & D. J. Kupfer (Eds.), *Psychopharmacology: The fourth generation of progress* (pp. 933–934). New York: Raven Press.
- Macht, M. (1996). Effects of high-and low-energy meals on hunger, physiological processes and reactions to emotional stress. *Appetite*, *26*(1), 71–88.
- Magkos, R., Arvaniti, F., & Zampelas, A. (2006). Organic food: Buying more safety or just peace of mind? A critical review of the literature. *Critical Reviews in Food Science and Nutrition*, *46*, 23–56.
- Malinauskas, B. M., Aeby, V. G., Overton, R. F., Carpenter-Aeby, T., & Barber-Heidal, K. (2007). A survey of energy drink consumption patterns among college students. *Nutrition Journal*, *6*(35), 1–7; Available online: <http://www.biomedcentral.com/content/pdf/1475-2891-6-35.pdf>
- Markus, C. R., Firk, C., Gerhardt, C., Klock, J., & Smolders, G. F. (2008). Effect of different tryptophan sources on amino acids availability to the brain and mood in health volunteers. *Psychopharmacology*, *201*, 107–114.
- Markus, C. R., Jonkman, L. M., Lammers, J. H., Deutz, N. E., Messer, M. H., & Rigtering, M. (2005). Evening intake of alpha-lactalbumin increases plasma tryptophan availability and improves morning alertness and brain measures of attention. *American Journal of Clinical Nutrition*, *81*, 1026–1033.
- Markus, C. R., Olivier, G., Paynuysen, G., Van der Gugten, J., Alles, M. S., Tuiten, A., ... de Hann, E. (2000). The bovine protein  $\alpha$ -Lactalbumin increases the plasma ratio of tryptophan to the other large neutral amino acids, and in vulnerable subjects raises brain serotonin activity, reduces cortisol concentration, and improves mood under stress. *American Journal of Clinical Nutrition*, *71*, 1536–1544.
- Markus, C. R., Panhuysen, G., Jonkman, L. M., & Bachman, M. (1999). Carbohydrate intake improves cognitive performance of stressprone individuals under controllable laboratory stress. *British Journal of Nutrition*, *82*, 457–467.
- Markus, C. R., Panhuysen, G., Tuiten, A., Koppenschaar, H., Fekkes, D., & Peters, M. L. (1998). Does carbohydrate-rich, protein-poor food prevent a deterioration of mood and cognitive performance of stress-prone subjects when subjected to a stressful task? *Appetite*, *31*, 49–65.
- McMurray, J., Chopra, M., Abdullah, I., Smith, W. E., & Dargie, H. J. (1993). Evidence of oxidative stress in chronic heart failure in humans. *European Heart Journal*, *14*, 1493–1498.
- Meyer, C. H., & Sekundo, W. (2005). Nutritional supplementation to prevent cataract formation. *Developmental Ophthalmology*, *38*, 103–119.
- Miller, L. L., Rasmussen, J. B., Palace, V. P., & Hontela, A. (2009). Physiological stress response in white suckers from agricultural drain waters containing pesticides and selenium. *Ecotoxicology and Environmental Safety*, *72*, 1249–1256.
- National Institutes of Health. (2011). *Overweight*. Retrieved from <http://www.nlm.nih.gov/medlineplus/ency/article/003101.htm>
- O'Brien, M. C., McCoy, T., Rhodes, S. D., Wagoner, A., & Wolfson, M. (2008). Caffeinated cocktails: Get wired, get drunk, get injured. *Academy of Emergency Medicine*, *15*, 453–460.

- Organic Trade Association. (2011). *Industry statistics and projected growth*. <http://www.ota.com/organic/mt/business.html>. Retrieved June 19, 2012
- Özcan Oruc, E., & Usta, D. (2007). Evaluation of oxidative stress responses and neurotoxicity potential of diazinon in different tissues of *Cyprinus carpio*. *Environmental Toxicology and Pharmacology*, *23*, 48–55.
- Pham, C. H., Min, J., & Gu, M. B. (2004). Pesticide induced toxicity and stress response in bacterial cells. *Bulletin of Environmental Contamination and Toxicology*, *72*, 380–386.
- Polivy, J., & Herman, C. P. (1999). Distress and eating: Why do dieters overeat? *International Journal of Stress Management*, *5*(1), 57–75.
- Reissig, C. J., Strain, E. C., & Griffiths, R. R. (2009). Caffeinated energy drinks – A growing problem. *Drug and Alcohol Dependence*, *99*(1), 1–10.
- Richardson, A. J., & Montgomery, P. (2005). The Oxford-Durham Study: A randomized, controlled trial of dietary supplementation with fatty acids in children with developmental co-ordination disorder. *Child: Care, Health, and Development*, *31*, 629–630.
- Riediger, N. D., Othman, R. A., Suh, M., & Moghadasian, M. H. (2009). A systematic review of n-3 fatty acids in health and disease. *Journal of the American Dietetic Association*, *109*, 668–679.
- Rogers, P. J. (2007). Caffeine, mood and mental performance in everyday life. *Nutrition Bulletin*, *32*(suppl 1), 84–89.
- Rosetti, R. G., Seiler, C. M., Deluca, P., Laposata, M., & Zurier, R. B. (1997). Oral administration of unsaturated fatty acids: Effects on human peripheral blood T lymphocyte proliferation. *Journal of Leukocyte Biology*, *62*, 438–443.
- Rutters, F., Nieuwenhuizen, A. G., Lemmens, S. G. T., Born, J. M., & Westerterp-Plantenga, M. S. (2008). Acute stress-related changes in eating in the absence of hunger. *Obesity*, *17*, 72–77.
- Rutters, F., Nieuwenhuizen, A. G., Lemmens, S. G. T., Born, J. M., & Westerterp-Plantenga, M. S. (2010). Hypothalamic-pituitary-adrenal (HPA) axis functioning in relation to body fat distribution. *Clinical Endocrinology*, *72*, 738–743.
- Ruxton, C. H. S., Gardner, E. J., & McNulty, H. M. (2010). Is sugar consumption detrimental to health? A review of the evidence 1995–2006. *Critical Reviews in Food Science and Nutrition*, *50*(1), 1–19.
- Ryan-Harshman, M., & Aldoori, W. (2005). The relevance of selenium to immunity, cancer, and infectious/inflammatory diseases. *Canadian Journal of Dietetic Practice and Research*, *66*, 98–102.
- Sawazaki, S., Hamazaki, T., Yazawa, K., & Kobayashi, M. (1999). The effect of docosahexaenoic acid on plasma catecholamine concentrations and glucose tolerance during long-lasting psychological stress: A double blind placebo-controlled study. *Journal of Nutritional Science and Vitaminology*, *45*, 655–665.
- Scholey, A. B., & Kennedy, D. O. (2004). Cognitive and physiological effects of an “energy drink”: An evaluation of the whole drink and of glucose, caffeine and herbal flavouring fractions. *Psychopharmacology*, *176*(3–4), 320–330.
- Seifert, S. M., Schachter, J. L., Hershorin, E. R., & Lipshulz, S. E. (2011). Health effects of energy drinks on children, adolescents, and young adults. *Pediatrics*, *127*, 511–528.
- Sies, H. (1993). Strategies of antioxidant defense. *European Journal of Biochemistry*, *215*, 213–219.
- Simopoulos, A. P. (2001). The Mediterranean diets: What is so special about the diet of Greece? The scientific evidence. *Journal of Nutrition*, *131*, 3065S–3073S.
- Simopoulos, A. P. (2006). Evolutionary aspects of diet, the omega-6/omega-3 ratio and genetic variation: nutritional implications for chronic diseases. *Biomedicine & Pharmacotherapy*, *60*, 502–507.
- Smit, H. J., & Rogers, P. J. (2002). Effects of “energy” drinks on mood and mental performance: Critical methodology. *Food Quality and Preference*, *13*(5), 317–326.

- Sofi, F., Cesari, F., Abbate, R., Gensini, F., & Casini, A. (2008). Adherence to Mediterranean diet and health status: Meta-analysis. *British Medical Journal*. Available Online: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2533524/pdf/bmj.a1344.pdf>
- Soh, N. L., & Walter, G. (2011). Tryptophan and depression: Can diet alone be the answer? *Acta Neuropsychiatrica*, 23, 3–11.
- Stoll, A. L., Locke, C. A., Marangell, L. B., & Severus, W. E. (1999). Omega-3 fatty acids and bipolar disorder: A review. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, 60(5–6), 329–337.
- Sun, X., Morris, K. L., & Zemel, M. B. (2008). Role of calcitriol and cortisol on human adipocyte proliferation and oxidative and inflammatory stress: a microarray study. *Journal of Nutrigenetics and Nutrigenomics*, 1(1–2), 30–48.
- Tadaka, E., Terao, J., Nakaya, Y., Miyamoto, K., Baba, Y., Chuman, H., & Rokutan, K. (2004). Stress control and human nutrition. *The Journal of Medical Investigation*, 51, 139–145.
- Tajalizadekhoob, Y., Sharifi, F., Fakhrazadeh, H., Mirarefin, M., Ghaderpanahi, M., Badamchizade, Z., & Azimipour, S. (2011). The effect of low-dose omega 3 fatty acids on the treatment of mild to moderate depression in the elderly: a double-blind, randomized, placebo-controlled study. *European Archives of Psychiatry and Clinical Neuroscience*. Advance Online Publication. Available from <http://www.ncbi.nlm.nih.gov/pubmed/>
- Torres, S., & Nowson, C. (2007). Relationship between stress, eating behavior and obesity. *Nutrition*, 23(11–12), 887–894.
- Tsaluchidu, S., Cocchi, M., Tonello, L., & Puri, B. (2008). Fatty acids and oxidative stress in psychiatric disorders. *BMC Psychiatry*, 8, S1–S5.
- U.S. Department of Veterans Affairs, Veterans Health Administration, Office of Public Health and Environmental Hazards. (2010). Analysis of VA health care utilization among U.S. Global War of Terrorism (GWOT) veterans. Unpublished quarterly report (cumulative through 4<sup>th</sup> quarter FY 2009). Washington, DC: Author.
- United States Department of Agriculture. (2010). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans*. Retrieved from <http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/DGAC/Report/2010DGACReport-camera-ready-Jan11-11.pdf>
- Valko, M., Izakovic, M., Mazur, M., Rhodes, C. J., & Telser, J. (2004). Role of oxygen radicals in DNA damage and cancer incidence. *Molecular Cell*, 266, 37–57.
- Valko, M., Leibfritz, D., Moncol, J., Cronin, M., Mazur, M., & Telser, J. (2007). Free radicals and antioxidants in physiological functions and human disease. *The International Journal of Biochemistry & Cell Biology*, 39, 44–84.
- Voigt, R. G., Llorente, A. M., Jensen, C. L., Fraley, J. K., Berretta, M. C., & Heird, W. C. (2001). A randomized, double-blind, placebo-controlled trial of docosahexaenoic acid supplementation in children with attention deficit/hyperactivity disorder. *The Journal of Pediatrics*, 139, 189–196.
- Walsh, R. (2011). Lifestyle and mental health. *American Psychologist*, 66(7), 579–592.
- Wertz, P. W. (2009). Essential fatty acids and dietary stress. *Toxicology and Industrial Health*, 25, 279–283.
- Whelton, P. K., He, J., Appel, L. J., Cutler, J. A., Havas, S., Kotchen, T. A., ... Karimbakas, J. (2002). Primary prevention of hypertension: Clinical and public health agency advisory from the National High Blood Pressure Education Program. *Journal of the American Medical Association*, 288(15), 1882–1888.
- Whitney, E. N., Hamilton, E. M., & Rolfes, S. R. (1990). *Understanding nutrition* (5th ed.). St. Paul, MN: West.
- Whitney, E., & Rolfes, S. R. (2011). *Understanding nutrition* (12th ed.). Belmont, CA: Wadsworth.
- Williams, M. H. (2007). *Nutrition for health, fitness, and sport* (8th ed.). Boston: McGraw-Hill.
- Wolraich, M. L., Wilson, D. B., & White, J. W. (1995). The effect of sugar on behavior or cognition in children. A meta-analysis. *Journal of the American Medical Association*, 274, 1617–1621.

- World Health Organization. (2008). *ICD-10: International statistical classification of diseases and related health problems* (10 Revth ed.). New York, NY: Author.
- Wurtman, J., & Suffes, S. (1997). *The serotonin solution: To achieve permanent weight control*. New York: Ballentine Books.
- Yehuda, S., Rabinovitz, S., & Mostofsky, D. I. (1997). Effects of essential fatty acids preparation (SR-3) on brain biochemistry and on behavioral and cognitive functions. In S. Yehuda & D. I. Mostofsky (Eds.), *Handbook of essential fatty acids biology: Biochemistry physiology and behavioral neurobiology* (pp. 427–452). Totawa, NJ: Humana.
- Yehuda, S., Rabinovitz, S., & Mostofsky, D. I. (2006). Essential fatty acids and stress. In S. Yehuda & D. I. Mostofsky (Eds.), *Nutrients, stress, and medical disorders* (pp. 99–110). Totawa, NJ: Humana.
- Zellner, D. A., Loaiza, S., Gonzalez, Z., Pita, J., Morales, J., Pecora, D., & Wolf, A. (2006). Food selection changes under stress. *Physiological Behavior*, *87*, 789–793.