Nutritional Aspects of Eating Disorders, Addictions, and Substance Use Disorders

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Laurie M. McCormick, Obiora E. Onwuameze, and Sergio Paradiso

Abstract

Substance use disorders and eating disorders can lead to unhealthy eating and malnutrition that is associated with varying degrees of medical morbidity and increased risk of death. Vitamin and mineral deficiencies are most common in patients with anorexia nervosa, bulimia nervosa, and alcohol use disorders but can be seen in other substance use disorders as well. These disorders may share some underlying pathophysiology and can co-occur, which further increases the risk of malnutrition. The specific nutritional deficits in eating disorders and substance use disorders, their clinical (especially neurological) manifestations, how commonly they occur, and their mechanisms of action will be discussed in this chapter.

Keywords

Eating disorders • Substance use disorders • Nutritional deficiency • Alcohol use disorders • Anorexia nervosa • Binge eating disorder • Bulimia nervosa

8.1 Introduction

The importance of nutrition for healthy living was discussed as early as the fifth century when Hippocrates suggested that many health problems could be prevented or alleviated with healthy diet (and exercise) (Simopoulos, 2001). Nutrition refers

L.M. McCormick (✉)
Department of Psychiatry, Carver College of Medicine, University of Iowa, 200 Hawkins Drive, W278 GH, Iowa City, IA 52242, USA
e-mail: laurie-mccormick@uiowa.edu

O.E. Onwuameze
Department of Psychiatry, Southern Illinois University Medical School, Springfield, IL, USA

S. Paradiso
Una Mano per la Vita – Association of Families and their Doctors, Catania, Italy

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to the supply of foods or supplements for the growth and maintenance of a living organism. There are six major classes of nutrients. These include carbohydrates, proteins, fats, water, vitamins, and minerals. Over the centuries, specific nutritional deficiencies have been shown to carry deleterious health effects. One of the earliest discoveries was made by Dr. James Lind who in 1747 treated scurvy among British sailors and seafarers with lime juice. In the early 1930s it was found that scurvy is a disease due to deficit of the essential nutrient vitamin C. Similarly, Japanese sailors whose diets consisted primarily of white rice developed beriberi (a constellation of neurological and cardiac deficits) from which they often died. This condition was later found to be due to thiamine deficiency. Since then, numerous medical disorders have been linked to malnutrition or specific nutrient deficiencies. Many governments throughout the world have set forth specific guidelines for food consumption and daily requirements of vitamins and minerals.

There are a number of psychiatric conditions that predispose people to develop malnutrition, most commonly addiction to substances substance use disorders (SUD) or disordered eating from eating disorders (ED) anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED). Malnutrition can be seen in other psychiatric conditions such as schizophrenia when delusions are related to food intake (McCormick, Buchanan, Onwuameze, Pierson, & Paradiso, 2011) or in children with attention-deficit hyperactivity disorder when appetite is decreased from stimulant use (Davis et al., 2007; Schachter, King, Langford, & Moher, 2001). This chapter will primarily focus on the mechanisms of malnutrition and their sequelae occurring among individuals suffering with SUD and ED and especially when these two conditions co-occur.

8.2 Mechanisms and Clinical Manifestations of Malnutrition in SUD

The first descriptions of addiction to substances were found in Sumerian writings as early as 6,000 years BC (Davenport-Hines, 2003; Krikorian, 1975). SUD have become known today as a cluster of cognitive, behavioral, and psychological symptoms indicating that an individual continues using substances despite substance-related problems (American Psychiatric Association, 2013). The most recent version of the Diagnostic and Statistical Manual of Mental Illness (DSM-5) considers what was formerly indicated as substance abuse and dependence as a continuum of disorders involving the use of substances from mild to severe, according to the number of endorsed symptoms (American Psychiatric Association, 2013). The threshold for diagnosing mild substance use disorders is set at two symptoms (or criteria) from a list of 11. Craving is a symptom not present in the previous version of the Manual (i.e., a strong desire to use a substance). Problems with law enforcement are eliminated due to cultural considerations.

Malnutrition and nutritional deficiencies are common among people with SUD (Abd El Gawad, Hassan, Ghanem, Awad, & Ali, 2011; Gueguen et al., 2003; Islam,

Hossain, Ahmed, & Ahsan, 2002; Saeland et al., 2008). SUD impact nutrition and diet in at least two ways (1) through reduced food intake and by the deleterious effects of the used substances on the body. It is plausible that combination effects may be multiplicative rather than simply additive. Perhaps because of the alteration in reward and impulse control pathways, people who suffer with SUD show poor decision-making in nutritional choices that can create a variety of deficiencies and/or excesses of certain nutrients. Diminished and/or poor nutritional choices may result in noxious effects on the body irrespective of the substance abused. With poor nutrition, the most common nutrients impacted are the vitamins and rare minerals that are supplied from fresh vegetables and fruits including vitamin A (e.g., carotene), B vitamins (thiamine, niacin, pantothenic acid, and biotin), vitamin C, vitamin E, folic acid, copper, potassium, manganese, magnesium, molybdenum, and selenium. Some drugs including cocaine, marijuana, amphetamine, and heroin may impact food intake by reducing appetite (Anglin, Burke, Perrochet, Stamper, & Dawud-Noursi, 2000; Mohs, Watson, & Leonard-Green, 1990). Some substances of abuse have been shown to be directly neurotoxic such as ecstasy, inhalants, hallucinogens, and methamphetamine (Ares-Santos, Granado, & Moratalla, 2013; Cairney et al., 2013; Capela et al., 2013; Cowan, Roberts, & Joers, 2008; Marona-Lewicka, Nichols, & Nichols, 2011; Takagi, Lubman, & Yucel, 2011). The specific nutritional impact of some of the more commonly used substances will be discussed in more detail below.

A nationwide study in 2008 showed that alcohol is the most commonly abused substance in the USA, followed by cannabis and methamphetamine abuse (Substance Abuse and Mental Health Services Administration, 2011). Excess use of this substance is known to cause deficiency of several vitamins and minerals through impaired absorption, metabolism, and utilization (Lieber, 2003; McClain, Barve, Barve, & Marsano, 2011; Strohle, Wolters, & Hahn, 2012). Alcohol can exert direct effects on metabolism or effects due to the progression of medical conditions associated with alcohol overuse (e.g., hepatitis and dilated cardiomyopathy). Impaired absorption is in the early stages secondary to the effects of alcohol to the gastric mucosa. In later stages of the illness, poor absorption develops due to gastritis secondary to cirrhosis and the relative venous stasis in the portal splanchnic district. Metabolism is generally altered in various degrees according to the severity of accompanying hepatic pathology. Vitamin deficiencies in alcohol use disorders (AUD) include vitamin A, vitamins of the B family (thiamine, riboflavin, niacin, pantothenic acid, pyridoxine, and cobalamin), folic acid, vitamin C, vitamin D, and vitamin E (Clugston & Blaner, 2012; Strohle et al., 2012). The mineral deficiencies associated with AUD include calcium, magnesium, and zinc (Devgun, Fiabane, Paterson, Zarembski, & Guthrie, 1981). These deficiencies are thought to play a role in some forms of dementia and depression as well as poor appetite (Coppen, 2005; Glick, 1990; Ramsey & Muskin, 2013; Rasmussen, Mortensen, & Jensen, 1989; Tiemeier et al., 2002). Men with AUD have been shown to have low levels of vitamin D and are at an increased risk of osteoporosis due to direct and indirect effects on bone remodeling and formation, which leads to increased risk for fractures (González-Reimers et al., 2011). Similarly, men with AUD may be at

risk for bleeding problems secondary to vitamin K deficiency by disturbed absorption through the liver, which is often impaired from heavy alcohol use (Iber, Shamszad, Miller, & Jacob, 1986).

The most serious and life-threatening deficiency that occurs in AUD is a deficiency of B vitamins including thiamine and folic acid. These deficiencies are known to be associated with dementia (Goebels & Soyka, 2000). Wernicke's encephalopathy and Korsakoff's syndrome are severe life-threatening conditions that can occur in a thiamine-deficient state induced by poor nutrition often caused by severe alcoholism (Lough, 2012). There are times in which the lack of one B vitamin impairs the utilization of another. For example, folic acid cannot be utilized in the body in the absence of B12 and when deficient folic acid can mask vitamin B12 deficiency as well.

The impact of addiction to tetrahydrocannabinol (THC) in cannabis on nutrients has not been studied to the same extent as alcohol. Results from the third national health and nutrition examination survey showed higher cigarette-smoking rates and higher consumption of sodas and alcohol, including beer, among current cannabis users than among non-current cannabis users (Smit & Crespo, 2001). Cannabis users also consumed more sodium, fewer fruits, and more pork, cheese, and salty snacks, which resulted in lower serum carotenoid levels (i.e., vitamin A deficiency) (Smit & Crespo, 2001). Vitamin A deficiency can result in night blindness (complete blindness in severe deficiency), impaired immunity to infections, predisposition to cancer, and even birth defects. While cannabis can increase appetite in some people, several studies and two national surveys have found that active cannabis users (using at least three times a week) actually have a lower body mass index (BMI) than persons who do not use cannabis (Le Strat & Le Foll, 2011; Penner, Buettner, & Mittleman, 2013; Rodondi, Pletcher, Liu, Hulley, & Sidney, 2006). The studies revealed that smoking cannabis was also associated with a smaller waist circumference and lower insulin levels than those who do not use cannabis (Penner et al., 2013; Rodondi et al., 2006).

Illicit stimulant substances such as cocaine and amphetamine (methamphetamine) have been shown to decrease appetite when using and when withdrawing from substance (Walsh, Donny, Nuzzo, Umbricht, & Bigelow, 2010). Calorie and protein malnutrition has been found in people who use cocaine chronically (Santolaria-Fernández et al., 1995). Rodent models of cocaine addiction have also found that to cocaine continued use of the substance and neglect food (Sanchis-Segura, & Spanagel, 2006). Cocaine and methamphetamine have been found to be especially neurotoxic to dopaminergic neurons in the basal ganglia and hippocampus in animal models (Aksenov et al., 2006; Ares-Santos et al., 2013; Capela et al., 2013; Olsen, 1995) and have been linked to myocardial infarctions in humans (Galasko, 1997; Isner et al., 1986; Lange & Hillis, 2001). More data is needed to fully understand the specific nutritional deficiencies that occur with addiction to stimulant substances, but there appears to be at least indirect effects of poor appetite causing reduced consumption of nutritious food.

Poor appetite leading to poor intake of nutrients is also the likely cause of malnutrition in people primarily addicted to opiates (Meleger, Froude, & Walker, 2013; Morabia et al., 1989; Neale, Nettleton, Pickering, & Fischer, 2012). In a study of 149 people addicted to heroin 25 % were found to have hypovitaminemia, primarily of B vitamins, which are water soluble and are most vulnerable during poor intake of regular food (el-Nakah, Frank, Louria, Quinones, & Baker, 1979). Hyperkalemia (or increased blood potassium) has been reported in heroin addiction, while calcium deficiency has been reported in morphine abusers (Mohs et al., 1990). Hyperkalemia can lead to deadly arrhythmias, while calcium deficiency can cause altered bone metabolism and reduced trabecular bone mass resulting in osteoporosis (Katz & Norman, 2009; Roberts, Finch, Pullan, Bhagat, & Price, 2002).

In summary, common nutritional deficiencies associated with SUD occur early in the addiction process and continue as the illness progresses, poor consumption of a balanced diet is but one mechanism of malnutrition. There is evidence that the acute intoxicating effects and long-term craving of particular substances also impair insight and decision-making leading to poor self-care and malnutrition. There is also an association between chronic substance addiction and unemployment as well as homelessness (Johnson, Freels, Parsons, & Vangeest, 1997), which may contribute to a lack of access to nutritious food (Beharry, 2012; Rehm et al., 2009). Studies suggest that having a SUD increases the chances of being addicted to more than one substance (Aggrawal, 2001; Perkonigg, Lieb, & Wittchen, 1998), which may theoretically increase the chances of malnutrition. More studies are needed to fully understand all the deleterious effects of SUD on the body and brain to improve strategies for treatment and prevention.

8.3 Mechanisms and Clinical Manifestations of Malnutrition in ED

Disordered eating that leads to clinical or subclinical malnutrition of macronutrients (e.g., carbohydrates, proteins, fats) and/or micronutrients (e.g., vitamins, minerals, trace elements) is often present to varying degrees in people suffering from AN and can be seen in some patients with BN (Casper, Kirschner, Sandstead, Jacob, & Davis, 1980; Rock & Vasantharajan, 1995; Setnick, 2010; Winston, 2012). Typical nutritional problems that occur with BED include excessive intake of highly caloric foods that can lead to significant weight gain but will not be discussed in detail here (Raymond, Bartholome, Lee, Peterson, & Raatz, 2007; Schneider, 2003). There are a number of medical consequences of vitamin and mineral deficiencies as well as severe electrolyte disturbances that have been described in patients with AN and BN, which can occur in SUD as well, particularly AUD (see Table 8.1). The most life-threatening electrolyte abnormality is hypokalemia, which can lead to life-threatening arrhythmias and cardiac arrest in AN and BN (Hofland & Dardis, 1992; Winston, 2012). This condition develops primarily due to metabolic alkalosis from purging behaviors that include vomiting, laxative

Table 8.1 Malnutrition and associated medical symptoms/complications in people with eating and/or substance use disorders

	ED	SUD	Medical conditions	Common symptoms
Protein	AN	AUD, cocaine, methamphetamine use disorders	Kwashiorkor, marasmus, anasarca, liver failure (fatty infiltrates)	Muscle wasting, enlarged abdomen, thinning hair, loss of teeth, skin depigmentation, dermatitis, edema
Glucose	AN	AUD	Hypoglycemic coma, central nervous system problems	Shakiness, anxiety, nervousness, tachycardia, palpitations. Perspiration, pallor, coldness, clamminess, dilated pupils, paresthesia
Sodium	AN	AUD	Central nervous system problems[a]	Nausea, vomiting, headache, confusion, lethargy, fatigue, anorexia, restlessness and irritability, muscle weakness, spasms or cramps, seizures, and decreased consciousness or coma
Potassium	AN and BN	AUD	Cardiac arrest	Muscle weakness, myalgia, and muscle cramps
Magnesium	AN and BN	AUD	Cardiac arrhythmias, central nervous system problems	Weakness, muscle cramps, tremors, athetosis, jerking, nystagmus, confusion, disorientation, hallucinations, depression, epileptic fits, hypertension, tachycardia, tetany
Calcium	AN and BN	AUD, opiate abuse	Cardiac arrhythmias	Muscle tetany, bruising, paresthesias
Zinc	AN	AUD	Anorexia	Poor appetite, growth retardation, acrodermatitis enteropathica, diarrhea, taste disorders, hypogonadism, increased risk of cancer
Vitamin B1 (thiamine)	AN	AUD	Wernicke's encephalopathy/ Korsakoff's syndrome/ dementia, beriberi heart failure	Mental confusion, amnesia, poor insight, confabulation, weakness, shortness of breath
Vitamin B2 (riboflavin)	AN	AUD	Angular cheilitis	Cracked and red lips, inflammation of the lining of mouth and tongue, mouth ulcers, cracks at the corners of the mouth, and a sore throat

(continued)

Table 8.1 (continued)

	ED	SUD	Medical conditions	Common symptoms
Vitamin B3 (niacin)	AN	AUD	Pellagra, dilated cardiomyopathy, dermatitis	Diarrhea, confusion, skin rash and sun sensitivity, mouth and tongue inflammation
Vitamin B6 (pyridoxine)	AN	AUD	Sideroblastic anemia, peripheral neuropathy	Lethargy, nerve damage, seizures, skin problems, and sores in the mouth
Vitamin B9 (folic acid)	AN	AUD	Macrocytic anemia	Diarrhea, weakness or shortness of breath, peripheral neuropathy, pregnancy complications, mental confusion, forgetfulness, depression, sore or swollen tongue, peptic or mouth ulcers, headaches, heart palpitations, behavioral disorders. Can lead to homocysteinemia and increased risk of cancer
Vitamin B12 (cobalamin)	AN	AUD	Pernicious anemia, dementia	Fatigue, depression, poor memory, symptoms of mania and/or psychosis
Vitamin C (ascorbic acid)	AN	AUD	Scurvy	Brown spots on the skin (thighs and legs), spongy gums, and bleeding from all mucous membranes. Pallor, depression, suppurating wounds, tooth loss
Vitamin D	AN	AUD	Rickets (children), osteopenia/osteoporosis (adults)	Muscle aches and/or twitching (secondary to hypocalcemia), bending of the spine, bowing of the legs, proximal muscle weakness, bone fragility, and increased risk for fractures
Vitamin E (tocopherol)	AN	AUD	Spinocerebellar ataxia, myopathies, anemia	Poor nerve conduction, lethargy, muscle pain

[a]Corrected too quickly can cause pontine myelinolysis

use, and/or diuretic use. Poor intake of calories and particularly of protein has been associated with liver damage, anemia, and neutropenia in AN (Sabel, Gaudiani, Statland, & Mehler, 2013; Tomita et al., 2013).

Patients suffering with AN are also at high risk of developing osteopenia or osteoporosis, conditions whose mechanisms are multifactorial including low estrogen and poor intake of calcium and/or vitamin D (Fonseca et al., 1988; Legroux-Gerot, Vignau, Collier, & Cortet, 2005; Misra et al., 2004). One study in Japan found that 40 % of adolescent patients with AN met criteria for osteoporosis and that an

earlier onset of AN increase the risk, since adolescence is such an important time for acquisition of peak bone mass development (Maesaka & Hasegawa, 2003). Vitamin D is a fat-soluble vitamin responsible for enhancing intestinal absorption of calcium and phosphate. It can be synthesized in the skin and is also absorbed in the gastrointestinal tract primarily from dairy products but is converted into its biologically active form through the liver and kidneys, and thus the presence of liver or kidney damage in AUD and/or AN can contribute to deficiency of this vitamin (De Caprio et al., 2006). Excess vitamin D taken in the form of supplements can lead to hypercalcemia and cause symptoms of anorexia, nausea, and vomiting, frequently followed by polyuria, polydipsia, pruritus, central nervous manifestations of insomnia, depression, nervousness, weakness, and ultimately renal failure (Vieth, 1999).

The B vitamins are water soluble and are present in many foods but can easily become depleted in the context of ED. Poor nutrition leading to vitamin B1 (thiamine) deficiency can occur in AN as with AUD and can lead to life-threatening Wernicke's encephalopathy and/or Korsakoff's syndrome in some patients (Handler & Perkin, 1982; McCormick, Buchanan, Onwuameze, Pierson, & Paradiso, 2011; Peters, Parvin, Petersen, Faircloth, & Levine, 2007; Saad, Silva, Banzato, Dantas, & Garcia, 2010; Sharma, Sumich, Francis, Kiernan, & Spira, 2002). Thiamine can be stored in the body for only 9–18 days and is an essential cofactor for glucose to enter the Krebs cycle for energy utilization. The brain and heart require a constant supply of glucose and are prone to severe damage when glucose by-products accumulate in the tissues and cannot be used for energy. Wernicke's encephalopathy occurs during the acute thiamine-deficient state and can be rapidly fatal especially when glucose is given without pretreatment with thiamine (Watson, Walker, Tomkin, Finn, & Keogh, 1981). The long-term sequela for those who survive thiamine deficiency is Korsakoff's syndrome. This syndrome is characterized by confabulatory amnesia (dementia), apathy, poor insight, and oculomotor manifestations such as eye-muscle weakness and/or nystagmus. Most cases of Korsakoff's syndrome occur in the absence of any clear encephalopathic episodes (Ogershok, Rahman, Nestor, & Brick, 2002). Both conditions are underrecognized in the medical setting, especially in the setting of starvation without AUD. One study of AN patients being hospitalized for treatment found that 1/3 were deficient in thiamine (Winston, Jamieson, Madira, Gatward, & Palmer, 2000). Thiamine deficiency is known to cause damage primarily to the thalamus (Zuccoli et al., 2009) even before any neurological signs are present in animals (Langlais & Zhang, 1997). In a sample of 14 patients with AN undergoing longitudinal brain imaging, two subjects had thiamine deficiency (thiamine diphosphate <70 nmol/L) and showed magnetic resonance imaging evidence of significantly increased thalamic volume (McCormick, McCann, & Keel, 2012), suggestive of osmotic damage that occurs in acute Wernicke's encephalopathy (Jung, Chanraud, & Sullivan, 2012).

Although rare, pellagra from vitamin B3 (niacin) deficiency has been shown to occur in AN and has a characteristic desquamation rash that primarily affects the face and periphery and is worsened with sun exposure (Jagielska, Tomaszewicz-Libudzic, & Brzozowska, 2007; MacDonald & Forsyth, 2005; Prousky, 2003; Rapaport, 1985). Riboflavin and/or biotin deficiency can also occur in AN and

AUD and can cause dermatitis that is similar to that in pellagra (Capo-Chichi et al., 1999; Gehrig & Dinulos, 2010; Rock & Vasantharajan, 1995). Similarly, vitamin B6 (pyridoxine) deficiency has been shown to occur with ED or AUD and can cause seborrheic dermatitis as well as peripheral neuropathy (Majumdar, Shaw, O'Gorman, Aps, Offerman, & Thomson, 1982; Schlosser, Pirigyi, & Mirowski, 2011). Folate and vitamin B12 deficiencies have been shown to occur in AN and BN (Eedy, Curran, & Andrews, 1986; Moyano, Vilaseca, Artuch, Valls, & Lambruschini, 1998) and can lead to megaloblastic anemia (Miller et al., 2005; Misra et al., 2004) and cognitive impairment (Katzman, Christensen, Young & Zipursky, 2001; McDowell et al., 2003; Tchanturia et al., 2004). The effects of folate and thiamine deficiency that lead to anemia and cognitive impairment have been well described in elderly patients who sometimes have dietary deficiencies in these vitamins as well (Morris, Jacques, Rosenberg, & Selhub, 2007; Roberts, Martin-Clavijo, Winston, Dharmagunawardena, & Gach, 2007; Selhub, Morris, Jacques, & Rosenberg, 2009). Vitamin E and other antioxidants have also been found to be reduced in some people with AN and are thought to be due to poor nutrition and oxidative stress from starvation (Moyano et al., 1999). Zinc deficiency can occur in AN and AUD, which has been associated with a number of physiological problems, including anorexia (poor appetite), growth retardation, acquired acrodermatitis enteropathica (peripheral and perioral dermatitis), taste disorder, diarrhea, and hypogonadism (Kim et al., 2010; Roberts, Martin-Clavijo, Winston, Dharmagunawardena, & Gach, 2007; Suzuki et al., 2011). Secondary zinc deficiency can occur in the presence of low levels of vitamins A and D (Potocnik et al., 2006). Vitamin K deficiency has been described in a patient with BN and was due to poor intake of green leafy vegetables (Niiya et al., 1983). Several studies have assessed whether iron, vitamin A, or essential fatty acids are deficient in ED and SUD and these appear to be normal in most patients with these conditions (Forbes & Parsons, 2012; Langan & Farrell, 1985; Lieb et al., 2011; Sabel et al., 2013).

Macronutrient deficiency in the presence of calorie restriction in its most severe form causes marasmus and has been seen in children and adults and is still prevalent in countries with poor access to food (Román, 2013). Protein deficiency in the presence of normal calories has been shown to cause Kwashiorkor in AN characterized by severe edema, muscle wasting, enlarged abdomen, thinning hair, loss of teeth, skin depigmentation, dermatitis, and edema (Esca, Brenner, Mach, & Gschnait, 1979; Grillet & Harms, 1980). Hypoglycemia can occur in AN and AUD, especially at very low weight (average BMI of 13) and/or in the presence of liver damage (Gaudiani, Sabel, Mascolo, & Mehler, 2012), which can lead to ketoacidosis, coma, and/or death (Fulop, Ben-Ezra, & Bock, 1986; Rich, Caine, Findling, & Shaker, 1990; Yanai, Yoshida, Tomono, & Tada, 2008). Cardiomyopathy or heart failure can occur in AN and can be due to deficiencies in protein, thiamine, phosphorus, magnesium, selenium, and/or ipecac poisoning (Birmingham & Gritzner, 2007). Some of the behaviors associated with ED can cause exposure to toxins. One such example was reported by one of the authors of this chapter. A 47-year-old woman with a 30-year history of AN who for 7 years had reduced her food intake to almost exclusively canned tuna was hospitalized at

53 % of her expected body weight showing marked depression and confusion and was found to have frank mercury poisoning (Ravneet & Paradiso, 2008).

Some of the complications from nutritional deficiencies do not emerge until refeeding efforts are instituted. For example, in AN it is rare for a person to have phosphate deficiency on admission for hospitalization, but this nutrient can be rapidly depleted and lead to heart failure within a few days of refeeding (O'Connor & Nicholls, 2013). A study of 50 hospitalized AN patients revealed that 60 % of patients had low magnesium and that magnesium levels should also be assessed and replaced when necessary during the first 3 weeks of refeeding (Birmingham, Puddicombe, Hlynsky, 2004). Low magnesium levels can also occur in BN and AUD and exacerbate hypocalcemia and prolong muscle tetany, cardiac arrhythmias, and even heart failure (Abbott, Nadler, & Rude, 1994; Fonseca & Havard, 1985; Hall, Beresford, & Hall, 1989). Similarly, hyponatremia is known to occur in AN and BN (Caregaro, Di Pascoli, Favaro, Nardi, & Santonastaso, 2005) and replacing sodium too quickly can lead to anasarca or refeeding edema (Rigaud, Boulier, Tallonneau, Brindisi, & Rozen, 2010). Rapid correction of hyponatremia in ED and AUD, especially in the context of hypokalemia, can also cause central pontine myelinolysis, which can manifest as acute paralysis and dysphagia (Amann, Schäfer, Sterr, Arnold, & Grunze, 2001; Heng et al., 2007; Malhotra & Ortega, 2013; Patel, Matthews, & Bruce-Jones, 2008). Guidelines on monitoring and supplementation of macro- and micronutrients during early refeeding are discussed in Chap. 23. In addition, many of these nutritional deficiencies can have adverse effects on a developing fetus, and malnutrition in AN and BN and obesity in BED can all lead to reduced fertility (Linna et al., 2013).

8.4 Co-occurrence of SUD and ED May Increase the Incidence of Malnutrition

SUD and ED have a high rate of co-occurrence (Bulik et al., 2004; Lilenfeld et al., 1998), especially between AUD and ED with bulimic features (Baker, Mitchell, Neale, & Kendler, 2010; Duncan et al., 2006; Hudson, Hiripi, Pope, & Kessler, 2007; Munn-Chernoff et al., 2013; Slane, Burt, & Klump, 2012). A meta-analysis of 21 studies reported a median rate of 23.0 % of alcohol use among individuals with BN (Holderness, Brooks-Gunn, & Warren, 1994). Another study found an AUD rate of 21 % in people with AN, most of which developed AN prior to an AUD (Baker et al., 2010). While the majority of people with AN do not develop AUD, when it does occur, there is a significantly increased risk of death and thus it is recommended that clinicians inquire about alcohol use in this population (Bulik et al., 2004). Alternatively, in a study of 140 people with SUD without organic pathology, compared to 50 healthy adults, over 90 % weighed under the mean weight for the population and over half had experienced weight loss and reduced appetite, while nearly 20 % met criteria for severe malnutrition (Santolaria-Fernández et al., 1995). While there are no studies that have specifically assessed increased risk of malnutrition when ED and SUD co-occur, it is likely that there is

an increased risk of malnutrition, electrolyte abnormalities, and/or vitamin and mineral deficiencies due to poor intake, absorption, and utilization of essential nutrients. More research in this area is greatly needed.

Conclusions

ED and SUD can lead to unhealthy eating and malnutrition that is associated with varying degrees of medical morbidity and increased risk of death. These disorders likely share some aspects of underlying pathophysiology related to reward processing in the brain as well as common medical sequelae due to nutritional deficits. Vitamin and mineral deficiencies are most common in patients with AN, BN, and AUD but can be seen in other SUD as well. The most widely appreciated nutritional deficiency is for B vitamins, particularly thiamine in AUD, but several research studies have shown that this occurs in AN as well and can cause severe neurological sequelae and/or death. More research is needed to fully understand the neurobiological consequences of malnutrition in SUD and ED and to determine the extent of the additive effects when these conditions co-occur.

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