Role of the Registered Dietitian in Treatment of Substance Use Disorder

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Introduction

Substance dependence and mental illness affect millions of adults and adolescents in the United States each year\(^1\). Substance use disorder, which includes substance dependence, presents a major public health concern given the current state of the opioid epidemic and the ongoing risk that alcohol use presents to adults and adolescents. The opioid epidemic involves heroin, illicitly-made fentanyl, and misuse of legal opioids in the form of prescription pain relievers. Centers for Disease Control and the National Institute on Drug Abuse report that 115 people in the United States die each day as a result of opioid overdose\(^2,3\). The estimated economic burden of this epidemic is $78.5 billion per year in costs of healthcare, lost productivity, addiction treatment, and criminal justice costs\(^2\). The National Institute on Alcohol Abuse and Alcoholism report an estimated 88,000 people die each year in the United States from alcohol-related causes, making alcohol the third leading cause of preventable death behind tobacco and diet and exercise\(^4\). SUD poses additional public health concerns due to the increased risk of chronic disease for individuals with SUD including cirrhosis, cardiovascular disease, diabetes, pulmonary disease, poor wound healing, and impaired immune function\(^5\), as well as common comorbidity with other mental illnesses and eating disorders.

Substance use and substance dependence are intimately tied to nutrition: the use of substances interferes with adequate food intake in the short term and can alter digestive function and metabolism of nutrients in the long term. Despite this established link between substance use and nutrition, nutrition services are not always employed in SUD treatment and if they are, the role of the registered dietitian in the treatment of SUD is not well defined. Like other chronic conditions, substance use disorders are characterized by a genetic vulnerability and biological processes that facilitate substance use. As such, treatment of substance use disorders warrants similar interventions from a registered dietitian aimed at promoting dietary and behavior changes to best facilitate recovery and long-term management. This paper explores how the Health at Every Size approach best fits as a model of nutrition practice in the setting of SUD treatment.

Definition and prevalence

The National Survey on Drug Use and Health (NSDUH) collects data related to alcohol, tobacco, and drug use among those 12 years and older in the civilian, noninstitutionalized population of the United States therefore presenting a picture of the current state of substance use in this country. The NSDUH reports an estimated 19.7 million people aged 12 and older had a substance use disorder related to alcohol or illicit drugs in the year 2017\(^1\). This estimate includes 14.5 million people with alcohol use disorder and 7.5 million people with illicit drug use disorder. Among the illicit drug use disorders, the most common disorders were for marijuana (4.1 million people) and for prescription pain relievers (1.7 million people)\(^1\). These estimates from the survey are based on individuals who meet the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria for dependence or abuse for alcohol or illicit drugs.

According to the NSDUH, respondents were defined as having alcohol use disorder or illicit drug use disorder if they met the DSM-IV criteria for either dependence or abuse. Respondents were defined as having substance dependence if they met three or more of the seven dependence criteria\(^1\):

1. Spent a lot of time engaging in activities related to substance use;
2. Used substance in greater quantities or for a longer time than originally intended;
3. Developed tolerance;
4. Made unsuccessful attempts to cut down on use;
5. Continued use despite physical health or emotional problems associated with use;
6. Reduced or eliminated participation in other important or previously pleasurable activities because of substance use;
7. Experienced withdrawal symptoms when cutting back or stopping use;

Respondents who did not meet criteria for dependence were defined as having abuse if they reported one or more of the following:

1. Problems at work, home, or school because of substance use;
2. Regularly using the substance and then doing something physically dangerous;
3. Repeated trouble with the law because of substance use;
4. Continued use of the substance despite problems with family or friends.

More recently, the DSM-V combines substance dependence and substance abuse under a single diagnosis of substance use disorder. Substance use disorder (SUD) is diagnosed based on a pathological pattern of behaviors related to the use of the substance. The diagnostic criteria match the DSM-IV dependence and abuse criteria listed previously with a few differences. The DSM-V does not include the criterion about repeated legal problems as a result of substance use. The DSM-V replaces this criterion with presence of cravings or a strong desire or urge to use the substance. In combining substance dependence and substance abuse under a single diagnosis, SUD is diagnosed based on 11 criteria. The presence of two to three symptoms indicates a mild substance use disorder, the presence of four to five symptoms is moderate, and the presence of six or more symptoms is designated a severe substance use disorder.

Substance misuse is a preferred term over substance abuse. Substance misuse is the use of a substance in ways or in quantities that are harmful. It is preferred over substance abuse because it conveys a similar meaning without the stigmatizing overtones that are associated with “abuse.” Addiction is another common term with its own definition. Addiction is a brain disease that is characterized by “compulsive substance abuse despite harmful consequences.” Addiction aligns with severe substance use disorder. Substance use disorder encompasses multiple levels of disordered substance use, from misuse to dependence to addiction.

**Alcohol**

The 2017 NSDUH reports that 14.5 million people aged 12 and older live with alcohol use disorder. This figure includes 3.4 million young adults aged 18 to 25 (10% of this age group) and 10.6 million adults 26 and older, about 5% of this age group. These prevalence numbers are similar to the prevalence of AUD in these age groups in 2016. Additionally, the NSDUH reports that alcohol had the highest rate of recent (in the last year) substance use initiation with 4.9 million new users.

**Opioids**

Opioids are a group of chemically similar drugs that includes heroin and prescription pain relievers such as hydrocodone, oxycodone, and morphine.
Opioid misuse and opioid addiction represent a major medical and public health concern with an average of 115 Americans dying from an opioid overdose everyday³. Preliminary numbers from the Centers for Disease Control estimate that more than 70,000 people died of drug overdoses in 2017 which represents a 10% increase in overdose deaths from 2016. Opioids were involved in the majority of those deaths, more than 48,000¹⁰.

The CDC recently released preliminary numbers that indicate a decrease in the number of deaths due to drug overdose. From December 2017 to March 2018, the pace of increase in overdose deaths decreased from 10 percent to 3 percent over the previous 12 months¹⁰. Despite the beginning of a plateau in overdose deaths, there is still work to be done in the prevention and treatment of opioid abuse. After alcohol and marijuana, prescription pain killers have the next highest rate of recent (in the last year) substance misuse initiation with 2.0 million new misusers¹. This equates to roughly 5,500 people per day misusing prescription pain relievers for the first time. The NSDUH defines “misuse” as using pain relievers without a prescription of one’s own or using a higher dose or using more often than is prescribed, even if the reason is to relieve pain¹. Among those misusing prescription pain relievers, the majority of misusers (62.6%) cited their primary reason for use was pain relief. After that, the most common primary reason (for 13.2% of misusers) was to feel good or get high¹.

In contrast, the NSDUH estimates that about 220 people per day used heroin for the first time in 2017 for a total of 81,000 people aged 12 and older initiating heroin use¹. This estimate is down from 2016 when the number of estimated new heroin users for the year was 170,000 people among those aged 12 and older¹.

The 2017 NSDUH estimates that 1.7 million people aged 12 and older (0.6% of this population) had a prescription pain reliever use disorder and that 652,000 people aged 12 and older (0.2% of this population) had a heroin use disorder. For both classes of opiates, the highest number of substance use disorders are seen in those aged 26 and older (compared to categories for individuals 12-17 and 18-25 years old)¹.

What is considered a current opioid epidemic can be attributed to medical and pharmaceutical industry downplay of the addictive potential of opioids in the late 20th century and physicians subsequently over-prescribing opioids to treat chronic pain. A letter to the New England Journal of Medicine in January 1980 by two researchers explained that in their analysis of 11,882 patients treated with opioids for pain, development of addiction was rare in individuals without history of addiction¹¹. This letter became one of several publications challenging the previous paradigm that opioids are addictive. Instead of physicians avoiding opioids, opioid treatment started to become the humane alternative to surgery or not treating a patient in chronic pain.

Oxycontin was introduced in 1996. Manufacturer Purdue Pharma advertised in medical journals and created videos directed at patients to market the drug. After the video, opioid prescriptions in the United States increased by 11 million. Resources printed in 2000 by the Joint Commission, the non-profit agency that accredits hospitals, and sponsored by Purdue Pharma would further validate the thought that opioids present low risk of addiction. Purdue Pharma executives would later be charged with downplaying Oxycontin’s addictive potential¹¹.

More recently, opioid users have turned to heroin instead of prescription pain relievers because heroin is cheaper, easier to use, and widely available¹¹. Making this transition more dangerous is the
introduction of illicitly-made fentanyl. Fentanyl is a synthetic opioid that is 50 times more potent than heroin\textsuperscript{12} and is used in surgical anesthesia and pain treatment. Most of the fentanyl in the drug supply is manufactured overseas and mixed with heroin or used to make counterfeit prescription pills\textsuperscript{13}. Because of its increased potency, use of fentanyl increases risk of an overdose.

**Substance impact on nutrition**

It is well established that substance use impacts nutrient intake, absorption, and metabolism\textsuperscript{5,13,14}. Substance misuse and substance use disorders are positively correlated with malnutrition\textsuperscript{5,13}, malnutrition worsening with severity of substance use disorder and polydrug use\textsuperscript{15}. Schenker (as quoted by Jeynes and Gibson, 2017) defines malnutrition as the “disturbance of form or function arising from the deficiency of one or more nutrients”\textsuperscript{95}. Ross and colleagues differentiate between primary malnutrition and secondary malnutrition\textsuperscript{13}, both of which apply to those with alcohol use disorder and drug use disorder. Primary malnutrition is the inadequate intake of food variety and/or volume while secondary malnutrition is an interference in digestion, absorption, metabolism and usage of nutrients\textsuperscript{13}.

Malnutrition in this population can be the result of a variety of factors. Variables at play in malnutrition include poor food security, low income, and lack of cooking skills\textsuperscript{5}. On a physiological level, malnutrition could result from compromised liver function in metabolism and energy storage, decreased appetite, inhibited gastric motility, and increased excretion of nutrients secondary to poor absorption\textsuperscript{5,15}.

**Alcohol**

An estimated 14.5 million people aged 12 and older had alcohol use disorder according to the 2017 NSDUH\textsuperscript{1}. This number corresponds to 5.3% of the population aged 12 and older or about 1 in 19 people of this age group\textsuperscript{1}. de Timary and colleagues report that epidemiological studies have shown that alcohol may account for 10% of total energy consumption in alcohol consumers and for more than 50% of total energy consumption in those with alcohol dependence\textsuperscript{16}. With such a significant amount of total energy coming from alcohol instead of food, it follows that those with alcohol use disorder are at increased risk for malnutrition.

Alcohol impacts nutrition from ingestion to nutrient use. Metabolism of ethanol in the liver consumes the electron acceptor NAD\textsuperscript{+}. Consumption of NAD\textsuperscript{+} in this reaction depletes NAD\textsuperscript{+} stores for macronutrient metabolism and hinders processes like glycolysis, gluconeogenesis, and lipid metabolism. When these processes are impaired, the liver cannot efficiently create energy for the rest of the body.

In addition to impaired macronutrient metabolism, micronutrient deficiencies are a concern in alcohol use disorder. Individuals with AUD are at increased risk for micronutrient deficiencies of many of the B-complex vitamins, vitamins A, C, D, E, K, potassium, magnesium, selenium, and zinc\textsuperscript{5}. Thiamine (vitamin B1) is of particular concern in those with AUD\textsuperscript{17}. Thiamine is required for metabolism of carbohydrates, the body’s most efficient source of energy and preferred fuel source for the brain. Alcohol inhibits thiamine uptake from the gastrointestinal tract by reducing transcription factors, thereby reducing synthesis, for the two transporters that absorb thiamine in the brush border of the small intestine\textsuperscript{5}. With fewer transporters, less thiamine is absorbed. Alcohol also reduces synthesis of the enzyme thiamine pyrophosphokinase. This protein changes the thiamine found in food to thiamine pyrophosphate, the active form that serves as a coenzyme in metabolic reactions\textsuperscript{5}. Thiamine deficiency is established as the primary cause of Wernicke’s encephalopathy and Korsakoff Syndrome\textsuperscript{5} which often co-occur as
Wernicke-Korsakoff syndrome. Wernicke’s encephalopathy is the first stage of the syndrome and is characterized by confusion, involuntary and impaired eye movement, and lack of muscle coordination. Korsakoff syndrome is characterized by amnesia and hallucinations. Thiamine supplementation has been shown to relieve symptoms of both, especially Wernicke’s encephalopathy.

Magnesium is also depleted in alcohol consumption. In those with AUD, deficiency levels vary from 13% to 50%. Magnesium is a required micronutrient for many metabolic reactions and is of particular concern as it relates to thiamine metabolism. Magnesium is a cofactor in the conversion of thiamine to thiamine pyrophosphate, the metabolically active form of thiamine. If sufficient thiamine is available without adequate magnesium, carbohydrate metabolism is impaired. This micronutrient relationship should be considered in those with Wernicke’s encephalopathy and others for whom thiamine supplementation is indicated.

Niacin, another member of the B-complex of vitamins, is likely to be deficient in those with AUD. Niacin is found in protein sources such as meat products which may be lacking in the diets of those with AUD given the likelihood of overall poor intake. The body can synthesize niacin from the amino acid tryptophan provided that tryptophan is included in the diet. This conversion of tryptophan to niacin takes place in the liver and requires micronutrient cofactors thiamine, riboflavin, pyridoxine, and zinc. If liver function is compromised as a result of long-term alcohol use or if any of these cofactors are unavailable, niacin deficiency can result.

Subjects with AUD are also found to have low levels of vitamins A, C, and E. Vitamin A functions as an antioxidant in the form of carotenoids and regulates expression of genes as retinoic acid. Levels of retinoic acid are found to be low in those with AUD. The conversion of ethanol to acetic acid and the conversion of retinol to retinoic acid compete for a metabolic pathway. Both conversions require alcohol dehydrogenase to catalyze the reaction in the liver and both require NAD+ as an electron acceptor. If NAD+ supplies are depleted in the course of metabolizing consumed ethanol, there will be insufficient NAD+ to convert retinol to retinoic acid. Increased levels of ethanol to detoxify could explain the decreased amounts of retinoic acid in individuals with AUD. Low levels of antioxidant vitamins C and E and carotenoids could be the result of poor overall diet observed in AUD and poor intake of foods that contain these nutrients. Decreased levels of antioxidants impair the body’s ability to remove toxins and regulate inflammation, tipping the body’s regulatory processes toward oxidative damage. Oxidative damage is involved in the development of chronic disease like diabetes and atherosclerosis as well as aging and damage to DNA.

Chronic alcohol use is also associated with physical changes that impact nutrition status. Chronic alcohol use can cause mucosal damage in the mouth, esophagus, stomach, and intestine, as well as inappropriately increase intestinal permeability. As alcohol consumption increases, gastric acid secretion may also increase, leading to hyperchlorhydria and atrophic gastritis. These conditions impair the absorption of nutrients, particularly thiamine and vitamin B12. Consequences of a vitamin B12 deficiency include pernicious anemia and nerve damage. Lack of vitamin B12 would result in numbness and tingling in extremities, poor muscle control, and impaired memory.

Replacing nutrients with alcohol and the maldigestion, malabsorption, and poor utilization of nutrients that occurs as a result of chronic alcohol use creates a pathological cycle of malnutrition in those with alcohol use disorder.
Individuals entering treatment for alcohol use disorder are likely coming from a place of appetite dysregulation and irregular eating patterns as a result of their substance use. As mentioned previously, alcohol may account for as much as 50% of energy intake in those with AUD, leaving little room for normal eating patterns. Researchers have observed that alcohol craving and blood levels of appetite-regulating hormones are correlated, again demonstrating a link between alcohol use and nutrition.

de Timary and colleagues conducted a study evaluating energy intakes, appetite-regulating hormones, and BMI among those admitted to inpatient for alcohol withdrawal and rehabilitation. The study compared alcohol-dependent subjects to control subjects. The researchers observed that alcohol-dependent subjects show a significantly lower frequency of eating breakfast and lunch at the beginning of withdrawal compared to controls, but frequency of dinner was similar to that of controls. When measured at Time 3 (16 days from the start of withdrawal), frequency of breakfast and lunch among alcohol-dependent subjects had increased. The study also observed that among alcohol-dependent subjects, there was a positive correlation between energy intake from non-alcoholic sources and energy intake from alcoholic sources. After splitting the study group along the median alcohol intake of 12.5 kcal ethanol/kg/day, the researchers observed a difference in total energy intake above and below this marker. Below 12.5 kcal/kg/day, energy intake from nonalcoholic sources is decreased to compensate for the alcohol intake, maintaining a balance in energy intake. Above this median value, total energy intake exceeded “norms” as energy from non-alcoholic sources increased with energy from alcoholic sources. Furthermore, the study found that as alcohol intake increases (in kcal of ethanol/kg/day), BMI and weight of alcohol-dependent subjects decrease, suggesting that there is an interaction between alcohol and energy metabolism. The study measured respiratory quotient among alcohol-dependent subjects at different times throughout the withdrawal period. Early in withdrawal the RQ of AD subjects was low, indicating the body favored lipid oxidation for energy. When measured after withdrawal, RQ had increased indicating a shift away from lipid metabolism. The study observed higher basal metabolic rates among AD subjects early in withdrawal and decreased basal metabolic rates after withdrawal. Favoring lipid oxidation and higher BMR during heavy alcohol use could explain the negative correlation between alcohol intake and BMI observed in AD subjects. This association could also be attributed to impaired nutrient absorption in the gastrointestinal tract secondary to alcohol use. Results from this study are consistent with previous research that reports complete recovery of fat mass within three months of abstinence.

Compared to controls, anorexigenic and orexigenic hormones differed among alcohol dependent subjects. de Timary and colleagues observed higher levels of anorexigenic hormones leptin, insulin, and PYY and decreased levels of the orexigenic hormone ghrelin in AD subjects. The researchers, however, also observed a significant decrease in the anorexigenic hormone GLP-1 which would be expected to have a positive effect on appetite. Taken together, these hormone level differences could help explain decreases in food intake in individuals with alcohol dependence. This finding contradicts the results of the study among heavy alcohol consumers that observed higher total energy intakes among individuals consuming more than 12.5 kcal ethanol/kg/day. This suggests that there is a deeper interaction between ethanol and appetite that remains to be explored.

**Opioids**

Opioid misusers are at increased risk of malnutrition, low body weight and BMI, and essential vitamin and nutrient deficiencies. In individuals with drug use disorders, malnutrition or nutrient deficiencies
may more often result from inadequate intake in food volume and variety and dysfunctional eating patterns. Unlike alcohol, drug use does not compromise the structure of the digestive tract, but drug users do experience poor nutrient absorption as a result of diarrhea, constipation, and vomiting secondary to substance use. Micronutrients of concern in opioid misuse include thiamine, riboflavin, niacin, vitamins A, C, D, and E, magnesium, calcium, copper, and iron – micronutrients essential for energy production, antioxidant function, bone health, and oxygen transport throughout the body.

Contributing to malnutrition among opioid misusers, particularly heroin users, is a preference for cheap, sweet, convenient foods. Cravings for sweet foods are typically higher in those actively using drugs compared to methadone maintenance, but individuals on methadone maintenance, a medication-assisted treatment for opioid addiction, still report high cravings for sweet foods. While sweet convenience foods certainly have a place in a healthy diet, they don’t provide many essential micronutrients. Irregular eating patterns noted during active heroin use further exacerbates malnutrition. Drug misusers report eating only once or twice per day and report deriving little pleasure from food or eating during active heroin use. Drug misusers also report little social eating which generally decreases food intake.

Irregular eating patterns during drug use could be a manifestation of a variety of factors contributing to lack of desire to eat. Opioids inhibit gastric motility, resulting in constipation which could decrease appetite. Individuals misusing drugs might also have a history of eating disorders or other mental health conditions like depression, which decreases regular food intake and reduces appetite. Other factors with nutrition implications noted in this population include lack of access to adequate food or cooking facilities (due to homelessness or poor housing) and preoccupation with the drug which results in giving little thought to food.

Dental caries among drug misusers further contributes to poor nutrition status. In general, dental caries contribute to poor nutrition status because caries can make eating painful and if left untreated, can cause infection of the tooth and gums and tooth loss. Factors driving dental caries among opioid misusers include less frequent brushing while using the substance and preference for sweet foods while using. From one report, 66% of users reported brushing teeth less often when using heroin than when not using and 84% reported increase in sweets consumption when using. Opiate use, methadone and heroin, is independently associated with dental pathology after controlling for frequency and quality of dental care. This report does not indicate whether the study controlled for personal or professional dental care, or both. In light of that, infrequent visits to the dentist could be another factor driving poor dentition in this population. Methadone is 50% sugar and could also contribute to dental caries, although there isn’t literature to support this. The sugar content of methadone is not likely the sole cause of caries.

**Pharmacological treatment**

Substance use disorders are chronic mental illnesses with multifactorial etiologies. As such, treatment should also be multifactorial with the collaboration of multiple disciplines. Pharmacological interventions to ease withdrawal symptoms in conjunction with behavioral therapy, social support, and counseling for lifestyle changes present a combination most likely to result in treatment adherence and prevent relapse.

**Alcohol Use Disorder**
There are three drugs approved by the United States Food and Drug Administration for the treatment of alcohol use disorder: disulfiram, naltrexone, and acamprosate.

Disulfiram was approved for the treatment of alcohol dependence in 1951. The medication works by inhibiting the enzyme acetaldehyde dehydrogenase in the liver that metabolizes acetaldehyde to acetic acid\textsuperscript{20}, a downstream reaction in the metabolism of alcohol. This causes buildup of acetaldehyde which produces unpleasant side effects that characterize the “disulfiram reaction” including flushing, headache, nausea, vomiting, diaphoresis, and lightheadedness\textsuperscript{21}. Disulfiram is an alcohol-deterrent meaning that rather than reducing cravings for alcohol or reducing euphoric feelings associated with alcohol intake, disulfiram produces these immediate and potentially severe effects when alcohol is consumed which should deter the individual from consuming alcohol. Even small amounts of alcohol are enough to elicit this response when taking disulfiram. Because it is a deterrent, medication adherence is required for disulfiram to be effective in treatment. This medication should not be prescribed early in treatment when an individual is struggling to maintain sobriety. Disulfiram is a useful pharmacologic treatment for individuals whose goal is complete abstinence from alcohol rather than a reduction in amount of drinking or heavy drinking days\textsuperscript{21}.

Naltrexone is the second drug approved by the FDA for the treatment of AUD. Naltrexone is an opioid receptor antagonist. Because it blocks the opioid receptors in the brain that alcohol targets, it inhibits the euphoric effects of alcohol consumption, thereby reducing alcohol cravings and facilitating abstinence from alcohol\textsuperscript{21}. This mechanism of action also means that naltrexone is an effective pharmacological treatment for opioid dependence. As an opioid receptor antagonist, naltrexone would be contraindicated for individuals in AUD recovery who take prescription opioids for pain management. When taken orally, naltrexone can have hepatotoxic effects. In response to this, a form of the drug that can be delivered via intramuscular injection was developed and approved by the FDA in 2006\textsuperscript{21}. The injection only needs to be administered monthly and may be a viable option in those at risk of not adhering to pharmacotherapy.

Acamprosate is the third FDA-approved drug for treatment of AUD, and its mechanism of action is still unknown. Some research indicates that acamprosate reduces the dopamine release in the brain in response to alcohol consumption, thereby reducing the reward associated with alcohol intake. Other research suggests that the drug prevents relapse by reducing alcohol withdrawal symptoms like sleep and mood instabilities. In spite of its still undefined mechanism, acamprosate was approved in 2006 and meta-analyses validate its efficacy in AUD treatment\textsuperscript{20}.

**Opioid Use Disorder**

The Food and Drug Administration has approved three drugs for the treatment of opioid abuse: buprenorphine, methadone, and naltrexone. Naloxone is another FDA-approved medication and is specifically used in the setting of an opioid overdose.

Experience of opioid withdrawal in treatment often leads to relapse. In light of this, supervised opioid maintenance with medication to mitigate withdrawal symptoms often becomes the first line of defense to prevent relapse and is considered an effective therapy in opioid use disorder\textsuperscript{20}. The goals of opioid maintenance with an opioid agonist are “prevention or reduction of withdrawal symptoms, drug craving, and relapse to addictive drug use, but also the restoration of physiological function disrupted by drug abuse”\textsuperscript{20}.
Two common medications used in medication-assisted treatment (MAT) for opioid use disorder are methadone and buprenorphine. Methadone is a full agonist at the µ-opioid receptor, the same receptor in the brain that opiate drugs target, and has a longer half-life (24-36 hours) than opioids involved in abuse, such as heroin or morphine. Methadone exerts a steady state effect compared to the more volatile effects of opiates of abuse. Methadone also lessens the pain associated with withdrawal symptoms while blocking the euphoric effects of opiates like heroin or prescription pain relievers. A second opioid receptor agonist is buprenorphine. Unlike methadone, buprenorphine is a partial agonist at the µ-opioid receptor, characterized by a slow dissociation from the mu receptor. This slow dissociation contributes to the long-acting nature of the drug. Long-acting buprenorphine has less abuse potential than methadone because lower doses can be used to achieve similar effects. Buprenorphine has shown to be effective in reducing opioid cravings and severity of opiate withdrawal symptoms without having the euphoric effects of full opioids. A known limitation of the medication is the limited efficacy in cases of severe opiate dependence.

In contrast to methadone and buprenorphine, naloxone is a µ receptor antagonist. Naloxone blocks the mu receptor and displaces opioids currently bound at those receptors. As a result of blocking the mu receptor and displacing any currently-bound opiate, administration of naloxone results in immediate withdrawal symptoms. Naloxone is used foremost in the treatment of opioid overdoses. Given as an injection or as a nasal mist, it quickly crosses the blood-brain barrier and can reverse opiate-induced respiratory depression within one to two minutes. In April 2018 the Surgeon General released a statement urging more Americans to carry the potentially life-saving drug as one step toward reducing deaths from opioid overdose. Many first responders already carry the drug. The Surgeon General’s recommendation is particularly applicable to friends and family of and professionals working with those who are at risk of an opioid overdose, including individuals in outpatient treatment for opioid misuse and those who take high doses of prescription opioids for medical conditions. All states have passed laws to increase access to naloxone such as allowing physicians to write third party prescriptions for friends and family of those at risk of an overdose and permitting the distribution of naloxone in community settings. In many states it is possible to request naloxone from a pharmacy without a prescription.

**RD role in treatment**

*Nutrition services in SUD treatment setting*

In 1990 the Academy of Nutrition and Dietetics (formerly the American Dietetic Association) published a statement advocating for the use of the registered dietitian in the treatment of substance use disorders, asserting that improved nutrition status during recovery improves brain health and emotional functioning to make treatment more effective, reduces alcohol and drug cravings, and prevents relapse. Little was done in the way of incorporating the dietitian into the SUD treatment setting on a broad scale so dietitians are still underutilized in SUD treatment facilities, despite what is known about malnutrition and SUD. In treatment centers that utilize nutrition services, nutrition education may not be delivered by a registered dietitian. Instead, a counselor, social worker, or non-credentialed nutritionist may take on the role of nutrition counseling. It is also not uncommon for the only nutrition information that patients receive in treatment to be written by Alcoholics Anonymous, which recommends eating sweet foods to curb alcohol cravings. This lack of adequate nutrition information in recovery creates a vacuum in addiction treatment that registered dietitians can fill with group and
individual nutrition education and become an integral part of the treatment team. Nutrition education and improving the food environment during treatment can be powerful tools in the treatment of substance use disorders by decreasing likelihood of relapse and empowering patients with the knowledge to make positive food choices within their means. A nourished brain will be more receptive to recovery, decreasing the likelihood of relapse. Studies show that nutrition education in the treatment facility improves dietary intake compared to baseline and individualized nutrition counseling within comprehensive nutrition education programs significantly improves the three-month success rate of substance abuse treatment units. Registered dietitians should be involved in treatment at the residential, partial hospitalization, and outpatient levels of care.

One of the initial goals in nutrition care during SUD treatment is adequate calorie intake. Patients often enter treatment in a state of malnutrition, having substituted the substance for nutrition or not making nutrition a priority. The registered dietitian plays a role here in ensuring that patients entering treatment are assessed and are being served meals that meet their estimated needs to support recovery.

Beyond the initial requirement for adequate nutrition, the dietitian has an important role in nutrition education. Patient education is routine when patients are newly diagnosed or need additional support for conditions like diabetes or congestive heart failure. Given that substance use disorder is a chronic condition, individuals with SUD should be afforded the same benefits as patients with other, less stigmatized chronic conditions. As is the case for other chronic conditions, nutrition education in this setting should be tailored to the specific risk factors in the SUD population such as potential for malnutrition in substance use and disordered eating behaviors. The dietitian should consider group or individual education topics focusing on proper diets for co-occurring conditions with SUD such as irritable bowel syndrome, hepatitis C, and chronic constipation. The AND proposes additional topics for nutrition education in SUD treatment: eating for mental health to discuss nutrient deficiencies that impact mental health; anti-inflammatory foods; emotional eating; cravings and physiologic appetite control; the importance of fiber; the gut microbiome; nutrition myths; exercise in recovery; mindful eating; budgeting and shopping. Caffeine and nicotine are often abused during recovery or active substance use. Use of nicotine is a disordered behavior if it is used to suppress appetite. AND recommends education in this setting about safe use of caffeine and nicotine and strategies for reduction or cessation.

Grant and colleagues conducted a study of treatment facilities to objectively measure the association of nutrition education in treatment and SUD treatment outcomes. Their study used changes in Addiction Severity Index (ASI) scores to quantify the association. The ASI is a validated instrument that measures addiction severity based on seven problem areas related to substance misuse: alcohol, drug, medical, legal, employment, family/social, and psychiatric problems. The ASI is administered to patients at admission and can be re-administered at follow-up points throughout treatment. Changes in ASI scores provide a measurable degree of change in each of the domains as a result of treatment. Grant and colleagues found that when nutrition education services were provided in treatment, significant improvements in ASI scores from 55% to 99% were noted for the medical, psychiatric, and family/social domains. In fact, when nutrition services were not provided in treatment, scores for these same domains worsened by a range of 67% to 104%.
In another intervention study, six different treatment facilities offered weekly nutrition and cooking education to male residents in SUD treatment. Participants in all study sites reported high levels of satisfaction with nutrition and cooking education, with percentage of individuals reporting satisfaction rarely dropping below 80\%\textsuperscript{31}. Nutrition often takes a back seat for someone during active substance misuse so patients will likely become more concerned with their nutritional status after drug or alcohol use is reduced when a patient realizes they look underweight, are given a drug use-related diagnosis like hepatitis C, or have difficulty sleeping\textsuperscript{18}. This increased interest in nutritional status lends itself to an interest in nutrition and cooking education. Cooking and eating a balanced diet in SUD treatment can have a therapeutic effect of increasing self-esteem, health, and social relationships\textsuperscript{18}. Patients enjoy an overall healthier diet when they can cook for someone else or eat in a social setting\textsuperscript{18}. In spite of the noted benefits of cooking, barriers to preparing food outside of treatment still need to be validated and addressed, such as poor access to cooking equipment at home or financial constraints.

The food service in a treatment program is a strong predictor of intake behavior among participants\textsuperscript{14}. The registered dietitian could play a key role in SUD treatment services by improving and managing the food services of the treatment center. Interventions that address the food environment in the treatment center can be another avenue to facilitate behavior change and good nutrition among patients. One nutrition intervention in six SUD treatment facilities added daily fruit and vegetable options and 100% fruit juice to the facility menus, reduced the number of sugary beverages available, and reduced the amount of fried food available. All six sites saw improvement in daily fruit and vegetable intake among residents, although results were not significantly different from baseline. The four sites with “high implementation” saw significant reduction in intake of daily servings of fats, oils, and sweets from baseline as a result of food service changes\textsuperscript{31}. There is a lack of research examining this type of intervention in SUD treatment\textsuperscript{14}, but studies like this provide a foundation for future investigations and provide early signs of evidence for the utility of a registered dietitian in food service in SUD treatment.

Eating disorder-informed treatment

The role of the registered dietitian in the treatment of SUD goes beyond providing general nutrition services. Part of what makes the dietitian’s role unique in the SUD treatment setting is the responsibility to provide nutrition guidance from an eating disorder-informed perspective. In other words, the dietitian should approach nutrition in this setting from a position that does not emphasize weight loss and dieting due to the high rate of comorbidity between eating disorders and substance use disorders. Eating disorders and substance use disorders often co-occur\textsuperscript{32–35} — those with eating disorders are at an increased risk for a substance use disorder and vice versa\textsuperscript{14}. In their review of the National Comorbidity Survey Replication (NCS-R), a nationally representative survey of the US household population, Hudson and colleagues found that occurrence of the eating disorders anorexia nervosa, bulimia nervosa, or binge eating disorder was positively correlated with co-occurrence of a substance use disorder\textsuperscript{32}. From their analysis, prevalence of SUDs in those with an anorexia nervosa, bulimia nervosa, and binge eating disorder was as high as 27\%, 37\%, and 23\%, respectively\textsuperscript{12}. People with SUD are 10 times more likely to have an eating disorder than the general population\textsuperscript{36}. Eating disorder symptoms in the SUD population appear to be nuanced. Available research suggests that individuals with bulimic ED symptoms such as binging and purging (present in bulimia nervosa, binge eating disorder, and anorexia nervosa — binge/purge subtype) are more likely to abuse substances than individuals whose primary ED symptom is food restriction. Available studies reveal that a median prevalence of 20\% of people with SUD have a
history of bulimia nervosa or other purging-related disorder. By comparison, anorexia nervosa or subclinical food restriction behaviors are present in an estimated 10% of those with SUD\textsuperscript{35}.

Eating disorders and substance use disorders present as unique diagnoses but they have similar roots in their etiologies. In addition to genetic predisposition for both, individuals with EDs or SUDs have often experienced trauma that contributed to the development of their ED or SUD. Experience of childhood sexual abuse is a particularly important risk factor for the development of bulimia nervosa and SUD, regardless of whether the ED or SUD manifests first\textsuperscript{35}. After experiencing trauma, behaviors associated with EDs and SUDs become mechanisms for the individual to cope with the subsequent anxiety, shame, or depression that develops. If the two diagnoses co-occur, symptom substitution is common in treatment of both ED and SUD. Treatment of the ED often sees resurgence of the substance misuse. Likewise, people entering SUD recovery may turn to other unhealthy coping mechanisms like eating disorder behaviors to numb uncomfortable emotions that resurface with abstinence from substance use\textsuperscript{37}. Alcohol and heroin are among the substances most frequently abused by people with eating disorders or sub-clinical ED symptoms\textsuperscript{37}. In a report of 204 women admitted for inpatient treatment of SUD, presence of eating pathology predicted poorer treatment outcomes and a greater probability of relapse\textsuperscript{35}.

Coming from a state of malnutrition or appetite dysregulation, it’s likely that individuals in SUD recovery will experience changes in their bodies when they’ve stopped using the substance and begun to normalize their eating and increase calories from food. Individuals on methadone maintenance report improved appetite and meal consumption. Individuals often gain weight and are often at a higher body mass index (BMI) during methadone maintenance, possibly secondary to cravings for sweet foods that persist during methadone treatment\textsuperscript{14,15}. Those on methadone maintenance tend to choose large amounts of dairy foods\textsuperscript{15} and sweet foods\textsuperscript{19} and low quantities of fruits and vegetables.

These body changes can be distressing, especially for those with pre-existing body image concerns. For many, there is a fear of weight gain in recovery. The Academy of Nutrition and Dietetics reports that in one sample of 297 females, 70% reported concerns about weight gain in recovery and reported using inappropriate restricting or purging behaviors to counteract weight gain. Examples of behaviors from this sample included exercising, dieting, starvation, vomiting, enemas, consumption of energy drinks, and use of diet pills, cigarettes, laxatives or drugs in efforts to lose weight\textsuperscript{14}. Excessive cigarette use during SUD recovery is common because nicotine can act as an appetite-suppressant\textsuperscript{14}. These behaviors, when used to manipulate weight or suppress appetite, are disordered eating behaviors.

Commonly-reported use of eating disorder behaviors in the SUD treatment setting underscores the delicate balance the dietitian must find between reinforcing normal eating patterns and having compassion for the real fears that patients have around weight gain in recovery. This population is also particularly vulnerable to dieting messages ubiquitous in the media and in public health messaging. Patients are often coming from a state of malnourishment and unable to think critically about nutrition messaging. They may feel guilty about the way they have treated their bodies while using substances. Patients may feel pressure from themselves or from family to “get healthy” and they may not view weight gain in abstinence as meeting that goal\textsuperscript{30}. Susceptibility to dieting messages increases risk for disordered eating behaviors and the dietitian must be able to screen for and recognize these behaviors.

Weight gain in recovery can be a trigger for substance use relapse. The dietitian can address these fears by normalizing regular eating patterns and providing nutrition education from a weight-neutral
perspective. Ethical treatment of eating disorders does not include dieting messaging or micromanagement of food intake, but rather reinforcement of healthy behaviors to restore a healthy body weight for the individual. Given the strong risk for overlap of EDs and SUDs, substance use disorder should also be treated without diet and weight loss messaging.

**Reasons to not promote dieting in SUD treatment**

High risk for co-occurrence of eating disorders and disordered eating is reason enough to take an approach to nutrition in the SUD treatment setting that does not focus on weight. To further make the case for a weight-neutral perspective, research shows that attempts to manipulate weight through diet and exercise are not only futile, but also harmful. Research indicates that diets do not lead to sustainable weight loss. “Dieting” is defined in the literature as the severe restriction of caloric intake in order to lose weight. Studies show that dieting leads to weight loss in the short term, but this weight loss is not maintained. In the long-term, evidence demonstrates that dieters regain the weight they initially lost, sometimes gaining more whether or not they continue the diet or exercise regimen. In their review of the long-term outcomes of dieting, Mann and colleagues identified 14 diet studies that followed participants for at least four years after a diet. In analyzing the outcomes for all of the subjects in the 14 studies, “The average weight loss on these diets was 14 kg (30.8 lb), and by the long-term follow-up participants had gained back all but 3 of those kilograms (6.6 lb)” In eight of the diet studies from this analysis, an average of 41% of participants weighed more at follow-up than they did before the diet. The rate of participants weighing more at follow-up compared to baseline ranged from 29% to 64% among the participants in the studies being compared in this analysis. In two of those studies, more than half of participants weighed more at the long-term follow-up after the diet than before. Studies that conclude that diets lead to long-term weight loss often confound diet and exercise. Participants in studies who engage in exercise are more successful at long-term weight loss compared to those participants who exclusively restrict calories.

What’s more, evidence does not show that long-term weight loss improves health independent of other health-promoting behaviors. This is partly because research has yet to prove that long-term weight loss is possible for a significant number of people. Without that, it cannot be definitively concluded that weight loss improves health in the long term. In studies that conclude that weight loss improves biomarkers, the weight loss is accompanied by changes in behavior like improved diet or increased exercise. In a study evaluating weight loss and type 2 diabetes, participants lost weight and improved glycemic control. The study found that participants’ glycemic markers reverted to starting values six to eighteen months after the intervention even when weight loss was maintained. This result suggests that weight itself is not responsible for improved health outcomes. Behaviors are more important than weight for improving health. Biomarkers like blood pressure, blood lipid profile, and insulin sensitivity can be improved with behavior change, whether or not weight is lost. Given the evidence, managing weight should not be the focus in SUD recovery, but rather the dietitian should reinforce healthy behaviors surrounding food and exercise to facilitate recovery independent of weight.

Available research rejects the traditional paradigm that dieting leads to better health and sustainable weight loss. What’s more, available research validates that weight cycling leads to poorer health outcomes than weight itself. Weight cycling is the repeated loss and gain of weight, causing the individual’s weight to cycle up and down over time. Cycling can occur at any weight, but repeated attempts to lose weight are more common among individuals in the obese BMI category. Weight
cycling is the most common result of dieting and is associated with increased cardiovascular morbidity and mortality\textsuperscript{40}. Weight cycling is known to increase inflammation which increases risk for other chronic diseases\textsuperscript{40}. Weight cycling is also positively associated with dyslipidemia and insulin resistance\textsuperscript{41}.

Beyond weight cycling, dieting is associated with other negative health outcomes. Dieting reduces bone mass, increasing risk for osteoporosis in people within normal weight and obese BMI categories. Dieting also results in increased cortisol production and psychological stress that comes with micromanaging food intake and exercise. Elevated cortisol and psychological stress increase risk for chronic disease\textsuperscript{40}.

Placing value on weight loss in the setting of SUD treatment, or any setting, reinforces anxiety and fear around weight gain, an outcome that many individuals in SUD treatment experience. It’s also possible that valuing weight loss or thinness might reinforce substance misuse if that’s how an individual has previously maintained a lower weight. The dietitian working in SUD treatment should be prepared to respond in a productive way to the sentiment “I’d rather be thin and addicted than fat and sober”\textsuperscript{30}.

**Health at Every Size and intuitive eating as models for nutrition practice in SUD treatment**

Because nutrition services are underutilized in the SUD treatment setting\textsuperscript{27,28}, there is not a well-defined scope of nutrition services for this population. This void creates the opportunity to establish an evidence-based protocol with the greatest potential for positive health outcomes. That nutrition practice is taking a weight neutral approach to nutrition in SUD treatment, using Health at Every Size and intuitive eating as models for practice. Given the vulnerability of this population and the co-occurrence of eating disorders, taking the focus in treatment off body shape and weight and instead focusing on positive behavior change creates a foundation for the most successful treatment outcomes. Redirecting focus away from diets and weight also complies with the registered dietitian’s responsibility to provide ethical and evidenced-based nutrition information based on the research that shows the harmful and ineffective nature of diets.

Health at Every Size is a philosophy that shifts focus from weight to health. HAES teaches that regardless of current body weight or changes in body weight, an individual can engage in behaviors that improve health.

“HAES encourages body acceptance as opposed to weight loss or weight maintenance”\textsuperscript{40}. Available research refutes the conventional belief that weight loss improves health. Encouraging body acceptance is particularly important for those in SUD recovery as their bodies change in response to adequate nutrition and normal eating. Body acceptance opens the door for folks in SUD treatment to pursue more effective avenues to health, instead of attaching a healthy sobriety to a certain weight. In encouraging body acceptance, the dietitian also teaches self-compassion which proves to be a far more effective motivator for behavior change than shame\textsuperscript{40}. Like in eating disorder treatment, patients are taught to accept their body as it is right now, even if appearance differs from a desired weight or shape. In this way, patients can move from trying to manipulate their bodies to engaging greater self-care. Armed with adequate nutrition knowledge, self-acceptance and self-compassion prove to increase an individual’s ability to care for themselves and implement lasting behavior change\textsuperscript{38,40,42}.

“HAES supports reliance on internal regulatory processes, such as hunger and satiety, as opposed to encouraging cognitively-imposed dietary restriction”\textsuperscript{40}. In other words, HAES encourages intuitive eating, the practice of making eating decisions based on internal cues rather than external diet or food
rules and recognizing that all foods serve a variety of purposes (energy, satisfaction, etc.)\textsuperscript{43}. HAES and intuitive eating encourage people to increase awareness of their body’s response to food and make food decisions based on this attunement to bodily cues, paying attention to hunger, fullness, satisfaction, mood, energy levels, bowel movements, and pleasure. Intuitive eating is associated with improved nutrient intake, reduced eating disorder symptomology, and improved body image\textsuperscript{40,43}. A HAES and intuitive eating model challenges black and white thinking around food. Folks in SUD treatment might feel shame around the way they treated their bodies while using substances and might view maintaining a “perfect diet” as the way to achieve health in sobriety – a way to “make up” for time spent using\textsuperscript{30}. Knowing that a perfect diet doesn’t exist, the dietitian can use HAES and intuitive eating to reduce this rigidity, challenge conventional thinking, and make room for all foods in a balanced diet.

Taking a HAES approach to nutrition in SUD treatment allows nutrition services to be adaptable to a variety of demographics among folks seeking treatment. Patients can hear the same weight-neutral message and implement recommendations in ways within their means to improve diet, sleep adequately, and engage in enjoyable exercise. Implementing HAES and intuitive eating-based nutrition services in SUD is also the ethical approach given the high likelihood of SUD and ED co-morbidity. It is well-documented that an intuitive eating approach reduces disordered eating behaviors\textsuperscript{38,43}. Reducing disordered eating behaviors, normalizing eating habits, and encouraging self-compassion has the greatest potential for positive SUD treatment outcomes.

### Barriers to nutrition services in SUD treatment

Despite the existing evidence of the benefits of nutrition services in SUD treatment, nutrition services are still underutilized. One of the reasons for this is the idea that folks seeking treatment are only looking to address the initial crisis of detoxing from their substance. They are not seeking additional services like nutrition education or counseling or ready for multiple health behavior changes\textsuperscript{14}. A study of SUD treatment facilities in the Los Angeles area reported additional barriers. This study reported that facilities were satisfied with the levels of care that they offered and did not perceive a need for additional care, facilities had budgeting constraints, or the facility perceived a difficulty in care team coordination with nutrition services. Other facilities had never considered nutrition as part of their treatment plan\textsuperscript{17}. Nutrition services in this setting may not be covered by insurance, leaving the cost of implementation to the facility\textsuperscript{14}. If nutrition services are included in the treatment setting, there could be additional barriers to efficacy. Resident turnover can change the group dynamic which could impact group education settings. Staff buy-in to the importance of nutrition services in this setting is also critical. As staff encourages and prioritizes nutrition services, patients are more likely to participate and benefit\textsuperscript{31}.

### Conclusion

Individuals with substance use disorder are at increased risk of malnutrition. Alcohol use disorder increases risk of malnutrition due to poor nutrient absorption and utilization, as well as decreased intake of food volume and variety. Malnutrition in opioid use disorder often results from irregular eating patterns that lead to overall inadequate food intake and persistent cravings for nutrient-poor foods. Adequate nutrition facilitates recovery and prevents relapse. Despite what is known about the links between substance use and nutrition, registered dietitians are still underutilized in the treatment of SUD. When nutrition services are implemented, available research touts the benefits of nutrition interventions in this setting. I propose that dietitians regularly participate in the treatment of SUDs to
provide nutrition education and nutrition counseling to patients seeking treatment, as well as manage the food service within treatment facilities. I also emphasize the need for dietitians in this space to be educated on the critical intersection of SUDs and eating disorders and to tailor any nutrition intervention accordingly. The most effective and evidenced-based way to do this is to use Health at Every Size and intuitive eating as models for practice, ultimately emphasizing healthy behavior change and removing focus from body weight and shape. There is certainly space for dietitians on the team in SUD treatment to lay a solid nutrition foundation for a healthy recovery and treat SUD not as a moral failure on the part of the individual, but rather as a chronic disease that requires education and long-term management.
References


