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Review

The importance of nutrition in aiding recovery from substance use disorders: A review

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ABSTRACT

Background: Nutrition is a prerequisite for health; yet, there is no special nutritional assessment or guidance for drug and alcohol dependent individuals, despite the fact that their food consumption is often very limited, risking malnutrition. Further, the premise is examined that malnutrition may promote drug seeking and impede recovery from substance use disorders (SUD).

Method: A narrative review addressed the relationship between substance use disorders and nutrition, including evidence for malnutrition, as well as their impact on metabolism and appetite regulation. The implications of the biopsychology of addiction and appetite for understanding the role of nutrition in SUD were also considered.

Results: The literature overwhelmingly finds that subjects with alcohol use disorder (AUD) and drug use disorder (DUD) typically suffer from nutrient deficiencies. These nutrient deficiencies may be complicit in the alcoholic myopathy, osteopenia and osteoporosis, and mood disorders including anxiety and depression, observed in AUD and DUD. These same individuals have also been found to have altered body composition and altered hormonal metabolic regulators. Additionally, brain processes fundamental for survival are stimulated both by food, particularly sweet foods, and by substances of abuse, with evidence supporting confusion (addiction transfer) when recovering from SUD between cravings for a substance and craving for food.

Conclusion: Poor nutritional status in AUD and DUD severely impacts their physical and psychological health, which may impede their ability to resist substances of abuse and recover their health. This review contributes to a better understanding of interventions that could best support individuals with substance use disorders.

1. Introduction

Alcohol and drug dependency are not merely matters of addiction and substance misuse, but are accompanied by serious comorbidities. It is estimated that every year worldwide, 3.3 million people die from harmful alcohol consumption and that 15.3 million people use drugs in a way that is harmful (World Health Organisation, 2017). In England, there are estimated to be 1.6 million people suffering from alcohol dependence (Fenton and Newton, 2016), and approximately 1.5 million adults take illegal drugs at least monthly (Lader, 2015). In 2014–15, just over 141,000 new clients accessed drug and/or alcohol recovery services in England for a total of just under 300,000 in all services (Public Health England, 2015b). Of those, 130,000 left treatment during the same period, with approximately 52% or 68,000 recorded as having completed treatment (Public Health England, 2015b): thus, 48% of those who left treatment were still substance dependent.

Substance use disorders increase the long-term risk of serious health complications (Hossain et al., 2007; Nazrul Islam et al., 2001; Quintero-Platt et al., 2015) and are linked to increased mortality (Quintero-Platt et al., 2015). Liver disease, cirrhosis, cardiovascular disease (Quintero-Platt et al., 2015; Zhang et al., 2008), diabetes, pulmonary disease (Zhang et al., 2008), poor wound healing (Guo and Dipietro, 2010), lowered immune function (Housova et al., 2005; Quintero-Platt et al., 2015) and depression (Tolliver and Anton, 2015) have all been linked to substance use disorders, as have the spread of HIV and hepatitis through illegal drug use (Nabipour et al., 2014). Drug use, not including alcohol, costs the UK National Health Service (NHS) almost £500 million every year; the cost of drug related crime alone is estimated to be £13.9 billion per year, with every £1 spent on drug treatment estimated to save £2.50 in health and criminal justice costs (NHS National Treatment Agency for Substance Misuse, 2016). Not only does recovery save public money, but society also benefits from the recovery of these individuals who are themselves, children, parents, neighbours, employees, employers and friends: successful treatment is important.

Supporting the nutritional status of individuals with alcohol use disorder (AUD) and drug use disorder (DUD) is often neglected or only

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a very small part of the recovery support offered by outpatient services. This is the case despite the fact that substance use disorders, in many cases, can lead to malnutrition, metabolic disorders that compromise nutrition (Nabipour et al., 2014), altered body composition (Tang et al., 2010) and poor mental health (Tolliver and Anton, 2015). Thus, nutrition should be an important part of the treatment of substance use disorders; however, it is not given much consideration in treatment guidelines, despite evidence that recovery outcomes can be improved by nutrition therapy and well-balanced nutrient intake (Biery et al., 1991; Grant et al., 2004). In the extensive UK National Institute for Health and Care Excellence (NICE) guidelines for alcohol use disorders, there is no mention of nutrition as a factor that needs to be specifically assessed or addressed (NICE, 2011). There are only three places in the guidelines where nutrition might be incorporated: the brief triage assessment category (NICE, 2011; Section 1.2.2.5) which specifies that 'presence of any comorbidities or other factors' be assessed and referred, the comprehensive assessment category (Section 1.2.2.6) which instructs that 'physical health problems' be assessed, and, the very last entry in the intervention guidelines (Section 1.3.8.5) which covers Wernicke-Korsakoff Syndrome (WKS) (NICE, 2011). It suggests thiamine supplementation for those at risk of Wernicke's Encephalopathy (WE), and for those who are entering inpatient services or prison *and* are at risk of malnutrition or who are already suffering from malnutrition (NICE, 2011). In April 2015, NICE did not see a need to update the guidelines. The drug use disorders guidelines by NICE do not mention nutrition (NICE, 2012), nor do the relevant Department of Health guidelines (Department of Health, 2007); they only mention physical health. Thus, this review examines the evidence for the nutritional status of subjects with alcohol and drug use disorders, and considers the implications of malnutrition for treatment of these disorders.

2. Malnutrition in substance use disorders

Malnutrition, as will be discussed herein, has been eloquently defined as, "disturbance of form or function arising from the deficiency of one or more nutrients" (Schenker, 2003, p. 91). Diagnostic criteria for malnutrition are inexact; therefore, it can be difficult to diagnose. The standard assessment takes into account an individual's body mass index (BMI) and the possibility of unintentional weight loss of between 5 and 10% of total body weight in the last 3–6 months (British Association for Parenteral and Enteral Nutrition, 2016; BAPEN). Symptoms of low mood, fatigue, muscle weakness and increased infection and/or illness can also be indicative of malnutrition (BAPEN, 2016). One thing that makes diagnosis difficult is the link to weight; it is possible to be overweight and malnourished (Gastelurrutia et al., 2011). Furthermore, individuals' behavioural symptoms related to malnutrition may be masked by drug and alcohol misuse.

It has been estimated that, of the British population, approximately 3 million are malnourished (BAPEN, 2016). This may be a result of a diet that is deficient in key macro- and micronutrients, insufficient intake, and/or poor nutrient digestion and absorption (BAPEN, 2016). Malnutrition can also result from a lack of: money, available outlets to purchase nutrient dense food, access to cooking facilities, knowledge about cooking, confidence in cooking, motivation/desire to eat and/or education about the importance of food, (Himmelgreen et al., 1998; Neale et al., 2012; Schenker, 2003). People suffering from substance use disorders may be particularly susceptible to compromised nutritional status and body composition. Substance use disorders have been shown to decrease appetite and taste for food (Neale et al., 2012) and physically impair the body's ability to access nutrients (Egerer et al., 2005). It is also possible that subjects with AUD and DUD were already nutrient deficient prior to chronic substance intake, as Schroeder and Higgins (2016) found that poor micronutrient status increased the likelihood of substance use disorders.

3. Alcohol use disorder and nutrition

3.1. Nutritional intake and absorption

Individuals with chronic AUD are generally malnourished (Chopra and Tiwari, 2012; Clugston and Blaner, 2012; Nair et al., 2015; Ross et al., 2012). Alcohol both inhibits the absorption of many nutrients directly (Badawy, 2014) and, with chronic alcohol intake, can also severely impact the health of the entire gastrointestinal (GI) tract. Chronic alcohol consumption has been linked to widespread physical injury and dysfunction including: mucosal damage in the mouth, oesophagus, and stomach, delayed gastric emptying, increased intestinal permeability and membrane damage, bacterial overgrowth and cancer (Egerer et al., 2005). This severely affects the digestion and absorption of essential nutrients (Chopra and Tiwari, 2012; Ross et al., 2012). As a result, nutrient deficiencies are prevalent in this population (Chopra and Tiwari, 2012; Stroehle et al., 2012). Research has shown that subjects with AUD are deficient in or have inadequate intake of most nutrients, including: thiamine (Dastur et al., 1976; de la Monte and Kril, 2014; Boyd et al., 1981; Stroehle et al., 2012), riboflavin, niacin (Chopra and Tiwari, 2012; Dastur et al., 1976), B_5 (Nabipour et al., 2014), pyridoxine (Dastur et al., 1976; de la Monte and Kril, 2014; Stroehle et al., 2012), folic acid (de la Monte and Kril, 2014; Stroehle et al., 2012; Wu et al., 1975), vitamin A (Clugston et al., 2015; Ross et al., 2012), vitamin C (Boyd et al., 1981), vitamin D (Boyd et al., 1981; Quintero-Platt et al., 2015; Santolaria et al., 2000a; Wijnia et al., 2013; Wilkens Knudsen et al., 2014) vitamin E (Chopra and Tiwari, 2012; Tanner et al., 1986), vitamin K (Iber et al., 1986), magnesium (Dingwall et al., 2015; McLean and Manchip, 1999; Wilkens Knudsen et al., 2014) selenium (Tanner et al., 1986) and zinc, (de la Monte and Kril, 2014; Stroehle et al., 2012; Wilkens Knudsen et al., 2014) (Table 1). Vitamin B_{12} may also be deficient; however, circulating levels may not accurately reflect the stores available for use (Kanazawa and Herbert, 1985). AUD subjects are also likely to become lactose intolerant because of a down-regulation in lactase (Egerer et al., 2005). AD subjects also have altered eating patterns (Santolaria et al., 2000b).

3.2. Effects of alcohol on nutrients

Alcohol inhibits thiamine uptake by reducing the transcription factors for the two transporters that absorb thiamine in the brush border cells (Kiela, 2010). In addition, alcohol limits the production of thiamine pyrophosphokinase, an enzyme that converts thiamine to thiamine pyrophosphate (TPP) which is a coenzyme for metabolic functions (Kiela, 2010). Thiamine deficiency has long been associated with cognitive dysfunction (de la Monte and Kril, 2014) and established as the primary cause of Wernicke's encephalopathy (WE) (Kiela, 2010; Rees and Gowing, 2013; Ross et al., 2012; Sechi and Serra, 2007), which affects mood, coordination, and ocular movement (Sechi and Serra, 2007). Korsakoff Syndrome (KS), often paired with WE (WKS) because it develops in some of these patients, limits working memory (Sechi and Serra, 2007). Thiamine deficiency has been shown to cause neurodegeneration (Yang et al., 2011), and supplementation has been shown to improve symptoms especially in WE (Kiela, 2010; Sechi and Serra, 2007).

Magnesium is depleted by alcohol consumption (McLean and Manchip, 1999) and deficiency levels have been found in subjects with AUD but prevalence varies between 13% and approximately 50% (Dingwall et al., 2015; Wilkens Knudsen et al., 2014). This deficiency has been found to play a particular role in WE and WKS. Magnesium is a cofactor in the conversion of thiamine to thiamine pyrophosphate (Bishai and Bozzetti, 1986). Some WE or WKS patients supplemented with thiamine alone did not improve (Bishai and Bozzetti, 1986) or improvement plateaued. In some of these cases, adding supplemental magnesium lead to an improvement in cognition (Bishai and Bozzetti, 1986; Dingwall et al., 2015). Among WKS patients, magnesium levels