# Nutrition and addiction – can dietary changes assist with recovery?

**Margherita Grotzkyj-Giorgi**
*Margherita Grotzkyj-Giorgi is completing a PhD on the role of nutrition in relation to alcohol misuse in socially excluded populations, at the Centre for Health Services Studies at the University of Kent.*

mg209@kent.ac.uk

## Abstract
While many of the most widely used treatment interventions engage with the psychological, social and spiritual dimensions of addiction, some of the biological aspects can at times be neglected. It is increasingly being recognised that there is a close, exacerbating relationship between problematic substance use and poor nutrition.

## Key words
Nutrition, alcohol and drugs addiction, brain function recovery, biology

In many cases, dietary imbalances relating to amino acid, vitamins, healthy fats, good carbohydrates and proteins, can lead to a wide variety of medical problems such as cognitive impairment (memory), heart disease or diabetes. These are often accompanied by a host of psychological and behavioural issues. For many people struggling with drug dependence, learning about good nutrition is critical in the recovery process and is often instrumental in securing long-term success.

Users of different substances are confronted by different challenges. Alcohol is so calorie-dense (with seven calories per gram of alcohol it is second only to fat, with nine calories per gram) that drinkers often experience a sense of being sated. In fact, these 'empty' calories do not support the health of the body and over time produce a chronic state of malnutrition. Opiate or cocaine users are often so distracted by the cycle of getting high, crashing and looking for another high, that eating and self-care are severely neglected.

Drug and alcohol abuse can significantly damage the digestive processes of the body, and many addicts experience digestive problems such as constipation, diarrhoea, indigestion and poor appetite (Holford *et al*, 2008).The damage of the delicate inner lining of intestines caused by chronic alcohol or heroin abuse impairs the absorption of amino acids, vitamins and minerals (Holford *et al*, 2008). A healthy gut means a healthy brain too. The gut's job is to extract nutrients from digested food, such as the amino acids from proteins to make neurotransmitters. Vitamins and minerals are needed to make them work properly, so having a healthy gut is key to getting the brain working properly. Addictive substances, mainly alcohol, caffeine, painkillers and heroin, can damage or even shut down the digestive processes, preventing the brain from being nourished properly. Conversely, a well-nourished brain means fewer withdrawal symptoms during the early stages of detoxification and higher possibility of achieving long-term recovery.

In some worst cases (ie. severe addicts or when intestines are severely damaged), short-term intravenous nutritional therapy may be required in order to bypass a severely damaged gut. Generally (ie. when the addiction is not severe), a good diet with short-term nutritional supplementation should recover a poor gut and in turn revitalise the brain. Regular intake of good-quality, fresh food should provide enough macronutrients and micronutrients to help in maintaining a drug-free lifestyle.

During the initial phases of recovery, however, special emphasis should be placed on educating service users about changing dietary habits and moving from a junk food- and convenience food-based diet to a healthier diet, without falling into food deprivation. Indeed, caloric intake should be substantial but calories should be coming from fresh, good quality food like fruits, vegetables, fish, nuts and pulses rather than trans-fat-laden, highly processed convenience food like ready meals, fish and chips, cakes and sweets. The processing of food and addition of colorants and preservatives depletes food of essential micronutrients and in the case of fats, transforms natural, beneficial fatty acids into 'synthetic' molecules (trans-fats) that are not recognised by enzymes and may end up stored in fat cells or, worse, they may interfere with biochemical processes. On the other hand, fresh food, or food properly prepared and cooked, retains macro- and micronutrients, fibres and water that are essential for correct functioning of the body and mind.

The education process of choosing healthy and nutritious food should be carried out within the context of psychological and social re-education (Battaglia, 2008) because learning to eat properly is part of learning to care for oneself. Good nutrition, relaxation, and exercise all play an important role in successful detoxification from alcohol and drugs abuse. Learning to make healthy food choices is important to achieve a healthy lifestyle. Because they have neglected their diet for long time, addicts experience gastrointestinal disorders such as diarrhoea, constipation and an inability to digest foods properly, along with a poor appetite. As a result, they have a special need for foods that are high in nutrients to rebuild damaged tissues and organs and to regain appropriate functioning of the various systems including the nervous and gastrointestinal systems (Battaglia, 2008).

Carefully selected nutrition with the right types of high protein and high carbohydrate foods can make a big difference in coping with cravings during the early stage of recovery (Bonner *et al*, 2004). There are also important implications for mood, as deficiency of nutrients like amino acids, folic acid and the other B-complex vitamins also have a serious impact. Sugar and caffeine can generate mood swings, so intake of both should be reduced during the early stages of recovery (Holford *et al*, 2008).

## Tyrosine and tryptophan

Alcohol and drug use prevents the body from properly processing two important amino acids, tyrosine and tryptophan. They are responsible for the production of the neurotransmitters adrenalin, dopamine and serotonin. These compounds are essential for emotional stability, mental clarity and a general state of well-being (Bandaret & Lieberman, 1989). Decreased levels of these neurotransmitters negatively affect mood and behaviour. In fact, drug craving and other abstinence symptoms are the result of malfunctions of reward centres of the brain involving the neurotransmitters and the enzymes that control them. Research indicates that it is possible to reduce stress, depression and anxiety, as increase glucose and neurotransmitters receptor sensitivity, and to restore serotonin, dopamine, enkephalins, taurine and gamma-aminobutyric acid (GABA) with amino acid supplementation, which will give the client a generally improved feeling of well-being (Brown *et al*, 1990; Bonner *et al*, 2004).

Phenylalanine is a precursor of tyrosine that is then used to produce dopamine and noradrenaline. Supplementation with phenylalanine has been shown to elevate mood, increase confidence, motivation and energy levels, and indirectly decrease drug cravings (Ehrenpreis, 1983). Tyrosine supplementation has been indicated to increase energy and emotional/mental alertness (Bandaret *et al*, 1989).

Tryptophan is integral to the production of serotonin, which has a calming effect and is important for proper sleep. It is found in foods such as bananas, milk and sunflower seeds, as well as turkey meat. Increased intake of tryptophan has been shown to stabilise mood, improve sleep and reduce frequency and severity of alcohol withdrawal symptoms (Sahley & Birkner, 2001; Bonner *et al*, 2004). Additionally, a small pilot study reported tryptophan supplementation to ameliorate cognitive functions, particularly visual memory, in abstinent chronic alcohol abusers (Grotzkyj-Giorgi *et al,* 2009). Tryptophan is metabolised along three metabolic pathways. Serotonin and melatonin pathways are physiologically relevant because

they produce an important neurotransmitter (serotonin) and sleep hormone/antioxidant (melatonin). However, serotonin and melatonin pathways of tryptophan metabolism account only for ~10% of tryptophan degradation (Badawy, 2002). In fact, more than 90% of dietary tryptophan is metabolised along the kynurenine-nicotinic acid pathway (Badawy, 2002). The kynurenine-nicotinic acid pathway produces a number of physiologically active metabolites, collectively known as kynurenines. Kynurenines are neuroactive molecules, with some of them causing damage to the brain (kynurenine, quinolinic acid) and some others protecting neurons (kynurenic acid). Kynurenines act on n-methyl-D-aspartate (NMDA) and nicotinic acid (NA) receptors. NMDA receptors are particularly important because it is involved in the process of long-term potentiation and depression, two physiological mechanisms involved in modulating learning behaviour and memory formation (Sweatt, 2003). Several animal studies have indicated that chronic and acute alcohol use interferes with tryptophan-serotonin metabolism (Badawy & Evans, 1973). Acute and chronic alcohol abuse increases cerebral serotonin concentration (LeMarquand *et al*, 1994a; 1994b). During alcohol withdrawal, cerebral serotonin concentrations drop dramatically (Badawy *et al*, 1980), thus increasing possibility of alcohol withdrawal symptoms. Recent research indicates that alcohol withdrawal symptoms may be caused by decreased serotonin levels but also by kynurenines. Kynurenines concentration during alcohol withdrawal increases, particularly those with neurotoxic activities (Morgan, 1991).

Tryptophan supplementation during early stages of alcohol detoxification may reduce the number and intensity of withdrawal symptoms and possibly improve cognitive functions of abstinent chronic alcohol abusers (Grotzkyj-Giorgi *et al*, 2009). A pilot study reported tryptophan supplementation to reduce alcohol withdrawal symptoms by acting on kynurenine pathway of tryptophan metabolism (increase of neuroprotective kynurenine) and to ameliorate cognitive functions, particularly visual memory, in 60 abstinent chronic alcohol abusers enrolled in a detoxification programme (Grotzkyj-Giorgi *et al*, 2009). Concentrations of the neuroprotective kynurenine kynurenic acid increased after tryptophan supplementation. This study also demonstrated that adding selected micronutrients (B-vitamins, antioxidant vitamins and minerals) to tryptophan supplementation improved cognitive outcomes and increased the level of neuroprotectant kynurenic acid (Grotzkyj-Giorgi *et al*, 2009).

In summary, amino acid therapy together with multivitamin and mineral supplementation should be considered during the early stages of alcohol and drugs recovery and possibly continued by following a healthy diet.

No drug currently in wide use – medical or recreational – addresses the root cause of neurotransmitter imbalance. Medical drugs merely stimulate temporary excessive release of pre-existing neurotransmitter stores or block the action of adrenalin, for example. They do not increase the production of neurotransmitters. How can they, without supplying the building materials – the amino acids? Optimising the intake of the relevant amino acids, vitamins, minerals and essential fatty acids is the long-term way out of addiction.

However important a correct amino acid and multivitamin supplementation might be, it should be remembered that nutritional supplementation is not an instant cure-all. This therapy is not a substitute for good nutrition or other aspects of healthy living (exercise, relaxation etc). That is why it is called supplementation and not substitution. Indeed, increased amino acid intake should be included into a wider nutritional strategy including the right intake of micronutrients (vitamins and minerals) and macronutritients (low glycaemic index carbohydrates, good quality proteins and fatty acids) and water.

Good quality proteins can be sourced from fish, and oily fish has an added bonus: it provides polyunsaturated fatty acids (PUFAs or omega fats). PUFAs are essential fatty acids (they must come from the diet). Omega fats (omega-3 and omega-6 fatty acids) are building blocks of cerebral membranes together with phospholids and cholesterol. Their presence is essential to maintain membrane fluidity. Membrane fluidity, in turn, guarantees a correct signal transduction in the synapses. Most addictive substances strip the brain of essential fats. Research has shown that relapses among cocaine- and alcohol-dependent people over the course of two years was significantly lowered with an increased intake of omega-6 and omega-3 foods (Buydens-Branchey *et al*, 2003a; 2003b). Equal quantities of omega-6 and omega-3 essential fats should be introduced with the diet as higher intake of omega-6 fats has been linked to a host of diseases,

among them chronic abstinence symptoms (also linked to omega-3 deficiency) (Holford *et al*, 2008).

Among micronutrients, B-vitamins and antioxidant vitamins (A, E and C) are particularly important because they play crucial roles in brain physiology. The latter protect the brain from the attacks of free radicals. These are a natural byproduct of the endogenous metabolism for which the body is protected by endogenous and exogenous mechanisms. Endogenous antioxidant system includes glutathione and other enzymatic systems. Exogenous, diet-derived antioxidants are the vitamins A, E and C. Unfortunately, brain cells require more diet-derived antioxidants than other organs because they are low in antioxidants defences (Zaidi *et al*, 2004) so high intake of antioxidants is amenable to protecting our brain from free radicals attack.

However, both antioxidants systems require substrate molecules derived from food (glutathione is synthesised from three amino acids). As a part of the intracellular, diet-derived antioxidant network, vitamin C acts in concert with other low-molecular-weight compounds, including glutathione and vitamin E, as well as antioxidant enzymes (Cohen, 1994). To date, an increasing body of evidence reports vitamin C as playing an important role in the antioxidant network in the brain (Spector & Lorenzo, 1973; Grunewald, 1993). Particularly, it has been suggested that vitamin C plays an essential role in protecting extracellular compartment from free radicals attack (Rice, 2000).

Alongside the neuroprotection exerted by endogenous antioxidant enzymes, micronutrients (vitamins and minerals) are particularly important to protect neurobiological structures associated with reward pathways and cognitive functions. Particularly, vitamin B1 (thiamine) plays a central role in preventing the development of Wernicke-Korsakoff psychosis, a neurodegenerative disorder affecting mainly alcoholics (Cook *et al*, 1998; Thomson *et al*, 2002; Bonner *et al*, 2004). People with Korsakoff syndrome suffer from severe memory impairments. Memory dysfunction is a very debilitating condition; fortunately, while Korsakoff syndrome is a virtually irreversible condition, the prodromal stage of it, Wernicke encephalopathy, is a reversible condition caused by a marked deficiency of the water-soluble vitamin thiamine (vitamin B1). When blood concentration of thiamine is brought back to physiological levels with a rapid intravenous infusion of high-concentration thiamine, symptoms quickly subside and permanent brain impairment is avoided (Bonner, 2006).

Generally, the vitamin B complex is essential for the overall cerebral cognitive performance (Bonner *et al*, 2004) as a lack of pyridoxine (vitamin B6) and riboflavin (vitamin B2) overloads the gamma-amino butyric acid (GABA) shunt, thus resulting in an excess of glutamate production and neuronal death caused by a glutamate overexcitement (excitotoxicity).

Thiamine, together with the other vitamins B, vitamins A, C, E and zinc, form a complex network of exogenous (diet-derived) antioxidant protection, which has been demonstrated to be essential in preventing age-related and alcohol-caused neurodegeneration, by acting in close collaboration with the endogenous antioxidant protection systems, such as the glutathione system (Casadesus *et al*, 2002; Esposito *et al*, 2002).

In summary, whatever the addiction, nutrition should be considered a powerful ally in the process of recovery, particularly during the initial stages of detoxification. A varied diet rich in good carbohydrates, good quality proteins (lean meat, fish and vegetable proteins), fresh fruit and vegetables, essential fats (oily fish, nuts) and plenty of water should be considered and utilised as a tool to help the recovery process.

Food provides the vast majority of metabolic co-factors essential for the correct functioning and recovery of brain structures, an example of which can be the case of Wernicke-Korsakoff encephalopathy. This is just one example of how (mal)nutrition can play a big role in the development and perpetuation of brain and cognitive dysfunctions often observed in addicts, and how simple changes in dietary habits (or, in more serious cases, nutritional supplementation) may produce a big difference in mental well-being and in recovery of brain functions during alcohol and drugs recovery.

## References

Badawy AA-B (2002) Tryptophan metabolism in alcoholism. *Nutrition Research Reviews* **15** (1) 123– 152.

Badawy A & Evans M (1973) Tryptophan pyrrolase in ethanol administration and withdrawal. *Advances in Experimental Medicine and Biology* **35** 105–123.

Badawy A, Punjani NF, Evans CM & Evans M (1980) Inhibition of rat brain tryptophan metabolism by ethanol withdrawal and possible involvement of the enhanced liver tryptophan pyrrolase activity. *Biochemical Journal* **192** 449–455.

Banderet LE & Lieberman HR (1989) Treatment with tyrosine, a neurotransmitter precursor, reduces enviromental stress in humans. *US Army Reseach Institute of Environmental Medicine Brain Research Bulletin* **22** (4) 759–762.

Battaglia E (2008) Proper nutrition for recovery. *Drug and Alcohol Addiction Recovery*. Available at: www.drugalcoholaddictionrecovery.com?p=33 (accessed May 2009).

Bonner AB (2006) *Social Exclusion and the Way Out*. London: John Wiley and Sons.

Bonner AB, Thomson AD & Cook CCH (2004) *Alcohol, Nutrition, and Recovery of Brain Function*. Oxford: CRC Press LLC.

Brown RJ, Blum K & Trachtenberg MC (1990) Neurodynamics of relapse prevention: a neuronutrient approach to outpatient DUI offenders. *Journal of Psychoactive Drugs* **22** (2) 173–187.

Buydens-Branchey L, Branchey M, McMakin DL & Hibbeln JR (2003a) Polyunsaturated fatty acid status and relapse vulnerability in cocaine addicts. *Psychiatry Research* **120** (1) 29–35.

Buydens-Branchey L, Branchey M, McMakin DL & Hibbeln JR (2003b) Polyunsaturated fatty acid status and aggression in cocaine addicts. *Drug and Alcohol Dependence* **71** (3) 319-323.

Casadesus G, Shukitt-Hale B & Joseph JA (2002) Qualitative versus quantitative caloric intake: are they equivalent paths to successful aging? *Neurobiology of Aging* **23** (3) 747–769.

Cohen G (1994) Enzymatic/nonenzymatic sources of oxyradicals and regulation of antioxidant defenses. *Annals of New York Academy of Science* **738** 8–14.

Cook CC, Hallwood PM & Thomson AD (1998) Vitamin deficiency and neuropsychiatric syndromes in alcohol misuse. *Alcohol and Alcoholism* **33** (4) 317–336.

Ehrenpreis S (1983) *Degradation of endogenous opioids: its relevance in human pathology and therapy*. New York: Raven Press.

Esposito E, Rotilio D, Di Matteo V, Di Giulio C, Cacchio M & Algeri S (2002) A review of specific dietary antioxidants and the effects on biochemical mechanisms related to neurodegenerative processes. *Neurobiology of Aging* **23** (5) 719–735.

Grotzkyj-Giorgi M, Martin C & Bonner AB (2009) *Tryptophan supplementation improves cognitive functions in chronic alcohol misusers enrolled in a detoxification programme*. Available from the authors.

Grunewald RA (1993) Ascorbic acid in the brain. *Brain Research Reviews* **18** (1) 69–80.

Holford P, Miller D & Braly J (2008) *How to quit without feeling s**t*. London: Piatkus Books.

LeMarquand D, Pihl RO & Benkelfat C (1994a) Serotonin and alcohol intake, abuse, and dependence: findings of animal studies. *Biological Psychiatry* **36** (6) 395–421.

LeMarquand D, Pihl RO & Benkelfat C (1994b) Serotonin and alcohol intake, abuse, and dependence: clinical evidence. *Biological Psychiatry* **36** (5) 326–337.

Morgan PF (1991) Is quinolinic acid an endogenous excitotoxin in alcohol withdrawal? *Medical Hypotheses* **36** (2) 118–121.

Rice ME (2000) Ascorbate regulation and its neuroprotective role in the rat brain. *Trends in Neuroscience* **23** (5) 209–216.

Sahley BJ & Birkner KM (2001) *Heal with amino acids and nutrients*. San Antonio: Pain and Stress Publications.

Spector R & Lorenzo AV (1973) Ascorbic acid homeostasis in the central nervous system. *American Journal of Physiology* **225** 757–763.

Sweatt JD (2003) *Mechanisms of memory*. San Diego, CA: Elsevier.

Thomson A, Cook CCH, Touquet R & Henry JA (2002) The Royal College of Physicians report on alcohol: guidelines for managing Wernicke's encephalopathy in the accident and emergency department. *Alcohol and Alcoholism* **37** (6) 513–521.

Zaidi & Banu N (2004) Antioxidant potential of vitamins A, E and C in modulating oxidative stress in rat brain. *Clinica Chimica Acta* **340** 229–233.