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Mini Review

Possible Benefits of Coconut Oil in Multiple Sclerosis

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Abstract

Multiple sclerosis (MS) is an immune-mediated neurodegenerative disease that results from progressive demyelination of axons. Currently the disease has no pharmacological cure. The immune-mediated neurogenic derangements result in inflammation resulting in neuronal death. This paper seeks to make use of findings from animal based work to demonstrate that to attenuate this neuronal death, an energetic alternative for neurons are the ketone bodies. In this line, the hepatic metabolism of coconut oil results in the production of a large amount of ketone bodies that are obtained from the hepatic metabolism of medium chain triglycerides, in which coconut oil is very rich. These ketone bodies are neuroprotective and have anti-inflammatory activity, being able to be an adequate source of energy of damaged neurons. Therefore, ketone bodies are able to impact in the progression of MS. In conclusion, it can be hypothesized that the oral administration of extra virgin coconut oil attenuates the progression of MS thus providing an alternative therapy for the disease.

INTRODUCTION

Multiple sclerosis (MS) is a neurodegenerative disease of increasing prevalence with no medical cure thus far. This pathology shows damage in the myelin sheath that covers the neurons. Manifestations of the disease include motor (such as gait instability, tingling, fatigue and sphincter disorders), cognitive (speed of information processing and attention, as well as memory disturbances and executive functions) and emotional problems (such as depression, anxiety or fatigue). To explain the neuronal alteration, three pathogenic mechanisms have been proposed. These mechanisms include excessive accumulation of intra axonal Ca^{2+} [1,2], demyelination of axons that evolves into a degenerative process due to the lack of trophic support provided by myelin or myelin-forming cells [3,4] and inflammatory process mediated by alterations in the immune system [5-7]. All these processes end up altering the activity at the level of mitochondrial energy utilization of the axons, producing a decrease in the concentration of ATP, which will have drastic effect on axonal damage. Based on the last mechanism, a therapeutic alternative to the drugs currently administered is a nutritional intervention that improves the energy balance of neurons by reversing mitochondrial activity. Therefore, an alternative to glucose has been linked with an improvement in energy efficiency and less oxidative stress. Moreover, in other neurodegenerative diseases, the contribution of ketone bodies has improved mitochondrial activity at the energy level. Thus, improving this energy balance of neurons would be possible to recover the reversible axonal damage, generating a neuroprotection. The relationship between anti-inflammatory effect and antioxidant is based on the increase in neuronal survival and axonal growth observed in animal model with the disease (experimental autoimmune encephalomyelitis (EAE)). This survival depends physiologically directly on neuroprotection. Likewise, the level of inflammation and oxidation would also be decreased since they are associated and possibly related [8].

COCONUT OIL

Unlike palm oil, coconut oil has the largest number of medium chain triglycerides (MCTs). While palm oil is extracted from the mesocarp of the coconut fruit, coconut oil is extracted from the white pulp (endosperm) of this fruit [9]. According to the way of extraction, different types of coconut oil can be found. Virgin coconut oil can be considered the most the most important one. This oil is obtained by cold pressure, which is better since the amount of long chain triglycerides (LCTs) is considerably reduced, less chemical is used and its lipidic profile changes increasing the percentage of MCTs. Due to the percentage of MCTs does not vary, it maintains its properties. So that the contribution of ketone bodies once these MCTs are metabolized in the liver remains constant throughout the duration of the intervention in these patients. Regarding to its appearance, coconut oil is found in liquid form from a temperature of 24°C. Below that temperature, the oil tends to solidify with greater intensity at a lower temperature, acquiring an off-white colour.

A very interesting aspect to note is that the properties of coconut oil are not altered when changing from solid to liquid form and vice versa. Therefore, heating coconut oil solidified to get it liquid, in order to consume it, keeps intact all its properties [10].

From the ingestion of coconut oil, when an individual has a negative energy balance, ketone bodies are obtained through β-oxidation in the liver [11]. MCTs, a carbon chain length between 6 and 12 carbons, such as caproic acid (6 carbons), caprylic acid (8 carbons), capric acid (10 carbons) and lauric acid (12 carbons), are the most important source of these ketone bodies. Coconut oil is one of the foods that most MCTs contain in its composition. Therefore, an energetic alternative for neurons, with a great performance in obtaining ATP, could be ketone bodies.

DISCUSSION

The ability to recover the correct mitochondrial activity and the energy production problems of the ketone bodies are evident. These ketone bodies can regulate mitochondrial activity and cell survival through the responses mediated by an increase of sirtuins [12]. The sirtuins allow an epigenetic regulation based on functional level changes related to the genome. This not implies changes in the nucleotide sequence. Among these changes, histone modification stands out. The ketone bodies are inhibitors of histone deacetylase, which causes changes in the folding of histones and an increase in the synthesis of antioxidant enzymes. This antioxidant activity improves mitochondrial functioning [13]. As a consequence of this improvement in mitochondrial activity, a neuroprotective effect is achieved. This neuroprotective effects of ketogenic diets have been demonstrated in several neurological disorders, including among others, epilepsy [14,15] in rodent models of Parkinson's disease [16], in pain and inflammation [17] and in juvenile traumatic brain injury [18,19].

Moreover, the ketone β-hydroxybutyrate, not only serves as an intermediary of energy metabolism but also regulates cellular function by activating G protein-coupled receptors HCA1/GPR81, HCA2/GPR109A and HCA3/GPR109B that are essential in the maintenance of homeostasis in changing metabolic and dietary conditions via their control of metabolic, immune and other bodily functions [20]. The work based on models of transgenic mice and synthetic HCA receptor ligands has shown that members of this family of receptors can serve as targets for the prevention and therapy of diseases, such as metabolic and inflammatory disorders [20]. As consequence, the activation of HCA2 induces a neuroprotective phenotype of monocytes and/or macrophages that depends on the production of prostaglandin D2 (PGD2), through the action of cyclooxygenase by COX1 and PGD2 hematopoietic synthetase. The activation of HCA2 through these Ly-6C monocytes and/or macrophages can be achieved through diet or pharmacological treatment, getting the wanted neuroprotection [21].

The anti-inflammatory effects must also be added to this neuroprotective capacity. Regarding this issue, recent studies show that HCA2 mediates profound anti-inflammatory effects in a variety of tissues. This can be observed by means of neuroimaging techniques or through the quantification of inflammatory markers in blood [22]. Therefore, HCA2 may be an important therapeutic target for the treatment of inflammation

Therefore, it is important to emphasize that ketone bodies represent an important alternative source of energy [23,24]. This is especially due to the fact that ketone bodies have a very rapid impact on these cells, since the absorption of the medium chain fatty acids (MCFAs), contained within MCTs, is faster and more efficient than other fatty acids, such as those with long chain. Therefore, MCFAs also stimulate the secretion of cholecystokinin, bile phospholipids and cholesterol, although less than large chain fatty acids (LCFAs). Cholecystokinin is necessary for the bile to leave the gallbladder on its way to the small intestine. This means that MCFAs can be absorbed in the same way in situations in which there is deficiency of bile salts. This also favors its absorption since it is not required to synthesize these bile salts in the liver. They do not need bile salts as much for the emulsion unlike LCFAs, for which bile is fundamental for full absorption [25].

The exact same thing would happen with the lack of pancreatic lipase in the duodenum (also derived from the lack of cholecystokinin). MCTs that contain caprylic acid (8C) and capric acid (10C) are hydrolyzed by gastric lipase, lingual lipase, and intestinal lipase [26]. These MCTs can be quickly absorbed without pancreatic lipase or the need of emulsifying with bile. In addition, these MCTs are not re-esterified in the enterocyte, which causes them not to circulate through the lymphatic system, but through the portal vein [27]. MCFAs are absorbed and transported by the portal vein to the liver instead of being part of the chylomicrons that reach the blood flow via the lymphatic system. Due the blood flow velocity of the portal vein is much greater than that of the lymph, the MCFAs are rapidly transported to the liver in blood following their absorption. Once in the liver, MCFAs are oxidized to ketone bodies much more efficiently than in the hepatic metabolism of long chain triglycerides.

CONCLUSION

Ketone bodies, obtained mainly from extra virgin coconut oil, have numerous benefits linked to MS pathogenic mechanisms. The most notable benefit is neuroprotective and anti-inflammatory activity. To this is added its easy obtaining after hepatic oxidation, due to a rapid intestinal absorption and arrival to the liver via the portal. All of this makes extra virgin coconut oil a nutritional alternative able to improve the altered energetic activity of neurons in patients with MS.

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