Review

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Alcohol: Effects on Nutritional Status, Lipid Profile and Blood Pressure

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Abstract

Alcohol consumption can be the cause of several diseases. Depending on the frequency and quantity, the consumption of alcohol may increase the risk of malnutrition, weight-gain, obesity and cardiovascular diseases. The aim of this study was to review the influence of alcohol intake on nutritional status, lipid profile and blood pressure. The review shows that the additional consumption of ethanol calories will favor weight gain and metabolic changes and may engrave the risk of cardiovascular diseases, altering the lipid profile and promoting hypertension. Recommending a moderate use of alcohol with the purpose of taking advantage of its health benefits must not be done.

Keywords: Nutritional status; Obesity; Ethanol; Cardiovascular disease

Introduction

Alcohol consumption can be the cause of several diseases, and it is, well known, the high burden of its consumption over mortality around the world [1]. Alcohol is the only psychoactive drug that provides energy (7.1 kcal/g). However, alcohol intake can increase the risk of weight-gain and the development of obesity or malnutrition. Additionally, the frequency and quantity in which alcoholic beverages are consumed are important factors that should be included in the evaluation of the risk for cardiovascular diseases.

Nutritional effects of alcohol are entangled with cardiovascular toxicity and there are many aspects to review on the subject. Depending on the age of the subject, its previous nutritional state, and dose of alcohol ingestion, the consumption of alcohol can lead to different and opposite effects. For these reason, the aim of this study was to review the influence of alcohol intake on nutritional status and the consequent effects on the lipid profile and hypertension.

Alcohol and Malnutrition

Alcohol is the only psychoactive drug that provides energy (7.1 kcal/g). This drug is an intermediate source of energy when compared to proteins and carbohydrates, which provide 4.0 kcal/g, and to the lipids, which provide 9.0 kcal/g [2]. However, its calories are considered "empty," because alcohol ingestion does not provide vitamins and minerals [3] and its use may cause alterations to the nutritional state [4].

Due to the high energy value of alcohol, it is common to assume that, when consumed in excess, alcohol increases the risk of weight-gain and the development of obesity [5]. However, despite its high calorie density, alcohol consumers do not appear to gain weight when compared to non-consumers [6].

When the alcohol caloric ingestion represents 50% or more of the total calorie ingestion, the body system may inefficiently utilize the energy provided by the ethanol ingestion due to the activation of the Microsomal Ethanol Oxidizing System (MEOS) [7]. At the cost of energy waste, the MEOS is the main liver pathway for ethanol oxidation [8]. Its induction is reversible after alcohol abstinence [9]. During the observational study of 181 hospitalized males that consumed more than 80 g/day of ethanol, Santolaria et al [10] observed that 63.1% suffered from anorexia, 58.7% presented weight loss, and 17.8% had a BMI below 20 kg/m^2. After a period of three months of alcohol abstinence, the use of caloric energy in alcoholics was normalized, just as the increase of the Body Mass Index (BMI) and the Waist Hip

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Ratio (WHP) [11]. The authors suggested the improvement in the nutritional state during abstinence may have occurred due to a less active MEOS, decreasing the waste of caloric energy.

Alcoholic patients presented metabolic and nutritional disorders due to alcohol consumption, and showed a high use of energy at a resting state [12], preferential use of lipids as energy source [13], and a 19% reduction of the adipose tissue when compared to a healthy control group [14]. Cohort studies have shown that women who consume alcohol at a small or moderate rate (up to 30 g/day) have a lower risk to gain weight and become obese than those who abstain from it [15, 16], while other studies have reached the same conclusion regardless of the subjects' gender [17, 18].

Martin-Gonzalez et al [19] evaluated the prognostic value of the long-term alterations to the adipose and muscular tissues of alcoholics and the effects of abstinence on these changes. The results demonstrate that a loss of lean body mass during the first six months after the first evaluation is associated to a worse prognosis, whether or not patients were abstinent during this same period. However, non-abstinence was greater associated to the loss of lean body mass.

The energy derived from the intake of alcoholic beverages may be added to the diet of those with a moderate consumption of alcohol or may become a substitute to other foods to chronic consumers [18] possibly decreasing the gain of body mass, while increasing alcohol's toxicity [2]. Therefore, the body's use of ethanol calories may be associated with the dosage of alcohol. The consumption of alcohol dosages below 25-35% of the daily calories may be utilized as an energy source; however, at higher consumption, the utilization may not be complete [20].

In humans, caloric malnutrition is a close reality to millions of people dying of hunger around the world and, due to low prices and high availability, ethanol is ingested in place of food. Animal models were created in order to clarify what happens when a malnourished organism is exposed to ethanol. However, results were not what one would have expected. In fact, in low doses, alcohol consumption represents an important source of calories, mitigating the effects of malnutrition. Animal studies show that the consumption of low to moderate doses of alcohol (up to 20% of total caloric intake) causes weight gain in malnourished rats, which suggests an effective utilization of alcohol energetic content. Eutrophic rats, however, lose weight when the energetic content of alcohol represents 10% or more of the total calorie intake [21, 22].

Could Alcohol be a Risk Factor for Obesity?

The nutritional state of an individual may influence the utilization of the energy derived from alcoholic beverages. It is possible that, in humans, lean individuals have a more inefficient utilization of ethanol calories, and that in obese individuals the calories contribute to an increase of body mass [23].

Although prospective studies have defended a positive [24, 25] or negative [26, 27] correlations between alcohol consumption and obesity, a 10-year long prospective study showed that alcohol consumers had a more stable weight than non-consumers, indicating that alcohol consumption is not a risk factor for obesity [28]. Romeo et al [29] verified that a chronic and moderate monthly consumption of beer did not modify the weight or the body composition of adult individuals.

The impact of alcohol drinks over body weight continues to be a controversial topic, as results vary, showing sometimes an inverse (or negative) correlation between alcohol quantity and the BMI or the weight gain in women [15, 28, 30-38] and, other times, a positive correlation [39, 40]. Moreover, studies found a positive association among men, [30, 39-48] as well as an inverse association [34, 36]. Actually, some studies, in both men [49] and women [35, 47], found no associations. Different patterns of consumption may be related to different outcomes [50].

Population studies demonstrate that the increase of body and abdominal fat is related to a moderate [49, 51] and frequent [52] alcohol consumption. There is also evidence that the alcohol can result in weight gain in different patterns to drink. The consumption of $\geq$ 30 g of alcohol/day may alter the balance of the energetic homeostasis, prompting an appetite increase and, consequently, body weight gain [53] and obesity, regardless of type of beverage consumed [43].

On a short-term, alcohol consumption is considered an appetite stimulant, influencing neurochemical and peripheral systems utilized to control appetite, such as, leptin inhibition, glucagon-like-peptide-1 and serotonin, and enhancing the effect of gamma-aminobutyric acid, endogenous opioids and neuropeptide Y. [54, 55] Hence, greater alcohol consumption with an absence of dependency, as well as binge drinking, may increase the risk for obesity. These effects may not be related to preferences for habitual drinks [56]. Toniolo et al [57] reported the increase of non-alcoholic energy consumption when an increase in the alcohol consumption was observed. However, Veenstra et al [58] showed that alcohol consumers and non-consumers had a similar intake of non-alcoholic caloric energy.

The association of alcohol consumption with a change in body weight and its development into obesity appears to be different depending on gender. Men add alcohol to the daily ingestion of calories, while women have a tendency to use alcohol as a substitute to other energy sources [30, 59], such as reducing the consumption of carbohydrates [16, 18] without an increase of the total calorie ingestion. These differences must be included when considering the energetic balance, as they may generate different results regarding bodyweight.

A variety of obesity risk factors are related to alcohol

consumption, such increased abdominal fat [60] decreased capability to oxidize lipids, consumption of high density calories diets [61, 62] and increase in cortisol secretion [63]. One may say that calories from alcohol are more utilized in individuals who are overweight or obese, and non-daily moderate consumers, than in those that are alcoholics and in a diet rich in fat [64].

Higher values of body, abdominal, and peripheral fat were also found in female, alcohol consumers, college students, showing a positive correlation between higher points in the AUDIT questionnaire and the BMI, the percentage of body fat, the waist circumference, the triceps skinfolds, and the arm circumference [65].

A study based on the data of the National Health Interview Surveys 1997 - 2001 demonstrated strong opposite effect between the quantity and frequency of alcohol consumption as related to the BMI. Individuals who drank more often but in small quantities (i.e. a drink a day, every day) had a lower BMI. On the other hand, individuals that drank less often, but did so in greater quantities (binge drinking), presented a higher BMI [39].

Jin et al [66] found an increase of the abdominal obesity risk factor in those individuals consuming ≥ 50 g/day. In agreement to this finding, in a study conducted with British individuals, it was noted that a consumption of 30 g or more of ethanol increased the risk of a high BMI and weight gain [43].

Similarly, the results provided after a study of the data of the Third National Health and Nutrition Examination Survey showed a lower chance of obesity among current alcohol consumers in relation to those abstinent, and a greater chance of obesity among binge drinking individuals or those who consume four or more doses a day. Obesity risk-factors were significantly lower among those reported drinking frequently but consuming less than five doses per week [40].

Alcohol and Lipid Profile

Alcohol consumption three to four days a week is associated with a lower risk of myocardial infarct among men and women. It is estimated that the cardio protector effect of alcohol may be attributed to 50% of the HDL-c increase [67, 68]. Moderate consumption of alcohol results (30 g ethanol/day) increases the concentration of HDL-c in approximately 4 mg/dL, and apoA-I in 8.82 mg/dL, with a reduction to the risk of cardiac disease estimated at 24.7% [69, 70]. This alteration may occur despite the quantity or type of drink consumed (wine, spirits or beer) [71, 72]. Furthermore, alcohol promotes less degradation of HDL-c and a greater liver metabolism of LDL-c [72].

Although alcohol consumption induces alterations to the lipid profile and reduces cardiovascular events, the incidence of strokes, such as brain hemorrhages and subarachnoid hemorrhage, has shown to be more elevated in heavy drinkers than in those who do not consume alcohol [73]. Additionally, there is a progressive increase of diseases attributed to greater alcohol consumption [74], such as diabetes mellitus, hypertensive cardiac disease, ischemic heart disease, ischemic and hemorrhagic stroke, among others [75].

The variation between alcohol's risks and benefits are unique to each individual and its utilization as a tool to cardiovascular protection should not be encouraged as a public health measure [76]. Filmore et al [77] described the reduction of the alcohol benefits to the prevention of coronary disease in a meta-analysis study where abstemious and light and moderate alcohol consumers show the same risk of any death caused by coronary disease.

Recently, Chen et al [78] found high levels of triglycerides in individuals consuming 10 g/day of alcohol. Consumption greater than 50 g/day significantly reduced the risk of developing low levels of HDL-c, but elevated the risks of developing high levels of cholesterol.

In the post-prandial period, the alcohol is responsible for an increase in the triglycerides, with the inhibition of the oxidation of free fat acids (FFA) [79]. It is important to highlight that individuals with coronary disease, the post-prandial hypertriglyceridemia is greater and longer [80]. The hypertriglyceridemia or the increase of FFA are associated to a reduction of the endothelial vasodilation in normal individuals [81] and those who are insulin-resistant [82].

Alcohol and Hypertension

The alcohol intake elevates the arterial pressure in a dose-dependent response. Consumption higher than two daily doses is one of the most common reversible causes of hypertension [83]. The renin-angiotensin system might be involved in the mechanisms that alcohol to induce hypertension [84].

A study showed that the acute alcohol intoxication changed the renin-aldosterone system to humans with normal hydric and sodium balances. The increase plasma renin was, probably, caused by dehydration due to ethanol diuresis or to the inhibiting action of the ethanol in the aldosterone secretion [85]. No significative change was found in the osmolarity, arterial pressure, and cardiac frequency. Later, an acute increase of renin activity when there is a moderate ingestion of alcohol was confirmed, inducing changes to the fluid and electrolyte balance and arterial blood pressure [86].

However, the answer to a low and moderate consumption of alcohol seems to be different, depending on the individual's gender. Again, the literature is not in agreement about the question. In a prospective study developed by the Women's Health Study with 28,848 women and in another one developed by the Physicians' Health Study with 13,455, the low and moderate consumption of alcohol decreased the risk of hypertension in women and increased it in men. The

threshold value above which alcohol became a risk factor for hypertension was equal to or larger than four doses a day in women versus a moderate level equal to or larger than one a day for men [87]. Nevertheless, some studies have been controversial when considering whether or not the arterial blood pressure sensitivity to alcohol is different in men and women [87-93].

Wakabayashi [94] suggests that an increase of the arterial blood pressure, as a consequence to habitual alcohol consumption, is more prominent in individuals with low body weight than in those with an elevated bodyweight. Therefore, the recommended alcohol consumption limit for the prevention of hypertension must be lower in women with low bodyweight than in those with a high bodyweight.

Stranges et al [95] investigated the association of alcohol consumption and the current drinking standards to the hypertension risk in healthy men and women without other cardiovascular diseases. When compared to abstemious, those who report alcohol drinks consumption had a higher risk of developing hypertension, despite the quantity of alcohol consumed.

A reduction in the ingestion of alcohol between heavy consumers significantly reduces the systolic and diastolic arterial pressures. Therefore, the reduction of alcohol consumption must be recommended as a life-style modification measure for the prevention and treatment of hypertension in heavy drinkers [96].

Conclusion

Alcohol is a drug of complex physiological effects that vary according to gender, age, race, body weight and consumption patterns. Recommending a moderate use of alcohol with the purpose of taking advantage of its health benefits must not be done. It must be reminded that there are groups of increased susceptibility to alcohol exposure. The present review shows that the additional consumption of ethanol calories will promote metabolic changes and weight gain increasing the risk of cardiovascular diseases, altering the lipid profile and promoting hypertension.

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