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A review on Influence of alcohol consumption on immunological status

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ABSTRACT: ***The purpose of this paper is to show and debate how various amounts of alcohol intake affect the immune system. In order to evaluate the effects on activity, number, distribution, balance, interaction, and responsiveness of immunocompetent cells, not only the quantity eaten but also the kind of alcoholic beverage must be taken into consideration. Alcohol exposure and the likelihood of getting an alcohol-related illness are linked by a number of factors. Age, gender, smoking habits, food consumption, and physical activity are all variables to consider. When the host's cellular and humoral immune responses were evaluated, it was shown that moderate alcohol intake may have some advantages. Furthermore, antioxidant-rich alcoholic drinks like red wine may act as immune cell defenders. According to the research reviewed, daily alcohol consumption of 10–12g for women and 20–24g for men is deemed moderate; the kind of beverage consumed is not a factor in determining moderation. The U- or J-shaped curve, which also indicates that light to moderate drinking has a protective impact, is frequently the subject of special study. This inverse connection suggests that light and moderate drinkers are at lower risk, whereas heavy drinkers and non-drinkers are at greater risk.***

KEYWORDS: ***Alcohol Consumption, Nutritional Status, Immune Function, Cytokines.***

1. INTRODUCTION

The link between alcohol consumption and the chance of getting an alcohol-related illness is complex, with significant individual variation in risk, with female vulnerability being particularly high. Apart from gender, age, ethnicity, hormones, body mass, personality, genetic and environmental variables all have an impact on individual vulnerability. Alcohol's net impact may also be influenced by lifestyle variables including smoking and overall health.

The U- or J-shaped curve, which indicates that mild to moderate drinking has a protective impact, is frequently the subject of special study. This inverse connection suggests that light and moderate drinkers are at lower risk, whereas heavy drinkers, as well as non-drinkers, are at higher risk. Moderate drinking may provide net health benefits that may be obtained in a less hazardous manner by not smoking, eating less dietary fat, and exercising regularly. The number of negative effects and their intensity are proportional to the amount of alcohol consumed [1]–[3].

1.1. Morbidity caused by alcohol:

Although high amounts of alcohol intake and extended periods of drinking are usually associated with more severe alcohol-induced morbidity, the timing and course of lesions is extremely varied. Even among communities of heavy drinkers and alcoholics, several kinds of organ damage usually associated with excessive alcohol use are not prevalent. Cardiomyopathy is uncommon, whereas cirrhosis of the liver and pancreatitis affect only a small percentage of people. Digestive problems, fatty liver infiltration, gastrointestinal hemorrhage, neuropsychiatric impairment, nutritional insufficiency, peripheral neuropathy, and skeletal myopathy are some of the more frequent conditions.

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Organ toxicity, carcinogenicity, genetic effects, teratogenicity, and immune system impacts may all be caused by hazardous chemicals. Reactions of alcohol or its metabolites with bodily components have detected all of these effects. Hepatoxicity and neurotoxicity are the two kinds of ethanol toxicity that are most often addressed, although there is widespread skepticism about identifying "the inherent toxicity of alcohol." Even today, it is well recognized that consuming large amounts of alcohol on a daily basis is harmful to virtually all bodily tissues. Researchers have learned more about the impact of moderate and high levels of alcohol use on the immune system in recent years. The current article's goal is to provide a state-of-the-art assessment.

1.2. Moderate alcohol consumption is defined as:

For many years, there has been a lack of agreement on how to define moderate alcohol intake, making it difficult to compare findings from research across the globe. In the United Kingdom, for example, moderation is defined as drinking no more than 21 units of alcohol per week for males and 14 units per week for women, with a unit equaling 10g of alcohol. In the United States, moderate drinking is defined as two drinks per day for males and one drink per day for women, according to the most current dietary recommendations.

Because there is no standard for determining the amount of alcohol in a drink, a difficulty arises. When it comes to defining moderation, the kind of beverage is ignored. Official public health recommendations for moderate alcohol intake do not exist in Spain or other European nations. The quantity of alcohol linked with the lowest mortality risk in the population is defined as moderate intake of alcohol in the sense of optimal. Our study group has suggested that moderate alcohol intake be classified as 10–12g/day for women and 20–24g/day for males, in line with other international organizations. Several studies have isolated beverage-specific relative hazards, but it has yet to be determined if one kind of beverage is more protective than another [4]–[6].

1.3. The immune system's relationship with alcohol consumption:

Age, race, gender, body composition, environmental variables, and the time, quantity, and kind of alcoholic beverage consumed all influence the degree of alterations in the immune system and nutritional status in alcoholics.

The effects of alcohol on the immune system have been ascribed mostly to a nutritional deficiency, which is often induced in alcoholics. Alcohol consumption has been linked to thymus and spleen shrinkage, loss or redistribution of peripheral blood leukocytes, and reduced humoral and cell-mediated immunological responses, among other things.

1.4. Cellular immunity and ethanol:

The effector stage of cell-mediated immunity begins with memory or effector T cells recognizing a particular antigen. The following characteristics in the cellular immunological state of alcoholic patients should be taken into account while researching them: The absolute number of circulating T lymphocytes, the nature of the sensitizing antigen, the potential experimental disparity between evaluating blood lymphocytes versus tissue-derived lymphocytes accumulating at the active site of liver cell damage, serum factors on cellular immunity, the effect of ethanol and its intermediate metabolites on lymphocytes and macrophages, and possible abnormalities in lymphocytes and macrophages In this respect, alcoholic patients have been shown to have a variety of dietary deficits, all of which may have an impact on lymphocyte function.

Indeed, utilizing pure protein derivatives such as histoplasmin, streptokinase-streptodornase, coccidiodin, and candida antigens, it was shown three decades ago that the delayed skin

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hypersensitivity reaction was frequently low in patients with ethanol-related liver disease. Only 32% of individuals with alcoholic illness had a favorable skin reaction.

When assessing the immunological response, it is important to remember that the amount of alcohol consumed should be taken into account. When the response to an intradermal injection of phytohemaglutinin is measured, changes in immune function in vivo are linked with variations in the quantity and duration of ethanol consumption. The region of induration (skin test reaction) has been shown to be substantially increased when ethanol consumption is minimal. High dosages of ethanol, on the other hand, reduced this reaction considerably.

Antioxidant-rich alcoholic drinks, such as red wine, may act as immune-system defenders. In mice given ethanol, baseline cell counts were substantially reduced. However, baseline cell counts in mice eating the same quantity of alcohol as wine stay the same as those in the control group who drink water. Similarly, the lymphocyte response to a lipopolysaccharide challenge was shown to be reduced in mice that consumed ethanol, but normal in mice who consumed the same quantity of ethanol as wine. In alcoholics, NK cells have been shown to have lower functional activity; alcoholics without liver illness may have normal numbers and activity, but in certain alcohol-related diseases, NK cell numbers are greatly decreased and NK activity is lost [7]–[9].

1.5. Humoral immunity and ethanol:

The humoral immune system is harmed by chronic alcohol use. Although the number of B cells in alcoholics without liver illness has been shown to be normal or slightly reduced, it seems to be substantially reduced in individuals with alcoholism. In alcoholics with liver disease, the immunoglobulins generated by B cells in response to infections are typically higher. An increased antigenic stimulation of B cells may be the cause of this hyper gamma globul inemia.

In alcoholic liver cirrhosis, total serum IgE is elevated. Increased IgE levels, as well as those of the other immunoglobulins, were originally thought to be the result of extensive liver damage. When compared to individuals with liver cirrhosis caused by other causes, serum IgE is exclusively elevated in patients with alcoholic cirrhosis.

In addition, following a period of abstinence from ethanol, blood IgE levels in alcoholics drop rapidly. As a result, chronic ethanol use has been proposed to have a role in IgE production. Similarly, both in alcoholics and in a specified healthy population, a rise in ethanol-related IgE has been demonstrated to be independent of concomitant behaviors such as smoking. Based on these findings, it's possible that IgE elevations in alcoholics are caused by alcohol and its metabolites, implying that alcohol may serve as a secondary antigen or allergy.

Furthermore, alcoholics' elevated blood IL-6, IL-10, and IL-8 levels have been shown to drop within days after abstinence from alcohol. In healthy individuals, on the other hand, IL-8 levels rose after they were exposed to alcohol. Increased IL-6 and Th2cytokines, such as IL-10 and IL-13, have been linked to IgE production in alcoholics. However, further research is needed to determine the impact of alcohol intake on the synthesis of interleukin and its implications for the development of IgE [10].

2. DISCUSSION

In 2016, alcohol use was projected to have caused 3.0 million fatalities globally, accounting for 5.3 percent of all deaths. Cancer accounts for a significant part of the harm caused by alcohol use. In 2016, alcohol was responsible for 376 200 cancer deaths (95 percent confidence interval: 324 900–439 700), accounting for 4.2 percent (95 percent confidence

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interval: 3.6–4.9 percent) of all cancer deaths and an age-standardized rate (ASR) of 4.8 deaths (95 percent confidence interval: 4.2–5.7) per 100,000 people. Malignancies caused by alcohol are referred to as "alcohol-attributable cancers" in this context. The percentage of cancers caused by alcohol is therefore determined by the fraction of cancers that would not have developed if alcohol had not been consumed. ICD-10 is the 10th edition of the International Statistical Classification of Diseases and Related Health Problems. Alcohol consumption was responsible for 10.3 million (95 percent uncertainty interval, 8.7 million–12.0 million) of the 245 million disability-adjusted life years lost in 2016 due to cancer, accounting for 4.2 percent (95 percent uncertainty interval, 3.6–4.9 percent) of all cancer DALYs lost. The bulk of these alcohol-related cancer DALYs (97.7%) were lost owing to years of life lost due to early death due to high cancer mortality rates. ICD-10, International Statistical Classification of Diseases and Related Health Problems, 10th edition; DALYs, disability-adjusted life years Colon, liver, and esophageal cancers were the leading causes of alcohol-related cancer fatalities in 2016, accounting for 23.9 percent, 22.3 percent, and 19.3 percent, respectively, of all alcohol-related cancer deaths.

The effect of alcohol use on cancer in 2016 varied by age group, with 13.9 percent of cancer deaths due to alcohol intake among individuals aged 30–34 years and 2.7 percent of cancer deaths among those aged 80–84 years. The liver, breast, and colorectal cancers were the major contributors to the alcohol-attributable cancer burden at younger ages, accounting for 32.2 percent, 19.4 percent, and 18.4 percent of all alcohol-attributable cancer fatalities among individuals aged 30–34 years, respectively. Colorectal, liver, and esophageal cancers were the major contributions to the alcohol-attributable cancer burden in individuals over the age of 80, accounting for 39.1%, 20.1 percent, and 14.9 percent of all alcohol-attributable cancer fatalities, respectively.

Because data are lacking and the etiology of these cancers is complex, the impact of alcohol on cancer deaths and DALYs lost among people aged 29 and younger is unknown; however, the proportion of alcohol-attributable cancers among this age group is hypothesized to be relatively small. In 2016, there were significant differences in the ASRs of alcohol-related cancer fatalities and cancer DALYs lost between nations and geographical areas. According to the Institute for Health Metrics and Evaluation's Global Burden of Disease study, the burden of alcohol-related cancers was lowest in North Africa and the Middle East (ASRs of 0.8 cancer deaths and 24.2 cancer DALYs lost per 100 000 people) and highest in Eastern Europe (ASRs of 0.8 cancer deaths and 24.2 cancer DALYs lost per 100 000 people) (ASRs of 12.0 cancer deaths and 360.4 cancer DALYs lost per 100 000 people). Similarly, the proportion of alcohol-related cancer deaths and DALYs lost varied significantly across countries and regions. The proportions were lowest in North Africa and the Middle East (0.8 percent of cancer deaths and 0.8 percent of cancer DALYs lost) and highest in Eastern Europe (0.8 percent of cancer deaths and 0.8 percent of cancer DALYs lost) (8.1 percent of cancer deaths and 8.6 percent of cancer DALYs lost). The cancer burden by site differed across geographical regions as well.

Alcohol-related colorectal cancers were particularly common in southern Latin America, high-income North America, high-income Asia Pacific, Australasia, and central, eastern, and western Europe, all of which have countries with high or very high Human Development Index scores (HDI). As countries develop, so does their alcohol consumption and cancer burden. In 2016, countries with a very high HDI (7.3 cancer deaths and 203.8 cancer DALYs lost per 100 000 people) had the highest ASRs, while countries with a medium HDI (2.5 cancer deaths and 78.8 cancer DALYs lost per 100 000 people) had the lowest (Fig. 5). The burden of alcohol-related cancer by HDI also differed by location. Colorectal cancer in countries with a very high HDI, liver cancer in both low and high HDI countries, and cancers

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of the lip and oral cavity in countries with a medium HDI contributed the most to the ASRs of alcohol-related cancer fatalities. The alcohol-related cancer fatalities and DALYs lost mentioned above only include cancer sites with adequate causative evidence, as assessed by the IARC Monographs, and do not include cancer sites with insufficient evidence of carcinogenicity in humans.

However, according to a 2015 study performed for France, the percentage of cancer incidence caused by alcohol rose from 7.9% when restricted to diseases with adequate causal evidence to 8.4% when considering cancers with at least minimal evidence of a direct relationship. Alcohol is a leading risk factor for cancer development and death in the United States, France, the United Kingdom, Australia, and the Nordic countries (Denmark, Finland, Iceland, Norway, Sweden, the Faroe Islands, and Greenland), according to country- and region-specific analyses of the relative contributions of risk factors to the cancer burden in the United States, France, the United Kingdom, Australia, and the Nordic countries (Denmark, Finland, Iceland, Norway, Sweden, the Faroe Islands, and Greenland Alcohol is the second most important risk factor for cancer development and death after tobacco in some studies and countries, such as a study that looked at nine behavioral and environmental risk factors for the Global Burden of Disease 2000 study and a study that looked at 13 risk factors for France in 2015.

The present burden of malignancies induced by alcohol use is considerable, and it is projected to grow in the future. As a result, initiatives aimed at reducing alcohol use in the general population are an effective and cost-efficient way of reducing cancer risk. The observed differences in alcohol-attributable fractions of cancer deaths and cancer DALYs lost between countries and regions provide evidence for how to reduce this burden through individual- and societal-level programs that reduce alcohol consumption, such as the WHO intervention strategies known as alcohol policy "best buys," which include raising excise taxes on alcoholic beverages.

Furthermore, interventions that target those risk factors that interact with alcohol use to raise the risk of cancer or that directly influence the risk of alcohol-related cancers, such as cigarette smoking, may decrease the burden of alcohol-related cancers. Furthermore, since early detection of cancer signs and symptoms, as well as early identification of precancerous lesions and tumors, are critical to patient survival in many instances, screening for colorectal and breast cancers may help to decrease the burden of alcohol-related malignancies. Finally, despite the proof of a causal connection between alcohol use and cancer development, the majority of the general public is ignorant of this association. Warning labels may be used to increase awareness about the connection between alcohol and cancer, but their efficacy in reducing alcohol intake is unclear at this time. Furthermore, medical practitioners in primary care may use short interventions to clarify the causal connection between alcohol and cancer in order to decrease alcohol use.

3. CONLUSION

In 2016, alcohol use was one of the main causes of cancer development and mortality worldwide, accounting for 376 200 cancer deaths (4.2 percent of all cancer deaths) and 10.3 million cancer disability-adjusted life years lost (4.2 percent of all cancer disability-adjusted life years lost). In 2016, the effect of alcohol intake on cancer varied by age group, with 13.9 percent of cancer deaths due to alcohol use among those aged 30–34 years and 2.7 percent of cancer deaths among those aged 80–84 years. It's worth noting that moderate intake seems to have a better effect on the immune system than excessive or non-consumption, as shown by a Gaussian-type curve. When comparing the effects of moderate and excessive alcohol consumption on the immune response, an inverse relationship is suggested. In reality, alcohol

use causes abnormally increased cell-mediated and humoral immune responses, resulting in damage to secondary lymphoid organs. When alcohol use is modest or low, however, immunological processes revert to normal, increasing immune competence.

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