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Alcohol Use and Non- Communicable Diseases, is there a Relationship?

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Abstract

Background and aims: *The relationship between alcohol use and non- communicable disease prevalence has puzzled researchers for many years. Alcohol use is widely practiced globally and is believed to be a major contributor to injuries, mortality and the burden of disease. This review updates knowledge on risk relations between dimensions of alcohol use and health outcomes such as; cardiovascular diseases, chronic obstructive pulmonary diseases, diabetes and cancers.*

Methods*: Systematic review of reviews and meta- analyses on chronic health outcomes attributable to alcohol use. For dimensions of exposure: volume of alcohol use, alcohol concentration and patterns of drinking, in particular heavy drinking occasions were studied. In total, 89 reviews and meta-analyses were searched in Google Scholar, PubMed, Medline but 28 reviews published from 2016 onwards were included in the study.*

Results*: Most partly imputable illness classes showed monotonic relationships with volume of alcohol use: additional alcohol consumed posed a higher risk of disease or death. Exceptions were ischemic diseases and polygenic disease, with curvilinear relationships, and with beneficial effects of light to moderate drinking in people without heavy irregular drinking occasions. Biological pathways counsel a control of significant drinking occasions on further diseases; but, the lack of medical epidemiological studies measuring this dimension of alcohol use precluded an in-depth analysis.*

Conclusions*: Research confirms alcohol use as both a risk factor for non-communicable diseases and at the same time protective when taken in moderation. However, further epidemiological studies should emphasize assessment of Health outcomes based on the quantity, frequency and concentration of the alcoholic drink.*

Keywords*: Alcohol use, non- communicable diseases, relationship.*

What is already known on this Topic?

Moderate alcohol consumption is thought to be associated with a lower risk of developing cardiovascular disease compared with abstinence or heavy drinking. There are ongoing debates about the role of combining different types of current non-drinkers in producing this apparent protective effect. Specifically, former or occasional drinkers might have reduced or ceased drinking because of ill health, making the aggregated non-drinking group artificially seem to have a higher risk of cardiovascular disease and mortality Less is known about the role of alcohol consumption in the etiology of particular non- communicable diseases; where studies exist they are often few in number, small in size, have combined different types of non-drinkers, and have not excluded all forms of non-communicable disease before the primary event.

What this study adds

This review debates the findings from various studies on the pathophysiological relationship between alcohol consumption and non-communicable diseases and also suggest focus for future engagements in the bid to mitigate health effects of harmful alcohol consumption.

Introduction

Non-communicable diseases (NCDs) are now a major focus of national and global preventative efforts because of global population ageing, with alcohol being considered one of the four key associated behavioral factors which contribute to this burden. The WHO Global action plan for the prevention and control of non- communicable diseases 2013–2020 focuses on cardiovascular diseases, cancers, chronic lung diseases and diabetes (WHO, 2018). These are diseases or

conditions that occur in, or are acknowledged to have an effect on, people over associate extended amount of your time, and that there aren't any acknowledged activating agents that are transmitted from one affected individual to another (Akinwale et al., 2017).

The National Cancer Institute defines alcohol as "a common term for ethanol or ethyl alcohol, a chemical substance found in alcoholic beverages such as beer, hard cider, malt liquor, wines, and distilled spirits (liquor), produced by the fermentation of sugars and starches by yeast and also found in some medicines, mouthwashes, and household products (including vanilla extract and other flavorings)". Alcohol has been consumed by most societies throughout human history, beginning 7000 to 9000 years ago in what is now present-day China, Armenia, and Iran. Alcohol is one of the most widely consumed beverages, and hence investigating whether alcohol consumption is associated with the risk of non-communicable diseases has important public health implications (Li et al., 2016; Kalla & Figueredo, 2017).

Regular consumption of alcohol by quantity, type or concentration has been found to either improve or worsen many risk factors for non-communicable diseases such as; increased triglycerides and reduced high density lipoproteins, cholesterol, hemostatic and fibrinolytic abnormalities that lead to hypercoagulable state, as well as endothelial dysfunction, inflammation, and heightened oxidative stress (Grant et al., 2017). Light to moderate alcohol intake has been associated with lower insulin levels and improved glycemic control, lower levels of fibrinogen, and reduced inflammation whereas the antioxidant polyphenols in red wine have been known to reduce arterial stiffness (Lie et al., 2016). Similarly, heavier alcohol intake, has been linked to increased blood pressure (Mori et al., 2016). This was also re-echoed by Taylor et al. (2017), who conducted a study in China and found a causal relationship between alcohol abuse and hypertension.

With the available evidence of increased dangers of heavy alcohol drinking such as; alcoholism, high blood pressure, obesity, stroke, breast cancer, suicide, and accidents, the American Heart Association advises those who drink, to do so in moderation and consult their doctors about the benefits and risks of consuming alcohol in moderation. The 2015–2020 Dietary Guidelines for Americans defines moderate alcohol consumption as upto1drink per day for women and up to 2 drinks per day for men, where a drink is defined as 12 ounces of beer, 4 ounces of wine, 1.5ounces of 80-proofspirits, or 1 ounce of 100-proof spirits (Benjamin et al., 2016). In 2016, harmful use of alcohol resulted in some 3 million deaths (5.3% of all deaths) worldwide and 132.6 million disability-adjusted life years (DALYs) – i.e. 5.1% of all DALYs. Mortality resulting from alcohol consumption was higher than that caused by diseases such as tuberculosis, HIV/AIDS and diabetes. During the same year, harmful use of alcohol caused loss of some 1.7 million lives to non- communicable diseases as a result of harmful alcohol use, this included up to 0.6 million deaths from digestive diseases, 0.6 million from cardiovascular diseases and 0.4 million deaths from cancers (WHO, 2018).

In the US alone, alcohol consumption affected between 4 to 5% of the population, with a 12.5% lifetime prevalence. Further still, excessive drinking is the third leading preventable cause of death (behind tobacco and obesity), accounting for 88 000 deaths annually in the United States, and resulting in approximately 2.5 million years of potential life lost (based on the Centers for disease control and prevention's alcohol- related disease impact application). Heavy drinkers (>6 drinks/day) are known to possess a doubled mortality rate, and consumption of 3 to 5 drinks is associated with a 50% higher mortality rate compared to nondrinkers (Kalla & Figueredo, 2017).

The purpose of this review is to update knowledge on risk and benefit relations between dimensions of alcohol use and health outcomes such as; cardiovascular diseases, chronic obstructive pulmonary diseases, diabetes and cancers. It also aims to outline the potential mechanisms of action whereby alcohol bestows benefits or induces harm, and suggest safe drinking practices, types and quantities of alcohol that optimize NCD outcomes and minimize risk and detriment for those choosing to consume.

Methods

The review was conducted as a systematic review following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Liberati et al., 2009). literature search was conducted in Google

Scholar, Medline, and PubMed for the relevant peer-reviewed studies published in English language between 2016 and 2019 using the search terms alcohol, ethanol, Non-communicable diseases, cardiovascular disease, Cancer, Diabetes and Chronic Obstructive Pulmonary diseases.

Inclusion and exclusion criteria

Studies included in this review were judged to be methodologically sound, high quality, objective, and reproducible; studies and reports previously conducted, peer reviewed and published between 2016 and 2019 relative to alcohol consumption and health outcomes such as; cardiovascular diseases, cancer, chronic obstructive pulmonary disease, diabetes. The relevant studies were identified by screening the titles, abstracts and papers. Findings associated with other modifiable behavioral risk factors like physical activity; diet and nutrition; smoking; and social activity other than alcohol consumption were excluded.

Fig 1 illustrates the flow chart for the study selection process. Studies excluded at the full paper screening stage are listed inS1 Table along with the reason for exclusion.

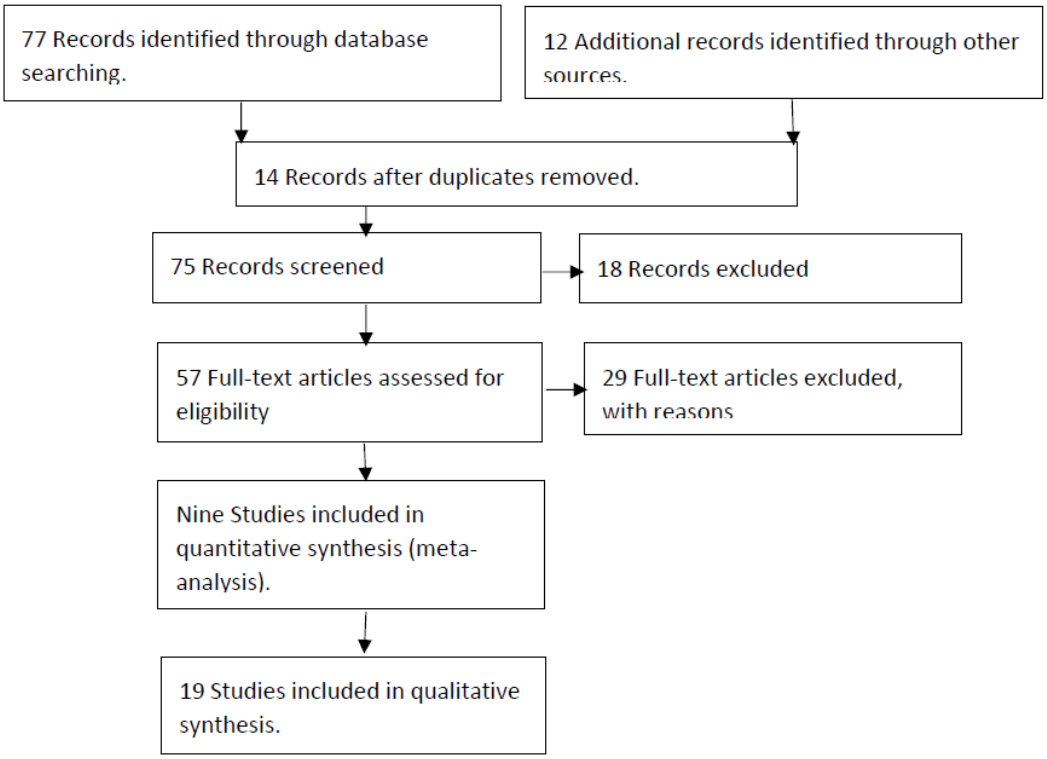

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta- analyses (PRISMA) flow chart (Liberati et al., 2009) search results on the relationship between alcohol and non- communicable diseases

Search results/ Findings

The searches for primary studies and the grey literature located 89 articles after removing duplicates, 57 of which had relevant titles and abstracts. In total, 29 studies that explicitly focus on alcohol consumption and outcomes of the main non- communicable diseases; CVDs, COPDs, Diabetes and Cancer informed the study review.

Alcohol use and cardiovascular diseases

The World Health Organization (WHO) defines Cardiovascular diseases as a group of disorders of the heart and blood vessels and include; coronary heart disease: disease of the blood vessels supplying the heart muscle; neural structure illness: illness of the blood vessels serving the brain; peripheral blood vessel disease: disease of blood vessels serving the arms and legs; rheumatic heart disease: injury to the heart muscle and heart valves from infectious disease, caused by true bacteria; inherent heart disease: malformations of heart structure existing at birth; deep vein occlusion and pulmonic embolism: blood clots within the leg veins, which can dislodge and move to the heart and lungs. Heart attacks and strokes are typically acute events

often caused by a blockage that forestalls blood from flowing to the guts or brain. The most common reason could be a build-up of fatty deposits on the inner walls of the blood vessels. Strokes will be caused by harm from a vessel within the brain or by blood clots.

In 2013, the global deaths due to cardiovascular diseases were estimated at 17.9 million, accounting for 48% of all NCD deaths and 31% of all global deaths and their incidence expected to increase to an estimated 23.6 million deaths by 2030 (WHO, 2017). Examples of cardiovascular diseases include; Ischemic heart disease, Ischemic stroke, hemorrhagic and other strokes, atrial fibrillation, Peripheral arterial disease (PAD), Aortic aneurysm, Cardiomyopathy and myocarditis, Hypertensive heart disease, Endocarditis, Rheumatic heart disease (RHD) (Benjamin et al., 2017).

A summary of evidence from a meta- analysis of 23 studies involving 29, 457 participants by Voskoboinik et al. (2017) demonstrated a complex physiological interplay of alcohol with cardiovascular events at different points after consumption, exhibiting both risky and cardio protective features. A further study by Whitman et al (2017) on alcohol abuse and Cardiac disease in California found a significant positive relationship between alcohol abuse and an increased risk of arterial fibrillation incidents, myocardial infarction and congestive heart failure, mimicking magnitudes of risk similar to other well-established risk factors. This implies that efforts to mitigate the addictive effects of alcohol could result in substantial reductions in cardiac disease and related conditions. Therefore, the findings demonstrated that alcohol in excess shouldn't be considered cardio protective but rather cardio toxic, contributing to heightened risk of all 3 major, yet distinctive, cardiac adverse health outcomes (Kalla & Figueredo, 2017).

Xi et al. (2017) conducted a study to establish the association between alcohol consumption and all-cause mortality among U.S adults >/= 18 years using the National Health Interview Survey which enrolled a total of 333,247 participants in an 8.2 year follow- up, findings revealed a reduced risk of all- cause mortality among abstainers, light and moderate alcohol consumers while there was an increased risk with heavy alcohol consumption.

Epidemiological studies over the last twenty years have however failed to confirm any linear associations between consumption of alcohol and cardiovascular (CV) conditions like peripheral arterial disease (PAD), stroke, hypertension (HTN), coronary heart disease (CHD), and cardiomyopathy. Probably, the dose and pattern of alcohol consumption determines the direction of the association. The characterization of dosage low-to-moderate daily alcohol consumption means <15 to 20 g/day and standard drink means 1 to 2 drinks said to be associated with a reduced risk of cardiovascular disease and mortality, whereas greater amounts of alcohol consumption and a binge pattern defined as equal or more than four drinks per day have been linked to an increased risk (Piano, 2017).

Further epidemiological studies have demonstrated the detrimental effects of heavy alcohol consumption citing a causative link with cardiovascular conditions such as arrhythmias, cardiomyopathy and hemorrhagic stroke, including accidents, hepatic cirrhosis, pancreatitis and other forms of malignancies. However, findings also reveal that consuming alcohol moderately especially with meals has a protective effect in coronary heart disease (CHD), ischemic stroke, peripheral arterial disease, CHD mortality, and all-cause mortality. Triglycerides are believed to play an independent role in the risk of CAD therefore, the alcohol association via triglycerides is likely to be unfavorable because some heavy drinkers have substantially increased blood triglyceride levels compared to light– moderate drinking which seldom causes increased triglyceride levels (Kalla & Figueredo, 2017).

The mechanism of this relationship has been linked by Klasky, 2015 to antithrombotic actions of alcohol which inhibits stickiness of platelets and lowers levels of fibrinogen, this results in an increased risk of hemorrhagic stroke and lowered risk of ischemic stroke making it both a risk and protective stroke factor. Evidence also suggest that the effect of alcohol on cardiovascular events has a genetic connotation; alcohol dehydrogenase polymorphism (ADH1C), a gene known to delay the metabolism of alcohol therefore, individuals who possess this gene tend to enjoy the protective effects of alcohol as compared to those who are deficient. It's important to note that some types of alcohol such as red wine contain polyphenols alongside other antioxidant and antithrombotic properties may qualify to provide protective benefits. However, differentiating the line of

action between the polyphenols and the ethanol in wine though other classes of similar compounds are present in grapes and other fruits and vegetables might have effects that promote endothelial health, remains a subject for further investigation.

Further efforts to confirm whether alcohol is protective or a risk factor in development of cardiovascular events should take into account; the genetic makeup of subjects which determine vulnerability, level of alcoholism, alcohol choice, amount of alcohol consumed, drinking pattern and interactions with medicines be ascertained individually). In the meantime, non-alcohol consumers should avoid drinking while the light to moderate drinkers should not change their habits.

Alcohol consumption and chronic obstructive pulmonary diseases

Chronic obstructive pulmonary disease is defined as airflow limitation that is not fully reversible. Chronic obstructive pulmonary disease is associated with an abnormal inflammatory response of the lung to harmful particles or gases. Diagnosis is based on post bronchodilator spirometry, which detects fixed airway obstruction; a forced expiratory volume in 1 second to forced vital capacity (FEV1/FVC) ratio of less than 0.70 is the current criterion for a positive COPD diagnosis. Persons with COPD often, but not always, have symptoms such as dyspnea (difficulty breathing or shortness of breath), chronic cough, and chronic sputum production. Patients often have a history of exposure to risk factors such as cigarette smoke or heating fuels or occupational exposure to dusts or chemicals. Although post bronchodilator spirometry is required to make a definitive diagnosis, prescreening questionnaires can elicit current symptoms and previous exposures to harmful particles or gases (Siu et al., 2016; Vogelmeier et al., 2017).

The burden of chronic metabolism diseases is mostly increasing across the world, chronic preventative pulmonic sickness (COPD) represent a significant category and are among the most causes of mortality and morbidity (Afshar et al., 2016). Numerous research findings have established a causative role of alcohol in the progression of COPDs, of which the ethanol (EtOH) component is a major suspect. The mechanism of EtOH effect on COPDs is demonstrated in its multiple deleterious effects on the lungs; EtOH contributes to lung dysfunction by altering barrier function in the airway epithelium, impairing the binding and phagocytic abilities of alveolar microphages (AMs), and deregulating mucociliary clearance in the airway.

EtOH exposure leads to decreased clearance of pathogens through several pathways, including a decrease in core binding factor (CBF) via protein-protein inhibitors (PP1) activation and impaired AM function through up regulation of NADPH oxidase (NOX); a family of enzymes implicated in reactive oxygen species generation and transforming growth factor (TGF) and down regulation of zinc transporters. EtOH acts on the adaptive immune system as well, via dendritic cells, T cells, and signaling cytokines. This combination of impaired CBF and a weakened immunologic response results in the pathological process of infectious respiratory organ sickness, including the bacterial pneumonia common among heavy drinkers. Additionally, EtOH-mediated epithelial permeability and impaired fluid clearance via ion channel dysregulation and increased TGF- predispose the lung to acute lung injury (ALI), and EtOH-induced matrix remodeling suggests a role for EtOH in lung fibrosis (Kaphalia and Calhoun, 2013). A thorough understanding of the results of EtOH and its metabolites on organic phenomenon, cell signaling, and oxidative stress will lead to better treatment options for EtOH-induced lung disease (Traphagen, Tian & Allen-Gipson, 2015).

Although an outline of proof by Kaluza et al., (2019), indicate an inverse association between moderate alcohol consumption and chronic inflammatory diseases; apparent association between alcohol consumption and chronic obstructive pulmonary disease (COPD) incidence was not been widely studied. However, reportage on the investigated associations of total alcohol consumption and intake of specific alcoholic beverages with risk of COPD in an exceedingly population-based prospective cohort study, the Cohort of Swedish Men (n = 44,254), using self-administered questionnaires in 1997 and during follow-up (1998–2014) involving 2,177 COPD cases, moderate alcohol consumption was found to be associated with the lowest risk of COPD. Furthermore, a J-shaped association was observed for ethanol consumption ($P = 0.003$) and beer consumption ($P < 0.001$); for wine consumption, a U-shaped association was

observed ($P < 0.001$). The revelation of the findings suggesting a decreased risk of COPDs with moderate beer and wine consumption, but not liquor consumption, is not conclusive enough and requires additional studies to confirm these associations.

Alcohol consumption and diabetes mellitus

This is a condition whereby; glucose accumulates in the blood and urine as a result of faulty glucose metabolism. Whether glucose is absorbed by the digestive tract or manufactured and released by the liver, very little glucose leaves the body once it has entered the bloodstream. The kidneys reabsorb virtually all glucose, so glucose does not appear in the urine. Genetic mutations as a result of inadequate insulin productions are believed to cause diabetes mellitus, some responsible genes have since then been identified. The synthesis of abnormal insulin molecules, or the production of defective receptor proteins produce comparable symptoms. These conditions have also been associated with obesity which is known to accelerate the onset and severity of the disease. Diabetes mellitus can also result from other pathological conditions, injuries, immune disorders, or hormonal imbalances (WHO, 2016).

Diabetes mellitus (DM) currently stands out as an important cause of morbidity and mortality, its prevalence almost doubled from 4.7% in 1980 to 8.5% in 2014. Consequently, the global adult population with diabetes mellitus rose from 108 million to 422 million over this time period and is estimated to reach 642 million people by 2040. The Institute of Health Monitoring and Evaluation indicated that an approximate 50.0% of patients with diabetes are believed not to be aware of their condition (Bommer et al., 2017)).

Diabetes mellitus is preceded by a pre-diabetes stage and classified into; type1, type 2 and gestational diabetes. Pre diabetes describes individuals who have blood glucose or A1C levels higher than normal but do not qualify to be classified as diabetics. Studies have shown that people with pre diabetes have an increased risk of developing type 2 diabetes, heart disease, and stroke. However, if they lose weight and increase their physical activity, they can prevent or delay type 2 diabetes and, in some cases, return their blood glucose levels to normal (Skyler et al., 2017).

Type one polygenic disorder is typically diagnosed in youngsters and young adults, previously known as juvenile diabetes. In this type of diabetes, the body does not produce insulin, it accounts for only 5% of diabetic people worldwide. The body breaks down the sugars and starches eaten into simple sugars called glucose and stored for energy use. Insulin is a hormone that the body needs to transform glucose into a storable form called glycogen and gets it from the bloodstream into the cells of the body. With the assistance of endocrine medical aid and different treatments, even young children can learn to manage their condition and live long, healthy lives. People with type 1 diabetes will therefore require daily administration of insulin to regulate the amount of glucose in their blood. If they are doing not have access to endocrine, they cannot survive. The reason behind sort one polygenic disorder isn't legendary and it's presently not preventable. Symptoms include excessive urination and thirst, constant hunger, weight loss, vision changes and fatigue (Insel et al., 2015).

According to Skyler et al. (2017), type 1 diabetes encompasses the majority of cases which are primarily due to pancreatic islet beta-cell destruction leading to absolute insulin deficiency and are prone to ketoacidosis. Type 1 diabetes includes those cases attributable to an autoimmune process, as well as those with beta cell destruction and who are prone to ketoacidosis for which neither an etiology nor a pathogenesis is known (idiopathic). It doesn't embody those sorts of beta-cell destruction or failure to that specific causes are allotted (e.g. cystic fibrosis, mitochondrial defects). Some subjects with this sort are known at earlier clinical stages than diabetes mellitus.

Type 2 diabetes (hyperglycemia) is the most common form of diabetes, it’s a problem with the body that causes blood glucose (sugar) levels to rise higher than normal. In kind 2diabetes, the body does not use insulin properly, this is called insulin resistance. At first, the pancreas compensates by producing extra insulin. But, over time it isn't able to keep up and can't make enough insulin to keep the blood glucose at normal levels (American Diabetes Association (ADA), 2019). Insulin resistance is typically the precursor to kind two polygenic disorder, a condition in which more insulin than usual is needed for glucose to enter cells. Insulin resistance in the liver results in more glucose

production while resistance in peripheral tissues means glucose uptake is impaired (Skyler et al., 2017).

The main risk factors for developing type 2 diabetes are both non- modifiable and modifiable: age; being over the age of 40 (over 25 for people of south Asian, Chinese, African-Caribbean or black African origin, even if you were born in the UK), genetic predisposition; having a close relative with the condition, such as a parent, brother or sister, weight; being overweight or obese and lack of physical activity (ADA, 2018).

Gestational DM (GDM) is outlined as any degree of aldohexose intolerance with onset or 1st recognition throughout gestation. The definition applies whether or not hormone or solely diet modification is employed for treatment and whether or not or not the condition persists when gestation. It doesn't exclude the chance that unrecognized aldohexose intolerance could have antedated or begun concomitantly with the gestation (A D A, 2019).

An anecdotal believe exists to the effect that alcohol increases the risk of diabetes however, there is also evidence of an inverse association between alcohol and the risk of type 2 diabetes. A myriad of studies reviewed by Lie et al., (2016) further reveal that the association between alcohol intake and type 2 diabetes risk, was both directly and inversely related. Inversely, 1 to 2 drinks per day for was found to be protective for women by increasing insulin sensitivity and lowering resistance of fasting insulin, corresponding to a 40% lower risk compared with lifetime alcohol abstainers. Alternatively, drinking 15 or more grams per day attenuated the positive association between glycemic load and type 2 diabetes incidence, potentially by improving insulin sensitivity. Alcohol consumption exhibits a U-shaped relationship with risk of T2DM in both males and females, with two drinks per (~50g/day) per day increasing the Relative Risks (RR) (Bertoglia et al., 2017).

A summary of evidence by Li et al., (2016) from various studies, found a relationship between heavy alcohol drinking and liver cirrhosis which is a risk factor in the development T2D. Furthermore, heavy daily consumption of alcohol was found to negatively affect the risk of diabetes in men coupled with the influence of other factors like age, BMI, smoking status, physical activity, and family history of T2D being significant contributors to the risk of T2D (Li et al., 2016). Although wine has been recommended to possess notably protecting effects in relevancy kind two polygenic disorder, epidemiological studies are inconclusive when it comes to the specific effects of beer and spirits. In addition, binge drinking has been associated with an increased risk of type 2 diabetes among men and women, while others have reported a tendency of reduced risk associated with binge drinking. Another space so that must be more addressed is whether or not the potential helpful result of alcohol on the danger of polygenic disorder pertains to specific food sorts.

Alcohol consumption and cancer

Cancer has been defined by the American Cancer Society as a group of diseases characterized by the uncontrolled growth and spread of abnormal cells in which uncontrolled spread can result in death. Although the reason for development of the disease remains unknown for many cancers, particularly those that occur during childhood, there are many known cancer causes, modifiable and non-modifiable factors (ACS, 2018). The International Agency for Research on Cancer (IARC) estimated the global cancer burden from 36 different cancer types to have risen to 18.1 million new cases and 9.6 million deaths in 2018. One in five men and one in half dozen girls worldwide develop cancer throughout their time period, and one in eight men and one in eleven girls die from the sickness.

Worldwide, the total number of people who are alive within 5 years of a cancer diagnosis, called the 5-year prevalence, is estimated to be 43.8 million (IARC, 2018). The trend is increasing and by 2030, the global burden is expected to reach 21.6 million new cancer cases and 13.0 million cancer deaths solely due to the growth and aging of the population. These projections may increase given the adoption of unhealthy behaviors and lifestyles associated with rapid income growth (e.g., smoking, poor diet, and physical inactivity) and changes in reproductive patterns (e.g., fewer children, later age at first childbirth) in LMICs (ACS, 2018).

Whereas the etiology of cancer is not clearly known, a dearth of previously conducted studies points to scientific evidence linking alcohol use to several cancer sites including; liver, breast, head and neck (mouth, oropharynx, hypopharynx, and esophagus), pancreas and colon cancers. Though the mechanism is not very

clear, the International Agency for Research on Cancer (IARC) suspects ethanol and its known metabolic product, acetaldehyde is understood to be carcinogenic in human beings. The acetaldehyde genotoxicity is believed to raise the concentration of estrogen, increases cellular stress, alteration in folate metabolism and causing inflammation. After absorption of ethanol in the small intestines, metabolism by alcohol dehydrogenase (ADH) enzyme into acetaldehyde takes place in the liver (Ratna &Mandekar, 2017).

Connors (2016) agrees that although measurement of associations exhibit gradients of effect of plausibility biologically, there is incomplete knowledge to justify the biological mechanisms however, the truth lies in the epidemiological evidence supporting the judgment that alcohol causes of various forms of cancer and potential reversibility of risk upon cessation of alcohol consumption. Similarly, the interplay between alcohol, obesity and other non-modifiable factors such as race in determining disease outcomes among alcohol users has been demonstrated. For example, studies indicate that black women found to be obese and consuming alcohol have higher incidences of breast cancer and more aggressive tumors among white women, black women found to be obese and using alcohol are more likely to have aggressive tumors with less favorable histology, and to have a poorer prognosis (Killelea et al., 2019).

Pathways of cancer causation in consumption of alcohol is demonstrated as;

- Inhibition of absorption of folate by altering one carbon metabolism, this leads to an increase in concentrations of homocysteine which in turn interrupts the cycle of the enzyme methionine synthase and also participates in the trans-methylation of the enzyme's DNA methyltransferase and methionine adenosyltransferase.
- It is possible that alcohol affects the levels of serum in hormones and associated pathways for signal thereby increasing the risk of breast cancer and partly prostate, ovarian and endometrial cancers.
- Consumption of alcohol may alter the levels of serum in insulin-like growth factor (IGF); the relationship may be a complex one, chronic consumption of alcohol in moderation is bound to increase serum levels of IGF, and acute consumption of alcohol causing a decrease in IGF levels.
- Alcohol has been shown to damage permanently the DNA strands in the cell, and to inhibit DNA repair processes from functioning, particularly through acetaldehyde – the immediate product of alcohol metabolism. Alcohol use may also lead to nutritional deficiencies that affect DNA processing pathways. Some genetic variations are also associated with an increased risk from alcohol in cancer development. Alcohol is also thought to modulate estrogen pathways, thus increasing the risk for development of breast cancer in females (WHO, 2018).
- Finally, there is a demonstrated interaction between alcohol and smoking of tobacco, tobacco is known for its carcinogenic effects on the oral cavity and oesophago (SCC) and in this case, alcohol aids in the absorption of tobacco carcinogens by acting as a solvent (Rehm et al., 2017).

It is worth noting that in 2016, the World Health Organization acknowledged that up to 80 countries had reported having written national alcohol policies, while a further eight countries had subnational policies and 11 others had a total ban on alcohol. Accordingly, the percentage of countries with a written national alcohol policy steadily increased from 2008, and many countries have revised their policies since the Global strategy to reduce the harmful use of alcohol was released. The majority of countries in Africa and the Americas do not have written national alcohol policies. The presence of national alcohol policies is highest among reporting high-income countries (67%) and lowest among low-income countries (15%). Principal responsibility for the policy lies with the health sector in 69% of countries with a national policy.

Conclusion

In observation of the current trends, projections point to an increase in total per capita consumption of alcohol worldwide in the next 10 years and this is likely to put the target of a 10% relative reduction by 2025 out of reach unless implementation of effective alcohol control measures reverse the situation in countries with high and increasing levels of alcohol consumption. The relationship between alcohol and NCDs is complex; heavy episodic drinking

has a causal relationship with onset of cancers such as; the oro-pharyngeal, laryngeal, esophageal, liver, colon, rectal and the female breast. Other conditions as; hypertensive heart disease, hemorrhagic stroke, alcoholic cardiomyopathy, cirrhosis of the liver, pancreatitis are also believed to be exasperated by heavy alcohol consumption. However, drinking in moderation is seen to be protective for disease conditions such as; Diabetes mellitus, ischemic heart disease, ischemic stroke. Contextualization, domestication and implementation at national levels to the dot of the recommendations entailed in the global strategy to reduce alcohol consumption by 10% by 2025 will go a long way in averting the negative effects of alcohol use on population health.

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