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ALCOHOL CONSUMPTION AND RISK FOR PARKINSON'S DISEASE : A SYSTEMATIC REVIEW

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ABSTRACT
The neurodegenerative disorder known as Parkinson disease (PD) is an illness that affects the brain. It is characterized by motor symptoms such as sluggish movement, tremor, stiffness, and imbalance, as well as other consequences such as cognitive impairment, mental health issues, sleep difficulties, and pain and sensory abnormalities. It is also characterized by additional complications. Over the course of many decades, lifestyle exposures such as smoking, drinking coffee or tea, and consuming alcohol have been the subject of study, and the findings have been variable and sometimes contradictory. Lifestyle exposures such as these have been identified as contributing factors to the risk of Parkinson's disease (PD), in addition to the genetic and familial environmental exposures that are often stated as contributing factors to the risk of the illness. Alcohol consumption has been linked to Parkinson's disease (PD), although the biochemical mechanisms behind this connection are poorly understood and open to conjecture. The fact that we identified different correlations with different types of alcoholic beverages led us to infer that the mechanisms at play involve components that are not the ethanol itself or that are in addition to it. Beer, but not wine or liquor, contains a significant amount of purine, which, when mixed with ethanol, has the ability to enhance plasma urate levels in a synergistic way. Wine and liquor, on the other hand, do not include purine. However, data regarding the relationship between alcohol consumption and PD are inconsistent.

Keyword: *Alcohol; Lifestyle; Parkinson' Disease; Urate Acid*

INTRODUCTION

Parkinson disease (PD) is a neurodegenerative condition that affects the brain. It is characterized by motor symptoms such as slowed movement, tremor, rigidity, and imbalance, as well as other complications such as cognitive impairment, mental health disorders, sleep disorders, and pain and sensory disturbances.[1,2] It is believed that at least one percent of people in the population over the age of 60 are affected by Parkinson disease. Both the death of dopaminergic neurons in the substantia nigra and the existence of Lewy bodies are linked to the illness. The vast majority of cases are idiopathic.[3,4] It's estimated that just approximately 10% of instances have a hereditary origin, and the majority of those occurrences occur in young individuals. The start of the condition is gradual, but it will worsen with time.[2] Tremor is often the first symptom to appear, and later on, bradykinesia and stiffness may be connected with the condition. Instability in one's posture often manifests itself late in the course of the illness and may have a significant negative influence on one's quality of life. The existence of autonomic symptoms, which in some individuals might come before motor symptoms, is another factor that should not be overlooked.[3–6]

Little is known about the aetiology of Parkinson's disease (PD), which is the second most common neurodegenerative disorder following Alzheimer's disease.[7] The etiology of Parkinson's disease is unknown or idiopathic, but is thought to be related to genetic and environmental factors.[8] Lifestyle exposures such as smoking, coffee/tea and alcohol consumption have been the focus of research for several decades with varying and often conflicting results. While genetic and familial environmental exposures are frequently cited as contributing factors to the risk of Parkinson's disease (PD), lifestyle exposures such as these have also been cited as contributing factors.[3,9,10]

This article discusses correlation alcohol consumtion and Parkinson's disease.

METHODS

The information used in this systematic review came from full-text English publications that were released in the 10 years before to this study's start date (range 2012-2022). Pubmed and Google Schoolar are the two databases that were employed during the production of this essay. In this inquiry, we analysis the correlation alcohol consumtions and Parkinson's diseaseThe study included both controlled clinical trials and randomized clinical trials as its component parts.

In accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) paradigm, the researchers initially input keywords into each database for this study. The phrases "alcohol consumtion" or "alcohol drinking" and "Parkinson's disease" were used in the search : (("alcohol drinking"[MeSH Terms] OR ("alcohol"[All Fields] AND "drinking"[All Fields]) OR "alcohol drinking"[All Fields] OR ("alcohol"[All Fields] AND "consumption"[All Fields]) OR "alcohol consumption"[All Fields]) AND ("parkinson disease"[MeSH Terms] OR ("parkinson"[All Fields] AND "disease"[All Fields]) OR "parkinson disease"[All Fields] OR "parkinson s disease"[All Fields])) AND ((y_10[Filter]) AND (clinicaltrial[Filter] OR randomizedcontrolledtrial[Filter])). The researchers received four articles, which will be discussed during the discussion **(Table 1)**.

RESULT

Eriksson, et al (2013) showed that women and men had a higher probability of admission with a diagnosis of Parkinson's disease if their medical history included a history of alcohol use disorder. In particular, it seemed that the risk was greater at younger ages when Parkinson's disease was diagnosed for the first time.[11] Liu, et al (2013) study showed a reduced to moderate intake of beer is associated with a decreased risk of PD, but a larger consumption of liquor is associated with an increased risk.[12]

Study conducted with 209,998 PD-free participants showed no associations between baseline or lifetime total alcohol consumption and PD risk were observed. Men with moderate lifetime consumption (5–29.9 g/day) were at ~50% higher risk compared with light consumption (0.1–4.9 g/day), but no linear exposure–response trend was observed. Analyses by beverage type also revealed no associations with PD.[13]

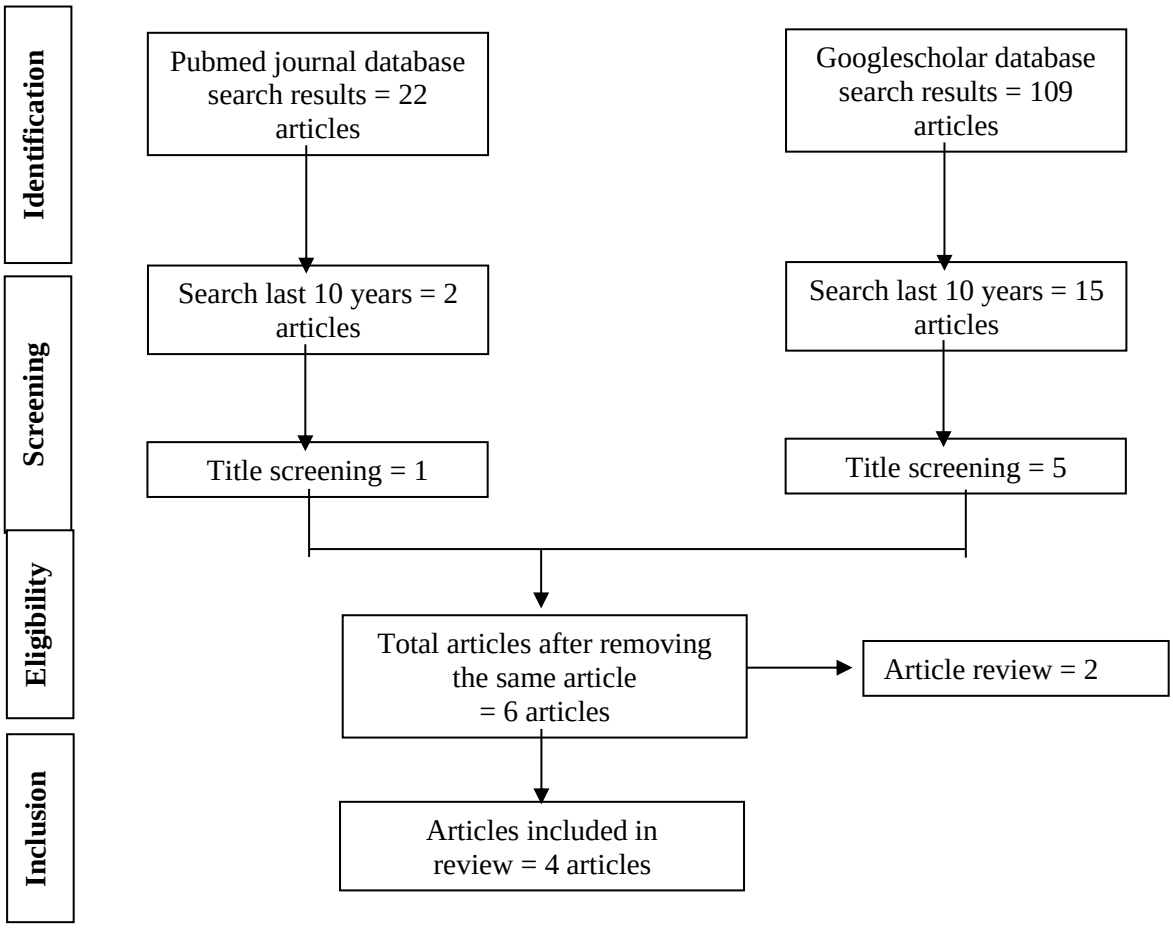

Figure 1. Article search flowchart

Table 1. The litelature include in this study

Author	Origin	Method	Sample Size	Period	Result
Eriksson, 2013[11]	Swedeen	Cohort retrospective	602,930 individuals	January 1, 1972 and December 31, 2008	1,741 (0.3%) of 602,930 people had PD, 1,083 (0.4%) had an alcohol use problem, and 658 (0.2%) had appendicitis. Follow-up averaged 13.6 and 17.1 years. Adjusted for age and sex, the HR for PD was 1.38 (CI 1.25-1.53) Adjusted for age at exposure and sex, the lowest age group <44, had the greatest risk for initial hospitalization with PD (HR 2.39, 0.96-5.93).
Liu, 2013[12]	USA	Cross sectional	306,895 participants	2000-2006	Between 2000 and 2006, 1,113 PD cases were analyzed. Total alcohol intake didn't affect PD. The connection varied by alcohol type. Beer drinkers' multivariate ORs were 0.79 (95 percent CI: 0.68, 0.92) for 1 drink/day, 0.73 (95 percent CI: 0.50, 1.07) for 1-1.99 drinks/day, and 0.86 (95 percent CI: 0.60, 1.21) for ≥2 drinks/day. For liquor intake, PD risk increased monotonically with 1, 1-1.99, and ≥2 drinks/day (P for trend 0.03). Additional investigations among exclusive one-drinkers validated similar results. Comparing wine drinkers of 1-1.99 drinks/day with nondrinkers showed a borderline decreased PD risk (OR = 0.74, 95% CI: 0.53-1.00).
Peters, 2020[13]	Netherlands	Cohort retrospective	209,998 PD-free participants	No date	No relationships were found between the total amount of alcohol consumed at baseline or during one's lifetime with the risk of PD. Men who had a moderate lifetime use of alcohol (5–29.9 g/day) had a risk that was about fifty percent greater than those who had a light consumption of alcohol (0.1–4.9 g/day), but there was no evidence of a linear exposure–response relationship. The kind of beverage was not a factor in any of the analyses that found a connection with PD.
Kim, 2020[14]	USA	Cross sectional	1,309,267 women	1996 and 2001	In 1,309,267 women followed for 17.9 years, 11,009 developed PD. In drinkers, the multivariable-adjusted relative risk for women who drank 14 or more drinks per week was 0.99. (95 percent confidence interval: 0.90, 1.10). Excluding the first 10 years of follow-up did not modify the results (risk-adjusted = 1.01; 95% CI: 0.90, 1.13). Never-smokers showed no alcohol-related PD risk trends. This connection was also null by alcohol type.

Among the 1,309,267 women who were followed for an average of 17.9 years, 11,009 of them were diagnosed with PD for the first time. In women who drank alcohol, the relative risk was 0.99 when comparing women who drank more than 14 drinks of alcohol per week to women who drank between 1 and 2 drinks of alcohol per week. This comparison was done among drinkers (95 percent confidence interval: 0.90, 1.10).[14]

After removing the first 10 years of follow-up, the findings did not significantly shift (relative risk adjusted = 1.01; 95 percent confidence interval: 0.90, 1.13). Never smokers did not show any significant upward or downward changes in their risk of Parkinson's disease (PD) due to alcohol use. In addition, analyzing this connection based on the kind of alcohol consumed also resulted in results that were inconclusive.[14]

DISCUSSION

Drinks that include ethanol are referred to as alcoholic beverages, however the abbreviation "Minol" is also often used. Consuming a chemical that produces psychoactive effects, such as ethanol, may lead to a loss of consciousness. In many nations, the purchase of alcoholic drinks is restricted to a specified demographic, which is often comprised of individuals

who have reached or beyond a minimum age requirement. Researchers have spent a significant amount of time looking at the possibility of a link between PD and aspects of one's lifestyle, such as the use of tobacco products, the consumption of coffee, and the drinking of alcohol.[15]

The role of alcohol use in the genesis of PD has not been well explored. Majority of prior epidemiologic investigations were case-control studies that did not discriminate between particular kinds of alcoholic beverages, and the majority did not discover a relationship between alcohol intake and PD risk. Inconsistent outcomes were also found in the limited prospective trials currently available.[15] Other study showed no associations between baseline or lifetime total alcohol consumption and PD risk were observed.[13,14]

The neurodegenerative ailment known as Parkinson disease is characterized by a widespread slowing of movements known as bradykinesia and at least one additional symptom like resting tremor or stiffness. Parkinson disease often manifests itself in later life. A loss of smell, dysfunctional sleep, mood issues, increased salivation, constipation, and excessive periodic limb movements in sleep are some of the other symptoms that are connected with this condition. It is believed that at least one percent of those over the age of 60 are affected by Parkinson disease.[1,16]

Both the death of dopaminergic neurons in the substantia nigra and the existence of Lewy bodies are linked to the illness. The vast majority of cases are idiopathic. It's estimated that just approximately 10% of instances have a hereditary origin, and the majority of those occurrences occur in young individuals. The start of the condition is gradual, but it will worsen with time. Tremor is often the first symptom to appear, and later on, bradykinesia and stiffness may be connected with the condition.[1,16]

Instability in one's posture often manifests itself late in the course of the illness and may have a significant negative influence on one's quality of life. The existence of autonomic symptoms, which in some individuals might come before motor symptoms, is another factor that should not be overlooked. The history of the patient and their current symptoms are the two primary factors that are considered while making a diagnosis.[1,16]

Intoxication, as well as abstinence from alcohol in certain situations, may cause neurological deficiencies that manifest as motor impairments, lapses in memory, and other cognitive problems. Consuming alcohol in excessive amounts over a prolonged period of time is another factor that contributes to more severe neurological diseases including Alzheimer's disease (AD) and Parkinson's disease (PD).[17]

According to the findings of a Swedish National Cohort Study that took place between 1978 and 2008, out of 276,527 individuals who were diagnosed with AUD and then monitored for 37 years, 1,083 (0.4 percent) were hospitalized with Parkinson's Disease. A different cohort research was carried out in France between the years of 2008 and 2013, and it found that AUD was the most significant modifiable risk factor for the development of dementia. This study included 38.9 percent of the 57353 people who were diagnosed with early-onset dementia.[8,18]

Consumption of alcohol, as shown by admission with an alcohol use problem, was associated with an elevated risk of Parkinson's disease in both females and males. The impact of alcohol intake seemed to be stronger at younger PD ages when the HRs were estimated for age groups of initial admission with PD and adjusted for age at exposure and sex.[11]

Other study showed low to moderate beer drinking is associated with a decreased risk of Parkinson's disease, whereas higher alcohol consumption is associated with a higher risk. These results and possible underlying processes need more research.[12,19]

The biological processes that connect alcohol use with PD are not well understood and are subject to speculation. The fact that we found diverse relationships with various kinds of alcoholic drinks leads us to believe that the processes at play include elements other than or in addition to the ethanol itself. Beer, but not wine or liquor, includes a significant quantity of purine, which, when combined with ethanol, has the potential to raise plasma urate levels in a synergistic manner.[20,21]

Patients with Parkinson's disease who take urate, which is a powerful free-radical scavenger, have a decreased chance of developing Parkinson's disease and have slower clinical development. Beer, in comparison to wine or liquor, has a relatively high concentration of the vitamin niacin, which has been shown to lower the probability of developing Parkinson's disease. Our result that heavy users of booze had a greater risk of Parkinson's disease is more difficult to explain.[15,22]

When compared with wine and beer, the percentage of pure ethanol that is contained in liquor is much higher. Study hypothesize that this may be the cause of this phenomenon. It has been proven that drinking pure ethanol may cause oxidative stress by functioning as a pro-oxidant, and it also has the potential to cause inflammation. In addition, liquor and other drinks made by the distillation process do not include any vitamins or antioxidants.[8,23]

It has been shown that the polyphenolic components of red wine, such as resveratrol, have the ability to reduce the neurotoxic 6-hydroxydopamine (6-OHDA)-induced toxicity in animal models of Parkinson's disease (PD) due to their antioxidant and anti-inflammatory potentials. However, previous epidemiologic research, including our own, has not been successful in finding a neuroprotective impact of wine on the risk of Parkinson's disease (PD).[8,23]

CONCLUSION

However, data regarding the relationship between alcohol consumption and PD are inconsistent.

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