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Article

Alcohol Intake and Prevalent Kidney Stone: The National Health and Nutrition Examination Survey 2007–2018

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Abstract: The association of alcohol intake with kidney stone disease (KSD) is not clear. We examined the National Health and Nutrition Examination Survey (NHANES) 2007–2018 and used logistic regression analyses to determine the independent association between alcohol intake and prevalent KSD. 29,684 participants were eligible for the final analysis including 2,840 prevalent stone formers (SF). Mean alcohol intake was 37.0±2.4 g/day among SF compared to 42.7±0.9 among non-SF ($p=0.04$). Beer [Odds ratio (OR)=0.76, 95% CI: 0.61–0.94, $p=0.01$] and wine (OR=0.75, 95% CI: 0.59–0.96, $p=0.03$) intakes were strongly associated with lower odds of prevalent KSD while liquor intake had no association. The effect from beer was dose dependent with an OR of 0.34 (95% CI: 0.20–0.57, $p<0.001$) for stone formation comparing participants drinking >56g/day of beer to non-drinkers. Interestingly, the effect from wine was only significant among participants drinking moderate amount, with an OR of 0.54 (95% CI: 0.36–0.81, $p=0.003$) compared to non-drinkers. These effects were consistent in spline models. This study suggests that both moderate to heavy beer intake and moderate wine intake are associated with a reduced risk of KSD. Future prospective studies are needed to clarify the causal relationship.

Keywords: alcohol intake; beer; wine; liquor; kidney stone

Introduction

Kidney stone (KS) disease is highly prevalent and carries a significant economic burden [1]. Although adequate hydration is essential for stone prevention, types of liquid may have different modifying effects on KS risk [2–4].

Alcohol may increase urinary concentrations of calcium [5–7], phosphorus [7], and uric acid [7–11] thereby increasing their supersaturations. It may also increase the urinary concentration of magnesium [7,12–15], an important inhibitor of stone formation. Furthermore, it contains a fair amount of liquid and has a diuretic effect [16–19] which can reduce KS formation. The overall effect of alcohol on KS disease is unclear. In existing clinical studies, the effect of alcohol on KS disease has been inconsistent with some studies showing that it reduces the risk [20–24], while others showing no significant association between alcohol intake and risk of KS disease [25–27].

Here, we used a large US population survey database, the National Health and Nutrition Examination Survey (NHANES) from 2007 to 2018, to examine the independent association between alcohol intake and KS disease.

Methods

Study Population

NHANES is an ongoing series of cross-sectional assessments of the health and nutritional status of adults and children in the US. The survey collects demographic, socioeconomic, dietary, and health-related information, in addition to the examination and laboratory data obtained by highly trained medical personnel. A total of 59,842 participants were interviewed for NHANES from 2007 to 2018. Of these, our analysis included 29,684 participants aged 20 years or older with complete data on alcohol intake, history of KS, and the covariates of interest. (Figure 1).

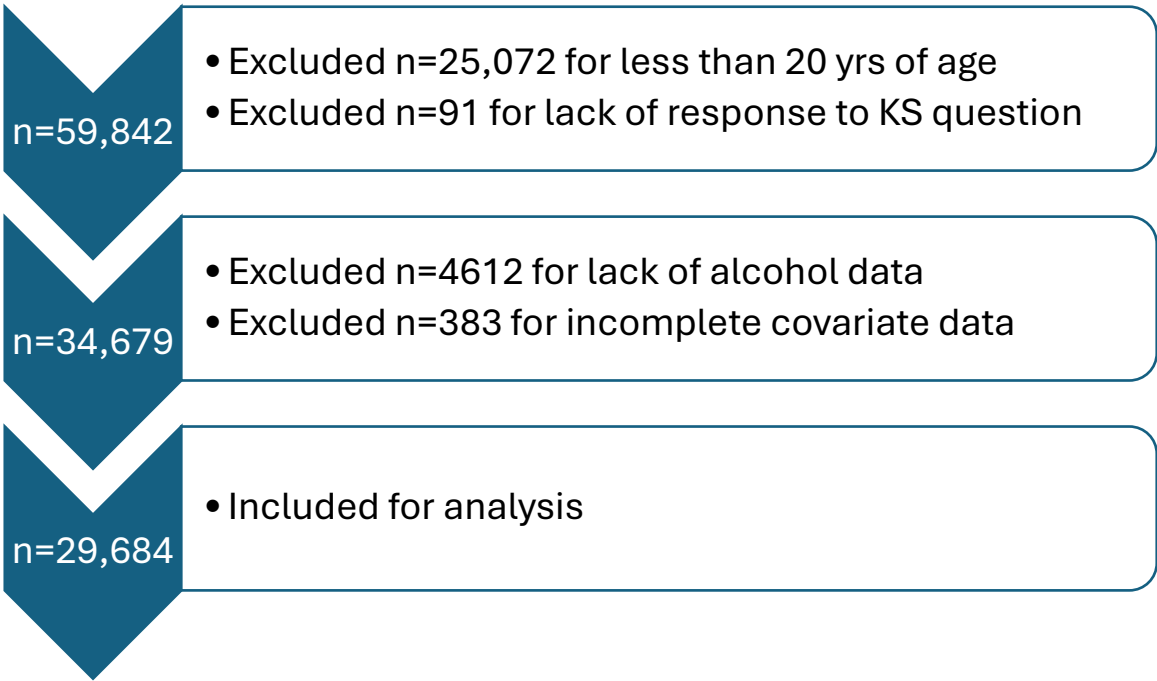


Figure 1. Selection of study population.

Primary Exposure and Outcome

Our primary exposure was amount and type of alcohol intake using specific foods reported on the 24-hour dietary recall interviews. Individual foods and beverages with a non-zero amount of alcohol were examined and classified as beer, wine, liquor, mixed drinks, and other sources of alcohol, such as prepared alcohol containing foods. Total alcohol in grams by type was summed for each participant. Alcohol drinking status was obtained by questionnaire and categorized as never, former (no drinks in the past 12 months), and current (at least one drink in the past 12 months). Never and former drinkers who reported any alcohol on the 24-hour recall interviews were excluded. Only data from day one out of the two 24-hour recall periods was included in the present analysis.

Primary Outcome

The outcome or dependent variable of interest was prevalent KS disease. It was extracted from the interview data file. ‘Have you ever had a kidney stone?’ was the question asked during the standardized home interview. Adult participants who responded ‘yes’ to the question were considered to have a history of KS.

Covariates

Age, sex, race, history of diabetes, hypertension, thiazide use, and smoking status was obtained from the questionnaire. Body mass index (BMI) was calculated from height and weight measured during the health examination. Information on total intakes of calories, protein, and fluids (excluding

alcohol) along with dietary intakes of sodium, potassium, and calcium were obtained from 24-hour dietary recall during the same day-one interview when data on the type and amount of alcohol intake was collected.

Analysis

Statistical analysis was performed with Stata MP 18 (Stata Corp, College Station TX). The complex sampling design was incorporated by applying strata, primary sampling units, and sampling weights via survey-specific procedures. Day one 24-hour recall weights were used for all analyses. Logistic regression was used to estimate unadjusted and multivariable-adjusted odds ratios (OR) and 95% confidence intervals (CI) for alcohol intake and prevalent KS disease. Alcohol intake was examined as both a categorical and a continuous predictor of KS formation. Categories were created from type of alcohol consumed and, within each type, from amounts reflecting ratios of a standard drink (14g) [28]. Alcohol intake of each type was also examined by including restricted cubic splines in the regression model [29]. Knots were specified at the 5th, 25th, 50th, 75th, and 95th percentiles of the distribution among participants drinking at least 1 gram of a specific type of alcohol. A binary indicator variable was added to the model to represent 0 to less than 1 gram of alcohol [30]. Deviations from linearity were assessed by testing coefficients for non-linear spline terms. The multivariable models included age (years), sex, race (non-Hispanic White, non-Hispanic Black, Hispanic/Latino, Non-Hispanic Other), BMI (<25, 25-<30, >30 kg/m²), diabetes (no, borderline/yes), hypertension, thiazide diuretic use, smoking (never, former, current), total dietary calories (kcal), total dietary protein (g), total fluid without alcohol contribution (g), dietary sodium (mg), potassium (mg), and calcium (mg). All p-values presented were two-tailed with p<0.05 considered statistically significant.

Results

A total of 29,684 participants were included in this analysis. 2,840 (9.7%) of these reported a history of stones. Among stone formers, 2,087 (78.1%) participants reported drinking alcohol currently, as compared to 19,985 (79.6%) participants among non-stone formers (p=0.002). Mean alcohol intake was also significantly lower with 37.0±2.4 g/day in stone formers compared to 42.7±0.91 g/day in non-stone formers (p=0.04). Stone formers tended to be older, predominantly male, Non-Hispanic White with a higher BMI compared to non-stone formers. They were also more likely to have a history of diabetes, hypertension, to use thiazides and had a history of smoking (Table 1).

Table 1. Baseline characteristics of study population.

	KS Former	Non-KS Former	p value
Total n, unweighted	9.7 (2,840)	90.3 (26,844)	
Male sex	54.6 (1,571)	47.4 (12,865)	<0.001
Age (y)	53.7 ± 0.38	46.8 ± 0.26	<0.001
Race			<0.001
Non-Hispanic White	76.2 (1,553)	65.3 (10,851)	
Non-Hispanic Black	5.9 (376)	11.8 (5,995)	
Hispanic/Latino	11.7 (690)	14.7 (6,844)	
Non-Hispanic other	6.1 (221)	8.2 (3,154)	
BMI (kg/m ²)			<0.001
<25.0	19.8 (542)	30.2 (7,803)	
25.0-<30.0	32.9 (962)	32.8 (8,784)	
30.0+	47.2 (1,336)	37.0 (10,257)	
History of diabetes	22.4 (736)	10.8 (3,895)	<0.001
History of hypertension	48.7 (1,487)	32.3 (9,841)	<0.001
Thiazide diuretic use	12.6 (377)	7.8 (2,498)	<0.001
Smoking status			<0.001
Never	49.8 (1,392)	56.2 (15,104)	
Former	30.6 (884)	24.0 (6,267)	
Current	19.6 (564)	19.8 (5,473)	

Total calories (kcal)	2,122.9 ± 28.7	2,142.9 ± 8.9	0.5
Protein intake (g)	80.8 ± 1.4	82.8 ± 0.42	0.16
Dietary sodium (mg)	3,534.0 ± 54.6	3,539 ± 15.9	0.93
Dietary potassium (mg)	2,644.0 ± 38.3	2,690.2 ± 15.5	0.22
Dietary calcium (mg)	934.3 ± 15.3	973.7 ± 6.6	0.02
Total fluid intake, excluding alcohol (g)	2,905.0 ± 35.4	2,885.9 ± 20.0	0.58
Alcohol drinking status			0.002
Never	12.3 (442)	13.6 (4,638)	
Former (0 drinks in past year)	9.7 (311)	6.9 (2,221)	
Current (>0 drinks in past year)	78.1 (2,087)	79.6 (19,985)	
Type of alcohol, if any			0.01
Beer only	43.8 (235)	43.5 (2,828)	
Wine only	22.5 (103)	23.3 (1,209)	
Liquor only	24.8 (110)	17.9 (1,103)	
Other/combination	8.9 (69)	15.3 (946)	

Values are expressed as weighted means ± SE or % (unweighted n). Abbreviations: BMI = body mass index, KS = kidney stone.

Among current drinkers, 235 (43.8%) stone formers drank beer, 103 (22.5%) drank wine, and 110 (24.8%) drank liquor as compared to 2,828 (43.5%) non-stone formers who drank beer, 1,209 (23.3%) drank wine, and 1,103 (17.9%) drank liquor.

In univariate analysis of alcohol types, consumption of only beer or only wine was associated with lower odds of prevalent KS when compared to never drinkers or current drinkers who did not report alcohol by dietary recall. These associations remained after adjustment for age, sex, race, BMI, histories of hypertension, diabetes, thiazide use, cigarette smoking, dietary intakes of calorie, protein, fluid without alcohol contribution, sodium, potassium, and calcium (Table 2).

Table 2. Odds ratios of prevalent kidney stone according to type of alcohol.

	Unadjusted Model		Adjusted Model 1		Adjusted Model 2		Adjusted Model 3	
	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value
Never/Currently none	REF		REF		REF		REF	
Beer only	0.79 (0.64-0.97)	0.02	0.76 (0.62-0.94)	0.01	0.79 (0.64-0.97)	0.03	0.76 (0.61-0.94)	0.01
Wine only	0.75 (0.58-0.99)	0.04	0.64 (0.49-0.84)	0.001	0.74 (0.57-0.96)	0.02	0.75 (0.59-0.96)	0.03
Liquor only	1.08 (0.77-1.52)	0.63	1.04 (0.74-1.47)	0.82	1.05 (0.75-1.49)	0.76	0.99 (0.69-1.42)	0.97

Abbreviations: OR = odds ratio, CI = confidence interval. REF= never drinkers and current drinkers who did not report alcohol intake on day 1 recall. Model 1: adjusted for demographics. Model 2: adjusted for BMI, histories of hypertension, diabetes, thiazide use, cigarette smoking, in addition to model 1. Model 3: adjusted for dietary intakes of calorie, protein, fluid (minus alcohol contribution), sodium, potassium, and calcium in addition to model 2.

We also evaluated KS risk among current drinkers with exclusive intake of alcohol modeled as a categorical variable in tertiles or quartiles after rounding to the nearest multiple of one standard drink (14g). The multivariate-adjusted OR for stone formation among participants drinking 1-≤14g/day of beer was 1.41 (95% CI: 0.97-2.05), >14-28g/day was 0.65 (95% CI: 0.42-1.00), >28-56g/day was 0.60 (95% CI: 0.39-0.93), and >56g/day was 0.34 (95% CI: 0.20-0.57) compared to those who did not drink beer (Table 3).

Table 3. Odds ratios of prevalent kidney stone according to exclusive **beer** intake **among current drinkers**.

Beer	Unadjusted Model		Adjusted Model 1		Adjusted Model 2		Adjusted Model 3	
	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value
0-<1g	REF		REF		REF		REF	
1-≤ 14g	1.46 (1.00-2.15)	0.05	1.35 (0.93-1.96)	0.11	1.45 (1.00-2.12)	0.05	1.41 (0.97-2.05)	0.07
>14-28g	0.67 (0.44-1.01)	0.06	0.65 (0.43-0.99)	0.04	0.66 (0.43-1.02)	0.06	0.65 (0.42-1.00)	0.05
>28-56g	0.60 (0.40-0.91)	0.02	0.61 (0.40-0.92)	0.02	0.64 (0.41-0.98)	0.04	0.60 (0.39-0.93)	0.02
>56g	0.39 (0.24-0.63)	<0.001	0.38 (0.24-0.62)	<0.001	0.38 (0.24-0.62)	<0.001	0.34 (0.20-0.57)	<0.001

Abbreviations: OR = odds ratio, CI = confidence interval. REF = No alcohol intake on day 1 recall or less than 1g of beer only. Model 1: adjusted for demographics. Model 2: adjusted for BMI, histories of hypertension, diabetes, thiazide use, cigarette smoking, in addition to model 1. Model 3: adjusted for dietary intakes of calorie, protein, fluid (minus alcohol contribution), sodium, potassium, and calcium in addition to model 2. 18,532 records included.

Interestingly, the multivariate-adjusted OR for stone formation was 1.14 (95% CI: 0.72-1.83) among participants drinking 1-≤14g/day of wine, 0.54 (95% CI: 0.36–0.81) among those drinking >14-28g/day, and 0.85 (95% CI: 0.54–1.33) among those drinking >28g/day compared to those who did not drink wine (Table 4), showing a unique effect of moderate wine intake on KS risk.

Table 4. Odds ratios of prevalent kidney stone according to exclusive **wine** intake **among current drinkers**.

Wine	Unadjusted Model		Adjusted Model 1		Adjusted Model 2		Adjusted Model 3	
	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value
0-<1g	REF		REF		REF		REF	
1-≤ 14g	0.98 (0.62-1.53)	0.91	0.90 (0.56-1.44)	0.66	1.10 (0.69-1.77)	0.68	1.14 (0.72-1.83)	0.57
>14-28g	0.52 (0.35-0.77)	0.001	0.43 (0.29-0.64)	<0.001	0.53 (0.36-0.78)	0.002	0.54 (0.36-0.81)	0.003
>28g	0.86 (0.52-1.44)	0.56	0.77 (0.47-1.27)	0.31	0.87 (0.54-1.4)	0.56	0.85 (0.54-1.33)	0.47

Abbreviations: OR = odds ratio, CI = confidence interval. REF = No alcohol intake on day 1 recall or less than 1g of wine only. Model 1: adjusted for demographics. Model 2: adjusted for BMI, histories of hypertension, diabetes, thiazide use, cigarette smoking, in addition to model 1. Model 3: adjusted for dietary intakes of calories, protein, fluid (minus alcohol contribution), sodium, potassium, and calcium in addition to model 2. 16,781 records included.

When beer and wine consumption were examined as continuous variables by restricted cubic splines, both exhibited non-linear associations with prevalent KS (Figures 2 and 3).

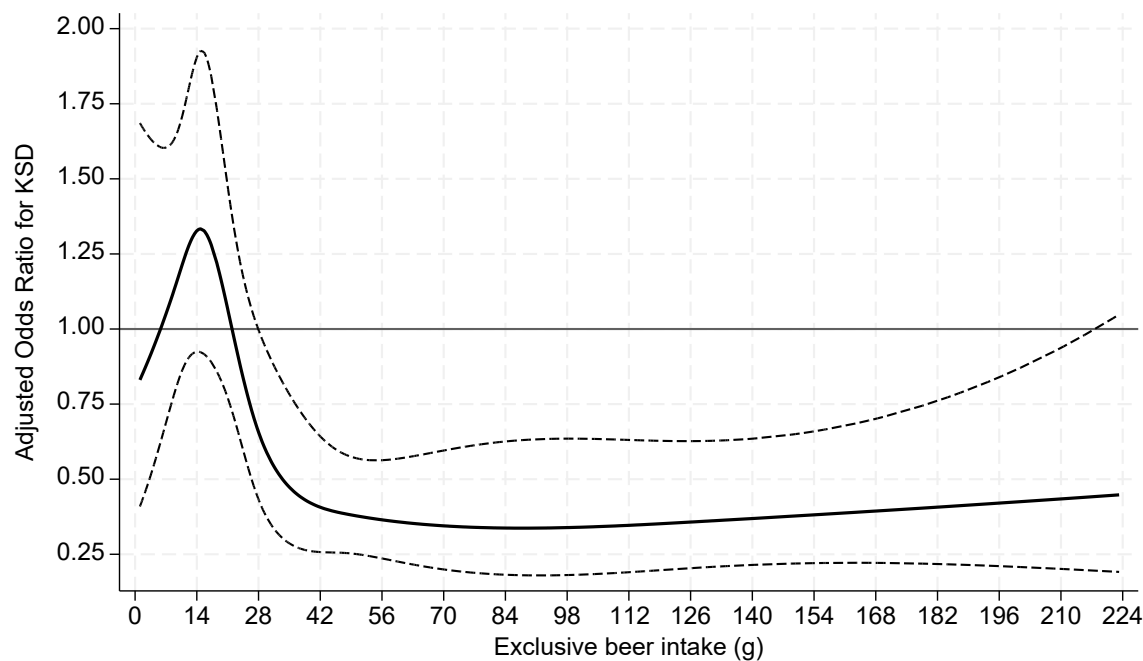


Figure 2. Odds ratios of prevalent kidney stone by restricted cubic splines for exclusive **beer** intake among **current drinkers**. Knots at 9.4, 14.9, 28.1, 47.95, and 126.4 g of beer with a binary indicator variable for 0-<1g. P=0.02 for test of linearity. The x-axis was truncated at 224 g, omitting 27 respondents with intake >224-832 g.

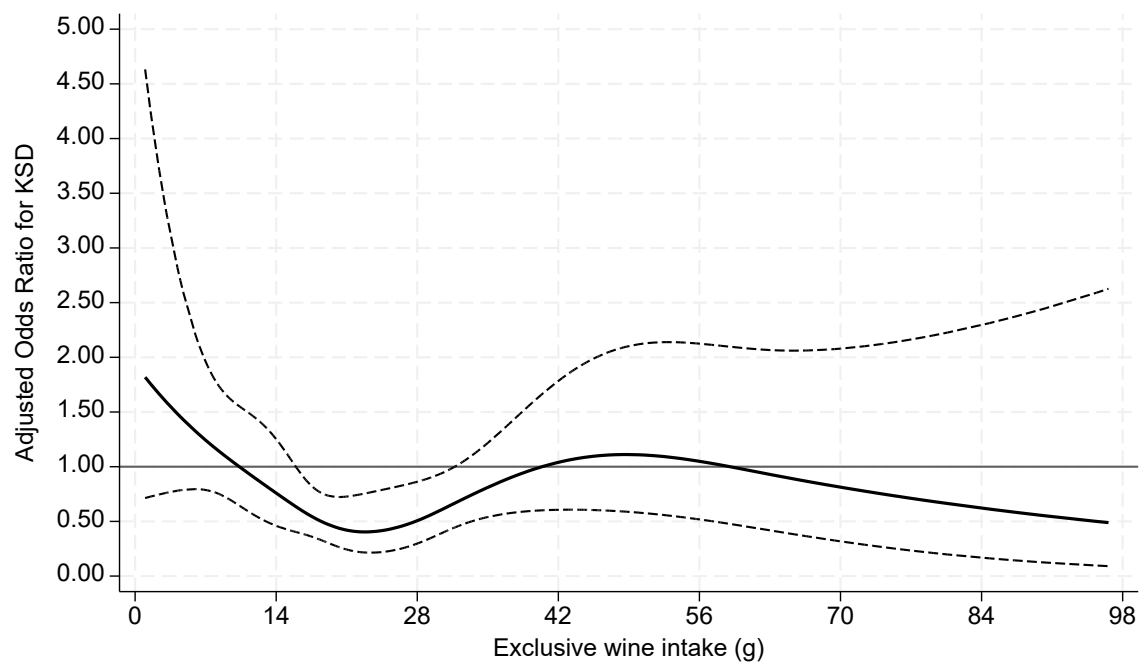


Figure 3. Odds ratios of prevalent kidney stone by restricted cubic splines for exclusive **wine** intake among **current drinkers**. Knots at 7.2, 15.1, 21.6, 31.2, and 63.6 g of wine with a binary indicator variable for 0-<1g. P=0.04 for test of linearity. The x-axis was truncated at 98 g, omitting 14 respondents with intake >98-167 g.

Exclusive liquor consumption was not associated with prevalent KS when analyzed as a categorical or continuous variable (Table 5 and Figure 4).

Table 5. Odds ratios of prevalent kidney stone according to exclusive **liquor** intake **among current drinkers**.

Unadjusted Model			Adjusted Model 1		Adjusted Model 2		Adjusted Model 3	
Liquor	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value
0-<1g	REF		REF		REF		REF	
1-≤28g	1.18 (0.70-1.99)	0.53	1.15 (0.67-1.96)	0.62	1.20 (0.71-2.02)	0.49	1.16 (0.69-1.97)	0.58
>28g	0.97 (0.65-1.44)	0.87	0.94 (0.62-1.40)	0.75	0.94 (0.62-1.43)	0.79	0.85 (0.56-1.30)	0.45

Abbreviations: OR = odds ratio, CI = confidence interval. REF = No alcohol intake on day 1 recall or less than 1g of liquor only. Model 1: adjusted for demographics. Model 2: adjusted for BMI, histories of hypertension, diabetes, thiazide use, cigarette smoking, in addition to model 1. Model 3: adjusted for dietary intakes of calorie, protein, fluid (minus alcohol contribution), sodium, potassium, and calcium in addition to model 2. 16,682 records included.

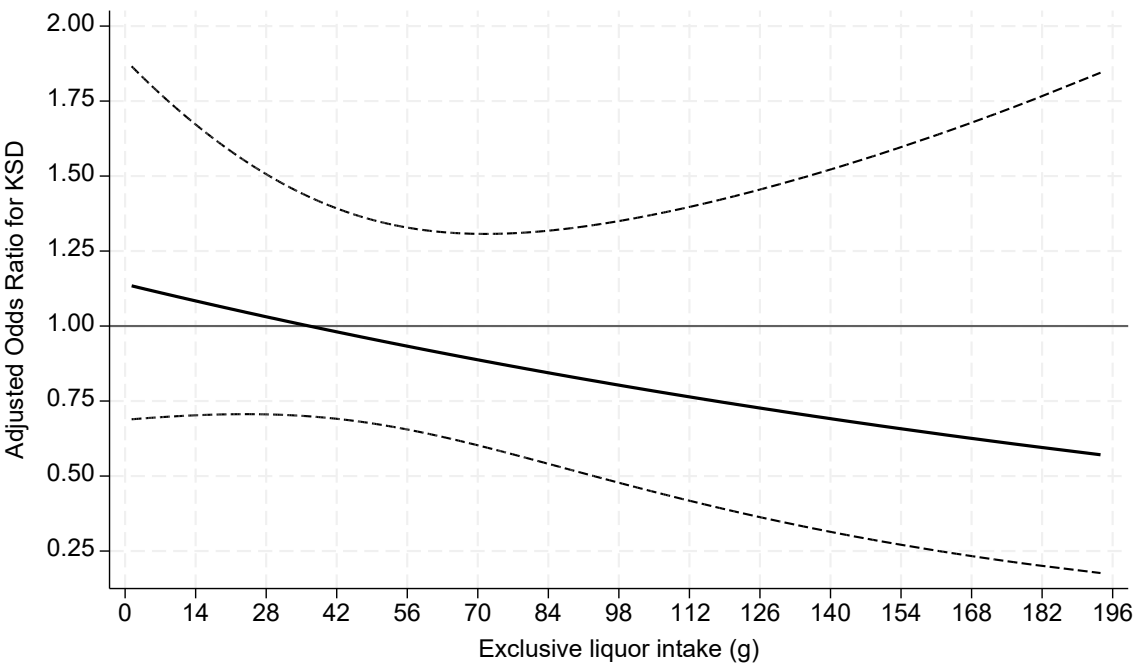


Figure 4. Odds ratios of prevalent kidney stone for continuous **liquor** intake **among current drinkers**. P = 0.29 for test of linearity. The x-axis was truncated at 196 g, omitting 17 respondents with intake >196-404 g.

Discussion

Alcohol has been implicated in many health problems including cardiovascular disease, liver damage, cancer and behavioral disorders. Its role in KS formation, however, remains unclear. Here, we analyzed a large cohort of US population and showed a strong protective effect from beer and wine intakes on the odds of prevalent KS. To the best of our knowledge, this is the largest population study examining specifically the role and type of alcohol intake on risk of KS formation independent of other known confounders.

KS formation occurs when stone forming elements, most commonly calcium, phosphorus, oxalate, and uric acid reach a supersaturation point followed by nucleation, aggregation, and growth. While some including citric acid and magnesium [31–33] inhibit KS formation, others including zinc [34–38] have been implicated as a promoter. Alcohol, in turn, has been shown to either directly affect the concentrations of stone forming elements or indirectly affect their promoters and inhibitors thereby potentially playing a role in KS formation. Alcohol can lead to suppressed osteoblast activity [39] and increased osteoclastic activity [40] thereby leading to bone loss and increasing urinary

calcium [5–7] and phosphorus [7]. Together these effects seem to increase the risk of KS formation. However, alcohol also promotes urinary magnesium excretion [7,12–15,41], and increases urine output [16–19], both can be protective against stone formation. The effect of these biochemical changes in urine constituents have raised uncertainty regarding the overall effect of alcohol on KS risk.

Beer has limited alcohol content, but contains large amount of guanosine [42] which is metabolized to uric acid [43,44]. As a result, higher beer intake may lead to an increased urinary excretion of uric acid and promote KS formation [11,42,43]. While the effect of purine in beer on stone risk has not been studied exclusively, water, which accounts for 95% of content in beer [28], is known to reduce the supersaturations of stone forming elements and crystal formation [45,46]. Indeed, optimal water intake is proven to be an effective intervention for KS prevention [47,48]. We found that beer intake is strongly associated with a reduced risk of prevalent KS by as much as 24%. Our findings are consistent with what has been previously reported from other large population-based studies [3,20,24]. It should be noted that our study found a direct dose response relationship between the number of standard drinks consumed and KS risk with participants drinking moderate (>14-28g) to large (>28-56g) amount of beer every day having a progressively lowered risk of KS formation. This protective effect is likely not driven simply by the high water content in beer, as the strong association between beer and prevalent KS persisted after we adjusted for water content from beer in the sensitivity analysis (Supplemental table 1). Indeed, beer water contains a wide range of mineral and other ingredients that could influence KS risk [49].

Despite having high water content, wine has unique features that may affect KS risk. Wine consumers tend to have a higher excretion of urinary calcium [50], phosphorus [50], zinc [51], and a lower urinary excretion of magnesium [50] compared to liquor consumers and this effect remained in people consuming dealcoholized wine [50] suggesting a role of congeners in wine affecting KS risk. Even though the alcohol component has an aquaresis effect [16–19], the volume of wine consumed is usually low. Therefore, wine consumption could theoretically increase KS risk. In this study, we found a 25% reduction in KS risk among participants who drank wine. But this protection appeared to be modified by the amount of wine intake. Drinking moderate amount (14g-28g /day) of wine was associated with reduced risk of KS disease, whereas no protective effects were found among low (<14g/day) or heavy (>28g/day) drinkers suggesting a U-shaped response. Previous studies have reported a reduced risk of prevalent KS formation by 39% in men [3], and 59% in women [52] and this was confirmed by Ferraro et al [24] in a prospective study. However, Curhan et al also reported a dose related linear reduction in risk of KS in both male [3] and female [52] wine drinkers. The cause of this discrepant finding among higher wine drinkers is not clear. It might reflect the differences in study cohort and analysis methods. In addition, the studies by Curhan et al did not adjust for dietary fluid intake. Lastly, the regression analyses in this study included many other essential confounders of KS disease. Since the water content in wine can be as high as 88% [28] and there are many other unique ingredients in wine [53], it is feasible that moderate wine drinking is associated with a more favorable balance between stone promoting factors and inhibitors.

Liquor, which is a concentrated form of ethanol, leads to increased urinary calcium [5–7], phosphorus [7], and uric acid [54] raising possibility of harmful effect on KS formation. However, it also leads to increased urinary magnesium [7,12–15,41] and while low in volume itself when consumed, it does lead to suppression of vasopressin [55,56] which in turn results in increased urine volume suggesting a protective role in KS formation. We found that drinking any amount of liquor has no association with risk of prevalent KS disease, reflecting a well-balanced effects from KS promoters and inhibitors. Our finding is consistent with what has been reported by Goldfarb et al in a study of Vietnam Era Twin Registry [20,25]. However, Wang et al [21] in their large population study, found that drinking any amount of liquor had a reduced risk of prevalent KS. This discrepancy could be due to the differences in study cohort.

Our study has limitations. First, this is a cross-sectional study and conclusions regarding causal or temporal relationship cannot be made. Second, it is possible that stone formers who are aware about their disease increase their alcohol intake to increase fluid volume. However, such practice is

generally not recommended due to the lack of solid clinical evidence and concerns of overall negative impact to health especially considering a high prevalence of hypertension among KS formers. Third, KS diagnosis was self-reported during interviews. Therefore, misclassification is possible as stone formers may have a recall bias and some may not be aware that they had a stone. Furthermore, alcohol consumers may underestimate and underreport the amount of alcohol consumed. Regardless, this should be biased toward null. Finally, we also could not evaluate the effect of alcohol intake on urinary risk profile or the type of stone since these data were not available in the NHANES.

Conclusions

Our study demonstrated that moderate to high beer and moderate wine intake is associated with a reduced prevalence of KS disease. Future prospective studies are needed to clarify the causal relationship and underlying mechanisms.

Author Contributions: Conceptualization, J.T., M.C.; Methodology, J.T., C.R.; Software, C.R.; Validation, J.T., C.R.; Data curation, C.R.; Analysis, C.R.; Writing -original draft preparation, S.S., C.R., J.T.; Writing-review and editing, J.T., M.C., C.R., S.S. CR and SS contributed equally to this study All authors have read and agreed and to the published version of the manuscript.

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Conflicts of Interest: Authors declare that they have no competing interests.

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