

Experimental and observational studies on alcohol use and dietary intake: a systematic review

Jenna R. Cummings^{1,2}  | Ashley N. Gearhardt^{1,2} | Lara A. Ray³ | Alyssa K. Choi³ |
A. Janet Tomiyama³

¹Department of Psychology, University of Michigan, Ann Arbor, Michigan

²Center for Human Growth and Development, University of Michigan, Ann Arbor, Michigan

³Department of Psychology, University of California, Los Angeles, Los Angeles, California

Correspondence

Jenna R. Cummings, 1465 East Hall,
530 Church St., Ann Arbor, MI 48109.
Email: jennarc@umich.edu

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Summary

The scientific literature on links among alcohol use, total energy intake, cardiometabolic disease and obesity is conflicting. To clarify the link between alcohol use and cardiometabolic health, this systematic review (PROSPERO CRD42016039308A) uses PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to synthesize how alcohol use affects dietary intake (carbohydrate, fat and protein intake) in humans. A search of Google Scholar, PsycINFO and PubMed from June 2016–March 2019 yielded 30 qualified studies. Experimental and observational studies allowed for inferences about effects of a single drinking occasion and of frequent drinking, respectively. Alcohol quantities were standardized according to the 2015–2020 Dietary Guidelines for Americans. On average, methodological quality of the studies was medium strength. Results indicated that a single occasion of light and moderate drinking as well as frequent light and moderate drinking were linked to greater fat and protein intake, albeit the majority of studies did not detect differences in dietary intake due to these drinking behaviours. Frequent heavy drinking, on the other hand, was linked to less carbohydrate intake in the majority of studies. Overall, alcohol use does not appear to uniformly affect diet but instead appears to affect intake of specific macronutrients in a dose-dependent manner, most consistently decreasing carbohydrate intake with heavier use.

KEYWORDS

alcohol, dietary intake, PROSPERO CRD42016039308, systematic review

1 | INTRODUCTION

The scientific literature on alcohol use and cardiometabolic health is conflicting. On one hand, any alcohol use causes greater total energy intake, which may harm cardiometabolic health.^{1,2} On the other hand, frequent moderate drinking – about 1–2 U.S. standard drinks/day for women and about 2–3 U.S. standard drinks/day for men – is associated with reduced risk of clinical events including atherosclerosis, myocardial infarction, ischemic stroke, and type II diabetes.³ Moreover, the association between alcohol use and obesity [body mass index (BMI) ≥ 30] is complex. Greater alcohol use is associated with

higher BMI in some studies, but lower BMI in other studies.⁴ Higher versus lower BMI is generally associated with poorer cardiometabolic health,⁵ although BMI categories can misclassify cardiometabolic health status.⁶ In sum, the scientific literature points to potential cardiometabolic health benefits and negative consequences of alcohol use, leaving clinicians and the public unclear as to how this behaviour fits into the prevention and treatment of cardiometabolic diseases and obesity.

One novel approach to clarifying the link between alcohol use and cardiometabolic health is to evaluate how alcohol use changes diet or the foods someone eats. Indeed, a growing movement in

cardiometabolic disease and obesity research emphasizes the importance of investigating the amount of different macronutrients (carbohydrates, fat, and protein) in an individual's diet, not just total energy intake, to understand disease risk.⁷ Carefully controlled experimental studies show that diets with different macronutrient compositions differentially affect energy expenditure even when calorically equivalent.^{8,9} Moreover, experimental and observational research indicates that diets consisting of greater intake of refined carbohydrates (e.g. breads and pastas) and fat heighten risk for cardiometabolic disease and obesity irrespective of total energy intake.^{7,10} Experimental research also indicates that greater intake of protein facilitates weight loss,¹⁰ and weight loss can predict reduced risk for cardiometabolic disease.¹¹; *c.f.*¹² Systematically evaluating the current evidence on whether alcohol use shifts dietary intake will therefore be clinically informative. For instance, does alcohol use confer cardiometabolic risk through increasing intake of refined carbohydrates and fat and decreasing protein intake? Or might the cardiometabolic consequences of alcohol use be attenuated through increased protein intake and decreased intake of refined carbohydrates and fat? Additionally, this evaluation will improve upon existing clinical and public guidelines on alcohol use and dietary intake,¹³ which is important because a high prevalence of alcohol use exists globally.¹⁴

No prior paper has synthesized and critiqued the scientific literature on whether alcohol use shifts dietary intake. The current paper therefore fills this gap by including a systematic review of the empirical literature investigating the effect of alcohol use on dietary intake in humans. Systematic reviews have strength because they use explicit methods to identify, select and appraise studies unlike narrative reviews, which can be affected by bias.¹⁵ Additionally, a systematic review can include studies that utilize different designs.¹⁵ This is particularly important for investigating how alcohol use affects dietary intake because, although experimental studies with alcohol administration allow for causal conclusions, these studies are practically constrained in multiple ways: to lighter alcohol quantities, to one drinking and eating occasion and to a laboratory setting.² Observational studies, on the other hand, are well suited to capture naturalistic patterns of drinking including frequent intake of heavier alcohol quantities and to test how these drinking behaviours are related to patterns of dietary intake. Given that cardiometabolic diseases and obesity develop after long-term shifts in dietary intake,¹⁶ evaluating associations between frequent drinking and dietary intake patterns has especially strong clinical implication. Moreover, evaluating the specific alcohol quantities and frequencies linked with changes in dietary intake will improve precision in clinical and public discourse on the topic.

In order for associations between alcohol use and dietary intake to be interpreted in the context of specific alcohol quantities and frequencies, alcohol use was standardized across studies into light drinking, moderate drinking and heavy drinking based on the 2015-2020 Dietary Guidelines for Americans.¹³ This standardization accounts for sex differences in alcohol pharmacokinetics. Also, the drinking behaviours tested in experimental studies were subsequently referenced to as single drinking occasions, given that experimental studies administered an alcoholic beverage at one drinking occasion in a short time

period. In contrast, the drinking behaviours evaluated in observational studies were subsequently referenced to as frequent drinking behaviours, given that observational studies measured typical patterns of drinking engaged in a longer time period (e.g. past 12 months). Changes in dietary intake were assessed by parsing out the influence of alcohol use on intake of carbohydrates versus fat versus protein. Given that greater proportional intake of refined carbohydrates not unrefined carbohydrates (e.g. fruits and vegetables) heightens risk for cardiometabolic diseases and obesity,¹⁰ the influence of alcohol use on intake of refined carbohydrates versus unrefined carbohydrates was further delineated. Finally, where possible, results were compared across male and female participants to identify potential moderation by biological sex.

2 | METHOD

2.1 | Search strategy

The systematic review was pre-registered at PROSPERO (CRD42016039308A) and conducted in accordance to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.¹⁵ The search for eligible studies occurred from 1 June 2016 through 28 February 2018 and was updated in March 2019. Databases included: (a) Google Scholar, PsycINFO and PubMed; (b) reference sections of relevant articles; and (c) tables of contents of *Appetite*, *Eating Behaviors*, *Health Psychology*, *Journal of Health Psychology* and *Journal of Studies on Alcohol and Drugs*. Search strings included 'alcohol' AND 'eating'; 'alcohol' AND 'food' AND 'intake'; 'alcohol' AND 'nutrition'; and 'alcohol' AND 'diet'.

2.2 | Inclusion criteria

Inclusionary study characteristics were: (a) included at least one measure of alcohol use (e.g. alcohol preload, 24-hour dietary recall); (b) included at least one measure of dietary intake (e.g. *ad libitum* food intake, 24-hour dietary recall); (c) included an explicit *a priori* investigation of how alcohol use impacts dietary intake; and (d) conducted in humans. Inclusionary report characteristics were reported in English. Studies were not excluded based on publication year (publication years of included studies ranged from 1985-2017).

2.3 | Study selection

Five trained research assistants conducted blind literature reviews. They examined titles and abstracts from the search, recorded citations, checked for duplicate inclusion, obtained relevant articles in full, reviewed the content of the full article and labelled each citation as eligible or ineligible. One lead research assistant verified eligibility. In

*Most included studies quantified dietary intake by (1) calculating the percentage of total energy intake that macronutrients contributed to and (2) calculating the number of calories and/or grams of high-carbohydrate, high-fat, and high-protein foods eaten. However, some included studies quantified dietary intake by only calculating one of these. For consistency, we refer to all quantifications as 'carbohydrate', 'fat', and 'protein' intake.

the case of discrepancies, the lead research assistant and the first author adjudicated the disagreement.

2.4 | Data extraction

The lead research assistant extracted data on: (a) sample characteristics including information on sample size, biological sex, age and BMI; (b) study methods including study design and the alcohol use and dietary intake measures; and (c) study results. The first author reviewed these data and fully read each eligible study in alphabetic order by citation.

Alcohol use was standardized as light, moderate or heavy drinking based on the 2015-2020 Dietary Guidelines for Americans.¹³ For female participants, light drinking was defined as <1 standard drink/day (operationally 0-0.49 standard drinks/day), moderate drinking was defined as 1 standard drink/day (operationally 0.5-1.49 standard drinks/day) and heavy drinking was defined as ≥ 2 standard drinks/day (operationally ≥ 1.5 standard drinks/day).¹⁷ For male participants, light drinking was defined as <1 standard drink/day (operationally 0-0.49 standard drinks/day), moderate drinking was defined as 1-2 standard drinks/day (operationally 0.5-2.49 standard drinks/day) and heavy drinking was defined as ≥ 3 standard drinks/day (operationally ≥ 2.5

standard drinks/day).¹⁷ In studies wherein female and male participants were not separated for analysis, alcohol use was standardized based on the guidelines for female participants to provide the most conservative estimation. Alcohol use was also referenced to as a single drinking occasion or frequent drinking based on study design (experimental = a single drinking occasion; observational = frequent drinking).

A U.S. standard drink contains roughly 14 g of alcohol, which is found in about 12 oz. of regular beer, 5 oz. of wine, and 1.5 oz. of distilled spirits. However, internationally, there is variation in the alcohol grams that determine a 'standard' drink. For example, most European and non-U.S. English-speaking nations identify standard drinks to contain roughly 10 g of alcohol. Therefore, when generalizing and applying these results in non-U.S. countries, this difference should be acknowledged.

2.5 | Assessment of methodological quality

The first author assessed risk of bias in individual studies according to the Downs and Black Quality Index scoring system¹⁸ with external review from the lead research assistant. In this system, authors use a validated checklist to assess the quality of randomized and non-randomized studies. The system consists of five subscales that

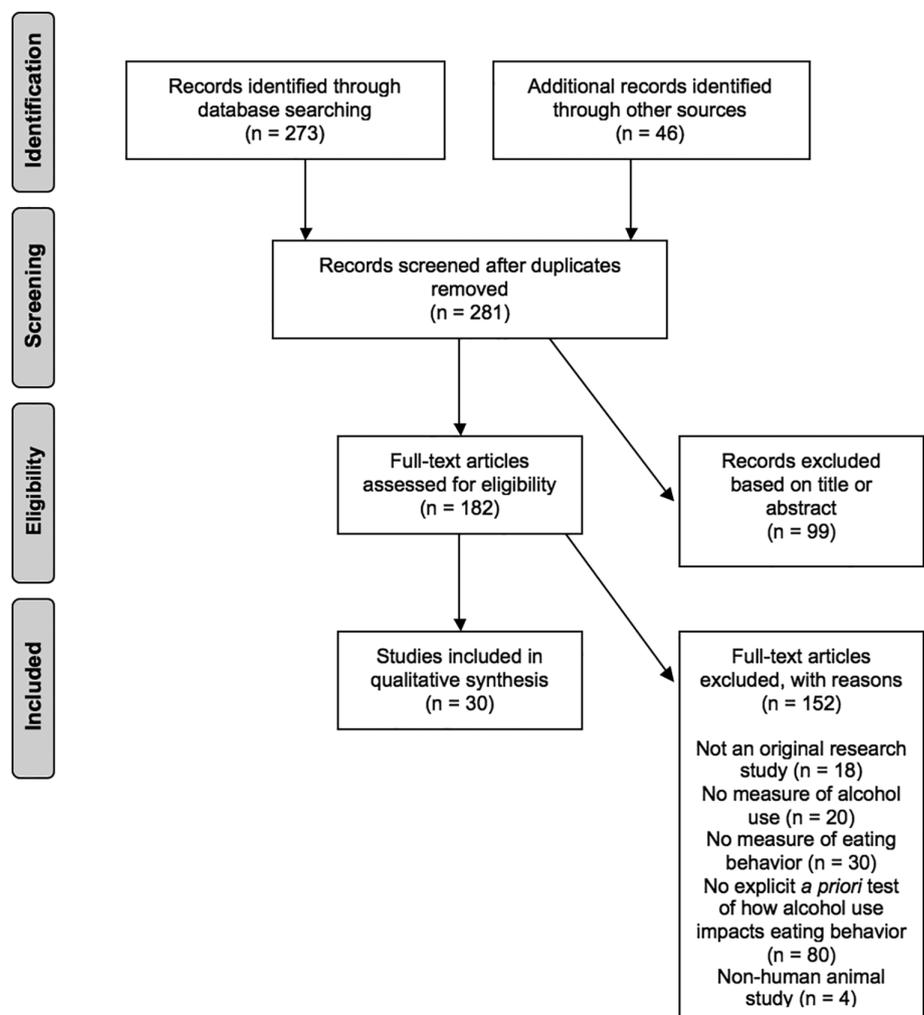


FIGURE 1 PRISMA flow chart for qualified studies

TABLE 1 Summary of results for included studies

Study	Quality Index score	Sample size	BMI M (SD)	Sex	Alcohol use	Refined carbohydrate intake	Fat intake	Protein intake	Unrefined carbohydrate intake
Experimental studies (single drinking occasion)									
Caton et al. (2004)	17	12	Not reported; inclusion criterion < 26	100% men	None Light Moderate	Referent <i>ns</i> <i>ns</i>	Referent <i>ns</i> <i>ns</i>	Referent <i>ns</i> <i>ns</i>	Referent <i>ns</i> <i>ns</i>
Caton et al. (2005)	19	12	Not reported; inclusion criterion < 27	100% men	None Moderate	Referent +	Referent +	Referent +	
Christian-sen et al. (2016)	21	60	20.82 (2.82)	100% women	None Heavy	Referent +	Referent +		
Eiler et al. (2015)	19	35	21.70 (1.80)	100% women	None Heavy	Referent +	Referent +	Referent +	
Hetherington et al. (2001)	18	26	Not reported; Inclusion criterion 20-27	100% men	None Moderate	Referent +	Referent +	Referent +	Referent +
Hofmann et al. (2008)	18	63	21.80 (2.18)	100% women	None Light	Referent +	Referent +		
Hollister (1971)	13	24	Not reported	95.83% men; 4.17% women	None Heavy	Referent <i>ns</i>	Referent <i>ns</i>	Referent <i>ns</i>	
Mattes (1996)	18	16	Not reported	50% men; 50% women	None Light Moderate	Referent <i>ns</i> <i>ns</i>	Referent + +	Referent + +	Referent <i>ns</i> <i>ns</i>
Ouwens et al. (2003)	16	116	23.10 (2.90)	100% women	None Heavy	Referent <i>ns</i>	Referent <i>ns</i>		
Polivy et al. (1976a)	15	40	Not reported	100% women	None Heavy	Referent <i>ns</i>	Referent <i>ns</i>		
Polivy et al. (1976b)	15	55	Not reported	100% women	None Heavy	Referent <i>ns</i>	Referent <i>ns</i>		
Poppit et al. (1996)	18	20	23.00 (2.80)	100% women	None Heavy	Referent <i>ns</i>	Referent <i>ns</i>	Referent <i>ns</i>	
Raben et al. (2003)	18	19	22.10 (0.40)	52.63% men; 47.37% women	None Moderate	Referent <i>ns</i>	Referent <i>ns</i>	Referent <i>ns</i>	Referent <i>ns</i>
Rose et al. (2015)	17	114	Not reported	57.89% women; 42.11% men	None Heavy	Referent <i>ns</i>	Referent <i>ns</i>		Referent <i>ns</i>
Schrieks et al. (2015)	18	23	23.00 (0.10)	100% men	None Moderate	Referent <i>ns</i>	Referent +	Referent <i>ns</i>	Referent
Yeomans et al. (1999)	15	22	23.20 (0.80)	100% men	None Moderate	Referent <i>ns</i>	Referent <i>ns</i>	Referent <i>ns</i>	
Yeomans et al. (2002)	18	18	22.64 (SD not reported)	100% men	None Moderate	Referent <i>ns</i>	Referent <i>ns</i>	Referent <i>ns</i>	
Yeomans (2010)	17	40	22.80 (SD not reported)	100% women	None Moderate	Referent +	Referent +	Referent +	Referent +

(Continues)

TABLE 1 (Continued)

Study	Quality Index score	Sample size	BMI M (SD)	Sex	Alcohol use	Refined carbohydrate intake	Fat intake	Protein intake	Unrefined carbohydrate intake
Observational studies (frequent drinking)									
Butler et al. (2017)	21	14,375	Not reported	48.27% women 51.73% men	Men				
					None	+	ns	ns	+
					Light	+	+	ns	+
					Moderate	Referent	Referent	Referent	Referent
					Heavy	-	-	-	-
					Women				
					None	+	+	ns	+
					Light	+	+	ns	+
					Moderate	Referent	Referent	Referent	Referent
					Heavy	-	-	ns	-
Colditz et al. (1991)	20	138,031	Not reported	64.87% women 35.13% men	Men				
					None	Referent	Referent	Referent	Referent
					Light	ns	ns	ns	ns
					Moderate	ns	ns	ns	ns
					Heavy	-	+	ns	-
					Women				
					None	Referent	Referent	Referent	Referent
					Light	ns	ns	ns	ns
					Moderate	ns	ns	ns	ns
					Heavy	-	ns	ns	-
Cummings et al. (2017)	17	2,379	23.41 (5.39)	100% women	None	Referent	Referent		
					Light	-	ns		
					Moderate	-	ns		
					Heavy	-	ns		
de Castro et al. (1990)	15	92	21.90 (SD not reported)	75% women 25% men	None	Referent	Referent	Referent	Referent
					Light	ns	ns	ns	ns
					Moderate	ns	ns	+	ns
Gruchow et al. (1985)	19	10,428	Not reported	59% women 41% men	Men				
					None	Referent	Referent	Referent	Referent
					Light	-	+	+	-
					Moderate	-	ns	ns	-
					Heavy	-	ns	ns	-
					Women				
					None	Referent	Referent	Referent	Referent
					Light	ns	+	+	ns
					Moderate	-	ns	ns	-
					Heavy	-	ns	ns	-
Hillers et al. (1985)	17	179	Not reported	100% men	None	Referent	Referent	Referent	Referent
					Light	ns	ns	ns	ns
					Moderate	ns	ns	ns	ns
					Heavy	-	-	-	-
Ishikawa et al. (2017)	21	711	Not reported	100% men	Moderate	Referent	Referent	Referent	Referent
					Heavy	-	-	-	-

(Continues)

TABLE 1 (Continued)

Study	Quality Index score	Sample size	BMI M (SD)	Sex	Alcohol use	Refined carbohydrate intake	Fat intake	Protein intake	Unrefined carbohydrate intake
Kesse et al. (2001)	19	72,904	Not reported	100% women	None	Referent	Referent	Referent	Referent
					Light	-	+	ns	-
					Moderate	-	+	ns	-
					Heavy	-	+	+	-
Ma et al. (2000)	22	6,745	Not reported	54.93% men 45.07% women	Men				
					None	Referent	Referent	Referent	Referent
					Light	ns	ns	ns	ns
					Moderate	ns	-	ns	ns
					Heavy	-	-	-	-
					Women				
					None	Referent	Referent	Referent	Referent
					Light	ns	ns	-	ns
Ruf et al. (2005)	21	24,894	Not reported	53.33% women 46.67% men	Men				
					None	Referent	Referent	Referent	Referent
					Light	-	-	-	-
					Moderate	-	-	-	-
					Heavy	-	-	-	-
					Women				
					None	Referent	Referent	Referent	Referent
					Light	-	+	ns	-
Ruidavets et al. (2004)	21	1,100	Not reported	100% men	None	Referent	Referent	Referent	Referent
					Light	ns	ns	ns	ns
					Moderate	-	+	+	-
					Heavy	-	+	+	-
Walmsley et al. (1998)	20	1,198	Not reported	52% men 48% women	None	Referent	Referent		Referent
					Light	+	+		+
					Moderate	+	+		+
					Heavy	-	+		-

Note. For each study, the drinking quantities that were tested are listed. If a drinking quantity (compared with the referent group) was significantly linked with greater intake of a macronutrient, the correspondent table cell is labelled with a plus sign; if significantly linked with less intake, it is labelled with a minus sign; and if a significant difference in intake was not detected, it is labelled 'ns' to indicate non-significance. Significance was determined where $P < .05$. Table cells are blank if the correspondent macronutrient was not tested. For some observational studies, analyses were separated by biological sex; this was noted with the text 'men' and 'women' above those results.

address bias from reporting, external validity, internal validity, confounding or power. For the current review, the power subscale was scored dichotomously as '0' for no report of a power analysis or '1' for a reported power analysis. Thus, total scores could range from 0-27. In the case of discrepancies, the first author and lead research assistant adjudicated the disagreement. One study¹⁹ involved a subset of the authors of the current paper, thus an impartial Ph.D.-level individual with no conflict of interest assessed the risk of bias for that study.

3 | RESULTS

3.1 | Study characteristics

Numbers of studies screened, assessed for eligibility and included in the review (with reasons for exclusions) are provided in Figure 1. The search yielded a final sample of 30 studies.^{17,19-48} Of these, 18 (60%) were experimental and 12 (40%) were observational studies. A total of 273,751 participants were included across studies, of which

195,389 (71%) were female. Twelve (40%) study samples were drawn from the United States, 9 (30%) from England, 2 (7%) from France, 2 (7%) from Germany, 2 (7%) from The Netherlands, 1 (3%) from Scotland, 1 (3%) from Denmark and 1 (3%) from Japan. Average unweighted age across samples was 24.93 ($SD = 5.79$) ranging from 15 to 85+. Average unweighted BMI was 22.37 ($SD = 0.72$) ranging from 16.20 to 29.90. For additional information about the study samples, please see Table 1 and Table S1 in Supporting Information.

Experimental studies administered alcohol at one drinking occasion, allowing participants to drink for 5–30 min. Then, to assess alcohol-induced changes in dietary intake, researchers observed *ad libitum* food intake 10 min to 5 h later. One experiment differed by using 24-h dietary recall to capture dietary intake in the 24 h after alcohol administration.³⁵ Observational studies investigated cross-sectional associations between frequent drinking behaviours and dietary intake in large, nationally representative datasets. However, one study instead assessed longitudinal associations between frequent drinking behaviours and dietary intake,¹⁹ and another aggregated daily diary data.²⁴ Measures of alcohol use and dietary intake in observational studies generally included 24-h dietary recall⁴⁹ and food frequency questionnaires (including questions about alcohol).⁵⁰

3.2 | Methodological quality

Table 1 presents Downs and Black Quality Index scores for each study. On average, the methodological quality of included studies was medium strength ($M = 18.10$, $SD = 2.18$, $Range = 13-22$). Bias from reporting was generally low but several studies did not report one or a few of the following: testing for covariates, estimates of random variability in outcomes, characteristics of participants lost to follow-up, and/or actual probability values.^{19-21,24,27,29,31,35-43,45,46,51,52} Bias from lack of internal validity was also generally low but the majority of experiments did not report an attempt to blind the participants and/or the experimenters measuring outcomes.^{20-22,25,28,30,35-41,44,46-48} Moreover, bias from confounding was low and only evident for observational studies because of no randomization. In contrast, bias from lack of external validity was generally high because the experimental study samples were not from representative sources and the representativeness of the study samples in a few observational studies was unable to be determined.^{19,24} Bias from lack of power was also generally high because only one study in the review reported a power analysis to determine sample size.³⁵

3.3 | Alcohol use and dietary intake

Table 1 presents a summary of the findings from included studies separated by study design/drinking frequency.

3.3.1 | Alcohol use and refined carbohydrate intake

Eighteen experimental studies tested whether a single occasion of drinking affected refined carbohydrate intake from foods such as bread, breadsticks, cake, chips, chocolate, chocolate chip cookies,

chocolate mini-rolls, cookies, crackers, fruit loaf, ice cream, milkshakes, M&Ms, noodles, pasta and tortilla chips. The majority (66.7%) did not detect differences in refined carbohydrate intake after a single occasion of drinking. The minority (33.3%) found that a single occasion of drinking increased refined carbohydrate intake. Experiments that tested a single occasion of light or moderate drinking were more likely to find an increase in refined carbohydrate intake (effect detected in 40% of those experiments) relative to experiments that tested a single occasion of heavy drinking (effect detected in 25% of those experiments). Additionally, 11 observational studies tested whether frequent drinking was associated with refined carbohydrate intake from foods such as added sugars, candies, cereal and chocolate. Some (45.5%) found that frequent light and moderate drinking were associated with less refined carbohydrate intake, some (27.3%) found that these behaviours were not associated with refined carbohydrate intake, and a few (18.2%) found that these behaviours were associated with greater refined carbohydrate intake. The majority (90.9%) found that frequent heavy drinking was associated with less refined carbohydrate intake with the minority (9.1%) finding it was not associated with refined carbohydrate intake.

3.3.2 | Alcohol use and fat intake

Eighteen experimental studies tested whether a single occasion of drinking affected fat intake from foods such as beef, cheese, chips, chocolate, crackers, cream cheese, ham, ice cream, milkshakes, M&Ms, paté, salami and tortilla chips. Most (55.6%) did not detect differences in fat intake after a single occasion of drinking. Some (44.4%) found that a single occasion of drinking increased fat intake. Experiments that tested a single occasion of light or moderate drinking were more likely to find an increase in fat intake (effect detected in 60% of those experiments) relative to experiments that tested a single occasion of heavy drinking (effect detected in 25% of those experiments). In addition, 11 observational studies tested whether frequent drinking was associated with fat intake including intake of eggs, cheese, chocolate, cottage cheese, lamb, milk, processed meat, poultry, unspecified meat, unspecified dairy products, vegetable oil and yogurt. Most (54.5%) found that frequent light and moderate drinking had no association with fat intake, and some (45.5%) found that these behaviours were associated with greater fat intake. Some (36.4%) found that frequent heavy drinking was associated with less fat intake, some (36.4%) found that it was associated with greater fat intake, and a few (27.3%) found that it had no association with fat intake.

3.3.3 | Alcohol use and protein intake

Twelve experimental studies tested whether a single occasion of drinking affected protein intake from foods such as beef, cheese, lean ham, paté, salami, smoked beef, tuna, unspecified meat and yogurt. The majority (58.3%) did not detect differences in protein intake after a single occasion of drinking. The minority (41.7%) found that a single occasion of drinking increased protein intake. Experiments that tested a single occasion of light or moderate drinking were more likely to find

an increase in protein intake (effect detected in 44.4% of those experiments) relative to experiments that tested a single occasion of heavy drinking (effect detected in 33.3% of those experiments). Also, 9 observational studies tested whether frequent drinking was associated with protein intake including intake of eggs, cheese, fish, lamb, processed meat, poultry, seafood, unspecified meat and yogurt. The majority (55.6%) found that frequent light and moderate drinking had no association with protein intake, whereas some (33.3%) found that these behaviours were associated with greater protein intake, and one study (11.1%) found that these behaviours were associated with less protein intake. Some (44.4%) found that frequent heavy drinking was associated with less protein intake, whereas a few (33.3%) found it was not associated with protein intake, and a few (22.2%) found that it was associated with greater protein intake.

3.3.4 | Alcohol use and unrefined carbohydrate intake

Six experimental studies tested whether a single occasion of drinking affected unrefined carbohydrate intake from foods such as cucumbers, grapes, tomatoes and unspecified vegetables. The majority (83.3%) did not detect differences in unrefined carbohydrate intake after a single occasion of drinking. The minority (16.7%) indicated that a single occasion of drinking increased unrefined carbohydrate intake. Specifically, an increase in unrefined carbohydrate intake was only found in an experiment that tested the effect of a single occasion of moderate drinking. Also, 10 observational studies tested whether frequent drinking was associated with unrefined carbohydrate intake from foods such as unspecified fruits, grains and vegetables. Some (40%) indicated that frequent light and moderate drinking were associated with less unrefined carbohydrate intake; others (40%) found these drinking behaviours were not associated with unrefined carbohydrate intake, and a few (20%) indicated that these drinking behaviours were associated with greater unrefined carbohydrate intake. The majority (90%) found that frequent heavy drinking was associated with less unrefined carbohydrate intake with the minority (10%) finding that it was not associated with unrefined carbohydrate intake.

3.4 | Biological sex differences

There was no strong evidence of differences in effects due to biological sex. The likelihood of detecting that a single occasion of drinking stimulated intake of refined carbohydrates, fat, protein or unrefined carbohydrates was equivalent between studies with samples including exclusively female participants (effects detected in 50% of those experiments) and studies with samples including exclusively male participants (effects detected in 50% of those experiments). Of the studies that tested whether frequent drinking was associated with dietary intake and separated results by biological sex, the majority (60%) found consistent results between male and female participants. However, one of these studies (20%) found that heavy drinking was associated with greater fat intake only in male participants, and another (20%) found that light, moderate and heavy drinking were associated

with less fat intake in male participants but greater fat intake in female participants.

3.5 | Potential confounds

Because observational studies provided results on how frequent drinking is related to dietary intake, it is possible that associations between alcohol use and dietary intake may be in part explained by confounding variables. For instance, a widely held belief is that smoking cigarettes can suppress eating (albeit empirical evidence does not support this belief⁵³), and those who engage in frequent heavy drinking sometimes also smoke cigarettes.³⁴ Thus, cigarette smoking may explain why, for instance, frequent heavy drinking was associated with less carbohydrate intake. Yet several of the observational studies ruled out this confound by adjusting for the effect of cigarette smoking in analyses.^{17,23,42,43,45} In one case, frequent light and heavy drinking were associated with greater cigarette smoking but only frequent heavy drinking was associated with less carbohydrate intake³⁴; this is inconsistent with the notion that smoking explains the association between frequent heavy drinking and less carbohydrate intake.

Another possible confounding variable is BMI because BMI can impact the percentage of alcohol in someone's bloodstream, which may alter the influence of certain alcohol quantities on dietary intake.⁵⁴ Three observational studies adjusted for BMI in analyses and found results consistent with studies that did not adjust for BMI.^{23,33,43} However, because the majority of observational studies did not adjust for BMI, it will be important for future research to address this confound. In addition to calculating standard alcoholic drinks, researchers could calculate blood alcohol content based on participant's biological sex and body weight and time course of their drinking.⁵⁴ Lastly, results were found above and beyond the adjustment of many other variables including age,^{17,23,32-34,43,45} age of menarche,¹⁹ chronic disease status,¹⁷ education,^{17,34,43} employment status,³⁴ income,^{19,34} liking of foods,⁴⁰ living area,^{43,45} physical activity,^{17,43} race/ethnicity,^{17,19,34} self-reported health⁴⁵ and biological sex.⁴⁵ This inspires confidence that the documented associations between alcohol use and dietary intake are not largely explained by confounding variables.

4 | DISCUSSION

To help clarify the link between alcohol use and cardiometabolic health, this paper offers the first systematic review of experimental and observational studies investigating how alcohol use affects dietary intake (carbohydrate, protein and fat intake) in humans. The inclusion of experimental and observational studies allowed for inferences about the effects of a single drinking occasion versus frequent drinking. Moreover, alcohol use was standardized into light drinking, moderate drinking and heavy drinking based on the 2015-2020 Dietary Guidelines for Americans to specify alcohol quantities implicated in effects, and this standardization accounts for sex differences in alcohol pharmacokinetics.^{13,17} This review adhered to PRISMA¹⁵ and

Downs and Black Quality Index¹⁸ guidelines to synthesize and critique the existing literature, yielding insights into associations among alcohol use and dietary intake that may guide clinicians and the public.

Although some assume that any alcohol use uniformly affects diet by increasing intake of all foods,⁵⁵ synthesized results from this review do not support this claim. To begin, a single occasion of drinking increased intake of foods, but studies did not consistently detect this effect. Counterintuitively, the effect was more likely to occur in response to a single occasion of light or moderate drinking compared with a single occasion of heavy drinking. Moreover, when an effect was detected, a single occasion of drinking most often increased intake of fat and protein and, to a lesser extent, increased intake of refined and unrefined carbohydrates. This pattern was observed in female and male participants, which suggests there was no moderation by biological sex.

Next, the majority of studies testing the link between frequent drinking and dietary intake observed associations, but these differed by alcohol dose. Similar to the observed effects of a single occasion of light and moderate drinking, frequent light and moderate drinking were linked with greater intake of fat and protein and, to a lesser extent, with greater intake of unrefined and refined carbohydrates. In contrast, frequent heavy drinking was linked with intake of fewer refined and unrefined carbohydrates and, to a lesser extent, with lower protein and fat intake. This pattern was observed in female and male participants, further suggesting of no moderation by biological sex.

Overall, these synthesized results suggest four important conclusions regarding how alcohol use affects dietary intake. The first is that a single occasion of drinking does not reliably shift diet but frequent drinking appears to more consistently influence diet. This is likely because the effect of a single occasion of drinking on dietary intake is small² and only through frequent recurrence does it become larger and more consistently observable. Because cardiometabolic diseases and obesity develop over time,¹⁶ the distinction between transient versus enduring alcohol-induced dietary changes is an important one. Future studies on the effects of alcohol use on dietary intake should prioritize repeated measures or hybrid designs that use experimental paradigms repeated several times across an individual's lifespan.

The second conclusion these results suggest is that alcohol use affects dietary intake in a dose-dependent manner. Most often, alcohol use was linked with greater dietary intake at light and moderate quantities; however, it was not linked with changes in dietary intake or was linked with less dietary intake at heavier quantities. This finding corroborates with a prior meta-analysis that shows that a single occasion of light/moderate drinking (operationally defined as <2 standard drinks) increased total nonalcoholic energy intake whereas a single occasion of 'heavy' drinking (operationally defined as 2-4 standard drinks) had no effect.² Why might alcohol use counterintuitively stimulate dietary intake at light and moderate doses but fail to stimulate or even decrease dietary intake at heavier doses? One potential explanation is that the dose-dependent effects on dietary intake are mediated through changes in dopaminergic activity in the brain.^{19,54} At low doses, alcohol stimulates dopamine release, which may increase

motivation for several rewarding substances including food.⁵⁶ At high doses, however, alcohol saturates neural pathways with dopamine,⁵⁷ which may decrease this motivation. Another potential explanation is that dose-dependent effects on dietary intake coincide with biphasic alcohol effects. At low doses, alcohol causes stimulatory effects (e.g. positive mood),^{58,59} which may increase the motivation for food. At high doses, alcohol causes sedative effects (e.g. nausea and central nervous system depression), which may decrease this motivation.^{58,59} At high doses, alcohol may also cause gastric expansion and satiation,²⁶ which could decrease food intake. Moreover, it is plausible that the different aforementioned mechanisms collectively cause alcohol use to affect dietary intake in a dose-dependent manner. Future research should directly test these dose-dependent mechanisms with advanced measurement methods. For instance, researchers could intravenously administer alcohol to observe subjective and dietary effects across different quantities of alcohol dose.⁶⁰ Also, paradigms are available that would allow for the effect of alcohol use on dietary intake to be observed while using neural imaging methods.⁶¹

The third conclusion these results suggest is that alcohol use differentially affects intake of specific macronutrients. In detail, when alcohol use was linked with greater dietary intake, it was most often intake of fat and protein and least often intake of carbohydrates. In contrast, when alcohol use was linked with less dietary intake, it was most often intake of carbohydrates and least often intake of fat and protein. An evident explanation for the macronutrient-specific pattern of findings is that alcohol itself is a refined carbohydrate (i.e. alcohol is fermented sugar); ingesting it may decrease motivation for carbohydrates and increase motivation for different macronutrients (e.g. fat and protein).⁶² Non-human animal models suggest that this could be mediated by gut-brain processing in response to carbohydrate intake and regulated by several hormones and neurotransmitters including insulin, serotonin and dopamine.⁶² Future research should directly test how these physiological systems are integrated into the seemingly dose-dependent effect of alcohol use on dietary intake in humans.

The fourth conclusion these results suggest is that alcohol use similarly influences dietary intake in female and male individuals. One might have expected biological sex to moderate effects because male and female individuals tend to show different patterns of alcohol¹⁴ and dietary intake (albeit sex differences in dietary intake are inconsistently observed⁶³). In the current review, biological sex differences in alcohol pharmacokinetics were accounted for by sex-specific definitions of light, moderate and heavy drinking, which might explain the lack of moderation. Furthermore, the mechanisms by which alcohol use influences dietary intake appear to be distinct from those explaining biological sex differences in alcohol⁶⁴ and dietary intake⁶³ more generally. Future research might consider testing this explicitly.

The current results should be interpreted in light of limitations. In terms of study-level limitations, on average, methodological quality of the studies was medium strength. Researchers can improve methodological quality by fully disclosing method and statistical choices, using the Downs and Black Quality Index checklist to determine what needs to be reported and pre-registering studies to increase accountability.

In addition, researchers can avoid risk of bias by invoking double-blind procedures or reporting what study information was disclosed to experimenters/participants. It is challenging to achieve experimenter blindness when an experimenter is serving alcoholic beverages/food. However, one approach is having researchers other than the experimenter serve beverages/food. To achieve participant blindness, researchers can deceive participants as to the true nature of the study by providing cover stories. A commonly used and validated cover story is that participants are invited to the lab to taste and rate new drink and food products.⁶⁵ Also, although most studies thoroughly quantified dietary intake by providing the percentage of total energy intake that macronutrients contributed to and/or the number of calories and/or grams of foods eaten, some did not. To reduce variability across studies, researchers should quantify dietary intake in each of the aforementioned ways and/or publicly share data so that other quantifications could be derived in the future. Lastly, this review yielded many studies that were more than a decade old, some more than two decades old and some more than three decades old. Even though there exists a large-enough literature to warrant this review, there are opportunities for future scientific growth and improvement.

Regarding review-level limitations, the current review did not summarize information on effect sizes for results. This was because, as mentioned above, studies were inconsistent in how they quantified dietary intake and also because some studies only presented inferential statistical estimates without effect size information. Thorough quantifications of dietary intake and reporting of effect sizes will allow for meta-analytic methods to be applied in this domain in the future. Another review-level limitation was that three of the current paper's authors were authors of one study included in the review. To reduce the possibility that this biased the review, a Ph.D. individual with no conflict of interest assessed the methodological quality of that study. Also, the selected review eligibility requirements may have limited the scope of included studies. For example, the initial search yielded two studies on dietary intake of those with alcohol use disorder.^{66,67} These studies were ineligible for the review because a diagnosis of alcohol use disorder confounds drinking with other behaviours (e.g. withdrawal symptoms and quit attempts) and because, in some, dietary intake was measured when individuals were undergoing treatment. Understanding how features of alcohol use disorder influence dietary intake – and how this may change during treatment – might nonetheless be of interest to clinicians.

Dietary intake was operationalized as the macronutrient composition of foods because – irrespective of total energy intake – diets consisting of greater proportional intake of refined carbohydrates and fat heighten risk for cardiometabolic disease and obesity and diets consisting of greater protein intake may facilitate weight loss.¹⁰ However, it may have been beneficial to also provide information on how alcohol use was associated with overall diet quality (e.g. summary measure of variety in nutrient intake). Also, greater intake of certain types of fat (i.e. industrial trans fats) may carry the most risk for cardiometabolic disease and obesity,⁶⁸ so it may have benefited the review to further parse fat intake into groups based on type of fat. Likewise, greater proportional intake of ultra-

processed foods (i.e. food-derived substances including sugar-sweetened beverages, sweet/savory packaged snacks, mass-produced breads, 'instant' meals and reconstituted meats) compared with minimally processed foods (i.e. foods obtained directly from plants/animals including fresh fruits and vegetables, eggs and milk) increases risk of cardiometabolic disease and obesity irrespective of total energy intake.^{69,70} The majority of included studies did not provide enough detail so that the association of alcohol use and overall diet quality could be summarized, so that fat intake could be parsed into different groups based on fat type and so that dietary intake could be operationalized based on processing level. Future research on how alcohol use affects diet should provide detail on the type of fat measured and should consider using standardized classification procedures to categorize overall diet quality⁷¹ and food processing levels.⁷²

Overall, alcohol use does not reliably increase intake of food. When alcohol stimulates food intake, the scientific literature suggests it is most often at low doses – whether at a single occasion or frequently – and is specific to stimulation of fat and protein intake. In contrast, the scientific literature suggests that frequent heavy drinking is consistently linked with intake of fewer carbohydrates. Understanding these nuanced effects of alcohol use on dietary intake may improve the precision of clinical and public discourse on the topic and may improve existing cardiometabolic disease and obesity prevention and treatment efforts.¹³ Indeed, findings from this review suggest that alcohol use may confer some risk for cardiometabolic disease and obesity through dietary change. Specifically, a single occasion of and frequent light/moderate drinking may confer risk by increasing fat intake; however, this may be partially offset by increased protein intake. Also, the cardiometabolic consequences of heavy drinking may be diminished due to decreases in refined carbohydrate intake. The latter finding might even explain why greater alcohol use is associated with lower BMI in several studies.⁴ Future research might test this explicitly by testing refined carbohydrate intake as a mediator or moderator of the link between alcohol use and obesity. It is important, however, that any findings suggesting that alcohol use mitigates risk for cardiometabolic disease and obesity through dietary changes be interpreted in the context of the broader literature on the consequences of drinking. Alcohol use increases risk for addiction,⁷³ depression,⁷⁴ interpersonal violence,⁷⁵ several cancers,⁷⁶ liver cirrhosis,⁷⁷ pancreatitis⁷⁸ and unintentional injuries (e.g. vehicle accidents, falls and drowning).⁷⁸

In sum, there are many questions to be answered about the links among alcohol use, dietary intake, cardiometabolic disease and obesity, but the scientific evidence challenges the lay assumption that any alcohol use increases intake of all foods.⁵⁵ Research replicating the observed findings will strengthen the scientific literature and shed light on precise behavioural targets (e.g. light/moderate drinking facilitating a high-fat diet) that may be relevant to those seeking guidance on the cardiometabolic health effects of alcohol use.

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CONFLICT OF INTEREST

None.

ORCID

Jenna R. Cummings  <https://orcid.org/0000-0001-8124-2666>

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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