

Beer, wine, and spirits differentially influence body composition in older white adults—a United Kingdom Biobank study

Brittany A. Larsen^{1,2}  | Brandon S. Klinedinst^{1,3} | Scott T. Le³ | Colleen Pappas³ | Tovah Wolf⁴ | Nathan F. Meier⁵ | Ye-Lim Lim⁶ | Auriel A. Willette^{1,2,3,7}

¹Neuroscience Graduate Program, Iowa State University, Ames, Iowa, USA

²Department of Biomedical Sciences, College of Veterinary Medicine, Iowa State University, Ames, Iowa, USA

³Department of Food Science and Human Nutrition, Iowa State University, Ames, Iowa, USA

⁴Department of Health Sciences, Western Carolina University, Cullowhee, North Carolina, USA

⁵Department of Kinesiology, Concordia University, Irvine, California, USA

⁶Department of Psychology, Virginia Polytechnic Institute, Blacksburg, Virginia, USA

⁷Department of Neurology, University of Iowa, Iowa City, Iowa, USA

Correspondence

Auriel A. Willette, Neuroscience Graduate Program, Iowa State University, 1109 HNSB 2302 Osborn Drive Ames 50011-1078, Ames, Iowa, USA.
Email: awillett@iastate.edu

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Abstract

BACKGROUND: Aging is characterized by body composition alterations, including increased visceral adiposity accumulation and bone loss. Alcohol consumption may partially drive these alterations, but findings are mixed. This study primarily aimed to investigate whether different alcohol types (beer/cider, red wine, white wine/Champagne, spirits) differentially associated with body composition.

METHODS: The longitudinal UK Biobank study leveraged 1869 White participants (40–80 years; 59% male). Participants self-reported demographic, alcohol/dietary consumption, and lifestyle factors using a touchscreen questionnaire. Anthropometrics and serum for proteomics were collected. Body composition was obtained via dual-energy X-ray absorptiometry. Structural equation modeling was used to probe direct/indirect associations between alcohol types, cardiometabolic biomarkers, and body composition.

RESULTS: Greater beer/spirit consumptions were associated with greater visceral adiposity ($\beta = 0.069$, $p < 0.001$ and $\beta = 0.014$, $p < 0.001$, respectively), which was driven by dyslipidemia and insulin resistance. In contrast, drinking more red wine was associated with less visceral adipose mass ($\beta = -0.023$, $p < 0.001$), which was driven by reduced inflammation and elevated high-density lipoproteins. White wine consumption predicted greater bone density ($\beta = 0.051$, $p < 0.005$).

DISCUSSION: Beer/spirits may partially contribute to the “empty calorie” hypothesis related to adipogenesis, while red wine may help protect against adipogenesis due to anti-inflammatory/eulipidemic effects. Furthermore, white wine may benefit bone health in older White adults.¹

KEYWORDS

adiposity, alcohol, body composition, wine

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1 | INTRODUCTION

Recreational alcohol use may partially drive weight gain. However, decades of epidemiological evidence have shown conflicting findings between greater alcohol intake and measures of adiposity. Greater alcohol use has been related to both higher^{2,3} and lower^{4,5} body mass index as well as both increased^{6,7} and decreased^{8,9} adipose mass measurements that were directly obtained via body composition analysis technology, such as dual-energy X-ray absorptiometry (DEXA). For example, a twin study which examined 334 female monozygotic twins found that moderate alcohol consumption was linked to 20% less visceral adiposity among twins with a high versus low genetic susceptibility to obesity,¹⁰ whereas another cotwin-control study that examined 1911 male monozygotic twins found no relationship between alcohol consumption and weight or obesity.¹¹ These inconsistent results imply that the relationship between alcohol consumption and adiposity may differ by sex, as has been observed previously.^{6,9}

Dietary intake may influence the relationship between alcohol consumption and adiposity. However, despite alcohol having an additive energy balance beyond food alone and containing 29.7 kJ/gram (7.1 kilocalories/gram), findings have been inconsistent in regards to whether alcohol use promotes short-term or long-term increases in food intake.^{12,13} A systematic review showed that light and moderate drinking was related to greater fat and protein intake but not total energy intake,¹³ whereas another systematic review showed that moderate drinking promoted short-term increases in dietary intake.¹² These inconsistent findings could be partially attributed to the contrasting physiologic effects alcohol has been found to have on energy metabolism and absorption that work to both encourage and discourage adiposity accumulation. On one hand, alcohol consumption has been shown to promote fat retention by reducing lipid oxidation^{14,15} and trigger appetite by stimulating neurochemical and peripheral systems, which may, in turn, encourage overeating.¹² On the other hand, alcohol intake may hinder caloric absorption¹⁶ and increase energy expenditure when consumed concomitantly with meals,¹⁵ which may, in turn, encourage weight loss. The latter effect can be partially attributed to alcohol's high thermogenic effect.¹⁷ Such alcohol-induced physiological alterations can be reflected in levels of cardiometabolic serum biomarkers, especially with long-term alcohol consumption. Nevertheless, these inconsistencies in the body of literature, in combination with prior findings linking moderate drinking to a reduced risk for obesity-related diseases,¹⁸ have motivated researchers to investigate novel factors and mechanisms that could help elucidate why the relationship between alcohol consumption and body composition has remained elusive.

To best assess how alcohol may influence body composition, one must consider the patterns of usage for different types of alcohol (i.e., beer, spirits, and wine) rather than simply gauging alcohol consumption as a whole.¹⁹ Each type of alcohol contains unique nutrient profiles and percentages of alcohol by volume. Therefore, a strong preference for one type of alcohol could contribute to different influences on body composition with long-term use. For example,

greater beer and spirit consumptions have been correlated with a higher waist-to-hip ratio.²⁰ Conversely, wine has largely shown null²¹ or inverse²⁰ associations with waist-to-hip ratio. Because red wine has a higher polyphenol content than white wine,²² the type of wine most frequently consumed may also be a distinguishing factor.

In order to help clarify these aforementioned inconsistencies in the body of literature, the first objective was to longitudinally assess self-reported patterns of drinking for different types of alcoholic beverages and body composition (including visceral adipose mass, subcutaneous adipose mass, somatic lean muscle mass, and bone mineral density, as measured by DEXA). Additionally, whether physiological biomarkers influence such associations as well as whether latent sub-groups of different drinker types have comparatively healthy or detrimental differences remains unclear. Thus, the second objective was to leverage a panel of serum biomarkers that reflected cholesterol trafficking, inflammation, vascular integrity, and insulin resistance to test whether these variables mechanistically influenced the relationships between the type of alcohol consumed and body composition in a structural equation model. It is hypothesized that drinking more beer and spirits would associate with greater visceral and subcutaneous adipose masses in older White participants, while drinking more wine would show null relationships with visceral and subcutaneous adipose masses in older White participants.

2 | METHODS

2.1 | Study design and participants

The UK Biobank prospective cohort study gathered baseline data on >500,000 individuals, aged 40–80 years, from 22 assessment centers throughout the United Kingdom between 2007 and 2010. To acquire access to the UK Biobank dataset, the researcher first registered for UK Biobank and subsequently applied for data access through UK Biobank's Access Management System, where the researcher then completed a brief application form and selected applicable data-fields of interest. After approval of the application, an access fee was paid, and an authorized individual provided a signature for a material transfer agreement on behalf of Iowa State University, which enabled access to the data.²³ UK Biobank periodically collected and published initial and follow up data from participants. For example, beginning in 2014, new imaging data from 5000 to 10,000 participants was uploaded to the UK Biobank database at 6- to 12-month intervals.²³

As summarized in Figure 1, participants from the UK Biobank cohort who had missing or incomplete data for DEXA imaging, serum biomarkers, the Food Frequency Questionnaire, the International Physical Activity Questionnaire, and demographic information ($n = 500,590$) were excluded from the study. Furthermore, evidence has reported that, compared to White British individuals, all other races/ethnicities consume significantly less alcohol.²⁴ In addition, approximately 94.4% of participants included in the UK Biobank study were comprised of British Whites.²⁵ Thus, in order to improve robustness in the main effect and interaction analyses, all non-white,

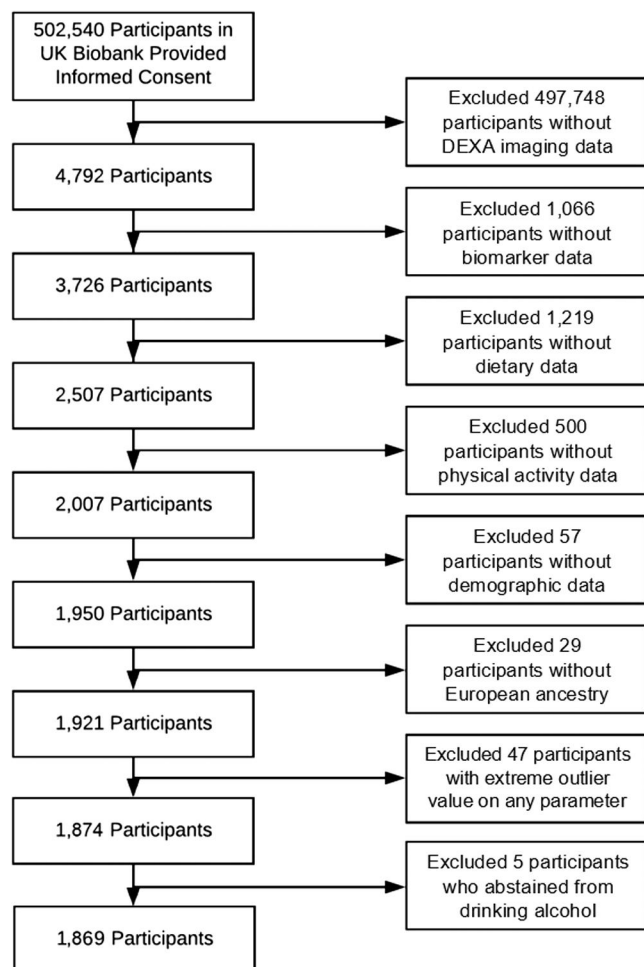


FIGURE 1 Flow diagram demonstrating the exclusion of participants. The flow chart diagram depicts the step-by-step exclusion of participants in order to obtain a final sub-sample with no missingness amongst the data of interest

Hispanic/Latino participants were excluded from the study ($n = 29$). Additionally, participants with a significant outlier value on any parameter ($n = 47$), which was defined as any observation that was greater than three standard deviations from the sample mean, as well as participants who abstained from alcohol ($n = 5$), were excluded from the analyses. Consequently, for this study, 1869 White participants of British ancestry were included from the United Kingdom (UK) Biobank cohort.²⁶

A subset of participants had a follow-up visit between August 2012 and June 2013. A visit to the assessment center involved: (1) consent, (2) touchscreen questionnaire, (3) verbal interview, (4) eye measures, (5) physical measures and (6) blood/urine sample collection. The touchscreen questionnaire collected data on sociodemographic, lifestyle, and health-related information.²⁷ Serum biomarkers were also collected, as was DEXA in order to image fat, bone, and muscle. Informed consent was obtained at baseline examinations. The UK Biobank protocol was approved by the North West Multi-Centre Research Ethics Committee (approval number: 11/NW/0382).

2.2 | Demographics and covariates

As described previously,²⁸ standard indices that were controlled for in the structural equation model included basic demographics (age, sex, education, socioeconomic status, height) and lifestyle factors (physical activity levels (moderate/vigorous), sleep duration, sunlight exposure, tobacco smoking status). The frequency, intensity, and duration of moderate-to-vigorous levels of leisurely and occupational physical activity were assessed using questions adapted from the validated, shortened version of the International Physical Activity Questionnaire.²⁹ Values were quantified as mean minutes per day. Data processing rules published by the International Physical Activity Questionnaire were followed.³⁰

2.3 | Alcohol/food intake and alcohol sub-groups

As previously described,³¹ participants self-reported weekly intake of various whole foods and alcohol over the past year on a Food Frequency Questionnaire²⁹ at three separate assessments in 2008, 2012, and 2014. Specifically, the dietary questionnaire consisted of 29 questions about dietary intake as well as 18 questions about alcohol consumption. More specifically, the whole foods included on the questionnaire were: fresh fruit, dried fruit, raw vegetables and salads, cooked vegetables, oily fish, non-oily fish, processed meat, poultry, beef, lamb, pork, cheese, bread, and cereal. The alcoholic beverages included on the questionnaire were: beer and cider, red wine, white wine and Champagne, and spirits. Bread, cereal, fruit, and vegetable responses were recorded in integer units (slices per week, bowls per week, pieces per day, and tablespoons per day, respectively). Total intake of meat, fish, and cheese responses were recorded as one of six ordinal categories ("never," "less than once a week," "once a week," "two to four times a week," "five or six times a week," or "once or more daily"). Total intake of similar food items was combined as follows: grain (bread, cereal), fruit (fresh, dried), vegetables (raw, cooked), and red meat (lamb, beef, pork). Alcohol consumption responses were recorded as an average weekly intake in pints for beer and cider (1 pint = 19.2 U.S. fluid ounces or 568 ml), glasses for wine/Champagne (1 glass = 5.9 U.S. fluid ounces or 175 ml), and standard measures for spirits (1 standard measure = 0.85 U.S. fluid ounces or 25 ml). One unit of alcohol was defined by UK Biobank as 8 g (10 ml), which is equivalent to a half pint of beer, one glass of wine, or one standard measure of spirits.³²

Based on prior work,³³ participants were categorized as beer/cider, spirits, red wine, or white wine/Champagne drinkers if $\geq 75\%$ of each participant's total alcohol intake came from one respective alcohol type. As a finer distinction for wine consumption, a "mixed wine" sub-group included people who showed the strongest preference for wine but drank roughly equal proportions of red and white wines (defined as $<25\%$ difference). Participants who showed no preference for one type of alcoholic drink were categorized as "no preference."

2.4 | Serum biomarker levels

At two separate visits in 2008 and 2012, plasma samples were collected in 4 ml EDTA vacutainers and analyzed within 24 h of sampling utilizing four Beckman Coulter LH750 instruments.³⁴ A total of 23 serum biomarkers were assessed (see Table 1).

2.5 | Body composition

DEXA imaging data collection began in May 2014 and occurred approximately 11 months after the follow-up visit. A trained radiographer delivered a five-minute, full-body DEXA (General Electric Lunar iDXA, Madison, WI) to each participant as the participant laid supine.³⁵ Compartment measurements included visceral adipose mass (in kilograms (kg)), subcutaneous adipose mass (kg), lean muscle mass (kg), and bone mineral density (g/cm²).

2.6 | Statistical analyses

In comparison to cross-sectional models, longitudinal computations constructed from observing variables over time enhance predictive performance with study outcomes.²⁸ Thus, for the dietary predictors, a mean was computed among the three observations to estimate the total amount consumed for each product.³⁶ As previously discussed, only participants with at least two of the three Food Frequency Questionnaire assessments were included in the study. This method demonstrated superior goodness-of-fit previously,²⁸ as this minimizes type I error and elucidates relationships between variables more robustly by capturing within-subject³⁷ and between-subject variations over time.³⁸

To test between-group differences among alcohol groups for the study measures, contingency chi-square or, when appropriate, Fisher's exact tests, one-way analysis of variance (ANOVA), and independent-samples *t*-tests were used. The frequency distributions for continuous variables were assessed, and all nonparametric continuous variables were transformed accordingly. After repetitions of statistical analyses were conducted using first the original variables followed by the transformed variables, however, results were not found to differ significantly. Therefore, the original variables were used for the analyses. For each significant ANOVA result, post-hoc comparisons were calculated via Tukey's honestly significant difference test.

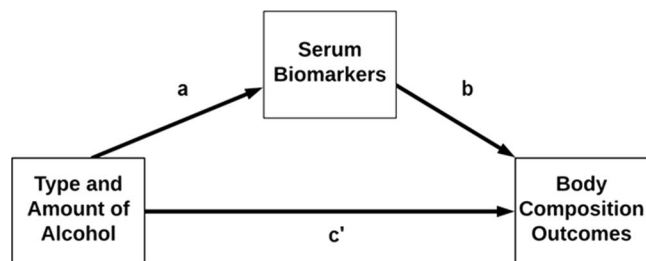
To examine how consumption patterns for each alcohol type were related to body composition, a structural equation model was leveraged using R (Version 3.4.1).³⁹ Graphs were prepared in *ggplot2* (Version 3.1.1).⁴⁰ Such structural equation model-based testing of direct associations, mediation, and moderation provides more statistical power than the standard regression procedure.⁴¹ Additionally, structural equation modeling is able to manage longitudinal data using the same framework.³⁸ Therefore, to best contain type I error

TABLE 1 List of serum biomarkers

Biomarker	Unit
Alanine aminotransferase (ALT)	U/L
Albumin	g/L
Alkaline phosphatase (ALP)	U/L
Apolipoprotein A (APOA)	g/L
Apolipoprotein B (APOB)	g/L
Aspartate aminotransferase (AST)	U/L
C-reactive protein (CRP)	mg/L
Creatinine	Umol/L
Cystatin C	mg/L
Gamma glutamyltransferase (GGT)	U/L
Glucose	mmol/L
High-density lipoproteins (HDL) cholesterol	mmol/L
Glycosylated hemoglobin (HbA1c)	mmol/mol (%)
Insulin-like growth factor I (IGF-I)	nmol/L
Low-density lipoproteins (LDL) cholesterol	mmol/L
Phosphate	mmol/L
Protein (total)	g/L
Sex hormone binding globulin (SHBG)	nmol/L
Testosterone	nmol/L
Triglycerides (TG)	mmol/L
Urate	Umol/L
Urea	mmol/L
Vitamin D	nmol/L

at a family-wise alpha of 0.05, an empirical model-building approach was utilized in order to select variables that influenced body composition. Specifically, using a backwards-elimination approach, a full structural equation model first included all variables. In descending order of significance level, variables were singularly removed from the structural equation model until all remaining variables were $p \leq 0.05$.

As shown in Figure 2, a structural equation model was built to best fit the covariance structure⁴³ between each alcohol type consumed and body composition, as well as indirect associations of 23 serum biomarkers, while controlling for height as well as demographic and lifestyle variables. Maximum likelihood was used to estimate standardized parameter estimates (β). Mediation tested whether different types of alcohol consumption was associated with body composition outcomes and whether changes in serum biomarkers influenced these associations. Specifically, parameter decomposition was used to distinguish indirect (λ) from direct (β) associations.^{42,43} To maintain a data-driven analysis and ensure robustness, participants with missing data were excluded from the analyses.



$$\text{Total Outcome from Alcohol consumption} = a + b \cdot c'$$

FIGURE 2 Mediation model. This model was used to test whether different types of alcohol consumption associated with body composition outcomes and whether changes in serum biomarkers influenced these associations, while controlling for demographics, height, and lifestyle variables

3 | RESULTS

3.1 | Participants and characteristics of alcohol sub-groups

Considering the marked sex-specific differences in body composition⁴⁴ as well as in age-associated physiological alterations,^{45,46} in addition to prior findings which imply that alcohol may have distinguishable sex-specific physiologic effects on men and women,^{6,9} participants were first stratified by sex. Table 2 summarizes demographic characteristics, lifestyle habits, and body composition differences by sex in the sample. Latent sub-groups were formed based on the primary alcohol that a participant most frequently consumed, then compared according to demographic characteristics, lifestyle habits, and body composition (Table 3). Approximately 60.8% of the sample showed a strong preference for one alcohol type. In summary, age, physical activity, and smoking status did not influence drinking habits. Men preferred beer or had no preference for one alcohol type while women preferred wine. Adults with lower educational attainment and lower socioeconomic status preferred beer/cider and spirits. Beer drinkers consumed the least amount of total alcohol, while participants with no preference consumed the most. In the overall sample, participants consumed a mean of 9.8 (± 7.6) alcoholic drinks per week and a median of eight alcoholic drinks per week.

Serum biomarkers among alcohol preference sub-groups are summarized in Table 4. In summary, beer and multi-alcohol drinkers had the highest levels of “bad” lipoproteins (e.g., triglycerides), HbA1c, as well as markers of liver function (e.g., ALT, AST, GGT), kidney function (e.g., creatinine, cystatin C, urate), and peripheral inflammation (e.g., C-reactive protein (CRP)) but showed the lowest levels of physiologically beneficial lipoproteins (e.g., Apolipoprotein A (APOA), High-density lipoproteins (HDL)). Similarly, spirit drinkers also had higher levels of kidney function biomarkers (e.g., urate, cystatin C). Conversely, in general, white wine drinkers had the lowest levels of most of the aforementioned biomarkers as well as the highest levels of physiologically beneficial lipoproteins (e.g., APOA, HDL) and phosphate.

3.2 | Dietary intake and alcohol consumption

Dietary intake patterns among alcohol sub-groups are detailed in Table 5. In summary, beer drinkers consumed more grains and fewer fruits than most other sub-groups. Overall, wine drinkers ate fewer processed food. Specifically, white wine drinkers ate fewer red/processed meats and grains than all other sub-groups except spirit drinkers. Red wine drinkers ate the lowest amounts of processed meats and poultry, while participants with no preference for one type of alcoholic beverage ate the most red/processed meats and spirit drinkers ate the most poultry.

3.3 | Body composition by most preferred alcohol types

As detailed in Table 6 and shown in Supplemental Figure S1, greater beer consumption over time was linked to greater visceral adipose mass ($p < 0.001$). Lipoprotein and metabolism factors influenced 54% of the beer consumption-visceral adipose mass association, including HDL ($p < 0.001$), urate ($p < 0.001$), Apolipoprotein B (APOB) ($p < 0.005$), APOA ($p < 0.001$), Insulin-like growth factor I (IGF-I) ($p = 0.02$), triglyceride ($p = 0.02$), and urea levels ($p = 0.04$). Conversely, greater beer consumption was associated with less lean muscle mass ($p = 0.04$). Lipoprotein factors as well as markers of peripheral inflammation and kidney/liver functions fully influenced the inverse beer consumption-lean muscle mass association, including HDL ($p < 0.001$), urate ($p < 0.001$), GGT ($p < 0.01$), and total protein levels ($p = 0.02$). Beer showed no relationships with subcutaneous adipose mass or bone mineral density in the model.

As shown in Table 7, white wine consumption over time was associated with lean muscle mass ($p < 0.001$) and bone mineral density ($p < 0.005$). Specifically, greater white wine consumption was related to less lean muscle mass, which was fully influenced by lipoprotein factors and markers of kidney and liver functions, including HDL ($p < 0.001$), urate ($p < 0.01$), GGT ($p = 0.01$), and creatinine ($p = 0.03$). Greater white wine consumption was also related to greater bone mineral density ($p < 0.005$). Although 106% of the total white wine-bone mineral density association was unspecified, 32% of the relationship was associated with lipoprotein factors and markers of kidney function, including HDL ($p < 0.005$) and urate levels ($p = 0.01$). White wine consumption showed no associations with visceral adipose mass or subcutaneous adipose mass in the model.

As shown in Table 8 and depicted in Supplemental Figure S2, red wine consumption showed inverse relationships with visceral adipose mass ($p < 0.001$), subcutaneous adipose mass ($p < 0.001$), and lean muscle mass ($p < 0.001$). The inverse red wine consumption-visceral adipose mass association was fully influenced by lipoprotein and kidney function factors, including HDL ($p < 0.001$), APOA ($p < 0.001$), and cystatin C levels ($p < 0.001$). Similarly, the inverse red wine consumption-subcutaneous adipose mass relationship was fully influenced by lipoprotein factors and markers of peripheral inflammation and kidney/liver functions, including HDL ($p < 0.001$), cystatin

TABLE 2 Demographics and data summary

Data (unit)	Total sample	Women	Men	T value/Chi-square ^a
Sample size (n)	1869	766 (41%)	1103 (59%)	
Age (years)	64.6 (7.5)	63 (7.5)	65.7 (7.3)	$t = 7.8^{***}$
Education level (n and sample %)				$\chi^2 = 42.91^{***}$
College/other higher level ^b	1294 (69.2%)	520 (67.9%)	774 (70.2%)	
Post-secondary/vocational ^b	303 (16.2%)	107 (14%)	196 (17.8%)	
Secondary ^b	189 (10.1%)	116 (15.1%)	73 (6.6%)	
Other ^b	83 (4.4%)	23 (3%)	60 (5.4%)	
Socioeconomic status (n and sample %)				$\chi^2 = 4.07$
Lower ^c	758 (40.6%)	330 (43.1%)	428 (38.8%)	
Middle ^c	1011 (54.1%)	393 (51.3%)	618 (56%)	
Upper ^c	100 (5.4%)	43 (5.6%)	57 (5.2%)	
Smoking status (n and sample %)				$\chi^2 = 3.5$
Never ^d	1099 (58.8%)	469 (61.2%)	630 (57.1%)	
Former ^d	651 (34.8%)	254 (33.2%)	397 (36%)	
Current ^d	119 (6.4%)	43 (5.6%)	76 (6.9%)	
Moderate exercise (mins/week)	58.2 (51.3)	56.3 (48)	59.6 (53.5)	$t = 1.49$
Vigorous exercise (mins/week)	39.8 (32.1)	38.1 (29.6)	41 (33.6)	$t = 2.03^*$
Beer/Cider (mean pints/week)	2.5 (3.9)	0.7 (1.8)	3.8 (4.4)	$t = 21.41^{***}$
White wine/Champagne (mean glasses/week)	2.3 (4.1)	3 (4.2)	1.7 (3.8)	$t = 6.79^{***}$
Red wine (mean glasses/week)	3.8 (4.9)	2.9 (3.5)	4.4 (5.6)	$t = 7.05^{***}$
Spirits (mean measures/week)	1.2 (2.6)	1 (2.1)	1.4 (2.9)	$t = 3.93^{***}$
Alcoholic drinks/Week	9.8 (7.6)	7.6 (5.8)	11.4 (8.3)	$t = 11.68^{***}$
Visceral adipose Mass (kg)	1.2 (0.9)	0.7 (0.5)	1.6 (0.9)	$t = 28.04^{***}$
Subcutaneous adipose Mass (kg)	23.2 (7.8)	24.6 (8.2)	22.3 (7.4)	$t = 6.27^{***}$
Muscle Mass (kg)	49.2 (9.6)	39.9 (4.5)	55.7 (6.3)	$t = 63.35^{***}$
Bone mineral density (g/cm ²)	1.2 (0.1)	1.1 (0.1)	1.3 (0.1)	$t = 29.25^{***}$

Notes: Values are Mean (Standard Deviation) unless stated otherwise.

Abbreviations: g/cm², grams/centimeter squared; kg, kilograms; Mins, minutes.

^aT-value and chi-square results reflect differences between men and women in the sample.

^bCollege/other higher level = earned at least a Bachelor's degree or ≥ 4 years of education beyond secondary education; post-secondary/vocational = earned an Associate's degree or attendance of postsecondary education for < 4 years; secondary = high school diploma or equivalent; other = less than a high school diploma or equivalent.

^cLower socioeconomic status = $< \text{£}31,000$ ($< \$51,441$ USD); middle socioeconomic status = $\text{£}31,000$ ($\$51,441$) - $\text{£}100,000$ ($\$165,940$ USD); upper socioeconomic status = $> \text{£}100,000$ ($\$165,940$ USD).

^dNever smokers = < 100 lifetime cigarettes ever smoked; former smoker = ≥ 100 lifetime cigarettes ever smoked but reported no longer smoking currently; current smokers = ≥ 100 lifetime cigarettes ever smoked and reported smoking cigarettes currently.

*, **, and *** denotes $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$.

C ($p < 0.001$), APOA ($p < 0.01$), albumin ($p = 0.01$), CRP ($p = 0.01$), and GGT levels ($p = 0.02$). The inverse red wine-lean muscle mass association was fully influenced by lipoprotein and liver function factors, including HDL ($p < 0.001$) and GGT levels ($p = 0.02$). No relationship between red wine consumption and bone mineral density was found in the model.

As shown in Table 9, greater spirit consumption was associated with greater visceral adipose mass ($p < 0.001$), subcutaneous adipose mass ($p < 0.001$), lean muscle mass ($p < 0.01$), and bone mineral density ($p = 0.01$). The spirit consumption-visceral adipose mass and -subcutaneous adipose mass associations were fully influenced by markers of kidney function, including urate ($p < 0.01$ and $p = 0.01$,

TABLE 3 Data summary by primary type of alcohol consumed

	Wine					F values/Chi-square ^g
	Beer	Red	White	Mixed	Spirits	
	<i>n</i> = 213 (11.4%)	<i>n</i> = 461 (24.7%)	<i>n</i> = 292 (15.6%)	<i>n</i> = 125 (6.7%)	<i>n</i> = 45 (2.4%)	<i>n</i> = 733 (39.2%)
Age (years)	64.14 (7.41)	64.89 (7.4)	64.52 (7.9)	64.54 (7.74)	64.24 (7.36)	64.65 (7.38)
Sex						<i>F</i> (51,863) = 0.33
Female	31 (14.6%)	232 (50.3%)	225 (77.1%)	72 (57.6%)	25 (55.6%)	181 (24.7%)
Male	182 (85.4%)	229 (49.7%)	67 (22.9%)	53 (42.4%)	20 (44.4%)	552 (75.3%)
Education level						$\chi^2 = 333.87^{***}$
College or higher	126 (59.2%)	327 (70.9%)	214 (73.3%)	101 (80.8%)	23 (51.1%)	503 (68.6%)
Post-secondary/vocational	47 (22.1%)	71 (15.4%)	33 (11.3%)	11 (8.8%)	8 (17.8%)	133 (18.1%)
Secondary	23 (10.8%)	42 (9.1%)	41 (14%)	13 (10.4%)	7 (15.6%)	63 (8.6%)
Other	17 (8%)	21 (4.6%)	4 (1.4%)	0 (0%)	7 (15.6%)	34 (4.6%)
Social class						$\chi^2 = 61.47^{***}$
Lower	113 (53.1%)	177 (38.4%)	102 (34.9%)	41 (32.8%)	29 (64.4%)	296 (40.4%)
Middle	93 (43.7%)	262 (56.8%)	161 (55.1%)	75 (60%)	16 (35.6%)	404 (55.1%)
Upper	7 (3.3%)	22 (4.8%)	29 (9.9%)	9 (7.2%)	0 (0%)	33 (4.5%)
Smoking status						$\chi^2 = 42.55^{***}$
Never	130 (61%)	278 (60.3%)	178 (61%)	85 (68%)	26 (57.8%)	402 (54.8%)
Former	69 (32.4%)	163 (35.4%)	97 (33.2%)	34 (27.2%)	13 (28.9%)	275 (37.5%)
Current	14 (6.6%)	20 (4.3%)	17 (5.8%)	6 (4.8%)	6 (13.3%)	56 (7.6%)
Alcoholic drinks (weekly)	8.18 (6.91) ^f	9.61 (7.94)	9.44 (8.2)	8.48 (6.55) ^f	9.29 (7.84)	10.83 (7.34) ^{af}
						<i>F</i> (51,863) = 5.65 ^{***}
Moderate exercise (mins/wk)	60.98 (57.61)	58.89 (51.38)	53.53 (45.28)	55.8 (51.71)	67.11 (52.71)	58.78 (51.43)
						<i>F</i> (51,863) = 0.97
Vigorous exercise (mins/wk)	40.42 (35.86)	41.34 (31.78)	36.7 (27.38)	39.21 (29.84)	37.78 (33.99)	40.14 (33.04)
						<i>F</i> (51,863) = 0.84
Height (cm)	174.8 (7.83) ^{b,c,d,e}	169.98 (8.97) ^{a,c,f}	166.26 (7.93) ^{ab,d,f}	169.45 (10.04) ^{a,c,f}	167.88 (9.14) ^{a,f}	173.56 (8.08) ^{b,c,d,e}
						<i>F</i> (51,863) = 43.7 ^{***}
VAM (kg)	1.53 (0.98) ^{b,c,d}	1.05 (0.79) ^{a,c,f}	0.83 (0.64) ^{ab,f}	1.02 (0.96) ^a	1.14 (0.91)	1.43 (0.9) ^{b,c,d}
						<i>F</i> (51,863) = 32.19 ^{***}
SAM (kg)	23.55 (8.64)	22.5 (7.57)	23.42 (7.6)	23.39 (9.31)	24.37 (7.74)	23.44 (7.54)
						<i>F</i> (51,863) = 1.21

(Continues)

TABLE 3 (Continued)

	Wine				Spirits	No preference	F values/Chi-square ^g
	Beer	Red	White	Mixed			
LMM (kg)	53.55 (8.38) ^{b,c,d,e}	47.38 (9.4) ^{a,c,f}	42.86 (7.63) ^{a,b,d,f}	47.18 (10.57) ^{a,c,f}	46.16 (9.68) ^{a,f}	52.22 (8.71) ^{b,c,d,e}	F (51,863) = 63.52***
BMD (g/cm ²)	1.28 (0.13) ^{b,c,d,e}	1.21 (0.14) ^{a,c,f}	1.17 (0.14) ^{a,b,f}	1.21 (0.13) ^{a,f}	1.17 (0.12) ^{a,f}	1.27 (0.14) ^{b,c,d,e}	F (51,863) = 29.67***

Notes: Values are Mean (Standard Deviation) unless stated otherwise. Superscript letters a through f in each column denote where the means of one alcohol preference group are significantly different from each another alcohol preference group according to Tukey's honestly significant difference test, where $p \leq 0.05$.

Abbreviations: BMD, bone mineral density; cm, centimeters; g/cm², grams/centimeter squared; kg, kilograms; mins, minutes; LMM, lean muscle mass; SAM, subcutaneous adipose mass; VAM, visceral adipose mass; wk = week.

^aDenotes the means in other groups that differ significantly from beer drinkers.

^bDenotes the means in other groups that differ significantly from red wine drinkers.

^cDenotes the means in other groups that differ significantly from white wine drinkers.

^dDenotes the means in other groups that differ significantly from red wine drinkers.

^eDenotes the means in other groups that differ significantly from spirit drinkers.

^fDenotes the means in other groups that differ significantly from drinkers with no preference for one type of alcoholic beverage.

^gChi-square results reflect differences in the distribution of subcategorical responses for each categorical variable (sex, education level, socioeconomic status, smoking status) between each of the preferred alcohol sub-groups in the sample.

*, **, *** denote $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$.

respectively) and cystatin C ($p = 0.03$ and $p = 0.02$, respectively) levels. The spirit consumption-lean muscle mass and -bone mineral density associations were fully influenced by urate levels (p 's < 0.001).

4 | DISCUSSION

Whether recreational alcohol use is a driver of weight gain has remained inconsistent in the prevailing body of literature.⁴⁷⁻⁴⁹ Additionally, few studies have assessed how specific types of alcohol may differentially influence body composition.^{20,21,50} Thus, the primary aim of this study was to model and generate testable hypotheses regarding associations between the consumption of preferred alcoholic beverage types (beer/cider, spirits, red wine, and white wine/Champagne) and body composition (visceral adipose mass, subcutaneous adipose mass, lean muscle mass, and bone mineral density) amongst a sample of older White British adults. Moreover, data-driven modeling was used to examine the interplay between the type of alcoholic beverage consumed, 23 serum biomarkers, and components of body composition.

Direct associations were identified between the consumption of different types of alcoholic beverages and components of body composition, as well as indirect associations from serum biomarkers. Specifically, associations with adiposity were found by alcohol type in the model. More specifically, greater beer consumption directly and indirectly associated with greater visceral adipose mass, which showed an inverse relationship with HDL cholesterol levels as well as positive relationships with urate, APOA, APOB, IGF-I, triglycerides, and urea levels. High-density lipoproteins cholesterol positively associated with beer consumption but inversely associated with visceral adipose mass, as found previously.⁵¹ Individuals with obesity have been found to have increased HDL cholesterol clearance, resulting in lower levels of HDL cholesterol.⁵² In addition, drinking beer may aggravate the expression of fatty acid oxidation- and metabolism-associated biomarkers, which may reduce the efficiency of energy expenditure and, in turn, encourage weight gain.⁵³

The direct beer consumption-visceral adipose mass association found in the model was in congruence with some prior findings,^{20,54} but not others.^{55,56} This direct association could be partially elucidated by the energy composition of beer in conjunction with differences in drinking patterns by classes of alcohol. Although beer contains the lowest alcohol by volume value (4%–6% in a standard beer)⁵⁷ of all alcoholic beverage types, beer contributes to energy intake predominantly through carbohydrates. One standard-sized beer contains approximately 251 kJ/500 g (60 kilocalories/500 g),⁵⁸ the consumption of which contributes to an increase in visceral adipose mass.

It should be noted, however, there has been a recent increase in the popularity of craft beer. The craft beer share of the retail beer market in the United States rose from 10.2% in 2012% to 23.6% in 2020, more than doubling in less than a decade.⁵⁹ Craft beers are

TABLE 4 Biomarkers by primary type of alcohol consumed

Biomarker (Unit)	Wine			Spirits	No preference	F values
	Beer	Red	White			
ALT (U/L)	24.85 (9.79) ^{b,c,d}	22.07 (11.37) ^{a,f}	20.75 (11.29) ^{a,f}	24.18 (13.33)	24.58 (11.81) ^{b,c,d}	$F(5,1863) = 7.89^{***}$
Albumin (g/L)	45.38 (2.49)	45.37 (2.27)	45.35 (2.44)	45.84 (2.31)	45.56 (2.31)	$F(5,1863) = 0.84$
ALP (U/L)	79.59 (19.13)	77.43 (19.32)	78.18 (21.94)	81.07 (19.63)	78.95 (19.33)	$F(5,1863) = 1$
APOA (g/L)	1.48 (0.23) ^{b,c,d}	1.59 (0.25) ^{a,c,f}	1.68 (0.27) ^{a,b,d,e,f}	1.55 (0.27) ^c	1.52 (0.25) ^{b,c,d}	$F(5,1863) = 24.36^{***}$
APOB (g/L)	1.02 (0.22)	1 (0.2)	1.02 (0.21)	0.98 (0.2)	1.03 (0.02)	$F(5,1863) = 1.58$
AST (U/L)	26.58 (6.19) ^c	25.58 (7.18)	24.71 (7.19) ^{a,f}	27.09 (9.29)	26.61 (7.23) ^c	$F(5,1863) = 4.63^{***}$
CRP (mg/L)	2.24 (3.65) ^b	1.59 (2.17) ^a	2 (2.75)	2.56 (3.38)	1.93 (2.74)	$F(5,1863) = 2.79^*$
Creatinine (umol/L)	79.72 (11.54) ^{b,c,d,e}	72.33 (13.58) ^{b,c,f}	67.11 (10.35) ^{a,b,d,e,f}	73.38 (12.58) ^{b,c}	77.74 (12.72) ^{b,c,d}	$F(5,1863) = 41.27^{***}$
Cystatin C (mg/L)	0.9 (0.12) ^{b,c,d}	0.86 (0.11) ^{a,f}	0.84 (0.11) ^{a,e,f}	0.87 (0.13) ^c	0.88 (0.11) ^{b,c}	$F(5,1863) = 9.78^{***}$
GGT (U/L)	40.22 (29.58) ^{b,c,d}	31.82 (26.29) ^{a,f}	31.91 (28.22) ^a	30.62 (25.28)	36.65 (27.05) ^{b,d}	$F(5,1863) = 5.69^{***}$
Glucose (mmol/L)	4.98 (0.83)	4.97 (0.82)	4.94 (0.77)	4.89 (0.5)	5.01 (0.78)	$F(5,1863) = 0.52$
HbA1c (mmol/mol)	34.73 (3.83)	35.08 (4.52)	34.26 (3.75) ^f	34.82 (5.06)	35.17 (4.44) ^c	$F(5,1863) = 2.23^*$
HDL (mmol/L)	1.35 (0.32) ^{b,c,d}	1.53 (0.36) ^{a,c,f}	1.65 (0.36) ^{a,b,d,e,f}	1.54 (0.35) ^{a,c,f}	1.41 (0.34) ^{b,c,d}	$F(5,1863) = 27.41^{***}$
IGF-1 (nmol/L)	22.86 (5.24)	22.07 (5.15)	21.54 (5.25)	22.32 (4.96)	22.21 (5.17)	$F(5,1863) = 1.92$
LDL (mmol/L)	3.49 (0.78)	3.47 (0.73)	3.6 (0.75)	3.58 (0.68)	3.55 (0.74)	$F(5,1863) = 1.68$
Phosphate (mmol/L)	1.16 (0.15) ^c	1.17 (0.14) ^c	1.2 (0.14) ^{a,b,f}	1.17 (0.14)	1.15 (0.14) ^c	$F(5,1863) = 4.93^{***}$
SHBG (nmol/L)	43.81 (19.95) ^{b,c,d}	54.08 (26.53) ^{b,c,f}	61.1 (29.19) ^{a,b,f}	54.66 (27.55) ^{a,f}	51.58 (20.09)	$F(5,1863) = 21.37^{***}$
Testosterone (nmol/L)	10.95 (5.2) ^{b,c,d,e,f}	6.68 (6.1) ^{a,c,f}	3.87 (5.07) ^{a,b,d,f}	5.88 (6) ^{a,c,f}	6.36 (6.48) ^{a,f}	$F(5,1863) = 61.17^{***}$
Total protein (g/L)	72.54 (3.67) ^d	72.16 (3.75) ^d	72.24 (3.92) ^d	71.06 (3.47) ^{a,b,c,f}	72.39 (3.7) ^d	$F(5,1863) = 3.09^{**}$
Triglycerides (mmol/L)	1.85 (0.98) ^{b,c,d}	1.55 (0.84) ^{a,f}	1.44 (0.76) ^{a,f}	1.51 (0.8) ^a	1.75 (0.89) ^{b,c}	$F(5,1863) = 9.71^{***}$
Urate (umol/L)	338.96 (71.01) ^{b,c,d,e}	300.9 (71.06) ^{a,c,f}	283.55 (68.74) ^{a,b,f}	292.37 (70.49) ^{a,f}	332.45 (70.52) ^{b,c,d}	$F(5,1863) = 31.82^{***}$
Urea (mmol/L)	5.49 (1.13)	5.38 (1.15)	5.32 (1.15)	5.34 (1.19)	5.5 (1.1)	$F(5,1863) = 1.73$
Vitamin D (nmol/L)	51.85 (19.09)	53.28 (19.84)	56.37 (18.98)	52.07 (20.59)	52.99 (19.43)	$F(5,1863) = 2.07$

Notes: Values are Mean (Standard Deviation) unless stated otherwise. Superscript letters a through f in each column denote where the means of one alcohol preference group are significantly different from each another alcohol preference group according to Tukey's honestly significant difference test, where $p \leq 0.05$.

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; APOA, apolipoprotein A; APOB, apolipoprotein B; AST, aspartate aminotransferase; CRP, C-reactive protein; GGT, gamma glutamyltransferase; HbA1c, glycated hemoglobin; HDL, high-density lipoproteins; IGF-1, insulin-like growth factor 1; LDL, low-density lipoproteins; SHBG, sex hormone binding globulin.

^aDenotes the means in other groups that differ significantly from beer drinkers.

^bDenotes the means in other groups that differ significantly from red wine drinkers.

^cDenotes the means in other groups that differ significantly from white wine drinkers.

^dDenotes the means in other groups that differ significantly from mixed wine drinkers.

^eDenotes the means in other groups that differ significantly from spirit drinkers.

^fDenotes the means in other groups that differ significantly from drinkers with no preference for one type of alcoholic beverage.

*, **, *** denote $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$.

TABLE 5 Dietary intake patterns by primary type of alcohol consumed

	Beer	Wine Red	White	Mixed	Spirits	No preference	F values/Chi-square ^j
Grains (servings/week)	18.4 (8.73) ^{b,c}	16.01 (6.82) ^a	14.42 (6.79) ^{a,f}	16.71 (7.69)	15.38 (8.13)	17.03 (7.84) ^c	$F(51,863) = 8.45^{***}$
Fruits (servings/day)	2.78 (2.27) ^d	3.16 (2.12)	3.22 (2.02)	3.62 (2.49) ^{a,f}	3.47 (2.01)	2.98 (1.93) ^d	$F(51,863) = 3.55^{***}$
Red meats							$\chi^2 = 64.86^{**}$
Never ^a	11 (5.2%)	42 (9.1%)	21 (7.2%)	7 (5.6%)	2 (4.4%)	32 (4.4%)	
<1 a week ^g	7 (3.3%)	9 (2%)	12 (4.1%)	4 (3.2%)	4 (8.9%)	12 (1.6%)	
Once a week ^g	13 (6.1%)	29 (6.3%)	26 (8.9%)	6 (4.8%)	4 (8.9%)	34 (4.6%)	
2-4 times/week ^g	77 (36.2%)	153 (33.2%)	121 (41.4%)	49 (39.2%)	15 (33.3%)	259 (35.3%)	
5-6 times/week ^g	44 (20.7%)	103 (22.3%)	50 (17.1%)	29 (23.2%)	6 (13.3%)	173 (23.6%)	
1(+) daily ^g	61 (28.6%)	125 (27.1%)	62 (21.2%)	30 (24%)	14 (31.1%)	223 (30.4%)	
Poultry							$\chi^2 = 46.72^{***}$
Never ^h	9 (4.2%)	33 (7.2%)	13 (4.5%)	5 (4%)	1 (2.2%)	20 (2.7%)	
<1 a week ^h	25 (11.7%)	45 (9.8%)	25 (8.6%)	13 (10.4%)	2 (4.4%)	55 (7.5%)	
Once a week ^h	75 (35.2%)	141 (30.6%)	75 (25.7%)	51 (40.8%)	14 (31.1%)	256 (34.9%)	
2-4 times/week ^h	99 (46.5%)	227 (49.2%)	168 (57.5%)	55 (44%)	27 (60%)	382 (52.1%)	
5-6 times/week ^h	5 (2.3%)	15 (3.3%)	10 (3.4%)	0 (0%)	0 (0%)	19 (2.6%)	
1(+) daily ^h	0 (0%)	0 (0%)	1 (0.3%)	1 (0.8%)	1 (2.2%)	1 (0.1%)	
Processed meats							$\chi^2 = 90.01^{***}$
Never ⁱ	9 (4.2%)	50 (10.8%)	28 (9.6%)	14 (11.2%)	4 (8.9%)	40 (5.5%)	
<1 a week ⁱ	60 (28.2%)	161 (34.9%)	126 (43.2%)	49 (39.2%)	15 (33.3%)	181 (24.7%)	
Once a week ⁱ	66 (31%)	133 (28.9%)	71 (24.3%)	37 (29.6%)	13 (28.9%)	218 (29.7%)	
2-4 times/week ⁱ	71 (33.3%)	108 (23.4%)	60 (20.5%)	23 (18.4%)	12 (26.7%)	256 (29.7%)	
5-6 times/week ⁱ	5 (2.3%)	9 (2%)	7 (2.4%)	2 (1.6%)	1 (2.2%)	29 (4%)	
1(+) daily ⁱ	2 (0.9%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	9 (1.2%)	

Notes: Values are Mean (Standard Deviation) unless stated otherwise. The table does not include non-significant food groups. Superscript letters a through f in each column denote where the means of one alcohol preference group are significantly different from each another alcohol preference group according to Tukey's honestly significant difference test, where $p \leq 0.05$.

^aDenotes the means in other groups that differ significantly from beer drinkers.

^bDenotes the means in other groups that differ significantly from red wine drinkers.

^cDenotes the means in other groups that differ significantly from white wine drinkers.

^dDenotes the means in other groups that differ significantly from mixed wine drinkers.

^eDenotes the means in other groups that differ significantly from spirit drinkers.

^fDenotes the means in other groups that differ significantly from drinkers with no preference for one type of alcoholic beverage.

^gIndicates the mean frequency per week for which a participant self-reported consuming at least one serving of red meats.

^hIndicates the mean frequency per week for which a participant self-reported consuming at least one serving of poultry.

ⁱIndicates the mean frequency per week for which a participant self-reported consuming at least one serving of processed meats.

^jChi-square results reflect differences in the distribution of subcategorical responses for each categorical variable (consumption frequencies of red meats, poultry, and processed) between each of the preferred alcohol sub-groups in the sample.

*, **, *** denote $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$.

produced by small, independent craft breweries that have the capacity to manufacture low volumes of beer, which are made using traditional ingredients as well as nontraditional ingredients in order to provide the beer with a distinctive flavor.⁶⁰ Craft beer has a higher mean percent alcohol by volume than traditional beer, with some craft beers containing as much as 15% alcohol by volume or higher.⁶¹

Although data about craft beer consumption was unavailable in the sample of participants examined from UK Biobank, craft beer may likely show an even stronger link to increased visceral adiposity among those who drink a higher proportion of craft beer compared to traditional beer, because the elevated percent alcohol by volume contained in craft beer equates to an increase in caloric content per

TABLE 6 Model results for beer

	Mechanisms of action	% of total association ^a	Beta coefficients of overall model
Visceral adiposity	(1) Unspecified:	46%	$\beta_{\text{total}} = 0.07^{***}$
	(2) HDL cholesterol levels:	35%	
	(3) Urate levels:	30%	
	(4) APOB levels:	22%	
	(5) APOA levels:	20%	
	(6) IGF-I levels:	7%	
	(7) triglyceride levels:	4%	
	(8) Urea levels:	3%	
Subcutaneous adiposity	(1) Urate levels:	208%	$\beta_{\text{total}} = 0.01$ n.s.
	(2) HDL cholesterol levels:	167%	
	(3) GGT levels:	50%	
	(4) IGF-I levels:	50%	
	(5) APOA levels:	50%	
Lean muscle Mass	(1) Urate levels:	114%	$\beta_{\text{total}} = -0.01^*$
	(2) HDL cholesterol levels:	114%	
	(3) GGT levels:	71%	
	(4) total protein levels:	29%	
Bone mineral density	(1) Urate levels:	233%	$\beta_{\text{total}} = 0.01$ n.s.
	(2) HDL cholesterol levels:	133%	

Notes: *, **, ***, and n. s. denote $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$, and non-significant beta coefficients, respectively. Model controlled for basic demographic (age, sex, education, socioeconomic status, height) and lifestyle (moderate and vigorous physical activity levels, sleep duration, sunlight exposure, tobacco smoking status) variables.

Abbreviations: APOA, apolipoprotein A; APOB, apolipoprotein B; HDL, high-density lipoproteins; GGT, gamma glutamyltransferase; IGF-1, insulin-like growth factor 1.

^aThe percentage of association for the mediation associations (serum biomarkers) are with respect to the total association that each serum biomarker composes. The table does not include non-significant serum biomarkers.

drink. Finally, demographic characteristics that associate with greater adiposity⁶² were more prevalent amongst beer drinkers in the cohort, which consisted predominantly of males who had less educational attainment and with lower socioeconomic status in comparison to all sub-groups except spirit drinkers.

In wine drinkers, adiposity was differentially correlated with the type of wine consumed. Specifically, greater red wine consumption showed inverse relationships with visceral adipose mass and subcutaneous adipose mass. In contrast, white wine consumption showed no association with adiposity. This finding corresponded with some prior findings,^{20,55} but not all.⁵⁴ The structural equation model suggested that red wine associated with visceral adipose mass through red wine's influence on HDL cholesterol, ApoA lipoprotein, and cystatin C levels. The red wine-adiposity association could be partially attributed to resveratrol, a polyphenol found in grapes and wine, with red wine having higher concentrations than white wine. Resveratrol may reduce inflammation and discourage fat storage in human adipocytes.⁶³

Spirit consumption associated with visceral adipose mass and subcutaneous adipose mass, which was consistent with some

studies,^{54,55} but contradicted others.^{21,56} Spirit drinkers in this cohort had less educational attainment and earned less income, which have been linked to greater adiposity.⁶² In the model, the spirit-adiposity relationships were fully influenced by urate and cystatin C levels; however, these relationships cannot imply causality. Thus, spirit consumption may contribute to an impairment in kidney function, consequently increasing uric acid and cystatin C levels, as found previously.⁶⁴

Interestingly, the consumption of white wine, but not red wine, showed direct and indirect associations with bone mineral density in the model. Indirectly, white wine consumption was positively associated with bone mineral density, which was partially predicted by serum urate levels. The direct white wine-bone mineral density association could be attributed to higher quantities of specific polyphenols found in white wine compared to red wine. Although red wine has higher polyphenol content overall, white wine contains nearly twice as much protocatechuic acid as red wine (0.33 mg/100 ml (0.4) versus 0.17 mg/100 ml (0.2), respectively).⁶⁵ Protocatechuic acid may attenuate osteoclast activity differentiation, as shown in animal-based trials, which consequently aids in the reduction of bone loss.⁶⁶ Alternatively,

TABLE 7 Model results for white wine

	Mechanisms of action	% of total association ^a	Beta coefficients of overall model
Visceral adiposity	(1) HDL cholesterol levels:	322%	$\beta_{\text{total}} = -0.01$ n.s.
	(2) APOA levels:	189%	
	(3) Urate levels:	111%	
	(4) Vitamin D levels:	44%	
	(5) cystatin C levels:	44%	
	(6) IGF-I levels:	44%	
	(7) HbA1c levels:	33%	
Subcutaneous adiposity	(1) HDL cholesterol levels:	267%	$\beta_{\text{total}} = -0.01$ n.s.
	(2) Urate levels:	133%	
	(3) cystatin C levels:	89%	
	(4) APOA levels:	89%	
	(5) creatinine levels:	89%	
	(6) Vitamin D levels:	56%	
	(7) IGF-I levels:	56%	
	(8) GGT levels:	44%	
Lean muscle Mass	(1) HDL cholesterol levels:	83%	$\beta_{\text{total}} = -0.01^{***}$
	(2) Urate levels:	33%	
	(3) GGT levels:	25%	
	(4) creatinine levels:	17%	
Bone mineral density	(1) Unspecified:	106%	$\beta_{\text{total}} = 0.05^{***}$
	(2) HDL cholesterol levels:	20%	
	(3) Urate levels:	12%	

Notes: *, **, ***, and n. s. denote $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$, and non-significant beta coefficients, respectively.

Abbreviations: APOA, apolipoprotein A; GGT, gamma glutamyltransferase; HbA1c, glycated hemoglobin; HDL, high-density lipoproteins; IGF-1, insulin-like growth factor 1.

^aThe percentage of association for the mediation associations (serum biomarkers) are with respect to the total association that each serum biomarker composes. The table does not include non-significant serum biomarkers.

there could be an unprecedented bone-supporting polyphenol that could be unique to white wine.

This study was not without limitations. First, DEXA-based body morphometry was cross-sectional, although this direct body composition measurement method performed well as an outcome because the data was collected several years after the predictors tested. Second, alcohol/dietary data were self-reported and may represent crude estimations. Third, the study included a homogeneous sample, consisting exclusively of older participants of European descent with greater educational attainment and lower-to middle-class socioeconomic statuses. Thus, these findings have limited generalizability to younger, non-White racial/ethnic, less educated, and higher socioeconomic status populations. Fourth, the study sample showed notably skewed sex-specific distribution differences in the preference for certain alcoholic beverage types, where women drank significantly more wine than male counterparts, while men drank significantly more beer/cider than female

counterparts. These skewed distributions by sex in preferred alcoholic beverage types may have influenced the alcoholic beverage type-body composition relationships. However, sex was included as a control variable in the overall structural equation models to reduce the influence of these skewed sex-specific distributions, thereby enhancing the internal validity of the models. Nevertheless, the study also contained several strengths. First, the measures obtained for body composition were directly quantified via DEXA rather than by indirect anthropometric measures (e.g., body mass index), which provided greater accuracy than indirect measures of body composition.^{67,68} Additionally, the included sample size was large ($n = 1869$) and tracked the cohort for up to 10 years, which provided stronger generalizability of these findings to the target population.

These findings may have several clinical implications. First, this study's findings regarding the inverse association between red wine consumption and lower visceral adiposity were consistent

TABLE 8 Model results for red wine

	Mechanisms of action	% of total association ^a	Beta coefficients of overall model
Visceral adiposity	(1) HDL cholesterol levels:	148%	$\beta_{\text{total}} = -0.02^{***}$
	(2) APOA levels:	87%	
	(3) cystatin C levels:	35%	
Subcutaneous adiposity	(1) HDL cholesterol levels:	57%	$\beta_{\text{total}} = -0.05^{***}$
	(2) cystatin C levels:	33%	
	(3) APOA levels:	18%	
	(4) albumin levels:	12%	
	(5) CRP levels:	10%	
	(6) GGT levels:	6%	
Lean muscle Mass	(1) HDL cholesterol levels:	80%	$\beta_{\text{total}} = -0.02^{***}$
	(2) GGT levels:	20%	
Bone mineral density	(1) HDL cholesterol levels:	275%	$\beta_{\text{total}} = -0.004$ n.s.
	(2) ALP levels:	200%	

Notes: *, **, ***, and n. s. denote $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$, and non-significant beta coefficients, respectively. Model controlled for basic demographic (age, sex, education, socioeconomic status, height) and lifestyle (moderate and vigorous physical activity levels, sleep duration, sunlight exposure, tobacco smoking status) variables.

Abbreviations: APOA, apolipoprotein A; ALP, alkaline phosphatase; CRP, C-reactive protein; GGT, gamma glutamyltransferase; HDL, high-density lipoproteins.

^aThe percentage of association for the mediation associations (serum biomarkers) are with respect to the total association that each serum biomarker composes. The table does not include non-significant serum biomarkers.

TABLE 9 Model results for spirits

	Mechanisms of action	% of total association ^a	Beta coefficients of overall model
Visceral adiposity	(1) Urate levels:	71%	$\beta_{\text{total}} = 0.01^{***}$
	(2) cystatin C levels:	29%	
Subcutaneous adiposity	(1) Urate levels:	60%	$\beta_{\text{total}} = 0.02^{***}$
	(2) cystatin C levels:	40%	
Lean muscle Mass	(1) Urate levels:	100%	$\beta_{\text{total}} = 0.004^{**}$
Bone mineral density	(1) Urate levels:	100%	$\beta_{\text{total}} = 0.01^*$

Notes: *, **, ***, and n. s. denote $p \leq 0.05$, $p \leq 0.01$, $p \leq 0.005$, and non-significant beta coefficients, respectively. Model controlled for basic demographic (age, sex, education, socioeconomic status, height) and lifestyle (moderate and vigorous physical activity levels, sleep duration, sunlight exposure, tobacco smoking status) variables.

^aThe percentage of association for the mediation associations (serum biomarkers) are with respect to the total association that each serum biomarker composes. The table does not include non-significant serum biomarkers.

with prior findings that red wine may be protective against weight gain and obesity-related health risks,⁶⁹⁻⁷¹ especially in older adults.⁷¹ The associations between beer and spirit consumptions and greater adiposity were also consistent with prior studies.^{20,54} Thus, for older adult drinkers, registered dietitians and physicians may find benefit in inquiring about the specific alcohol types consumed, as opposed to inquiring merely about holistic alcohol intake, as part of patient consultations or routine visits. Second, practitioners should encourage older adult alcohol drinkers to consume alcohol in moderation and to consume a higher

proportion of total alcohol intake from red wine while concomitantly discouraging the usage of beer and spirits in order to help reduce the risk for weight gain, adiposity-associated health risks. Third, for older adult drinkers who have an elevated risk for osteopenia-associated diseases, practitioners should consider encouraging this population of older adult drinkers to consume a higher proportion of total alcohol intake from white wine. However, more research is needed to examine how white wine may be linked to bone mineral density before practitioners can confidently make such recommendations. Furthermore, these

recommendations are not generalizable to older adult nondrinkers, as prior studies have recommended that abstainers of alcohol should not commence alcohol consumption solely for the proposed health benefits.^{72,73}

5 | CONCLUSION

Despite these limitations, these models offered new *post-priori* hypotheses that encourage future experimental trials to further investigate whether beer/cider, red wine, white wine/Champagne, and spirits may differentially influence body composition, and how these effects could be mechanistically occurring at the biomolecular level. These results suggest that drinking more beer and spirits could be linked to greater adiposity-associated weight gain in older White adults, while the consumption of red wine, but not white wine, could be inversely linked to adiposity-associated weight gain in older White adults. These results furthermore imply that the consumption of white wine in moderation may help curb age-associated bone mineral loss in older White adults. Future work should confirm variable loadings in these models and should also work to determine whether older adults that belong to non-White race/ethnicity groups as well as individuals in midlife at risk for metabolic disorders show different physiology and lifestyle association patterns. Additionally, more research is needed on different types of alcoholic beverages consumed as well as distinguishable effects that different alcoholic beverage types could have on health.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

AUTHOR CONTRIBUTIONS

The authors confirm contribution to the paper as follows: study conception and design: Brandon S. Klinedinst, Auriel A. Willette; statistical analysis and results interpretation: Brittany A. Larsen, Brandon S. Klinedinst, Scott T. Le, Colleen Pappas, Tovah Wolf, Nathan F. Meier, Ye-Lim Lim; draft manuscript preparation: Brittany A. Larsen, Brandon S. Klinedinst, Colleen Pappas, Tovah Wolf, Auriel A. Willette; oversaw the study: Auriel A. Willette. All authors reviewed the results and approved the final version of the manuscript.

ORCID

Brittany A. Larsen  <https://orcid.org/0000-0002-6088-5176>

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