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## Prospective Study of Alcohol Consumption and Self-reported Hearing Loss in Women

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### Abstract

Chronic excess alcohol intake has been associated with irreversible hearing loss and acute alcohol intake may temporarily impair auditory function; however, some evidence suggests that long-term moderate alcohol intake may be related to lower risk of hearing loss. This study prospectively examined the association between total alcohol and individual alcoholic beverage consumption and risk of hearing loss in women. Data were prospectively collected from 65,424 participants in the Nurses' Health Study II (NHS II) aged 27–44 years at baseline (follow-up 1991–2009). Alcohol consumption was assessed using a validated questionnaire every 4 years. An incident case was defined as self-reported hearing problem that began after 1991. Cox proportional hazards multivariate regression was used to adjust for potential confounders. During 1,024,555 person-years of follow-up, 12,384 cases of hearing loss occurred. After multivariate adjustment, there was no significant association between total alcohol consumption and risk of hearing loss. In exploratory analyses, beer consumption was associated with increased risk and wine consumption was associated with reduced risk. No significant association was observed for consumption of liquor. Total alcohol consumption is not associated with risk of hearing loss in women. The modest associations observed for beer (direct) and wine (inverse) may be due to chance or residual confounding but merit further study.

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## Keywords

Alcohol; hearing loss; prospective study; epidemiology

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## Introduction

Hearing loss is a highly prevalent and disabling chronic condition that can impair communication, quality of life and health (Agrawal et al., 2008). Although it is often perceived as an inevitable companion of aging, recent evidence suggests modifiable factors can potentially aid in prevention or slow progression of hearing loss (Curhan et al., 2012; Durga et al., 2007; Fransen et al., 2008).

Alcohol consumption may influence several mechanisms that have been proposed to underlie age-related hearing decline. Compromise of the cochlear blood supply, with resultant hypoxia and ischemic damage, oxidative stress and associated mitochondrial dysfunction and loss of neurosensory cochlear cells, and neurodegeneration of central auditory pathways, may all contribute to hearing loss during aging (Fitzpatrick and Eviatar, 1980; Gates and Mills, 2005; Han and Someya, 2013; Meerton et al., 2005; Someya and Prolla, 2010). Alcohol intake may protect cochlear blood flow (Seidman et al., 1999), promote cytoprotective and anti-inflammatory mechanisms that strengthen cellular survival pathways, and directly enhance neuroprotective mechanisms that preserve hearing (Collins et al., 2009). However, alcohol intake may also adversely alter central processing of auditory information (Fitzpatrick and Eviatar, 1980; Meerton et al., 2005; Upile et al., 2007).

Although chronic excess alcohol intake has been associated with irreversible hearing loss (Rosenhall et al., 1993) and acute alcohol intake may temporarily impair auditory processing and worsen auditory thresholds (Fitzpatrick and Eviatar, 1980; Hienz et al., 1989; Kahkonen et al., 2005; Liu et al., 2004; Pearson et al., 1999; Robinette and Brey, 1978; Upile et al., 2007), some evidence suggests that long-term moderate alcohol intake may protect against hearing loss (Gopinath et al., 2010; Popelka et al., 2000). Reversible auditory changes that have been demonstrated following acute alcohol ingestion include temporary worsening of auditory thresholds, poorer speech discrimination, elevation of the acoustic reflex threshold, and impaired processing of tones, frequency change and novel sounds (Fitzpatrick and Eviatar, 1980; Kahkonen et al., 2005; Pearson et al., 1999; Robinette and Brey, 1978; Upile et al., 2007). In guinea pigs, acute intraperitoneal alcohol injection was associated with temporary worsening of hearing thresholds, however no significant change in hearing thresholds was observed following 60 days of alcohol intake administered via drinking water (Liu et al., 2004). In humans, some cross-sectional studies reported an inverse association between alcohol consumption and hearing loss (Gopinath et al., 2010; Popelka et al., 2000), although others did not (Brant et al., 1996; Sousa et al., 2009). In a recent large population-based cross-sectional study of 164,770 adults in the UK aged 40 and 69 years who completed a speech-in-noise hearing test (the Digit Triplet Test), those who were current consumers of alcohol were approximately 40% less likely to have a hearing loss than lifetime teetotalers, and the inverse association was similar across 3 levels of alcohol intake: from 1–118.4 g/week, 118.4–196.8 g/week, and 196.8 g/week or more (Dawes et al., 2014)

Few studies have examined this relation prospectively. In a prospective study of 870 men and women age 49 and older, no association was observed between alcohol consumption and the 5-year incidence of measured hearing loss, however there was insufficient power to be conclusive (Gopinath et al., 2010). In a prospective study of 26,809 older men, we found no association between total alcohol consumption and the risk of self-reported hearing loss (Curhan et al., 2011). To our knowledge, this relation has not been studied prospectively in younger women. Thus, the influence of long-term alcohol consumption on hearing remains unclear. Therefore, we prospectively examined the relation of alcohol consumption and risk of hearing loss in 65,424 women in the Nurses Health Study II (NHS II). In addition, as the relations between alcohol consumption and other health outcomes have varied by type of beverage consumed (Choi et al., 2004; Wannamethee et al., 2003), we explored the relations between beer, white wine, red wine and liquor consumption and risk of hearing loss.

## Methods

### Study Participants

The Nurses' Health Study II is an ongoing cohort study of 116,430 female nurses who were aged 25 to 42 years and living in 14 US states at cohort inception in 1989. Participants have been followed by biennial mailed questionnaires that elicit information on dietary and lifestyle factors and various health outcomes; the follow-up rate over 22 years exceeds 90% of eligible person-time. Detailed information on diet, including alcohol consumption, has been obtained every 4 years (Questionnaires can be viewed online at [www.channing.harvard.edu/nhs/?page\\_id=70](http://www.channing.harvard.edu/nhs/?page_id=70)). The 2009 long-form questionnaire asked women whether they have a hearing problem and at what age a change in hearing was first noticed. Those who reported a hearing problem that began before the study baseline or a history of cancer other than non-melanoma skin cancer (due to possible exposure to ototoxic chemotherapeutic agents) were excluded from the analysis. The number of women included in the analysis was 65,424. The final study population was arrived at as follows: 90,488 participants answered the 2009 long form questionnaire, the version that included the hearing question; 12,156 participants were excluded from the analysis because they had not answered the 1991 baseline FFQ; 2,584 were excluded because they reported hearing loss prior to 1991 biennial questionnaire (study baseline); 9,191 were excluded because they did not answer the hearing loss questions; 173 reported mild, moderate or severe hearing loss but were excluded because they did not report the age of onset; 859 reported cancer (other than non-melanoma skin cancer) and were excluded due to the possibility of exposure to ototoxic chemotherapy; and 101 participants were pregnant at baseline and again prior to onset of hearing loss during follow-up, thus were excluded.

Although some participants were lost to follow-up since the inception of this cohort and/or did not answer the 2009 questionnaire, the baseline characteristics of participants who did and did not answer the 2009 questionnaire did not differ appreciably (data not shown). The study protocol was approved by the Institutional Review Board of the Partners Health Care System.

### Assessment of Alcohol Consumption

In 1991, and every 4 years thereafter, participants were asked to complete a food frequency questionnaire (FFQ) on their intake of specific foods and beverages, each with a specified portion size, during the previous 12 months. Separate questions asked about the consumption of regular beer (1 glass, bottle, can), light beer (1 glass, bottle, can), red wine (4 oz [120 mL] glass), white wine (4 oz [120 mL] glass), and liquor (e.g. whiskey, gin) (1 drink or shot). The response categories were “never or <once/month,” “1–3/month,” “1/week,” “2–4/wk,” “5–6/wk,” “1/day,” “2–3/day,” “4–5/day,” and “6+/day.” Regular beer was considered to contain 13g of alcohol per serving, light beer 11g, wine 11g per 4 oz serving, and spirits 14g per serving. Total grams per day of alcohol were calculated by summing the frequency of consumption multiplied by the alcohol content of the reported beverages. Information on alcohol consumption obtained in this way was validated in a similar cohort of women by comparison of the information collected on 2 FFQs with four 1-week diet records collected 3 months apart; the correlation for alcohol consumption between questionnaires and dietary records was  $r = 0.90$ . Reported alcohol intake was significantly correlated with serum high-density lipoprotein levels ( $r = 0.40$ ;  $p < 0.001$ ) (Giovannucci et al., 1991).

### Ascertainment of Hearing Loss

Self-reported hearing loss, the primary outcome, was ascertained based on responses to the 2009 questionnaire. Participants responded to the questions: “Do you have a hearing problem?” (“no,” “mild,” “moderate,” “severe”) and, “If so, at what age did you first notice a change in your hearing?”

We defined a case as a hearing problem first noticed after 1991. Although hearing loss is often insidious in onset, incident cases were defined as hearing loss at the age it was first noticed by the participant. We did not have information on severity of hearing loss at the time of onset, thus we were not able to perform prospective analyses that considered severity of hearing loss as the outcome. Pure-tone audiometry is considered the gold standard for hearing loss evaluation; however, cost and logistic limitations precluded such assessment in this large population. Previous evaluations of the reliability of using a single question to assess self-reported hearing loss have reported this method to be a reasonably reliable measure (Gomez et al., 2001; Schow and Gatehouse, 1990; Sindhusake et al., 2003). A validity study in a population of individuals aged 30–65 years evaluated the performance of a single question compared with the results of pure-tone audiometry and found the sensitivity and specificity were 79.6% and 77.4%, respectively (Ferrite et al., 2011).

### Assessment of covariates

We considered covariates that may be potential confounders of the relation between alcohol consumption and hearing loss. These included: age (Agrawal et al., 2008); race (Agrawal et al., 2008), as assessed from the baseline questionnaire and categorized as Caucasian/White, Hispanic, African-American, Asian, Native American or Other; smoking status (Itoh et al., 2001), based on updated self-reported smoking status on the biennial questionnaires and categorized as never smoker, past smoker, current smoker (1–4 cigarettes per day), current smoker (5–14 cigarettes per day), or current smoker (15+ cigarettes per day); body mass

index (Curhan et al., 2013), calculated at each 2-year cycle from the current weight and the height reported on the baseline questionnaire and categorized in the primary analysis as less than 25, 25–29, 30–34, 35–39, 40+ and also continuously in additional analyses; waist circumference (Curhan et al., 2013), assessed at 2 time points, 1993 and 2005, when participants were sent a measuring tape and asked to report their waist circumference to the nearest ¼ inch, and categorized in the primary analysis as less than 71 cm, 71–79 cm, 80–88 cm, 88+ cm and also continuously in additional analyses; level of physical activity (Curhan et al., 2013), assessed in 1991, 1997, 2001, 2005, and 2009, when participants completed a physical activity grid and reported average time spent per week in the previous year in a variety of recreational activities (e.g. walking for exercise, walking to work, hiking outdoors, jogging, running, bicycling, calisthenics, aerobics, aerobic dance, rowing machine tennis, squash, racquetball, lap swimming, other aerobic activity, lower-intensity exercise such as yoga or stretching, other vigorous activities such as weight training or resistance exercise, and flights of stairs climbed daily), calculated as energy expenditure in metabolic-equivalent tasks (METs) in hours/week using the Ainsworth classification (Ainsworth et al., 2011), and categorized in quintiles according to total METS expended per day; intake of folate (Durga et al., 2007), categorized in categories; intake of vitamin B12 (Houston et al., 1999), categorized in quintiles; intake of magnesium (Haupt et al., 2003), categorized in quintiles; intake of potassium, categorized in quintiles; intake of total vitamin A (Le Prell et al., 2007), categorized in quintiles of retinol activity equivalents (a measure of vitamin A activity based on the capacity of the body to convert provitamin carotenoids); history of hypertension (Gates et al., 1993), categorized as “yes” or “no” based on participant report of whether a clinician had made a new diagnosis of hypertension during the preceding 2 years and updated biennially; history of diabetes (Bainbridge et al., 2008), categorized as “yes” or “no” based on participant report of whether a clinician had made a new diagnosis of diabetes during the preceding 2 years and updated biennially; acetaminophen use and ibuprofen use (Curhan et al., 2012), categorized in days per week of use based on frequency of use reported beginning in 1995 and every 2 years thereafter; history of tinnitus (Nondahl et al., 2011), categorized as “yes” or “no” based on participants’ response to the question on 2009 questionnaire that queried, “In the past 12 months, have you had ringing, roaring, or buzzing in your ears?” (never, once per week or less, a few days per week, or daily) and, “At what age did this first begin?” (age <30, 30–39, 40–49, 50–59, and 60 years) with tinnitus defined as participant report of tinnitus occurring a few days per week or daily. Covariate information that was obtained from the biennial questionnaires was updated with each new analytic time period. Intakes of dietary factors were calculated from the FFQ and the intakes of the nutrients of interest were calculated by multiplying the portion size of a single serving of each food by its reported frequency of intake, multiplying the total amount consumed by the nutrient content of the food, and then summing the nutrient contributions of all food items, using US Department of Agriculture food composition data (Chug-Ahuja et al., 1993; Mangels et al., 1993; Nutrient Data Laboratory, 1999). Intakes of all nutrients were adjusted for total energy intake using the residual method (Willett, 1998). Intake of dietary supplements as assessed by the FFQ was also taken into account.

In 2012, a web-based Hearing Study Supplementary Questionnaire (HSSQ) that elicited additional information related to hearing health was completed by a subcohort of

participants for whom we had email addresses (n=23,815). Information on degree, shape, and pattern of onset of hearing loss, family history, evaluation and treatment history, potential underlying causes, tinnitus, and detailed information on lifetime recreational, occupational and impulse noise exposure was obtained.

To assess whether overall diet quality influenced the relation between intake of the various alcoholic beverages and hearing loss, we performed a secondary analysis that adjusted for the Alternate Healthy Eating Index (AHEI-2010), a measure based on the intake of foods and nutrients that are predictive of risk of major chronic diseases, including cardiovascular disease and diabetes (Chiuve et al., 2012).

### Statistical Analysis

Descriptive analyses for baseline characteristics in 1991 were examined by categories of total alcohol and also specific beverage intake. All analyses were prospective using data on alcohol consumption and covariates that were collected before the onset of hearing loss. Total alcohol consumption was calculated as the sum of all the alcoholic beverage types and was categorized according to level of average daily alcohol intake in grams: none, 0.1 to 4.9 grams/day, 5.0 to 14.9 grams/day, 15–29.9 grams/day, and 30 grams/day or more. Consumption of the individual alcoholic beverages (beer, wine, liquor) was categorized according to frequency of intake: less than one serving per month, 1 serving/month to 1 serving/week, 2–4 servings/week, 5 servings/week or more (Choi et al., 2004).

In our primary analyses, we modeled cumulative average of total alcohol consumption and of individual beverage consumption over follow-up to evaluate the relation of long-term exposure to alcohol and hearing loss. Thus, the incidence of hearing loss in each interval between FFQs was related to the cumulative average intake calculated from all preceding measures. In secondary analyses, we updated alcohol intake with each new assessment (simple updating) to examine the relation of the most recent alcohol intake and risk of hearing loss. Due to the possibility of pregnancy-related temporary changes in alcohol consumption, dietary habits, anthropometric measurements and other lifestyle habits, women who were pregnant at the start of each follow-up interval were skipped for that time period, but reentered the analyses in subsequent follow-up periods. Women pregnant at baseline were excluded from analyses that examined baseline characteristics.

For the primary analyses, we included women who reported tinnitus and adjusted for tinnitus in our analysis. However, as tinnitus is associated with hearing loss (Nondahl et al., 2011) and the relation between alcohol consumption and tinnitus remains unclear (Pugh et al., 1995), we performed secondary analyses that excluded participants who reported tinnitus at baseline or during follow-up that occurred more than once/week and before the onset of hearing problem (n=7,569).

Alcohol intake as well as dietary and other exposure information was updated every 4 years. The date reported for the onset of hearing loss was taken as the event date. Participants were censored at the report of cancer other than non-melanoma skin cancer, or the end of follow-up (date of return of the 2009 questionnaire), whichever came first. Cox proportional hazards regression models adjusted for potential confounders were used to estimate the

multivariate-adjusted relative risk (RR) of hearing loss by category of alcohol intake and individual beverage intake compared with those who did not. For all RRs, 95% confidence intervals were calculated and all *P* values were 2-sided. Statistical analyses were performed using SAS software, version 9.3 (SAS Institute Inc., Cary, North Carolina).

### Human Participant Protection

The study protocol was approved by the Institutional Review Board of the Partners Health Care System. Participants provided informed consent.

### Results

Characteristics of non-pregnant participants at baseline according to level of daily alcohol intake are summarized in Table 1. Among the women at baseline, 41% were non-drinkers, 39% consumed 0.1–4.9 g of alcohol per day, 16% consumed 5.0–14.9 g/d, 3% consumed 15.0 to 29.9 g/d, and 1% consumed 30 grams or more per day. Overall, non-drinkers of alcohol tended to be slightly heavier, less physically active, and more likely to have never smoked than women who consumed alcohol. Other health, dietary and lifestyle characteristics did not vary substantially according to level of total alcohol intake or intake of specific beverages (characteristics according to level of intake for each specific beverage are presented in supplementary Tables 1a–c).

We evaluated the consistency of alcohol intake over time by comparing the reported level of alcohol intake on the baseline 1991 FFQ and the 2003 FFQ (Table 2). Alcohol consumption remained relatively consistent over time; 54% of the women reported the same category of intake of total alcohol and 91% reported within one category higher or lower. Similarly, for beer, wine and liquor, 90–95% of the women reported intake within one category.

After 1,024,555 person-years of follow-up, 12,384 cases of hearing loss were reported to have occurred. After adjusting for potential confounders, there was no significant association between total alcohol consumption and the risk of hearing loss (Table 3).

The age- and multivariate-adjusted relative risks (RRs) of hearing loss according to level of consumption of each specific beverage are presented in Table 4. After adjusting for potential confounders as well as the other alcoholic beverages, more frequent consumption of beer was independently associated with an increased risk of hearing loss. Compared with women who consumed less than one serving of beer per month, the multivariate-adjusted RR for hearing loss for those who consumed 5+ servings/week of regular beer was 1.15 (95% CI 1.04,1.28; *p*-trend<0.001). In contrast, more frequent consumption of wine was independently associated with a lower risk of hearing loss. Compared with women who consumed less than one serving of wine per month, the multivariate-adjusted RR for hearing loss was 0.84 (95% CI 0.77,0.92) for those who consumed 5+ servings/week (*p*-trend<0.001). No independent association was observed for the consumption liquor (*p*-trend=0.09).

The relations between total alcohol or specific type of alcoholic beverage consumed and hearing loss did not vary by age (*p*-interaction >0.19). The multivariate adjusted results were

not appreciably changed after adjusting for overall dietary quality or in models that excluded women with tinnitus (data not shown). Results from analyses that modeled consumption of total alcohol, as well as consumption of the individual alcoholic beverages, using simple updating or using baseline intake measures did not materially differ.

In the subcohort of women for whom we had detailed information on self-reported lifetime noise exposure (n=23,815), the proportion of women who reported loud noise exposure was small (<4%) and did not differ by level of total alcohol or individual beverage intake (data not shown).

## Discussion

In this study of 65,424 women, we found no overall association between total consumption of alcohol and the risk of hearing loss. In exploratory analyses, we observed an increased risk of hearing loss associated with consumption of beer, and the risk tended to increase with increasing beer consumption. We also observed an inverse association between wine consumption and risk of hearing loss. No significant association was observed with consumption of liquor.

In cross-sectional studies, the prevalence of hearing loss was higher in those with chronic alcohol abuse (Ribeiro et al., 2007; Rosenhall et al., 1993). Some cross-sectional studies have suggested an inverse association between moderate alcohol consumption and hearing loss (Fransen et al., 2008; Itoh et al., 2001; Popelka et al., 2000), however, others have found no association (Helzner et al., 2005; Sousa et al., 2009). Notably, as these studies were cross-sectional, no conclusions regarding temporal relations can be drawn. Although no associations were observed between moderate alcohol consumption and the risk of audiometrically measured hearing loss in two other prospective studies, the sample sizes (n=531 men; n= 870 men and women) and case numbers in these studies were small; thus, these studies may not have had sufficient power to be conclusive (Brant et al., 1996; Gopinath et al., 2010). The current report is the first large prospective study of the relation between the consumption of alcohol and risk of hearing loss in younger women. The findings of no overall association between total alcohol consumption and risk of hearing loss are consistent with our previous observations in a prospective study of 26,809 older men, aged 40–75 years at study baseline and followed for 18 years (Curhan et al., 2011). Overall, there does not appear to be an association between total alcohol consumption and risk of hearing loss.

Our findings that the relation between alcohol consumption and risk of hearing loss might potentially vary by type of alcoholic beverage are intriguing. The differences in the associations observed for individual alcoholic beverages may be due to other unmeasured factors associated with choice of beverage, such as drinking patterns or aspects of lifestyle correlated with choice of beverage. Nevertheless, each type of alcoholic beverage has many non-alcohol components that could potentially be associated with risk of hearing loss. For example, the protective association observed with wine consumption may be mediated by the antioxidants, vasorelaxants, or factors that stimulate anti-aggregatory mechanisms that can be found in wine. Beer may be associated with larger increases in serum uric acid levels



than wine or liquor, and also contains factors related to the fermentation process that differ from wine or liquor. Whether these factors are associated with hearing loss remains unknown, however these findings present intriguing possibilities for further study. Although the alcohol content per serving of wine is comparable to that per serving of beer, previous studies have observed other disease outcomes that have varied by type of alcoholic beverage consumed (Choi et al., 2004; Djousse et al., 2002). A prospective study on alcohol consumption at midlife and successful aging in women in the Nurses' Health Study I found that the associations between alcohol consumption and successful aging varied by type of alcoholic beverage consumed; wine, but not beer or liquor, was associated with increased odds of successful aging. We previously found no association between intake of individual alcoholic beverages and hearing loss in older men (Curhan et al., 2011). Whether a relation between specific alcoholic beverage intake and risk of hearing loss varies by sex is unclear. In addition, we did not have information on exposure to noise, an important risk factor for hearing loss, in the full cohort; in the subcohort of participants who provided information on lifetime noise exposure, the proportion of women who reported loud noise exposure was small and did not differ by level of total alcohol or individual beverage intake. Further studies are needed to assess whether non-alcohol ingredients in beer or wine, and/or other lifestyle factors associated with consumption of these beverages, are associated with risk of hearing loss.

Our study has limitations. We relied on self-reported consumption of alcohol, the validity of which was described above. Assessment of hearing loss was also based on self-report. Although sudden hearing loss does occur, hearing decline is commonly insidious in onset, thus there is imprecision in the assessment of date of onset. Standard pure-tone audiometry is the gold standard measure for evaluation of hearing loss; however, assessment of hearing loss based on self-report has been found to be reasonably reliable (Coren and Hakstian, 1992; Ferrite et al., 2011; Gomez et al., 2001; Schow and Gatehouse, 1990). Assessment of hearing loss was based on participant report in 2009 regarding date of onset, however all information on exposures and covariates was collected prior to reported date of hearing loss onset; therefore, the relations were examined prospectively. We had limited power to evaluate heavy drinking; only 1% of women reported alcohol consumption of 30 g/d or more. We did not have information on hearing loss severity at the time of onset, thus severity of hearing loss could not be considered. Our study was limited to predominantly non-Hispanic white women and we cannot necessarily generalize these findings to women outside of this age range, thus further research in additional populations is warranted.

In conclusion, total alcohol consumption is not associated with risk of hearing loss in women. The modest associations we observed for beer (direct) and wine (inverse) may be due to chance or uncontrolled confounding factors but merit further study.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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**Table 1**  
Age-Adjusted Baseline Characteristics of Women in 1991 According to Category of Total Alcohol Intake

	Total alcohol intake, in grams per day				P-value	
	None (n=25850)	1-4.9 g/d (n=24654)	5-14.9 g/d (n=9729)	15-29.9 g/d (n=1773)		30+ g/d (n=692)
Age, years <sup>a</sup>	36.6(4.6)	36.2(4.6)	36.4(4.7)	37.1(4.6)	37.7(4.3)	<0.001
Body mass index, kg/m <sup>2</sup>	25.2(5.8)	24.2(4.9)	23.2(3.9)	23.2(3.8)	23.8(4.4)	<0.001
Waist Circumference (1993), cm	79.6(13.6)	77.3(12.1)	75.7(10.6)	75.9(10.4)	78.1(11.6)	<0.001
Physical activity, MET <sub>s</sub> <sup>b</sup>	18.5(25.4)	21.5(26.4)	24.3(29.1)	24.7(28.3)	21.8(27.6)	<0.001
Smoking status						
- Never, %	75.5	64.7	53.0	42.3	32.6	<0.001
- Past, %	16.0	23.9	31.8	36.1	30.8	<0.001
- Current, %	8.4	11.2	15.0	21.6	36.5	<0.001
Alcohol, g/d	0.0(0.0)	2.1(1.2)	8.8(2.7)	20.5(4.7)	40.9(13.5)	<0.001
Vitamin B12 intake, mcg/d	9.7(13.7)	9.5(11.1)	9.4(11.4)	9.1(12.8)	8.8(10.1)	0.01
Folate intake, mcg/d	462(279)	458(256)	452(242)	425(212)	408(206)	<0.001
Potassium intake, mg/d	2891(558)	2979(527)	2998(498)	2967(483)	2804(487)	<0.001
Magnesium intake, mg/d	310(77)	319(72)	323(69)	321(63)	313(62)	<0.001
Vitamin A intake, IU/d	12682(8428)	12825(7677)	12686(7650)	11912(7088)	10736(6646)	<0.001
History of hypertension, %	7.2	5.6	4.8	6.5	8.1	0.004
History of diabetes, %	1.1	0.6	0.4	0.5	0.6	<0.001
Caucasian, %	93.3	96.1	96.8	96.8	96.4	<0.001
Ibuprofen use 2+ d/wk (1995), %	17.9	19.5	20.6	22.8	21.6	<0.001
Acetaminophen use 2+ d/wk (1995), %	21.3	20.7	19.3	19.8	23.0	0.03
History of Tinnitus, %	2.4	1.9	1.6	2.0	1.3	<0.001
AHEI-2010 <sup>c</sup> score	45.7(10.8)	49.8(10.3)	55.1(10.3)	51.7(10.7)	43.2(9.9)	<0.001

Values are means(SD) or percentages and are standardized to the age distribution of the study population. Values of polytomous variables may not sum to 100% due to rounding. Women who were pregnant at baseline are not included.

<sup>a</sup> Value is not age adjusted

<sup>b</sup> Metabolic equivalents from recreational and leisure-time activities.

<sup>c</sup> AHEI-2010 = Alternate Healthy Eating Index-2010

**Table 2**

Comparison of Alcohol Intake at 1991 Baseline with Alcohol Intake in 2003

<b>Beverage</b>	<b>% same category</b>	<b>% within 1 category</b>
Total alcohol	54	91
Beer	36	90
All wine	58	91
Liquor	73	95

Categories of alcohol intake (grams/day) were: 0, 0.1–4.9, 5.0–14.9, 15.0–29.9, 30.0+ Categories of beer, wine, and liquor (servings): <1/month, 1/month–1/week, 2–4/week, 5+/week

**Table 3**

Relative Risks of Hearing Loss According to Alcohol Intake

Alcohol Intake (grams/day)	Cases	Person-years	Age-adjusted Relative Risk (95% CI)	Multivariate-adjusted Relative Risk <sup>a</sup> (95% CI)
0	3382	320860	1.00 (ref)	1.00 (ref)
0.1–4.9	5997	470357	0.98 (0.94,1.03)	1.00 (0.96,1.04)
5.0–14.9	2349	183901	0.95 (0.90,1.00)	0.99 (0.94,1.05)
15.0–29.9	530	39200	0.91 (0.83,1.00)	0.93 (0.85,1.02)
30.0+	126	10237	0.93 (0.77,1.11)	0.91 (0.76,1.09)
p-trend			0.01	0.09

<sup>a</sup> Adjusted for age, body mass index, waist circumference, physical activity, folate, vitamin B12, magnesium, potassium, vitamin A, smoking, hypertension, diabetes, race, nonsteroidal anti-inflammatory use, acetaminophen use, and tinnitus.

**Table 4**

Relative Risks of Hearing Loss According to Individual Alcoholic Beverage Intake

Frequency of Intake (servings)	Cases	Person-years	Age-adjusted Relative Risk (95% CI)	Multivariate-adjusted Relative Risk <sup>a</sup> (95% CI)
<b>Beer</b>				
<1/month	7598	635644	1.00 (ref)	1.00 (ref)
1/mo–1/wk	3409	274238	1.00 (0.96,1.04)	1.04 (1.00,1.09)
2–4/wk	961	78464	1.06 (0.99,1.13)	1.12** (1.04,1.20)
5+/wk	402	32848	1.13* (1.02,1.25)	1.15* (1.04,1.28)
p-trend			<0.001	<0.001
<b>Wine</b>				
<1/month	5594	475704	1.00 (ref)	1.00 (ref)
1/mo–1/wk	4722	387693	0.93** (0.89,0.96)	0.94* (0.90,0.98)
2–4/wk	1408	106670	0.90** (0.85,0.95)	0.93* (0.87,0.99)
5+/wk	651	52183	0.81** (0.75,0.88)	0.84** (0.77,0.92)
p-trend			<0.001	<0.001
<b>Liquor</b>				
<1/month	9157	762476	1.00 (ref)	1.00 (ref)
1/mo–1/wk	2752	219965	1.00 (0.95,1.04)	1.00 (0.95,1.05)
2–4/wk	322	27424	0.92 (0.82,1.03)	0.93 (0.83,1.04)
5+/wk	144	11990	0.95 (0.81,1.12)	0.93 (0.78,1.09)
p-trend			0.23	0.09

<sup>a</sup> Adjusted for age, body mass index, waist circumference, physical activity, folate, vitamin B12, magnesium, potassium, vitamin A, smoking, hypertension, diabetes, race, nonsteroidal anti-inflammatory use, acetaminophen use, tinnitus, and intake of the other types of alcoholic beverages.

\* Denotes statistically significant higher or lower hazard ratio compared with a reference level of 1.00 (\*,  $p < 0.05$ ; \*\*,  $p < 0.001$ ).