American Journal of Epidemiology
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Vol. 164, No. 11
DOI: 10.1093/aje/kwj323
Advance Access publication September 18, 2006

## Original Contribution

# Coffee and Sweetened Beverage Consumption and the Risk of Type 2 Diabetes Mellitus

## The Atherosclerosis Risk in Communities Study

**Nina P. Paynter[1], Hsin-Chieh Yeh[1], Sari Voutilainen[2], Maria Ines Schmidt[3,4], Gerardo Heiss[4], Aaron R. Folsom[5], Frederick L. Brancati[6], and W. H. Linda Kao[1]**

[1] Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.
[2] Research Institute of Public Health, University of Kuopio, Kuopio, Finland.
[3] Department of Social Medicine, School of Medicine, Federal University of Rio Grande do Sul, Porto Alegre, Rio Grande do Sul, Brazil.
[4] Department of Epidemiology, University of North Carolina School of Public Health, Chapel Hill, NC.
[5] Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, Minneapolis, MN.
[6] Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

*Received for publication November 1, 2005; accepted for publication April 25, 2006.*

The authors analyzed data from a prospective, community-based cohort to assess the risk of incident type 2 diabetes mellitus associated with coffee and sweetened beverage consumption. They included 12,204 nondiabetic, middle-aged men and women in the Atherosclerosis Risk in Communities (ARIC) Study (1987–1999). Consumption of each beverage was assessed by food frequency questionnaire and classified into categories of cups per day. They found an inverse association, after adjusting for potential confounders, between increased coffee consumption and risk of type 2 diabetes mellitus in men (for ≥4 cups (≥0.95 liter)/day compared with almost never: hazard ratio = 0.77, $p_{trend} = 0.02$) with no significant association in women (hazard ratio = 0.89, $p_{trend} = 0.32$) using a combination of self-report of physician-diagnosed diabetes, diabetes treatment, and a fasting or nonfasting blood glucose test. When self-reported diabetes or diabetes treatment alone was used, a stronger and significant inverse association was seen in men and women. Sweetened beverage consumption (men: hazard ratio = 1.03, $p_{trend} = 0.94$; women: hazard ratio = 1.01, $p_{trend} = 0.58$) showed no consistent association with the incidence of type 2 diabetes mellitus. In summary, increased coffee consumption was significantly associated with a decreased risk of diagnosed type 2 diabetes mellitus in community-based US adults.

beverages; coffee; cohort studies; diabetes mellitus, type 2

Abbreviations: ARIC, Atherosclerosis Risk in Communities; CI, confidence interval.

Of the approximately 18 million people with diabetes in the United States, the majority have type 2 diabetes mellitus (1). The identification of modifiable risk factors is of paramount importance. To date, obesity and physical activity are the only well-established modifiable risk factors (2–4). Nutrition and diet have received considerable attention with mixed results. One potential dietary component is nonalcoholic beverage consumption, including coffee and sweetened beverages. If these nonalcoholic beverages were found to be related to the development of type 2 diabetes,

Correspondence to Dr. W. H. Linda Kao, 615 North Wolfe Street, Room W6513, Baltimore, MD 21205 (e-mail: wkao@jhsph.edu).

the potential effect of dietary modification on diabetes risk could be quite large because of the widespread consumption of these beverages in the general population.

Previous prospective studies of coffee consumption have shown mixed results (5–13). A series of studies showing an inverse relation (5, 7, 9–13) have been reported, paralleled by those showing no association (6, 8), with a recent meta-analysis showing an overall inverse association (14). The studies have varied in consumption levels, as well as in assessment of incident type 2 diabetes and the measurement of important covariates, such as body mass index. There have also been substantial variations by geography, race, and gender in the populations studied. Overall, studies with reported physician diagnosis- or medical record-based outcomes have tended to find a stronger association than those with a screening test. To date, no large population-based, multiethnic US study has been conducted.

Sweetened beverage consumption has been shown to be associated with obesity incidence in children (15, 16), obesity prevalence cross-sectionally in young adults (17), and temporally with national obesity rates (18). Moreover, the association between sweetened beverage consumption (defined as sugar-sweetened soft drinks) and type 2 diabetes risk, as well as a relation between change in consumption and change in body mass index, was recently reported in the Nurses' Health Study (19).

We examined the association between coffee and sweetened beverage consumption in a US community-based cohort of middle-aged Black and White men and women. Our study addresses the question of whether the incidence of type 2 diabetes is related to consumption of the two beverages, whether these associations are independent of obesity and other potential confounders, and whether the relation is influenced by the method of ascertainment of diabetic status.

## MATERIALS AND METHODS

### Study population

The Atherosclerosis Risk in Communities (ARIC) Study is an ongoing, prospective study that examines clinical and subclinical atherosclerotic diseases in a cohort of 15,792 persons, aged 45–64 years at baseline examination, selected by probability sampling from four US communities: Forsyth County, North Carolina; Jackson, Mississippi; the northwest suburbs of Minneapolis, Minnesota; and Washington County, Maryland (20). Our analysis was based on a maximum of 9 years of follow-up, which included three clinic visits scheduled 3 years apart. For this analysis, we excluded participants who reported ethnicity other than Black or White ($n = 48$), had diabetes at baseline ($n = 1,860$), had missing exposure or diabetes information ($n = 134$), and had missing information on any of the potential confounders ($n = 683$), as well as individuals who did not return after the baseline visit ($n = 814$). Participants who did not return after the baseline visit were slightly more likely to drink 1 cup (0.24 liter) of coffee per day and more than 2 cups (0.48 liter) of sweetened beverages. After exclusions, 12,204 participants remained for this analysis.

### Exposure assessment

An interviewer-administered, modified version of the 61-item food frequency questionnaire developed by Willett et al. (21) was used to assess food and beverage consumption during the previous 12 months. Questions about intakes of beer, wine, and spirits were included separately. Participants were asked how often they had consumed 1 cup (8 ounces or 0.24 liter) of each of the beverages, and they could choose from nine responses (almost never, 1–3 per month, 1 per week, 2–4 per week, 5–6 per week, 1 per day, 2–3 per day, 4–5 per day, and $\geq$6 per day). Consumption of cups of coffee (caffeinated only) was analyzed using the following collapsed categories: almost never, <1 per day, 1 per day, 2–3 per day, and 4 or more per day. To create the sweetened beverage category, we combined the consumptions of fruit punch, nondiet soda, and orange or grapefruit juice using the midpoint daily consumption for the nine possible response categories.

### Covariate assessment

Information on age, gender, race, family history of diabetes, smoking status, alcohol intake, and education was obtained from interviews conducted at the baseline visit (22). Body mass index (weight (kg)/height (m)$^2$) and the waist: hip ratio were determined by the anthropometric measurements taken at the baseline clinic visit. Measurements were made with the participants wearing light-weight, nonconstricting clothing and no shoes following standard protocol. Physical activity was assessed using a modified interviewer-administered version of Baecke's questionnaire (23) and characterized as a leisure activity index with scores ranging from 1 to 5 (1 indicating least active). Total caloric intake and percent of diet composed of fiber were derived from the same food frequency questionnaire used in exposure assessment (21).

### Outcome assessment

Type 2 diabetes mellitus was defined as the presence of any one of the following: 1) fasting glucose of $\geq$126 mg/dl, 2) nonfasting glucose of $\geq$200 mg/dl, 3) current use of hypoglycemic medication, or 4) a positive response to the question, "Has a doctor ever told you that you had diabetes (sugar in the blood)?" Those with diabetes or unknown diabetes status at baseline were excluded from the prospective analyses. Individuals without diabetes at baseline who met any of these conditions during follow-up were considered incident cases. All incident cases were classified as type 2 diabetes, because the age of onset in this cohort was between 45 and 70 years.

### Alternate outcome definition

An alternate definition of type 2 diabetes was also developed for comparison with studies where diabetes was measured by self-report. For this purpose, type 2 diabetes was defined as either a positive response to the question, "Has a doctor ever told you that you had diabetes (sugar in the blood)?'', or current use of hypoglycemic medication. This criterion was then applied to each visit to generate the study population for the alternate analysis. Individuals with no self-report of diagnosed diabetes but with a high serum glucose level were considered nondiabetic until they reported a diagnosis or medication use. The population available for this analysis consisted of 12,747 people with 913 cases of incident self-reported diagnoses of type 2 diabetes.

### Statistical analysis

All analyses were stratified by gender both for comparison with previous studies and because the confounding effect of body mass index was different for the two groups. Incidence rate ratios were calculated for each consumption group by use of a person-years approach. For the standard type 2 diabetes definition, the date of diabetes incidence was estimated by linear interpolation using glucose values at the ascertaining visit and the previous one, as previously described (24). For the self-reported definition, incident cases were assigned to the midpoints between the last visit where they reported no physician-diagnosed diabetes or medication use and the first visit in which they reported either of the two.

Multivariate analysis was done using Cox proportional hazards models, and hazard ratios and corresponding 95 percent confidences intervals were estimated. Tests for trend were done using consumption categories as ordinal variables. For coffee, three models were assessed: 1) adjusted for study center, age, race, education, and family history of diabetes; 2) model 1 additionally adjusted for body mass index by quartile, waist:hip ratio quartile, total caloric intake quartiles, dietary fiber quartiles, smoking, alcohol consumption (never, former, current), leisure activity, and hypertension; and 3) model 2 without adjustment for body mass index or waist:hip ratio. An additional model for coffee consumption added serum magnesium, for its putative mediation role, to the fully adjusted model 2 above. For sweetened beverages, because body mass index and the waist:hip ratio were thought to mediate rather than confound the relation with incident type 2 diabetes, the following three models were assessed: 1) same as model 1 for coffee; 2) model 1 additionally adjusted for total caloric intake, dietary fiber, smoking, alcohol consumption, leisure activity, and hypertension; and 3) model 2 additionally adjusted for body mass index and waist:hip ratio quartiles.

All analyses were also performed using the alternate outcome of self-reported diabetes. All analyses were done using STATA, version 8, software (25).

## RESULTS

### Baseline characteristics and beverage consumption patterns

A total of 5,414 men and 6,790 women from the ARIC Study were included in the main analyses. The average age at baseline of both men and women was 54 years. Approximately 18 percent of the men and 25 percent of the women were African American. Both men and women had a mean body mass index of 27.2 kg/m$^2$, with a mean waist:hip ratio of 0.96 in the men and 0.89 in the women. Of the men, 29 percent reported never smoking at baseline compared with 53 percent never smokers in the women. As shown in table 1, drinking patterns differed both by beverage and across gender. For both coffee and sweetened beverages, there were more men in the highest consumption category and more women in the lowest. The median coffee consumption was 1 cup (0.24 liter) per day for both men and women.

### Relation of beverage consumption to type 2 diabetes risk

Over the follow-up interval, 1,437 participants developed incident diabetes (718 men and 719 women). Type 2 diabetes incidence rates differed among the six coffee consumption groups in both men and women (table 1). Although no statistically significant trend was seen in the age- and race-adjusted hazard ratio, in both men and women the groups with highest consumption had the lowest risk. For men, the crude incidence rates per 1,000 person-years from lowest coffee consumption to highest were 18.99, 18.46, 21.36, 15.84, and 14.78. For women, the crude incidence rates per 1,000 person-years were 15.86, 12.84, 15.70, 12.35, and 10.51, respectively. Sweetened beverage consumption showed a trend of increased risk with increased consumption in women and no association in men. Removing juice from the sweetened beverage definition did not affect the results.

### Association between coffee consumption and potential confounders and mediators of type 2 diabetes

In men, coffee consumption was strongly related to many characteristics previously found to be associated with incident type 2 diabetes in the ARIC Study cohort (table 2). Race, smoking status, educational level, alcohol consumption, hypertension, and energy intake were significantly associated with coffee consumption, with White participants and smokers drinking more coffee. In men, lower intake of fiber and younger age were also significantly associated with higher coffee consumption, while body mass index, obesity, and the waist:hip ratio were not. Serum magnesium levels also increased with increased coffee consumption, as expected. Higher coffee intake was also associated with lower serum insulin levels but showed no association with serum glucose.

Table 2 also shows the relation of coffee consumption to the same set of factors in women. Race, smoking status, educational level, alcohol consumption, hypertension, and energy intake are significantly associated with coffee consumption in women. Lower body mass index, waist:hip ratio, and higher leisure activity were associated with higher coffee consumption in women, while age and fiber intake were not. In women, higher coffee intake was also associated with lower levels of serum insulin and glucose at baseline.

### Type 2 diabetes risk and coffee consumption

The association between type 2 diabetes and coffee was not significant in any single category and did not show in the minimal model a significant inverse trend when minimally

**TABLE 1. Incidence of type 2 diabetes mellitus by consumption level and type of beverage, follow-up in person-years, and adjusted hazard ratio* and 95% confidence interval by gender, Atherosclerosis Risk in Communities Study, 1987–1999**

| | No. | % | Cases | Person-years | Adjusted hazard ratio* | 95% confidence interval | $p_{trend}$ |
|---|---|---|---|---|---|---|---|
| *Coffee* (*cups*† *per day*) | | | | | | | |
| Men | | | | | | | |
| Almost never | 1,183 | 22 | 167 | 8,795 | 1 | Referent | 0.09 |
| <1 | 780 | 14 | 109 | 5,866 | 1.00 | 0.78, 1.27 | |
| 1 | 958 | 18 | 152 | 7,073 | 1.12 | 0.90, 1.39 | |
| 2–3 | 1,372 | 25 | 165 | 10,378 | 0.88 | 0.71, 1.09 | |
| ≥4 | 1,121 | 21 | 125 | 8,414 | 0.84 | 0.66, 1.06 | |
| Women | | | | | | | |
| Almost never | 1,968 | 29 | 238 | 14,909 | 1 | Referent | 0.24 |
| <1 | 908 | 13 | 90 | 6,977 | 0.89 | 0.70, 1.14 | |
| 1 | 1,276 | 19 | 151 | 9,526 | 0.94 | 0.77, 1.15 | |
| 2–3 | 1,554 | 23 | 150 | 12,082 | 0.89 | 0.72, 1.09 | |
| ≥4 | 1,084 | 16 | 90 | 8,546 | 0.88 | 0.68, 1.13 | |
| *Sweet beverages* (*cups per day*) | | | | | | | |
| Men | | | | | | | |
| <1 | 2,557 | 47 | 331 | 19,205 | 1 | Referent | 0.68 |
| 1 | 504 | 9 | 67 | 3,706 | 1.03 | 0.80, 1.35 | |
| 1.1–1.9 | 1,415 | 26 | 182 | 10,665 | 0.96 | 0.80, 1.15 | |
| ≥2.0 | 938 | 17 | 138 | 6,892 | 1.09 | 0.89, 1.33 | |
| Women | | | | | | | |
| <1 | 3,510 | 52 | 320 | 27,438 | 1 | Referent | 0.05 |
| 1 | 896 | 13 | 103 | 6,815 | 1.21 | 0.97, 1.51 | |
| 1.1–1.9 | 1,490 | 22 | 182 | 11,255 | 1.20 | 1.00, 1.44 | |
| ≥2.0 | 894 | 13 | 114 | 6,533 | 1.17 | 0.94, 1.46 | |

* Hazard ratios are adjusted for race and age.
† One cup = 0.24 liter.

adjusted (table 3). However, after adjustment for potential confounders, men showed a significant inverse association ($p_{trend} = 0.02$). Compared with men who did not drink coffee, those who drank 4 or more cups (0.95 liter) per day were less likely to develop diabetes (relative hazard = 0.77, 95 percent confidence interval (CI): 0.61, 0.98). The corresponding relative hazard for women was 0.89 (95 percent CI: 0.69, 1.15) after full adjustment, and the trend was nonsignificant ($p_{trend} = 0.32$).

Because body mass index and the waist:hip ratio are potential mediators as well as confounders, we also examined the full model without adjusting for body mass index and the waist:hip ratio and found it to have little change on the relative hazard estimates within each category. Stratifying by obesity status (body mass index: $\geq 30$ kg/m$^2$) did not show a clear pattern of modification, and tests for interaction were not significant. In addition, further adjustment for either serum magnesium or fasting serum insulin and glucose levels (data not shown) did not affect the association in both men and women. Restricting the analysis to only the Caucasian participants did not affect the results.

When only the self-reported incident cases were used in order to correspond to previous literature, a significant inverse association was observed ($p_{trend} = 0.046$), as well as a significant relative hazard of 0.66 (95 percent CI: 0.45, 0.96) for women who drank 4 or more cups (0.95 liter) per day compared with those who drank none. This increase in the strength of association was also observed in men when using the alternative criteria for diagnosed diabetes as the outcome ($p_{trend} = 0.04$), as well as a decrease in the relative hazard to 0.69 for men who drank 4 or more cups per day compared with those who drank none.

### Multivariate analysis of type 2 diabetes risk and sweetened beverage consumption

Next, we examined the relation between sweetened beverage consumption and type 2 diabetes risk using the minimal and full models previously described. No pattern of association was seen in either men or women (table 4). Adjustment for body mass index and the waist:hip ratio

**TABLE 2. Selected baseline characteristics (mean and standard deviation or %) of men ($n$ = 5,414) and women ($n$ = 6,790) by coffee consumption, Atherosclerosis Risk in Communities Study, 1987–1989**

| | Cups† of coffee per day | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Men | | | | | | Women | | | | | |
| | Almost never | <1 | 1 | 2–3 | ≥4 | $p_{trend}$ | Almost never | <1 | 1 | 2–3 | ≥4 | $p_{trend}$ |
| No. | 1,183 | 780 | 958 | 1,372 | 1,121 | | 1,968 | 908 | 1,276 | 1,554 | 1,084 | |
| Age, years | 54.4 (5.8) | 54.7 (5.7) | 55.1 (5.8) | 54.3 (5.7) | 53.6 (5.4) | <0.01 | 53.3 (5.8) | 53.4 (5.6) | 54.0 (5.9) | 53.7 (5.5) | 53.2 (5.4) | 0.36 |
| Black, % | 27 | 21 | 28 | 13 | 6 | <0.01 | 32 | 23 | 37 | 17 | 6 | <0.01 |
| Currently smoking, % | 20 | 19 | 24 | 26 | 40 | <0.01 | 17 | 18 | 21 | 26 | 44 | <0.01 |
| Body mass index, kg/m$^2$ | 27.3 (4.2) | 27.4 (3.9) | 27.3 (4.1) | 27.1 (3.9) | 27.1 (3.9) | 0.06 | 27.8 (6.1) | 27.3 (5.6) | 27.9 (6.0) | 26.6 (5.4) | 25.8 (4.7) | <0.01 |
| Obesity (≥30 kg/m$^2$), % | 21 | 23 | 22 | 19 | 21 | 0.30 | 30 | 27 | 31 | 22 | 17 | <0.01 |
| Waist:hip ratio | 0.957 (0.055) | 0.962 (0.051) | 0.957 (0.054) | 0.960 (0.052) | 0.963 (0.054) | 0.06 | 0.890 (0.080) | 0.887 (0.078) | 0.896 (0.080) | 0.883 (0.077) | 0.876 (0.080) | <0.01 |
| Less than high school education, % | 23 | 19 | 29 | 17 | 16 | <0.01 | 20 | 18 | 25 | 17 | 17 | 0.03 |
| Family history of diabetes, % | 19 | 20 | 23 | 23 | 21 | 0.09 | 24 | 22 | 23 | 24 | 24 | 0.73 |
| Consuming alcohol, % | 58 | 68 | 64 | 73 | 73 | <0.01 | 43 | 55 | 46 | 60 | 68 | <0.01 |
| Hypertensive, % | 35 | 32 | 37 | 27 | 22 | <0.01 | 35 | 31 | 35 | 27 | 20 | <0.01 |
| Leisure activity index | 2.39 (0.56) | 2.37 (0.55) | 2.31 (0.57) | 2.38 (0.54) | 2.35 (0.52) | 0.32 | 2.39 (0.59) | 2.47 (0.58) | 2.36 (0.58) | 2.44 (0.56) | 2.47 (0.57) | <0.01 |
| Energy intake, kcal/day | 1,711 (636) | 1,723 (602) | 1,779 (631) | 1,772 (631) | 1,854 (668) | <0.01 | 1,470 (537) | 1,461 (529) | 1,498 (530) | 1,496 (524) | 1,586 (546) | <0.01 |
| Fiber, g/day | 18.0 (8.8) | 17.6 (8.0) | 17.7 (8.1) | 17.1 (8.1) | 17.0 (7.9) | <0.01 | 17.3 (8.7) | 16.9 (7.5) | 17.1 (8.2) | 17.0 (7.6) | 17.0 (7.7) | 0.29 |
| Serum magnesium, milliequivalent/liter | 1.64 (0.16) | 1.65 (0.16) | 1.64 (0.15) | 1.66 (0.14) | 1.67 (0.14) | <0.01 | 1.63 (0.15) | 1.64 (0.16) | 1.64 (0.15) | 1.64 (0.15) | 1.66 (0.14) | <0.01 |
| Serum insulin, pmol/liter† | 82 (61) | 81 (54) | 86 (66) | 78 (52) | 73 (49) | <0.01 | 81 (68) | 78 (56) | 86 (69) | 69 (46) | 61 (43) | <0.01 |
| Blood glucose, mmol/liter† | 5.58 (0.50) | 5.58 (0.51) | 5.61 (0.53) | 5.58 (0.49) | 5.58 (0.49) | 0.72 | 5.40 (0.52) | 5.38 (0.50) | 5.42 (0.52) | 5.38 (0.48) | 5.35 (0.48) | 0.03 |

* One cup = 0.24 liter.

† On fasting participants only (men: $n$ = 5,302; women: $n$ = 6,689).

**TABLE 3. Risk of incident type 2 diabetes mellitus by level of coffee consumption at baseline, Atherosclerosis Risk in Communities Study, 1987–1999**

| Covariate adjustment | Cups* of coffee per day | | | | | | | | | | $p_{trend}$ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Almost never | | <1 | | 1 | | 2–3 | | ≥4 | | |
| | Hazard ratio | 95% confidence interval | Hazard ratio | 95% confidence interval | Hazard ratio | 95% confidence interval | Hazard ratio | 95% confidence interval | Hazard ratio | 95% confidence interval | |
| Men | | | | | | | | | | | |
| Minimal† | 1 | Referent | 0.99 | 0.78, 1.26 | 1.09 | 0.87, 1.36 | 0.85 | 0.69, 1.06 | 0.83 | 0.65, 1.05 | 0.06 |
| Full‡ | 1 | Referent | 0.94 | 0.74, 1.20 | 1.04 | 0.83, 1.30 | 0.82 | 0.66, 1.03 | 0.77 | 0.61, 0.98 | 0.02 |
| Full‡ − body mass index and waist:hip ratio | 1 | Referent | 0.98 | 0.78, 1.26 | 1.01 | 0.81, 1.27 | 0.82 | 0.66, 1.02 | 0.77 | 0.61, 0.99 | 0.02 |
| Full‡ + serum magnesium | 1 | Referent | 0.94 | 0.74, 1.20 | 1.04 | 0.83, 1.30 | 0.83 | 0.66, 1.03 | 0.77 | 0.61, 0.99 | 0.02 |
| Full‡ using diagnosis/ medications§ | 1 | Referent | 1.02 | 0.76, 1.38 | 1.01 | 0.76, 1.34 | 0.93 | 0.70, 1.22 | 0.69 | 0.50, 0.96 | 0.04 |
| Women | | | | | | | | | | | |
| Minimal† | 1 | Referent | 0.90 | 0.70, 1.14 | 0.94 | 0.76, 1.15 | 0.88 | 0.72, 1.08 | 0.85 | 0.66, 1.09 | 0.16 |
| Full‡ | 1 | Referent | 0.91 | 0.71, 1.16 | 0.90 | 0.73, 1.10 | 0.92 | 0.75, 1.14 | 0.89 | 0.69, 1.15 | 0.32 |
| Full‡ − body mass index and waist:hip ratio | 1 | Referent | 0.91 | 0.71, 1.16 | 0.94 | 0.77, 1.15 | 0.88 | 0.71, 1.08 | 0.83 | 0.64, 1.08 | 0.14 |
| Full‡ + serum magnesium | 1 | Referent | 0.91 | 0.71, 1.15 | 0.90 | 0.73, 1.11 | 0.92 | 0.75, 1.14 | 0.91 | 0.70, 1.18 | 0.38 |
| Full‡ using diagnosis/ medications§ | 1 | Referent | 1.23 | 0.93, 1.62 | 1.06 | 0.83, 1.36 | 0.89 | 0.68, 1.16 | 0.66 | 0.45, 0.96 | 0.046 |

* One cup = 0.24 liter.

† Adjusted for age, race, education, and family history of diabetes.

‡ Adjusted for age, race, education, family history of diabetes, body mass index, waist:hip ratio, total caloric intake, dietary fiber, smoking, alcohol consumption, leisure activity, and hypertension.

§ Uses diagnosis or medication use only for outcome classification.

did not affect the results, nor did use of the alternative criteria for diabetes of self-reported physician's diagnosis or medication use. Stratification by age, body mass index, or physical activity did not affect the results, nor did removing juice from the definition.

## DISCUSSION

This study examined the risk of type 2 diabetes associated with nonalcoholic beverage consumption in a US community-based group of middle-aged African-American and Caucasian adults. This cohort reflects the distribution of consumption patterns in four communities with moderate consumption of coffee and sweetened beverages. High coffee intake was inversely associated with type 2 diabetes risk after adjustment for multiple potential confounders and mediators, including serum magnesium level. On the other hand, there was no association between sweetened beverage consumption and diabetes risk. Of interest, the inverse association between coffee consumption and diabetes risk was most evident for diabetes defined by self-report of a physician's diagnosis or current use of hypoglycemic medications. For both men and women, individuals who drank more than 4 cups (0.95 liter) of coffee per day had a risk of developing type 2 diabetes that was about 67 percent of that of their counterparts who drank no coffee.

Previous prospective studies of coffee consumption and the risk of type 2 diabetes have shown mixed results (5–10). Some differences in results may be attributable to differences in diabetes ascertainment, assessment of covariates, and study populations. As shown in our results, the definition of diabetes has a substantial impact on the strength of the association, especially in women. Differences in the measurement of and adjustment for obesity-related traits could also have led to differences in results, as suggested by the effect of adjustment for body mass index in our study. Finally, different coffee consumption patterns may create different associations with diabetes among studies.

In light of these differences, results from this and previous prospective studies have, in fact, been fairly consistent regarding the association between coffee consumption and type 2 diabetes risk, as summarized in table 5. Although the recent meta-analysis by van Dam and Hu (14) presented an overall picture of the research, we focus instead on placing our study into the context of the literature. Although variation in outcome ascertainment and reference groups makes direct comparisons difficult, two qualitative trends

**TABLE 4. Risk of incident type 2 diabetes mellitus by level of sweetened beverage consumption at baseline, Atherosclerosis Risk in Communities Study, 1987–1999**

| | Cups* of sweetened beverages per day | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | <1 | | 1 | | 1.1–1.9 | | ≥2.0 | | $p_{trend}$ |
| | Hazard ratio | 95% confidence interval | Hazard ratio | 95% confidence interval | Hazard ratio | 95% confidence interval | Hazard ratio | 95% confidence interval | |
| Men | | | | | | | | | |
| Full† – body mass index and waist:hip ratio | 1 | Referent | 1.03 | 0.79, 1.34 | 0.95 | 0.79, 1.15 | 1.03 | 0.82, 1.28 | 0.94 |
| Full† – body mass index and waist:hip ratio using diagnosis/ medications‡ | 1 | Referent | 1.09 | 0.79, 1.52 | 1.06 | 0.84, 1.34 | 1.02 | 0.76, 1.36 | 0.76 |
| Women | | | | | | | | | |
| Full† – body mass index and waist:hip ratio | 1 | Referent | 1.13 | 0.91, 1.42 | 1.10 | 0.91, 1.33 | 1.01 | 0.79, 1.29 | 0.58 |
| Full† – body mass index and waist:hip ratio using diagnosis/ medications‡ | 1 | Referent | 0.77 | 0.56, 1.06 | 1.05 | 0.83, 1.32 | 1.07 | 0.79, 1.43 | 0.63 |

* One cup = 0.24 liter.

† Adjusted for age, race, education, family history of diabetes, total caloric intake, dietary fiber, smoking, alcohol consumption, leisure activity, and hypertension.

‡ Uses diagnosis or medication use only for outcome classification.

in the studies emerge: 1) higher consumption of coffee seems to associate with decreased hazard of diabetes; and 2) measurement of disease using clinically diagnosed diabetes or medication use is associated with stronger associations. Our highest category (≥4 cups (≥0.95 liter) per day), using the self-reported criteria, showed similar hazard ratios to those in comparable groups in other populations.

There are several possible explanations for the difference in the strength of the association between coffee and incident diabetes according to the different diabetes definitions. If there is a true causal association between coffee and diabetes, physician-diagnosed or medically treated diabetes might be a more specific measure that results in less misclassification of diabetes (fewer individuals with false positive diabetes), thus leading to stronger associations. Moreover, if caffeine consumption leads to lower blood glucose levels, then regular coffee drinkers in the ARIC Study who fasted and did not have coffee on the day of the study visit could have experienced an increase in their tested blood glucose as a result of caffeine withdrawal. This would lead to a greater number of false positive diabetic cases, thus weakening the association between coffee consumption and diabetes risk. However, the effect of increased blood glucose as a result of caffeine withdrawal has not been shown in other settings (26). Alternatively, coffee consumption may not be causally associated with the risk of developing diabetes at all. In this case, coffee drinkers could appear to be less likely to have diagnosed diabetes because coffee consumption might either be associated with less health-seeking behavior or mask some symptoms of diabetes, both of which would lead to less diagnosis.

The consistency across populations adds evidence that a causal component of the association between coffee consumption and risk of type 2 diabetes should be considered. Several biologic mechanisms for a protective effect of coffee have been proposed. Coffee consumption is hypothesized to decrease risk of diabetes through its effect on obesity and insulin sensitivity—the two most important risk factors for diabetes. A small clinical study showed that coffee consumption in healthy volunteers can stimulate thermogenesis (27). A study of the effect of caffeinated coffee on energy metabolism in women also indicated that there is a prolonged thermogenic effect of caffeine, especially among lean women (28). Coffee contains caffeine, magnesium, and chlorogenic acid, which may influence glucose metabolism and insulin sensitivity. Although a short-term study has shown that caffeine administration can lead to greater insulin sensitivity (29), others have shown the opposite (30–32). Previous experimental studies have shown that magnesium supplementation can improve glucose metabolism (33–35), while several large epidemiologic studies have shown that low serum/dietary magnesium levels can predict a higher incidence of type 2 diabetes (36, 37). Finally, the chlorogenic acid found in coffee may lead to decreased glucose absorption (38, 39). A recent case-crossover trial of coffee consumption with a 4-week follow-up found that coffee consumption increased fasting insulin, but had no effect on glucose, suggesting that further research on the biologic effects of coffee is necessary (32).

The lack of association between sweetened beverage consumption and type 2 diabetes risk is surprising. However, in the study by Schulze et al. (19), the mean body

**TABLE 5. Summary of Atherosclerosis Risk in Communities Study (1987–1999) results in the context of prospective studies to date**

| First author, year (reference) | Population | Measure of type 2 diabetes | Highest/lowest category of coffee consumption (cups*/day) | Hazard ratio for highest to lowest categories in study | Hazard ratio for closest categories to ≥4 cups/day in study |
|---|---|---|---|---|---|
| Current study | Men and women, ARIC† Study, United States | Fasting blood glucose or self-report of physician's diagnosis or medication use | ≥4/almost never | Men: fasting blood glucose and self-report; hazard ratio = 0.81<br>Women: fasting blood glucose and self-report; hazard ratio = 0.92 | |
| | | Self-report of physician's diagnosis or medication use | ≥4/almost never | Men: self-report only; hazard ratio = 0.69<br>Women: self-report only; hazard ratio = 0.66 | |
| van Dam, 2006 (13) | Women, Nurses' Health Study II, United States | Self-report of physician's diagnosis with follow-up questionnaire | ≥4/none | Hazard ratio = 0.61 | Hazard ratio = 0.61 |
| van Dam, 2004 (11) | Men and women, Hoorn Study, Netherlands | Oral glucose tolerance test, blood glucose | ≥7/≤2 | Hazard ratio = 0.69 | 3–4 cups/day—hazard ratio = 0.94<br>5–6 cups/day—hazard ratio = 0.92 |
| Carlsson, 2004 (5) | Men and women, Finnish Twin Cohort | Drug registry and hospital discharge | ≥7/≤2 | Hazard ratio = 0.65 | 3–4 cups/day—hazard ratio = 0.70<br>5–6 cups/day—hazard ratio = 0.71 |
| Tuomilehto, 2004 (9) | Men and women, Finland | Drug registry and hospital discharge | ≥10/≤2 | Men: hazard ratio = 0.45<br>Women: hazard ratio = 0.21 | 3–4 cups/day<br>Men: hazard ratio = 0.73<br>Women: hazard ratio = 0.71<br>5–6 cups/day<br>Men: hazard ratio = 0.70<br>Women: hazard ratio = 0.39 |
| Salazar-Martinez, 2004 (7) | Men and women, Nurses' Health Study and Health Professionals Follow-up Study, United States | Self-report of physician's diagnosis with follow-up questionnaire | ≥6/none | Men: hazard ratio = 0.46<br>Women: hazard ratio = 0.71 | 4–5 cups/day<br>Men: hazard ratio = 0.71<br>Women: hazard ratio = 0.70 |
| Rosengren, 2004 (12) | Women (BEDA‡), Göteborg, Sweden | Blood or urine glucose, self-report of physician's diagnosis, hospital discharge | >6/≤2 | Hazard ratio = 0.57 | 3–4 cups/day—hazard ratio = 0.56<br>5–6 cups/day—hazard ratio = 0.45 |
| Saremi, 2003 (8) | Men and women, Pima Indians, United States | Oral glucose tolerance test | "Occasionally heavy"/none | Hazard ratio = 0.81 | Hazard ratio = 0.81 |
| Reunanen, 2003 (6) | Men and women, Finland | Drug registry | ≥7/≤2 | Hazard ratio = 0.92 | 3–4 cups/day—hazard ratio = 1.01<br>5–6 cups/day—hazard ratio = 0.98 |
| van Dam, 2002 (10) | Men and women, Netherlands | Self-report of diagnosis or drug use | ≥7/≤2 | Hazard ratio = 0.50 | 3–4 cups/day—hazard ratio = 0.79<br>5–6 cups/day—hazard ratio = 0.73 |

* One cup = 0.24 liter.

† ARIC, Atherosclerosis Risk in Communities.

‡ "BEDA" is not an acronym but the name of the archetypal Göteborg woman—tough, sharp witted, and with a generous heart.

mass index at baseline was 24.6 kg/m$^2$ and the mean age at baseline was 36.1 years, whereas in the current study the mean body mass index at baseline for women was 27.2 kg/m$^2$ and the mean age at baseline was 53.6 years. One possibility for the differences in results is that the effect of sweetened beverage consumption is mediated largely through increases in body mass index and that, once body mass index is increased, the additional effect of continued sweetened beverage consumption is diminished. Sweetened beverage consumption may be a more important risk factor for younger or leaner people and should be studied further in that population.

Our study does have several limitations. There is potential nondifferential misclassification of beverage consumption, both because the questions were neither as specific nor as sensitive as possible and because of the one-time assessment. We would expect this misclassification to bias our results on average toward a null finding, which might have dampened our results for coffee and masked an association between incident diabetes and sweetened beverages. We also had limited ability to examine the association between coffee and incident diabetes at very high levels of coffee consumption, such as those seen in the studies from Finland and the Netherlands. However, since our cohort was population based, it reflects the actual patterns of coffee drinking in our communities. We also note that there was no measurement of milk, cream, or sugar added to coffee. The effect of these additions might be expected to be positively associated with both increased consumption (the more coffee drunk, the more additions consumed) and increased diabetes, thus acting in opposition to the pattern observed.

In conclusion, a higher consumption level of coffee was associated with a decreased risk of type 2 diabetes in middle-aged adults. Although several observational prospective studies have yielded consistent findings and although biologic explanations for decreased diabetes risk with increased coffee consumption have been postulated, further research, particularly experimental studies that can examine the long-term effect of coffee consumption on glucose metabolism, insulin sensitivity, and diabetes risk, is needed before recommendations can be made about coffee drinking with respect to the prevention of type 2 diabetes.

## ACKNOWLEDGMENTS

The Atherosclerosis Risk in Communities Study is carried out as a collaborative study supported by National Heart, Lung, and Blood Institute contracts N01-HC-55015, N01-HC-55016, N01-HC-55018, N01-HC-55019, N01-HC-55020, N01-HC-55021, and N01-HC-55022. W. H. L. K. was supported by grant 1 K01 DK067207-01A1. N. P. P. was supported by Cardiovascular Epidemiology Training Grant T32HL07024 from the National Heart, Lung, and Blood Institute.

The authors thank the staff of the ARIC Study for their important contributions.

Conflict of interest: none declared.

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