White Rice, Brown Rice, and Risk of Type 2 Diabetes in US Men and Women

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Abstract

Context—Because of a different degree of processing and nutrient contents, brown rice and white rice may have different effects on risk of type 2 diabetes.

Objective—To prospectively examine white rice and brown rice consumptions in relation to type 2 diabetes risk in US men and women aged 26–87 yr.

Design and Setting—The Health Professionals Follow-up Study (1986–2006) and the Nurses' Health Study I (1984–2006) and II (1991–2005).

Participants—We prospectively ascertained diet, lifestyle practices, and disease status among 39,765 men and 157,463 women in these cohorts. All participants were free of diabetes, cardiovascular disease, and cancer at baseline. Intake of white rice, brown rice, other foods, and nutrients was assessed at baseline and updated every 2–4 years.

Results—During 3,318,196 person-years of follow-up, we documented 10,507 incident cases of type 2 diabetes. After multivariate adjustment for age and other lifestyle and dietary risk factors, higher intake of white rice was associated with a higher risk of type 2 diabetes. The pooled relative risk (95% confidence interval) of type 2 diabetes comparing ≥5 servings/week with <1 serving/month of white rice was 1.17 (1.02, 1.36). In contrast, high brown rice intake was associated with a lower risk of type 2 diabetes: The pooled multivariate relative risk (95% confidence interval) was 0.89 (0.81, 0.97) for ≥ 2 servings/week of brown rice as compared with <1 serving/month. We estimated that replacing 50 grams/day (cooked, equivalent to ⅓ serving/day) intake of white rice with the same amount of brown rice was associated with a 16% (95% confidence interval: 9%, 21%) lower risk of type 2 diabetes, whereas the same replacement with whole grains as a group was associated with a 36% (95% confidence interval: 30%, 42%) lower diabetes risk.

Conclusions—Substitution of whole grains, including brown rice, for white rice may lower risk of type 2 diabetes. These data support the recommendation that most carbohydrate intake should come from whole grains rather than refined grains to facilitate the prevention of type 2 diabetes.

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Rice has been a staple food in Asian countries for centuries. By the twentieth century, the advance of grain-processing technology made it possible for large scale production of refined grains.[1] Through refining processes, the outer bran and germ portions of intact rice grains (i.e., brown rice) is removed to produce white rice that primarily consists of starchy endosperm. Although not entirely consistent, consumption of white rice, in general, generates a stronger postprandial blood glucose response as measured by the glycemic index (GI) than the same amount of brown rice. A systematic review found that the mean GI was 64 ± 7 for white rice and 55 ± 5 for brown rice.[2] Higher dietary GI has been consistently associated with elevated risk of type 2 diabetes (T2D) in prospective cohort studies.[3–6] In addition, brown rice consumption may also impart beneficial effects on T2D risk by virtue of its high contents of multiple nutrients, such as fiber, vitamins, and minerals, the majority of which are lost during refining and milling processes.[7] In line with these observations, high intake of white rice was associated with a monotonically elevated risk of developing T2D in a Chinese population, in which white rice consumption was the primary source of carbohydrate (74% of dietary glycemic load).[6]

Compared to Asian countries, rice consumption is much lower in the U.S., but is increasing rapidly. According to the U.S. Department of Agriculture 2009 food supply and disappearance data, rice consumption has increased more than 3 fold since the 1930s', to reach 20.5 lbs per capita, and more than 70% of rice consumed is white rice.[8] However, little is known whether rice intake is associated with diabetes risk in U.S. populations. We, therefore, evaluated the associations between intake of white rice and brown rice and risk of T2D in three large cohort studies with repeated prospective dietary assessments. We have previously observed an inverse association between whole grain consumption and risk of T2D in these cohorts.[9, 10] In the present study, we extended the follow-up of these previously reported studies and evaluated whether substituting whole grains for white rice is associated with a lower risk of diabetes.

METHODS

Study Populations

We used data from three prospective cohort studies: the Health Professionals Follow-up Study (HPFS; age range, 32–87 yr) and the Nurses' Health Study (NHS) I (age range, 37–65 yr) and II (age range, 26–45 yr). Detailed descriptions of these three cohorts were introduced elsewhere.[11–13] In all three cohort studies, questionnaires were administered at baseline, as well as biennially after baseline, to collect and update information on lifestyle practice and occurrence of chronic diseases. The follow-up rates of the participants in these cohorts are all above 90%.

In the current analysis, we excluded men and women who had diagnoses of diabetes, cardiovascular disease, and cancer at baseline for the dietary analyses (1986 for HPFS, 1984 for NHS I, and 1991 for NHS II, when we first assessed white rice and brown rice consumption in these cohorts). In addition, we excluded HPFS participants who left more than 70 of the 131 food items blank on the baseline food frequency questionnaire (FFQ), or who reported unusual total energy intake levels (i.e., daily energy intake <800 or >4,200 kcal/day). For NHS I and II participants, we excluded those who left more than 10 (NHS I) or 9 (NHS II) items blank on baseline FFQs or whose total energy intake was <500 or >3,500 kcal/day. After exclusions, data from 39,765 (out of 51,530) HPFS participants, 69,120 (out of 81,755) NHS I participants, and 88,343 (out of 95,452) NHS II participants were available for the analysis. The study was approved by the Human Research Committee of Brigham and Women's Hospital and the Human Subjects Committee Review Board of Harvard School of Public Health. The completion of the self-administered questionnaire was considered to imply informed consent.

Assessment of Rice Consumption

In 1984, a 116-item FFQ was administered among the NHS I participants to collect information on their usual intake of foods and beverages in the previous year. In 1986–2002, similar but expanded FFQs were sent to these participants to update their diet every 4 years. Using the expanded FFQ employed in the NHS I, dietary data were collected every four years during 1986–2002 among the HPFS participants, and during 1991–2003 among the NHS II participants. In all FFQs, we asked the participants how often, on average, they consumed each food of a standard portion size. In the current study, based on the distribution of responses to rice intake questions, we categorized participants into 5 categories (<1 serving/month, 1–3 servings/month, 1 serving/week, 2–4 servings/week, and ≥5 servings/week) of white rice intake and 3 categories (<1 serving/month, 1–4 servings/month, and ≥2 servings/week) of brown rice intake to warrant appropriate variation in rice consumption while preserving enough statistical power to make stable estimate for each category. The reproducibility and validity of these FFQs have been demonstrated in detail elsewhere.[14–17] In a validation study conducted among a subsample of HPFS participants, assessments of white rice and brown rice intake were moderately correlated with diet record assessments. The corrected Pearson correlation coefficients between these two assessments were 0.53 for white rice and 0.41 for brown rice.[14] Assessment of whole grain intake was described in detail elsewhere.[9] We considered any intact or milled form of grain that consisted of the expected proportions of bran, germ, and endosperm as whole grains. By definition, brown rice is a whole grain.

Study Outcome

The study outcome was incident T2D that occurred between the return of the baseline FFQ and January 31, 2006 (HPFS), June 30, 2006 (NHS I), or June 30, 2005 (NHS II). In all three cohorts, men and women who reported a diagnosis of T2D in the biennial follow-up questionnaires were sent a supplementary questionnaire to confirm the diagnosis. In this supplementary questionnaire, information on symptoms, diagnostic tests, and treatment was collected. We utilized the criteria from the National Diabetes Data Group to confirm self-reported diagnosis of T2D.[18] For cases of T2D identified after 1998, we applied the American Diabetes Association criteria.[19] The validity of the supplementary questionnaire for the diagnosis of diabetes has been described previously.[20, 21] Out of a random sample of 62 nurses reporting type 2 diabetes in the supplementary questionnaire, 61 (98%) of them were confirmed after their medical records were reviewed by an endocrinologist blinded to the supplementary questionnaire information.[20] In another validation study conducted in HPFS participants, 97% (57/59) of self-reported type 2 diabetes cases were confirmed using medical record review.[21] Deaths were identified by reports from next of kin, postal authorities, or by searching the National Death Index. At least 98% of deaths among the study participants were identified.[22]

Statistical Analysis

We counted each individual's person-years of follow-up from the date of return of the baseline FFQ to the date of death, the date of diagnosis of T2D, or January 31, 2006 (HPFS), June 30, 2006 (NHS I), or June 30, 2005 (NHS II), whichever came first. The relative risks (RRs) were estimated using Cox proportional hazard regression,[23] in which we stratified the analysis jointly by age in months at baseline and calendar year to control for confounding by these factors as finely as possible. In multivariate analysis, we further adjusted for ethnicity, body mass index (BMI), smoking status, alcohol intake, multivitamin use, physical activity, and family history of diabetes. Among nurses, we adjusted for oral contraceptive use (NHS II participants only), postmenopausal status, and hormone use. To minimize confounding by other dietary factors, we further adjusted for total energy intake and intake of red meat, fruits and vegetables, coffee, and whole grains. All of these covariates are established risk factors

for type 2 diabetes and were correlated with white rice or brown rice consumption in these cohorts.

To address missing values of dietary variables in the follow-up FFQs, we replaced missing values with those from a previous FFQ without missing data. On average, 12.7% of NHS I, 11.3% of NHS II, and 23.1% of HPFS participants had missing data after baseline assessment. To better represent long-term diet and to minimize the within-person variation, we created cumulative averages of food and nutrient intake from baseline to the censoring events.[24] We stopped updating diet when participants first reported having a diagnosis of hypertension, hypercholesterolemia, cardiovascular disease, or cancer. For these participants, we carried forward the cumulative averages of dietary intake prior to the occurrence of these diseases to represent diet for later follow-up. We have used this approach in our previous studies to avoid systematic errors in dietary assessment due to potential biased recall after occurrence of chronic diseases.[24, 25] To estimate the association of substituting brown rice intake for the same amount of white rice, we included both white rice and brown rice intake as continuous variables (50 grams/day; equivalent to ⅓ serving/ day of white rice) in the same multivariate model. We used the difference between regression coefficients for brown rice and white rice to derive the RR measuring this association of substitution. We used the same approach to examine such an association for whole grains treated as a single food item. This method has been used in our previous studies.[26]

Tests for trend were conducted by assigning the median value to each category and modeling this value as a continuous variable. To summarize the estimates of association across the three studies, we conducted a meta-analysis using fixed effects models. P values for heterogeneity of study results were calculated using the Cochran Q test.[27] All P values were two-sided. Ninety-five percent confidence intervals (95% CI) were calculated for RRs. Data were analyzed with the Statistical Analysis Systems software package, version 9.1 (SAS Institute, Inc., Cary, North Carolina). The pooling analysis was conducted using STATA 10.0 (StataCorp, College Station, Texas).

Role of the Funding Source

The National Institutes of Health provided funding for this study. The funding source had no role in the collection, analysis, and interpretation of the data or in the decision to submit the manuscript for publication.

RESULTS

We documented 2,648 incident T2D cases during 20 years of follow-up in the HPFS, 5,500 cases during 22 years in the NHS I, and 2,359 cases during 14 years in the NHS II. TABLE 1 describes the distribution of baseline characteristics according to intake of white rice and brown rice. Men and women who had high white rice intake were less likely to have European ancestry or to smoke and more likely to have a family history of diabetes. In addition, high white rice intake was associated with high fruit and vegetable intake and low intake of whole grains, cereal fiber, and trans fat. In contrast, brown rice intake was not associated with ethnicity but with a more health-conscious lifestyle and dietary profile. For example, participants with higher brown rice intake were more physically active, leaner, less likely to smoke or have a family history of diabetes, and had higher intake of fruits, vegetables, and whole grains and lower intake of red meat and trans fat. Both white rice and brown rice intake were positively associated with a higher glycemic load in all three cohorts.

TABLE 2 shows the RRs of T2D according to white rice intake. In age-adjusted models, white rice intake was associated with an elevated risk of developing T2D across the three

studies. After multivariate adjustment for lifestyle and dietary risk factors, these associations were attenuated, but a trend of increased risk associated with high white rice intake still remained. After summarizing the multivariate estimates across the three studies, in comparison to those in the lowest category of white rice intake, participants who ate white rice ≥5 servings/week had 17% (95% CI: 2%, 36%; P for trend < 0.0001) higher risk of developing T2D.

In contrast to white rice, brown rice intake was associated with a lower risk of T2D in age-adjusted models (TABLE 3). After multivariate adjustment for covariates, these associations were attenuated but the statistical significance remained. When compared to the participants who ate less than one serving per month of brown rice, the pooled RR (95% CI) of T2D was 0.89 (0.81, 0.97) for intake of ≥2 servings/week with a P for trend of 0.005.

We observed a monotonically decreasing risk of diabetes associated with increasing consumption of whole grains, which include brown rice (TABLE 4). In comparison to the lowest quintile, the pooled RR (95% CI) for the highest quintile of whole grains was 0.73 (0.68, 0.78; P for trend < 0.0001). We further estimated the RRs of T2D associated with bran and germ intake (Table 4). Bran intake, but not germ intake, was associated with a lower risk of developing T2D. In comparison to those in the lowest quintile of bran intake, men and women in the highest quintile had a pooled RR (95% CI) of 0.76 (0.71, 0.82; P for trend < 0.0001). For germ intake, the corresponding RR was 0.95 (0.88, 1.03; P for trend = 0.40).

We subsequently examined the RR associated with the replacement of 50 grams/day (⅓ serving/day) of white rice with the same amount of brown rice intake. In all three cohorts, substituting brown rice for white rice was consistently associated with a lower risk of T2D (FIGURE). In the pooled analysis, each 50 grams/day intake of brown rice replacing white rice was associated with an RR (95% CI) of 0.84 (0.79, 0.91). We further examined the RR associated with replacing 50 grams/day of white rice intake with the same amount of whole grains: the RR (95% CI) was 0.64 (0.58, 0.70).

Since ethnicity was associated with both white rice consumption and diabetes risk,[28] the observed associations can be a consequence of confounding by ethnicity. However, in secondary analyses, when we repeated these associations among white participants only, we found similar results. For example, after excluding non-white participants (African American, Hispanic, and Asian), the pooled RRs (95% CI) of T2D associated with white rice intake were 1.00 (0.93, 1.07) for 1–3 servings/month, 1.04 (0.96, 1.13) for 1 serving/week, 1.10 (1.01, 1.19) for 2–4 servings/week, and 1.19 (1.00, 1.41; P for trend < 0.0001) for ≥5 servings/week. The pooled RRs (95% CI) for brown rice intake levels were 0.94 (0.90, 0.99) for 1–4 servings/month and 0.87 (0.79, 0.96; P for trend = 0.003) for ≥ 2 servings/week. Among white participants, the pooled RRs (95% CI) of T2D were 0.84 (0.78, 0.92; P < 0.0001) for replacing 50 grams/day of white rice intake with the same amount of brown rice, and 0.64 (0.58, 0.71; P < 0.0001) for substituting 50 grams/day of whole grains for the same amount of white rice. When we restricted our analysis within minority groups only, although we observed largely similar results, most associations became non-significant due to the dramatically diminished power (we identified 624 T2D among 9,644 non-white participants).

When we used more recent intake of white rice or brown rice instead of the cumulative average in the analyses, the results did not substantially change. For example, the pooled RR (95% CI) was 1.25 (1.08, 1.45; P for trend = 0.001) for ≥2 servings/week vs. <1 serving/month of white rice intake, 0.90 (0.85, 0.95; P < 0.0001) for replacing 50 grams/day of white rice intake with the same amount of brown rice, and 0.76 (0.71, 0.82; P < 0.0001) for

substituting 50 grams/day of whole grains for the same amount of white rice. Because rice consumption for most of our study participants was relatively stable over time (data not shown), these results indicated that the cumulative averages could better represent long-term rice consumption because of reduced random within-person measurement errors.[24] Lastly, we did not find any interactions between rice consumption and other diabetes risk factors, including age, BMI, and various co-morbidities.

COMMENT

In these three prospective cohort studies of U.S. men and women, we found that regular consumption of white rice was associated with higher risk of T2D, whereas brown rice intake was associated with lower risk. In addition, our data suggest that replacing white rice intake with the same amount of brown rice or whole grains was associated with a lower risk. These associations were independent of lifestyle and dietary risk factors for T2D, as well as ethnicity.

In Asian populations in whom rice is a staple food, higher white rice consumption has been associated with elevated risk of diabetes or metabolic syndrome.[6, 29, 30] For example, white rice consumption was prospectively associated with developing type 2 diabetes in Chinese women living in Shanghai.[6] In addition, in Asian Indians and Japanese, higher intake of refined grain including white rice was associated with metabolic risks in cross-sectional analyses.[29, 30] In comparison to Asian populations, white rice intake in Western populations was much lower. White rice consumption contributed, on average, less than 2% of total energy intake in our study populations. In contrast, in the aforementioned Chinese female population, white rice consumption accounted for 53.7% of total energy intake (Xiao-Ou Shu, personal communication).[6] Likewise, according to the Japanese National Nutrition Survey, white rice accounted for 29% of daily total energy intake in Japanese.[31] Consumption of rice or food groups consisting of rice in relation to risk of type 2 diabetes was also evaluated in Western populations, but mixed results were observed.[32–34] However, brown rice was not separated from white rice or other refined grains in these studies [32–34]. To our knowledge, the current studies are the first prospective investigations conducted among Western populations that have specifically evaluated white rice and brown rice intake in relation to T2D risk. In our cohorts only 0.9% (NHS I) to 2.2% (NHS II) of total participants reported having five or more servings per week (≥107 grams/day) of white rice, which was within the lowest reference level (<200 grams/day) in the prospective study of Chinese women.[6] However, by pooling data from the three studies, we detected a significant association for white rice intake. Our data are consistent with the Chinese study, in which white rice intake of 300 grams/day or more (equivalent to 2 servings/day in our analysis) was associated with a 78% increased risk of T2D in comparison to intake levels of less than 200 grams/day.[6]

We observed a moderate, inverse association for brown rice intake. Because brown consumption levels were rather low in our participants, we could not determine whether brown rice intake at much higher levels is associated with a further reduction of diabetes risk. Nonetheless, we found that substitution of brown rice for white rice was associated with a significantly lower risk of developing diabetes. Consistent with our previous analyses,[9, 10] we found a significant inverse association between whole grain consumption and diabetes risk. Substitution of whole grains for white rice was more strongly associated with diabetes risk than the substitution of brown rice. This observation may result from the more reliable estimates of the association with diabetes for whole grains than those for brown rice, because of the low overall consumption of brown rice. In addition, whole grains included multiple grains with various nutrient compositions and, thus, possibly various effects on glucose response. For example, whole wheat and barley generate lower glucose

response than brown rice: the GI values were 41 ± 3 for whole wheat, 25 ± 1 for barley, and 55 ± 5 for brown rice.[2] As a consequence, in comparison to whole wheat and barley, the same amount of brown rice likely bears a higher glycemic load, which is an established risk factor for T2D.[35]

Depending on the botanical structure, amylase contents, and processing methods, both white rice and brown rice demonstrated a wide variety of GI values,[2, 36–38] which made it difficult to directly compare white rice with brown rice for effects on postprandial glucose response.[2] Despite of this inconsistency inherent to rice GI values, in general, white rice consumption generates a relatively stronger postprandial glucose response than the same amount of brown rice[2] This notion was corroborated by the observation that isocaloric replacement of white rice with whole grains (66.6%; primarily composed of brown rice and barley) and legume powder (22.2%) significantly decreased postprandial glucose and insulin levels in a randomized clinical trial.[39] The high GI of white rice consumption is likely the consequence of disrupting the physical and botanical structure of rice grains during the refining process, in which almost all of the bran and some of the germ are removed.[40] The other consequence of the refining process includes loss of fiber, vitamins, magnesium and other minerals, lignans, phytoestrogens, and phytic acid,[7] many of which may be protective factors for diabetes risk. Intact rice grains contain nearly exclusively insoluble fiber.[7] In both observational and experimental studies, insoluble fiber intake was consistently associated with improved insulin sensitivity and decreased risk of developing T2D.[4, 5, 41, 42] In addition, higher magnesium intake has been consistently associated with reduced risk of T2D in cohort studies or improved glucose metabolism in clinical trials.[43–45] The combination of these mechanisms may explain the beneficial effects of replacing white rice with brown rice or other whole grains.

The strengths of the current study include a large sample size, high rates of follow-up, and repeated assessments of dietary and lifestyle information. The consistency of the results across all three cohorts indicates that our findings are unlikely due to chance. The current study was subject to a few limitations as well. First, our study populations primarily consisted of working health professionals with European ancestry. Although the homogeneity of socioeconomic status helps reduce confounding, the generalizability of the observed associations may be limited to similar populations. However, the biological mechanisms underlying the positive associations observed in both our study populations and the Chinese study[6] are likely to be the same in other populations. Second, because diet was assessed by FFQs, some measurement error of rice intake assessment is inevitable. However, the FFQs used in these studies were validated against multiple diet records, and reasonable correlation coefficients between these assessments of rice intake were observed. [14] Since we employed a prospective study design, any measurement errors of rice intake are independent of study outcome ascertainment, and, therefore, are likely to attenuate the associations towards the null. Moreover, we calculated cumulative averages of rice intake to minimize the random measurement errors caused by within-person variation. To minimize the possibility of systemic measurement error incurred by recall bias, we not only excluded participants with a history of major chronic diseases at baseline but also stopped updating dietary intake after participants reported having diagnoses of diseases that might influence their subsequent report of diet. Third, we did not perform oral glucose tolerance tests to confirm diabetes diagnoses because this is infeasible in large cohort studies. However, the supplementary questionnaire that we used for the confirmation of self-reported diabetes diagnoses has been demonstrated to be highly accurate.[20, 21] Lastly, although we adjusted for established and potential risk factors for T2D, residual confounding is still possible.

Our data suggest that regular consumption of white rice is associated with an increased risk of T2D, whereas replacement of white rice by brown rice or other whole grains is associated

with a lower risk. The current Dietary Guidelines for Americans identifies grains, including rice, as one of primary sources for carbohydrate intake and recommends at least half of carbohydrate intake should come from whole grains.[46] Rice consumption in the U.S. population is increasing.[8] However, most rice consumption is refined white rice,[8] as seen in our studies. From a public health point of view, replacing refined grains such as white rice by whole grains, including brown rice, should be recommended to facilitate the prevention of type 2 diabetes.

Acknowledgments

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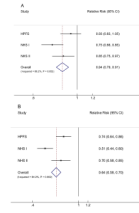
FIGURE.
Pooled fixed effects relative risk (95% CI) of type 2 diabetes of substituting 50 grams/day brown rice (A) or whole grains (B) intake for the same amount of white rice intake. Bars indicate 95% CIs and P values are P for heterogeneity. Individual associations were controlled for the same set of covariates for model 3 in Table 3.

TABLE 1

Baseline characteristics of study participants by levels of white rice and brown rice intake.

Characteristics	White rice intake			Brown rice intake		
	<1/mo	1/wk	≥5/wk	<1/mo	1/mo-1/wk	≥2/wk
HPFS						
n	9386	9968	795	21433	16134	2198
Age (year)	54.5	51.2	51.1	53.6	51.7	51.5
Physical activity (MET-hr)	22.0	22.0	20.8	19.3	23.4	28.4
BMI (kg/m^2)	24.8	24.9	24.2	25.0	24.8	24.4
White (%)	96.7	96.2	49.0	94.5	96.4	93.0
Current smoker (%)	9.6	7.5	7.0	9.9	7.2	5.8
Hypertension (%)	19.0	18.0	21.9	20.0	17.5	17.0
High cholesterol (%)	9.7	10.4	11.5	9.7	10.5	12.9
Family history of diabetes (%)	12.3	13.5	14.6	13.2	12.4	13.6
Total energy (kcal/day)	1851	2099	2200	1943	2042	2229
Red meat intake (servings/wk)	7.6	8.3	8.3	8.6	7.6	5.7
Fruit and vegetable intake (servings/day)	5.0	5.8	5.8	4.8	5.8	7.6
White rice (servings/wk)	0	1.0	7.5	0.9	0.8	1.2
Brown rice (servings/wk)	0.5	0.5	0.6	0	0.7	3.6
Whole grains (g/day)	23.7	21.5	17.8	16.2	25.2	49.6
P:S ratio	0.57	0.58	0.62	0.54	0.59	0.71
Glycemic load	121	125	143	122	126	138
trans fat (% of total energy)	1.3	1.3	1.0	1.4	1.2	0.9
Cereal fiber (g/day)	5.9	5.9	5.7	5.4	6.2	7.8
Magnesium (mg/day)	357	351	359	335	364	426
Alcohol (g/day)	11.2	11.7	8.6	11.3	11.7	10.5
Coffee (cups/day)	1.9	1.9	1.7	2.0	1.9	1.6
Multivitamin supplement user (%)	44.3	40.6	44.8	39.1	43.7	51.6
NHS I						
n	14690	17839	600	51673	16010	1437
Age (year)	50.9	49.2	49.8	50.0	50.2	50.9

Characteristics	White rice intake			Brown rice intake		
	<1/mo	1/wk	≥5/wk	<1/mo	1/mo-1/wk	≥2/wk
Physical activity (MET-hr)	14.7	14.5	15.8	13.1	17.2	22.3
BMI (kg/m^2)	24.8	24.8	24.1	24.9	24.4	24.0
White (%)	98.8	98.2	51.0	97.8	98.3	96.0
Current smoker (%)	25.1	23.8	16.1	25.4	20.8	15.0
Postmenopausal (%)	45.9	44.3	44.2	45.2	44.1	43.3
Postmenopausal hormone use (%) [*]	33.9	33.5	36.8	33.1	37.9	39.7
Hypertension (%)	19.1	20.1	19.9	19.7	18.8	17.4
High cholesterol (%)	7.3	7.6	8.4	7.2	7.8	10.2
Family history of diabetes (%)	24.6	24.6	27.6	25.4	23.8	22.1
Total energy (kcal/day)	1585	1852	1981	1724	1791	1958
Red meat intake (servings/wk)	8.1	9.3	9.6	9.2	8.2	6.3
Fruit and vegetable intake (servings/day)	4.4	4.8	5.4	4.4	5.2	6.5
White rice (servings/wk)	0	1.0	7.3	0.8	0.7	0.7
Brown rice (servings/wk)	0.3	0.2	0.3	0	0.6	3.5
Whole grains (g/day)	16.4	13.2	10.3	11.6	19.1	41.1
P:S ratio	0.56	0.56	0.61	0.54	0.57	0.68
Glycemic load	98	99	114	99	100	109
trans fat (% of total energy)	1.9	1.9	1.5	2.0	1.8	1.4
Cereal fiber (g/day)	4.3	4.1	3.7	4.0	4.5	6.0
Magnesium (mg/day)	296	288	293	278	312	368
Alcohol (g/day)	6.3	7.7	4.2	6.8	7.8	6.6
Coffee (cups/day)	2.4	2.5	2.2	2.5	2.4	2.0
Multivitamin supplement user (%)	39.6	36.2	37.8	34.6	43.4	51.7
NHS II						
n	15753	25808	1901	51217	32382	4744
Age (year)	36.0	36.3	36.3	36.2	36.0	36.3
Physical activity (MET-hr)	22.2	20.7	21.8	18.5	23.5	30.4
BMI (kg/m^2)	24.5	24.5	24.0	24.8	24.2	24.0
White (%)	94.5	94.2	51.6	92.1	94.4	92.1
Current smoker (%)	13.4	11.9	8.6	13.0	11.1	10.1

Characteristics	White rice intake			Brown rice intake		
	<1/mo	1/wk	≥5/wk	<1/mo	1/mo-1/wk	≥2/wk
Postmenopausal (%)	4.1	3.3	3.0	3.7	3.4	3.2
Postmenopausal hormone use (%) *	81.9	82.4	72.4	82.3	82.2	80.9
Hypertension (%)	6.2	5.7	6.1	6.3	5.5	5.1
High cholesterol (%)	14.3	13.7	16.3	14.6	13.7	13.3
Family history of diabetes (%)	15.8	15.7	18.4	16.7	14.8	14.7
Total energy (kcal/day)	1633	1852	2083	1737	1829	2034
Red meat intake (servings/wk)	4.8	5.7	5.6	5.9	5.0	3.9
Fruit and vegetable intake (servings/day)	4.1	4.8	5.7	4.0	5.0	6.8
White rice (servings/wk)	0	1.0	7.8	1.1	0.9	1.2
Brown rice (servings/wk)	0.6	0.4	0.5	0	0.7	3.5
Whole grains (g/day)	23.3	20.1	16.5	15.5	24.4	46.4
P:S ratio	0.52	0.52	0.57	0.50	0.54	0.61
Glycemic load	121	121	137	120	122	131
trans fat (% of total energy)	1.7	1.6	1.3	1.7	1.5	1.2
Cereal fiber (g/day)	5.9	5.6	5.1	5.2	6.0	7.7
Magnesium (mg/day)	321	316	316	299	331	385
Alcohol (g/day)	2.8	3.3	2.4	2.8	3.6	3.8
Coffee (cups/day)	1.5	1.6	1.3	1.5	1.6	1.6
Multivitamin supplement user (%)	44.6	43.1	44.6	40.9	46.9	51.5

Abbreviations: HPFS, Health Professionals Follow-up Study; NHS, Nurses' Health Study; MET, metabolic equivalent task; BMI, body mass index; P:S, polyunsaturated to saturated fat ratio.

*
Current and past use of hormone among postmenopausal women.

TABLE 2

Relative risk (95% CI) of type 2 diabetes risk according to white rice intake in the HPFS, NHS I, and NHS II.

	White rice (servings/time)					
	<1/mo	1–3/mo	1/wk	2–4/wk	≥5/wk	P for trend
HPFS						
Number of cases	320	1150	537	553	88	
Person-year	101899	299985	161667	120197	19172	
Model 1 *	1.0	1.12 (0.99, 1.27)	1.06 (0.92, 1.22)	1.35 (1.17, 1.55)	1.37 (1.08, 1.74)	<0.0001
Model 2 †	1.0	1.08 (0.95, 1.22)	1.07 (0.93, 1.23)	1.30 (1.12, 1.49)	1.06 (0.81, 1.39)	0.03
Model 3 ‡	1.0	1.09 (0.96, 1.24)	1.07 (0.93, 1.23)	1.30 (1.12, 1.50)	1.02 (0.77, 1.34)	0.08
NHS I						
Number of cases	415	2744	1183	1062	96	
Person-year	145718	691609	327215	221681	18150	
Model 1 *	1.0	1.03 (0.93, 1.14)	1.08 (0.96, 1.20)	1.12 (1.00, 1.26)	1.34 (1.07, 1.67)	0.0005
Model 2 †	1.0	1.02 (0.92, 1.14)	1.13 (1.01, 1.26)	1.16 (1.03, 1.30)	1.22 (0.96, 1.56)	0.0004
Model 3 ‡	1.0	1.00 (0.90, 1.11)	1.07 (0.96, 1.20)	1.09 (0.97, 1.23)	1.11 (0.87, 1.43)	0.02
NHS II						
Number of cases	248	924	585	500	102	
Person-year	134505	480405	324961	237765	33267	
Model 1 *	1.0	0.82 (0.72, 0.95)	0.83 (0.71, 0.96)	0.82 (0.71, 0.96)	1.27 (1.01, 1.60)	0.03
Model 2 †	1.0	0.93 (0.81, 1.07)	0.95 (0.82, 1.11)	0.96 (0.82, 1.12)	1.43 (1.12, 1.83)	0.005
Model 3 ‡	1.0	0.93 (0.81, 1.07)	0.94 (0.81, 1.10)	0.95 (0.81, 1.11)	1.40 (1.09, 1.80)	0.01
Pooled						
Fixed effects model ‡	1.0	1.01 (0.94, 1.08)	1.04 (0.96, 1.12)	1.11 (1.03, 1.20)	1.17 (1.02, 1.36)	<0.0001
P_heterogeneity	-	0.26	0.38	0.02	0.20	0.87

*
Age-adjusted.

†
Age (yr), ethnicity (Caucasian, African American, Hispanic, and Asian), BMI (<21 kg/m², 21–22.9 kg/m², 23–24.9 kg/m², 25–26.9 kg/m², 27–29.9 kg/m², 30–32.9 kg/m², 33–34.9 kg/m², or ≥35 kg/m²), smoking status (never smoked, past smoker, currently smoke 1–14 cigarettes/day, 15–24 cigarettes/day, or ≥25 cigarettes/day), alcohol intake (0 g/day, 0.1–4.9 g/day, 5.0–9.9 g/day, 10.0–14.9 g/day, 15.0–29.9 g/day, and ≥30 g/day for men; 0 g/day, 0.1–4.9 g/day, 5.0–9.9 g/day, 10.0–14.9 g/day, ≥15 g/day for women), multivitamin use (yes, no), physical activities (quintiles), family history of diabetes. For women, postmenopausal status and hormone use, and oral conceptive use were further adjusted for.

‡Based on model 2, model 3 was further adjusted for total energy (kcal/day) and intake of red meat, fruits and vegetables, whole grains, and coffee (all in quintiles).

TABLE 3

Relative risk (95% CI) of type 2 diabetes risk according to brown rice in the HPFS, NHS I, and NHS II.

	Brown rice intake (serving/time)			
	<1/mo	1/mo-1/wk	≥2/wk	P for trend
HPFS				
Number of cases	1142	1296	210	
Person-year	283230	358085	61606	
Model 1*	1.0	0.84 (0.77, 0.91)	0.77 (0.67, 0.90)	0.0002
Model 2†	1.0	0.95 (0.88, 1.03)	0.96 (0.83, 1.12)	0.53
Model 3‡	1.0	0.96 (0.89, 1.04)	0.96 (0.82, 1.12)	0.51
NHS I				
Number of cases	3127	2167	206	
Person-year	785713	554634	64026	
Model 1*	1.0	0.78 (0.74, 0.83)	0.63 (0.54, 0.72)	<0.0001
Model 2†	1.0	0.92 (0.87, 0.97)	0.83 (0.72, 0.96)	0.001
Model 3‡	1.0	0.92 (0.87, 0.98)	0.83 (0.72, 0.96)	0.003
NHS II				
Number of cases	1271	941	147	
Person-year	580179	537974	92750	
Model 1*	1.0	0.71 (0.65, 0.78)	0.62 (0.52, 0.73)	<0.0001
Model 2†	1.0	0.92 (0.85, 1.01)	0.88 (0.74, 1.05)	0.10
Model 3‡	1.0	0.95 (0.87, 1.04)	0.89 (0.75, 1.07)	0.17
Pooled				
Fixed effects model‡	1.0	0.94 (0.90, 0.98)	0.89 (0.81, 0.97)	0.005
$P_{heterogeneity}$	-	0.77	0.45	0.21

*
Age-adjusted.

†
Adjusted for the same set of covariates for model 2 in Table 2.

‡
Based on model 2, model 3 was further adjusted for total energy (kcal/day) and intake of red meat, fruits and vegetables, coffee (in quintiles), and white rice (see Table 2 for intake categories).

TABLE 4

Relative risk (95% CI) of type 2 diabetes risk according to whole grain, as well as bran or germ intake in the HPFS, NHS I, and NHS II.

| | Intake quintiles | | | | | |
	Q1	Q2	Q3	Q4	Q5	P for trend
Whole grain						
HPFS						
Intake levels*	5.1 (2.7–7.2)	12.6 (10.4–14.9)	20.4 (17.9–22.7)	29.9 (27.0–32.9)	47.1 (41.1–57.6)	
Number of cases	667	574	559	475	373	
Person-year	132594	142608	143546	144486	139687	
RR (95% CI)†	1.0	0.82 (0.73, 0.92)	0.86 (0.77, 0.97)	0.78 (0.69, 0.88)	0.72 (0.63, 0.83)	<0.0001
NHS I						
Intake levels*	3.6 (2.2–5.0)	8.2 (6.7–10.0)	13.0 (11.2–15.1)	19.4 (17.0–21.5)	31.3 (27.0–38.3)	
Number of cases	1346	1246	1158	971	779	
Person-year	271378	281807	283347	285978	281864	
RR (95% CI)†	1.0	0.89 (0.83, 0.97)	0.85 (0.79, 0.92)	0.76 (0.70, 0.83)	0.70 (0.64, 0.77)	<0.0001
NHS II						
Intake levels*	6.2 (4.0–8.0)	12.6 (11.0–14.3)	18.8 (16.9–20.4)	26.2 (23.9–28.6)	40.0 (35.1–48.4)	
Number of cases	676	524	462	377	320	
Person-year	236653	241524	243592	244875	244259	
RR (95% CI)†	1.0	0.89 (0.80, 1.00)	0.89 (0.79, 1.00)	0.81 (0.71, 0.92)	0.81 (0.70, 0.94)	0.002
Pooled RR (95% CI)						
Fixed effects model†	1.0	0.87 (0.83, 0.93)	0.86 (0.81, 0.91)	0.78 (0.73, 0.83)	0.73 (0.68, 0.78)	<0.0001
$P_{heterogeneity}$	-	0.41	0.86	0.78	0.23	0.01
Bran						
HPFS						
Intake levels*	0.6 (0.3–1.0)	2.1 (1.5–2.8)	4.3 (3.3–5.3)	7.5 (6.2–8.8)	14.3 (11.8–18.6)	
Number of cases	634	616	546	480	372	
Person-year	133254	144206	143049	142852	139558	
RR (95% CI)†	1.0	0.90 (0.79, 1.01)	0.84 (0.74, 0.96)	0.79 (0.68, 0.91)	0.69 (0.60, 0.81)	<0.0001
NHS I						

	Intake quintiles					P for trend
	Q1	Q2	Q3	Q4	Q5	
Intake levels*	0.6 (0.3–0.9)	1.5 (1.1–2.1)	2.8 (2.1–3.6)	4.8 (3.9–5.8)	9.5 (7.7–12.6)	
Number of cases	1278	1262	1182	1009	769	
Person-year	270994	278327	284764	285877	284412	
RR (95% CI)†	1.0	0.96 (0.88, 1.04)	0.92 (0.84, 1.01)	0.85 (0.77, 0.94)	0.77 (0.69, 0.86)	<0.0001
NHS II						
Intake levels*	1.0 (0.7–1.4)	2.6 (2.2–3.0)	4.3 (3.8–4.8)	6.7 (5.9–7.5)	12.1 (10.0–15.9)	
Number of cases	667	512	446	393	341	
Person-year	234309	244811	242569	245347	243867	
RR (95% CI)†	1.0	0.87 (0.77, 0.98)	0.84 (0.74, 0.96)	0.85 (0.74, 0.98)	0.83 (0.71, 0.97)	0.07
Pooled RR (95% CI)						
Fixed effects model†	1.0	0.92 (0.87, 0.98)	0.88 (0.82, 0.94)	0.84 (0.78, 0.90)	0.76 (0.71, 0.82)	<0.0001
$P_{heterogeneity}$	-	0.39	0.43	0.68	0.28	0.17
Germ						
HPFS						
Intake levels*	0.2 (0.1–0.3)	0.5 (0.4–0.6)	0.8 (0.7–0.9)	1.2 (1.1–1.4)	2.3 (1.9–3.4)	
Number of cases	577	592	563	506	410	
Person-year	129975	147061	141461	145060	139362	
RR (95% CI)†	1.0	1.00 (0.88, 1.13)	1.06 (0.93, 1.21)	1.04 (0.90, 1.20)	1.04 (0.89, 1.21)	0.65
NHS I						
Intake levels*	0.2 (0.1–0.2)	0.3 (0.3–0.4)	0.6 (0.5–0.6)	0.8 (0.7–0.9)	1.5 (1.3–2.1)	
Number of cases	1224	1163	1237	1038	838	
Person-year	256119	295315	287868	288588	276483	
RR (95% CI)†	1.0	0.93 (0.85, 1.02)	0.99 (0.90, 1.09)	0.92 (0.83, 1.02)	0.88 (0.79, 0.97)	0.02
NHS II						
Intake levels*	0.3 (0.2–0.4)	0.6 (0.5–0.7)	0.9 (0.8–1.0)	1.2 (1.1–1.4)	2.0 (1.7–2.4)	
Number of cases	617	464	484	391	403	
Person-year	234430	245739	241509	245747	243479	
RR (95% CI)†	1.0	0.93 (0.82, 1.05)	1.04 (0.91, 1.19)	0.90 (0.78, 1.04)	1.04 (0.90, 1.21)	0.56

	Intake quintiles					
	Q1	Q2	Q3	Q4	Q5	P for trend
Pooled RR (95% CI)						
Fixed effects model[†]	1.0	0.95 (0.89, 1.01)	1.02 (0.95, 1.09)	0.94 (0.88, 1.01)	0.95 (0.88, 1.03)	0.40
$P_{heterogeneity}$	-	0.65	0.67	0.29	0.09	0.06

[*] Median (interquartile range)

[†] Adjusted for the same set of covariates for model 3 in Table 3. Bran and germ intake were mutually adjusted in the analysis.