

Associations of Breakfast Cereal Consumption With Risk of Cardiovascular Disease and Mortality in Individuals With Diabetes

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Abstract

Background

While whole-grain cereals reduce cardiovascular and mortality risk in the general population, it remains unclear whether these benefits extend uniformly to all whole-grain breakfast cereals, particularly in individuals with diabetes. We aimed to investigate the associations of breakfast cereal consumption with cardiovascular disease (CVD) events, CVD and all-cause mortality in diabetes.

Methods

This study included 10 146 individuals with diabetes from the UK Biobank. Breakfast cereal intake was assessed via at least one 24-hour dietary recall. Cox proportional hazards models were used to estimate hazard ratios (HR) for CVD events, CVD and all-cause mortality. Mediation analyses were performed to explore underlying mechanisms.

Results

Over a median follow-up of 12.8 years, 3 264 CVD events and 1 882 deaths (515 cardiovascular) occurred. Compared with non-consumers, breakfast cereal intake (≥ 1.5 bowl) was associated with reduced risks of CVD incidence (HR, 0.86; 95% CI, 0.77–0.95), CVD mortality (HR, 0.66; 95% CI, 0.51–0.85) and all-cause mortality (HR, 0.85; 95% CI, 0.77–0.97). However, these associations varied substantially by cereal type. Higher intake of porridge (≥ 1 bowl) was associated with lower CVD incidence (HR, 0.90; 95% CI, 0.82–0.98), CVD mortality (HR, 0.77; 95% CI, 0.60–0.99), and all-cause mortality (HR, 0.80; 95% CI, 0.71–0.91). Muesli consumption (≥ 1 bowl) was associated with reduced CVD incidence (HR, 0.88; 95% CI, 0.77–0.99) and CVD mortality (HR, 0.70; 95% CI, 0.45–0.97). Conversely, sweetened cereal intake (≥ 0.5 bowl) was associated with higher CVD incidence (HR, 1.36; 95% CI, 1.11–1.64) and all-cause mortality (HR, 1.29; 95% CI, 1.01–1.71). C-reactive protein, BMI, and waist-to-hip ratio accounted for 4.99%, 4.14%, and 3.75% of the association between breakfast cereal intake and all-cause mortality.

Conclusion

Breakfast cereal intake was associated with lower CVD and mortality risk in diabetes; however, this concealed opposing associations by cereal type, with benefits confined to porridge and muesli, harms associated with sweetened cereals.

Introduction

Global prevalence of diabetes continues to rise, presenting a substantial chronic disease burden that threatens both public health and the sustainability of healthcare systems. According to the Global Burden of Disease study, the diabetic population is projected to increase further in the coming decades, underscoring the ongoing challenges in diabetes prevention and control ^{1,2}. Concurrently, cardiovascular disease represents one of the most prevalent and severe complications among individuals with diabetes ³. Evidence indicates that approximately 32.2% of patients with type 2 diabetes mellitus globally are affected by co-existing CVD. Notably, these individuals exhibit a two- to four-fold increased risk of mortality, myocardial infarction, and stroke compared to the general population ⁴. Therefore, identifying sustainable dietary behaviors with potential cardiovascular benefits within the diabetic population holds substantial public health significance for improving prognosis and reducing the risk of premature mortality.

Within the broad category of modifiable dietary factors, breakfast cereals represent a uniquely important food group. Their high accessibility and stable, habitual consumption patterns make them a practical and impactful target for public health guidance. However, from a clinical perspective, they cannot be treated as a single entity. The nutritional profiles of these products range dramatically, from fiber-rich, minimally processed bran and porridge to highly refined, sugar-sweetened varieties. This inherent heterogeneity raises a clinically important question: within the umbrella of commonly consumed grain-based foods, do nutritionally distinct subtypes of a staple product differentially affect health?

This question is of particular urgency for the management of type 2 diabetes. Current American Diabetes Association guidelines recommend prioritizing high-fiber, nutrient-dense carbohydrates and advise that at least half of grain intake come from whole grains ⁵. However, translating this quantitative target into specific food choices is challenging, precisely because of the nutritional complexity described above. While the benefits of cereal fiber and specific grains like oats for improving lipid profiles, glycemic control, and inflammation are well-established from general population studies and single-food intervention trials ^{6–11}, a critical gap remains in translating these principles into actionable, food-based advice for individuals with diabetes. A recent UK Biobank analysis demonstrated that the associations between breakfast cereal consumption and mortality differed markedly by cereal type, with fiber-rich varieties such as muesli, bran cereal, and porridge showing protective associations, while refined or sweetened cereals did not ¹². Yet, this study was conducted in the general population and did not examine whether these associations extend to individuals with diabetes, a group at substantially higher absolute cardiovascular risk. Consequently, prospective evidence is lacking on whether specific breakfast cereal subtypes translate into tangible differences in hard endpoints, namely cardiovascular events and mortality, within this high-risk group.

To address this gap, we used data from the UK Biobank to prospectively evaluate the associations of total and type-specific breakfast cereal intake with incident cardiovascular events, cardiovascular mortality, and all-cause mortality among individuals with diabetes. We also explored potential underlying mechanisms to provide an evidence-based foundation for more tailored dietary recommendations at breakfast in this high-risk population.

Methods

Study participants

We used data from the UK Biobank, a prospective, population-based cohort of over 500 000 adults recruited between 2006 and 2010 across 22 assessment centres in England, Scotland, and Wales. At baseline, participants completed touchscreen questionnaires, verbal interviews, and physical and functional measurements. Blood samples were also collected for biomarker and genetic analyses¹³. Diabetes was defined based on a validated algorithm using UK Biobank self-reported medical history and medication information (diagnosis, age at diagnosis, diabetes type, diabetes medications, and diabetes-related complications), or a baseline glycated hemoglobin A1c (HbA1c) level $\geq 6.5\%$ (≥ 48 mmol/mol) according to American Diabetes Association criteria¹⁴.

In this study, we initially excluded participants who did not complete any online 24-hour dietary recall questionnaire, leaving 210 540 individuals who completed at least one 24-hour dietary assessment. We further excluded participants lost to follow-up ($n = 559$), those with implausible dietary intake (defined as total energy intake > 20 MJ/day for men or > 18 MJ/day for women; $n = 3\ 068$), those in the top 1% of the cereal intake distribution ($n = 3\ 522$), and those without diabetes ($n = 193\ 245$). Ultimately, 10 146 participants were eligible for the subsequent analyses (Fig. 1).

Assessment of breakfast cereal consumption

Dietary intake was assessed using a web-based 24-hour recall questionnaire administered on up to five occasions between April 2009 and June 2012. In each recall, participants reported whether they had consumed any 'breakfast cereal' in the preceding 24 hours. In the UK Biobank, this term denoted a food category consumed at any time of day, encompassing porridge, muesli, oat crunch, sweetened cereal, plain cereal, whole-wheat cereal, bran cereal, and other cereal, but excluding cereal bars. Participants reported the type of cereal consumed and the amount (bowl). Mean daily intake of each cereal type was calculated across all available recalls. Participants who reported any breakfast cereal consumption in at least one recall were classified as consumers. For these individuals, we also recorded whether they added milk, sugar, artificial sweeteners, or dried fruit to their cereal, to explore the potential modifying effects of these additions on the cereal-outcome associations.

Outcomes assessment

Information on the date and cause of death was obtained through linkage to national datasets from the NHS Information Centre (England and Wales) and the NHS Central Register (Scotland). The study focused on the outcomes of incident CVD, CVD mortality, and all-cause mortality. Outcomes were defined according to ICD-10 codes: incident CVD (I21–I25, I48, I50, I60–I64, I70, I73), CVD mortality (I00–I99), and all-cause mortality (death from any cause). Participants were followed until the date of incident CVD, death, withdrawal from the study, or the end of follow-up (October 31, 2023), whichever came first.

Covariates

Potential confounding covariates were collected from the baseline questionnaire and assessment data of the UK Biobank. The covariates considered in this study included age, sex, ethnicity, household income, education level, Body Mass Index (BMI), smoking status, physical activity level, Townsend deprivation index (TDI), and various lifestyle, and clinical, dietary factors.

Age and BMI were treated as a continuous variable. Categorical covariates included sex (male or female), ethnicity (White or other), education level (degree or no degree), and smoking status (current, former, or never). Household income was grouped into five categories: $>£100,000$, $£52,000–100,000$, $£31,000–51,999$, $£18,000–30,999$, and $<£18,000$. Physical activity level was classified as low, moderate, or high based on a standard questionnaire-based assessment. The TDI was used as a composite indicator of socioeconomic deprivation.

Binary covariates (yes or no) comprised vitamin use, mineral/supplement use, family history of CVD or cancer, hypertension, hypercholesterolemia, and aspirin use. A healthy sleep pattern was defined as a score of ≥ 4 , derived from a 5-item healthy sleep score (each trait coded as 1 if met and 0 otherwise). Dietary covariates included continuous measures of coffee and tea intake, alongside overall diet quality assessed via a modified Alternative Healthy Eating Index (AHEI) score (with higher scores indicating a healthier dietary pattern)¹⁵.

Statistical analysis

Baseline characteristics of the study population were described according to cereal consumption status (non-consumers and cereal-consumers). Continuous variables were expressed as means with standard deviations (SDs), whereas categorical variables were presented as frequencies and percentages. Between-group differences were assessed using Student's t-test for continuous variables and chi-square tests for categorical variables. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the associations of breakfast cereal consumption with incident CVD, CVD mortality, and all-cause mortality among participants with diabetes. Total breakfast cereal intake was categorized as 0, 0 to < 1.5 , and ≥ 1.5 bowls/day. In addition, consumption of different types of breakfast cereals was further categorized into three groups. Intake of porridge, muesli, plain cereal, bran cereal, and whole wheat cereal was classified as 0 bowl/day, 0 to < 1 bowl/day, and ≥ 1 bowl/day. Because few participants consumed more than 1 bowl/day of oat crunch, sweetened cereal, and other cereal, intake of these cereal types was classified into three categories: 0 bowl/day, 0 to 0.5 bowl/day, and > 0.5 bowl/day. All models used 0 bowl/day as the reference group. The proportional hazard assumption was assessed using Schoenfeld residuals and log-log plots based on Kaplan–Meier estimates, and all evaluations satisfied the predetermined criteria. To visualize the

dose–response relationships between specific breakfast cereal intake and the risks of study outcomes, restricted cubic spline regression models were applied, with knots placed at the 5th, 45th, and 95th percentiles of the intake distribution.

Subgroup analyses were conducted to examine whether the associations of total and type-specific breakfast cereal intake with all-cause mortality differed across population subgroups. All-cause mortality was selected as the primary endpoint for these analyses, as it is the most clinically definitive outcome and the least susceptible to misclassification bias among the three endpoints examined. Stratified analyses were performed according to age (< 60 or ≥ 60 years), sex (male or female), hypertension status (yes or no), ethnicity (White or non-White), physical activity level (low, moderate, or high), body mass index (BMI; <30 or ≥ 30 kg/m²), smoking status (yes or no), and TDI (low or high).

Several sensitivity analyses were further performed to test the robustness of the findings. First, we performed a complete-case analysis by excluding participants with missing covariates. Second, participants who developed the study outcomes within the first 2 years of follow-up were excluded to minimize potential reverse causation. Third, participants who reported not following their usual diet on the previous day in any of the five 24-hour dietary recalls were excluded, to better capture their habitual breakfast cereal consumption. Fourth, BMI was excluded from the covariates to assess its potential mediating effect. Fifth, in the analyses of specific types of breakfast cereal, we mutually adjusted for the intakes of other cereal types to evaluate their independent effects.

To investigate whether C-reactive protein (CRP), BMI, and waist-to-hip ratio (WHR) mediated the association between breakfast cereal intake and all-cause mortality, mediation analyses were performed using a counterfactual-based framework. CRP, BMI, and WHR were evaluated separately as potential mediators in single-mediator analyses. For each mediator, two regression models were fitted adjusting for the aforementioned covariates: (1) a mediator model ($A \rightarrow M$), in which multivariable linear regression was used to assess the association between breakfast cereal intake and the mediator; and (2) an outcome model ($A + M \rightarrow Y$), in which Cox proportional hazards regression was used to estimate the associations of breakfast cereal intake and the mediator with all-cause mortality. The total effect (TE), natural direct effect (NDE), and natural indirect effect (NIE) were estimated, and the proportion mediated (PM) was calculated as $(NIE/TE) \times 100\%$. Hazard ratios (HRs) and 95% confidence intervals (CIs) derived via the delta method were reported. Mediation analyses were conducted using the R package *regmedint*.

Results

Baseline characteristics

Table 1 presents the baseline characteristics of the study population according to breakfast cereal consumption status. Among the 10 146 participants included in the analysis, 2 823 were non-consumers and 7 323 were cereal-consumers. Compared with non-consumers, cereal-consumers were older, more likely to be female, more likely to be White, and had a lower Townsend deprivation index, indicating less socioeconomic deprivation. They were also characterized by higher household incomes, advanced educational attainment, a lower mean BMI, and a higher basal metabolic rate. Regarding lifestyle and diet, cereal-consumers generally exhibited healthier profiles. They were less likely to be current smokers and reported healthier sleep patterns, more regular physical activity, and increased use of vitamins and minerals. Additionally, they consumed more coffee and tea and scored higher on the AHEI.

Associations of breakfast cereal consumption with cardiovascular disease and mortality outcomes

Over a median follow-up of 12.8 years, 3 264 incident CVD events and 1 882 deaths (including 515 CVD-related deaths) were documented. Table 2 presents the associations between breakfast cereal, the specific types and the risks of CVD incidence, CVD mortality, and all-cause mortality. Using non-consumers (0 bowl/day) as the reference group, higher consumption of breakfast cereals was significantly associated with decreased risks across multiple endpoints. Regarding CVD incidence, in the fully adjusted model (Model 3), participants consuming ≥1.5 bowls/day exhibited a lower risk of developing CVD (HR, 0.86; 95% CI, 0.77–0.95). However, moderate consumption (>0 to 1.5 bowls/day) did not show a statistically significant association with CVD incidence (HR, 0.99; 95% CI, 0.91–1.08). For mortality outcomes, a more pronounced inverse association was observed. In Model 3, compared to the reference group, breakfast cereal intake of >0 to 1.5 bowls/day and ≥ 1.5 bowls/day were associated with a 25% (HR, 0.75; 95% CI, 0.62–0.92) and 34% (HR, 0.66; 95% CI, 0.51–0.85) reduction in CVD mortality risk, respectively. Similarly, an inverse relationship was evident for all-cause mortality; the corresponding hazard ratios in the fully adjusted model were 0.88 (95% CI, 0.78–0.98) for participants consuming >0 to 1.5 bowls/day and 0.85 (95% CI, 0.77–0.97) for those consuming ≥ 1.5 bowls/day, indicating a consistent protective effect against premature death (Table 2).

In the fully adjusted model, different breakfast cereal types exhibited divergent associations with health outcomes. Compared with non-consumers (0 bowl/day), participants consuming ≥1 bowl/day of porridge had a 10% lower risk of CVD incidence (HR, 0.90; 95% CI, 0.82–0.98), a 23% lower risk of CVD mortality (HR, 0.77; 95% CI, 0.60–0.99), and a 20% lower risk of all-cause mortality (HR, 0.80; 95% CI, 0.71–0.91). Similarly, muesli intake was associated with significant risk reductions; specifically, ≥1 bowl/day was linked to a 12% reduction in CVD incidence (HR, 0.88; 95% CI, 0.77–0.99) and a 30% reduction in CVD mortality (HR, 0.70; 95% CI, 0.45–0.97), and a decreasing trend in all-cause mortality (HR, 0.83; 95% CI, 0.70–1.01). Notably, moderate muesli intake (0 to <1 bowl/day) was significantly associated with a 26% lower risk of all-cause mortality (HR, 0.74; 95% CI, 0.58–0.91) (Table 2).

In contrast, sweetened cereal consumption was associated with increased risks. In Model 3, consuming > 0.5 bowl/day of sweetened cereal was associated with a 36% higher risk of CVD incidence (HR: 1.36; 95% CI: 1.11–1.64) and a 29% higher risk of all-cause mortality (HR: 1.29; 95% CI: 1.01–1.71). Regarding other varieties, bran cereal intake at moderate levels (0 to <1 bowl/day) was significantly associated with a 26% lower risk of all-cause mortality (HR: 0.74; 95% CI: 0.59–0.92). However, no statistically significant associations were observed for plain cereal, whole-wheat cereal, oat crunch, or other cereal types after full adjustment (Table 2).

In restricted cubic spline analyses, a significant linear inverse association was observed between total breakfast cereal intake and CVD mortality (P for overall = 0.002; P for nonlinear = 0.055), whereas the association with all-cause mortality was nonlinear (P for overall = 0.006; P for nonlinear = 0.012). For specific cereal types, muesli intake exhibited nonlinear associations with both CVD mortality (P for overall = 0.017; P for nonlinear = 0.015) and all-cause mortality (P for overall = 0.009; P for nonlinear = 0.072). A similar nonlinear association was detected between other cereal intake and all-cause mortality (P for overall = 0.042; P for nonlinear = 0.039). Porridge intake showed a significant linear inverse association with all-cause mortality (P for overall = 0.048; P for nonlinear = 0.308). In contrast, sweetened cereal consumption displayed a significant linear positive association with CVD incidence (P for overall = 0.044; P for nonlinear = 0.385). No significant associations were observed for the remaining cereal-outcome pairs (Figure S2).

Additionally, we further investigated the associations of breakfast cereal consumers with CVD incidence, CVD mortality, and all-cause mortality according to reported additions of milk, sugar, artificial sweeteners, and dried fruit (Table 3). For all-cause mortality, among consumers who reported adding sugar, artificial sweeteners, milk, and dried fruit, the HR (95% CI) was 0.86 (0.77–0.97), 0.98 (0.75–1.27), 0.87 (0.76–0.98), and 0.78 (0.68–0.91), respectively. In participants who did not add these ingredients, the corresponding estimates were 0.78 (0.64–0.95), 0.84 (0.75–0.95), 0.76 (0.55–0.86), and 0.89 (0.77–0.96), respectively. For CVD-specific mortality, more pronounced inverse associations were observed. Among breakfast cereal consumers who reported adding sugar, artificial sweeteners, milk, and dried fruit, the HR (95% CI) was 0.71 (0.57–0.87), 0.91 (0.57–0.88), 0.72 (0.34–0.81), and 0.58 (0.45–0.76), respectively. In the corresponding non-addition groups, the HR (95% CI) was 0.63 (0.44–0.91), 0.70 (0.57–0.87), 0.53 (0.58–0.88), and 0.76 (0.61–0.95), respectively. However, for CVD incidence, no significant associations were generally observed across all stratified analyses.

Subgroup and sensitivity analysis

No significant interactions were observed between intakes of porridge, muesli, plain cereal, whole-wheat cereal, bran cereal, or other cereal and all-cause mortality across any of the examined subgroups (all P for interaction > 0.05). For oat crunch intake, the association with all-cause mortality differed by hypertension status: an inverse association was observed in participants with hypertension (HR, 0.63; 95% CI, 0.42–0.95), whereas a positive association was found in those without hypertension (HR, 1.64; 95% CI, 1.11–2.43; P for interaction = 0.01). Similarly, the positive association between sweetened cereal intake and all-cause mortality was more pronounced in non-smokers (HR, 1.80; 95% CI, 1.26–2.57) than in smokers (HR, 1.01; 95% CI, 0.73–1.39; P for interaction = 0.01). No other significant interactions were detected for oat crunch or sweetened cereal across the remaining subgroups (all P for interaction > 0.05) (Figure S3).

In sensitivity analyses, the findings did not materially change after excluding participants with missing covariates (Table S2), after excluding participants who experienced death events within the first 2 years of follow-up (Table S3), after excluding participants who reported not following their usual diet on the previous day in any of the five 24-hour dietary recalls (Table S4), after excluding BMI from the covariates (Table S5), or after mutually adjusting for the intakes of other cereal types in the analyses of specific types of breakfast cereal (Table S6).

Mediation analyses

Mediation analyses indicated that CRP, BMI, and WHR each partially mediated the association between breakfast cereal intake and all-cause mortality. Breakfast cereal intake was inversely associated with these three mediators, whereas higher levels of these mediators were associated with an increased risk of all-cause mortality. The natural indirect effects through these pathways were all statistically significant, with mediation proportions of 4.99%, 4.14%, and 3.75%, respectively. Furthermore, BMI was also found to partially mediate the association between breakfast cereal intake and CVD mortality, with a statistically significant natural indirect effect and a mediation proportion of 3.09% (Figure 2).

Discussion

In this prospective analysis of patients with diabetes from the UK Biobank, fully adjusted models revealed that higher breakfast cereal consumption (≥ 1.5 bowls/day) was significantly associated with reduced risks of CVD incidence, CVD mortality, and all-cause mortality compared to non-consumers. When disaggregating breakfast cereals by subtype, these favorable associations remained pronounced for porridge and muesli; conversely, sweetened cereal was associated with an increased CVD incidence and all-cause mortality risk. These associations were consistent across subgroups and sensitivity analyses. Moreover, adding dried fruit appeared to further strengthen the protective association with mortality, while adding sugar or artificial sweeteners weakened it. Mediation analyses further suggested that CRP, BMI, and WHR partially underlie the associations between breakfast cereal consumption and all-cause mortality. For CVD mortality, only BMI reached statistical significance as a mediator.

Among patients with diabetes in the UK Biobank, higher total breakfast cereal intake (≥ 1.5 bowls/day) was associated with a lower risk of CVD and all-cause mortality. These findings add to the existing evidence on breakfast cereals and health, which has largely been derived from general populations. A systematic review of 232 articles reported that regular breakfast cereal consumption is associated with higher diet quality, but noted that most studies were cross-sectional and that cereal intake may simply be a marker of an overall healthy lifestyle¹⁶. The same review found limited evidence for cardiovascular disease¹⁶. Our study extends these observations to individuals with diabetes, a population at disproportionately high cardiovascular risk. Several observational studies have reported that breakfast cereal consumption is associated with a lower body mass index and reduced risk of overweight and obesity^{17–19}. A 12-year longitudinal study of mid-age Australian women further found that oat-based cereal, muesli, and all-bran were specifically associated with a significant reduction in obesity risk²⁰. These findings suggest that the protective associations of total breakfast cereal intake may be partly mediated by favorable effects on body weight. Our mediation analyses indicate that BMI, WHR, and CRP accounted for only a modest fraction of the observed associations, implying that additional pathways, such as modulation of the gut microbiota, or improvements in vascular function, may also contribute. These alternative mechanisms warrant further investigation in future studies.

When we disaggregated total breakfast cereal intake by subtype, porridge was associated with lower risks of CVD incidence, CVD mortality, and all-cause mortality. Muesli also showed protective associations. In contrast, sweetened cereals were associated with higher risks of CVD incidence and all-cause mortality. These subtype-specific patterns are broadly consistent with the prior study to examine breakfast cereal subtypes and mortality in a general UK Biobank cohort, which reported that porridge and muesli were associated with lower mortality, whereas sweetened cereals were associated with elevated risks¹². The protective associations observed for porridge and muesli are biologically plausible. Both provide a food matrix that preserves grain integrity. Porridge, made from minimally processed whole oats, is particularly rich in β -glucan, a soluble fiber that improves insulin sensitivity and lowers LDL cholesterol^{6,21}. Its fermentable fiber also serves as a substrate for gut microbiota, generating short-chain fatty acids that suppress systemic low-grade inflammation^{22,23}. Muesli extends this nutritional base by incorporating raw oats, nuts, and seeds, thereby delivering unsaturated fatty acids and phytochemicals that confer additional cardiovascular protection through anti-inflammatory and antioxidant pathways^{7,24}. Furthermore, its rich array of phytochemicals and minerals enhances immune function and improves metabolic syndrome indices²⁵. Additionally, both porridge and muesli are high whole-grain and low in added sugar, which may explain the protective effect. A consistent body of research supports our speculation. For instance, meta-analyses of randomized controlled trials have demonstrated that whole grain consumption significantly decreases total cholesterol, low-density lipoprotein cholesterol, and specific inflammatory markers, such as CRP and interleukin-6^{9,26,27}. Dose-response meta-analyses have unequivocally established an inverse association of higher intakes of whole grains and cereal fiber with both CVD incidence and all-cause mortality^{6,21}. Epidemiological studies have also indicated that higher whole grain intake was inversely associated with body weight and the risk of obesity^{7,28}. Moreover, a Mendelian randomization analysis revealed a robust causal relationship between the habitual consumption of mixed cereals (enriched with nuts, and dried fruit) and a substantial reduction in the risk of coronary heart disease compared to refined grains²⁹. Likewise, a systematic review and meta-analysis of cohort studies found that higher added sugar intake was associated with increased risks of all-cause mortality and CVD mortality³⁰. Extending these findings, another review indicates that high dietary sugar intake was linked to a range of adverse health outcomes, including obesity, type 2 diabetes, CVD, and mortality³¹.

Meta-analyses of randomized controlled trials have demonstrated that cereal bran and specific oat-based soluble fibers can improve key cardiovascular risk markers, including blood pressure, total cholesterol, and postprandial glycemic control, particularly in diabetic populations^{7,32}. Yet, in our study, several predominantly whole-grain cereal types, including whole-wheat cereal and bran cereal, showed no significant associations with any outcome, in stark contrast to the protective associations observed for porridge and muesli. This discrepancy likely reflects critical differences in food matrix integrity and processing methods that are not captured by the whole-grain label alone. Unlike minimally processed porridge and muesli, many whole-wheat and bran cereals are extensively processed. Extrusion, puffing, or fine milling disrupts grain structure, accelerates starch gelatinization, and may produce higher glycemic responses despite their whole-grain label. Emerging evidences strongly support this interpretation. A systematic review of oat-based products has shown that milling intensity and particle size markedly alter postprandial glycemic responses; even when derived from the same whole grain source, more finely processed products produce significantly higher glycemic indices than minimally processed forms such as steel-cut oats or traditional porridge³³. Similarly, controlled crossover trials in adults with type 2 diabetes have demonstrated that less-processed whole grains with intact structural matrices result in lower postprandial glucose excursions compared with finely milled counterparts, despite equivalent macronutrient composition. These differences are attributed to slower starch gelatinization and reduced digestive enzyme accessibility in intact grain structures³⁴. Importantly, when wheat-based products were matched for grain origin and nutrient composition and both finely milled, whole-grain and refined formulations elicited comparable glycemic responses³⁵. Therefore, dietary recommendations may move beyond the binary classification of whole versus refined grains and instead consider particle size, processing intensity, and food form when guiding carbohydrate choices, particularly in individuals with impaired glucose regulation.

In subgroup analyses, the association between oat crunch and the outcome appeared to be modified by hypertension status, whereas the association between sweetened cereals and the outcome varied by smoking status. Oats are rich in soluble dietary fiber, which has been shown to exert a blood pressure-lowering effect³⁶. Therefore, compared with normotensive individuals, those with hypertension or a higher burden of cardiometabolic risk might derive greater benefit from oat consumption. Nonetheless, differences in processing methods and dietary fiber density among various oat products could attenuate or even alter the expected protective association. The observed differential findings for sweetened cereals by smoking status may partly reflect residual confounding by smoking, a major cardiometabolic risk factor in diabetes that can mask or dilute dietary effects³⁷.

Strengths and limitations

This study possesses several distinct strengths. First, based on a large-scale prospective analysis with comprehensive confounder adjustment, a key novel insight is that not all breakfast whole-grain cereals conferred equal benefit, a discrepancy likely reflecting differences in food matrix integrity and processing methods not captured by the whole-grain label alone. Second, repeated 24-hour dietary recalls allowed for a detailed assessment of overall breakfast cereal intake, cereal subtypes, and added ingredients. In addition, sensitivity analyses and mediation analysis strengthened the reliability and biological interpretation of our findings. Several limitations, however, should be noted. As with any observational study, causal relationships cannot be definitively inferred, and residual confounding may remain. Breakfast cereal intake was self-reported and thus subject to recall bias or measurement error. Furthermore, we lacked granular information on specific cereal products, processing levels, and exact nutrient composition. Moreover, the healthy volunteer bias inherent in the UK Biobank cohort may limit the generalizability of our findings.

Conclusion

In conclusion, this study shows that the health effects of breakfast cereals in patients with diabetes vary substantially by cereal type. Porridge was associated with lower risks of CVD incidence, CVD and all-cause mortality, whereas muesli showed protective associations. In contrast, sweetened cereals were associated with higher risks of CVD incidence and all-cause mortality. No significant associations were observed for plain cereal, bran cereal, whole-wheat cereal, or oat crunch. These findings underscore the importance of distinguishing between cereal subtypes, rather than treating all breakfast

cereals as a single food group, when advising patients with diabetes on dietary choices. Future clinical trials are warranted to confirm these observations and to establish whether substituting sweetened cereals with porridge or muesli can improve cardiovascular outcomes in this high-risk population.

Declarations

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Availability of data and materials: Data from the UK Biobank are available in a public, open access repository. This research has been conducted using the UK Biobank Resource under Application Number 160957. The data that support the findings of this study are available on request from the UK Biobank. (<https://www.ukbiobank.ac.uk/>).

Author Contributions:SD, LZP, WQX, YAM, YHX and LGY conceived and designed the research; LZP wrote the manuscript; LZP and WQX performed the data analysis. All authors contributed to the interpretations of the findings.

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Competing Interests:The authors declare that they have no competing interests.

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Tables

Table 1. Baseline characteristics of participants.

Characteristic	Total	Non-consumers	Cereal-consumers	P-Values
Participants, n(%)	10146(100)	2823(27.8)	7323(72.2)	
Mean age (SD), y	58.9(7.2)	57.7(7.6)	59.4(7.0)	<0.001
Male, n (%)	6242(61.5)	1800(17.7)	4442(43.8)	<0.001
BMI (kg/m ²), n (%)				<0.001
<25	1337(13.3)	304(10.9)	1033(14.2)	
≥25 & <30	3626(35.9)	911(32.6)	2715(37.2)	
≥30	5124(50.8)	1583(56.6)	3541(48.6)	
Mean basal metabolic rate (SD), kJ	7399.9(1493.6)	7528.9(1547.9)	7350.3(1469.4)	<0.001
Ethnicity, n (%)				<0.001
White	9070(89.4)	2354(83.4)	6716(91.7)	
Other	1076(10.6)	469(16.6)	607(8.3)	
Mean Townsend deprivation index (SD)	-0.96(3.2)	-0.43(3.3)	-1.17(3.1)	<0.001
Household income (£), n (%)				<0.001
<18 000	2312(22.8)	676(27.4)	1636(25.4)	
18 000–30 999	2528(24.9)	667(27.1)	1861(28.9)	
31 000–51 999	2213(21.8)	591(24.1)	1622(25.2)	
52 000–100 000	1517(14.9)	425(17.3)	1092(20.0)	
>100 000	326(3.2)	101(4.1)	225(3.5)	
Education				<0.001
Degree	6767(66.7)	1873(66.3)	4896(66.9)	
No degree	3379(33.3)	952(33.7)	2427(33.1)	
Smoking status, n (%)				<0.001
Never	4642(45.6)	1202(42.9)	3440(47.2)	
Former	4546(44.8)	1239(44.2)	3307(45.4)	
Current	907(8.9)	364(12.9)	543(7.4)	
Physical activity level, n (%)				0.003
Low	3391(33.4)	1010(36.2)	2381(32.7)	
Moderate	4215(41.5)	1134(40.7)	3081(42.3)	
High	2470(24.3)	645(23.1)	1825(25.0)	
Vitamin use, n (%)	2978(29.4)	813(8.1)	2165(21.3)	<0.001
Mineral use, n (%)	4043(39.9)	1024(10.1)	3019(29.8)	<0.001
Coffee use, n (%)	7958(78.4)	2157(21.3)	5801(57.2)	0.002
Tea use, n (%)	8312(81.9)	2228(30.0)	6084(59.9)	<0.001
Healthy sleep pattern, n (%)	4477(44.1)	1154(11.4)	3323(32.8)	<0.001
Energy, kcal	2090.9(738.8)	2016.5(804.2)	2078.1(711.2)	<0.001
Family history of CVD, n (%)	6233(61.4)	1682(16.6)	4551(44.9)	0.02
Family history of cancer, n (%)	3410(33.6)	922(9.1)	2488(24.5)	0.03
Mean AHEI (SD)	32.3(8.1)	31.9(8.6)	32.4(7.9)	0.01
Hypertension, n (%)	5873(57.9)	1730(17.1)	4143(40.8)	<0.001
Hypercholesterolemia, n (%)	232(2.3)	69(0.6)	163(1.6)	0.57
Aspirin use, n (%)	4357(42.9)	1190(11.7)	3167(31.2)	0.33

BMI: body mass index; CVD: cardiovascular disease; AHEI: alternative healthy eating index.

Table 2. Dose-response associations of specific types of breakfast cereals with CVD incidence, CVD mortality and all-cause mortality.

		CVD incidence			CVD mortality			All-cause mortality
		Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1
Breakfast cereal, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-1.5	0.89(0.82-0.96)	0.99(0.91-1.07)	0.99(0.91-1.08)	0.68(0.56-0.82)	0.75(0.62-0.92)	0.75(0.62-0.92)	0.79(0.71-0.87)
	≥ 1.5	0.77(0.70-0.85)	0.85(0.76-0.94)	0.86(0.77-0.95)	0.58(0.45-0.75)	0.65(0.51-0.85)	0.66(0.51-0.85)	0.75(0.66-0.86)
Porridge, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-1	0.97(0.78-1.20)	0.96(0.77-1.19)	0.88(0.77-1.11)	0.81(0.46-1.44)	0.86(0.47-1.56)	0.81(0.43-1.52)	0.86(0.64-1.15)
	≥ 1	0.84(0.77-0.92)	0.88(0.80-0.96)	0.90(0.82-0.98)	0.71(0.57-0.90)	0.77(0.60-0.98)	0.77(0.60-0.99)	0.77(0.68-0.86)
Muesli, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-1	0.86(0.74-0.99)	0.89(0.76-1.05)	0.89(0.76-1.05)	0.66(0.43-0.99)	0.66(0.41-1.05)	0.68(0.42-1.07)	0.68(0.55-0.84)
	≥ 1	0.78(0.68-0.89)	0.83(0.73-0.95)	0.88(0.77-0.99)	0.59(0.41-0.86)	0.66(0.45-0.95)	0.70(0.45-0.97)	0.80(0.67-0.95)
Plain cereal, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-1	0.97(0.81-1.16)	0.97(0.80-1.18)	0.97(0.79-1.19)	0.83(0.46-1.47)	0.79(0.46-1.38)	0.82(0.46-1.47)	1.03(0.73-1.35)
	≥ 1	1.01(0.90-1.13)	0.95(0.89-1.13)	1.02(0.90-1.15)	1.05(0.77-1.13)	1.03(0.76-1.40)	1.05(0.77-1.43)	1.08(0.95-1.32)
Bran cereal, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-1	0.87(0.74-1.03)	0.94(0.79-1.11)	0.93(0.78-1.10)	0.73(0.47-1.13)	0.70(0.43-1.14)	0.70(0.43-1.14)	0.87(0.70-0.97)
	≥ 1	0.89(0.78-1.02)	0.91(0.79-1.04)	0.91(0.79-1.04)	0.67(0.45-0.99)	0.73(0.49-1.08)	0.72(0.59-1.08)	0.84(0.70-0.98)
Whole wheat cereal, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-1	1.08(0.92-1.28)	1.06(0.98-1.13)	1.08(0.92-1.28)	0.97(0.63-1.52)	0.90(0.58-1.40)	0.97(0.63-1.52)	0.86(0.67-1.02)
	≥ 1	0.99(0.90-1.09)	1.02(0.93-1.11)	0.93(0.90-1.09)	0.87(0.78-1.03)	0.88(0.85-1.05)	0.84(0.68-1.13)	0.98(0.96-1.22)
Oat crunch, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-0.5	0.73(0.52-1.03)	0.82(0.58-1.16)	0.84(0.59-1.18)	0.42(0.14-1.31)	0.50(0.16-1.56)	0.52(0.18-1.63)	0.80(0.52-1.23)
	>0.5	1.01(0.78-1.28)	0.98(0.75-1.27)	1.02(0.76-1.34)	0.79(0.39-1.59)	0.81(0.39-1.72)	0.92(0.44-1.95)	0.91(0.64-1.27)
Sweetened cereal, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-0.5	1.09(0.76-1.53)	1.23(0.68-1.46)	1.34(0.66-1.39)	2.06(1.06-3.09)	1.88(0.93-3.80)	1.18(0.49-2.87)	1.52(1.02-2.28)
	>0.5	1.34(1.13-1.61)	1.33(1.11-1.62)	1.36(1.11-1.64)	1.56(1.01-2.41)	1.50(0.93-2.41)	1.15(0.66-2.00)	1.39(1.09-1.77)
Other cereal, bowls/d	0	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)	1.00(Reference)
	>0-0.5	1.10(0.83-1.44)	0.92(0.88-1.57)	1.09(0.80-1.47)	0.82(0.39-1.72)	1.06(0.50-2.25)	0.97(0.63-1.52)	0.73(0.48-1.11)
	>0.5	0.93(0.77-1.13)	0.98(0.80-1.20)	0.98(0.80-1.21)	0.75(0.44-1.27)	0.89(0.51-1.55)	0.88(0.68-1.33)	1.02(0.80-1.03)

Model 1: adjusted for age (continuous), sex (male, female), ethnicity (White or other), household income (>100000, 52 000–100 000, 31 000–51 999, 18 000–30 999, or <18 000£), education level (degree or no degree). Model 2 further adjusted for BMI (continuous), smoking status (current, former or never), physical activity level (low, moderate, or high), the Townsend deprivation index, vitamin use (yes or no), mineral and other dietary supplement use (yes or no), healthy sleep pattern (yes or no), family history of CVD (yes or no), family history of cancer (yes or no), coffee and tea intake, energy intake (continuous) and a modified AHEI score. Model 3 further adjusted for hypertension (yes or no), hypercholesterolemia (yes or no), and aspirin use (yes or no).

Table 3. Associations of breakfast cereals with risk of CVD incidence, CVD and all-cause mortality stratified by added sugar, artificial sweetener, milk, and dried fruit.

Outcome	None-consumer	Breakfast cereal consumer	
		Add	Not add
All-cause mortality			
Sugar	1(reference)	0.86(0.77-0.97)	0.78(0.64-0.95)
Artificial sweetener	1(reference)	0.98(0.75-1.27)	0.84(0.75-0.95)
Milk	1(reference)	0.87(0.76-0.98)	0.76(0.55-0.86)
Dried fruit	1(reference)	0.78(0.68-0.91)	0.89(0.77-0.96)
CVD-cause mortality			
Sugar	1(reference)	0.71(0.57-0.87)	0.63(0.44-0.91)
Artificial sweetener	1(reference)	0.91(0.57-0.98)	0.70(0.57-0.87)
Milk	1(reference)	0.72(0.34-0.81)	0.53(0.58-0.88)
Dried fruit	1(reference)	0.58(0.45-0.76)	0.76(0.61-0.95)
CVD incidence			
Sugar	1(reference)	0.95(0.80-1.06)	0.93(0.88-1.04)
Artificial sweetener	1(reference)	0.96(0.85-1.09)	0.95(0.87-1.03)
Milk	1(reference)	0.96(0.88-1.04)	0.90(0.77-1.04)
Dried fruit	1(reference)	0.89(0.81-0.99)	0.99(0.90-1.08)

Adjusted for age (continuous) and sex (male, female), ethnicity (white or other), household income (>100000, 52 000–100 000,31 000–51 999, 18 000–30 999, or <18 000£), education level (degree or no degree), BMI (continuous), smoking status (current, former, or never), physical activity level (low, moderate, or high), the Townsend deprivation index, vitamin use (yes or no), mineral and other dietary supplement use (yes or no), healthy sleep pattern (yes or no), family history of CVD (yes or no), family history of cancer (yes or no), coffee and tea intake, energy intake (continuous) and a modified AHEI score, hypertension (yes or no), hypercholesterolemia (yes or no), and aspirin use (yes or no).

Figures

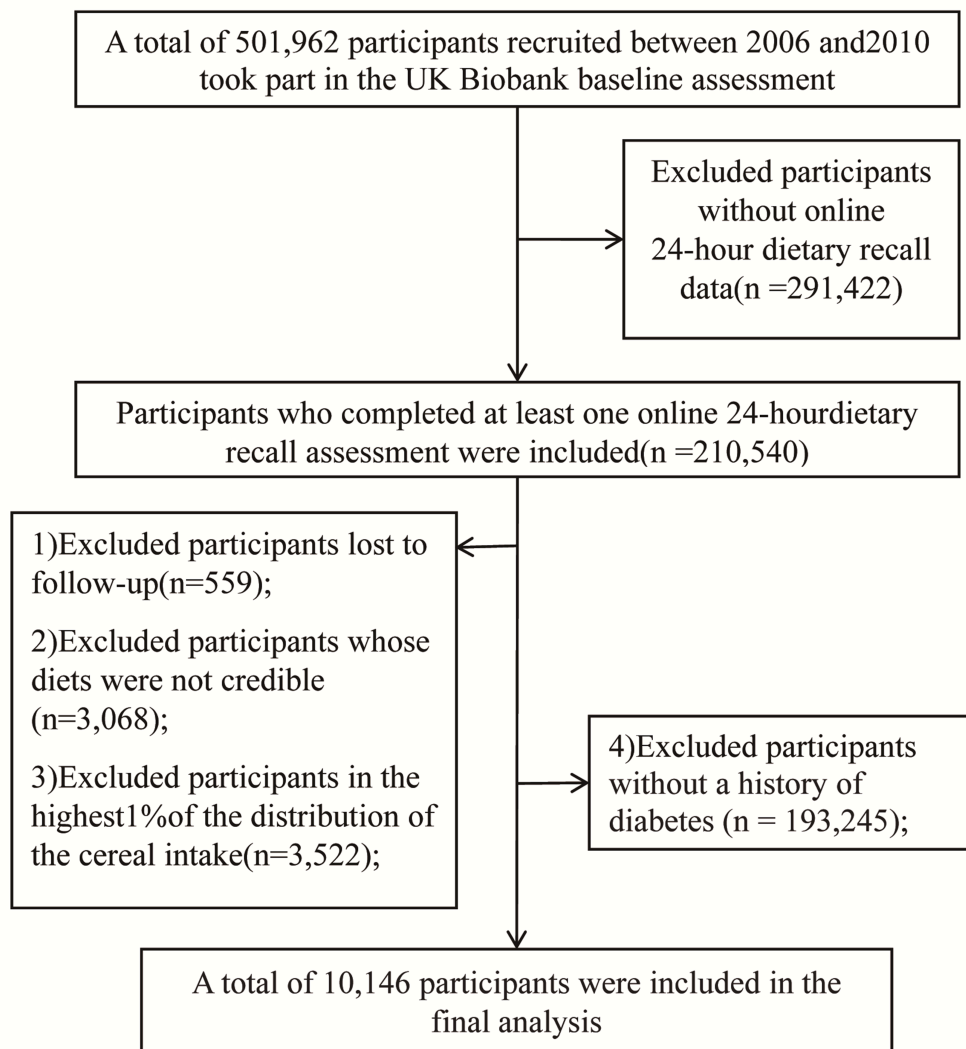


Figure 1

Flowchart of study design and participant enrollment.

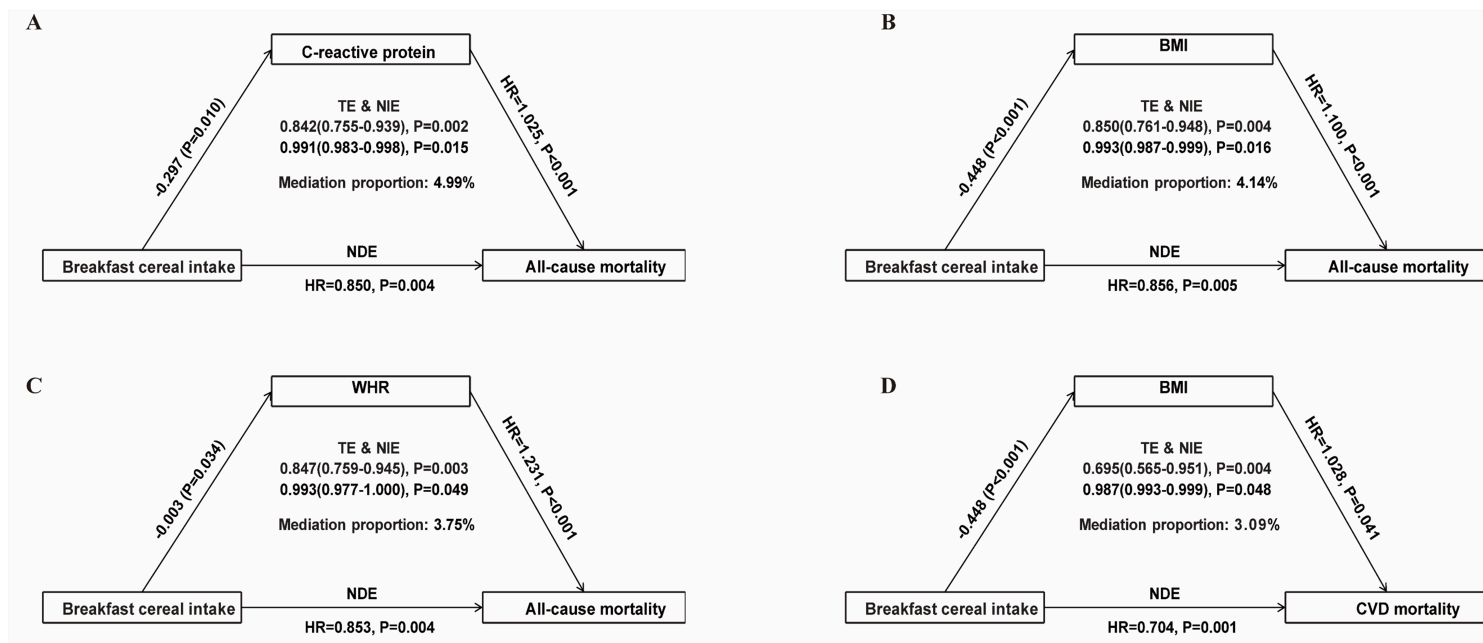


Figure 2

Mediation analysis of breakfast cereal intake and mortality.

Adjusted for age (continuous), sex (male, female), ethnicity (White or other), household income (>100000, 52 000–100 000, 31 000–51 999, 18 000–30 999, or <18 000£), education level (degree or no degree), BMI (continuous), smoking status (current, former, or never), physical activity level (low, moderate, or high), the Townsend deprivation index, vitamin use (yes or no), mineral and other dietary supplement use (yes or no), healthy sleep pattern (yes or no), family history of CVD (yes or no), family history of cancer (yes or no), coffee and tea intake, energy intake(continuous) and a modified AHEI score, hypertension (yes or no), hypercholesterolemia (yes or no), and aspirin use (yes or no).

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