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Meat Preference–Intake Discordance and Incident Cardiometabolic Disease in UK Biobank: A Prospective Cohort Study

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Abstract

Background

Dietary intake and food preference are examined separately in nutritional epidemiology. Whether meat preference-intake discordance is associated with cardiometabolic disease (CMD) risk remains unclear.

Methods

We included 168,163 UK Biobank participants without prevalent CMD. Meat preference was assessed using the Food Preference Questionnaire, and habitual intake from repeated food-frequency assessments. Standardised preference minus standardised intake defined preference-intake discordance. Cox models, stratified analyses, generalised random forests, and counterfactual mediation analyses assessed incident CMD, heterogeneity, and pathways.

Results

During follow-up, 39,428 incident CMD cases occurred. Overall meat preference > intake was not significantly associated with CMD after full adjustment (HR 1.027, 95% CI 0.994–1.061). Preference > intake for beef, lamb, and pork was associated with higher CMD risk (HRs 1.045, 1.041, and 1.052), especially for hypertension and type 2 diabetes. Overall discordance was associated with CMD among low meat consumers (HR 1.078, 95% CI 1.022–1.136). HbA1c was a consistent positive mediator; liver- and bilirubin-related markers showed negative indirect effects suggestive of statistical suppression.

Conclusions

Meat preference-intake discordance was not uniformly associated with CMD risk; relevance varied by meat subtype and intake context. Preference-aware dietary assessment may refine individualised CMD risk evaluation.

Background

Cardiometabolic diseases (CMD), including cardiovascular diseases and type 2 diabetes, remain leading causes of global morbidity, disability, and premature mortality. The Global Burden of Disease (GBD) 2023 study estimated 626 million prevalent cases and 19.2 million deaths from cardiovascular diseases worldwide, while the GBD 2021 study estimated 529 million people living with diabetes, with prevalence projected to rise substantially by 2050 [1, 2]. Much of this burden is attributable to modifiable metabolic and behavioural risks, including elevated blood pressure, dysglycaemia, excess adiposity, dyslipidaemia, and poor diet quality [1]. Because diet influences adiposity, glycaemic regulation, blood pressure, lipid

metabolism, inflammation, and vascular function, food-based prevention remains central to reducing CMD risk [3].

Meat consumption is a major dietary exposure of interest, but its cardiometabolic relevance differs by meat subtype and dietary context. The most consistent evidence implicates processed meat, which is associated with higher risks of cardiovascular disease and type 2 diabetes in large prospective cohorts and meta-analyses [4, 5, 6]. Unprocessed red meat is generally associated with higher cardiometabolic risk, although these associations tend to be weaker and more heterogeneous, whereas evidence for poultry or white meat remains less consistent [4, 5, 6]. Biological evidence provides plausible pathways linking meat intake to CMD, while also underscoring the importance of meat type and context. Red meat supplies L-carnitine and other substrates for gut microbial production of trimethylamine N-oxide, and controlled feeding studies have shown that red meat can increase circulating and urinary trimethylamine N-oxide [7, 8]. Haem iron, processed-meat additives, sodium, nitrites, and high-temperature cooking products may further contribute to oxidative stress, endothelial dysfunction, dysglycaemia, inflammation, and adverse lipid profiles [9, 10]. Trial evidence also suggests that cardiometabolic responses to red meat depend strongly on the comparison diet, particularly when red meat is replaced by high-quality plant proteins [11]. Thus, meat-related CMD risk is unlikely to be captured by intake quantity alone.

Beyond actual intake, food preference may represent an upstream behavioural phenotype relevant to cardiometabolic health. Food preference, often operationalised as food liking, captures hedonic valuation, reward-based inclination, and learned affective responses toward foods before consumption occurs. It is shaped by sensory properties, repeated exposure, associative learning, post-ingestive feedback, social meaning, cultural norms, and genetic predisposition [12, 13, 14]. Preference measures correlate with food-frequency data, but they are not interchangeable with intake and may provide complementary information on dietary motivation and vulnerability [12, 13]. Large-scale biobank studies further support the biological and behavioural relevance of food liking: UK Biobank analyses have identified a hierarchical genetic architecture of food liking linked to neurophysiological traits [15], while twin data suggest that food-liking patterns, including meat-related liking, are partly heritable [16]. More recent UK Biobank studies have also linked food-preference profiles and specific food-liking traits with cardiometabolic outcomes, inflammatory and metabolic biomarkers, and shared genetic architecture [17].

The discordance between food preference and actual intake may therefore carry substantive information beyond methodological measurement differences. Preference reflects internal valuation, whereas intake reflects realised behaviour under real-world constraints. High meat preference with low intake may indicate intentional restriction, health concerns, affordability barriers, limited access, ethical considerations, or strong self-regulation [18, 19]. Conversely, meat intake exceeding preference may reflect convenience, household norms, social eating, habitual automaticity, or limited alternatives [19]. Behavioural evidence shows that dietary intentions often fail to translate into eating behaviour, particularly under craving, stress, social cues, or easy availability of goal-incongruent foods [20]. This issue may be especially relevant to meat consumption, where reduction intentions often coexist with

persistent intake driven by habits, favourable attitudes toward meat, and psychological barriers to dietary change [21]. At the same time, stronger meat preference may partly reflect nutrient-state-related appetitive signalling in addition to preference itself, as experimental evidence indicates that protein dilution or deficit can elicit compensatory increases in intake and enhance preference for savoury high-protein foods [22]. Yet nutritional epidemiology has largely examined meat intake, meat preference, or meat-reduction intentions separately. Whether meat preference–intake discordance is prospectively associated with incident CMD remains unknown.

To address this gap, we used UK Biobank data to quantify discordance between meat preference and habitual meat intake and examine its association with incident CMD [23, 24]. We hypothesised that meat preference–intake discordance would capture cardiometabolic risk beyond preference or intake alone, and that opposite directions of discordance would reflect distinct behavioural phenotypes with different cardiometabolic implications.

Methods

Study design and population

This study was conducted using data from the UK Biobank (UKB), a large population-based prospective cohort established to investigate associations between a wide range of environmental, lifestyle, genetic, and biological factors and health outcomes. Between 2006 and 2010, approximately 500,000 participants aged 40–69 years were recruited from 22 assessment centres across the United Kingdom. At baseline, participants completed touchscreen questionnaires, underwent physical examinations, and provided biological samples.

Participants who completed at least one Food Frequency Questionnaire (FFQ) and the Food Preference Questionnaire (FPQ) were eligible. We excluded individuals with > 25% “prefer not to answer” responses in the FPQ, those lost to follow-up, and those with prevalent CMD at baseline, resulting in a final analytic sample of 168,163 participants. The inclusion and exclusion process is illustrated in Fig. 1.

Assessment of meat preference and intake

Food preferences were assessed using the FPQ, developed by the University of Edinburgh in collaboration with international researchers to investigate genetic and environmental determinants of food liking. Unlike traditional dietary assessment tools that measure consumption frequency or quantity, the FPQ captures hedonic liking, thereby reducing recall bias and social desirability bias. The FPQ has demonstrated high test–retest reliability (intraclass correlation coefficients 0.8–0.9) and temporal stability in adult populations. Participants rated 150 food items on a 9-point scale ranging from 1 (“dislike extremely”) to 9 (“like extremely”), with intermediate values representing graded preferences. Items were presented in random order to minimise sequence effects, covering major food groups and sensory dimensions.

Ten FPQ items related to meat preference were extracted, including beef, pork, lamb, poultry, ham, salami, sausage, bacon, hamburger patties, and fried chicken. To preserve ordinal information while improving interpretability, responses were recoded into a six-level ordered variable based on the underlying structure of the 9-point hedonic scale. Intermediate values (2–4 and 6–8) were collapsed into adjacent categories. Responses indicating “never tried” were coded as 0 to reflect a pre-exposure state along a try–preference continuum, whereas “prefer not to answer” responses (− 818) were treated as missing. The detailed recoding scheme is presented in Supplementary Table 1. Following the NOVA food classification system, six items (ham, salami, sausage, bacon, hamburger patties, and fried chicken) were classified as processed meats. A composite processed meat preference score was calculated as the arithmetic mean of the recoded values across these items, allowing for missing values under the assumption of missing at random, which resulted in 421 missing observations. To maintain consistency with single-item measures and enhance interpretability, the composite score was further categorised into a six-level ordered variable using left-open, right-closed intervals aligned with the original hedonic scale structure (Supplementary Table 2). All meat preference variables, including both single-item and composite measures, were standardised using Z-score transformation to facilitate comparability across exposure variables.

Actual meat intake was assessed using the UK Biobank FFQ (Data Coding 100377), which records consumption frequency using an integer-based coding system. Five meat categories were examined: processed meat, poultry, beef, lamb, and pork. When multiple assessment instances were available, the mean value across repeated measurements was calculated to better capture habitual intake over time. According to UK Biobank coding guidelines, responses coded as “−1” (never consumed) were treated as valid zero intake, whereas “−3” (prefer not to answer) responses were treated as missing. All meat intake variables were standardised using Z-score transformation to ensure comparability with preference measures.

To quantify discordance between meat preference and intake, a standardised discordance score (D) was calculated by subtracting the Z-score of intake (I) from the Z-score of preference (P) (D = P - I). Positive values indicate unmet preference (preference > intake), whereas negative values indicate relative overconsumption (preference < intake). For interpretability, the continuous discordance score was further categorised into three ordered groups based on the empirical distribution (± 1 SD around the mean): preference lower than intake (preference < intake), preference concordant with intake (preference = intake), and preference higher than intake (preference > intake). This data-driven classification avoids arbitrary thresholds while preserving behavioural interpretability and statistical robustness.

Ascertainment of cardiometabolic diseases

The primary outcome was incident CMD, defined as the first occurrence of any of the following conditions during follow-up: angina pectoris (Angina), acute myocardial infarction (AMI), cerebral infarction (CI), chronic ischaemic heart disease (CIHD), atrial fibrillation or flutter (AFF), heart failure (HF), essential hypertension, intracerebral haemorrhage (IH), or type 2 diabetes mellitus (T2DM).

Outcomes were ascertained through linkage to hospital inpatient records using the International Classification of Diseases, 10th Revision (ICD-10). For participants with multiple events, only the earliest diagnosis was considered, and the date of first occurrence was used to define time-to-event.

Covariate and candidate mediator assessment

Covariates were selected a priori based on biological plausibility and prior literature. Sociodemographic variables included age, sex, and ethnicity (White, Mixed, Asian, Black, Chinese, and Other; “prefer not to answer” and “do not know” were retained as separate categories). Body mass index (BMI) was calculated as the mean of repeated baseline measurements. Socioeconomic status was assessed using educational attainment and the Townsend deprivation index (categorised into quartiles). Lifestyle factors included physical activity (low, moderate, or high, based on the International Physical Activity Questionnaire), smoking status (current, former, never), and alcohol consumption (current, former, never). Dietary covariates included total energy intake, carbohydrate intake, and overall diet quality assessed using the Alternative Healthy Eating Index (AHEI), calculated from repeated dietary assessments as the sum of component scores based on population deciles [25], which was additionally examined in sensitivity analyses.

Additional biomarkers and behavioural measures were extracted as candidate mediators for counterfactual mediation analyses. Chronic glycaemic burden was assessed using glycated haemoglobin (HbA1c). Liver enzyme and bilirubin-related markers included gamma-glutamyl transferase (GGT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin, and direct bilirubin. Systemic inflammatory and immune markers included C-reactive protein (CRP), white blood cell count, and rheumatoid factor. Protein and nutritional-status markers included albumin and total protein, and renal-metabolic status was represented by Cystatin C. Psychobehavioural factors included neuroticism score and sleep duration. NMR-derived metabolic markers included apolipoprotein B, remnant cholesterol, LDL particle concentration, small LDL particle concentration, total triglycerides, VLDL triglycerides, saturated fatty acids, polyunsaturated fatty acids, degree of unsaturation, docosahexaenoic acid (DHA), total branched-chain amino acids, valine, and tyrosine. For biomarkers or behavioural measures with repeated UK Biobank assessment instances, non-missing values from the same field were averaged across available instances; neuroticism was taken from the baseline touchscreen assessment. Candidate mediators were analysed in mediator-specific complete-case datasets and were not included as covariates in the primary Cox models unless otherwise specified.

Statistical analysis

Baseline characteristics were summarised according to categories of meat preference–intake discordance (preference lower than intake, concordant, and preference higher than intake). Continuous variables are presented as means ± standard deviations (SDs), and categorical variables as counts and percentages. Between-group differences were assessed using the Kruskal–Wallis test for continuous variables and chi-square tests for categorical variables.

Missing values in baseline covariates were handled using multiple imputation by chained equations (MICE). Five imputed datasets (m = 5) were generated with 100 iterations using the mice package in R. Imputation models were specified according to variable type, including predictive mean matching for continuous variables, logistic regression for binary variables, and polytomous regression for categorical variables. The imputation model included all exposure variables, prespecified covariates, and outcome indicators with corresponding survival times to preserve the underlying data structure. Outcome variables were included as predictors but were not imputed. Estimates from the imputed datasets were combined using Rubin's rules.

Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the associations of meat preference, meat intake, and preference–intake discordance with incident CMD. Analyses were performed for overall CMD and individual disease subtypes. Person-years of follow-up were calculated from the date of recruitment to the date of first CMD diagnosis, death, or the administrative end of follow-up (whichever occurred first). The proportional hazards assumption was evaluated using Schoenfeld residuals. A prespecified, stepwise adjustment strategy was applied using a series of Cox models. Model 1 included the exposure only. Model 2 was additionally adjusted for sociodemographic and lifestyle factors, including age, sex, ethnicity (White, Mixed, Asian, Black, Chinese, and Other; "prefer not to answer" and "do not know" retained as separate categories), body mass index (BMI, continuous), socioeconomic status (Townsend deprivation index, quartiles), educational attainment, physical activity (low, moderate, high), smoking status (current, former, never), and alcohol consumption (current, former, never). Model 3 was further adjusted for dietary factors, including total energy intake (continuous) and carbohydrate intake (continuous). The fully adjusted model (Model 3) was considered the primary model for inference.

Stratified analyses were conducted to assess whether meat intake level modified the association between preference–intake discordance and incident CMD. Participants were categorised into tertiles of standardised meat intake amount (low, medium, and high). Within each stratum, associations were estimated using Cox proportional hazards models based on model 3 described above. Effect modification was further evaluated by including multiplicative interaction terms between preference–intake discordance and meat intake categories. Statistical significance of interactions was assessed using likelihood ratio tests.

To move beyond average treatment effects and explore individualised patterns of association between preference–intake discordance and incident CMD, we applied a machine learning framework based on generalised random forests (GRF) to estimate conditional average treatment effects (CATEs). Preference–intake discordance was operationalised as a binary exposure comparing individuals with preference exceeding intake versus those with concordant preference and intake. Models were adjusted for prespecified covariates, including age at recruitment, sex, body mass index (BMI), total energy intake, and carbohydrate intake. Continuous covariates were winsorised at the 1st and 99th percentiles. A two-stage procedure was implemented to accommodate the survival outcome. First, a survival forest with 2,000 trees was used to estimate outcome risk conditional on covariates. Second, a causal forest was

applied using the residualised outcome as the response variable and treatment assignment as the exposure. For interpretability, participants were categorised into tertiles of the CATE distribution. Variable importance was assessed using regression forests applied to the absolute CATE values, with stability evaluated through repeated subsampling.

Causal mediation analysis was conducted within a counterfactual framework to investigate potential biological and behavioural pathways linking preference–intake discordance with incident CMD. Potential mediators were prespecified across three domains: cardiometabolic and inflammatory biomarkers, neuropsychological traits, and metabolic profiles derived from nuclear magnetic resonance (NMR) spectroscopy. For each mediator, regression-based models were fitted to estimate the association between preference–intake discordance and the mediator, and between the exposure and mediator jointly with CMD risk. The total effect was decomposed into the average causal mediation effect (ACME) and the average direct effect (ADE). Skewed biomarkers were natural log-transformed prior to analysis. Uncertainty was assessed using non-parametric bootstrapping with 1500 resamples to derive 95% confidence intervals. Mediation models were repeated among participants with low baseline meat intake to evaluate whether observed pathways were independent of absolute intake levels. All models were adjusted for the prespecified covariates.

Several sensitivity analyses were conducted to evaluate the robustness of the findings. First, to address potential reverse causation, primary analyses were repeated after excluding participants who developed CMD within the first two years of follow-up. Second, competing risks of non-CMD mortality were accounted for using Fine–Gray sub-distribution hazard models. Third, to assess the impact of missing data, complete-case analyses were performed by restricting the sample to participants with complete information on all covariates. Results from these analyses were compared with those from the primary imputed dataset.

All analyses were conducted using R (v4.2.2). All tests were two-sided, with statistical significance set at $P < 0.05$.

Use of generative AI and AI-assisted technologies

During the preparation of this work, the authors used OpenAI ChatGPT/Codex to assist with language editing and journal-format compliance checking. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Results

Baseline characteristics of study participants

Table 1 summarises the baseline characteristics of the 168,163 participants, stratified by preference–intake discordance. Participants were categorised into three groups: preference < intake (n = 25,881),

concordant (n = 104,690), and preference > intake (n = 37,592). Baseline characteristics differed across groups for all variables examined (all $P \leq 0.003$).

Table 1
Baseline characteristics of participants by preference–intake discordance categories

Characteristics	Preference–Intake discordance			
	Preference < Intake (n = 25881)	Preference = Intake (n = 104690)	Preference > Intake (n = 37592)	P
Sex, n (%)				0.003
Female	14833 (57.3)	61171 (58.4)	21980 (58.5)	
Male	11048 (42.7)	43519 (41.6)	15612 (41.5)	
Age, mean (SD), years	55.2 (7.7)	55.4 (7.6)	55.7 (7.6)	< 0.001
BMI, mean (SD), kg/m^2	26.3 (4.4)	26.5 (4.4)	26.9 (4.5)	< 0.001
Ethnicity, n (%)				< 0.001
White	24454 (94.5)	101604 (97.1)	36733 (97.7)	
Asian or Asian British	644 (2.5)	1007 (0.9)	224 (0.6)	
Black or Black British	277 (1.1)	643 (0.6)	201 (0.5)	
Other	506 (1.9)	1436 (1.4)	434 (1.2)	
Socioeconomic Status, n (%)				< 0.001
High	4207 (16.3)	17947 (17.1)	7447 (19.8)	
Upper-Middle	6047 (23.4)	24574 (23.5)	9138 (24.3)	
Lower-Middle	6785 (26.2)	28108 (26.8)	9797 (26.1)	
Low	8204 (31.7)	31423 (30.0)	10281 (27.3)	
Missing	638 (2.5)	2638 (2.5)	929 (2.5)	
Education Status, n (%)				< 0.001
High	3708 (14.3)	16705 (16.0)	7527 (20.0)	
Upper-Middle	5585 (21.6)	23495 (22.4)	9055 (24.1)	
Lower-Middle	6997 (27.0)	28458 (27.2)	9768 (26.0)	
Low	8953 (34.6)	33394 (31.9)	10313 (27.4)	
Missing	638 (2.5)	2638 (2.5)	929 (2.5)	

Characteristics	Preference–Intake discordance			
	Preference < Intake (n = 25881)	Preference = Intake (n = 104690)	Preference > Intake (n = 37592)	P
Physical activity level, n (%)				< 0.001
Low	3656 (14.1)	15929 (15.2)	6133 (16.3)	
Moderate	9764 (37.7)	38367 (36.6)	12794 (34.0)	
High	8791 (34.0)	34658 (33.1)	12377 (32.9)	
Missing	3670 (14.2)	15736 (15.0)	6288 (16.7)	
Smoking status, n (%)				< 0.001
Never	15404 (59.5)	61968 (59.2)	21196 (56.4)	
Former	8642 (33.4)	35226 (33.6)	13301 (35.4)	
Current	1776 (6.9)	7253 (6.9)	3022 (8.0)	
Missing	59 (0.2)	343 (0.3)	73 (0.2)	
Alcohol drinking status, n (%)				< 0.001
Never	918 (3.5)	2705 (2.6)	983 (2.6)	
Former	669 (2.6)	2570 (2.5)	1024 (2.7)	
Current	24272 (93.8)	99312 (94.9)	35568 (94.6)	
Missing	21 (0.1)	103 (0.1)	17 (0.0)	
AHEI score, mean (SD)	60.9 (11.6)	61.1 (11.5)	60.2 (11.2)	< 0.001
Carbohydrate intake, mean (SD), g/day	256.8 (76.1)	253.7 (73.7)	250.0 (74.7)	< 0.001
Energy, mean (SD), kJ/day	8796.1 (2406.4)	8653 (2303.9)	8573 (2360.9)	< 0.001

Across the three groups, participants with preference > intake were older (mean age 55.7) and had a higher mean body mass index (BMI) (26.9 kg/m²). The proportion of females was highest in the preference > intake group (58.5%), and the majority of participants were of White ethnicity across all groups. Participants in the preference > intake group also had higher socioeconomic and educational attainment, with 19.8% classified as high socioeconomic status and 20.0% having higher education,

compared with 17.1% and 16.0% in the concordant group and 16.3% and 14.3% in the preference < intake group, respectively. In terms of lifestyle and dietary factors, the preference > intake group had the lowest proportion of high physical activity (32.9%) and the highest prevalence of current smoking (8.0%). Both discordant groups (preference < intake and preference > intake) had lower diet quality, as indicated by lower mean AHEI scores. The preference > intake group also had the lowest mean carbohydrate intake (250.0 g/day) and total energy intake (8573 kJ/day).

Associations of preference–intake discordance, actual intake, and preference with cardiometabolic diseases

Table 2 presents the HRs and 95% CIs for the associations between meat preference–intake discordance, actual meat intake, stated meat preference, and incident CMD. In analyses of discordance categories, using the concordant group (preference = intake) as the reference, participants with preference > intake exhibited a higher risk of CMD in the crude model and after adjustment for demographic, socioeconomic, and lifestyle factors. However, this association was attenuated and no longer statistically significant after further adjustment for dietary factors (Model 3: HR 1.027 (95% CI 0.994–1.061)). No significant association was observed for preference < intake across models.

Table 2
Association of CMD with preference–intake discordance, intake level, and preference level

Preference–Intake discordance	Model 1	Model 2	Model 3
Preference = Intake (n = 104690)	1 (ref)	1 (ref)	1 (ref)
Preference < Intake (n = 25881)	1.019 (0.991, 1.048)	1.023 (0.992, 1.056)	1.022 (0.984, 1.062)
Preference > Intake (n = 37592)	1.112 (1.086, 1.139)	1.035 (1.008, 1.063)	1.027 (0.994, 1.061)
Intake Level			
Low(n = 56028)	1 (ref)	1 (ref)	1 (ref)
Medium(n = 56028)	1.165 (1.136, 1.194)	1.034 (1.006, 1.063)	1.040 (1.005, 1.075)
High(n = 56028)	1.312 (1.281, 1.345)	1.077 (1.048, 1.108)	1.082 (1.045, 1.119)
Preference Level			
Low(n = 55923)	1 (ref)	1 (ref)	1 (ref)
Medium(n = 55922)	1.208 (1.178, 1.239)	1.028 (0.999, 1.057)	1.033 (0.999, 1.069)
High(n = 55922)	1.358 (1.325, 1.391)	1.078 (1.049, 1.109)	1.088 (1.052, 1.126)

Note: Model 1 was a crude model. Model 2 was adjusted for baseline age (continuous), sex (female or male), and ethnicity (White, Mixed, Asian, Black, Chinese, Other, with “prefer not to answer” or “do not know”), BMI (continuous), socioeconomic status (high, upper-middle, lower-middle, low), education status (high, upper-middle, lower-middle, low), physical activity (low, moderate and high level), smoking status (current, former, or never), alcohol drinking status (current, former, or never). Model 3 was further adjusted for energy intake (continuous), carbohydrate intake (continuous) based on Model 2.

When examined independently, both meat intake and preference showed more robust associations with CMD risk. In the fully adjusted model, higher meat intake was associated with increased risk in a dose–response manner (medium vs. low: HR 1.040 (95% CI 1.005–1.075); high vs. low: HR 1.082 (95% CI 1.045–1.119)). Similarly, high meat preference was associated with an elevated risk of CMD, HR 1.088 (95% CI 1.052–1.126), whereas no significant association was observed for medium preference.

Associations between preference–intake discordance and specific cardiometabolic outcomes varied substantially across meat types and disease subtypes (Fig. 2). For preference > intake, no significant association was observed between total meat discordance and overall CMD risk. However, positive associations were consistently identified for red meat subtypes. Specifically, a preference exceeding

intake for beef, HR 1.045 (95% CI 1.005–1.086), lamb, HR 1.041 (95% CI 1.008–1.076), and pork, HR 1.052 (95% CI 1.011–1.094) was associated with increased risk of CMD. When examining individual outcomes, this pattern was primarily driven by higher risks of essential hypertension for beef, HR 1.054 (95% CI 1.009–1.101) and pork, HR 1.054 (95% CI 1.008–1.102), and a higher risk of type 2 diabetes for lamb, HR 1.095 (95% CI 1.007–1.192). In contrast, inverse associations were observed for selected ischaemic outcomes, including AMI and CIHD, for total meat and beef (all HRs < 1.0; Fig. 2).

For preference < intake, associations were less pronounced. No significant associations were identified between any meat type and overall CMD risk. A higher risk of AFF was observed for total meat, HR 1.100 (95% CI 1.008–1.201), and a higher risk of AMI was associated with beef, HR 1.185 (95% CI 1.000–1.403). Conversely, lamb intake was associated with a reduced risk of angina, HR 0.854 (95% CI 0.761–0.957). No consistent associations were observed for poultry or processed meat across cardiometabolic subtypes (all P > 0.05; Fig. 2).

Stratified analyses

We further examined whether the association between meat preference–intake discordance and CMD risk varied across levels of actual meat intake (low, medium, and high; Fig. 3). For individuals with preference > intake, the association with CMD risk differed substantially by baseline intake level. While no significant association was observed for total meat in the overall analysis, stratified analyses revealed a significant increase in CMD risk among participants with low meat intake, HR 1.078 (95% CI 1.022–1.136), with no corresponding association in the medium or high intake groups. This pattern was consistently observed for several meat subtypes, including lamb, HR 1.073 (95% CI 1.014–1.136), pork, HR 1.069 (95% CI 1.001–1.142), and poultry, HR 1.059 (95% CI 1.004–1.116), where preference exceeding intake was associated with a higher CMD risk only in the low intake group. In contrast, for beef, a significant positive association was observed only among individuals with high intake, HR 1.089 (95% CI 1.003–1.183), with no evidence of association in the low or medium intake groups.

Analyses of specific cardiometabolic outcomes further revealed outcome-specific heterogeneity (Supplementary Fig. 1). For type 2 diabetes, preference > intake for total meat was associated with increased risk in the high intake group, HR 1.215 (95% CI 1.026–1.438), whereas lamb, HR 1.278 (95% CI 1.098–1.488) and pork, HR 1.193 (95% CI 1.002–1.421) showed significant associations only in the low intake group. For essential hypertension, increased risks were primarily observed in the low intake group for total meat, HR 1.098 (95% CI 1.034–1.165), lamb, HR 1.083 (95% CI 1.015–1.154), and pork, HR 1.092 (95% CI 1.014–1.177), whereas beef showed a significant association only in the medium intake group, HR 1.096 (95% CI 1.023–1.175).

For individuals with preference < intake, associations were generally weaker and less consistent across intake strata. A significant inverse association was observed only for processed meat in the low intake group, where intake exceeding preference was associated with a reduced risk of CMD, HR 0.907 (95% CI 0.841–0.979). No consistent associations were identified across other intake levels.

Data-driven heterogeneity and effect modifiers

To further characterise heterogeneity in the association between meat preference–intake discordance and CMD risk, we applied causal forest models to estimate individual-level treatment effects (Fig. 4). Variable importance analysis indicated that anthropometric and dietary factors were the primary drivers of effect heterogeneity. In particular, body mass index (BMI) emerged as the most influential contributor, followed by total energy intake and carbohydrate intake, whereas demographic factors, including age and sex, contributed comparatively less to the observed variation (Fig. 4A). Consistent with these findings, stability analyses based on bootstrap resampling showed that energy intake, BMI, and carbohydrate intake were the most frequently selected predictors among the top contributors (each appearing in the top three in > 30% of subsamples), whereas age was selected less frequently (< 10%), and sex did not appear among the top-ranking variables (Fig. 4B).

The distribution of conditional average treatment effects (CATEs) further revealed substantial inter-individual variability in the association between preference–intake discordance and CMD risk (Fig. 4C). Positive CATE values—indicating a stronger adverse association of discordance, particularly for the preference > intake pattern—were observed across wide ranges of BMI and dietary intake. Nonlinear patterns were evident across continuous covariates. For BMI, the estimated effect increased at lower levels (< 25 kg/m²), reached a local minimum around 28 kg/m², and then rose progressively at higher levels. For energy intake, the estimated effect was highest at the lowest intake levels (~ 4,000 kJ/day), declined progressively to a nadir near 7,000 kJ/day, and rose again to a secondary peak around 12,000 kJ/day. Carbohydrate intake exhibited a similar U-shaped profile, with the highest estimated effects near 100 g/day, a nadir around 200 g/day, and a secondary peak near 400 g/day. In contrast, differences by sex were minimal. Overall, these findings demonstrate pronounced and nonlinearly structured heterogeneity in the association between meat preference–intake discordance and CMD risk, primarily driven by BMI and dietary intake rather than demographic characteristics.

Potential biological pathways: causal mediation analysis

To elucidate potential biological and behavioural pathways underlying the observed associations, we applied a counterfactual-based mediation framework to quantify indirect effects of candidate mediators spanning glycaemic, hepatic, bilirubin-related, inflammatory, renal-metabolic, psychobehavioural, and nutritional-metabolomic domains. Analyses compared individuals in the preference > intake group with those in the concordant preference = intake group, with confidence intervals derived from 1,500 bootstrap simulations (Fig. 5, Supplementary Table 3A, B).

In the overall population, HbA1c showed a significant positive indirect effect. Preference > intake was associated with higher HbA1c levels, and higher HbA1c was associated with greater CMD risk in the outcome model, resulting in a positive ACME of 0.578×10-³ (95% CI [0.057×10 − 3, 1.230×10 − 3], P = 0.03). In contrast, several hepatic and bilirubin-related markers exhibited significant negative indirect effects. GGT showed the largest negative ACME among these markers (ACME − 0.564×10-³, 95% CI [-0.931×10 − 3, -0.133×10 − 3], P < 0.001), followed by AST (ACME − 0.308×10 − 3, 95% CI [-0.510×10 − 3, -0.072×10 − 3], P < 0.001), DBIL (ACME − 0.282×10 − 3, 95% CI [-0.457×10 − 3, -0.086×10 − 3], P < 0.001), and TBIL (ACME − 0.131×10 − 3, 95% CI [-0.261×10 − 3, -0.036×10 − 3], P < 0.001). Among supplementary

protein-related markers, albumin also showed a significant negative indirect effect (ACME − 0.201×10 − 3, 95% CI [-0.338×10 − 3, -0.044×10 − 3], P < 0.001).

In the low meat-intake subgroup, the mediation pattern became more specific. HbA1c remained a significant positive mediator and showed a larger indirect effect than in the overall population (ACME 0.875×10 − 3, 95% CI [0.124×10 − 3, 1.820×10 − 3], P = 0.03), corresponding to approximately 7.72% of the total adverse association. AST continued to show a significant negative indirect effect (ACME − 0.384×10 − 3, 95% CI [-0.685×10 − 3, -0.130×10 − 3], P < 0.001), again reflecting a suppression pattern. By contrast, the negative indirect effects of GGT, TBIL, and DBIL observed in the overall population were no longer statistically significant in the low-intake subgroup. Cystatin C also did not show a significant indirect effect in this stratum. WBC demonstrated a borderline positive indirect effect (ACME 0.340×10 − 3, 95% CI [0.019×10 − 3, 0.791×10 − 3], P = 0.05), suggesting a possible but exploratory inflammatory signal.

Psychobehavioural factors, including neuroticism and sleep duration, CRP, and other lipidomic, fatty acid, and amino acid markers did not demonstrate significant mediation in either the overall cohort or the low-intake subgroup.

Sensitivity analysis

A series of sensitivity analyses supported the robustness of the primary findings. The associations between meat preference–intake discordance and CMD risk remained materially unchanged after excluding CMD events occurring within the first two years of follow-up, accounting for competing non-CMD mortality using Fine–Gray sub-distribution hazard models, and restricting analyses to participants with complete covariate data. Results were also consistent after additional adjustment for overall dietary quality using the Alternative Healthy Eating Index, modified dietary quality scores excluding meat components, major non-meat food groups, and alternative dietary composition variables. Collectively, these analyses yielded effect estimates comparable in direction and magnitude to the primary models, indicating that the observed associations were robust to reverse causation, competing risk, missing data handling, and alternative dietary adjustment specifications (Supplementary Fig. 2).

Discussion

In this large prospective cohort study, we propose meat preference–intake discordance as a behaviourally informed dietary exposure and evaluate its association with CMD risk. At the aggregate level, overall meat preference–intake discordance was not significantly associated with CMD risk. This null finding, however, obscured a pattern of subtype- and context-specific associations. For red meat specifically, greater preference than intake was independently associated with elevated CMD risk. Stratified analyses further revealed that this association was confined to individuals with lower habitual meat intake and was not apparent among those with higher consumption. These findings suggest that the degree of alignment between consumption and underlying preference may provide additional

information about cardiometabolic vulnerability, supporting its potential incorporation into personalised and precision nutrition frameworks [26].

While prior epidemiological investigations have consistently linked both higher meat intake and stronger meat preference to elevated cardiometabolic risk [17, 27], our primary analyses observed no significant aggregate association between overall preference–intake discordance and CMD risk. Two complementary explanations may account for this null finding. First, preference–intake discordance may not operate independently of the background diet in which it occurs. The marked attenuation of the association after adjustment for total energy and carbohydrate intake suggests that discordance is partly embedded within broader dietary patterns, including overall energy balance and macronutrient composition. Second, the effects of preference–intake discordance are likely context-contingent, with heterogeneous subgroup-level effects attenuating toward the null in aggregate analyses. Behavioural evidence supports this: intention–behaviour gaps vary systematically with the nature of the target food and the direction of restraint—implementation intentions more effectively close the gap for increasing desired food consumption than for suppressing preferred intake [28], and self-regulatory capacity differentially moderates discordance across food categories [29]. Thus, pooling all forms of discordance may obscure subgroup-specific associations.

Consistent with this interpretation, our subsequent analyses showed that the association between discordance and CMD risk was not uniform, but was concentrated in specific disease subtypes and intake amount strata, particularly among individuals with lower meat intake. The subtype-specific association observed for red meat, whereby preference exceeding intake was associated with higher risks of CMD, particularly hypertension and type 2 diabetes, may reflect the distinctive biological and behavioural salience of red meat. From a cardiometabolic perspective, red meat has been more consistently linked to type 2 diabetes and cardiovascular outcomes than several other meat subtypes, with proposed mechanisms involving haem iron–related oxidative stress, saturated fat, and gut microbiome-derived metabolites such as trimethylamine N-oxide (TMAO) [4, 30]. These pathways may render red meat-related dietary patterns more metabolically consequential than meat consumption considered in aggregate. At the behavioural level, red meat consumption operates on a potent hedonic and reward-based axis, introducing a parallel psychological and physiological strain. Red meat consumption is deeply rooted in both biological reward mechanisms and socio-cultural practices. Its sensory properties, particularly flavour, tenderness, and juiciness, are consistently identified as key drivers of consumer preference. Cultural traditions and habitual household dietary practices further contribute to its continued consumption. As a result, red meat intake often remains high despite awareness of potential adverse health consequences [31]. This behavioural inertia aligns closely with the clinical paradigm of "meat attachment," wherein profound hedonic enjoyment and a perceived sense of dependence systematically erode an individual's willingness or self-regulatory capacity to transition toward lower-meat dietary patterns [32]. In this context, high red meat preference combined with lower realised intake may indicate unresolved appetitive motivation, with low intake not necessarily reflecting a uniformly health-conscious dietary pattern. Food reward models distinguish hedonic “liking” from incentive “wanting,” suggesting that motivational drive toward a desired food can persist despite

intentional or external constraint [33]. Consistently, selective deprivation of liked foods, particularly among restrained eaters, has been linked to greater craving and compensatory intake, while dietary restraint has been associated with heightened reward-related neural responses to palatable food cues [34, 35]. Moreover, the cardiometabolic consequences of reducing red meat intake may depend on the quality of dietary substitutions [36]. Thus, red meat preference > intake may identify individuals in whom constrained intake coexists with persistent appetitive drive and potentially less favourable compensatory dietary patterns. This discordance may be diluted when total meat is considered, because aggregate meat intake combines subtypes with heterogeneous nutritional profiles, sensory attributes, and social meanings. The concurrent inverse associations with ischaemic outcomes may reflect a protective effect of lower realised red meat consumption. Specifically, reduced risks of AMI and CIHD were observed when preference exceeded intake for beef and total meat. This pattern is consistent with evidence showing that reducing red meat intake and replacing it with plant-based protein sources can lower ischaemic cardiovascular risk [4, 36].

The observation that significant associations in stratified analyses were confined to individuals with lower meat intake is consistent with an intake-dependent model of dietary discordance. At higher consumption levels, the cardiometabolic burden attributable to intake itself likely predominates, limiting the marginal detectability of preference–intake discordance, consistent with prospective evidence linking higher red and processed meat consumption with elevated cardiovascular and metabolic risk [6, 27]. Among lower consumers, this direct metabolic burden is attenuated, allowing discordance to emerge as a more salient risk correlate. The co-occurrence of low intake and high preference may reflect sustained dietary restraint or unresolved appetitive drive: experimental evidence indicates that protein deficit activates compensatory mechanisms that increase intake and preference for high-protein foods [22], and when such restraint is chronic or unsuccessful, these responses may become maladaptive. Restrictive dieting has been associated with greater food cravings and binge-eating symptoms [37], while loss-of-control or binge-type eating has been linked to adverse cardiometabolic profiles including adiposity, dyslipidaemia, hypertension, and impaired glycaemic markers [38]. Collectively, these findings suggest that preference–intake discordance may be most cardiometabolically relevant under conditions of sustained dietary constraint. In this context, low realised intake signals a vulnerable state of compensatory dysregulation rather than low appetitive motivation—a pattern whose dependence on intake context is further supported by the structure of individual-level heterogeneity identified in the causal forest analyses.

Causal forest analyses revealed that this heterogeneity was structured primarily by metabolic and dietary rather than demographic characteristics: BMI, total energy intake, and carbohydrate intake consistently emerged as dominant contributors, while age and sex explained comparatively little variation.The non-linear CATE profiles are particularly informative in light of the restraint-based vulnerability model described above. For BMI, the adverse conditional effect was strongest at lower values (< 25 kg/m²), where intentional dietary restriction to maintain body weight may coexist with persistent appetitive drive. The effect reached a local minimum near 28 kg/m² and increased progressively at higher values, where adiposity-related metabolic vulnerability may represent a distinct contributing mechanism. Because BMI

does not distinguish fat mass from lean mass, these estimates should be interpreted as BMI-related rather than adiposity-specific. Moderation by carbohydrate intake further supported the substitution hypothesis. Adverse effects were stronger at higher carbohydrate intake levels, suggesting that restriction of preferred foods may be accompanied by increased consumption of less favourable carbohydrate-rich alternatives. This interpretation is consistent with the HbA1c-mediated pathway identified in the mediation analyses. The observed heterogeneity is also consistent with precision nutrition frameworks, which propose that dietary effects are modified by individual metabolic and anthropometric characteristics [26, 39, 40]. Together, these findings support preference–intake discordance as a behaviourally informed risk signal, although they should be regarded as hypothesis-generating until externally validated.

The mediation analyses revealed a context-dependent pathway structure with no single dominant biological mechanism. In the overall population, HbA1c showed a significant positive indirect effect: preference > intake was associated with higher chronic glycaemic burden, which was strongly linked to greater CMD risk. This glycaemic pathway became more pronounced among low-intake individuals, where HbA1c accounted for a larger absolute indirect effect. The dietary composition of this subgroup (Supplementary Table 4A) provides a mechanistic account of both findings: consistent with the carbohydrate-related heterogeneity identified in the causal forest analyses, low-intake individuals with preference > intake had the lowest intakes of vegetables, fruits, nuts, whole grains, n-3 fatty acids, and polyunsaturated fat, and the highest intakes of sodium, trans fat, alcohol, added sugars, and free sugars. This profile supports the compensatory-substitution interpretation. Constrained consumption of a highly preferred food may be accompanied by increased intake of nutritionally inferior, carbohydrate-rich alternatives rather than equivalent substitutes. Such a pattern could plausibly link preference–intake discordance to HbA1c-mediated glycaemic dysregulation, given the established associations of poor carbohydrate quality and low plant-food intake with type 2 diabetes risk [41, 42]. In contrast, GGT, AST, TBIL, and DBIL exhibited negative indirect effects. These findings are more consistent with statistical suppression, a well-recognised form of inconsistent mediation, than with biological mediation. Specifically, preference > intake was associated with lower marker levels, whereas higher marker levels predicted greater CMD risk. This pattern does not support a hepatoprotective interpretation [43, 44]. Among participants with low intake, suppression effects for GGT, TBIL, and DBIL were no longer significant. AST remained suppressive, while WBC showed a borderline positive indirect effect [45]. Overall, preference–intake discordance appears to be most consistently associated with CMD risk through an HbA1c-mediated glycaemic pathway, particularly when low realised intake is accompanied by unfavourable dietary substitution. This pathway may represent a measurable biological manifestation of the compensatory dysregulation proposed in the stratified analyses. By contrast, liver- and bilirubin-related biomarkers primarily appear to reflect suppressive relationships within a correlated metabolic biomarker network.

This study has several strengths. First, by operationalising preference–intake discordance as the primary exposure, we extend conventional intake-centric approaches in nutritional epidemiology to incorporate a behaviourally meaningful dimension of diet. Food liking and preference are recognised contributors to

food choice and consumption, yet they are rarely examined jointly with realised intake as a marker of behavioural concordance or constraint [46]. Second, the large prospective cohort design, repeated dietary assessments, and ascertainment of incident CMD outcomes provided substantial statistical power and helped reduce, although not eliminate, the possibility of reverse causation. Third, the use of complementary analytical strategies, including multivariable regression, stratified analyses, mediation models, and generalised random forests, allowed us to evaluate the association from multiple perspectives. This analytic triangulation strengthens the credibility of the observed pattern, while recognising that these approaches remain embedded within a single observational cohort and therefore do not substitute for evidence from independent designs or randomised interventions [47].

Several limitations should also be considered. Dietary intake and preference were self-reported and are therefore susceptible to recall error, social desirability bias, and misclassification, limitations that are well recognised in food-frequency and other self-report dietary instruments [48]. In addition, habitual intake was derived from repeated assessments, whereas preference was captured at a single time point. Although some adult food preference patterns show moderate temporal stability, preferences can also shift with ageing, health status, food environment, and intentional dietary change, introducing potential measurement discordance [46]. Residual confounding remains possible despite extensive adjustment, particularly from unmeasured factors such as intentional weight control, food insecurity, psychosocial stress, cultural dietary norms, and local food availability. The mediation and causal forest analyses should therefore be interpreted as exploratory and hypothesis-generating, and should not be taken as definitive evidence of causal mechanisms. Finally, UK Biobank participants are healthier and less socioeconomically deprived than the general UK population, consistent with a healthy-volunteer selection process; this may limit transportability to more diverse or disadvantaged populations and could affect association estimates if selection is related to both dietary behaviour and CMD risk [49, 50].

Overall, this study integrates dietary preference and actual intake into a unified framework and identifies preference–intake discordance as a behaviourally informed dimension of dietary assessment. Although overall meat discordance was not associated with CMD risk at the aggregate level, subtype-specific and stratified analyses revealed more specific signals: red meat preference exceeding intake was associated with higher CMD risk, and preference–intake discordance was more relevant among individuals with lower habitual meat intake. These findings suggest that realised intake alone may not fully capture diet-related vulnerability, and that dietary recommendations may benefit from considering behavioural sustainability, motivational conflict, and long-term adherence. Future studies should validate preference–intake discordance in independent and more representative cohorts, incorporate repeated preference measures, objective dietary biomarkers, digital dietary records, and high-resolution metabolic profiling, and test whether preference-aware dietary strategies improve adherence and cardiometabolic outcomes. More broadly, the health consequences of diet may depend not only on dietary content, but also on whether realised consumption is behaviourally sustainable and concordant with underlying motivational drive.

Conclusions

This study introduces preference–intake discordance as a behaviourally informed dimension of dietary assessment and shows that its cardiometabolic relevance varies by meat subtype and intake context. Although no aggregate association was observed for overall meat discordance, subtype-specific and stratified analyses revealed more specific signals, particularly for red meat and among individuals with lower habitual meat intake. These findings suggest that realised intake alone may not fully capture diet-related vulnerability, and that future dietary risk assessment may benefit from considering whether consumption is behaviourally sustainable and aligned with underlying preference. Further studies are needed to validate this framework in diverse cohorts and to test whether preference-aware dietary strategies improve long-term adherence and cardiometabolic outcomes.

Abbreviations

ACME
average causal mediation effect
AFF
atrial fibrillation or flutter
AHEI
Alternative Healthy Eating Index
ALP
alkaline phosphatase
AMI
acute myocardial infarction
AST
aspartate aminotransferase
BMI
body mass index
CATE
conditional average treatment effect
CI
confidence interval (or cerebral infarction in outcome labels)
CIHD
chronic ischaemic heart disease
CMD
cardiometabolic disease
CRP
C-reactive protein
DBIL
direct bilirubin
FFQ
food-frequency questionnaire

FPQ
Food Preference Questionnaire
GBD
Global Burden of Disease
GGT
gamma-glutamyl transferase
GRF
generalised random forest
HbA1c
glycated haemoglobin
HF
heart failure
HR
hazard ratio
IH
intracerebral haemorrhage
MICE
multiple imputation by chained equations
NMR
nuclear magnetic resonance
SD
standard deviation
T2DM
type 2 diabetes mellitus
TBIL
total bilirubin
TMAO
trimethylamine N-oxide
UKB
UK Biobank
WBC
white blood cell count.

Declarations

Ethics approval and consent to participate

Ethical approval for the original UK Biobank study was granted by Northwest Multi-Centre Research Ethics Committee (11/NW/0382). Informed consent was obtained from each participant. Our study was conducted in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable.

Availability of data and materials

The individual-level UK Biobank data used in this study cannot be publicly deposited by the authors because access is governed by UK Biobank data-use terms. Bona fide researchers may apply for access through the UK Biobank Access Management System (https://www.ukbiobank.ac.uk/enable-your-research/apply-for-access). Derived analytic materials and code are available from the corresponding author upon reasonable request, subject to UK Biobank data-use terms.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

All authors conceived and supervised the study. QZ completed the data analyses and drafted the manuscript, with significant contributions from YT, YY, XS, SC, LZ, ZS and AZ. QZ, ZS and AZ had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. All authors made substantial contributions to the content of the paper. All authors read and approved the final manuscript.

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Authors' information

Not applicable.

Sex, gender, and diversity reporting

The analysis included sex, ethnicity, socioeconomic status, education, and lifestyle variables available in UK Biobank. Sex was included as a covariate, and ethnicity and socioeconomic variables were retained in adjusted models as described in the Methods. The interpretation recognises the healthy-volunteer profile of UK Biobank participants and the resulting limits to transportability.

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Figures

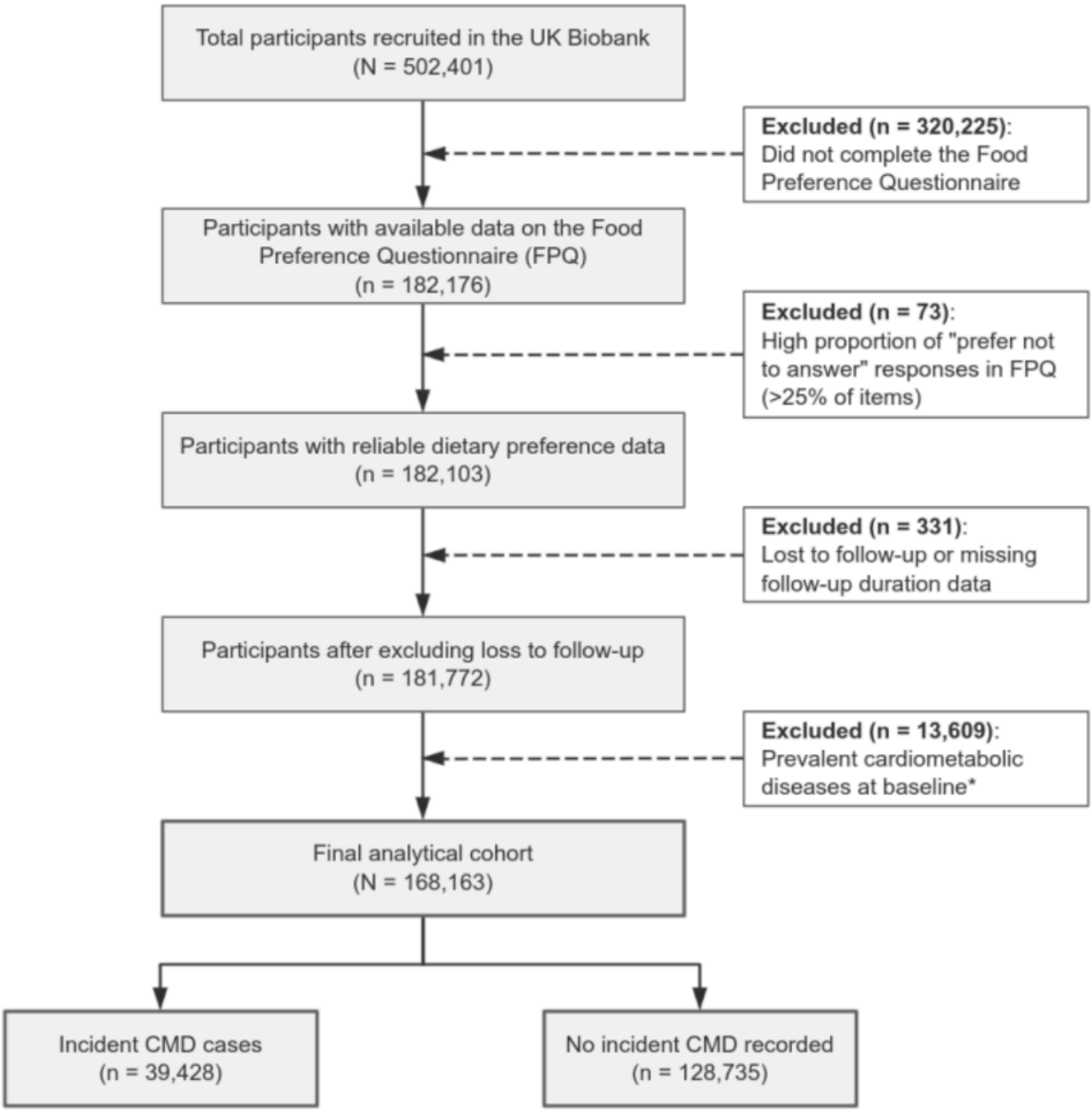


Figure 1

Flowchart of the study population selection process. CMD, cardiometabolic disease; FPQ, Food Preference Questionnaire; UK, United Kingdom. Prevalent cardiometabolic diseases at baseline were defined as a documented diagnosis of angina pectoris (Angina), acute myocardial infarction (AMI),

cerebral infarction (CI), chronic ischaemic heart disease (CIHD), atrial fibrillation or flutter (AFF), heart failure (HF), essential hypertension, intracerebral haemorrhage (IH), or type 2 diabetes mellitus (T2DM).

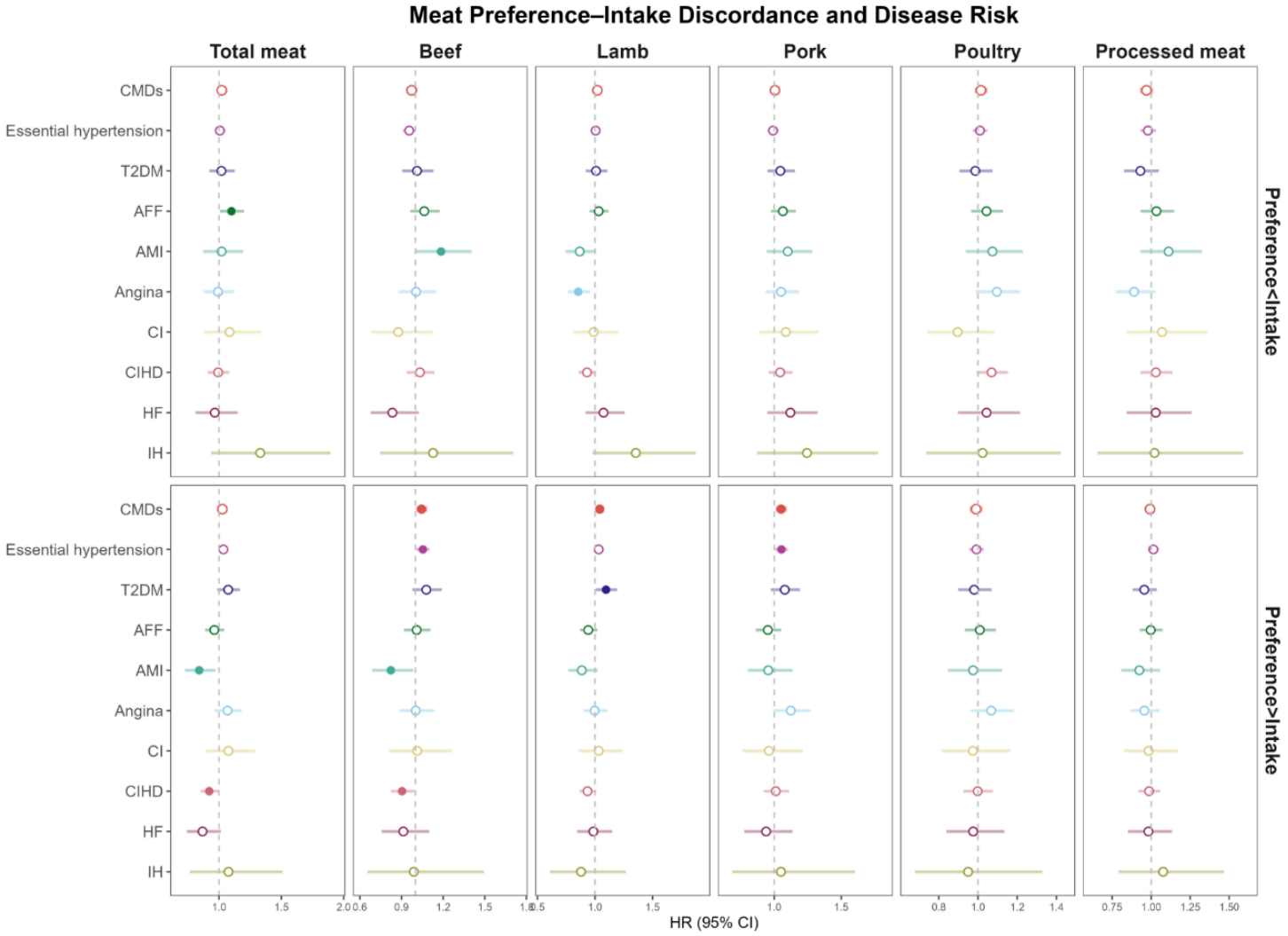


Figure 2

Associations of discordance between meat preference and intake with specific CMD outcomes. Hazard ratios (HRs) and 95% confidence intervals (CIs) for nine cardiometabolic disease outcomes were estimated using Cox proportional hazards models according to preference–intake discordance categories. The concordant group (preference = intake) was the reference. The model was adjusted for baseline age (continuous), sex (female or male), and ethnicity (White, Mixed, Asian, Black, Chinese, Other, with “prefer not to answer” or “do not know”), BMI (continuous), socioeconomic status (high, upper-middle, lower-middle, low), education status (high, upper-middle, lower-middle, low), physical activity (low, moderate and high level), smoking status (current, former, or never), alcohol drinking status (current, former, or never), energy intake (continuous) and carbohydrate intake (continuous).

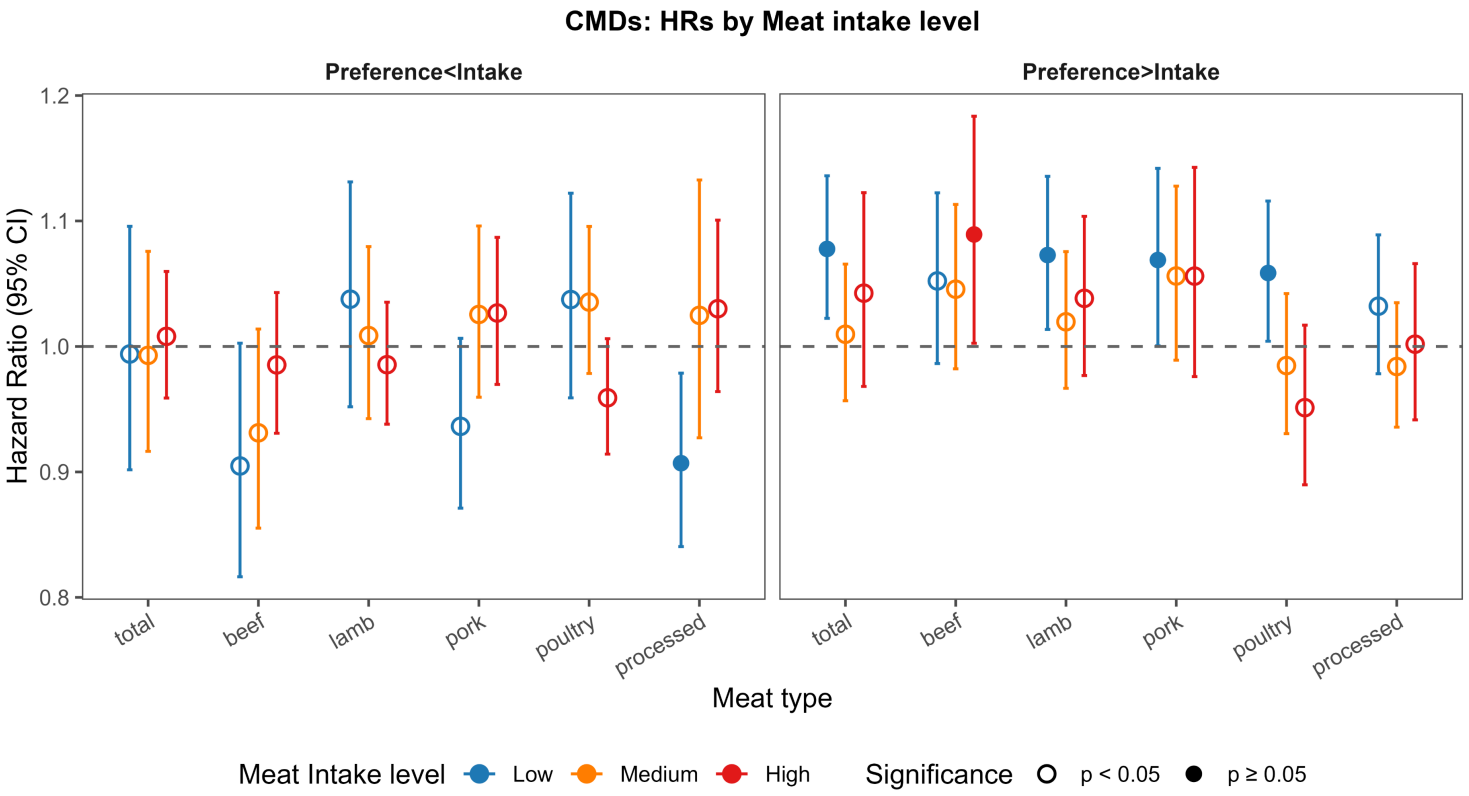


Figure 3

Stratified associations of preference–intake discordance with CMD. Associations between discordance of meat preference and actual intake and cardiometabolic disease risk were examined across subgroups defined by actual meat intake levels (low, medium, high). Hazard ratios (HRs) and 95% confidence intervals (CIs) were estimated using multivariable-adjusted Cox models. Models were adjusted for baseline age (continuous), sex (female or male), and ethnicity (White, Mixed, Asian, Black, Chinese, Other, with “prefer not to answer” or “do not know”), BMI (continuous), socioeconomic status (high, upper-middle, lower-middle, low), education status (high, upper-middle, lower-middle, low), physical activity (low, moderate and high level), smoking status (current, former, or never), alcohol drinking status (current, former, or never), energy intake (continuous) and carbohydrate intake (continuous).

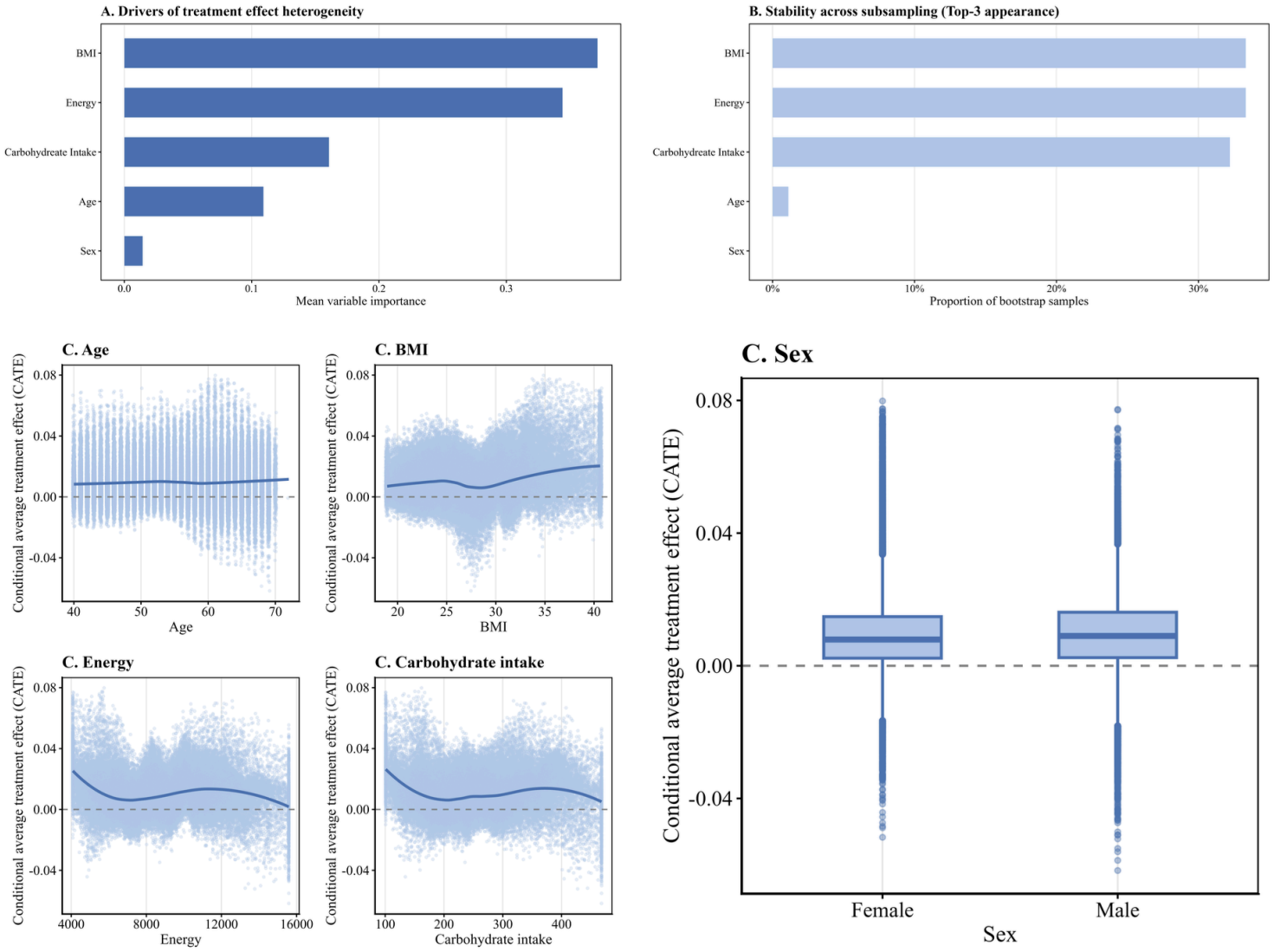


Figure 4

Heterogeneity in meat preference-intake discordance and cardiometabolic disease risk. (A) Mean variable importance of covariate groups (sex, age, BMI, energy intake and carbohydrate intake) in explaining heterogeneity of the treatment effect, estimated from causal forest models. (B) Stability of the top-ranked covariate groups across 30 bootstrap subsamples, expressed as the proportion of times each group appeared among the top three contributors. (C) Distribution of individual conditional average treatment effects (CATEs) across continuous (age, BMI, energy intake and carbohydrate intake) and categorical (sex) covariates. Scatter plots with locally weighted smoothing (LOESS) curves are shown for continuous variables, and box plots summarize the distribution for categorical variables. The horizontal dashed line indicates a null treatment effect (CATE = 0). Positive CATE values indicate a stronger adverse association of meat preference–intake discordance with cardiometabolic disease risk in the corresponding subgroup.

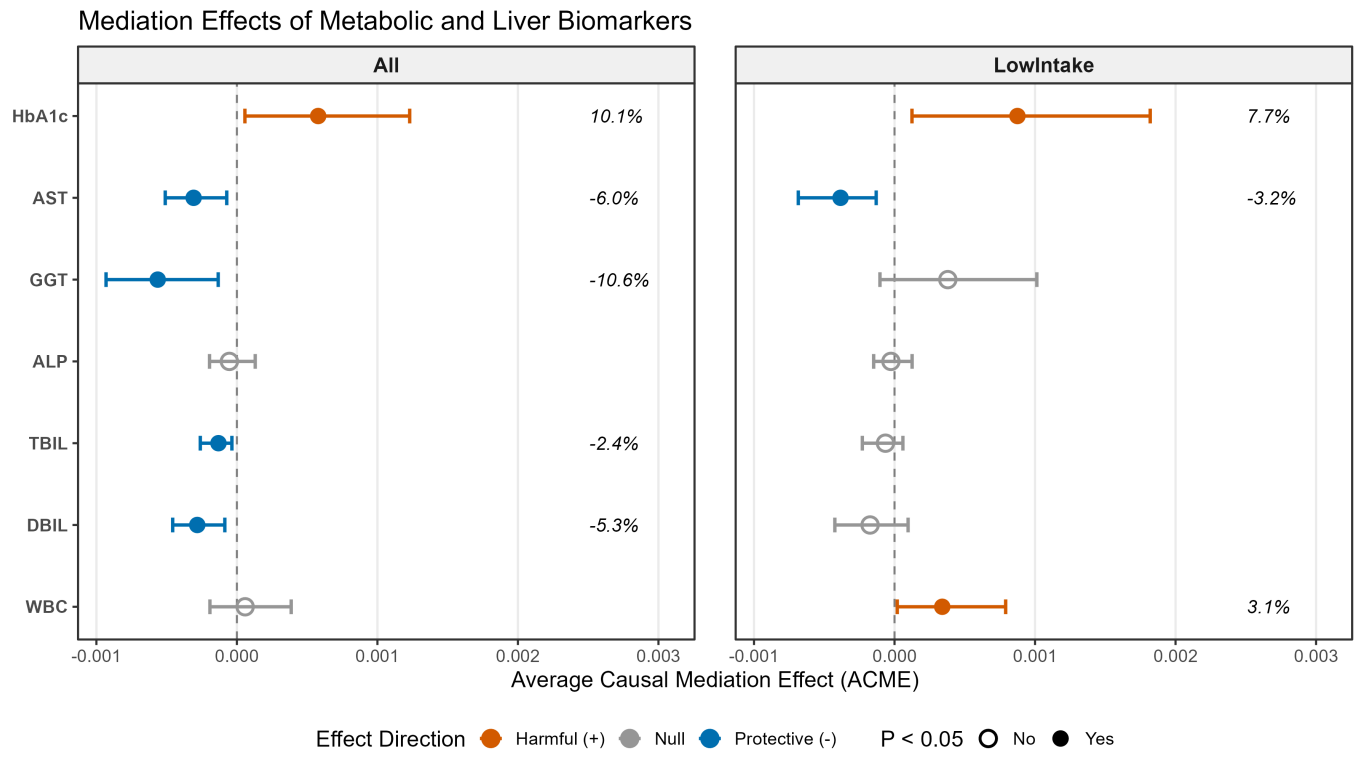


Figure 5

Mediation of the association between discordant meat preference and intake and cardiometabolic disease risk. Forest plots show average causal mediation effects (ACMEs) estimated using counterfactual-based mediation analysis comparing individuals reporting “Preference > Intake” with those reporting “Preference = Intake.” Models were adjusted for demographic, socioeconomic, lifestyle, and dietary covariates as described in the Methods. Confidence intervals were derived using 1,500 bootstrap simulations. Points indicate ACME estimates and horizontal lines represent 95% confidence intervals. Colours denote the direction of the indirect effect based on confidence intervals: red indicates positive mediation, blue indicates negative mediation or suppression, and grey indicates confidence intervals crossing zero. Filled circles indicate nominal statistical significance at $P < 0.05$. Percentages represent the estimated proportion mediated or suppressed and are shown only for statistically significant indirect effects; these estimates should be interpreted cautiously when the ACME and total effect have opposite signs or when mediator-specific total effects are imprecisely estimated. Identical x-axis scaling was applied across panels to facilitate comparison.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- tables.docx
- Supplementary.docx