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Wenbin Nan, Qiong Huang, Baimei He, Yinglan Liu & Zhenyu Peng

Adherence to the EAT-Lancet Diet and Risk of Sepsis: A Prospective Cohort Study from the UK Biobank

Wenbin Nan[1,2#], Qiong Huang [3,4#], Baimei He[5], Yinglan Liu[6], Zhenyu Peng[1,2 *]

[1] Department of Emergency Medicine, Second Xiangya Hospital, Central South University, Changsha 410011, China;

[2] Department of Emergency Medicine, Guilin Hospital of the Second Xiangya Hospital, Central South University, Guilin 541002, China;

[3] Department of Metabolism and Endocrinology, Second Xiangya Hospital, Central South University, Changsha 410011, China;

[4] National Clinical Research Center for Metabolic Diseases, Key Laboratory of Diabetes Immunology, Ministry of Education, and Department of Metabolism and Endocrinology, The Second Xiangya Hospital, Central South University, Changsha 410011, China;

[5] Department of Geriatric Respiratory and Critical Care Medicine, Xiangya Hospital, Central South University, Changsha 410008, China;

[6] Department of Endocrinology, The Second Affiliated Hospital of the University of South China, Hengyang, Hunan, 421001, China;

***Correspondence: Dr. Zhenyu Peng is the corresponding author**

 # These authors have contributed equally to this work and share the first authorship.

E-mail:

Wenbin Nan: nanwb2022@csu.edu.cn

Qiong Huang: huangqiong1998@csu.edu.cn

Baimei He: hebaimei@csu.edu.cn

Yinglan Liu: 2013020035@csu.edu.cn

Zhenyu Peng: pengzhenyu1999@csu.edu.cn

Abstract： Sepsis remains a significant global health challenge due to its high incidence, mortality, and disability. While adherence to the EAT–Lancet diet has been shown to reduce the risk of various inflammatory and metabolic diseases, its impact on sepsis is not well understood. This study aimed to investigate the association between adherence to the EAT–Lancet diet and sepsis risk, considering genetic susceptibility and potential proteomic mechanisms. Data from 199,085 participants in the UK Biobank were analyzed, with sepsis cases identified using ICD-10 codes from hospital records. Higher adherence to the EAT–Lancet diet was linked to a significantly reduced risk of sepsis (HR 0.85, 95% CI: 0.78–0.93; *P* for trend < 0.01). This protective effect was consistent across different genetic risk levels. Proteomic analysis revealed 43 plasma proteins that mediated this relationship, primarily involved in immune and inflammatory pathways. These findings suggest that greater adherence to the EAT–Lancet diet may lower sepsis risk, independent of genetic predisposition, through the modulation of immune-inflammatory proteins.

Key words： EAT-Lancet diet; Sepsis; Genetics; Proteomics; UK Biobank.

1 Introduction

Sepsis reflects life-endangering organ impairment caused by a dysregulated reaction of the host to infection(1). In 2017, it was estimated to affect 48.9 million individuals globally and account for nearly 11.0 million deaths(2). Survivors frequently suffer from long-term sequelae, such as recurrent infections, neurocognitive and functional deficits, and elevated cardiovascular risk(3). Given its substantial incidence, mortality, and disability rates, sepsis represents a major public health challenge worldwide(4). Significant progress has been made in understanding its pathogenesis, which encompasses immune dysregulation, systemic inflammation, endothelial injury, and ultimately multi-organ failure(5,6). Nevertheless, effective primary prevention remains limited. Thus, the identification of novel disease-modifying interventions is essential for reducing both the occurrence and long-term burden of sepsis.

Accumulating evidence underscores diet as a pivotal modifiable factor influencing both the risk and progression of sepsis. REGARDS data indicated that fruit- and vegetable-rich plant eating

corresponded to a 32% drop in sepsis risk(7). Additionally, high adherence to a traditional Mediterranean pattern centered on produce and pulses, with higher fish and grain consumption, demonstrated a 26% lower risk of sepsis (8). Nonetheless, the majority of dietary-pattern studies have centered exclusively on human health outcomes, largely overlooking planetary health implications. To address this, the EAT-Lancet Commission advocates a predominantly plant-based global reference diet to balance nutritional adequacy with environmental sustainability(9). Growing research indicates that following the EAT-Lancet dietary pattern has been linked to reduced risks of various inflammatory and metabolic diseases(10–12). However, its direct association with sepsis risk remains unexamined.

Genetic factors play a fundamental role in the pathogenesis of sepsis(13). This hereditary dimension is evidenced by clinical observations including familial case clustering and pronounced interindividual heterogeneity in susceptibility, severity, and outcomes(14). Genome-wide association studies (GWAS) have substantiated this genetic dimension by identifying a growing number of susceptibility loci, thereby elucidating the genetic architecture underlying sepsis risk and outcomes(15). As a multifactorial disease, sepsis emerges from complex interactions between inherited genetic predispositions and diverse environmental influences, such as infections, comorbidities, and lifestyle factors(16). Among these modifiable environmental determinants, dietary patterns stand out as key regulators to modulate sepsis susceptibility and clinical progression(17). However, the potential interplay between EAT-Lancet diet and genetic susceptibility in the context of sepsis risk remains entirely unexplored. Elucidating this environment-gene interaction is essential to advance our understanding of sepsis etiology and novel personalized preventive strategies.

Proteomics, as a high-throughput technology, allows comprehensive profiling of protein expression, function, and interactions(18,19). It has emerged as an essential tool for deciphering complex pathological mechanisms in diseases such as cancer, metabolic disorders, and infections(20,21). Recent plasma proteomic studies have uncovered numerous sepsis-related protein alterations and identified potential biomarkers for early diagnosis and therapeutic targeting(22,23). Additionally, proteomics informs understanding of diet–protein interaction pathways in sepsis pathogenesis, highlighting opportunities for dietary interventions in managing this critical illness(12). However, the influence of adherence to the EAT-Lancet diet on protein expression

profiles in sepsis remains largely unexplored.

To address these knowledge gaps, we designed a large prospective analysis within the UK Biobank cohort, which recruited participants through multiple assessment centers, to quantify the relationships between adherence to the EAT-Lancet diet score, genetic susceptibility, proteomic profiles, and incident sepsis. Our primary objective was to assess whether EAT-Lancet adherence showed an inverse relation to sepsis incidence. Subsequently, we evaluated potential interactions between dietary adherence and genetic predisposition in modifying sepsis risk. We also integrated plasma proteomics to identify protein signatures associated with EAT-Lancet diet compliance and sepsis risk, providing exploratory, hypothesis-generating clues that are broadly consistent with established immunoinflammatory processes in sepsis.

2 Results

2.1 Baseline characteristics

The primary analysis included 199,085 participants with median age 57 years (IQR 50-62) and 44.87% male (**Table 1**), and 5,026 incident sepsis cases were documented. The median EAT-Lancet diet index score was 11 (IQR 10–11). At baseline, individuals who developed sepsis tended to be older, more often male, smokers and non-current alcohol consumers, and had higher abnormal BMI, TDI, total energy intake, and prevalence of hypertension, diabetes, and ischemic heart disease, alongside lower physical activity levels. **Supplementary Table 3** summarized baseline characteristics by EAT-Lancet index categories, with additional comparisons between the groups regarding socioeconomic status, physical activity, smoking, alcohol consumption, and comorbidities.

2.2 Association between EAT-Lancet and sepsis risk

Table 2 summarized the inverse link between adherence to the EAT–Lancet diet and the likelihood of developing sepsis. After controlling for demographic and anthropometric factors (Model 1), individuals with the highest adherence had a notably lower risk compared with those with the lowest (HR 0.80; 95% CI 0.73–0.87; *P*-trend < 0.001). This relationship persisted after additional adjustment for socioeconomic and lifestyle variables (Model 2) and remained evident in the fully adjusted model (Model 3) (HR 0.81; 95% CI 0.74–0.89; *P*-trend<0.01). As shown in **Supplementary Table 4**, sepsis incidence decreased across increasing EAT–Lancet categories

(incidence rates per 1,000 person-years: 256.24 for ≤ 9, 238.59 for 10, 219.38 for 11, and 182.81 for ≥ 12). The median time to sepsis onset among cases was 6.28 years (IQR 4.12–8.47) overall, and was broadly similar across categories (range of medians: 5.96–6.51 years). RCS supported a linear dose–response (*P* overall<0.001; *P* nonlinearity=0.236), as illustrated in **Figure 1**.

2.3 Subgroup analysis and sensitivity analysis

Consistency was observed across all prespecified subgroups (**Supplementary Figure 2**). Interaction testing showed no significant modification by demographic, socioeconomic, behavioral, or clinical factors, with all *P*-interaction values exceeding 0.05. Consistent results were obtained in sensitivity analyses, as the inverse association remained following exclusion of participants with only one dietary recall or early-onset sepsis cases (**Supplementary Table 5 and Supplementary Table 6**). When accounting for death as a competing event using Fine–Gray models, the inverse association remained (per 1-point increment: adjusted sHR 0.94, 95% CI 0.92–0.97; $P < 0.001$; ≥ 12 vs ≤ 9: sHR 0.81, 95% CI 0.74–0.89; $P < 0.001$; *P* for trend < 0.001), indicating robustness to competing risk by mortality (**Supplementary Table 7**). Consistent results were obtained using alternative EAT-Lancet scores (**Supplementary Table 8**): each one-unit rise in the Stubbendorff score corresponded to roughly a 2 percent reduction in sepsis incidence (HR 0.98, 95% CI 0.98–0.99, $P < 0.001$), with significantly reduced risk at ≥ 24 points (*P*-trend < 0.001). A similar pattern was noted for the Kesse-Guyot score, where every 100-point increment was linked to about a 4 percent lower risk (HR 0.96, 95% CI 0.93–0.99, $P < 0.01$), and participants in the top quintile experienced the greatest risk reduction. Baseline characteristics were broadly comparable between included and excluded participants, although excluded participants were slightly older, less likely to be White, had higher BMI, were more socioeconomically deprived, more often current smokers, had higher cardiometabolic comorbidity, and showed higher sepsis incidence (**Supplementary Table 9**). In summary, higher dietary adherence was robustly and nearly linearly associated with reduced sepsis risk.

2.4 Joint effects of the EAT-Lancet diet index and genetic predisposition on sepsis risk

According to **Supplementary Table 10**, sepsis risk increased with higher polygenic scores. Individuals with moderate risk showed minimal change (HR 1.04, 95% CI 0.97–1.12; *P* 0.228), while those with high genetic susceptibility had a modest increase in risk (HR 1.10, 95% CI 1.02–1.17; *P* 0.010). The trend confirmed a dose-response link between genetic liability and sepsis

development (*P* for trend 0.010). However, the effect size of the PRS was small, and we caution that overfitting may have occurred due to PRS tuning in the target dataset, warranting caution in interpretation and the need for replication in independent datasets. **Supplementary Table 11** further showed that higher EAT-Lancet diet adherence was associated with lower sepsis risk across all genetic-risk groups, with significant reductions observed in the high-risk group (≥ 12 points: HR 0.82; *P* 0.010). The inverse associations appeared stronger in the middle and low genetic-risk groups (≥ 12 points: HR 0.81 and 0.77, respectively). As shown in **Table 3**, higher adherence to the EAT-Lancet diet consistently correlated with lower sepsis risk, particularly among those with lower genetic risk. However, no clear, significant interaction between diet and genetic risk was found. Comparing the extremes of the joint exposure, participants with low PRS and EAT-Lancet ≥ 12 had about 19% lower hazard than those with low PRS and EAT-Lancet ≤ 9 (HR ratio 0.81). These findings suggest that better adherence to the EAT-Lancet diet may reduce sepsis risk, particularly in individuals with lower genetic risk. However, further research is needed to clarify the potential role of diet in mitigating genetic risk for sepsis.

2.5 Proteomic signatures linking the EAT-Lancet diet and sepsis risk

Proteomic analysis identified significant plasma proteins associated with the EAT–Lancet diet index and sepsis. Among 2,911 proteins analyzed, 1,018 proteins were significantly associated with the diet index, with 276 proteins showing positive associations and 742 showing inverse associations (**Figure 2A and Supplementary Table 12**). In prospective analyses, 708 proteins were associated with sepsis risk, with 616 proteins showing positive associations and 92 showing inverse associations (**Figure 2B and Supplementary Table 13**). Following correction for multiple testing (FDR < 0.05 and Bonferroni < 0.05), 43 overlapping proteins were identified as potential mediators mediating the inverse association between diet adherence and sepsis risk (**Supplementary Table 14**). Significant mediators were determined based on the bootstrap 95% CI for indirect effects not crossing zero and P-values for indirect effects. Functional enrichment indicated that GO terms were enriched for positive regulation of leukocyte chemotaxis and migration, membrane microdomain and endoplasmic reticulum lumen, apical plasma membrane, extracellular matrix and external encapsulating structure, cell–cell junction, and for chemokine activity, chemokine receptor binding, cytokine receptor activity, and signaling receptor activator activity (**Figure 3A**). KEGG pathways in cytokine–cytokine receptor interaction, Toll-like receptor signaling, chemokine signaling,

apoptosis, influenza A, pathogenic E. coli infection, and lipid and atherosclerosis (**Figure 3B**). These results suggest that the EAT–Lancet diet adherence is tied to a distinct proteomic signature relevant to sepsis.

3 Discussion

Sepsis remains a critical global health challenge due to its high incidence, significant mortality, and substantial demand for intensive care(29). The EAT-Lancet diet, a "planetary health" dietary pattern aimed at improving population health while reducing environmental impacts, provides a promising preventive approach(30). However, its association with sepsis risk and potential biological mechanisms remains unclear. In this extensive cohort, stronger adherence to the EAT-Lancet dietary pattern was linked to a progressively lower likelihood of developing sepsis. This relationship remained robust across subgroup and sensitivity analyses. Adherence to the EAT-Lancet diet appeared to lessen the impact of inherited vulnerability on sepsis development, indicating a potential modification of genetic risk. Proteomic analyses further identified circulating protein signatures associated with both EAT-Lancet diet adherence and sepsis risk, providing hypothesis-generating clues that are broadly consistent with established immunoinflammatory biology in sepsis. Collectively, these results suggest that higher adherence to the EAT-Lancet diet is associated with a lower risk of sepsis.

Diet modulates key processes in sepsis pathogenesis, including inflammation, immune regulation, and infection susceptibility(31). The EAT-Lancet diet, which emphasizes plant-based and nutrient-rich foods, offers a sustainable framework for advancing human and planetary health(32). Accumulating evidence indicates that adherence to this dietary pattern is linked to a lower incidence of immune-related and infectious disorders, supporting its potential in sepsis prevention(33). For example, Wang et al. found that individuals with stronger adherence to the EAT-Lancet dietary pattern had a lower likelihood of developing rheumatoid arthritis, and metabolomic profiling suggested anti-inflammatory and immune-modulating mechanisms(30). Similarly, Kendrick et al. observed that individuals with higher compliance to the EAT-Lancet dietary pattern had fewer hospitalizations due to respiratory infections(34). In addition, closer alignment with the EAT-Lancet framework linked to lower Crohn's disease risk, potentially via beneficial modulation of gut microbiota and suppression of proinflammatory pathways. Collectively, these data provide consistent epidemiological and mechanistic support for the EAT-Lancet diet in fostering immune

balance and reducing infection-related outcomes. Our findings further extend prior evidence by showing that higher adherence to the EAT-Lancet dietary pattern was consistently associated with lower sepsis risk across progressively adjusted models. Spline analyses suggested an approximately linear dose–response pattern, supporting the robustness of the observed association. These results indicate that the EAT-Lancet diet may be relevant for identifying dietary patterns linked to lower sepsis risk.

Genetic background substantially influences an individual's vulnerability to developing sepsis(14). Genetic variants within key regulators of innate and adaptive immunity, particularly in genes encoding toll-like receptors and pro-inflammatory cytokines, have been robustly implicated in sepsis susceptibility and disease severity(35). Although GWAS has revealed multiple risk loci, PRS provides a more integrated assessment of cumulative genetic susceptibility(36). Growing evidence indicates that dietary patterns can modify genetic effects through regulation of inflammatory signaling, metabolic homeostasis, and host defense. For instance, individuals with strong compliance to the EAT-Lancet dietary pattern has been shown to attenuate colorectal cancer risk among genetically predisposed individuals(37). Similarly, healthy dietary patterns can mitigate the effects of genetic predisposition in those with cardiovascular disease and type 2 diabetes(10). In our analysis, individuals who followed the EAT-Lancet dietary pattern more closely showed a lower occurrence of sepsis across all genetic-risk groups, with the strongest benefit seen among those at high genetic susceptibility. However, the observed effect of diet in modifying sepsis risk was modest, and the interaction between diet and genetic susceptibility did not reach statistical significance. These results suggest a potential role for diet in modulating sepsis risk, particularly in genetically high-risk groups, but further investigation is needed to confirm whether diet can truly attenuate genetic risk, especially when assessed on both multiplicative and additive scales. In conclusion, while a nutrient-dense, plant-predominant diet may help reduce sepsis risk in high-risk groups, the current analysis does not provide conclusive evidence for a significant interaction between diet and genetic risk.

Proteomics enables the quantification of thousands of circulating proteins, offering a powerful tool to dissect the complex biological processes of sepsis(20). In clinical research, proteomics has been used to improve diagnostic accuracy. Gibot et al. demonstrated that combined measurement of sTREM-1, NGAL, and IL-6 effectively distinguished sepsis from non-infectious critical illness(38).

Beyond diagnosis, proteomics also enhances risk stratification. For instance, Ricci et al. incorporated endothelial and coagulation-related proteins such as angiopoietin-2 and complement components into mortality prediction models, significantly enhancing their prognostic performance(39). Mechanistically, proteomic profiling has highlighted key pathways driving organ injury in sepsis, including dysregulated complement activation, coagulation-inflammation cross-talk, neutrophil degranulation and NETosis, and activation of Toll-like receptor/NF-κB signaling(40). Furthermore, plant-based dietary patterns have been associated with beneficial proteomic shifts, including elevated anti-inflammatory proteins and reduced pro-inflammatory markers. Dryer-Beers et al. revealed that dietary components such as fiber and polyphenols induced favorable changes in the plasma proteins, suggesting possible mechanisms through which diet attenuated sepsis risk(41). However, whether adherence to the EAT-Lancet diet reshapes the plasma proteins relevant to sepsis remains unclear. In this analysis, key mediators and pathways—including immune cell migration, chemokine/cytokine receptor activity, and cytokine and Toll-like receptor signaling—were consistent with known mechanisms of dietary influence on sepsis risk. These findings suggest that adherence to the EAT–Lancet diet reduces sepsis risk potentially through modulation of immune-inflammatory plasma proteins.

So far as we know, this is the first analysis linking EAT-Lancet diet scoring to sepsis incidence. Strengths include the prospective design, large sample size, extensive covariate data, and integration of genetic and proteomic measures to explore potential pathways. Several limitations should be noted. First, diet was assessed using 24-hour recalls, which may not capture usual intake and could introduce random measurement error that attenuates associations toward the null, making estimates conservative. Second, residual confounding may persist despite extensive adjustment. Third, the predominantly European ancestry of participants may limit generalizability. Fourth, the PRS was used to stratify susceptibility within the cohort and to evaluate joint effects with diet rather than as a clinical prediction tool, and given the heterogeneity of sepsis we expected modest model fit and therefore report PRS quality control and model-fitting summaries in the Supplementary Materials. Fifth, PRS tuning within the target dataset raises the possibility of overfitting, so fit metrics should be interpreted cautiously. Sixth, the proteomic analysis was underpowered due to small sample size, limiting precision and generalizability and requiring replication. Seventh, data limitations, including reliance on a limited set of ICD-10 codes, may have led to sepsis misclassification and prevented

reliable subtype differentiation, and case definitions may not fully align with Sepsis-3 criteria. Finally, as an observational study, causality cannot be inferred.

In summary, our findings provide novel evidence that closer compliance with the EAT-Lancet dietary framework is related to decreased sepsis incidence in a large prospective population. This link remained stable across varying genetic-risk categories, suggesting that a EAT-Lancet diet may help reduce the impact of genetic risk. Proteomic analysis further identified key circulating proteins linked to both dietary adherence and sepsis risk, highlighting immune and inflammatory pathways such as cytokine signaling and leukocyte migration. These findings strengthen the mechanistic rationale for the beneficial effects of the EAT-Lancet diet and underscore its relevance as a feasible preventive measure at the population scale.

4 Methods

4.1 Study design and participants

UK Biobank is a nationwide longitudinal cohort initiated between 2006 and 2010, enrolling more than 500,000 adults aged 37–73 from multiple UK assessment centers(24). At baseline, participants completed a touchscreen questionnaire, underwent physical measurements, and provided biological samples, including blood and urine. The UK Biobank received ethical approval from the North West Multi-Centre Research Ethical Committee. All participants provided written informed consent. No specific ethical approval was required for this study due to use of publicly available data. This research has been conducted using the UK Biobank Resource under Application Number 617672.

From the initial 502,001 participants, we excluded those lacking at least one valid 24-h dietary recall (n=291,232), those with abnormal total energy intake (n=2,923), and individuals with pre-existing sepsis at baseline (n=1,341). We further excluded those with missing data on the following covariates: ethnicity (n=97), body mass index (BMI; n=558), education level (n=919), Townsend Deprivation Index (TDI; n=253), smoking status (n=430), alcohol drinking (n=97), and physical activity (n=5,066), resulting in 199,085 participants for primary analyses. For genetic predisposition analyses, we excluded individuals lacking polygenic risk score (PRS; n=3,757), yielding 195,328 participants. For proteomic analyses, we excluded individuals without proteomic data (n=181,779), leaving 17,306 participants. The study flowchart was shown in **Supplementary Figure 1**.

Ethical clearance for the UK Biobank was granted by the Community Health Index Advisory Group in Scotland, the North West Multi-Centre Research Ethics Committee, and the National Information Governance Board for Health and Social Care for England and Wales, with all procedures conducted in line with the Declaration of Helsinki. Written informed consent was obtained from each participant. The present study was reported according to the STROBE recommendations for observational research.

4.2 Dietary assessment and the EAT-Lancet diet index

Dietary intake was assessed using the UK Biobank Oxford WebQ, a web-based 24-hour dietary recall questionnaire administered at baseline and repeated in a subset of participants. Adherence was quantified using a 14-component EAT-Lancet dietary index based on Commission recommendations (**Supplementary Table 1**). Each component was scored as 1 if the criterion was met and 0 otherwise (**Supplementary Table 2**), yielding a total score of 0–14. For participants with more than one Oxford WebQ assessment, we calculated the EAT–Lancet score for each administration and used the mean of all available scores as the primary exposure to better approximate usual dietary intake and reduce random day-to-day variation; participants with a single recall contributed one score. Following prior work, scores were categorized as $\leqslant$ 9, 10, 11, and $\geqslant$ 12 points(25).

4.3 Definition of sepsis

Sepsis cases were ascertained via ICD diagnostic codes in line with previous literature (26). We extracted ICD-10 codes A02, A39, A40, and A41 from linked hospital inpatient records in England, thereby defining the outcome as incident hospitalized sepsis identified through hospital admission records, following enrollment in the UK Biobank.

4.4 Assessment of covariates

Sociodemographic characteristics were collected by standardized touchscreen questionnaire and nurse interview, including age, sex, self-reported ethnicity, and highest education. Lifestyle factors were recorded in the same way, including smoking, alcohol use, and physical activity. Total energy intake was estimated from the Oxford WebQ 24-h dietary recall. Prevalent hypertension, diabetes, and ischemic heart disease were ascertained from self-report, medication records, and linked hospital and primary care data. Socioeconomic status was indexed by TDI derived from postcode information, and higher scores indicate greater deprivation.

4.5 PRS calculation

Genotyping, imputation and QC workflows were performed as previously described (27). We constructed PRS for sepsis using PRSice-2 with the clumping-and-thresholding (C+T) approach. Base GWAS summary statistics were taken from FinnGen Release 12 for the sepsis endpoint (harmonized to the target genome build); SNPs were aligned to the effect allele and ambiguous variants were excluded. LD clumping used the UK Biobank target genotypes as the LD reference with an r2 threshold of 0.1, a ± 250-kb window, and clump-p set to 1. PRS were computed at multiple p-value thresholds, weighting alleles by GWAS betas; the threshold showing the strongest association in the target dataset was selected as the primary PRS. PRSice-2 model-fitting and association statistics across candidate *P*-value thresholds are provided as PRS QC results in the Supplementary Materials. For risk stratification, the PRS was grouped into quantile-defined thirds—low, medium, and high.

4.6 Plasma proteomics

Proteomic measurements in UK Biobank were generated within the UKB Pharma Proteomics Project (PPP) using a high-throughput, multiplex proximity-extension platform that assays ~3,000 circulating proteins (Olink Explore)(28). Quality control removed underperforming assays and samples, harmonized signals across plates, and corrected for batch effects. Protein abundances were supplied as vendor log2-scaled relative units and were mean-centered and standardized for analysis. Methodological details were available in the UK Biobank Showcase (field ID 1839). We analyzed the baseline proteomics release.

4.7 Statistical analysis

In the main analysis, associations were quantified using Cox proportional hazards; hazard ratio (HR) was calculated, alongside 95% confidence interval (CI), across EAT-Lancet diet index categories for sepsis incidence. Sequential covariate adjustments were performed as follows: Crude model adjusted for none covariates; Model 1 controlled for basic demographic and anthropometric factors, including age, sex, ethnicity, and BMI. Model 2 was further adjusted for socioeconomic and lifestyle characteristics such as deprivation index, education, smoking, alcohol intake, physical activity, and total energy consumption. Model 3 further incorporated baseline hypertension, diabetes, and ischemic heart disease. Trend across tertiles was evaluated using each tertile's median EAT–Lancet value as a continuous regressor in the Cox model. Additionally, the dose-response relationship was evaluated using restricted cubic splines (RCS) model, and Wald test was used to

examine potential nonlinearity. To aid interpretation of time-to-event results, we reported sepsis cases/total participants, person-years, and crude incidence rates (per 1,000 person-years) by EAT–Lancet categories, together with the median time to sepsis onset among cases (median [IQR]).

Subgroup analysis evaluated the link between EAT–Lancet adherence and sepsis risk across prespecified covariates. Interaction effects were evaluated using likelihood ratio tests to determine statistical significance. Model proportionality was assessed based on Schoenfeld residuals and by visualizing survival trends using log–log transformations. Sensitivity analyses included: (1) performing a comparison of baseline characteristics between participants who were included and excluded from the analysis due to missing dietary information; (2) excluding participants with only one 24-h dietary recall; (3) incident sepsis identified in the early 1–2-year follow-up window; and (4) repeating the primary analyses using two alternative EAT-Lancet scoring (Stubbendorff and Kesse-Guyot). In addition, to account for the potential competing risk of death, we conducted Fine–Gray sub-distribution hazard models treating all-cause mortality as the competing event, adjusting for the same covariates as Model 3.

To assess the joint effects of diet and genetic susceptibility, participants were cross-classified by EAT-Lancet adherence score and polygenic-risk strata. HR for each combined category was estimated using Model 3, and interaction on the multiplicative scale between the diet score and the PRS was tested.

In proteomic analysis, multiple linear regression assessed links between the EAT–Lancet index and plasma protein levels, while Cox proportional hazards models evaluated protein-sepsis risk relationships. Proteins with a missing value rate greater than 20% were excluded, leaving 2,911 proteins for subsequent analysis. Significant results were displayed as volcano plots (ggplot2, R). For proteins linked to both diet and sepsis, causal mediation analysis was performed to estimate the proportion of association mediated by protein levels. Statistical significance for protein associations was defined as a P value below 0.05, with False Discovery Rate (FDR) correction applied at < 0.05 and Bonferroni correction applied at $P < 0.05$ to account for multiple testing. GO and KEGG pathway enrichment analyses were conducted using KOBAS and clusterProfiler with FDR correction.

Declarations

Availability of data and materials

The UK Biobank data used in this study can be accessed through the UK Biobank website (https://www.ukbiobank.ac.uk/), following approval for access (Application: 617672). The code used for data analysis in this study is available upon request. Interested parties may contact the corresponding author via email, or the code can be provided as part of a collaborative arrangement.

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Author contributions

Data extraction and statistical analysis: WBN; Writing manuscript: QH; Conceptualization and supervision: BMH; Reviewing and editing: ZYP and YLL. All authors read and approved the final manuscript. ZYP was the guarantor of this work, had full access to and verified all the data in the study, and took responsibility for data integrity and the accuracy of the data analysis.

Competing interests

The authors declare that they have no competing interests.

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Tables and figure legends

Table 1 Baseline characteristics of included participants.

Characteristics	Total (N=199085)	Non-sepsis (N=194059)	Sepsis (N=5026)	*P*-value
Age, (years)	57 (50, 62)	57 (50, 62)	62 (56, 65)	**< 0.001**
Gender, n (%)				**< 0.001**
Female	109753 (55.13)	107658 (55.48)	2095 (41.68)	
Male	89332 (44.87)	86401 (44.52)	2931 (58.32)	
Ethnicity, n (%)				0.079
White	186636 (93.75)	181894 (93.73)	4742 (94.35)	
Other	12449 (6.25)	12165 (6.27)	284 (5.65)	
BMI, (kg/m^2)				**< 0.001**
18.5 - 24.9	71989 (36.16)	70712 (36.44)	1277 (25.41)	
< 18.5 or ≥ 25.0	127096 (63.84)	123347 (63.56)	3749 (74.59)	
TDI	-2.35 (-3.74, 0.01)	-2.35 (-3.75, 0.00)	-2.29 (-3.70, 0.19)	**0.027**
Education level, n (%)				0.070
High school graduate or higher	123186 (61.88)	120014 (61.84)	3172 (63.11)	
Less than high school	75899 (38.12)	74045 (38.16)	1854 (36.89)	
Smoking status, n (%)				**< 0.001**
Never	112966 (56.74)	110657 (57.02)	2309 (45.94)	
Previous	70769 (35.55)	68530 (35.31)	2239 (44.55)	
Current	15350 (7.71)	14872 (7.66)	478 (9.51)	
Alcohol drinking, n (%)				**< 0.001**
Never	6125 (3.08)	5936 (3.06)	189 (3.76)	
Previous	5901 (2.96)	5685 (2.93)	216 (4.30)	
Current	187059 (93.96)	182438 (94.01)	4621 (91.94)	
Physical activity, days/week				**< 0.001**
0	69505 (34.91)	67381 (34.72)	2124 (42.26)	
1-2	65009 (32.65)	63524 (32.73)	1485 (29.55)	
3-4	42603 (21.40)	41709 (21.49)	894 (17.79)	

≥5	21968 (11.03)	21445 (11.05)	523 (10.41)	
Total energy intake (Kcal/day)	2001 (1675, 2374)	2000 (1674, 2372)	2041 (1702, 2418)	< **0.001**
Hypertension	10899 (5.47)	10302 (5.31)	597 (11.88)	< **0.001**
Diabetes	2629 (1.32)	2424 (1.25)	205 (4.08)	< **0.001**
Ischemic heart disease	5173 (2.60)	4862 (2.51)	311 (6.19)	< **0.001**
The EAT-Lacet index	11 (10, 11)	11 (10, 11)	10 (10, 11)	< **0.001**
≤ 9	40541 (20.36)	39377 (20.29)	1164 (23.16)	
= 10	56065 (28.16)	54563 (28.12)	1502 (29.88)	
= 11	60794 (30.54)	59293 (30.55)	1501 (29.86)	
≥ 12	41685 (20.94)	40826 (21.04)	859 (17.09)	

Continuous variables were expressed as mean ± standard deviation or median (interquartile range), and differences were assessed using the Student's t-test or Kruskal–Wallis test, as appropriate. Categorical variables were presented as counts (percentages) and compared using the χ^2 test. BMI, body mass index; TDI, Townsend Deprivation Index.

Table 2 Association between the EAT-Lancet index and the risk of sepsis.

The EAT-Lancet index	Case/Total	Crude Model		Model 1		Model 2		Model 3	
		HR (95%CI)	*P*-value	HR (95%CI)	*P*-value	HR (95%CI)	*P*-value	HR (95%CI)	*P*-value
1-point increment	5026/199085	0.91 (0.89, 0.93)	< **0.001**	0.92 (0.90, 0.94)	< **0.001**	0.94 (0.92, 0.96)	< **0.001**	0.94 (0.92, 0.96)	< **0.001**
≤ 9	1164/40541	Reference		Reference		Reference		Reference	
= 10	1502/56065	0.93 (0.86, 1.01)	0.071	0.92 (0.86, 1.00)	**0.044**	0.96 (0.89, 1.04)	0.291	0.96 (0.89, 1.04)	0.304
= 11	1501/60794	0.86 (0.79, 0.92)	< **0.001**	0.85 (0.79, 0.92)	< **0.001**	0.90 (0.83, 0.97)	**0.007**	0.90 (0.83, 0.98)	**0.010**
≥ 12	859/41685	0.71 (0.65, 0.78)	< **0.001**	0.74 (0.68, 0.81)	< **0.001**	0.80 (0.73, 0.88)	< **0.001**	0.81 (0.74, 0.89)	< **0.001**
P for trend			< **0.001**		< **0.001**		< **0.001**		< **0.001**

The crude model did not adjust for any covariates. Model 1 adjusted for age, gender, ethnicity and BMI. Model 2 adjusted for model 1 plus TDI, education level, smoking status, alcohol drinking, physical activity and total energy intake. Model 3 adjusted for model 2 plus hypertension, diabetes and ischemic heart disease. HR, hazard ratio; CI, confidence interval; BMI, body mass index; TDI, Townsend Deprivation Index.

Table 3 Association between EAT-Lancet indiex and sepsis risk stratified by PRS.

The EAT-Lancet index	Low genetic risk		Medium genetic risk		High genetic risk	
	HR (95%CI)	*P*-value	HR (95%CI)	*P*-value	HR (95%CI)	*P*-value
≤ 9	Reference		Reference		Reference	
= 10	1.03 (0.90, 1.18)	0.673	0.90 (0.79, 1.03)	0.143	0.96 (0.84, 1.10)	0.539

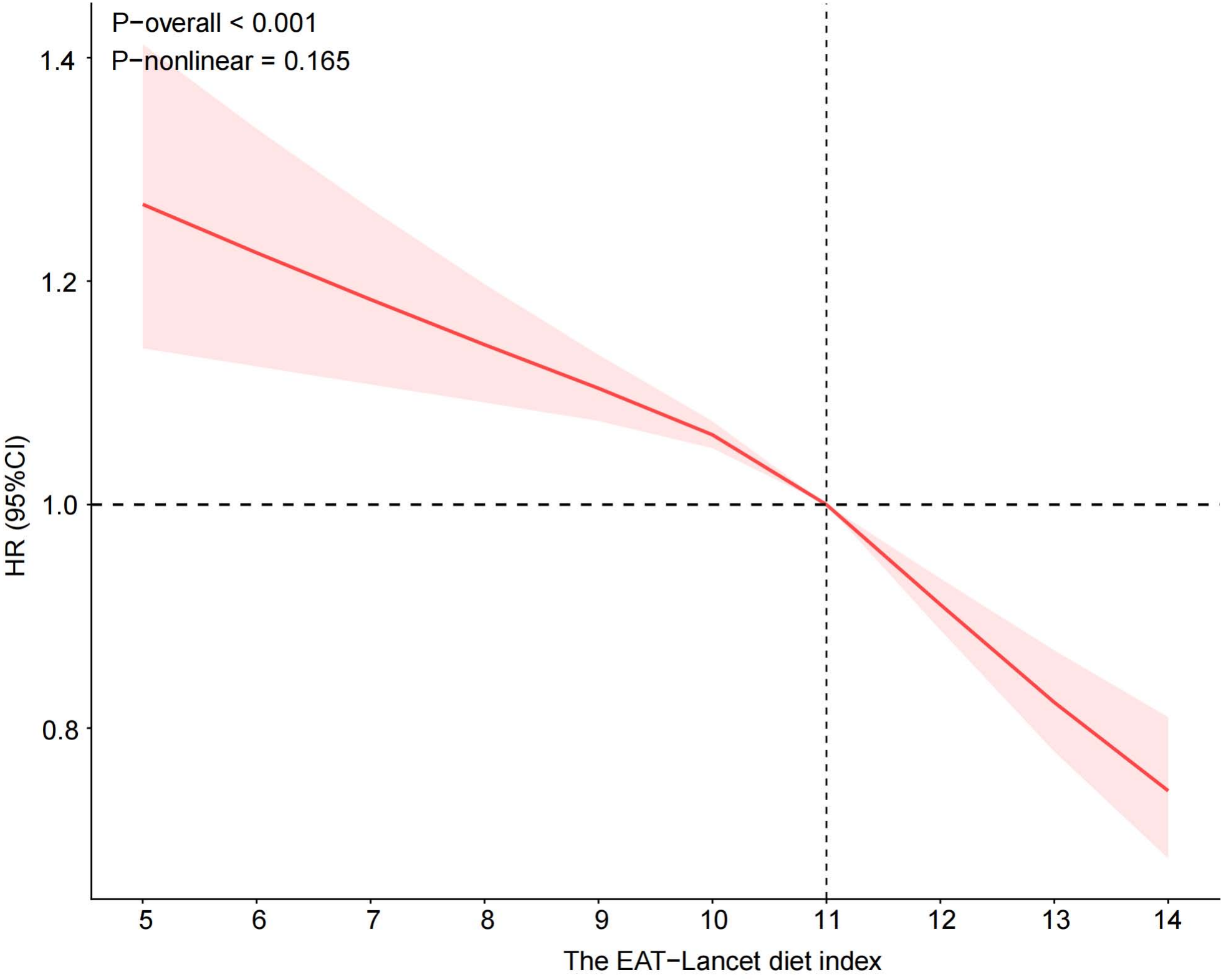
= 11	0.86 (0.74, 0.99)	**0.034**	0.86 (0.75, 0.98)	**0.025**	1.00 (0.88, 1.15)	0.951
≥ 12	0.81 (0.69, 0.96)	**0.014**	0.79 (0.67, 0.92)	**0.003**	0.83 (0.71, 0.97)	**0.021**
P for trend		**0.001**		**0.002**		**0.013**

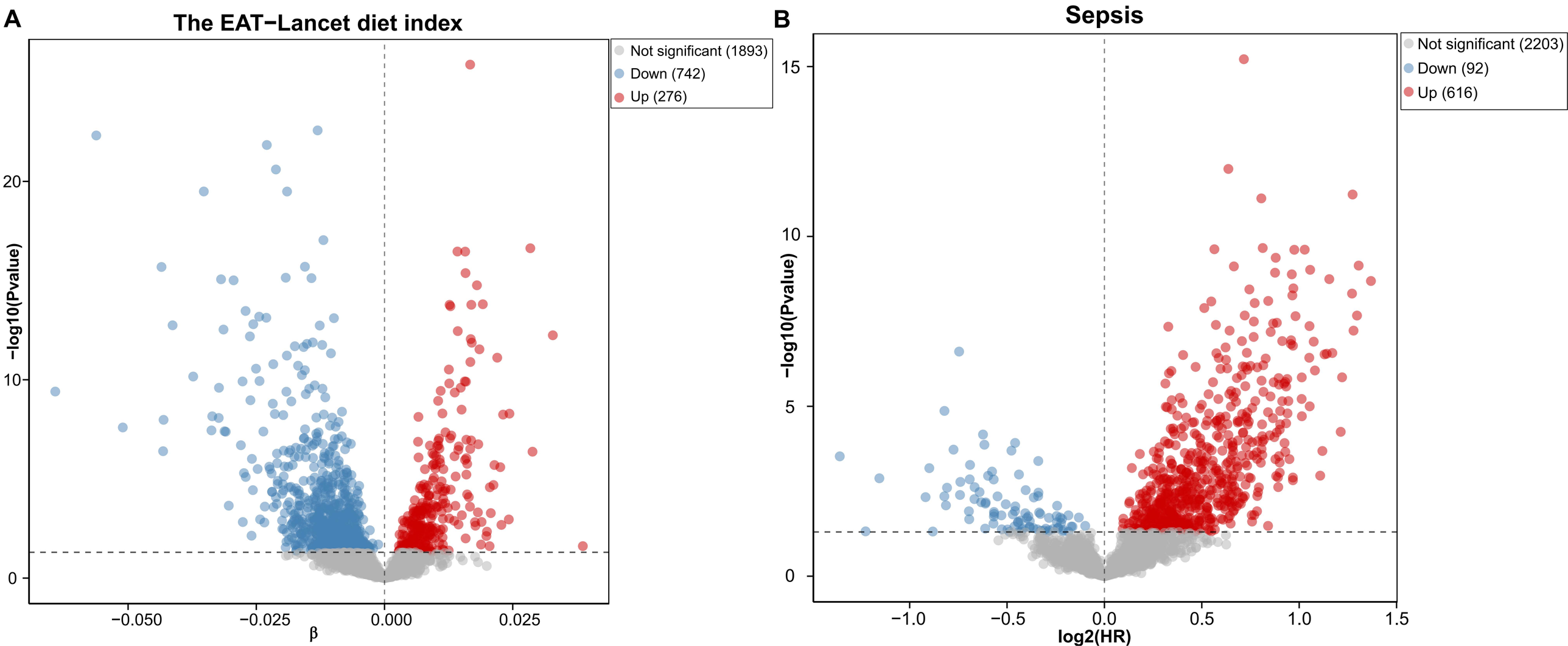
The model adjusted for age, gender, ethnicity, BMI, TDI, education level, smoking status, alcohol drinking, physical activity, total energy intake, hypertension, diabetes, ischemic heart disease, the first 10 genetic principal components and genotyping batch. PRS, polygenic risk score; HR, hazard ratio; CI, confidence interval; BMI, body mass index; TDI, Townsend Deprivation Index.

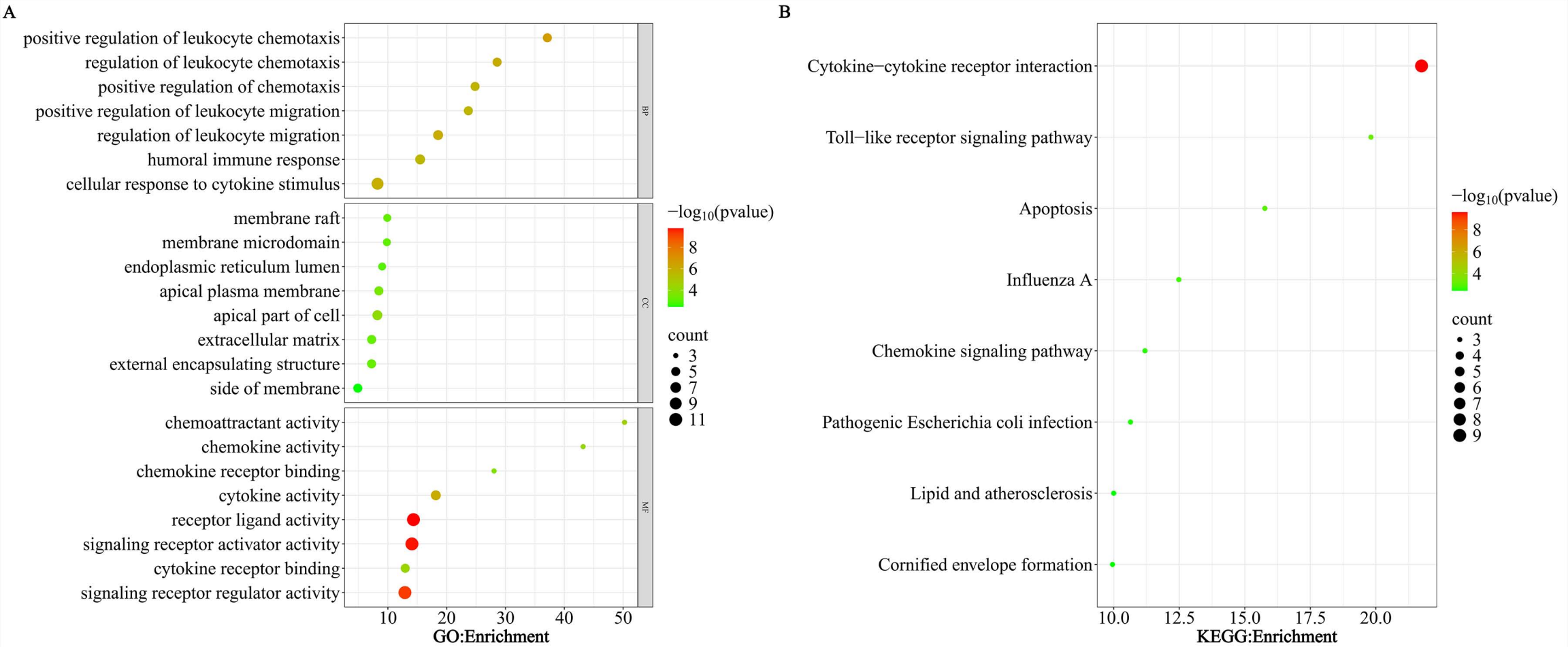
Figure 1 The dose-response association between the EAT-Lancet diet index and sepsis risk assessed by restricted cubic splines regression.

Figure 2 Volcano plots of the associations of the EAT-Lancet diet index and plasma proteins with sepsis risk.

Figure 3 Enriched pathways from GO analysis (A) and KEGG analysis (B) of mediating proteins.


P−overall < 0.001
P−nonlinear = 0.165
1.4
1.2
1.0
0.8
HR (95%CI)
5
6
7
8
9
10
11
12
13
14
The EAT−Lancet diet index


A
The EAT−Lancet diet index
Not significant (1893)
Down (742)
Up (276)
−log10(Pvalue)
20
10
0
−0.050
−0.025
0.000
0.025
β
B
Sepsis
Not significant (2203)
Down (92)
Up (616)
−log10(Pvalue)
15
10
5
0
−1.0
−0.5
0.0
0.5
1.0
1.5
log2(HR)


A
positive regulation of leukocyte chemotaxis
regulation of leukocyte chemotaxis
positive regulation of chemotaxis
positive regulation of leukocyte migration
regulation of leukocyte migration
humoral immune response
cellular response to cytokine stimulus
membrane raft
membrane microdomain
endoplasmic reticulum lumen
apical plasma membrane
apical part of cell
extracellular matrix
external encapsulating structure
side of membrane
chemoattractant activity
chemokine activity
chemokine receptor binding
cytokine activity
receptor ligand activity
signaling receptor activator activity
cytokine receptor binding
signaling receptor regulator activity
BP
CC
MF
10
20
30
40
50
GO:Enrichment
−log10(pvalue)
8
6
4
count
3
5
7
9
11
B
Cytokine−cytokine receptor interaction
Toll−like receptor signaling pathway
Apoptosis
Influenza A
Chemokine signaling pathway
Pathogenic Escherichia coli infection
Lipid and atherosclerosis
Cornified envelope formation
10.0
12.5
15.0
17.5
20.0
KEGG:Enrichment
−log10(pvalue)
8
6
4
count
3
4
5
6
7
8
9