

The synergistic impact of anti-inflammatory and nutrient-rich dietary patterns on long term prognosis among individuals with osteoarthritis

Keywords

dietary patterns, NHANES, Dietary Inflammatory Index, mortality, Osteoarthritis, Healthy Eating Index

Abstract

Introduction

To investigate the associations between healthy and anti-inflammatory dietary patterns and mortality risk in osteoarthritis (OA) patients, and to identify key dietary factors influencing long-term outcomes.

Material and methods

We analyzed data from 3,012 OA patients participating in the National Health and Nutrition Examination Survey (NHANES) between 2007 and 2018. Dietary patterns were assessed using two validated indices: the Healthy Eating Index-2015 (HEI-2015) for overall diet quality and the Dietary Inflammatory Index (DII) for inflammatory potential, both derived from 24-hour dietary recall data. Mortality outcomes were ascertained through linkage with the National Death Index. Weighted Cox proportional hazards regression models were employed to evaluate associations between dietary patterns and all-cause mortality, with adjustments for demographic, lifestyle, and clinical confounders. To identify key dietary predictors, we applied Least Absolute Shrinkage and Selection Operator (LASSO) regression. A prognostic nomogram was developed to assess predictive performance.

Results

A healthy, anti-inflammatory diet (high HEI-2015, low DII) was associated with a 14% lower mortality risk (HR: 0.86, 95% CI: 0.75–1.00, p-trend = 0.045) compared to an unhealthy, pro-inflammatory diet. LASSO regression highlighted 21 key dietary factors, including vitamins (B2, B6, B12, D, E), minerals (zinc, selenium, iron), fatty acids (n-3, n-6, monounsaturated), and whole foods (fruits, vegetables, grains). The nomogram showed reliable predictive accuracy (AUC: 0.65 at 3 years, 0.70 at 12 years). Subgroup analyses indicated stronger effects in non-smokers (HR: 0.79, 95% CI: 0.65–0.97).

Conclusions

Adopting a healthy, anti-inflammatory dietary pattern can reduce mortality rates in individuals with arthritis, with specific micronutrients and food groups playing a critical role.

1 **The synergistic impact of anti-inflammatory and nutrient-**
2 **rich dietary patterns on long term prognosis among**
3 **individuals with osteoarthritis**

4 *Fuyue Yang^{1,#}, Yan Wang^{2,#}, Qingyuan Li^{3,4,5,#}, Tong Feng⁶, Wenjing Dai^{3,4,5,*}*

¹*Department of Rheumatology, Chengdu Fifth People's Hospital, Chengdu, China*

²*School of Nursing, Chengdu Medical College, Chengdu, 610500, China*

³ *School of Clinical Medicine, Chengdu Medical College, Chengdu, People's Republic of China*

⁴*Department of Respiratory and Critical Care Medicine, The First Affiliated Hospital of Chengdu Medical College, Chengdu, People's Republic of China*

⁵*Laboratory of Geriatric Respiratory Diseases of Sichuan Higher Education Institute, Chengdu, People's Republic of China⁴*

⁶*Department of Respiratory and Critical Care Medicine, Deyang People's Hospital, Affiliated Hospital of Chengdu, College of Medicine, Deyang, China*

These authors contributed equally to this work.

Corresponding author:

Wenjing Dai

Email Address: daiwenjing168@outlook.com

Department of Respiratory and Critical Care Medicine, The First Affiliated Hospital of Chengdu Medical College, Chengdu, People's Republic of China

Abstract

5 Objective: To investigate the associations between healthy and anti-inflammatory
6 dietary patterns and mortality risk in osteoarthritis (OA) patients, and to identify key
7 dietary factors influencing long-term outcomes.

8 Methods: We analyzed data from 3,012 OA patients participating in the National
9 Health and Nutrition Examination Survey (NHANES) between 2007 and 2018. Dietary
10 patterns were assessed using two validated indices: the Healthy Eating Index-2015
11 (HEI-2015) for overall diet quality and the Dietary Inflammatory Index (DII) for
12 inflammatory potential, both derived from 24-hour dietary recall data. Mortality
13 outcomes were ascertained through linkage with the National Death Index. Weighted
14 Cox proportional hazards regression models were employed to evaluate associations
15 between dietary patterns and all-cause mortality, with adjustments for demographic,
16 lifestyle, and clinical confounders. To identify key dietary predictors, we applied Least
17 Absolute Shrinkage and Selection Operator (LASSO) regression. A prognostic
18 nomogram was developed to assess predictive performance.

19 Results: A healthy, anti-inflammatory diet (high HEI-2015, low DII) was
20 associated with a 14% lower mortality risk (HR: 0.86, 95% CI: 0.75–1.00, p-trend =
21 0.045) compared to an unhealthy, pro-inflammatory diet. LASSO regression
22 highlighted 21 key dietary factors, including vitamins (B2, B6, B12, D, E), minerals
23 (zinc, selenium, iron), fatty acids (n-3, n-6, monounsaturated), and whole foods (fruits,
24 vegetables, grains). The nomogram showed reliable predictive accuracy (AUC: 0.65 at
25 3 years, 0.70 at 12 years). Subgroup analyses indicated stronger effects in non-smokers
26 (HR: 0.79, 95% CI: 0.65–0.97).

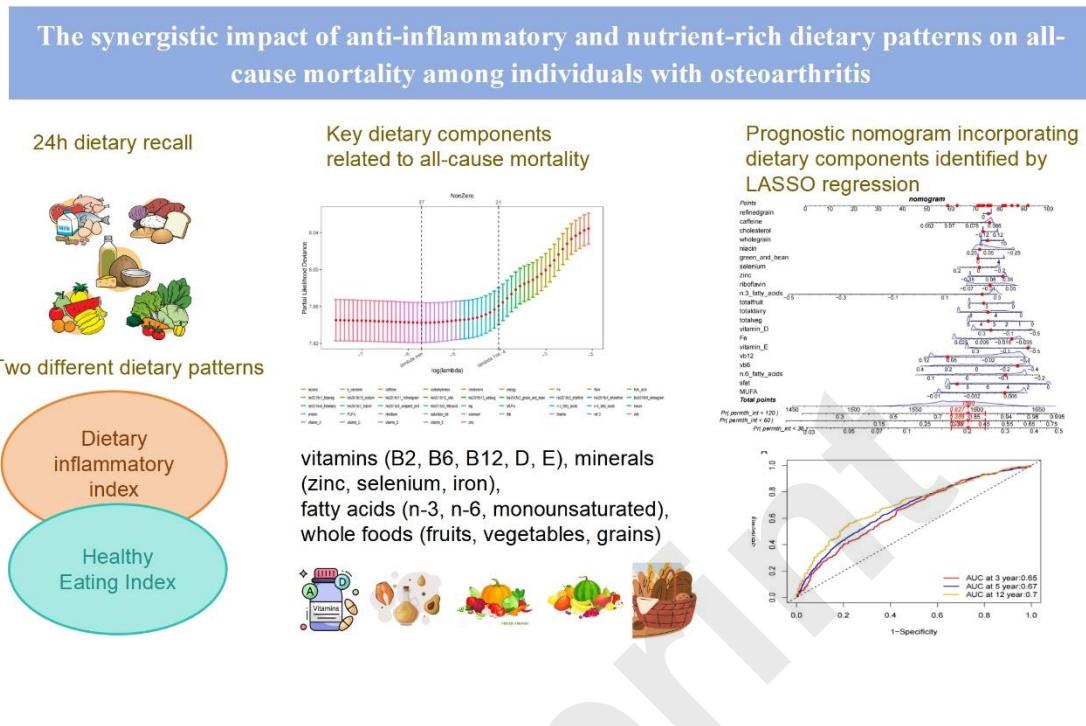
27 Conclusion: Adopting a healthy, anti-inflammatory dietary pattern can reduce
28 mortality rates in individuals with arthritis, with specific micronutrients and food
29 groups playing a critical role. Dietary interventions are beneficial for OA management,
30 highlighting the need for further longitudinal and clinical research to establish causal
31 relationships.

32 Keywords: Osteoarthritis, dietary patterns, Healthy Eating Index, Dietary

33 Inflammatory Index, mortality, NHANES

34

35 Graphical abstract



36

37

38

39

40

1. Introduction

42 Osteoarthritis (OA) is a common degenerative joint disorder characterized by
43 progressive cartilage breakdown, osteophyte formation, and chronic synovial
44 inflammation, leading to persistent pain, stiffness, and reduced mobility, significantly
45 impacting daily function and well-being[1-3]. It impacts about 303 million people
46 worldwide, with prevalence rising due to aging populations and increasing obesity rates
47 [4]. OA affects approximately 30.8 million adults, accounting for 13.4% of the adult
48 population, making it one of the most common chronic joint conditions[5]. Furthermore,
49 OA is frequently associated with multimorbidity, including cardiovascular diseases,

50 metabolic disorders such as diabetes, and systemic low-grade inflammation. These
51 comorbidities not only exacerbate disease progression but also contribute to an
52 increased risk of mortality among affected individuals [6-8].

53 Dietary factors significantly influence the onset, progression, and clinical
54 outcomes of OA[9]. Consuming foods abundant in antioxidants, omega-3 fatty acids,
55 and dietary fiber has been shown to support joint health by mitigating systemic
56 inflammation and oxidative stress[10-12]. For example, the Mediterranean diet—
57 renowned for its anti-inflammatory effects—has demonstrated associations with
58 symptomatic relief and delayed disease progression in OA patients [13].

59 Dietary quality and inflammation are measured using the Healthy Eating Index
60 (HEI-2015) and Dietary Inflammatory Index (DII). The HEI-2015 evaluates food group
61 balance and nutrient intake, with higher scores linked to lower risks of cancer,
62 cardiovascular disease, and mortality[14]. Conversely, the DII measures the
63 inflammatory properties of an individual's diet, categorizing it as either pro-
64 inflammatory or anti-inflammatory. Elevated DII scores have been associated with
65 higher mortality risks, particularly from cardiovascular disease, cancer, and metabolic
66 syndrome[15, 16]. In non-OA populations, higher HEI-2015 scores and lower DII
67 scores are associated with reduced long-term mortality. However, the application of
68 these dietary indices in OA patients and their impact on mortality risk remain
69 underexplored, particularly due to a lack of evidence from large-scale, representative
70 cohorts.

71 This study aims to explore potential associations between dietary patterns, as

72 measured by HEI-2015 and DII, and all-cause mortality in OA patients using NHANES
73 2007–2018 data. We hypothesize that higher HEI-2015 and lower DII scores may be
74 associated with lower mortality risk, though the observational design limits causal
75 inferences. These exploratory findings aim to inform future research on dietary
76 interventions for OA management.

77

78 **2. Materials and methods**

79 **2.1 Study design and population**

80 NHANES, a nationally representative cross-sectional survey by the CDC's NCHS,
81 assesses the health and nutrition of the U.S. population. Using a stratified, multistage
82 probability design, it collects data biennially from ~10,000 participants via interviews,
83 physical exams, and lab tests, supporting public health research and policy[17].

84 This study utilized data from the 2007–2018 NHANES, initially including 59,842
85 respondents. First, individuals under 18 years of age (n=23,262) were excluded, leaving
86 36,580 adult participants. Subsequently, those diagnosed with non-osteoarthritis
87 conditions or with unclear arthritis etiology (n=32,938) and those with missing dietary
88 data (n=624) were excluded, resulting in 3,018 eligible rheumatoid arthritis patients.
89 Finally, participants with missing survival data (n=6) were excluded, yielding 3,012
90 subjects for analysis (Figure 1).

91

92 **2.2 Definition of osteoarthritis**

93 OA was identified using the NHANES Medical Conditions Questionnaire (MCQ).
94 Participants were asked, “Have you ever been told by a doctor or other health

95 professional that you had arthritis?” (Question code: MCQ160A). Those responding
96 “yes” were further asked to specify the type of arthritis (MCQ190/191/195: “Which
97 type of arthritis was it?”), with response options including osteoarthritis, rheumatoid
98 arthritis, and others. Only participants reporting a diagnosis of osteoarthritis were
99 included in this study[18].

100

101 **2.3 Dietary assessment**

102 Dietary intake data from NHANES (2007–2018) were collected via 24-hour
103 dietary recall interviews under the What We Eat in America initiative. Trained
104 professionals used the Automated Multiple-Pass Method to ensure accurate recall of all
105 foods and beverages consumed from midnight to midnight the previous day, including
106 portion sizes and preparation methods. Each participant completed two non-
107 consecutive recalls: one in-person at the Mobile Examination Center with measurement
108 aids (e.g., cups, spoons, visual models) and one by telephone 3–10 days later with a
109 provided food measurement guide. Mean consumption values from both recalls were
110 used to reduce variability, with single-day data included for participants with only one
111 recall.

112 The HEI-2015 was used to assess overall diet quality in relation to the 2015–2020
113 Dietary Guidelines for Americans[19]. The HEI-2015 evaluates diet quality based on
114 the 2015–2020 Dietary Guidelines for Americans, comprising 13 components (total
115 score: 0–100). Key adequacy components include: (1) hei2015c1_totalveg (total
116 vegetables, 0–5 points), which measures intake of all vegetables (excluding legumes

117 and starchy vegetables like corn) in cup equivalents per 1,000 kcal, with higher intake
118 yielding higher scores; (2) hei2015c2_green_and.Bean (greens and beans, 0–5 points),
119 which scores intake of dark green vegetables (e.g., spinach, broccoli) and legumes (e.g.,
120 soybeans, lentils), encouraging their consumption as key healthy diet components; (3)
121 hei2015c3_totalfruit (total fruits, 0–5 points), which includes whole fruits and fruit
122 juices, with higher intake scoring higher but prioritizing whole fruits to limit added
123 sugars; and (4) hei2015c4_wholefruit (whole fruits, 0–5 points), which specifically
124 evaluates non-juice fruit intake to promote direct fruit consumption and minimize added
125 sugar intake. Other adequacy components include whole grains, dairy, total protein
126 foods, seafood and plant-based proteins, and fatty acids ratio. Moderation components
127 include refined grains, sodium, added sugars, and saturated fats. Higher HEI-2015
128 scores indicate better diet quality.

129 The DII was calculated to evaluate the inflammatory potential of the diet, which
130 has been associated with chronic disease outcomes, including mortality[20]. The
131 Dietary Inflammatory Index (DII) quantifies the inflammatory impact of 45 dietary
132 components, based on literature linking diet to inflammation markers (e.g., C-reactive
133 protein, interleukin-6). This study used 28 NHANES components, including energy,
134 macronutrients (carbohydrates, proteins, fats, cholesterol), micronutrients (vitamins A,
135 B1, B2, B6, B12, C, D, E, niacin, folate, iron, magnesium, selenium, zinc), and others
136 (caffeine, alcohol). DII scores were calculated by standardizing intake into z-scores
137 using a global reference database, converting to percentiles, centering with (percentile
138 × 2) – 1, and multiplying by component-specific inflammatory effect scores from

139 literature. Component scores were summed for the overall DII score.

140

141 **2.4 Ascertainment of mortality**

142 Mortality status in the NHANES was determined through probabilistic linkage
143 with the National Death Index (NDI), a comprehensive database of death records
144 maintained by the NCHS. Additional data sources, including Social Security
145 Administration records and Medicare/Medicaid Services data, were used to enhance
146 linkage accuracy. NHANES participants provided personal identifiers (e.g., name,
147 Social Security Number, date of birth, sex, and state of residence) during the survey,
148 which were used to match records with NDI entries via a probabilistic algorithm. The
149 linked dataset included vital status, date of death (month and year), and cause of death
150 coded. Follow-up extended from the NHANES examination date to December 31 of
151 2019.

152

153 **2.5 Assessment of covariates**

154 The NHANES collected comprehensive covariate data through structured
155 questionnaires, clinical examinations, and laboratory tests to adjust for potential
156 confounding factors in mortality analyses. These covariates encompassed demographic
157 factors (age as a continuous variable, self-reported gender, race/ethnicity categorized
158 as Non-Hispanic White, Non-Hispanic Black, Hispanic, or Other, and marital status
159 classified as married, widowed, divorced, separated, never married, or living with a
160 partner), socioeconomic indicators (family income level via the Poverty Income Ratio

161 and educational attainment as less than high school, high school graduate, or college or
162 higher), lifestyle and behavioral factors (smoking status as never, former, or current
163 smoker, alcohol consumption as none, mild, moderate, or heavy based on dietary
164 interviews, and physical activity measured in MET-minutes per week), medical history
165 and health status (diabetes defined by self-reported diagnosis, fasting glucose ≥ 126
166 mg/dL, HbA1c $\geq 6.5\%$, or antidiabetic medication use, and hypertension identified by
167 self-reported diagnosis, blood pressure $\geq 140/90$ mmHg, or antihypertensive
168 medication use), and nutritional and metabolic factors (dietary supplement use in the
169 past 30 days, total caloric intake from 24-hour dietary recalls, and estimated glomerular
170 filtration rate calculated using the CKD-EPI equation incorporating serum creatinine,
171 age, gender, and race)[21] (Supplementary materials).

172

173 **2.6 Statistical analysis**

174 All statistical analyses were performed utilizing data from the National Health and
175 Nutrition Examination Survey (NHANES). To ensure the accuracy and generalizability
176 of the findings, appropriate survey weights were applied to adjust for the complex,
177 stratified, multistage probability sampling design inherent in NHANES. Additionally,
178 first-day dietary recall weights were incorporated for dietary data analysis to produce
179 nationally representative estimates. Continuous variables were summarized as means \pm
180 standard errors (SE) when normally distributed, while non-normally distributed
181 variables were expressed as medians with interquartile ranges (IQR). Categorical
182 variables were reported as weighted frequencies and percentages to reflect their

183 distribution accurately. Group comparisons were assessed using weighted analysis of
184 variance (ANOVA) for continuous measures and weighted chi-square tests for
185 categorical variables. To address missing covariates within the NHANES 2007–2018
186 dataset, the Multivariate Imputation by Chained Equations (MICE) method was
187 employed, ensuring robust handling of incomplete data through multiple imputation
188 techniques. (Supplemental figure1 and 2).

189 The HEI-2015 and DII were categorized into quartiles: HEI-2015 as Q1 (≤ 47.17),
190 Q2 (47.17–55.93], Q3 (55.93–66.10], Q4 (> 66.10); DII as Q1 (≤ -0.089), Q2 (-0.089–
191 1.42], Q3 (1.42–2.77], Q4 (> 2.77). Dietary patterns were defined based on the median
192 values of the HEI-2015, and DII as follows: Unhealthy and Pro-Inflammatory (HEI-
193 2015 ≤ 55.93947 , DII > 1.424381), Healthy and Pro-Inflammatory (HEI-2015 $>$
194 55.93947, DII > 1.424381), Unhealthy and Anti-Inflammatory (HEI-2015 ≤ 55.93947 ,
195 DII ≤ 1.424381), and Healthy and Anti-Inflammatory (HEI-2015 > 55.93947 , DII \leq
196 1.424381). Weighted Cox proportional hazards regression models were used to assess
197 the association between dietary patterns (HEI-2015, DII, and their composite effects)
198 and all-cause mortality, with hazard ratios (HRs) and 95% confidence intervals (CIs).
199 The p-value for trend was calculated to evaluate dose-response relationships.

200 Subgroup analyses were conducted to evaluate effect modification by key
201 demographic and clinical variables with interaction p-values calculated to assess
202 heterogeneity across subgroups. Sensitivity analyses mitigated reverse causation by
203 excluding participants who died within the first year of follow-up. The relationships
204 between dietary indices (HEI-2015 and DII) and mortality were examined using

205 restricted cubic spline (RCS) models to assess non-linear associations, while Least
206 Absolute Shrinkage and Selection Operator (LASSO) regression, with 10-fold cross-
207 validation to determine the optimal tuning parameter (λ), identified influential dietary
208 factors linked to mortality. A prognostic nomogram, developed from LASSO-selected
209 dietary components, predicted mortality risk, with predictive accuracy evaluated using
210 the area under the receiver operating characteristic curve (AUC) and time-dependent
211 ROC analysis. Participants were stratified into high- and low-risk groups based on the
212 median risk score from the nomogram, and Kaplan-Meier survival curves compared
213 mortality risk between these groups. All statistical analyses were performed using R
214 software (version 4.2.0), with a two-sided p-value <0.05 considered statistically
215 significant.

216

217 **3. Results**

218 **3.1 Patient characteristics**

219 A cohort of 3,012 osteoarthritis patients (63.73% female, median age 63 years,
220 IQR: 54–72) was stratified into four dietary pattern groups: Unhealthy and Pro-
221 Inflammatory (n=1,070), Healthy and Pro-Inflammatory (n=548), Unhealthy and Anti-
222 Inflammatory (n=427), and Healthy and Anti-Inflammatory (n=967). Demographic and
223 clinical characteristics, as detailed in Table 1, showed significant variations ($p < 0.05$)
224 across groups for age, sex, race/ethnicity, marital status, family income poverty ratio,
225 education level, smoking status, alcohol consumption, physical activity (MET scores),
226 diabetes mellitus, dietary supplement use, and total energy intake. The Healthy and

227 Anti-Inflammatory group had the highest median family income poverty ratio (4.12,
228 IQR: 2.23–5.00) and the largest proportion of college-educated individuals (74.69%),
229 while the Unhealthy and Anti-Inflammatory group had the highest mean daily energy
230 intake (2503.04 ± 46.79 kcal). No significant difference was observed in hypertension
231 prevalence across groups ($p = 0.5$).

232

233 **3.2 Impact of a healthy and anti-inflammatory diet on mortality rates in**
234 **individuals with OA**

235 Table 2 illustrates the correlations between dietary patterns and mortality risk in
236 osteoarthritis patients, evaluating the DI and HEI-2015 stratified by quartiles, which
237 showed no statistically significant association between lower DII scores (indicating
238 reduced inflammation) or higher HEI-2015 scores (reflecting better diet quality) and
239 decreased long-term mortality risk, with hazard ratios in Model 2 of 1.11 (95% CI:
240 0.94–1.30, p-trend = 0.109) for DII and 0.91 (95% CI: 0.77–1.07, p-trend = 0.13) for
241 HEI-2015. Participants were classified into four groups based on median DII and HEI-
242 2015 values, and after comprehensive adjustment for confounders in Model 2—
243 including age, gender, race, marital status, family income, education, smoking status,
244 alcohol consumption, physical activity, diabetes, hypertension, supplement use, total
245 caloric intake, and estimated glomerular filtration rate—those following a Healthy and
246 Anti-Inflammatory Diet exhibited a 14% reduction in long-term mortality risk
247 compared to those with an Unhealthy and Pro-Inflammatory Diet (HR: 0.86, 95% CI:
248 0.75–1.00, p-trend = 0.045). Restricted Cubic Spline analysis revealed no significant

249 nonlinear associations, indicating a consistent trend across the observed ranges of
250 dietary scores (Figure 2).

251

252 **3.3 Subgroup analyses**

253 Subgroup analyses, as detailed in Table 3, were conducted to evaluate variations
254 in the association between composite dietary patterns and mortality across demographic
255 and clinical factors. No statistically significant interaction effects were found for age,
256 gender, race/ethnicity, alcohol consumption, hypertension, or diabetes mellitus (p-
257 interaction > 0.05). However, a significant interaction was observed for smoking status
258 (p-interaction = 0.04), with never-smokers adhering to a Healthy and Anti-
259 Inflammatory dietary pattern showing a significantly lower mortality risk compared to
260 those following an unhealthy dietary pattern (HR: 0.79, 95% CI: 0.65–0.97, p-trend =
261 0.04).

262

263 **3.4 Key dietary factors linked to mortality**

264 Through LASSO regression analysis (Figure 3), key dietary components
265 associated with the long-term mortality risk of OA patients were identified. Figure 3A
266 illustrates the coefficient shrinkage trajectories of 41 dietary variables, while Figure 3B
267 determines the tuning parameter (λ) via ten-fold cross-validation, selecting 1 standard
268 deviation, ultimately identifying 21 key dietary factors: caffeine, cholesterol, niacin,
269 selenium, zinc, vitamin B2, n-3 fatty acids, vitamin D, iron, vitamin E, vitamin B6,
270 vitamin B12, n-6 fatty acids, monounsaturated fatty acids, refined grains, whole grains,

271 greens and beans, total fruits, whole fruits, total vegetables, and saturated fats. Figure
272 4 presents a prognostic nomogram constructed based on the LASSO regression results,
273 further analyzing the relationship between intake and mortality. The area under the
274 receiver operating characteristic (ROC) curve indicates: the AUC at 3 years is 0.65, at
275 5 years is 0.67, and at 12 years is 0.70, with predictive accuracy increasing over time
276 (Figure 5A). Time-dependent AUC shows high predictive accuracy for long-term
277 mortality (10 years) (Figure 5B).

278 A dietary risk score was computed for each participant in the study, with
279 individuals scoring below the median categorized as "low risk" and those above the
280 median designated as "high risk." Subsequent Kaplan-Meier survival analysis
281 demonstrated a statistically significant increase in long-term mortality rates among
282 high-risk patients when compared to their low-risk counterparts (Figure 6).

283

284 **3.5 Sensitivity analysis**

285 Individuals who passed away within the initial year of follow-up were
286 systematically excluded to minimize potential bias. The findings demonstrated that
287 consistent adherence to a health-promoting, anti-inflammatory dietary pattern was
288 associated with a statistically significant 14% reduction in overall mortality risk
289 (adjusted HR: 0.86; 95% CI: 0.75–0.99). This protective association remained robust
290 even after comprehensive adjustment for all relevant confounding variables, thereby
291 reinforcing the reliability and validity of the analytical model (Table 4).

292

293 **4. Discussion**

294 This study evaluates the combined effect of HEI-2015 and DII on mortality in
295 osteoarthritis patients, finding that a healthy, anti-inflammatory diet (high HEI-2015,
296 low DII) is associated with a 14% lower mortality risk (HR=0.86, 95% CI: 0.75–1.00,
297 p-trend=0.045). LASSO regression identified 21 key dietary predictors, and a
298 nomogram showed reliable prognostic accuracy (AUC=0.70 at 12 years). Neither
299 HEI-2015 nor DII alone was significantly linked to mortality.

300 Recent studies link dietary patterns to OA outcomes. The Western dietary pattern,
301 rich in red meat, processed foods, and refined grains, is consistently associated with
302 increased OA progression. A large-scale cohort study from the Osteoarthritis Initiative
303 (n=2,842, mean age: 60.5 years) found that adherence to a Western diet elevated the
304 risk of radiographic knee OA progression (Kellgren and Lawrence grade ≥ 2) over 72
305 months (HR = 1.69, 95% CI: 1.13 – 2.52), while a prudent dietary pattern, high in fruits,
306 vegetables, whole grains, and lean proteins, was linked to a reduced risk (HR = 0.70,
307 95% CI: 0.50–0.98)[22]. These findings are supported by a systematic review of six
308 cohort studies, which confirmed that prudent and Mediterranean diets mitigate OA
309 symptom progression, whereas Western diets exacerbate clinical outcomes[23]. An
310 umbrella review further emphasized the Mediterranean diet's protective effects on OA-
311 related measures, such as pain and joint stiffness, attributed to its anti-inflammatory
312 and antioxidant properties[24]. Additionally, a 2025 systematic review and meta-
313 analysis of nine randomized controlled trials (n=898) evaluating dietary
314 interventions—including energy-restricted, Mediterranean, low-fat, anti-inflammatory,

315 low-carbohydrate, and plant-based diets—demonstrated significant improvements in
316 pain and physical function (SMD = -0.62, 95% CI: [-0.94, -0.30]), underscoring the
317 potential of anti-inflammatory dietary strategies for effective OA management[25].

318 The DII associates higher scores (pro-inflammatory diets rich in saturated fats and
319 refined carbohydrates) with worse OA outcomes. A longitudinal study (n=1,127) from
320 the Osteoarthritis Initiative found higher DII scores linked to lower quality of life (QoL),
321 with increased odds of low physical-low psychological QoL (OR=1.163, p=0.014) or
322 low physical-high psychological QoL (OR=1.131, p=0.013), and reduced overall QoL
323 ($\beta=-0.117$, $p<0.001$)[26]. A cross-sectional study (n=4,358) found a dose-dependent
324 link between higher DII scores and increased radiographic symptomatic knee OA
325 prevalence, rising from 24.0% in the lowest quartile to 35.4% in the highest ($p<0.0001$),
326 with an adjusted odds ratio of 1.40 (95% CI: 1.14–1.72, $p=0.002$) for the highest versus
327 lowest quartile[27].

328 The HEI-2015 shows limited direct evidence linking it to OA but suggests benefits
329 through its inverse correlation with the DII. A study of 937 White postmenopausal
330 women found higher HEI-2015 scores correlated with lower DII scores ($r = -0.347$, p
331 < 0.001) and reduced waist circumference ($r = -0.152$, $p < 0.001$), a key OA risk factor.
332 Women in the highest HEI-2015 quartile had lower waist circumference (80.73 cm)
333 than the lowest (85.89 cm, $p < 0.001$), with HEI-2015 independently predicting waist
334 circumference ($B = -0.168$, $p < 0.001$). Given DII's link to OA symptoms, higher HEI-
335 2015 scores may indirectly mitigate OA outcomes by reducing inflammation and
336 obesity risk. However, the lack of direct HEI-2015-OA studies necessitates further

337 longitudinal research[28] .

338 This research leverages several key strengths to ensure robust and reliable findings.

339 Utilizing data from the 2007–2018 NHANES with its stratified, multistage probability

340 sampling design, the study achieves national representativeness, allowing

341 generalization to the U.S. adult population with osteoarthritis. Comprehensive dietary

342 assessment through two non-consecutive 24-hour dietary recalls provides high-quality

343 data for calculating the HEI-2015 and DII. Advanced statistical techniques, including

344 LASSO-Cox regression and a prognostic nomogram, enhance the precision and

345 predictive accuracy of the analysis. Additionally, the study accounts for an extensive

346 range of covariates—demographic, lifestyle, and clinical factors—to minimize

347 confounding bias, while subgroup and sensitivity analyses further strengthen the

348 robustness and clinical relevance of the findings.

349 Despite its strengths, this study has several limitations. The reliance on self-

350 reported OA diagnoses from the NHANES Medical Conditions Questionnaire may

351 introduce misclassification bias, as diagnoses were not verified through clinical or

352 radiographic methods. The use of 24-hour dietary recalls captures short-term intake,

353 which may not fully represent long-term dietary patterns, potentially affecting the

354 accuracy of HEI-2015 and DII scores. As an observational study, it cannot establish

355 causality between dietary patterns and mortality risk. Furthermore, the DII calculation

356 was restricted to 28 of the 45 possible components due to limited data availability in

357 NHANES, excluding parameters such as certain bioactive compounds (e.g., flavonoids,

358 spices) that may influence inflammatory potential. This incomplete coverage may

359 reduce the DII's sensitivity and accuracy in capturing the full spectrum of dietary
360 inflammatory effects, potentially underestimating or misrepresenting associations with
361 mortality. Furthermore, the reliance on 24-hour dietary recalls, while validated for
362 short-term intake, may not adequately reflect long-term dietary patterns due to day-to-
363 day variability in food consumption. This could introduce measurement error in both
364 HEI-2015 and DII scores, affecting the precision of observed associations. Future
365 research should incorporate comprehensive DII components and longitudinal dietary
366 assessments, such as food frequency questionnaires, to enhance the robustness and
367 reliability of dietary pattern analyses in osteoarthritis cohorts.

368

369 **Conclusion**

370 This NHANES 2007–2018 study shows that a healthy, anti-inflammatory diet
371 (high HEI-2015, low DII) is associated with reduced mortality risk in osteoarthritis
372 patients. Key dietary factors include vitamins, minerals, fatty acids, and whole foods.
373 The prognostic nomogram showed reliable accuracy. Further research is needed to
374 explore causal links and optimize dietary recommendations.

375

376 **Author declarations**

377 None.

378

379 **Sources of support**

380 This research did not receive any specific grant from funding agencies in the public,
381 commercial, or not-for-profit sectors.

382

383 **Authors' contributions**

384 Fuyue Yang, Yan Wang, and Qingyuan Li contributed significantly to the study design,
385 data analysis, and initial drafting of the manuscript. Tong Feng and Wenjing Dai
386 provided critical revisions to the document, ensuring its accuracy and coherence, and
387 approved the final version for submission. All authors thoroughly reviewed and
388 endorsed the manuscript prior to its submission.

389

390 **Funding**

391 None.

392

393 **Availability of data and materials**

394 The survey data are publicly available on the internet for data users and researchers
395 throughout the world (www.cdc.gov/nchs/nhanes/).

396

397 **Acknowledgements**

398 None

399

400 **Declarations**

401 **Competing interests**

402 The authors declare no conflict of interest.

403

404 **Consent for publication**

405 Not applicable

406

407 **Ethics approval and consent to participate**

408 The Institutional Review Board (IRB) of the National Center for Health Statistics

409 (NCHS) rigorously reviewed and approved all study protocols associated with the

410 National Health and Nutrition Examination Survey (NHANES). Prior to participation,

411 written informed consent was secured from all enrolled individuals. For participants

412 under the age of 18, consent was obtained from legally authorized proxies or guardians.

413 The research adhered strictly to the ethical principles established in the 1964

414 Declaration of Helsinki, ensuring compliance with all relevant amendments and

415 contemporary ethical guidelines for human subject research.

416

417 **Declaration of generative AI and AI-assisted technologies in the writing process**

418 In the process of preparing this work, the author(s) employed GPT-4, an advanced AI

419 language model, to assist with linguistic refinement and stylistic improvements.

420 Following the use of this tool, the author(s) conducted a thorough review, made

421 necessary revisions, and ensured the accuracy, originality, and integrity of the content.

422 The author(s) assume complete responsibility for the final version of this publication,

423 including its scholarly validity and adherence to ethical standards.

424

425 **Clinical trial number:** not applicable.

426

427 Table 1 Characteristic of study sample.

Characteristic	Unhealthy and Pro-Inflammatory diet N = 1070	Healthy and Pro-Inflammatory diet N = 548	Unhealthy and Anti-Inflammatory diet N = 427	Healthy and Anti-Inflammatory diet N = 967	p-value
Age, years	60.00(52.00,71.00)	67.00(58.00,75.00)	61.00(51.00,69.00)	65.00(58.00,73.00)	< 0.001
Sex					< 0.001
Male	339(30.13)	114(17.67)	230(52.42)	390(41.03)	
Female	731(69.87)	434(82.33)	197(47.58)	577(58.97)	
Race/Ethnicity					< 0.001
Non-Hispanic Black	209(9.70)	95(6.99)	59(4.92)	122(4.85)	
Non-Hispanic White	636(77.76)	314(82.50)	286(86.32)	635(84.33)	
Mexican American	67(2.89)	49(3.72)	42(3.30)	60(2.19)	
Other Hispanic	70(2.63)	57(2.81)	17(1.08)	76(3.14)	
Other Race - Including					
Multi-Racial	88(7.01)	33(3.98)	23(4.37)	74(5.49)	
Marital					0.004
Never married	98(8.01)	31(5.45)	38(9.38)	51(5.00)	
Married/ Living with partner	586(61.10)	289(60.75)	272(68.67)	617(68.58)	
Divorced/ Widowed/					
Separated	386(30.89)	228(33.80)	117(21.95)	299(26.42)	
Family income poverty ratio	2.64(1.52,4.71)	2.95(1.54,4.92)	3.40(1.84,5.00)	4.12(2.23,5.00)	< 0.001
Education					< 0.001
Less than high school	93(4.68)	61(4.73)	19(2.85)	42(1.76)	
High school or equivalent	435(39.81)	204(36.98)	146(31.17)	258(23.55)	
College and above	542(55.52)	283(58.29)	262(65.98)	667(74.69)	
Smoking status					< 0.001
Never	448(44.32)	317(53.96)	186(43.72)	500(52.01)	
Former	345(31.30)	167(31.45)	167(40.70)	385(39.22)	
Now	277(24.38)	64(14.60)	74(15.59)	82(8.77)	
Drinking status					< 0.01
Never	132(10.25)	107(14.68)	32(5.42)	111(9.13)	
Former	262(20.46)	113(17.05)	88(17.06)	155(13.63)	
Mild	410(42.12)	229(45.85)	197(47.50)	508(54.98)	
Moderate	153(14.69)	57(11.88)	59(16.45)	131(15.36)	
Heavy	113(12.49)	42(10.55)	51(13.57)	62(6.90)	
MET scores, min/week	3201.08 (1440.00,5063.27)	2271.89 (1000.00,3532.35)	2880.00 (900.00,5280.00)	2460.00 (1080.00,4320.00)	0.001
DM					0.01
No	738(76.32)	359(71.35)	303(73.62)	740(81.61)	

Yes	332(23.68)	189(28.65)	124(26.38)	227(18.39)	
Hypertension					0.5
No	333(35.01)	165(36.48)	145(37.06)	332(39.80)	
Yes	737(64.99)	383(63.52)	282(62.94)	635(60.20)	
Use of Supplement					< 0.001
No	421(35.65)	168(24.29)	125(24.21)	205(21.50)	
Yes	649(64.35)	380(75.71)	302(75.79)	762(78.50)	
Energy,kcal	1715.77±24.41	1387.61±23.24	2503.04±46.79	2104.70±29.80	< 0.001

428

429 Abbreviations: MET: Metabolic Equivalent of Task; DM: diabetes mellitus.

430

431 Table 2 Relationship between dietary habits and mortality in osteoarthritis patients

Outcomes	Crude Model		Model 1	Model 2
	HR (95%CI)	HR (95%CI)	HR (95%CI)	HR (95%CI)
HEI-2015	Continuous	1.00(0.99,1.00)	1.00(0.99,1.00)	1.00(0.99,1.00)
	Categories			
	Q1 (≤ 47.17)	Ref.	Ref.	Ref.
	Q2 (47.17,55.93]	1.10(0.98,1.24)	1.08(0.95,1.23)	1.08(0.94,1.24)
	Q3 (55.93,66.10]	1.02(0.91,1.15)	0.99(0.87,1.11)	0.98(0.86,1.11)
	Q4 (> 66.10)	0.99(0.84,1.15)	0.92(0.78,1.09)	0.91(0.77,1.07)
	p for trend	0.646	0.206	0.13
	Continuous	1.00(0.97,1.03)	1.01(0.98,1.04)	1.03(1.00,1.06)
	Categories			
	Q1 (≤ -0.089)	Ref.	Ref.	Ref.
DII	Q2 (-0.089,1.42]	1.04(0.92,1.18)	1.08(0.95,1.22)	1.10(0.97,1.24)
	Q3 (1.42,2.77]	1.13(0.99,1.29)	1.17(1.02,1.35)	1.22(1.06,1.40)
	Q4 (> 2.77)	0.97(0.84,1.11)	1.01(0.88,1.15)	1.11(0.94,1.30)
	p for trend	0.947	0.598	0.109
	Categories			
Composition effect	Unhealthy and Pro-Inflammatory diet	Ref.	Ref.	Ref.
	Healthy and Pro-Inflammatory diet			
	Unhealthy and Anti-Inflammatory diet	1.10(0.98,1.24)	1.08(0.95,1.23)	0.95(0.83,1.10)
	Healthy and Anti-Inflammatory diet	1.02(0.91,1.15)	0.99(0.87,1.11)	0.95(0.82,1.10)
	p for trend	0.646	0.206	0.045

432

433 Crude Model: no covariates were adjusted.

434 Model 1: Adjusted covariates for model 1 included age, gender, race, marital status, family income level, and educational level.

435 Model 2: Adjusted covariates for model 2 included the covariates for model 1 plus smoking status,

437 alcohol intake, physical activity, diabetes, hypertension, use of supplements, total Kal intake and
438 eGFR.

439 HR, Hazard Ratio; 95%CI, 95% confidence interval; HEI: Healthy Eating Index; DII: Dietary
440 Inflammatory Index

441

442

443 Table 3 Stratified analyses of the relationships of dietary patterns and mortality among OA patients.

Characteristics	Unhealthy	Healthy	and Unhealthy	and Healthy	and	P for trend	P for interaction
	and Inflammatory	Pro-Inflammatory	Anti-Inflammatory	Anti-Inflammatory	and Inflammatory		
	N = 1070	N = 548	N = 427	N = 967			
Age						0.68	
<60	ref	0.92(0.72,1.17)	1.25(0.96,1.62)	1.02(0.80,1.31)	0.69		
≥60	ref	0.95(0.80,1.13)	0.74(0.59,0.91)	0.76(0.66,0.89)	<0.001		
Gender						0.35	
Male	ref	1.08(0.81,1.44)	0.86(0.67,1.11)	0.84(0.66,1.07)	0.12		
Female	ref	0.93(0.79,1.09)	1.03(0.81,1.31)	0.87(0.74,1.04)	0.13		
Race/Ethnicity						0.28	
Non-Hispanic							
Black	ref	0.72(0.48,1.07)	0.88(0.59,1.32)	0.47(0.32,0.69)	<0.001		
Non-Hispanic							
White	ref	0.98(0.83,1.14)	0.94(0.79,1.11)	0.86(0.74,1.00)	0.05		
Mexican							
American	ref	1.09(0.76,1.55)	1.58(0.88,2.81)	1.21(0.78,1.89)	0.39		
Other Hispanic	ref	0.68(0.43,1.06)	1.07(0.50,2.32)	1.23(0.86,1.76)	0.12		
Other Race	-						
Including	Multi-						
Racial	ref	1.26(0.65,2.44)	1.13(0.50,2.51)	0.98(0.60,1.62)	0.92		
Smoke status						0.04	
Never	ref	0.76(0.60,0.95)	0.79(0.63,0.97)	0.79(0.65,0.97)	0.04		
Former	ref	1.16(0.87,1.55)	1.17(0.90,1.53)	0.91(0.73,1.15)	0.39		
Now	ref	1.22(0.86,1.74)	0.72(0.49,1.07)	0.92(0.65,1.30)	0.49		
Drinking status						0.18	
Never	ref	0.91(0.56,1.48)	2.42(1.49,3.92)	1.13(0.75,1.69)	0.4		
Former	ref	1.01(0.73,1.40)	1.07(0.75,1.53)	0.87(0.62,1.22)	0.44		
Mild	ref	0.85(0.68,1.05)	0.73(0.60,0.90)	0.73(0.62,0.87)	<0.001		
Moderate	ref	1.14(0.77,1.70)	1.01(0.71,1.45)	0.96(0.66,1.38)	0.76		
Heavy	ref	1.51(0.88,2.59)	1.37(0.74,2.52)	1.36(0.93,1.98)	0.09		
Hypertension						0.24	
No	ref	1.07(0.82,1.41)	1.10(0.86,1.40)	1.03(0.81,1.31)	0.85		
Yes	ref	0.89(0.75,1.05)	0.88(0.72,1.09)	0.77(0.65,0.91)	0.002		

DM					0.72
No	ref	0.95(0.81,1.10)	0.92(0.77,1.09)	0.85(0.73,1.00)	0.06
Yes	ref	1.03(0.75,1.42)	1.06(0.74,1.53)	0.92(0.72,1.17)	0.51

444 Adjusted covariates included age, gender, race, marital status, family income, education, smoking
 445 status, alcohol consumption, physical activity, diabetes mellitus (DM), hypertension, supplement
 446 use, total calorie intake, and eGFR, excluding stratified variables.

447

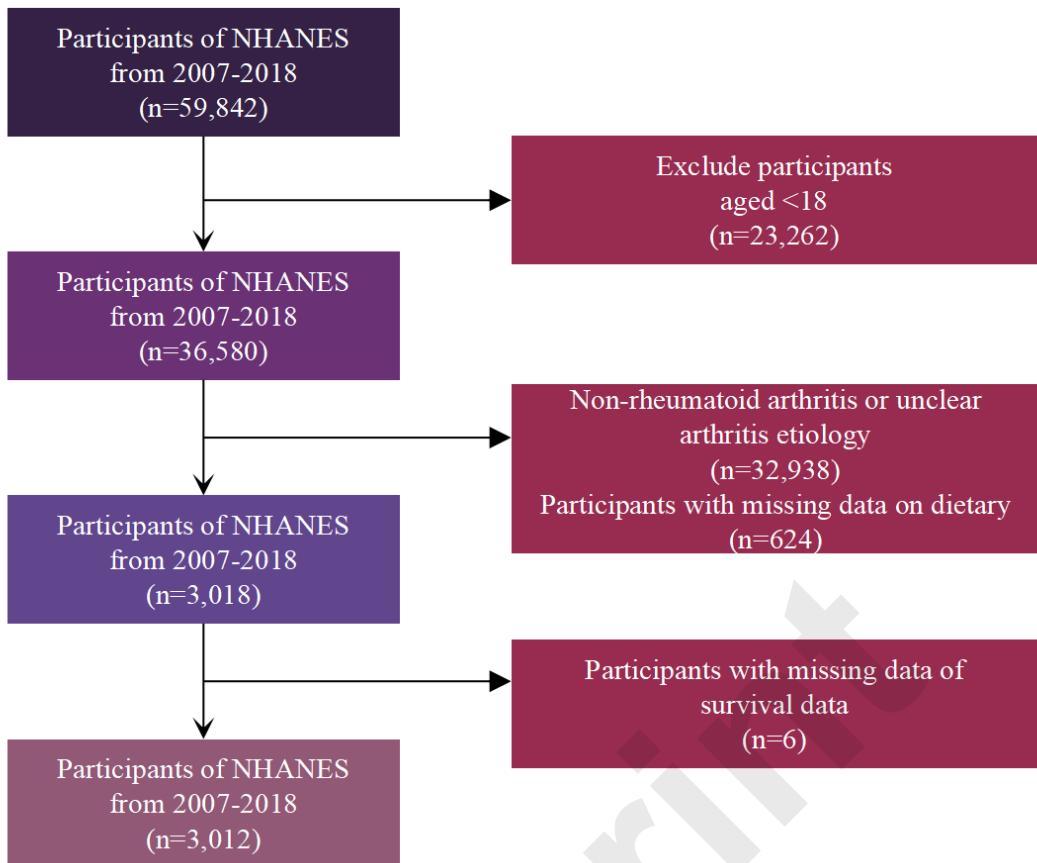
448 Table 4. Cox regression analysis of dietary habits and mortality in osteoarthritis patients excluding
 449 deaths within one year

Outcomes	Crude Model	Model 1	Model 2
	HR (95%CI)	HR (95%CI)	HR (95%CI)
Categories			
Unhealthy and Pro-Inflammatory diet	Ref.	Ref.	Ref.
Healthy and Pro-Inflammatory diet	1.00(0.86,1.15)	0.97(0.85,1.11)	0.95(0.83,1.10)
Composition effect			
Unhealthy and Anti-Inflammatory diet	1.03(0.89,1.19)	1.03(0.90,1.18)	0.95(0.82,1.10)
Healthy and Anti-Inflammatory diet	0.95(0.83,1.09)	0.90(0.78,1.04)	0.86(0.75,1.00)
<i>p</i> for trend	0.544	0.201	0.045

450

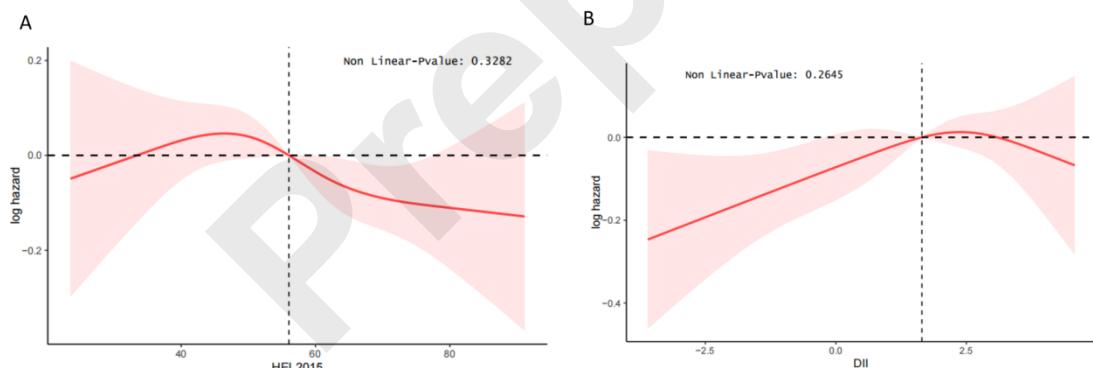
451

452



453

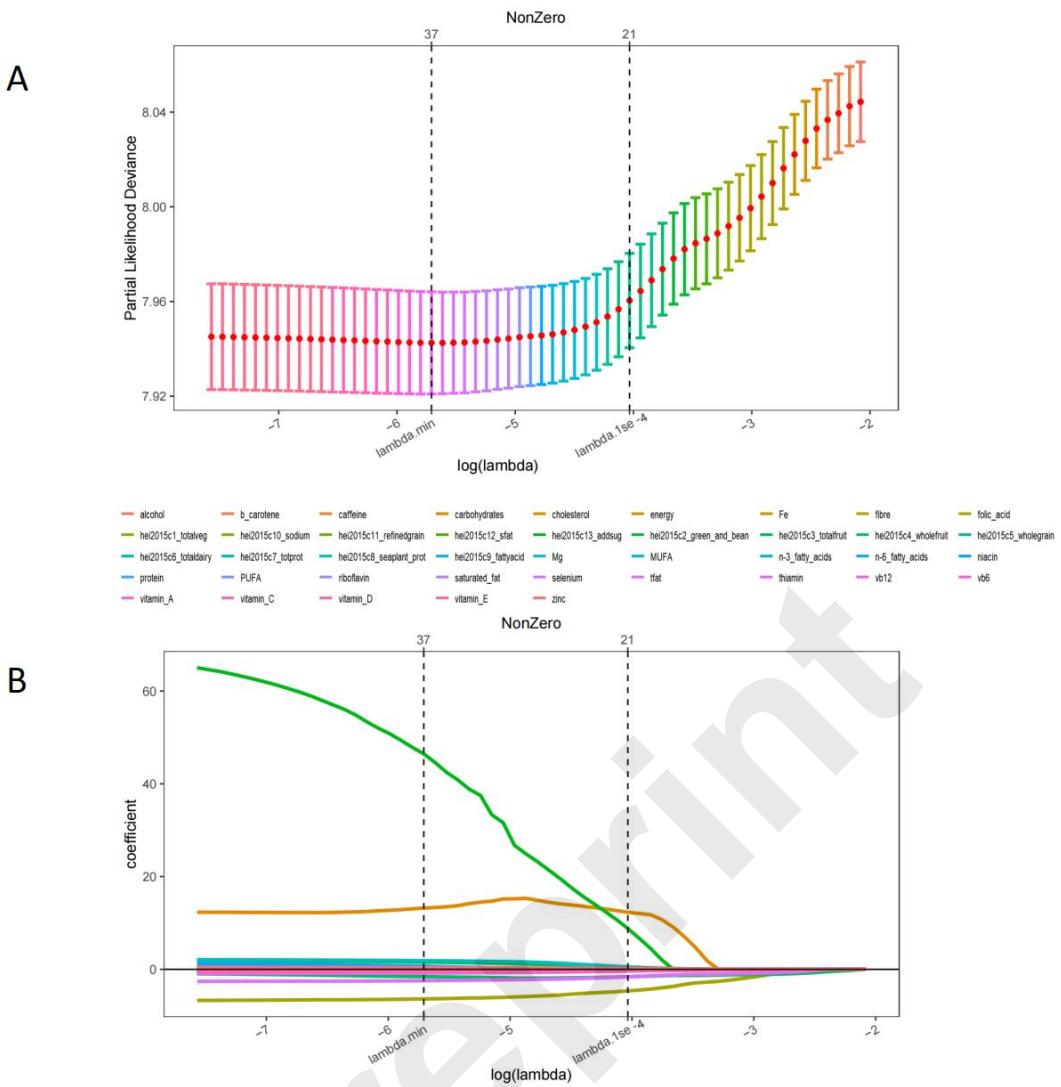
454 Figure 1. Flowchart for Selecting Eligible Study Participants



455

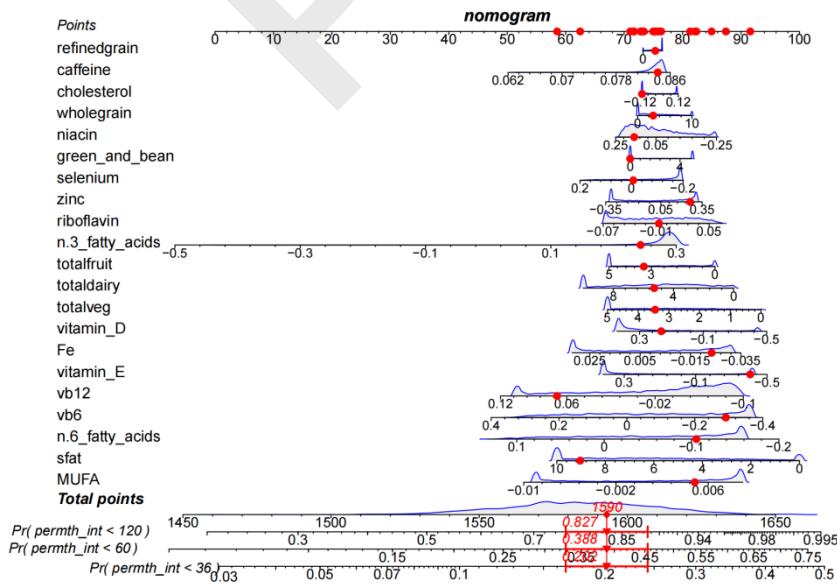
456 Figure 2. Restricted cubic spline (rcs) curves illustrating the relationship between dietary Indices
457 and long-term mortality in osteoarthritis patients. (A) Dietary Inflammatory Index (DII). (B)
458 Healthy Eating Index-2015 (HEI-2015).

459



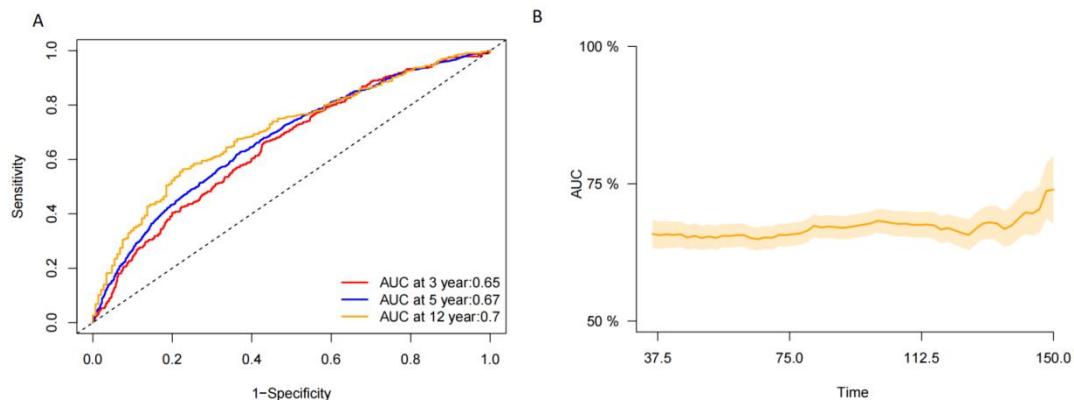
460

461 Figure 3. LASSO regression analysis for screening dietary factors linked to OA mortality. (A)
 462 Trajectory of coefficient shrinkage across 41 dietary variables. (B) Ten-fold cross-validation curve

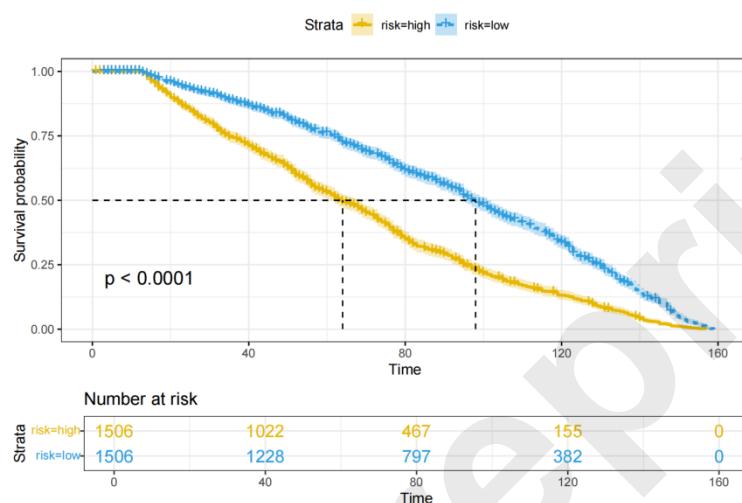


463

464 Figure 4. Prognostic nomogram incorporating dietary components identified by LASSO regression.



465
466 Figure 5. Predictive performance of the nomogram model for OA mortality. (A) ROC curve for all-
467 cause mortality prediction. (B) Time-dependent ROC analysis for overall survival.



468
469 Figure 6. Kaplan-Meier estimates comparing mortality risk in patient subgroups identified by
470 LASSO regression modeling.

471

472 Reference

1. Hunter DJ, Bierma-Zeinstra S: **Osteoarthritis**. *Lancet* 2019, **393**:1745-1759.
2. Liu W, Ma G, Zhu X, Liufu Q, Lai J: **Epidemiological trends and burden of osteoarthritis in United States: Insights from the GBD 2021 study**. *Archives of Medical Science* 2025.
3. Yang S, Zhou L, Gong W, Guo B, Wang L: **Global burden of osteoarthritis from 1990 to 2019 attributable to high body mass index**. *Archives of Medical Science* 2024, **20**:1841-1853.
4. Safiri S, Kolahi AA, Smith E, Hill C, Bettampadi D, Mansournia MA, Hoy D, Ashrafi-Asgarabad A, Sepidarkish M, Almasi-Hashiani A, et al: **Global, regional and national burden of osteoarthritis 1990-2017: a systematic analysis of the Global Burden of Disease Study 2017**. *Ann Rheum Dis* 2020, **79**:819-828.
5. Cisternas MG, Murphy L, Sacks JJ, Solomon DH, Pasta DJ, Helmick CG: **Alternative Methods for Defining Osteoarthritis and the Impact on Estimating Prevalence in a US Population-Based Survey**. *Arthritis Care Res (Hoboken)* 2016, **68**:574-580.
6. Veronese N, Cereda E, Maggi S, Luchini C, Solmi M, Smith T, Denkinger M, Hurley M, Thompson

486 T, Manzato E, et al: **Osteoarthritis and mortality: A prospective cohort study and systematic**
487 **review with meta-analysis.** *Semin Arthritis Rheum* 2016, **46**:160-167.

488 7. Veronese N, Trevisan C, De Rui M, Bolzetta F, Maggi S, Zambon S, Musacchio E, Sartori L,
489 Perissinotto E, Crepaldi G, et al: **Association of Osteoarthritis With Increased Risk of**
490 **Cardiovascular Diseases in the Elderly: Findings From the Progetto Veneto Anziano Study**
491 **Cohort.** *Arthritis Rheumatol* 2016, **68**:1136-1144.

492 8. Xing X, Wang Y, Pan F, Cai G: **Osteoarthritis and risk of type 2 diabetes: A two-sample**
493 **Mendelian randomization analysis.** *J Diabetes* 2023, **15**:987-993.

494 9. Wen S, Huang Z, Zhang B, Huang Y: **The effect of cheese intake on osteoarthritis: a Mendelian**
495 **randomization study.** *Archives of Medical Science* 2024, **20**:1943-1956.

496 10. Dai Z, Niu J, Zhang Y, Jacques P, Felson DT: **Dietary intake of fibre and risk of knee osteoarthritis**
497 **in two US prospective cohorts.** *Ann Rheum Dis* 2017, **76**:1411-1419.

498 11. Tang Y, Xu X, Zhang S, Kong W, Zhang W, Zhu T: **Genetic liability for diet-derived circulating**
499 **antioxidants, oxidative stress, and risk of osteoarthritis: a Mendelian randomization study.**
500 *Front Nutr* 2023, **10**:1233086.

501 12. Wei N, Dai Z: **The Role of Nutrition in Osteoarthritis: A Literature Review.** *Clin Geriatr Med*
502 2022, **38**:303-322.

503 13. Veronese N, Koyanagi A, Stubbs B, Cooper C, Guglielmi G, Rizzoli R, Punzi L, Rogoli D, Caruso
504 MG, Rotolo O, et al: **Mediterranean diet and knee osteoarthritis outcomes: A longitudinal**
505 **cohort study.** *Clin Nutr* 2019, **38**:2735-2739.

506 14. Wang YB, Page AJ, Gill TK, Melaku YA: **The association between diet quality, plant-based diets,**
507 **systemic inflammation, and mortality risk: findings from NHANES.** *Eur J Nutr* 2023, **62**:2723-
508 2737.

509 15. Zahedi H, Djalalinia S, Asayesh H, Mansourian M, Esmaeili Abdar Z, Mahdavi Gorabi A, Ansari
510 H, Noroozi M, Qorbani M: **A Higher Dietary Inflammatory Index Score is Associated with a**
511 **Higher Risk of Incidence and Mortality of Cancer: A Comprehensive Systematic Review and**
512 **Meta-Analysis.** *Int J Prev Med* 2020, **11**:15.

513 16. Namazi N, Larijani B, Azadbakht L: **Dietary Inflammatory Index and its Association with the**
514 **Risk of Cardiovascular Diseases, Metabolic Syndrome, and Mortality: A Systematic Review**
515 **and Meta-Analysis.** *Horm Metab Res* 2018, **50**:345-358.

516 17. Chen TC, Clark J, Riddles MK, Mohadjer LK, Fakhouri THI: **National Health and Nutrition**
517 **Examination Survey, 2015-2018: Sample Design and Estimation Procedures.** *Vital Health Stat*
518 2 2020:1-35.

519 18. Xiao Q, Cai B, Yin A, Huo H, Lan K, Zhou G, Shen L, He B: **L-shaped association of serum 25-**
520 **hydroxyvitamin D concentrations with cardiovascular and all-cause mortality in individuals**
521 **with osteoarthritis: results from the NHANES database prospective cohort study.** *BMC Med*
522 2022, **20**:308.

523 19. Krebs-Smith SM, Pannucci TE, Subar AF, Kirkpatrick SI, Lerman JL, Tooze JA, Wilson MM, Reedy
524 J: **Update of the Healthy Eating Index: HEI-2015.** *J Acad Nutr Diet* 2018, **118**:1591-1602.

525 20. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hébert JR: **Designing and developing a literature-**
526 **derived, population-based dietary inflammatory index.** *Public Health Nutr* 2014, **17**:1689-
527 1696.

528 21. Levey AS, Stevens LA, Schmid CH, Zhang YL, Castro AF, 3rd, Feldman HI, Kusek JW, Eggers P, Van
529 Lente F, Greene T, Coresh J: **A new equation to estimate glomerular filtration rate.** *Ann Intern*

530 22. Xu C, Liu T, Driban JB, McAlindon T, Eaton CB, Lu B: **Dietary patterns and risk of developing**
531 **knee osteoarthritis: data from the osteoarthritis initiative.** *Osteoarthritis Cartilage* 2021,
532 **29**:834-840.

533 23. Zeng J, Franklin DK, Das A, Hirani V: **The effects of dietary patterns and food groups on**
534 **symptomatic osteoarthritis: A systematic review.** *Nutr Diet* 2023, **80**:21-43.

535 24. Buck AN, Vincent HK, Newman CB, Batsis JA, Abbate LM, Huffman KF, Bodley J, Vos N, Callahan
536 LF, Shultz SP: **Evidence-Based Dietary Practices to Improve Osteoarthritis Symptoms: An**
537 **Umbrella Review.** *Nutrients* 2023, **15**.

538 25. Asadi S, Grafenauer S, Burley CV, Fitzgerald C, Humburg P, Parmenter BJ: **The effectiveness of**
539 **dietary intervention in osteoarthritis management: a systematic review and meta-analysis**
540 **of randomized clinical trials.** *Eur J Clin Nutr* 2025.

541 26. Chen Z, Zhang H, Jin J, Su C, Chen H, Li B: **A longitudinal study of dietary inflammatory index**
542 **and quality of life in people with osteoarthritis: data from the Osteoarthritis Initiative**
543 **database.** *Sci Rep* 2025, **15**:6024.

544 27. Veronese N, Shivappa N, Stubbs B, Smith T, Hébert JR, Cooper C, Guglielmi G, Reginster JY,
545 Rizzoli R, Maggi S: **The relationship between the dietary inflammatory index and prevalence**
546 **of radiographic symptomatic osteoarthritis: data from the Osteoarthritis Initiative.** *Eur J Nutr*
547 2019, **58**:253-260.

548 28. Arikawa AY, Kurzer MS: **Associations between Diet Quality and Anthropometric Measures in**
549 **White Postmenopausal Women.** *Nutrients* 2021, **13**.

550

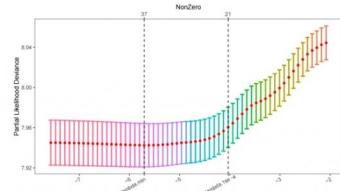
551

The synergistic impact of anti-inflammatory and nutrient-rich dietary patterns on all-cause mortality among individuals with osteoarthritis

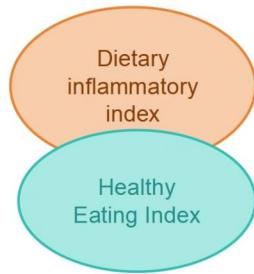
24h dietary recall



Key dietary components related to all-cause mortality



Two different dietary patterns



vitamins (B2, B6, B12, D, E), minerals (zinc, selenium, iron), fatty acids (n-3, n-6, monounsaturated), whole foods (fruits, vegetables, grains)



Prognostic nomogram incorporating dietary components identified by LASSO regression

