Preventive Medicine Reports 45 (2024) 102826

Contents lists available at ScienceDirect

Preventive Medicine Reports

journal homepage: www.elsevier.com/locate/pmedr

Association between dietary inflammatory index and all-cause mortality in patients with osteopenia or osteoporosis: A retrospective cohort study from the NHANES 2007–2018

Chenrong Ke [a,b,c], Xiaolei Zhang [a,b,c,*], Xiangyang Wang [a,b,c,*]

[a] Department of Orthopaedics, The Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University, Wenzhou 325000, Zhejiang Province, China
[b] Key Laboratory of Orthopaedics of Zhejiang Province, Wenzhou 325000, Zhejiang Province, China
[c] The Second School of Medicine, Wenzhou Medical University, Wenzhou 325000, Zhejiang Province, China

ARTICLE INFO

Keywords:
Dietary inflammatory index
Osteoporosis
Osteopenia
NHANES database

ABSTRACT

Objective: Osteoporosis is an inflammatory disease that causes a large disease burden worldwide. Dietary inflammation index (DII), a comprehensive assessment index that reflects the pro-inflammatory/anti-inflammatory level of diet was related to multiple inflammatory diseases. This study aimed to explore the association between DII and all-cause mortality in patients with osteoporosis or osteopenia.

Methods: In this retrospective cohort study, data of patients aged ≥ 45 years diagnosed as osteopenia or osteoporosis and had complete dietary intake information were extracted from the National Health and Nutrition Examination Survey (NHANES 2007–2010, 2013–2014, 2017–2018). Dietary intake information was obtained from 24-h dietary recall interviews and was used to calculate the DII score. Weighted univariate and multivariate Cox proportional hazard models were utilized to explore the association between DII and all-cause mortality in patients with osteoporosis or osteopenia, with hazard ratios (HRs) and 95 % confidence intervals (CIs). Subgroup analyses based on different age, gender and complications were further assessed this association.

Results: A total of 5,381 patients were included. Until December 31, 2019, 1,286 all-cause deaths occurred. After adjusting all covariates, high DII was associated with the high odds of all-cause mortality among patients with osteoporosis or osteopenia (HR=1.28, 95 %CI: 1.10–1.48), especially in the male (HR=1.38, 95 %CI: 1.06–1.78), aged < 65 years (HR=1.49, 95 %CI: 1.09–2.02), and without the history the cardiovascular disease (HR=1.30, 95 %CI: 1.03–1.65), diabetes mellitus (HR=1.27, 95 %CI: 1.06–1.52) and chronic kidney disease (HR=1.28, 95 %CI: 1.03–1.58).

Conclusion: A pro-inflammatory diet may have an adverse effect on the prognosis of osteoporosis patients.

1. Introduction

Osteoporosis, a systemic bone disorder, is featured by osteopenia and skeletal fragility (Anam and Insogna, 2021; Gregson et al., 2022; Reid and Billington, 2022). By report, osteoporosis enhances the risk of fragility fractures, capable of causing increased risk of all-cause, cardiovascular disease (CVD) and cancer mortality (Lorentzon et al., 2019; Wooltorton, 2006). An estimated 10.2 million middle-aged and older American suffer from osteoporosis in 2010 and the cases will reach 13.5 million by 2030 (Carlson et al., 2019; Wright et al., 2014). Osteoporosis affects people's daily life and quality of life and the health costs caused by the condition prevention and treatment also put pressure on socio-economic development. It is essential to detect the preclinical manifestations of osteoporosis as early as possible and to conduct the intervention.

Osteoporosis is a bone metabolic disorder involving systemic inflammatory activation and higher inflammation level was a risk factor for fracture (Livshits and Kalinkovich, 2022). Dysfunctional inflammation level in *vivo* may lead to increase bone resorption and suppress bone formation (Loi et al., 2016). Over the past few decades, the relationship between dietary intake and the inflammation has received attention, and good nutritional status may help decrease the inflammation of the

* Corresponding authors at: Department of Orthopaedics, The Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University, No. 109 West Xueyuan Road, Lucheng District, Wenzhou, Zhejiang Province 325000, China.
E-mail addresses: xiangyangwang@wmu.edu.cn (X. Zhang), zhangxiaolei@wmu.edu.cn (X. Wang).

https://doi.org/10.1016/j.pmedr.2024.102826
Received 6 February 2024; Received in revised form 10 July 2024; Accepted 10 July 2024
Available online 20 July 2024

body (Eckart et al., 2020; Wieringa and Thurnham, 2023). Studies reported that the intake of several dietary nutrients, such as magnesium, iron and calcium, are beneficial for maintaining bone health (Rondanelli et al., 2021; Tuomi et al., 1991; Varenna et al., 2007). Evidence have shown the relationship between overall dietary patterns, rather than just individual nutrients, and chronic diseases (Cavicchia et al., 2009; Shivappa et al., 2014a). Dietary inflammatory index (DII) included 45 types of food parameter was a quantitative method to assess the level of dietary inflammation emerged as time and conditions required, which can reflect the whole diet of an individual from the most anti-inflammatory to the most pro-inflammatory (Cao et al., 2023). Previous studies found that DII was associated with the risk or prognosis of several diseases (Garcia-Arellano et al., 2019; Zhang et al., 2022). Garcia-Arellano A et al. (Garcia-Arellano et al., 2019) reported that compared with the lowest category of DII, highest DII was significantly associated with an increase of 23 % in all-cause mortality. Zhang et al. (Zhang et al., 2022) shown that DII was associated with the risk of all-cause, cancer and CVD mortality in a linear manner. Dietary intervention might be a promising method in the prevention and treatment of most non-communicable diseases. However, the association between DII and all-cause mortality in osteoporosis patients with low bone mass remains unclear.

Herein, we explored the association between DII and all-cause mortality in patients with osteopenia or osteoporosis. This study aimed to lay a theoretical foundation for improving the prognosis of osteopenia or osteoporosis patient from the perspective of eating habits.

2. Methods

2.1. Study design and participates

Data of this study were extracted from the National Health and Nutrition Examination Survey (NHANES) (2007–2018). NHANES was a cross-sectional study conducted by National Centers for Health Statistics (NCHS) and aim to assessment the overall health and nutritional status of non-institutional population in the United States. The NHANES survey used complex, multistage, probability sampling methods based on broad population distributions. NHANES was a publicly available dataset and was approved by the NCHS Ethics Review Board, and all participants provided written informed consent. According to the Ethics Review Board of The Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University, retrospective cohort studies have been exempted from the ethical review.

In present study, 13,869 subjects aged $\geq$ 45 years old were extracted from the NHAES database 2007–2010, 2013–2014 and 2017–2018. Of these 13,869 subjects, 5,699 subjects had the diagnostic information of osteopenia or osteoporosis and with complete dietary intake data. 304 subjects missing DII assessment information and 14 subjects missing survival information were excluded from the study. Finally, 5,381 eligible patients were included for analysis of the association between DII and all-cause mortality risk in osteopenia or osteoporosis patients.

2.2. Outcome

In present study, all-cause mortality in osteoporosis or osteopenia patients was designed as the exposure variable including caused by malignant tumor, CVD, respiratory disease, Alzheimer's disease, diabetes, nephropathy-related diseases, accidental death, and other causes (Li et al., 2023). All-cause mortality derived from the records of the National Death Index (NDI) before 31 December 2019, which were linked with NHANES data.

2.3. Dietary intake and DII assessment

Dietary intake information was obtained from 24-h dietary recall interview. The interview was conducted through face-to-face communication of professional technicians at the Mobile Test Center. Each participant was asked to recall the type and amount of food, drink and dietary supplements consumed in the 24 h preceding the interviews from midnight to midnight. Then, the intake of each food component was estimated from the United States Department of Agriculture (USDA) Survey Nutrient Database (Zhang et al., 2022). DII were calculated using the average nutrient intake on the first day of the 24-h dietary recall information according to the protocol published by N. Shivappa et al. (Shivappa et al., 2014a). Twenty-eight food parameters in NHANES including carbohydrates, protein, total fat, alcohol, fiber, cholesterol, saturated fat, monounsaturated fatty acids (MUFAs), polyunsaturated fatty acids (PUFAs), niacin, vitamin A, B6, B12, C, D and E, thiamin, riboflavin, ferrum, magnesium, zinc, selenium, folic acid, β-carotene, caffeine, energy, n-3 fatty acids and n-6 fatty acids were calculated the DII score. The predictive ability based on less than 30 food parameters for DII calculated was reported in the previous studies (Shivappa et al., 2014b). In simple terms, the Z score was calculated by comparing the average and standard deviation of common dietary nutrients with the individual dietary intake assessed by 24-h dietary review method. Z score was converted to a percentile value, then the percentile value was doubled, and '1′ was subtracted for centralization (from −1 to + 1, centered on 0). Definitively, the value was multiplied by the inflammation effect score of the corresponding food nutrient presented in the previous literature to calculate each nutrient's DII score. The DII of all food nutrients were added to obtain each individual overall DII scores.

DII calculation formula (Jandari et al., 2023):

$$Z\ score = [(daily\ mean\ intake - global\ daily\ mean\ intake) / standard\ deviation].$$

$$Z\ score^1 = Z\ score \rightarrow (converted\ to\ a\ percentile\ score) \times 2 - 1.$$

$$DII = \sum Z\ score^1 \times the\ inflammatory\ effect\ score\ of\ each\ dietary\ nutrient.$$

As an objective tool to assess the body's overall dietary inflammatory potential, a higher DII indicated a more pro-inflammatory diet, while a lower DII score suggested an anti-inflammatory effect of diet. DII score was categorized into tertiles in present study.

2.4. Osteopenia and osteoporosis assessment

The study participants' bone mineral density (BMD) (g/cm^2) of femoral neck, trochanteric, intertrochanteric and total femoral areas were examined by dual-energy x-ray absorptiometry (DXA). According to the World Health Organization (WHO), we classify the bone health status into low BMD (osteopenia)/osteoporosis/normal. BMD at the femoral neck equal to or less than 2.5 standard deviations below the mean for a young person of the same sex was diagnostic of osteoporosis. Low BMD was reported as a T-score $<$ 1.0 and $>$ -2.5, while osteoporosis is defined as T-score $\leq$ -2.5. T-score was calculated using the formula: T-score = BMD $_{respondent}$ –mean BMD $_{reference\ group}$) /SD $_{reference\ group}$ (1994).

2.5. Potential covariates

The potential covariates included age, gender, race, education level, marital status, smoking, drinking, body mass index (BMI) and poverty-to-income ratio (PIR). Age, gender, race and marital status were obtained from self-reported demographic information. Race included Mexican American, other Hispanic, Non-Hispanic White, Non-Hispanic Black and Multi-Racial. Education level was assessed by the question "What is the highest grade or level of school you have completed or the highest degree you have received?" (less than 9th grade/9-11th grade, high school grad/GED or equivalent and some college or AA degree/college graduate or above) (Patel et al., 2019). "Smoking: cigarette use' questionnaire" was used to assess the smoking status. Never smoker was

defined as smoking less than 100 cigarettes in their whole life. Current smoker was classified as smoking less than 100 cigarettes in s/he whole life and still smoking when s/he answered this questionnaire. Former smoker was defined as smoking at least 100 cigarettes in their life, and had quit smoking when s/he answered this questionnaire (Shen et al., 2021). Drinking status was assessed by the question "Had at least 12 alcohol drinks?" (yes/or) (Gay et al., 2018). BMI was based on clinic-measured weight and height (kg/m^2) (Peterson et al., 2016).

Fracture, arthritis, hypertension, diabetes, dyslipidemia, CVD, chronic kidney disease (CKD) and cancer were defined by laboratory tests, self-reported and medication history. Hypertension was defined as systolic blood pressure (SBP) $\geq$ 130 mmHg, diastolic blood pressure (DBP) $\geq$ 80 mmHg, self-reported high blood pressure or taking blood pressure medication (Li and Shang, 2021). Diabetes was defined as total cholesterol (TC) $\geq$ 200 mg/dL (5.2 mmol/L), triglyceride (TG) $\geq$ 150 mg/dL (1.7 mmol/L), low-density lipoprotein cholesterol (LDL-C) $\geq$ 130 mg/dL (3.4 mmol/L), high-density lipoprotein cholesterol (HDL-C) $\leq$ 40 mg/dL (1.0 mmol/L), self-reported hypercholesterolemia or receiving cholesterol-lowering therapy (Flack and Adekola, 2020). CVD was assessed by the question "Ever told you had angina, heart failure, heart attack, coronary heart disease, stroke or congestive heart failure?" (Gay et al., 2018). Equations to estimate glomerular filtration rate (eGFR) < 60 ml/min/1.73 m^2 was defined as CKD. The eGFR calculation formula: eGFR=141 * min (Scr/k,1) α * max (Scr/k,1)1.029 * 0.993 age *1.108 (if female) *1.159 (if black), k is 0.7 for females and 0.9 for males, α is −0.329 for females and −0.411 for males, min indicates then minimum of Scr/k or 1, and max indicates the maximum of Scr/k or 1 (Levey et al., 2009). The history of cancer was assessed by the question "Ever told you had cancer or malignancy?" (Seo and Strauss, 2020). The family history of osteoporosis was assessed by the question "Has a doctor or any other health professional ever told your blood relatives including living and decreased that they had osteoporosis or brittle bones?"(Tang et al., 2022). The history of fracture was assessed by the question "Has a doctor ever told you or your blood relatives that you/s/he had broken or fractured hip?"(Xiao et al., 2022). The history of arthritis was assessed by the question "Has a doctor ever told you or your blood relatives that you/s/he had arthritis?"(Sule and Fontaine, 2018). Physical activity was expressed as the metabolic equivalent task (MET) and calculated as follows: physical activity (met·min/week) = recommended MET×exercise time for corresponding activities (min/day) × the number of exercise days per week (day) (Mendes et al., 2018).

2.6. Statistical analysis

Continuous data were expressed as mean and standard error (S.E.), and the weighted *t*-test was used for comparison between groups. Categorical data were described by case number and percentage [N (%)], and comparisons between groups used the weighted χ^2 test. Multivariate imputation by chained equations (MICE) was used to missing data imputation. Sensitivity analysis was performed before and after missing data imputation (Table S1). Weighted univariable and multivariable cox proportional hazard models were used to explore the association between DII and all-cause mortality in osteoporosis or osteopenia patients, with hazard ratios (HRs) and 95 % confidence intervals (CIs). Model 1 was a crude model. Model 2 was adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, CVD, cancer, regularity of menstruation, BMI, CKD, white blood cell (WBC), uric acid and glucocorticoid.

SDMVPSU, SDMVSTRA and WTMEC2TR were weighted for the final sample size using the proc surveyfreq in SAS software. *P*-value < 0.05 was considered statistically significant. All statistical analyses were performed by SAS 9.4 (SAS Institute Inc., Cary, NC, USA).

3. Results

3.1. Characteristics of study population

The flow chart of population screening was shown in Fig. 1. After the screening, total 5,699 patients diagnosed as osteopenia or osteoporosis were screened. Among them, 304 patients missing DII assessment information and 14 patients missing the survival data were excluded. Finally, 5,381 patients were included, with the mean age of 63.19 (0.21) years old. US NHANES documented 1,286 deaths over a mean follow-up of 92.12 months. The characteristics of study patients were shown in Table 1. The proportion of patients with high DII ($\geq$1.78) in all-cause mortality group was higher than that in survival group (39.43 % *vs.* 31.55 %). Differences were found in age, race, the level of education, physical activity, WBC, marital status, AST, uric acid, BMI and DII, smoking, drinking, the history of fracture, arthritis, hypertension, diabetes, CVD, cancer and CKD, whether menstruation regular, the use of bone resorption inhibitors and glucocorticoid between two groups (P<0.05).

3.2. Association between DII and all-cause mortality

Table 2 shows the association between DII and all-cause mortality in osteoporosis or osteopenia patients. After adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, CVD, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid in model II, compared with low DII score group, all-cause mortality was increased, respectively, by 15 % and 28 % at the DII score of 0.30–1.78 (HR=1.15, 95 %CI: 0.96–1.37) and $\geq$ 1.78 (HR=1.28, 95 %CI: 1.10–1.48).

Using restricted cubic splines (RCS), a non-linear relationship between DII and the all-cause mortality of osteoporosis or osteopenia patients was explored, as exhibited in Fig. 2. The RCS curves suggested that the nonlinear association between DII and the all-cause mortality risk was not significant (P for nonlinear = 0.779).

3.3. Association between DII and all-cause mortality stratified by different age, gender, and history of CVD, hypertension, diabetes and CKD

The subgroup analyses in Table 3 suggested that the association of DII and all-cause mortality was consistent and reliable after being stratified by age, gender, hypertension, diabetes, CVD and CKD. All adjusted model II suggested that, compared with the low DII score (<0.30), high DII group ($\geq$1.78) was still associated with the high risk of all-cause mortality in patients with osteoporosis or osteopenia, especially among patients with male (HR=1.38, 95 %CI: 1.06–1.78), aged < 60 years (HR=1.49, 95 %CI: 1.09–2.02) and without the history of CVD (HR=1.30, 95 %CI: 1.03–1.65), diabetes (HR=1.27, 95 %CI: 1.06–1.52) and CKD (HR=1.28, 95 %CI: 1.03–1.58) (all P<0.05). While, no significant association between DII and all-cause mortality was observed in female, aged $\geq$ 60 years old, and with the history of CVD, diabetes and CKD (all P>0.05). Moreover, we found no interaction effect between DII and each subgroup (all P>0.05) (Table 4).

4. Discussion

This study provided evidence that DII was related to the all-cause mortality in a representative sample of U.S. osteoporosis or osteopenia patients based on the NHANES database. After adjustment for potential confounders, high DII ($\geq$1.78) was associated with higher risk of all-cause mortality among osteoporosis or osteopenia patients, especially among patients with male, aged < 60 years, without the history of CVD, diabetes and CKD.

Osteoporosis can be viewed as a systemic inflammatory status and inflammation is the driving pathophysiological process in multiple

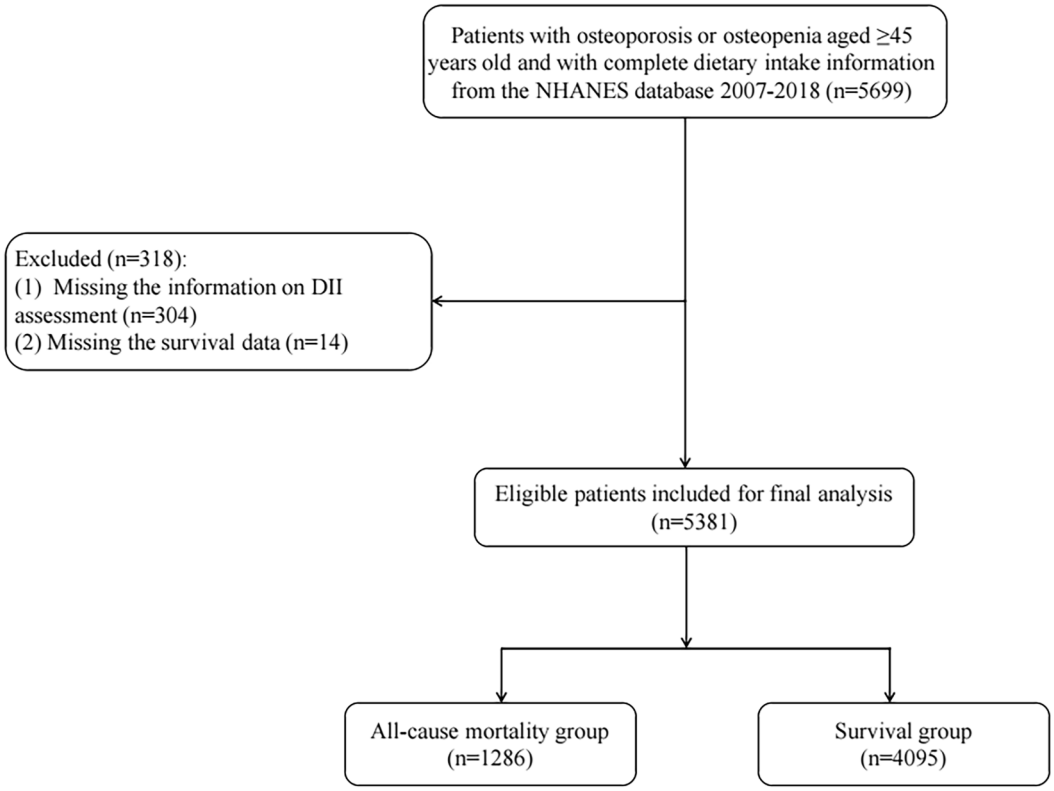

Fig. 1. The flow chart of population screening.

chronic diseases (Singh et al., 2019). Chronic inflammation was characterized by the continuous presence of pro-inflammatory cytokines through increased blood flow during tissue injury due to histamine release from damaged mast cells (Keibel et al., 2009). Human and animal studies suggested that pro-inflammatory cytokines are major mediators of accelerated bone loss, including interleukin-1, tumor necrosis factor-alpha and interleukin-6 (Mundy, 2007). Several researches reported that inflammation-mediated osteoporosis and low bone mass increase the all-cause and cause-specific mortality in the general adult population (Domiciano et al., 2016; Mussolino and Armenian, 2007). Michael et al. (Mussolino and Armenian, 2007) found that compared with the highest quartile of BMD, subjects in the lowest quartile of BMD in total femur increased 53 % all-cause mortality in U.S. population aged 50 years and older. Diogo et al. (Domiciano et al., 2016) reported that participants at the lowest quartile of BMD in total hip displayed a 110 % increase in all-cause mortality, as compared with those participants at the highest quartile in Brazil population aged over 65 years old. Identifying modifiable factors contributing to osteoporosis and good health management of osteoporosis are important to improve the prognosis of prognosis.

The direct role of whole diet and various dietary components on inflammation has confirmed by previous studies (Barbaresko et al., 2013; Bordoni et al., 2017; Santos et al., 2013). However, there was no tool that could take the whole diet into account and evaluate its inflammatory potential until 2009. Researchers of public health from University of South Carolina have proposed a dietary tool called the DII that puts individuals into a continuum from maximally pro-inflammatory to maximally anti-inflammatory diet and further improved this quantitative assessment tool in 2014 (Cavicchia et al., 2009). There is a significant relationship between DII score and the pro-inflammatory effects of the dietary and vice versa, therefore suggesting that the anti-inflammatory effects are stronger the higher the DII score (Park et al., 2018). DII has been shown to be related to various chronic inflammation-related health outcomes including cancer incidence, all-cause and specific-cause mortality (Shivappa et al., 2016). Several studies mainly from Australia, Europe, Japan and America have shown an increased risk of CVD-related mortality associated with a pro-inflammatory diet (Gao et al., 2023; Ji et al., 2020). For instance, a large NHANES study focused on the general population in U.S. reported that all-cause and CVD-related mortality are linked to high DII scores, independently (Gao et al., 2023). Moreover, as of 2018, the association between a highest quartiles of DII scores (pro-inflammatory diet) and all-cause mortality seemed robust in two large Spanish cohorts, which also has been consistent with present study (Correa-Rodríguez et al., 2018; Flores et al., 2019). In our study, we found that DII score at highest quartiles has increased 28 % all-cause mortality among osteoporosis. Although fewer studies about dietary management in osteoporosis focused on DII, previous studies urged us to connect dietary intervention with DII in osteoporosis. Consumption of fresh fruits and vegetables may be beneficial for decreasing the levels of inflammation, while consumption red meat has been shown to be increase the inflammation (Almeida-de-Souza et al., 2018). Several dietary nutrients have been related to play a major role in the development of osteoporosis. Ensuring the adequate dietary calcium, vitamin D and protein intake, performing regular physical activity and giving up harmful life habits, such as excessive smoking and drinking may help to improve bone quality (Tucker, 2009).

In the subgroup analysis, we found that highest DII scores was associated with the high all-cause mortality among male, aged less than 65 years and without the history of CVD, diabetes and CKD. Osteoporosis in male was a common bone metabolism related disease but often overlooked disorder. Most male generally have stronger bones than female and have less bone loss during their whole lifetime. Male often fracture less than female, while they have a higher rate mortality of fracture (Vilaca et al., 2022). A prospective cohort from the Dubbo Osteoporosis Epidemiology Study focused on the female and male aged more than 60 years reported that there were 952 low-trauma fractures followed by 461 deaths (48.4 %), and in men, 343 fractures were

Table 1
Distribution of included characteristics of U.S. adults over 45 years old between 2007–2010, 2013–2014 and 2017–2018, National Health and Nutrition Examination Survey (n = 5,381).

Variables	Total (n = 5381)	Alive (n = 4095)	Death (n = 1286)	*P*
Age, years, Mean (S.E)	63.19 (0.21)	61.94 (0.22)	68.73 (0.48)	0<.001
Gender, n (%)				0.075
Male	2469 (42.42)	1783 (41.69)	686 (45.62)	
Female	2912 (57.58)	2312 (58.31)	600 (54.38)	
Race/Ethnicity-Recode, n (%)				0.009
Mexican American	723 (4.96)	583 (5.06)	140 (4.50)	
Other Hispanic	513 (3.97)	425 (4.30)	88 (2.49)	
Non-Hispanic White	2930 (78.46)	2103 (77.97)	827 (80.65)	
Non-Hispanic Black	673 (5.51)	494 (5.13)	179 (7.23)	
Other Race-Including Multi-Racial	542 (7.09)	490 (7.54)	52 (5.13)	
Education level, n (%)				0<.001
Less than 9th Grade/9-11th Grade (Includes 12th grade with no diploma)	1479 (16.66)	1044 (14.53)	435 (26.09)	
High school grad/GED or equivalent ;	1298 (25.35)	948 (24.58)	350 (28.76)	
Some college or AA degree/ college graduate or above	2604 (57.98)	2103 (60.89)	501 (45.15)	
PIR, n (%)				0.233
0–1	840 (9.22)	631 (8.87)	209 (10.79)	
≥1	4025 (82.82)	3047 (83.05)	978 (81.81)	
Unknown	516 (7.96)	417 (8.08)	99 (7.40)	
Marital status, n (%)				0<.001
Married	3027 (61.68)	2400 (64.11)	627 (50.97)	
Widowed	874 (13.05)	515 (9.96)	359 (26.72)	
Divorced	826 (15.15)	670 (15.95)	156 (11.65)	
Separated	154 (1.82)	121 (1.80)	33 (1.90)	
Never married	334 (5.47)	255 (5.24)	79 (6.48)	
Living with partner	166 (2.83)	134 (2.95)	32 (2.28)	
Smoking, n (%)				0<.001
Never smoker	2717 (52.37)	2202 (54.99)	515 (40.81)	
Former smoker	1733 (31.28)	1235 (30.01)	498 (36.89)	
Current smoker	931 (16.34)	658 (14.99)	273 (22.31)	
Drinking, n (%)				0<.001
No	1832 (28.27)	1374 (26.92)	458 (34.26)	
Yes	3549 (71.73)	2721 (73.08)	828 (65.74)	
Physical activity, met·min/ week, n (%)				0<.001
<750	1091 (20.61)	837 (20.71)	254 (20.19)	
≥750	2479 (51.77)	2021 (54.88)	458 (38.06)	
Unknown	1811 (27.62)	1237 (24.41)	574 (41.75)	
Fracture history, n (%)				0.030
No	4558 (82.89)	3509 (83.55)	1049 (79.97)	
Yes	823 (17.11)	586 (16.45)	237 (20.03)	
Arthritis, n (%)				0.004
No	3051 (57.57)	2398 (58.81)	653 (52.12)	
Yes	2330 (42.43)	1697 (41.19)	633 (47.88)	
Hypertension, n (%)				0<.001
No	1326 (30.48)	1121 (33.13)	205 (18.79)	
Yes	4055 (69.52)	2974 (66.87)	1081 (81.21)	
Diabetes, n (%)				0<.001
No	4061 (81.59)	3186 (83.46)	875 (73.36)	
Yes	1320 (18.41)	909 (16.54)	411 (26.64)	
CVD, n (%)				0<.001
No	3907 (77.02)	3166 (80.63)	741 (61.07)	
Yes	1474 (22.98)	929 (19.37)	545 (38.93)	
Dyslipidemia, n (%)				0.716
No	926 (17.35)	694 (17.25)	232 (17.82)	
Yes	4455 (82.65)	3401 (82.75)	1054 (82.18)	
Cancer, n (%)				0<.001
No	4421 (80.54)	3457 (82.05)	964 (73.86)	
Yes	960 (19.46)	638 (17.95)	322 (26.14)	
CKD, n (%)				0<.001
No	4127 (82.41)	3357 (86.06)	770 (66.32)	
Yes	1254 (17.59)	738 (13.94)	516 (33.68)	
WBC, 1000 cells/uL, Mean (S.E)	6.99 (0.05)	6.94 (0.05)	7.20 (0.08)	0.007
ALT, U/L, Mean (S.E)	23.29 (0.30)	23.29 (0.28)	23.30 (0.90)	0.989
AST, U/L, Mean (S.E)	25.20 (0.30)	24.49 (0.23)	28.33 (1.18)	0.002
Uric acid, mg/dL, Mean (S.E)	5.34 (0.03)	5.27 (0.03)	5.63 (0.04)	0<.001
Regularity of menstruation, n (%)				0.037
No	5210 (95.20)	3945 (94.76)	1265 (97.13)	
Yes	171 (4.80)	150 (5.24)	21 (2.87)	
BMI, kg/m^2, n (%)				0<.001
0–25	1947 (36.97)	1418 (35.70)	529 (42.58)	
≥25	3434 (63.03)	2677 (64.30)	757 (57.42)	
Bone resorption inhibitors, n (%)				0.004
No	5119 (95.16)	3915 (95.63)	1204 (93.06)	
Yes	262 (4.84)	180 (4.37)	82 (6.94)	
Glucocorticoid, n (%)				0.014
No	4933 (91.52)	3777 (92.01)	1156 (89.33)	
Yes	448 (8.48)	318 (7.99)	130 (10.67)	
Follow-up, Mean (S.E)	92.12 (2.58)	92.62 (2.83)	89.90 (3.01)	0.394
DII, n (%)				0<.001
<0.30	1522 (32.99)	1203 (34.37)	319 (26.88)	
0.30–1.78	1783 (34.00)	1371 (34.07)	412 (33.68)	
≥1.78	2076 (33.01)	1521 (31.55)	555 (39.43)	

S.E: standard error; PIR: poverty income ratio; met: metabolic equivalent; CVD: cardiovascular disease; CKD: chronic kidney stones; WBC: white blood cell; BMI: body mass index; ALT: alanine aminotransferase; AST: aspartate aminotransferase; DII: dietary inflammation index; P: P-values (come from Chi-Squared test and *t*-test).

Table 2
The association between DII score and all-cause mortality in patients with osteoporosis or osteopenia in NHANES 2007–2010, 2013–2014 and 2017–2018.

Variables	Model I		Model II	
	HR (95 %CI)	*P*	HR (95 %CI)	*P*
DII				
1: <0.30	Ref		Ref	
2: 0.30–1.78	1.23 (1.04–1.45)	0.015	1.15 (0.96–1.37)	0.138
3: ≥1.78	1.56 (1.34–1.80)	<0.001	1.28 (1.10–1.48)	0.001

Ref: reference; HR: hazard ratio; CI: confidence interval.
NHANES: National Health and Nutrition Examination Surveys; DII: dietary inflammation index.
Model I: crude model;
Model II: adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, CVD, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid.

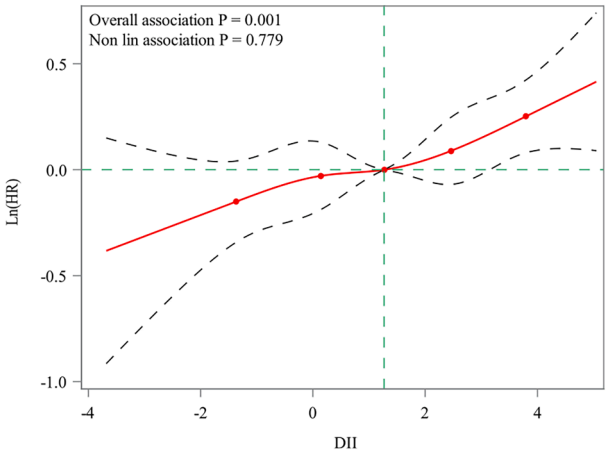

Fig. 2. Restricted cubic splines curve of DII and all-cause mortality of osteoporosis or osteopenia patients.

followed by 197 deaths (57.4 %) without 10 years (Bliuc et al., 2009). Lifestyle changes, adequate calcium, vitamin D intake and moderate physical activity to reduce inflammation are beneficial to management of bone health in male. The prevalence of osteoporosis is increasing with the aging population. As a silent disease, which no clinical manifestations until a facture occurs, the condition of osteoporosis is often underdiagnosed. Therefore, early lifestyle can predict the risk of future osteoporosis and a healthy diet and lifestyle in youth and middle age are the best means to maintain good bone mineral density. The poor quality of diet often occurred during the transition stage of adolescence and young adulthood (Winpenny et al., 2017). More and more evidence shown that fruit and vegetables can meditation the body's acid-base balance thereby protecting the bone health. The major harmful factors to bone health including an excessive intake of alcohol, sodium and caffeine have been reported in previous study (Cashman, 2007). Any diet and lifestyle that was considered to be anti-inflammatory in young adulthood and middle age may have beneficial effects on bone health and reduce the risk of osteoporosis in the later life. CVD, CKD and diabetes were the result of the accumulation of chronic inflammatory processes in the body and are commonly associated with osteoporosis (Libby, 2012). There was a clinical consensus that an unhealthy diet could

Table 3
Association between DII and all-cause mortality in osteoporosis or osteopenia patients stratified by age, gender, history of CVD, CKD, diabetes and hypertension in NHANES 2007–2010, 2013–2014 and 2017–2018.

Variables	Model II			
	HR (95 %CI)	*P*	HR (95 %CI)	*P*
Gender	Male (n = 2469)		Female (n = 2912)	
DII				
<0.30	Ref		Ref	
0.30–1.78	1.35 (1.03–1.78)[a]	0.032	0.97 (0.75–1.27)[a]	0.840
≥1.78	1.38 (1.06–1.78)[a]	0.016	1.17 (0.98–1.40)[a]	0.089
Age	≥65 (n = 2780)		<65 (n = 2601)	
DII				
<0.30	Ref		Ref	
0.30–1.78	1.04 (0.84–1.30)[b]	0.716	1.38 (0.96–1.99)[b]	0.078
≥1.78	1.19 (0.97–1.44)[b]	0.088	1.49 (1.09–2.02)[b]	0.013
CVD	NO (n = 3907)		YES (n = 1474)	
DII				
<0.30	Ref		Ref	
0.30–1.78	1.25 (0.94–1.66)[c]	0.122	0.99 (0.73–1.34)[c]	0.932
≥1.78	1.30 (1.03–1.65)[c]	0.031	1.26 (0.93–1.70)[c]	0.137
Hypertension	NO (n = 1326)		YES (n = 4055)	
DII				
<0.30	Ref		Ref	
0.30–1.78	1.17 (0.74–1.86)[d]	0.488	1.13 (0.91–1.40)[d]	0.263
≥1.78	1.53 (1.01–2.33)[d]	0.047	1.24 (1.02–1.50)[d]	0.031
Diabetes	NO (n = 4061)		YES (n = 1320)	
DII				
<0.30	Ref		Ref	
0.30–1.78	1.20 (0.97–1.49)[e]	0.085	1.01(0.78–1.55)[e]	0.580
≥1.78	1.27 (1.06–1.52)[e]	0.009	1.33 (0.95–1.87)[e]	0.100
CKD	NO (n = 4127)		YES (n = 1254)	
DII				
<0.30	Ref		Ref	
0.30–1.78	1.14 (0.89–1.48)[f]	0.298	1.12 (0.87–1.46)[f]	0.374
≥1.78	1.28 (1.03–1.58) [f]	0.024	1.26 (0.99–1.60)[f]	0.062

Ref: reference; HR: HR: hazard ratio; CI: confidence interval.
NHANES: National Health and Nutrition Examination Surveys; DII: dietary inflammation index; CVD: cardiovascular disease; CKD: chronic kidney stones.
Model I: crude model;
[a] : adjustment for age, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, CVD, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid;
[b] : adjustment for gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, CVD, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid;
[c] : adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid;
[d] : adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, diabetes, CVD, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid;
[e] : adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, CVD, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid;
[f] : adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, CVD, cancer, regularity of menstruation, BMI, WBC, uric acid and glucocorticoid.

increase the level of inflammation in the body, at least in part. Low DII scores, that was, healthy diet habits, may be beneficial to prevent the occurrence of chronic inflammatory disease events of CVD, CKD and diabetes, thereby reducing the incidence of osteoporosis.

The strengths of present study include novel finding of association between DII as an assessment tool for inflammatory in *vivo* and all-cause mortality in osteoporosis with large sample size and reliable mortality status. In addition, we accounted for complex survey design in statistical analysis, which was representative of the non-institutionalized civilian population in the US. Several limitations need caution in interpreting

Table 4
The interaction effect between DII and each subgroup.

Groups	Model II
	P
DII*Gender	0.6502
DII*Age	0.1163
DII*CVD	0.6869
DII*Hypertension	0.5457
DII*Diabetes	0.2663
DII*CKD	0.8679

DII: DII: dietary inflammation index; CVD: CVD: cardiovascular disease; CKD: chronic kidney stones.
Model II: adjustment for age, gender, race, education level, marital status, smoking, drinking, physical activity, fracture history, arthritis, hypertension, diabetes, CVD, cancer, regularity of menstruation, BMI, CKD, WBC, uric acid and glucocorticoid.

our findings. First, it was an observational study, so residual confounding cannot be excluded. However, various covariates have been altered to reduce confounding. Moreover, the dietary intake information was obtained from 24-h dietary recall interview and may be biased by recall. Finally, due to the limited of database, information on diet and drug use was collected in a single survey, and the impact of changes in diet and drug use on outcomes needs to be further explored in more large-scale studies.

5. Conclusion

High DII was associated with high odds of all-cause mortality in osteoporosis or osteopenia patients. Anti-inflammatory dietary intervention may have potential benefit on the health outcomes of patients with osteoporosis or osteopenia.

Funding

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

CRediT authorship contribution statement

Chenrong Ke: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Xiaolei Zhang:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Xiangyang Wang:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.pmedr.2024.102826.

References

Almeida-de-Souza, J., Santos, R., Lopes, L., Abreu, S., Moreira, C., Padrão, P., Mota, J., Moreira, P., 2018. Associations between fruit and vegetable variety and low-grade inflammation in Portuguese adolescents from LabMed Physical Activity Study. Eur. J. Nutr. 57, 2055–2068.
Anam, A.K., Insogna, K., 2021. Update on osteoporosis screening and management. Med. Clin. North Am. 105, 1117–1134.
Barbaresko, J., Koch, M., Schulze, M.B., Nöthlings, U., 2013. Dietary pattern analysis and biomarkers of low-grade inflammation: a systematic literature review. Nutr. Rev. 71, 511–527.
Bliuc, D., Nguyen, N.D., Milch, V.E., Nguyen, T.V., Eisman, J.A., Center, J.R., 2009. Mortality risk associated with low-trauma osteoporotic fracture and subsequent fracture in men and women. J. Am. Med. Assoc. 301, 513–521.
Bordoni, A., Danesi, F., Dardevet, D., Dupont, D., Fernandez, A.S., Gille, D., Nunes Dos Santos, C., Pinto, P., Re, R., et al., 2017. Dairy products and inflammation: a review of the clinical evidence. Crit. Rev. Food Sci. Nutr. 57, 2497–2525.
Cao, Y., Li, P., Zhang, Y., Qiu, M., Li, J., Ma, S., Yan, Y., Li, Y., Han, Y., 2023. Dietary inflammatory index and all-cause mortality in older adults with hypertension: results from NHANES. J. Clin. Med. 12.
Carlson, B.C., Robinson, W.A., Wanderman, N.R., Sebastian, A.S., Nassr, A., Freedman, B. A., Anderson, P.A., 2019. A review and clinical perspective of the impact of osteoporosis on the spine. Geriatr. Orthop. Surg. Rehabil. 10, 2151459319861591.
Cashman, K.D., 2007. Diet, nutrition, and bone health. J. Nutr. 137, 2507s–s2512.
Cavicchia, P.P., Steck, S.E., Hurley, T.G., Hussey, J.R., Ma, Y., Ockene, I.S., Hébert, J.R., 2009. A new dietary inflammatory index predicts interval changes in serum high-sensitivity C-reactive protein. J. Nutr. 139, 2365–2372.
Correa-Rodríguez, M., González-Jiménez, E., Rueda-Medina, B., Tovar-Gálvez, M.I., Ramírez-Vélez, R., Correa-Bautista, J.E., Schmidt-RioValle, J., 2018. Dietary inflammatory index and cardiovascular risk factors in Spanish children and adolescents. Res. Nurs. Health 41, 448–458.
Domiciano, D.S., Machado, L.G., Lopes, J.B., Figueiredo, C.P., Caparbo, V.F., Oliveira, R. M., Scazufca, M., McClung, M.R., Pereira, R.M., 2016. Bone mineral density and parathyroid hormone as independent risk factors for mortality in community-dwelling older adults: a population-based prospective cohort study in Brazil. The São Paulo Ageing & health (SPAH) Study. J. Bone Miner. Res. 31, 1146–1157.
Eckart, A., Struja, T., Kutz, A., Baumgartner, A., Baumgartner, T., Zurfluh, S., Neeser, O., Huber, A., Stanga, Z., et al., 2020. Relationship of nutritional status, inflammation, and serum albumin levels during acute illness: a prospective study. Am J Med 133: 713-22.e7.
Flack, J.M., Adekola, B., 2020. Blood pressure and the new ACC/AHA hypertension guidelines. Trends Cardiovasc. Med. 30, 160–164.
Flores, J.C., Gracia-Lavedan, E., Benavente, Y., Amiano, P., Romaguera, D., Costas, L., Robles, C., Gonzalez-Barca, E., de la Banda, E., et al., 2019. The dietary inflammatory index and chronic lymphocytic leukaemia in the MCC Spain study. Nutrients 12.
Gao, Y., Wang, Y., Zhang, D., Wu, T., Li, Q., 2023. The relationship between dietary inflammatory index and all-cause, cardiovascular disease-related, and cancer-related mortality. J. Multidiscip. Healthc. 16, 2543–2556.
Garcia-Arellano, A., Martínez-González, M.A., Ramallal, R., Salas-Salvadó, J., Hébert, J. R., Corella, D., Shivappa, N., Forga, L., Schröder, H., et al., 2019. Dietary inflammatory index and all-cause mortality in large cohorts: the SUN and PREDIMED studies. Clin. Nutr. 38, 1221–1231.
Gay, I.C., Tran, D.T., Paquette, D.W., 2018. Alcohol intake and periodontitis in adults aged ≥30 years: NHANES 2009–2012. J. Periodontol. 89, 625–634.
Gregson, C.L., Armstrong, D.J., Bowden, J., Cooper, C., Edwards, J., Gittoes, N.J.L., Harvey, N., Kanis, J., Leyland, S., et al., 2022. UK clinical guideline for the prevention and treatment of osteoporosis. Arch. Osteoporos. 17, 58.
Jandari, S., Rezvani, R., Yousefian, S., Mosalmanzadeh, N., Bagherniya, M., Soleimani, D., Mousavian, S.Z., Shivappa, N., Hébert, J.R., et al., 2023. The effect of low dietary inflammatory index score formula on inflammatory, metabolic, and clinical outcomes in critically ill traumatic brain injury patients: a single-blind randomized controlled pilot study. Food Sci. Nutr. 11, 3365–3375.
Ji, M., Hong, X., Chen, M., Chen, T., Wang, J., Zhang, N., 2020. Dietary inflammatory index and cardiovascular risk and mortality: a meta-analysis of cohort studies. Medicine (Baltimore) 99, e20303.
Keibel, A., Singh, V., Sharma, M.C., 2009. Inflammation, microenvironment, and the immune system in cancer progression. Curr. Pharm. Des. 15, 1949–1955.
Levey, A.S., Stevens, L.A., Schmid, C.H., Zhang, Y.L., Castro 3rd, A.F., Feldman, H.I., Kusek, J.W., Eggers, P., Van Lente, F., et al., 2009. A new equation to estimate glomerular filtration rate. Ann. Intern. Med. 150, 604–612.
Li, B., Chen, L., Hu, X., Tan, T., Yang, J., Bao, W., Rong, S., 2023. Association of serum uric acid with all-cause and cardiovascular mortality in diabetes. Diabetes Care 46, 425–433.
Li, C., Shang, S., 2021. Relationship between sleep and hypertension: findings from the NHANES (2007–2014). Int. J. Environ. Res. Public Health 18.
Libby, P., 2012. Inflammation in atherosclerosis. Arterioscler. Thromb. Vasc. Biol. 32, 2045–2051.
Livshits, G., Kalinkovich, A., 2022. Targeting chronic inflammation as a potential adjuvant therapy for osteoporosis. Life Sci. 306, 120847.
Loi, F., Córdova, L.A., Pajarinen, J., Lin, T.H., Yao, Z., Goodman, S.B., 2016. Inflammation, fracture and bone repair. Bone 86, 119–130.
Lorentzon, M., Nilsson, A.G., Johansson, H., Kanis, J.A., Mellström, D., Sundh, D., 2019. Extensive undertreatment of osteoporosis in older Swedish women. Osteoporos Int. 30, 1297–1305.

Mendes, M.A., da Silva, I., Ramires, V., Reichert, F., Martins, R., Ferreira, R., Tomasi, E., 2018. Metabolic equivalent of task (METs) thresholds as an indicator of physical activity intensity. PLoS One 13, e0200701.
Mundy, G.R., 2007. Osteoporosis and inflammation. Nutr. Rev. 65, S147–S151.
Mussolino, M.E., Armenian, H.K., 2007. Low bone mineral density, coronary heart disease, and stroke mortality in men and women: the Third National Health and Nutrition Examination Survey. Ann. Epidemiol. 17, 841–846.
Park, S.Y., Kang, M., Wilkens, L.R., Shvetsov, Y.B., Harmon, B.E., Shivappa, N., Wirth, M. D., Hébert, J.R., Haiman, C.A., et al., 2018. The dietary inflammatory index and all-cause, cardiovascular disease, and cancer mortality in the multiethnic cohort study. Nutrients 10.
Patel, J.S., Oh, Y., Rand, K.L., Wu, W., Cyders, M.A., Kroenke, K., Stewart, J.C., 2019. Measurement invariance of the patient health questionnaire-9 (PHQ-9) depression screener in U.S. adults across sex, race/ethnicity, and education level: NHANES 2005–2016. Depress. Anxiety 36, 813–823.
Peterson, C.M., Thomas, D.M., Blackburn, G.L., Heymsfield, S.B., 2016. Universal equation for estimating ideal body weight and body weight at any BMI. Am. J. Clin. Nutr. 103, 1197–1203.
Reid, I.R., Billington, E.O., 2022. Drug therapy for osteoporosis in older adults. Lancet 399, 1080–1092.
Rondanelli, M., Faliva, M.A., Tartara, A., Gasparri, C., Perna, S., Infantino, V., Riva, A., Petrangolini, G., Peroni, G., 2021. An update on magnesium and bone health. Biometals 34, 715–736.
Santos, S., Oliveira, A., Lopes, C., 2013. Systematic review of saturated fatty acids on inflammation and circulating levels of adipokines. Nutr. Res. 33, 687–695.
Seo, J.Y., Strauss, S.M., 2020. Association of cancer history and health care utilization among female immigrants using NHANES 2007–2016 data. J. Immigr. Minor. Health 22, 1208–1216.
Shen, Q., Xu, Q., Li, G., Ren, L., Zhang, Z., Zhang, Y., Zhong, Z., Li, X., Wang, Q., 2021. Joint effect of 25-hydroxyvitamin D and secondhand smoke exposure on hypertension in non-smoking women of childbearing age: NHANES 2007–2014. Environ. Health 20, 117.
Shivappa, N., Steck, S.E., Hurley, T.G., Hussey, J.R., Hébert, J.R., 2014a. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr. 17, 1689–1696.
Shivappa, N., Steck, S.E., Hurley, T.G., Hussey, J.R., Ma, Y., Ockene, I.S., Tabung, F., Hébert, J.R., 2014b. A population-based dietary inflammatory index predicts levels of C-reactive protein in the seasonal variation of blood cholesterol study (SEASONS). Public Health Nutr. 17, 1825–1833.
Shivappa, N., Blair, C.K., Prizment, A.E., Jacobs Jr., D.R., Steck, S.E., Hébert, J.R., 2016. Association between inflammatory potential of diet and mortality in the Iowa Women's Health study. Eur. J. Nutr. 55, 1491–1502.
Singh, N., Baby, D., Rajguru, J.P., Patil, P.B., Thakkannavar, S.S., Pujari, V.B., 2019. Inflammation and cancer. Ann. Afr. Med. 18, 121–126.
Sule, S., Fontaine, K., 2018. Metabolic syndrome in adults with a history of juvenile arthritis. Open Access Rheumatol. 10, 67–72.
Tang, Y., Liu, Z., Wang, S., Yi, Q., Xia, Y., Geng, B., 2022. Development and validation of a novel screening tool for osteoporosis in older US adults: the NHANES cross-sectional study. Endocrine 76, 446–456.
Tucker, K.L., 2009. Osteoporosis prevention and nutrition. Curr. Osteoporos. Rep. 7, 111–117.
Tuomi, T., Huuskonen, M.S., Tammilehto, L., Vanhala, E., Virtamo, M., 1991. Occupational exposure to asbestos as evaluated from work histories and analysis of lung tissues from patients with mesothelioma. Br. J. Ind. Med. 48, 48–52.
Varenna, M., Binelli, L., Casari, S., Zucchi, F., Sinigaglia, L., 2007. Effects of dietary calcium intake on body weight and prevalence of osteoporosis in early postmenopausal women. Am. J. Clin. Nutr. 86, 639–644.
Vilaca, T., Eastell, R., Schini, M., 2022. Osteoporosis in Men. Lancet Diabetes Endocrinol 10, 273–283.
Wieringa, F.T., Thurnham, D.I., 2023. Inflammation, biomarkers, and real nutritional status. J. Nutr. 153, 605–607.
Winpenny, E.M., Penney, T.L., Corder, K., White, M., van Sluijs, E.M.F., 2017. Change in diet in the period from adolescence to early adulthood: a systematic scoping review of longitudinal studies. Int. J. Behav. Nutr. Phys. Act. 14, 60.
Wooltorton, E., 2006. Osteoporosis treatment: raloxifene (Evista) and stroke mortality. CMAJ 175, 147.
Wright, N.C., Looker, A.C., Saag, K.G., Curtis, J.R., Delzell, E.S., Randall, S., Dawson-Hughes, B., 2014. The recent prevalence of osteoporosis and low bone mass in the United States based on bone mineral density at the femoral neck or lumbar spine. J. Bone Miner. Res. 29, 2520–2526.
Xiao, S., Zhou, Y., Wu, Q., Wang, X., Hu, Y., Pan, Q., Liu, Q., Liu, A., Liu, J., et al., 2022. Prevalence of cardiovascular diseases in relation to total bone mineral density and prevalent fractures: a population-based cross-sectional study. Nutr. Metab. Cardiovasc. Dis. 32, 134–141.
Zhang, J., Feng, Y., Yang, X., Li, Y., Wu, Y., Yuan, L., Li, T., Hu, H., Li, X., et al., 2022. Dose-response association of dietary inflammatory potential with all-cause and cause-specific mortality. Adv. Nutr. 13, 1834–1845.
Zhang, C., Ren, W., Li, M., Wang, W., Sun, C., Liu, L., Fang, Y., Liu, L., Yang, X., et al., 2022. Association between the children's dietary inflammatory index (C-DII) and markers of inflammation and oxidative stress among children and adolescents: NHANES 2015–2018. Front. Nutr. 9, 894966.