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The association between diverse dietary quality measures and the presence of acute or chronic pain in the UK Biobank

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ABSTRACT

Chronic pain affects approximately 20% of adults worldwide and is a leading cause of disability. Emerging evidence suggest that nutrition may influence pain perception. However, the association between dietary quality and various pain conditions remains underexplored. This study examines associations between multiple dietary indices and the presence of acute and chronic pain analysing data from 55,721 UK Biobank participants. Multinomial logistic regression models were used to assess associations between diet quality (Dietary Inflammatory Index [DII], Healthy Diet Index [HDI], Healthful Plant-Based Diet Index [hPDI], Mediterranean Diet Score [MDS], and Eat-Lancet Diet Score [EATL]) and presence of acute or chronic pain versus pain-free individuals. Subgroup analyses examined chronic pain by number of pain sites and pain types. In fully adjusted models, higher hPDI (OR: 0.993, 95% CI: 0.989–0.997), EATL (OR: 0.969, 95% CI: 0.955–0.983), and MDS (OR: 0.981, 95% CI: 0.969–0.992) scores were positively associated with lower odds of having chronic pain, whereas only hPDI was linked to lower odds of having acute pain. Subgroup analyses showed that hPDI and EATL were consistently associated with lower odds of MSK and more localised pain while lower MDS and higher DII scores were positively associated with increased odds of widespread and non-MSK pain. This population-based study demonstrates an association between diet quality and chronic pain and extends the existing literature by showing that multiple dietary indices capture distinct aspects of this relationship. The results highlight the importance of considering different dietary indices and pain subtypes when evaluating diet-pain relationships.

Perspective: This large-scale study reveals that diverse dietary patterns associate uniquely with the risk of chronic pain and its distinct subtypes. Distinct dietary indices capture unique nutritional dimensions associated with pain type and distribution, underscoring the importance of diet and precision in understanding and managing chronic pain.

Introduction

Chronic pain affects approximately 20% of the global population and is a leading cause of disability, reduced quality of life, and financial burden.[1] A multimodal lifestyle approach, incorporating factors such as physical activity, sleep, alcohol consumption, smoking, and nutrition has shown promise.[2] Emerging evidence suggests dietary patterns may influence pain perception through mechanisms including

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neuroinflammation, oxidative stress, glucose metabolism, epigenics changes, brain-gut axis, and effects on overweight/obesity.[3] However, the diet–pain relationship remains complex, requiring further study to identify patterns that may aid in prevention or improvement.

Over the past decade, an increasing number of pre-clinical, clinical, experimental, and observational studies have examined the relationship between dietary intake and pain, with particular attention to plant-based, anti-inflammatory, pro-inflammatory, Western, calorie restriction, and elimination diets, as well as specific nutrient intakes.[4,5] For example, a population-based Mendelian randomization study identified a causal relationship between higher intake of alcohol, salt, pork, and poultry and the risk of chronic pain.[6] Additionally, higher dietary inflammatory scores have been associated with increased pain sensitivity in individuals with chronic low back pain.[7] Conversely, adherence to plant-based or Mediterranean diets, or higher intake of magnesium, antioxidants, and vitamin D, has been linked to improved chronic pain outcomes.[4,5]

Existing research is often limited by being observational, not having well enough characterised populations in terms of confounders, small sample sizes, and lack of an appropriate comparison group, and/or inconsistent dietary assessments. Historically there has been a predominant focus on estimating associations with individual nutrients as opposed to comprehensive dietary patterns and such approaches would potentially overlook the synergistic effects of foods and their components.[8] Different dietary indices have diverse methodologies that capture distinct aspects of dietary quality. These include measures of inflammatory potential (Dietary Inflammatory Index – DII), adherence to specific diets (Mediterranean Diet Score – MDS), food group emphasis (healthful Plant-Based Diet Index – hPDI), or alignment with dietary guidelines (Healthy Diet Index – HDI; Eat-Lancet Diet Score – EATL).[9] To date, existing research has relied on a single dietary index, which may not adequately reflect the complexity of diet–pain interactions.[4] Therefore, investigating multiple dietary indices within the same study may offer a more comprehensive understanding of the utility of distinct dietary patterns in relation to chronic pain.

To address these gaps, this study uses data from the UK Biobank,[10] to examine the association between dietary patterns and both acute and chronic pain. The UK Biobank provides a unique opportunity due to its large sample size, breadth of phenotypic data and diversity in lifestyle factors. Also, while UK Biobank is limited in ethnic diversity, it provides extensive data on diet, pain characteristics, confounders, and subgroups. Therefore, this cross-sectional analysis explores associations between dietary patterns, measured using five different dietary indices (DII, HDI, hPDI, MDS, and EATL), and the presence of acute and chronic pain, with comparisons made to pain-free individuals. Additionally, subgroup analyses will also assess chronic pain by number of pain sites (including localised versus widespread) and pain type (i.e., musculoskeletal (MSK) vs non-MSK pain).

Methods

Study design, settings, and population

This cross-sectional study used data from UK Biobank (Application Number: 179513), a large-scale biomedical database and research resource containing genetic, lifestyle, and health information from 500,000 UK participants aged 40–69 years recruited between 2006 and 2010. Participants attended one of 22 assessment centres across the UK, where they provided health and lifestyle information, underwent physical assessments, and contributed biological samples. UK Biobank was approved by the Northwest Multi-Center Research Ethics Committee [10] and obtained consent obtained from each participant prior to data collection. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline for cross-sectional studies was followed to ensure transparent and accurate reporting.[11]

Although the UK Biobank study began in 2006, the Oxford WebQ, a web-based 24-hour dietary assessment tool was introduced in April 2009 to collect dietary intake data. Therefore, we included only participants who had dietary intake data from the baseline assessment between April 2009 and September 2010. Additionally, participants were excluded if they were pregnant at the time of participation, had missing data for pain, diet, covariates, or were not following their usual diet on the day they completed the dietary diary due to illness, fasting, or other unspecified reasons.

Cohort selection and subgroup identification

This study included three main groups defined as chronic pain, acute pain, and pain-free participants. To define pain groups, responses to questions asked using a touchscreen during the UK-Biobank baseline visit were analysed. Participants were asked the following two questions to assess their pain experience. The first question was: “In the last month, have you experienced any of the following that interfered with your usual activities? (You can select more than one answer).” The response options included “headache”, “facial pain”, “neck or shoulder pain”, “back pain”, “stomach or abdominal pain”, “hip pain”, “knee pain”, “pain all over the body”, “none”, or “prefer not to answer”. The second question was: “For each type of pain selected, have you had that pain for more than three months?”. The response options were “Yes”, “No”, “Do not know”, or “Prefer not to answer”.

Those who reported experiencing pain in any body location and answered “Yes” to having it for more than three months were classified into the chronic pain group. Participants who selected one or more body locations but answered “No” to the three-month pain question(s) were assigned to the acute pain group. Lastly, individuals who reported no pain in response to the first question were categorized as pain-free.

Additionally, we conducted two subgroup analyses within the chronic pain group. The first subgrouping was based on the number of pain sites. Participants were classified as having widespread pain if they selected “pain all over the body”, single-site pain if they reported only one pain condition, multisite (2–3) if they reported pain in two to three locations, and multisite (>3) if they had pain in more than three locations.[12]

The second subgrouping classified participants based on the pain type. Those who reported pain in the neck/shoulder, back, hip, knee, or widespread pain conditions were categorized as having musculoskeletal (MSK) pain. Participants who selected stomach/abdominal pain, headaches, or facial pain were classified as having non-MSK pain. Individuals who reported at least one MSK pain condition and one non-MSK pain condition were categorized as having mixed pain.[13]

Dietary intake analysis and diet quality assessment

The Oxford WebQ, a web-based 24-hour dietary assessment tool, was used to gather data on dietary intake and was completed by participants using a touchscreen at assessment centers. This questionnaire recorded the frequency of consumption for 206 common foods and 32 types of beverages over the previous 24 h. It has been validated against traditional interviewer-administered multiple-pass 24-hour dietary recalls.[14] Energy and nutrient intakes were estimated by combining the reported frequency of consumption for each food or beverage item with its standard portion size and associated nutrient content.[15,16]

Based on the food and beverage intake information collected using the Oxford WebQ, five dietary indices were calculated: Dietary Inflammatory Index (DII), Healthy Diet Index (HDI), Healthful Plant-Based Diet Index (hPDI), Mediterranean Diet Score (MDS), and Eat-Lancet Diet Score (EATL).

DII: The DII is used to assess the inflammatory potential of dietary patterns. It was developed based on 45 dietary components identified as significantly influencing systemic inflammation, as determined from an analysis of 6500 published studies on six inflammatory cytokines, including C-reactive protein, TNF-alpha, and interleukins (IL) 1b, IL4,

IL6, and IL10[17]. This study incorporates 30 food items available in the UK Biobank.[18] A complete list of these items can be found in the supplementary file (Supplementary Table S1). Detailed explanation of the DII calculation has been explained elsewhere.[17] First, individual intake was standardized using Z-scores, adjusted to percentile scores, and scaled between −1 (anti-inflammatory) and +1 (pro-inflammatory). Each score was then weighted by its specific inflammatory effect. The overall DII score was obtained by summing the scores of all 30 food parameters. Higher scores indicated a more pro-inflammatory diet.

HDI: The HDI consists of 12 components reflecting adherence to the WHO's dietary recommendations published in 1990 and was developed by Maynard et al.[19] However, in this study, the cholesterol component was omitted because cholesterol intake is not included in the 2020 WHO recommendations and corresponding intake data were unavailable in the UK Biobank, resulting in an 11-item HDI consistent with the WHO's 2020 dietary guidelines.[20] These components include saturated fatty acids, polyunsaturated fatty acids, protein, total carbohydrates, dietary fiber, fruits and vegetables, pulses and nuts, total non-milk extrinsic sugars, fish, red meat and meat products, and calcium. Each component was scored based on specific cut-off values, as detailed in the supplementary file (Supplementary Table S2). A higher score indicated a higher diet quality. The total score varied from 0 to 11.

hPDI: In this study, the hPDI methodology outlined in previous research was followed.[21] The 17-item hPDI version was used, excluding vegetable oil intake due to its unavailability in the UK Biobank. Food components were categorized into sex-specific quintiles, with higher scores (1−5) assigned to healthy components—such as whole grains, fruits, vegetables, nuts, legumes, vegetarian protein alternatives, tea, and coffee. In contrast, lower scores were given to unhealthy components, including fruit juices, refined grains, potatoes, sugar-sweetened beverages, sweets and desserts, animal fat, dairy, eggs, fish and seafood, meat, and other animal-based foods (Supplementary Table S3). Higher scores indicated greater adherence to a healthy plant-based diet and higher diet quality. The total hPDI score ranged from 17 to 85.

EATL: The EATL, proposed by the EAT-Lancet Commission, is a planetary healthy diet designed to foster a sustainable balance between human nutrition and environmental impact.[22] The EATL was developed based on 14 key dietary recommendations, including rice/wheat/corn, potatoes, all vegetables, all fruits, milk and dairy products, red meat, poultry, eggs, fish, legumes, soy foods, nuts, unsaturated fats, and sweeteners. Each dietary component was assigned a score of 0 or 1, depending on whether it met the specified criteria. The criteria and scoring system for the EATL are detailed in the supplementary file (Supplementary Table S4). Higher scores indicated a higher diet quality and greater adherence to the EAT-Lancet dietary recommendations. The total EATL score ranged from 0 to 14.

MDS: In this study, the 9-item MDS developed by Trichopoulou et al. was utilised [23] This score includes nine dietary components: vegetables, legumes, fruits and nuts, cereals, fish and seafood, the monounsaturated-to-saturated fat ratio, dairy products, meat, and alcohol. The total MDS score was calculated based on sex-specific median intake values. Participants received a score of 1 if their intake exceeded the median for vegetables, legumes, fruits and nuts, cereals, fish and seafood, and the monounsaturated-to-saturated fat ratio. Conversely, a score of 1 was assigned for dairy and meat consumption below the median. For alcohol, participants received a score of 1 if their intake ranged between 0 and 2 units per day (Supplementary Table S5). Higher scores indicated greater adherence to the Mediterranean diet. The total MDS score varied from 0 to 9.

Covariate selection guided by directed acyclic graph

At the baseline assessment participants completed a touchscreen questionnaire. The recorded variables included age, sex, ethnicity (white, black, Asian, mixed, other), education level, physical inactivity (hours), sleep duration (hours), insomnia (never/rarely, sometimes, usually), smoking status (never, previous, current), and alcohol intake (special occasions only, one to three times a month, once or twice a week, three or four times a week, daily or almost daily).

Physical activity level was also collected via the baseline touchscreen questionnaire. It was derived using the International Physical Activity Questionnare (IPAQ) algorithm and categorised as low, moderate, or high physical activity according to the standard scoring.[24] Missing responses weer present due to "do not know" or "prefer not to answer" options. In line with the IPAQ scoring rules, incomplete responses to the time and day-based components resulted in missing classifications.[24]

Additionally, socioeconomic status was assessed using the Townsend Deprivation Index, an area-based measure derived from participants' residential postal codes, incorporating data on unemployment, car ownership, home ownership, and household overcrowding. Body mass index was also calculated based on weight and height measurements taken at the assessment centre.

Lastly, to account for potential confounding by chronic disease, multimorbidity was included as a covariate. Multimorbidity was determined by counting long term conditions (LTCs) a commonly used and validated approach in epidemiological studies to capture overall disease burden.[25] As the standard list includes 43 LTCs, conditions related to pain were excluded, as pain is a primary outcome in this study. As a result, 42 LTCs were included, with full details provided in Supplementary File (Supplementary Table S6).

A directed acyclic graph (DAG) was constructed to explicitly visualize our hypothesized causal relationships and identify potential confounding pathways between dietary patterns and pain outcomes (Supplementary Figure S1). This DAG guided the selection of covariates for adjustment in our statistical models. The DAG was created using DAGitty software (version 3.1.2.). The inclusion of covariates was informed by prior literature and theoretical considerations regarding their potential to confound the relationship between diet and chronic pain.

Data analysis

All analyses were conducted using Python (version 3.9.12) and R (version 4.3.3) on the UK Biobank Research Analysis Platform. Baseline characteristics were summarized as means and standard deviations for continuous variables, and as percentages for categorical variables. Group differences were assessed using one-way ANOVA for continuous variables and chi-square tests for categorical variables. Due to the large sample size, formal normality tests were considered overly sensitive and therefore not conducted. Instead, normality assumptions for ANOVA were assessed visually using Q-Q plots and density plots to detect any severe deviations, extreme outliers, or pronounced skewness.

Multinomial logistic regression was used to examine the association between diet quality and the presence of chronic and acute pain, with pain-free individuals as the reference group. Three models were constructed: Model 0 (unadjusted), Model 1 (adjusted for age, sex, ethnicity, education level, and Townsend Deprivation Index), and Model 2 (further adjusted for body mass index, physical activity and inactivity levels, smoking status, alcohol consumption, sleep duration, insomnia, and multimorbidity). Collinearity between covariates was assessed prior to model fitting.

Two subgroup analyses were performed among individuals with chronic pain. The first categorized participants based on the number of pain sites while the second classified them based on pain type (MSK, non-MSK, and mixed pain). Multinomial logistic regression was applied in both analyses, using the same covariate adjustments as in the main models. Results were presented as odds ratios (OR) with 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant for all analyses.

Sensitivity analyses

Given high missingness for physical activity (n=13,291), this variable was coded as an additional factor level in the main models, as in prior studies dies.[26,27] Sensitivity analyses were conducted excluding participants with missing physical activity to assess robustness.

Results

Characteristics of participants

Among the 502,161 participants in the UK Biobank study, a total of 55,721 individuals who had baseline dietary data and met the eligibility criteria were included in the final analysis. Participants were excluded for reasons including pregnancy, irregular dietary patterns due to fasting or illness, and missing or invalid data entries (see Fig. 1). Specifically, from the full cohort (502,161), participants with baseline dietary dairy were selected (70,655). Next participants based on pregnancy (confirmed or unsure) and based on irregular dietary patterns were excluded leading to an additional 12,847 exclusions. The full statistical regression models (one for each dietary exposure type) required fourteen variables in their estimation (twelve covariates, one exposure and one target variable). Excluding the physical activity covariate, we chose complete cases from the remaining 57,808 individuals leading to the removal of an additional 254 participants. The final exclusion was based on those variables where "Prefer not to answer" was the registered response. Specifically, the variables where such responses were observed were the Pain outcome response, alcohol intake, smoking status, insomnia, sleep duration, employment status, ethnicity, physical inactivity, and level of qualification. This criterion filtered out an additional 1833 individuals leading to a final 55,721 participants. Since physical activity was a key variable in our analysis and because of its

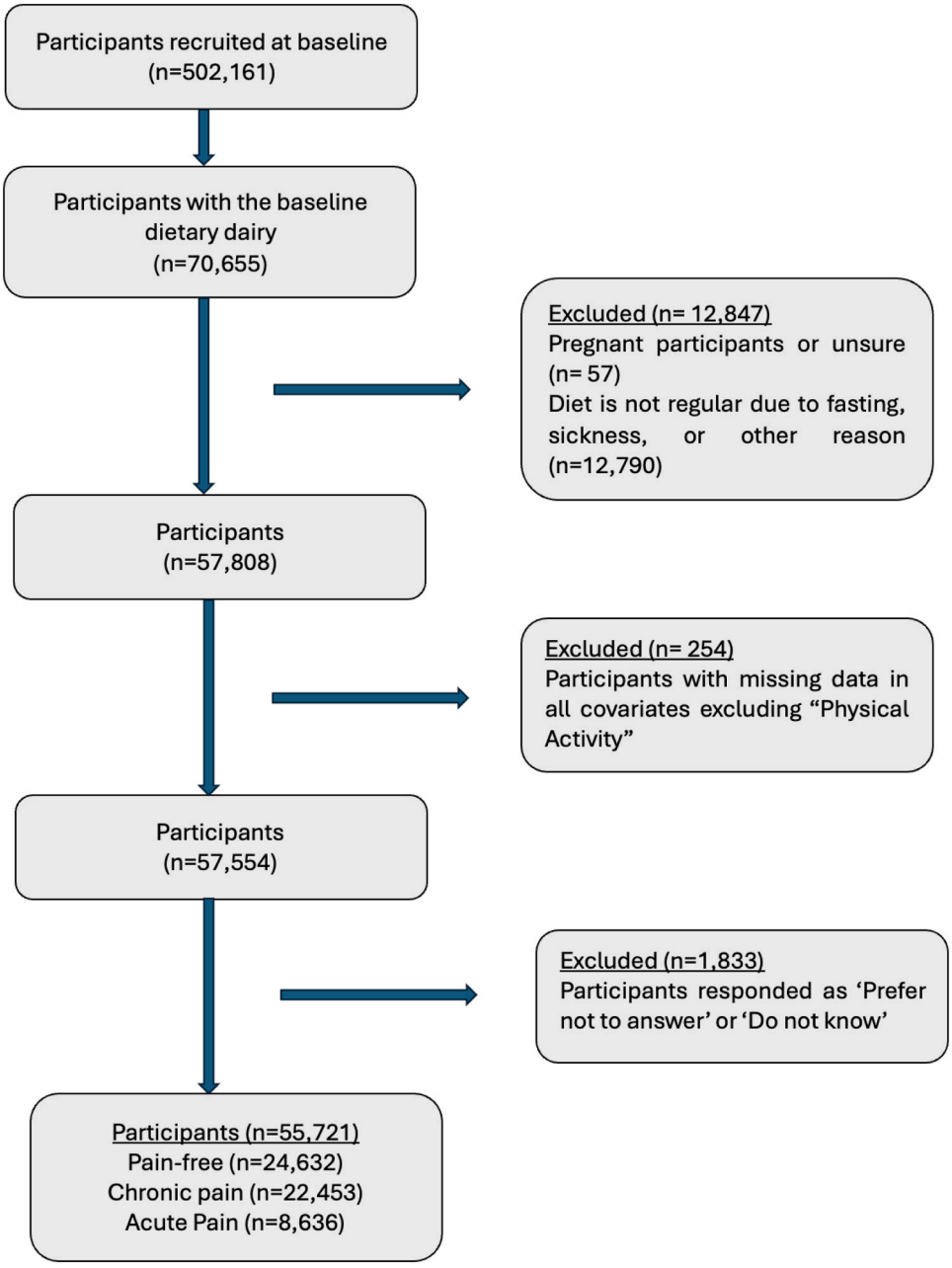

Fig. 1. Flow chart of the study. PF, Pain-free individuals; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index.

proportionately large missingness rate, we coded its missing values (n=13,291) as an additional factor level in our regression model. This rationale has been followed in other closely related studies.[26,27]

Of the 55,721 eligible participants, 22,453 (40.3%) reported experiencing chronic pain, 8636 (15.5%) had acute pain, and 24,632 (44.2%) were pain-free. The mean age of the participants was 56.4 years (SD = 8.14), with a mean BMI of 27.1 kg/m^2 (SD = 4.7). Females comprised 54.2% of the study population. Baseline characteristics for each group are presented in Table 1. Individuals with chronic pain, compared to those with acute pain and those pain-free individuals, were predominantly female (58.1 %), had lower socioeconomic status as measured by the Townsend Deprivation Index (mean = −1.18, SD = 2.89), were less likely to have completed a college or university degree and overall tended to have lower educational attainment, tended to have a higher BMI (mean = 27.8 kg/m,^{2} SD = 5.15), demonstrated lower levels of physically activity (mean = 4.12 h/day, SD = 2.23), reported less sleep hours (mean = 7.09 h/day, SD = 1.14), reported higher levels of insomnia, had more comorbidities, and were more likely to smoke (Table 1).

Pain and dietary quality

In a multinomial logistic regression analysis, the unadjusted results (Model 0) indicated that four diet quality indices HDI, hPDI, MDS, and the EATL were associated with lower odds of both acute and chronic pain compared to the pain-free group, whereas DII was associated with the increased odds of chronic pain but not with acute pain (Table 2).

After adjusting for sociodemographic covariates including age, sex, ethnicity, and Townsend Deprivation Index (Model 1), these associations generally persisted. However, when additional adjustments (Model 2) were made for BMI, physical activity level, smoking status, alcohol intake, sleep duration, insomnia, and multimorbidity, associations for HDI and DII were attenuated. For every one-unit increase in the MDS score, the odds of chronic pain were reduced by approximately 2% (OR: 0.981, 95% CI: 0.969–0.992). Similarly, a one-unit increase in hPDI score was associated with a 0.8% reduction in the odds of chronic pain (OR: 0.992, 95% CI: 0.988–0.996), and for EATL, a 3.1% reduction was observed per unit increase (OR: 0.969, 95% CI: 0.955–0.983). For acute pain, only the hPDI remained consistently associated with lower odds, showing a 0.7% reduction per unit increase (OR: 0.993, 95% CI: 0.989–0.997) (Table 2, Fig. 2).

Chronic pain subgroup analysis based on pain site

The initial subgroup analysis investigated associations across different pain sites: single-site pain, multisite pain (2–3 sites), multisite pain (>3 sites), and widespread pain (pain all over body) (Table 3). In the unadjusted model (Model 0) and the partly adjusted model (Model 1), all five dietary indices were associated with the odds of experiencing all pain site subgroups. In the fully adjusted model (Model 2), DII was associated with a notable 7% increase in the odds of widespread pain (OR: 1.07, 95% CI: 1.027–1.125) compared to pain-free individuals. Conversely, DII did not show an association with other chronic pain subgroups. MDS demonstrated a notable 6.5% and 5.3% reduction in the odds of widespread pain (OR: 0.935, 95% CI: 0.884–0.988) and multisite pain (3> sites) (OR: 0.947, 95% CI: 0.912–0.983) per unit increase respectively. Furthermore, hPDI and Eat-Lancet were consistently associated with lower odds of both single-site and multisite (2–4 sites) chronic pain groups (Table 3, Fig. 3).

Chronic pain subgroup analysis based on pain type

The second subgroup analysis focused on the type of pain and categorized participants into MSK, non-MSK, and mixed pain groups (Table 4). Across all models, both before and after adjustments, hPDI and EATL consistently demonstrated negative associations with the odds

Table 1
Baseline characteristics.

	Pain-free (n= 24,632)	Acute pain (n= 8636)	Chronic pain (n= 22,453)	p<
Age (years)[a]	56.8 (8.05)	55.1 (8.38)	56.5 (8.10)	0.001
Sex[b]				0.001
Female	51.9%	50.4%	58.1%	
Male	48.1%	49.6%	41.9%	
Ethnicity[b]				0.001
White	95.2%	91.49%	93.5%	
Black	1.56%	2.56%	2.06%	
Asian	1.97%	3.61%	2.75%	
Mixed	0.64%	1.03%	0.66%	
Other	0.65%	1.30%	1.00%	
Education Level[b]				0.001
O levels/GCSE/CSEs or equivelant	26.2%	27.9%	29.0%	
A levels/AS levels or equivalent	12.8%	13.4%	12.4%	
College or University Degree	41.7%	38.5%	33.1%	
Professional Qualifications	10.5%	10.2%	12.0%	
None of the Above	8.87%	10.0%	13.5%	
Townsend Deprivation Index	-1.48 (2.74)	-1.20 (2.87)	-1.18 (2.89)	0.001
BMI (kg/m^2)[a]	26.5 (4.31)	27.0 (4.52)	27.8 (5.15)	0.001
Physical Activity[b]				0.001
Low	12.66%	12.70%	14.89%	
Moderate	34.5%	34.2%	32.65%	
High	36.68%	35.66%	32.66%	
Missing	16.11%	17.37%	19.80%	
Physical Inactivity (hours)[a]	3.73(2.01)	3.95(2.15)	4.12(2.23)	0.001
Sleep Duration (hours)[a]	7.20(0.96)	7.14(1.00)	7.09(1.14)	0.001
Insomnia[b]				0.001
Never/rarely	30.94	26.10	19.42	
Sometimes	47.76	50.54	46.02	
Usually	21.30	23.36	34.56	
Smoking Status[b]				0.001
Never	58.14%	58.26%	54.6%	
Previous	34.4%	33.64%	36.18%	
Current	7.42%	8.10%	9.27%	
Alcohol Intake[b]				0.001
Never	6.04	7.05	9.1	
Special occasions only	9.46	11.1	12.9	
One to three times a month	10.9	12.1	12.3	
Once or twice a week	25.11	25.3	24.7	
Three or four times a week	24.4	23.3	21.0	
Daily or almost daily	24.13	21.1	20.0	
Comorbidities[b]				0.001
0	45.96%	43.7%	33.71%	
1	33.54%	33.9%	33.41%	
2–3	19.12%	20.5%	28.30%	
4>	1.37%	1.9%	4.58%	
DII[a]	0.433 (2.05)	0.476 (2.09)	0.546 (2.14)	0.001
HDI[a]	4.04 (1.44)	4.00 (1.43)	3.96 (1.43)	0.001
HPDI[a]	59.47 (4.72)	59.08 (4.72)	59.07 (4.71)	0.001
MDS[a]	4.05 (1.59)	4.00 (1.59)	3.92 (1.59)	0.001
EATL[a]	9.10 (1.30)	9.02 (1.30)	9.02 (1.32)	0.001

Difference between continuous variables was assessed using analysis of variance (ANOVA) while difference between categorical variables were assessed using chi-square test.
p<0.05 was considered statistically significant.
BMI, Body mass index; n, number of participants; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index
[a] Numeric variables
[b] Categorical variables

Table 2
Unadjusted, partly adjusted, and fully adjusted multinominal logistic regression analysis - Main Cohort.

		Model 0			Model 1			Model 2		
		OR	95% CI	p<	OR	95% CI	p<	OR	95% CI	p<
DII	Pain-free (n=24,632)	Reference			Reference			Reference		
	Acute Pain (n=8636)	1.010	0.998–1.022	0.103	0.999	0.985–1.010	0.634	0.991	0.979–1.003	0.129
	Chronic Pain (n=22,453)	1.026	1.017–1.035	**0.001**	1.015	1.006–1.024	**0.001**	1.002	0.993–1.011	0.725
HDI	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.980	0.963–0.997	**0.021**	0.998	0.981–1.016	0.863	1.011	0.993–1.029	0.231
	Chronic Pain	0.959	0.947–0.971	**0.001**	0.967	0.954–0.979	**0.001**	0.994	0.981–1.008	0.402
HPDI	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.983	0.977–0.988	**0.001**	0.989	0.984–0.994	**0.001**	0.993	0.987–0.998	**0.007**
	Chronic Pain	0.981	0.978–0.985	**0.001**	0.983	0.979–0.986	**0.001**	0.993	0.989–0.997	**0.001**
MDS	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.978	0.963–0.993	**0.005**	0.984	0.968–0.999	**0.037**	0.995	0.979–1.011	0.515
	Chronic Pain	0.950	0.939–0.960	**0.001**	0.955	0.944–0.966	**0.001**	0.981	0.969–0.992	**0.001**
EATL	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.958	0.940–0.976	**0.001**	0.975	0.957–0.994	**0.011**	0.984	0.965–1.003	0.101
	Chronic Pain	0.958	0.945–0.972	**0.001**	0.948	0.935–0.962	**0.001**	0.969	0.955–0.983	**0.001**

The association between dietary quality indices and the presence of acute and chronic pain, with the pain-free group as the reference, was assessed using multinomial logistic regression. p<0.05 was considered statistically significant.
Model 0: Unadjusted model. **Model 1:** Adjusted for sociodemographic covariates including age, sex, ethnicity, and social deprivation index. **Model 2:** In addition to model 1, adjusted for body mass index, physical activity level, physical inactivity level, smoking status, alcohol intake, sleep duration, insomnia, and multimorbidity. OR Odds ratio; CI, Confidence interval; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index

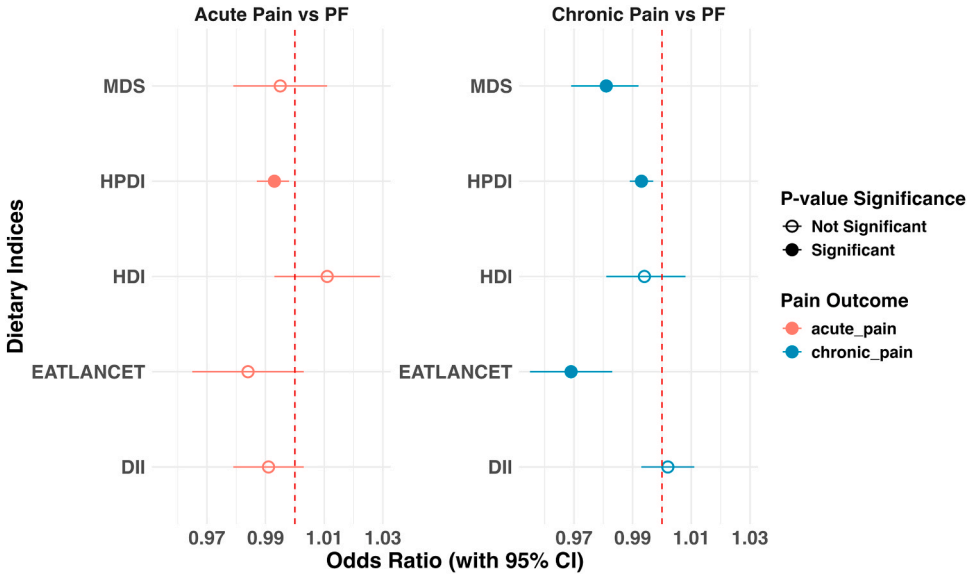

Fig. 2. Combined Results of Fully Adjusted Model-2 for Dietary Indices - Main Cohort. PF, Pain-free individuals; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index.

of experiencing all three pain subgroups. Similarly, MDS showed an inverse associations with all three subgroups in Models 0 and 1, but in fully adjusted Model 2, these associations remained evident only for non-MSK and mixed groups. While the DII was positively associated with all three subgroups in Models 0 and 1, after full adjustment in Model 2, it showed a positive association only with the odds of non-MSK pain. Specifically, a higher DII score was linked to a 2.8% increase in the odds of non-MSK pain for every unit increase in the index (OR: 1.028, 95% CI: 1.008–1.049). HDI was the only index that showed no association with all subgroups after fully adjusting the model with its confidence intervals consistently encompassing the null effect (Table 4, Fig. 4).

Sensitivity analyses

Sensitivity analyses excluding participants with missing physical activity data yielded results that were largely consistent with the main findings. The protective associations between higher scores on the hPDI, MDS, and EATL diet indices and reduced odds of chronic pain remained evident. The hPDI also continued to show a modest association with lower odds of acute pain (Supplementary Table S7).

Discussion

This population-based study showing a close link between diet and chronic pain. While there have been no studies comparing different dietary indices with pain outcomes including presence of acute and chronic pain within the same cohort, the main findings indicate that, in the fully adjusted model which accounted for both sociodemographic and lifestyle factors, only the hPDI, MDS, and EATL remained associated with lower odds of experiencing chronic pain. In contrast, associations for DII and HDI were weaker and more uncertain after adjustment,

Table 3
Unadjusted, partly adjusted, and fully adjusted multinominal logistic regression analysis - Pain Sites Chronic Pain Subgroup.

		Model 0			Model 1			Model 2		
		OR	95% CI	p<	OR	95% CI	p<	OR	95% CI	p<
DII	Pain-free (n= 24,632)	Reference			Reference			Reference		
	Acute Pain (n=8636)	1.010	0.998–1.022	0.103	0.997	0.985–1.009	0.641	0.991	0.979–1.003	0.132
	Single site (n=12667)	1.016	1.006–1.027	**0.002**	1.009	0.999–1.021	0.075	1.001	0.990–1.011	0.890
	Multisite 2–3 (n=8020)	1.027	1.015–1.043	**0.001**	1.015	1.010–1.034	**0.019**	0.998	0.985–1.001	0.722
	Multisite >3 (n=1225)	1.069	1.040–1.099	**0.001**	1.041	1.012–1.070	**0.005**	1.010	0.982–1.039	0.480
	Widespread (n= 541)	1.150	1.103–1.199	**0.001**	1.110	1.065–1.157	**0.001**	1.070	1.027–1.115	**0.001**
HDI	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.980	0.963–0.997	**0.021**	0.998	0.981–1.016	0.858	1.011	0.993–1.029	0.234
	Single site	0.977	0.963–0.992	**0.003**	0.984	0.969–0.999	**0.031**	1.002	0.987–1.018	0.768
	Multisite 2–3	0.945	0.929–0.962	**0.001**	0.952	0.935–0.969	**0.001**	0.985	0.967 – 1.003	0.110
	Multisite >3	0.898	0.863–0.935	**0.001**	0.915	0.879–0.953	**0.001**	0.971	0.931–1.012	0.158
	Widespread	0.890	0.839–0.945	**0.001**	0.912	0.859–0.969	**0.003**	0.980	0.923–1.042	0.526
HPDI	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.983	0.977–0.988	**0.001**	0.989	0.984–0.994	**0.001**	0.993	0.987–0.998	**0.007**
	Single site	0.984	0.980–0.989	**0.001**	0.986	0.981–0.990	**0.001**	0.993	0.988–0.997	**0.002**
	Multisite 2–3	0.980	0.975–0.985	**0.001**	0.981	0.975–0.986	**0.001**	0.993	0.988–0.999	**0.020**
	Multisite >3	0.966	0.955–0.978	**0.001**	0.969	0.958–0.981	**0.001**	0.991	0.978–1.003	0.146
	Widespread	0.967	0.950–0.985	**0.001**	0.971	0.953–0.988	**0.001**	0.996	0.978–1.015	0.709
MDS	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.978	0.963–0.993	**0.004**	0.984	0.968–0.999	**0.037**	0.995	0.979–1.010	0.508
	Single site	0.965	0.952–0.978	**0.001**	0.969	0.956–0.982	**0.001**	0.986	0.973 – 1.000	0.051
	Multisite 2–3	0.942	0.927–0.957	**0.001**	0.948	0.933–0.963	**0.001**	0.979	0.963–0.995	**0.011**
	Multisite >3	0.884	0.852–0.917	**0.001**	0.897	0.865–0.930	**0.001**	0.947	0.912 – 0.983	**0.004**
	Widespread	0.862	0.816–0.910	**0.001**	0.876	0.829–0.925	**0.001**	0.935	0.884–0.988	**0.018**
EATL	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.958	0.940–0.976	**0.001**	0.975	0.956–0.994	**0.010**	0.984	0.965–1.003	0.098
	Single site	0.965	0.949–0.981	**0.001**	0.961	0.944–0.977	**0.001**	0.974	0.958–0.991	**0.003**
	Multisite 2–3	0.961	0.943–0.980	**0.001**	0.943	0.925–0.962	**0.001**	0.968	0.949–0.988	**0.002**
	Multisite >3	0.905	0.866–0.946	**0.001**	0.881	0.842–0.921	**0.001**	0.921	0.880–0.964	**0.001**
	Widespread	0.910	0.853–0.972	**0.005**	0.892	0.835–0.954	**0.001**	0.945	0.884–1.011	0.102

The association between dietary quality indices and the presence of acute and chronic pain, with the pain-free group as the reference, was assessed using multinomial logistic regression. $p<0.05$ was considered statistically significant.
Model 0: Unadjusted model. **Model 1:** Adjusted for sociodemographic covariates including age, sex, ethnicity, and social deprivation index. **Model 2:** In addition to model 1, adjusted for body mass index, physical activity level, physical inactivity level, smoking status, alcohol intake, sleep duration, insomnia, and multimorbidity. OR, Odds ratio; CI, Confidence interval; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index

indicating these indices may be less informative for capturing diet–pain relationships. In the subgroup analysis based on pain sites, the fully adjusted models showed that higher scores on hPDI, MDS, and EATL were positively associated with reduced odds of both single-site and multisite (2–3 sites) chronic pain. Additionally, a higher DII, indicating a more pro-inflammatory diet, and a lower MDS scores were positively associated with increased odds of widespread pain. In the second subgroup analysis, which focused on pain types including MSK, non-MSK, and mixed pain, hPDI and EATL consistently showed positive associations with lower odds across all three pain type categories in all models. Furthermore, a higher DII and lower MDS were both positively associated with the odds of non-MSK chronic pain. Notably, associations involving the HDI were consistently weak and imprecise across all subgroups after adjustment. Overall, although the observed associations were modest in magnitude, they were consistent across several pain subtypes and patterns, including musculoskeletal and widespread pain.

While the individual odds reductions (e.g., 2–3% per unit increase in hPDI, MDS, or EATL) may appear small, they are comparable to the effect sizes of other established lifestyle factors in large-scale epidemiological studies. For example, the association between diet quality and chronic pain in our model is similar in magnitude to the observed protective effect of physical activity within the same cohort. In the UK Biobank, the difference between the highest and lowest quartiles of physical activity often yields odds ratios in the range of 0.85–0.95 for chronic pain; our findings for top-tier diet quality fall within a similar range.[28] Therefore, given the large sample size and the diverse range of dietary exposures captured, these findings likely reflect meaningful population-level effects with potential clinical relevance over time.

Our findings align with a growing body of literature supporting the association between diet quality and chronic pain. Previous studies have reported that individuals with chronic spinal or low back pain tend to have lower overall diet quality and higher intake of pro-inflammatory foods compared to pain-free individuals.[7,29] For instance, Zick et al. [29] found that chronic spinal pain was associated with lower scores on the HEI-2015, and that lower sugar intake, particularly from added sugars, reduced the odds of spinal pain by more than 25%. Similarly, individuals with chronic low back pain were reported to have poorer diet quality and greater consumption of pro-inflammatory foods.[7] Our study extends these findings by showing that diet quality indices emphasizing anti-inflammatory and healthy whole-food components, such as hPDI, MDS, and EATL, are associated with lower odds of chronic pain, even after adjusting for a broader set of potential confounders including sleep quality, smoking, and multimorbidity factors not consistently accounted for in earlier studies. Furthermore, the positive association between DII and widespread or non-MSK pain observed here is in line with previous research linking pro-inflammatory diet measured with DII to abdominal and non-MSK pain conditions.[27]

This study evaluates and compares five distinct dietary indices within a single, large population-based cohort in relation to chronic pain, revealing that associations between diet and pain are not uniform but vary depending on pain phenotypes and the dietary construct assessed. The differences observed between dietary indices in our analysis likely reflect their distinct methodological foundations and the specific dietary components they capture.[30] Indices such as the hPDI and EATL emphasize nutrient-dense, minimally processed plant foods and make clear distinctions between fruits and vegetables, different meat types, and healthful and unhealthful food groups, for example, separating whole from refined grains. hPDI also uses quantile-based,

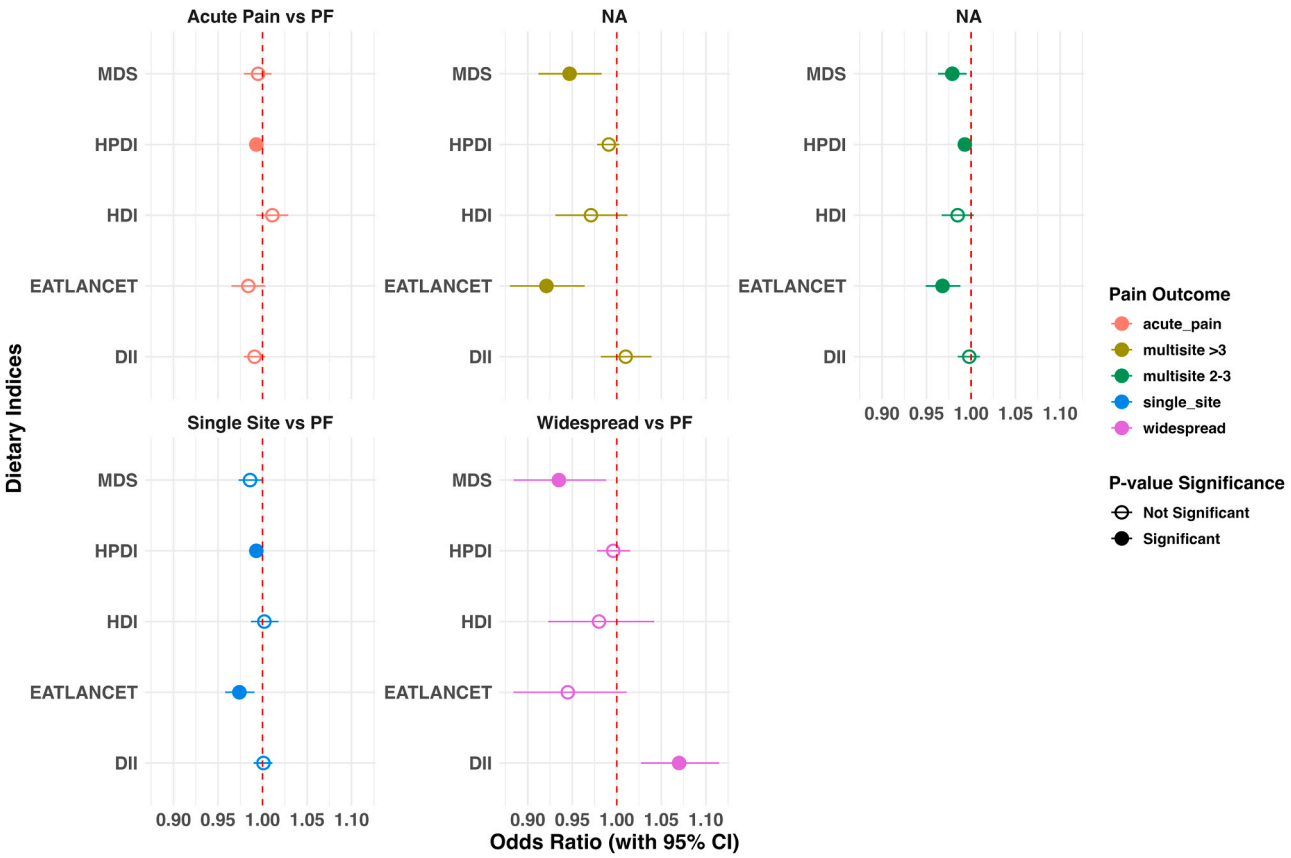

Fig. 3. Combined Results of Fully Adjusted Model-2 for Dietary Indices - Pain Sites Subgroup. PF, Pain-free individuals; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index.

sex-specific scoring that focuses on whole foods. Thereby they both are better at capturing dietary aspects that might influence pain-related mechanisms such as glycemic regulation,[31] gut microbiota composition,[32] and inflammation.[33] In contrast, the HDI, which is based on broader dietary guideline, does not adequately differentiate food quality or levels of processing. It primarily focuses on individual nutrients rather than whole food components, which may explain its weaker associations with chronic pain and its loss of significance after adjustment. The MDS also showed some protective associations, but its performance varied across subgroups, suggesting it may not fully capture dietary elements most relevant to chronic pain. Meanwhile, the DII's unique focus on the inflammatory potential of the diet provides specific insights into how individual nutrients influence systemic inflammation, which may be particularly relevant to pain types such as widespread or non-MSK pain.

The differing associations across chronic pain subgroups highlight the importance of considering pain phenotypes in nutritional research and interventions. hPDI and EATL were consistently linked to lower odds in most subgroups, except non-MSK and widespread pain, suggesting a stronger role in more localized MSK pain. In contrast, DII and MDS showed stronger associations with widespread and non-MSK pain, which are often linked to systemic inflammation, cardiovascular risk, and neuroinflammatory pathways. This aligns with evidence that widespread pain is associated with higher risks of cardiovascular events such as stroke, myocardial infarction, and heart failure,[34] with a greater number of pain sites also correlating with cardiovascular dysfunction.[35] Additionally, multimorbidity significantly increases the likelihood of chronic and widespread pain, with UK Biobank data showing participants with four or more long-term conditions were three times more likely to report chronic pain and twenty times more likely to experience chronic widespread pain compared to those without comorbidities.[25]

Strength and limitations

A major strength of this study is its large-scale design and population-based nature, utilizing data from the UK Biobank, enhancing the generalizability of the findings. The use of a validated dietary assessment tool (Oxford WebQ) allowed for detailed analysis of food and nutrient intake, providing robust and reliable dietary data. Moreover, the study applied five different dietary indices, offering a comprehensive evaluation of diet quality and its association with various pain conditions. The analysis also adjusted for an extensive set of sociodemographic and lifestyle factors as well as multimorbidity helping to minimize confounding.

However, several limitations should be noted. The cross-sectional design of this analysis limits the ability to draw causal inferences, as dietary intake and chronic pain were assessed at the same time, making it difficult to determine directionality. Reverse causality is a potential concern, for example, individuals with chronic pain may change their dietary habits in response to their symptoms, but even in that case results are important in a way that poor dietary habits are likely to perpetuate chronic pain.

Residual confounding remains a possibility. Although the UK Biobank provides rich phenotypic data, and we adjusted for an extensive set of covariates guided by a directed acyclic graph to, it is still possible that relevant variables were either unmeasured or not available in the dataset. Chronic disease was accounted for using a multimorbidity measure based on the count of long-term conditions (LTCs), which is a commonly used approach in epidemiological studies to capture overall disease burden and adjust for confounding25. However, this method does not account for differences in disease type, severity, or duration, nor for specific conditions that may relate differently to both diet and pain.

Table 4
Unadjusted, partly adjusted, and fully adjusted multinominal logistic regression analysis - MSK Chronic Pain Subgroup.

		Model 0			Model 1			Model 2		
		OR	95% CI	p<	OR	95% CI	p<	OR	95% CI	p<
DII	Pain-free (n= 24,632)	Reference			Reference			Reference		
	Acute Pain (n=8636)	1.010	0.998–1.022	0.099	0.997	0.986–1.009	0.647	0.991	0.979–1.003	0.133
	MSK (n-16,114)	1.016	1.007–1.026	**0.001**	1.010	1.000–1.019	**0.048**	0.996	0.986–1.006	0.442
	Non-MSK (n=2588)	1.052	1.032–1.073	**0.001**	1.034	1.013–1.054	**0.001**	1.028	1.008–1.049	**0.006**
	Mixed (n=3751)	1.052	1.035–1.070	**0.001**	1.028	1.011–1.045	**0.001**	1.008	0.991–1.025	0.384
HDI	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.980	0.963–0.997	**0.020**	0.998	0.981–1.016	0.862	1.011	0.993–1.029	0.232
	MSK	0.963	0.949–0.976	**0.001**	0.968	0.955–0.982	**0.001**	0.996	0.982–1.011	0.626
	Non-MSK	0.979	0.952–1.007	0.147	0.988	0.960–1.017	0.417	0.998	0.970–1.028	0.916
	Mixed	0.931	0.909–0.954	**0.001**	0.944	0.922–0.967	**0.001**	0.982	0.958–1.006	0.145
HPDI	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.983	0.977–0.988	**0.001**	0.989	0.984–0.994	**0.001**	0.993	0.987–0.998	**0.006**
	MSK	0.983	0.979–0.987	**0.001**	0.983	0.978–0.987	**0.001**	0.994	0.989–0.998	**0.005**
	Non-MSK	0.983	0.974–0.991	**0.001**	0.989	0.981–0.998	**0.013**	0.990	0.982–0.999	**0.030**
	Mixed	0.973	0.966–0.980	**0.001**	0.978	0.971–0.986	**0.001**	0.990	0.983–0.998	**0.012**
MDS	Pain-free	Reference			reference			Reference		
	Acute Pain	0.978	0.963–0.993	**0.004**	0.981	0.966–0.997	**0.036**	0.995	0.979–1.010	0.503
	MSK	0.956	0.944–0.968	**0.001**	0.962	0.950–0.974	**0.001**	0.989	0.977–1.002	0.105
	Non-MSK	0.944	0.921–0.969	**0.001**	0.943	0.919–0.968	**0.001**	0.949	0.925–0.974	**0.001**
	Mixed	0.925	0.905–0.945	**0.001**	0.932	0.912–0.953	**0.001**	0.965	0.944–0.987	**0.002**
EATL	Pain-free	Reference			Reference			Reference		
	Acute Pain	0.958	0.940–0.976	**0.001**	0.975	0.957–0.994	**0.007**	0.984	0.965–1.003	0.097
	MSK	0.959	0.945–0.974	**0.001**	0.955	0.940–0.970	**0.001**	0.951	0.921–0.982	**0.002**
	Non-MSK	0.973	0.943–1.003	0.080	0.948	0.918–0.979	**0.001**	0.951	0.921–0.982	**0.002**
	Mixed	0.945	0.921–0.971	**0.001**	0.920	0.896–0.946	**0.001**	0.946	0.920–0.972	**0.001**

The association between dietary quality indices and the presence of acute and chronic pain, with the pain-free group as the reference, was assessed using multinomial logistic regression. p<0.05 was considered statistically significant.
Model 0: Unadjusted model. **Model 1:** Adjusted for sociodemographic covariates including age, sex, ethnicity, and social deprivation index. **Model 2:** In addition to model 1, adjusted for body mass index, physical activity level, physical inactivity level, smoking status, alcohol intake, sleep duration, insomnia, and multimorbidity. OR, Odds ration; CI, Confidence interval; MSK, Chronic musculoskeletal pain; Non-MSK, Chronic non-musculoskeletal pain; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index

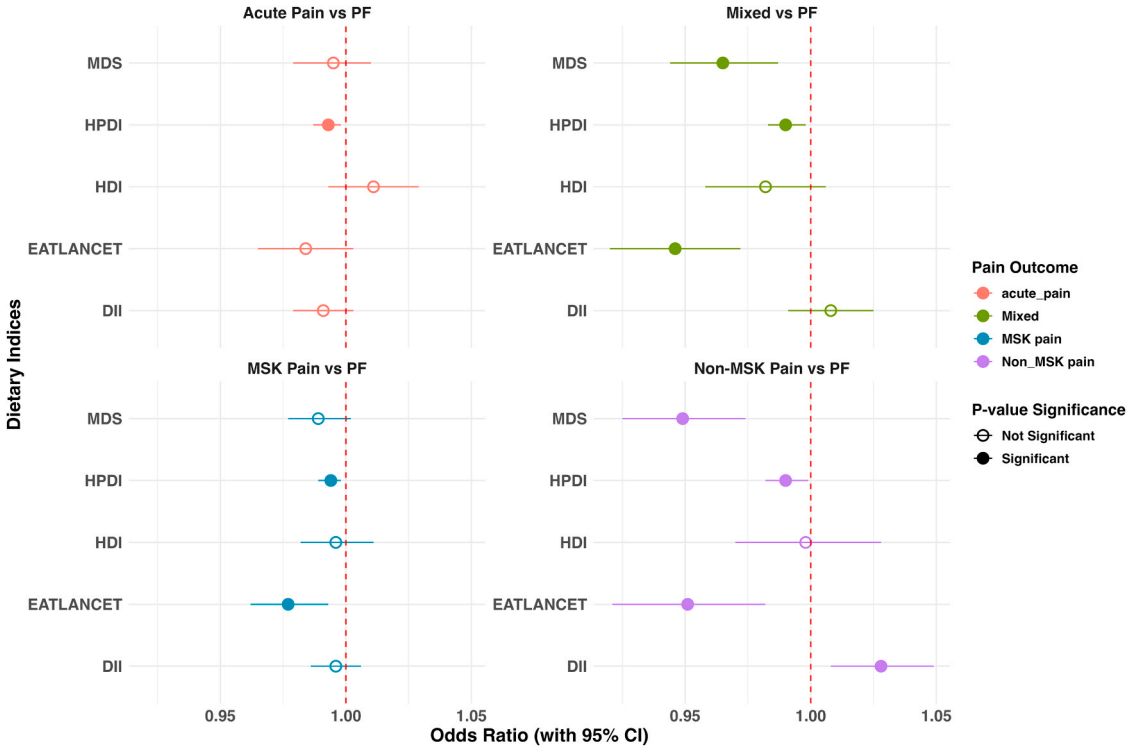

Fig. 4. Combined Results of Fully Adjusted Model-2 for Dietary Indices - MSK Pain Subgroup. PF, Pain-free individuals; DII, Dietary inflammatory index; HDI, Healthy diet index; HPDI, Healthful plant-based diet index; MDS, Mediterranean diet score; EATL, Eat-Lancet dietary index.

Another limitation of this study is the generalizability of the findings to more ethnically and socioeconomically diverse populations due to the characteristics of the UK Biobank cohort. The sample is predominantly White (95.2%) and demonstrates higher levels of affluence (mean

Townsend Deprivation Index < 0) compared to the general UK population. While these factors may limit the external validity of our results to more ethnically and socioeconomically diverse populations, the internal validity is supported by our use of DAGs to identify and statistically adjust for these variables in our multivariable models specifically Model 1 and 2.

Finally, dietary intake was assessed using a single slef-reported 24-hour dietary recall which may be subject to recall bias and does not capture long-term dietary patterns or within-person variation over time.

Clinical and research implications

These findings highlight an association between diet quality and chronic pain, highlighting dietary modification as a potential complementary management approach. Lower odds of chronic pain with hPDI and EATL suggest benefits of plant-based, whole-food diets rich in nutrients and anti-inflammatory properties. Positive associations between DII and widespread and non-MSK pain point to a role of systemic inflammation. Targeting pro-inflammatory diets and tailoring recommendations by pain type may enhance treatment strategies. On the other hand, it is also important to distinguish between individual clinical significance and public health significance; while a 3% reduction in odds per unit of diet improvement might not change a single patient's immediate clinical prognosis, a shift in diet quality across the entire population could significantly reduce the total burden of chronic pain.

Given the cross-sectional design, future research should focus on longitudinal and interventional studies to clarify temporal and causal links between diet and chronic pain. Prospective studies with repeated diet and pain measures can help address reverse causality, while randomized controlled trials are needed to test whether dietary changes reduce pain and improve quality of life. Mechanistic studies should explore metabolic, inflammatory, and neurobiological pathways, and personalized nutrition approaches, based on metabolic profiles, inflammation, gut microbiome, or pain phenotypes, may enhance intervention effectiveness and support precision pain management.

Conclusion

These findings suggest that different dietary indices capture distinct aspects of diet quality, which may have varying utility on assessing chronic pain outcomes. The methodological differences between these indices such as the hPDI and EATL emphasizing whole, plant-based foods, while the DII focuses on inflammatory potential likely explain their differential associations with pain phenotypes. Notably, the hPDI and EATL were consistently linked to lower odds of chronic pain, particularly musculoskeletal and localized pain, whereas the DII showed stronger associations with widespread and non-MSK pain. These results underscore the link between dietary quality and chronic (widespread) pain and highlight the importance of considering different diet quality indices and pain subtypes when evaluating diet-pain relationships. Controlled longitudinal and interventional studies are needed to confirm these associations and evaluate the potential of dietary modification as a strategy for pain management.

CRediT authorship contribution statement

Conception and design: All authors. Data access: OE and CL. Data cleaning and pre-processing: OE and CL. Data analysis: OE and CL. Interpretation of the results: All authors. Writing the first draft of the manuscript: OE. Reviewed and approved the final version of the manuscript: All authors.

Disclosures

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Conflict of Interest

The authors declare no conflict of interest.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jpain.2026.106331.

Data Availability

The data used in this study are not publicly available but can be accessed upon application and approval from the UK Biobank (https://www.ukbiobank.ac.uk)

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