



OPEN Dietary inflammation and its impact on congestive heart failure in older adults with depression

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Depression is recognized as a major contributor to global disability, significantly impacting individuals' quality of life. Recent studies suggest a link between depression and cardiovascular diseases, including congestive heart failure (CHF), possibly influenced by dietary patterns that promote chronic inflammation. This cross-sectional study investigated the relationship between the dietary inflammatory index (DII) and congestive heart failure in older adults with depressive symptoms. Data from the National Health and Nutrition Examination Survey (NHANES) collected between 2005 and 2018 were analyzed. The participants were classified according to their depressive symptoms and congestive heart failure status. DII scores were calculated from dietary intake data, reflecting the inflammatory potential of participants' diets. The associations between the DII and the status of CHF among individuals with depression were assessed using generalized linear models (GLM), trend tests, and subgroup analyses. Among individuals displaying depressive symptoms, particularly those aged over 60 years, a significant positive correlation was found between DII scores and the incidence of CHF [OR: 1.122 (95% CI: 1.041, 1.209)]. Subgroup analyses indicated that older adults (≥ 60 years) were especially vulnerable to the effects of dietary inflammation on CHF risk, while no significant association was observed in younger participants (< 60 years). Higher DII scores are linked to an increased risk of CHF among older adults with depressive symptoms. This highlights the need for dietary interventions to address inflammation, potentially reducing CHF risk in this vulnerable population.

Keywords Cross-sectional study, Dietary inflammation index, Congestive heart failure, Depression, NHANES

As the prevalence of depression continues to rise annually, it has emerged as a prominent mental illness, with major depression now being a leading cause of disability¹. Individuals suffering from depression often exhibit emotional symptoms such as a lack of motivation and persistent low mood², significantly impacting their quality of life and diminishing social productivity. Projections indicate that depression will burgeon into a major global disease burden by 2030³.

Moreover, mortality rates among depression patients significantly surpass those of the general population⁴, with research suggesting a marked increase in the incidence of cardiovascular diseases among this demographic population^{5,6}, potentially contributing to this elevated mortality rate. Congestive heart failure, which is prevalent as the foremost syndrome in the advanced stages of various cardiovascular ailments, is characterized by high mortality rates and affects approximately 23 million individuals worldwide⁷. Therefore, unraveling the risk factors associated with congestive heart failure development in depressed patients could have profound implications for mitigating mortality rates in this vulnerable group. Chronic inflammation links diet, depression, and congestive heart failure (CHF) through several pathways. Inflammatory diets can increase pro-inflammatory cytokines, negatively affecting mood and potentially leading to depression. This inflammation also contributes to oxidative stress and endothelial dysfunction, raising cardiovascular risks. Moreover, it disrupts neurotransmitter systems, worsening both mood and inflammation. Addressing these interconnected factors through dietary changes could improve health outcomes for individuals with depression and cardiovascular conditions^{8–11}.

Recent studies have revealed a positive correlation between dietary habits fostering systemic chronic inflammation and depression incidence^{11–14}. The progression of congestive heart failure may be linked to the perpetuation of systemic chronic inflammation^{8,15}, with emerging evidence suggesting that dietary habits

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play a pivotal role in modulating chronic inflammation levels, subsequently influencing disease onset and progression^{16,17}. The dietary inflammatory index (DII) serves as a quantifiable measure of the inflammatory properties inherent in one's diet, facilitating the guidance of dietary adjustments and the evaluation of potential associations between diet-induced chronic inflammation and various diseases^{18,19}. Despite the known connection between depression and cardiovascular diseases^{5,6}, there is a notable gap in research specifically addressing how dietary habits, particularly those that influence systemic inflammation, affect this relationship. While existing literature establishes a significant association between depression and cardiovascular issues, few studies have explored the impact of dietary inflammatory factors, as measured by the DII, on the development of congestive heart failure among depressed individuals.

Conducted under the auspices of the United States, the National Health and Nutrition Examination Survey (NHANES) cross-sectional study boasts attributes of high quality, substantial sample size, and representativeness, rendering its analytical findings more robust. This study aims to scrutinize DII scores among individuals with depression via the NHANES database, shedding light on the significance of dietary inflammatory factors in the interplay between depression and congestive heart failure. It is envisaged that by refining dietary habits in the future, the prevention of congestive heart failure among individuals with depression can be more targeted and effective.

Methods

Data sources

The NHANES is a significant and comprehensive survey undertaken in the United States. "The NHANES dataset is a robust resource that combines self-reported health information with objective health measurements, although it is important to note that reliance on self-reported data may introduce biases. Its primary goal is to evaluate the nation's population's health and nutritional status and to supply copious amounts of data on a range of health-related indicators, such as obesity, diabetes, and cardiovascular disease. Using a special design that combines interviews and physical examinations, the National Center for Health Statistics (NCHS), a division of the Centers for Disease Control and Prevention (CDC) in the United States, administers the NHANES. The datasets used and analyzed in the current study are publicly available on the NHANES website (<https://www.n.cdc.gov/nchs/nhanes/Default.aspx>). Comprehensive data regarding lifestyle, nutrition, and health status are gathered in the survey interview section. For example, participants may be asked about their diet, physical activity levels, smoking habits, and access to healthcare. The physical examination section is conducted at a specially equipped mobile examination center, providing medical, dental, and physiological measurements, as well as laboratory testing of blood and urine samples. This comprehensive approach enables the NHANES to collect data reflecting self-reported and objectively measured health and nutritional status indicators. The National Center for Health Statistics Ethics Review Board authorized the procedure, and all participants provided written, informed consent. Our data was obtained from a cross-sectional survey (NHANES), which limits our ability to explore risk factors prospectively. Additionally, we excluded study subjects who did not have biochemical indicators measured, which may affect the generalizability of our findings to the broader American population. The dietary recall information and some covariates were derived from interviews and questionnaires, which may lead to inaccuracies or recall bias. Physical activity levels and sedentary behavior were evaluated using questionnaires rather than objective measures like accelerometers. Finally, there may be unknown confounders that are not accounted for in the NHANES data. The inclusion criteria for the study are participants aged 60 years and older who exhibit depressive symptoms as assessed by the Patient Health Questionnaire-9 (PHQ-9), have available dietary intake data to calculate DII scores, and have no prior diagnosis of CHF at enrollment; conversely, the exclusion criteria include individuals younger than 60 years, those without depressive symptoms, participants with incomplete dietary intake data or health records, and individuals with any prior diagnosis of CHF or significant cardiovascular diseases.

Participants

This study uses a 14-year period of data (2005–2018) to conduct the survey. First, we eliminated 33,853 individuals whose PHQ-9 questionnaire data were missing. Next, 27,362 people who did not have depression were excluded. Samples with extreme energy intakes (females < 500 kcal/d or > 5,000 kcal/d; males < 500 kcal/d or > 8,000 kcal/d)²⁵; samples with missing main indicators and dietary data were excluded. Finally, the study resulted in the exclusion of 559 and 340 participants, respectively, because of a lack of conservative heart failure and inadequate dietary information. In the end, 8076 subjects composed the study (Fig. 1).

The Patient Health Questionnaire-9 (PHQ-9) is a widely used tool for diagnosing, monitoring, and measuring the degree of depression. It consists of 9 items, each of which is related to depressive symptoms, selected according to the diagnostic criteria for depression in the American Diagnostic of Mental Disorders and Statistical Manual Fourth Edition (DSM-IV). The PHQ-9 is not only used in the field of mental health but also widely used as a rapid tool in primary healthcare and other medical settings. When used to evaluate the frequency of depressive symptoms in the past 2 weeks, it has shown high reliability and effectiveness in the general population, with scores ranging from 0 to 27. In this study, a population with a score of 5 or above was selected as the population with depressive symptoms²⁰.

The DII is a scientifically developed tool aimed at evaluating the inflammatory potential of an individual's diet. The DII is calculated by summing the total potential inflammatory levels associated with individual dietary intake^{21,22}. When the dietary data of 1 day was missing, the average value was replaced by another day value. Energy, protein, alcohol β -carotenoids, carbohydrates, cholesterol, total fat, fiber, folate, Fe, MUFAs, n-3 fatty acids, caffeine, PUFA, niacin, riboflavin, saturated fats, selenium thiamine, vitamins A/C/E/B12/B6, Mg, and zinc are all used to generate DII scores. The calculation of the DII takes into account various food conditions

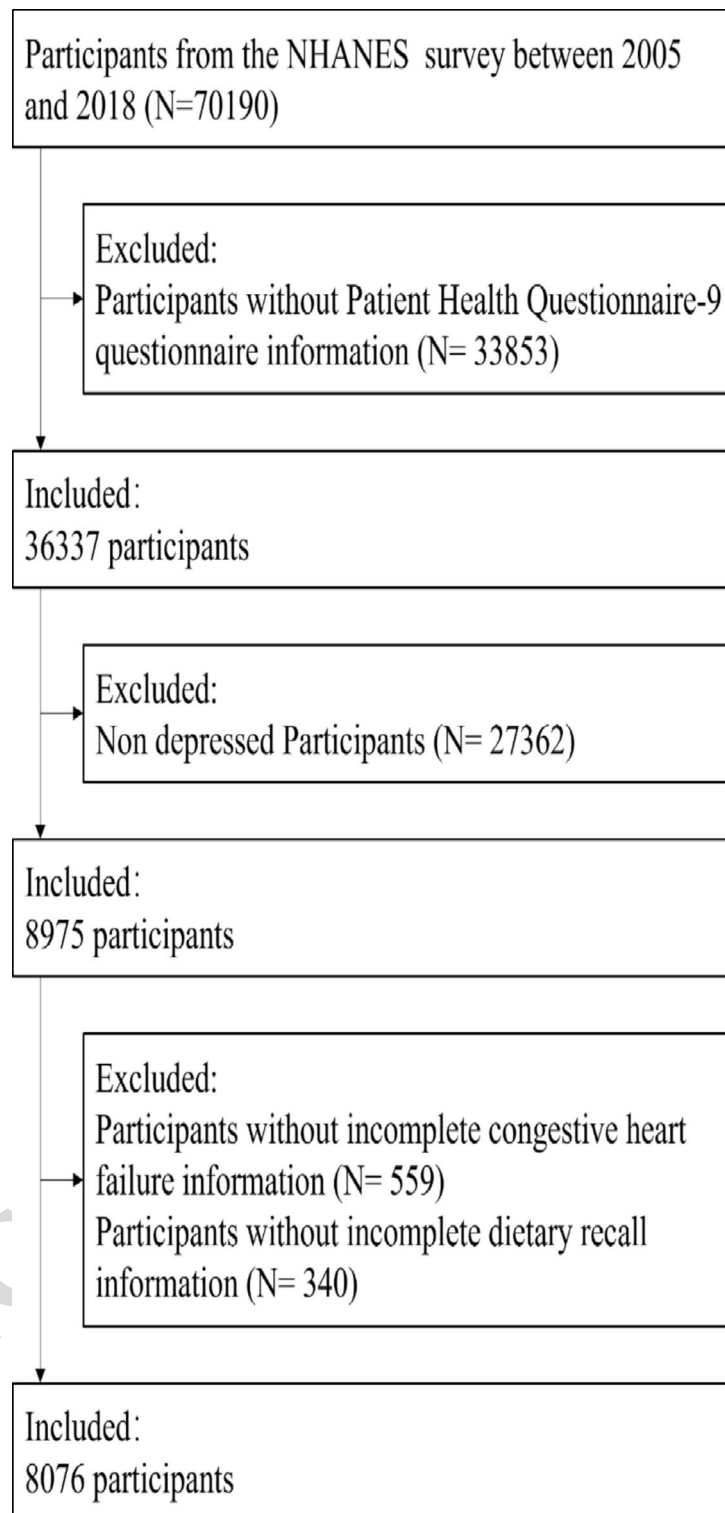


Fig. 1. Flow chart of participant selection. *NHANES* National Health and Nutrition Examination Survey.

and provides positive and negative scores on the basis of the ability of the DII to promote inflammation or anti-inflammatory properties. First, we calculated the dietary parameters and z scores for each participant. The individual's z score is then converted to the median percentile. Third, for each median percentile, the standardized overall inflammatory impact score was calculated. To control for the total energy intake effect, the **DII** was calculated per 1000 calories consumed. Higher E-DII scores indicate more pro-inflammatory diets, while more negative values suggest a more anti-inflammatory diet. Attachment 1 (Additional file 1) shows the

dietary inflammation scores for each nutrient used in the calculation and then sums the DII scores for each participant.

If participants answered yes to doctors or other health professionals informing them that they have congestive heart failure, they were classified as having congestive heart failure. If the participant answers “yes” to the following question, it is considered congestive heart failure: “Have doctors or other health professionals told you that you have congestive heart failure?”. This self-reported method for diagnosing heart disease by doctors has been proven to be sufficiently effective for monitoring purposes²³. We excluded participants with missing data on the independent variable (DII score) or the dependent variable (CHF status), as well as those without depressive symptoms. For missing covariate information, we used the mean value for continuous variables or the missing-indicator method for categorical variables^{24,25}. As a sensitivity analysis, we repeated the logistic regression analysis without the NHANES sampling weight. The covariates included age, sex, race, body mass index, smoking status, drinking status, diabetes status, and hypertension status.

According to the definition by the United Nations and the World Health Organization (WHO), individuals aged 60 and above are classified as the elderly^{26,27}. The pathogenesis, clinical manifestations, and prognosis of CHF in this age group may differ from those in individuals under 60²⁸. By conducting stratified research, we can more accurately understand the association between the DII and CHF at different stages of life, providing a basis for targeted interventions.

Significant differences exist in the incidence and prognosis of CHF among different races. Studies have shown that the prevalence of CHF is highest among non-Hispanic Black individuals, followed by non-Hispanic White and Mexican American individuals²⁹. Differences in inflammatory responses may exist among different races, which could be related to genetic background and environmental factors³⁰. Stratified research by race can more accurately assess the relationship between DII and CHF in different racial groups, providing support for precision medicine.

Statistical analysis was performed via R (version 4.2) and Empower Stats (version 4.1). The weighted chi square test and weighted linear regression were used to evaluate the demographic characteristics of the sample. To investigate the linear relationship between DII and congestive heart failure, a generalized linear model was used for evaluation. The relationships between different DII groups and CHF were explored through subgroup analysis experiments. Statistical significance was defined as a two-tailed *P* value < 0.05. The relationships between different DII groups and CHF were explored through subgroup analysis experiments. A trend test was conducted to assess the linear association between DII scores and the incidence of CHF. Trend analyses have also been conducted in previous studies on CHF³¹.

Results

The participant characteristics categorized CHF status are summarized in Table 1. Participants with CHF had a mean age of 63.974 years (SE = 0.674), significantly higher than the 46.194 years (SE = 0.298) of those without CHF (*p* < 0.001). The average body mass index (BMI) for participants with CHF was 33.630 (SE = 0.521), compared to 30.174 (SE = 0.149) for those without CHF (*p* < 0.001). Among CHF participants, 58.023% were female, in contrast to 60.581% in the non-CHF group (*p* = 0.323). In terms of race/ethnicity, 4.598% of CHF participants were Mexican American, compared to 8.386% in the non-CHF group (*p* = 0.005). The prevalence of diabetes was significantly higher among those with CHF at 43.649%, compared to 11.912% in the non-CHF group (*p* < 0.001). Hypertension was reported by 84.775% of CHF participants, starkly contrasting with 42.367% in the other group (*p* < 0.001). Lifestyle factors also showed significant differences: 31.181% of CHF participants were former drinkers compared to 15.090% in the non-CHF group (*p* < 0.001), and smoking prevalence was higher among CHF participants, with 37.210% being former smokers compared to 23.894% without CHF (*p* < 0.001). These findings underscore the distinct health profiles and risk factors associated with CHF status.

The relationship between the dietary inflammation index (DII) and congestive heart failure was assessed through three generalized linear regression models, as detailed in Tables 2 and 3. Each model consistently demonstrated a significant positive association between DII and congestive heart failure. In Model 1, the odds ratio (OR) for DII was 1.134 (95% CI: 1.055, 1.218), indicating that each unit increase in DII was associated with a 13.4% increase in the odds of developing congestive heart failure. After adjusting for relevant demographic factors in Model 2, the OR increased to 1.159 (95% CI: 1.077, 1.247), reinforcing the strength of this association. In the fully adjusted Model 3, a unit increase in the DII score corresponded to an OR of 1.122 (95% CI: 1.041, 1.209), suggesting a 12.2% increase in the odds of congestive heart failure. The trend test confirmed the stability of this linear relationship across all the models, with a *P* value for trend < 0.001, highlighting the robustness of the findings. Repeated logistic regression analysis as a sensitivity analysis without weighting revealed similar trends in the results (Additional file 2).

This Fig. 2 illustrates the comparison of Odds Ratios (OR) among different groups across various models, specifically analyzing the distribution of certain factors categorized by DII quartiles (Q1 to Q4), where the horizontal axis represents risk distribution from low to high risk, each of the three models (Model 1, Model 2, Model 3) corresponds to a separate data column, and the color-coded bars indicate the OR and 95% confidence intervals (CIs) for different quartile groupings under model adjustments; Q1 represents the lowest risk level while Q4 represents the highest, with the OR indicating the risk ratio—where OR > 1 suggests increased risk and OR < 1 suggests decreased risk—and the 95% CI representing the confidence interval for the OR, wherein if the CI includes 1, the risk ratio is not statistically significant; the specific results show that in Model 1, Q1 has an OR of 1.373 (95% CI: 0.919–2.052, *P* = 0.125) while Q4 has an OR of 1.772 (95% CI: 1.239–2.533, *P* = 0.002) with a trend *P*-value of 0.002, indicating a significant increase in risk for Q4 compared to Q1; in Model 2, Q1 has an OR of 1.385 (95% CI: 0.911–2.108, *P* = 0.131) and Q4 has an OR of 1.960 (95% CI: 1.370–2.804, *P* < 0.001) with a trend *P*-value of < 0.001, further suggesting an increased risk ratio for Q4 after variable adjustment; in Model 3, Q1 has an OR of 1.245 (95% CI: 0.815–1.900, *P* = 0.313) and Q4 has an OR of 1.684 (95% CI: 1.160–2.444,

Characteristics	Without congestive heart failure	With congestive heart failure	P value
DII	1.716 ± 0.035	2.124 ± 0.109	< 0.001
Age	46.194 ± 0.298	63.974 ± 0.674	< 0.001
Body mass index	30.172 ± 0.148	33.630 ± 0.521	< 0.001
Gender, (%)			0.323
Female	60.581	58.023	
Male	39.419	41.977	
Race/ethnicity, (%)			0.005
Mexican American	8.386	4.598	
Non-Hispanic	78.221	84.843	
Other race/multiracial	13.393	10.559	
Diabetes, (%)			< 0.001
No	88.040	56.351	
Yes	11.912	43.649	
Unrecorded	0.048	0	
Alcohol drinking status, (%)			< 0.001
Never	9.014	14.488	
Former	15.090	31.181	
Heavy	25.790	8.290	
Mild	28.827	31.779	
Moderate	17.634	8.548	
Unrecorded	3.645	5.713	
Smoking status, (%)			< 0.001
Never	45.080	38.583	
Former	23.894	37.210	
Now	31.026	24.207	
Hypertension			< 0.001
No	57.633	15.225	
Yes	42.367	84.775	

Table 1. Baseline characteristics of the participants.

	Model 1	Model 2	Model 3
Congestive heart failure	OR (95% CI) P value	OR (95% CI) P value	OR (95% CI) P value
DII	1.134 (1.055, 1.218) < 0.001	1.159 (1.077, 1.247) < 0.001	1.122 (1.041, 1.209) 0.003

Table 2. Associations between dietary inflammatory indices and congestive heart failure in the population with depressive symptoms (with sampling weights). Model 1: no covariates were adjusted. Model 2: Age, sex, and race were adjusted. Model 3: Age, sex, race, body mass index, smoking status, diabetes status, hypertension status and alcohol consumption status were adjusted for.

	Model 1	Model 2	Model 3
Congestive heart failure	OR (95% CI) P value	OR (95% CI) P value	OR (95% CI) P value
DII quartile			
Q1	Ref.	Ref.	Ref.
Q2	1.373 (0.919, 2.052) 0.125	1.385 (0.911, 2.108) 0.131	1.245 (0.815, 1.900) 0.313
Q3	1.379 (0.947, 2.009) 0.097	1.405 (0.966, 2.042) 0.078	1.213 (0.821, 1.792) 0.335
Q4	1.772 (1.239, 2.533) 0.002	1.960 (1.370, 2.804) < 0.001	1.684 (1.160, 2.444) 0.007
P for trend	0.002	< 0.001	0.009

Table 3. Trend test between dietary inflammatory indices and congestive heart failure in the population with depressive symptoms (with sampling weights). Model 1: no covariates were adjusted. Model 2: Age, sex, and race were adjusted. Model 3: Age, sex, race, body mass index, smoking status, diabetes status, hypertension status and alcohol consumption status were adjusted for.

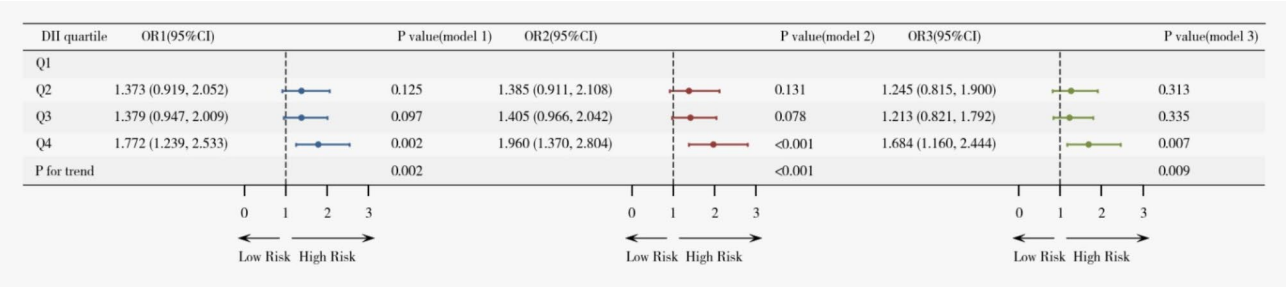


Fig. 2. Illustrates odds ratios (OR) by dietary inflammatory index (DII) quartiles (Q1–Q4), with each model (Model 1, Model 2, Model 3) shown in separate columns.

Subgroup	OR (95% CI) P value
Age	
> 60	1.119 (1.024, 1.222) 0.013
≤ 60	1.113 (0.986 1.256) 0.085
Race/ethnicity	
Mexican American	1.312 (1.033, 1.666) 0.031
Non-Hispanic	1.111 (1.020, 1.211) 0.018
Other race/multiracial	1.170 (0.970, 1.411) 0.104

Table 4. Subgroup analysis of the associations between dietary inflammatory indices and congestive heart failure in the population with depression symptoms.

Congestive heart failure	Model with sampling weights	Model without sampling weights
	OR (95% CI) P value	OR (95% CI) P value
DII	1.295 (1.003, 1.671) 0.0496	1.271 (1.020, 1.585) 0.0328
DII quartile		
Q1	Ref.	Ref.
Q2	3.021 (0.585, 15.596) 0.1896	3.289 (0.885, 12.224) 0.0755
Q3	2.472 (0.478, 12.787) 0.2830	3.280 (0.863, 12.465) 0.0812
Q4	5.130 (0.927, 28.390) 0.0638	4.416 (1.197, 16.291) 0.0257
P for trend	0.0379	0.0320

Table 5. Associations between dietary inflammatory indices and congestive heart failure in the population with depressive symptoms (exclude the population with HTN, DM, and cancer).

$P=0.009$) with a trend P -value of 0.007, indicating that although the risk for Q4 is lower compared to previous models, it remains significantly higher than Q1; finally, trend analysis across all models shows that the OR tends to increase as DII quartile levels rise from Q1 to Q4, particularly evident in the Q4 group, which represents the highest quartile.

Subgroup analyses stratified by age and race revealed differing relationships between DII and congestive heart failure. Among participants aged over 60 years, there was a significant positive association between DII and congestive heart failure, with an OR of 1.116 (95% CI: 1.022, 1.218), suggesting that older adults may be particularly susceptible to the inflammatory effects of diet, which could elevate their risk of heart failure. In contrast, no significant correlation was observed in individuals under 60 years of age, indicating that the relationship between the DII and congestive heart failure may differ across various age groups. Furthermore, the findings did not reveal statistical significance among other racial groups, highlighting the necessity for more in-depth studies to explore the dietary influences on congestive heart failure across diverse populations. This underscores the importance of tailoring dietary recommendations and interventions to account for age and racial differences in susceptibility to diet-related inflammation and heart failure risk, as shown in Table 4.

Table 5 presents the associations between dietary inflammatory indices and CHF in individuals exhibiting depressive symptoms, while excluding those with hypertension (HTN), diabetes mellitus (DM), and cancer. We conducted an analysis on the remaining population, and the results are summarized in the following table. Both the weighted logistic regression model and the sensitivity analysis (conducted without weighting) reveal consistent trends. In the weighted model, the dietary inflammatory index (DII) is linked to an elevated risk of CHF, with an odds ratio (OR) of 1.295 (95% confidence interval [CI]: 1.003 to 1.671), and a P -value of 0.0496.

Similarly, in the unweighted model, the DII is associated with an increased risk of CHF, presenting an OR of 1.271 (95% CI: 1.020 to 1.585) and a P-value of 0.033. The convergence of findings from both analyses indicates the robustness of the results.

Discussion

In this study, we conducted an analysis of data retrieved from the NHANES database spanning the years 2005–2018. Our findings suggest that individuals who are male, non-Hispanic, obese, and afflicted with depression, alongside engaging in detrimental habits such as smoking and excessive alcohol consumption, as well as possessing underlying medical conditions such as hypertension and diabetes, exhibit a heightened propensity toward developing congestive heart failure. Notably, a significant positive correlation emerged between DII scores and the incidence of congestive heart failure among depressed patients, particularly among individuals aged 60 years and above.

Previous research has demonstrated a marked positive association between depression and cardiovascular disease^{9,32}. CHF, a prevalent cardiovascular ailment, often coexists with depression and anxiety in patients³³. Despite mounting evidence indicating a significant link between depression and cardiovascular disease, the precise risk factors governing the interaction between these two conditions remain elusive. Unhealthy lifestyle choices, such as inadequate exercise³⁴ or failure to complete cardiac rehabilitation subsequent to the onset of cardiovascular disease³⁵, may constitute contributing risk factors.

Furthermore, unhealthy dietary patterns have been posited as potential risk factors for cardiovascular disease among depressed patients³⁵. However, few studies exist on this subject, leaving the role of dietary habits in the interplay between depression and congestive heart failure ambiguous. In recent years, the DII has emerged as a widely utilized metric for assessing the impact of dietary habits on health outcomes^{36,37}. Depressed individuals frequently consume diets rich in high-calorie foods³⁸, consequently increasing their DII scores. This increase in DII score has been associated with increased incidence of cardiovascular disease^{39,40}. Hence, we postulated that dietary habits might mediate the interaction between depression and congestive heart failure, a hypothesis corroborated by our findings. In our study, we observed significant variability in the association between the DII and CHF across different age and racial/ethnic groups. Specifically, older adults (over 60 years) exhibited a stronger positive association between DII and CHF compared to younger individuals. This finding may be attributed to several biological and lifestyle factors. Aging is associated with a decline in immune function and an increase in chronic low-grade inflammation^{41,42}, which could amplify the impact of pro-inflammatory diets on cardiovascular health. Additionally, reduced physical activity levels and changes in gut microbiota observed in older adults^{43,44} can both influence inflammation and CHF risk. A multicenter cohort study conducted in China revealed that individuals over 55 years of age with depression presented a heightened incidence of dietary inflammation¹⁰, echoing the notion that individuals over 60 years old who adhere to a proinflammatory diet are more predisposed to developing CHF.

The precise mechanism by which dietary habits mediate the interaction between depression and congestive heart failure remains elusive. However, systemic chronic inflammation likely serves as a primary causative factor. Patients with depression often endure prolonged inflammatory stimulation⁴⁵, and an elevated DII score indicative of heightened proinflammatory dietary habits may exacerbate chronic inflammation¹⁸. Chronic inflammatory stimuli increase the risk of congestive heart failure and impede cardiac repair⁸. Additionally, DII scores have been linked to increased obesity risk⁴⁶, with heightened DII scores among depressed patients potentially contributing to obesity onset and, consequently, congestive heart failure^{47,48}. Furthermore, dietary patterns, such as the Mediterranean diet or a diet low in DII, may influence the development of both depression and CHF by mitigating inflammation and oxidative stress—two key contributing factors. A healthy diet's positive impact on gut health may further contribute to both mood regulation and cardiovascular function. While these mechanisms offer promise, further research is crucial to fully elucidate the complex interplay between diet, depression, and CHF. This will enable the development of more targeted dietary recommendations for individuals experiencing these co-occurring conditions. Specifically, further research is warranted to explore the impact of dietary factors on CHF occurrence and progression in individuals with depression^{8,45,49}.

Studies have shown that depressive symptoms not only affect the quality of life in the elderly population but also may have a significant impact on the prognosis of heart failure⁵⁰. In patients with heart failure, depressive symptoms are considered a strong predictor of mortality, indicating that depressive symptoms may exacerbate the condition of patients with heart failure⁵⁰. Moreover, there is an association between depressive symptoms and cognitive dysfunction in patients with heart failure. Inflammation is believed to be an important factor leading to cognitive dysfunction, as it may affect cognitive function by reducing cerebral blood flow⁵¹. In patients with heart failure, depressive symptoms are also related to the relationship between appetite and health status. Studies have found that depressive symptoms may moderate the relationship between appetite and health status, indicating that in patients without depressive symptoms, higher levels of appetite are associated with better health status⁵². This suggests that in managing patients with depression, the recognition and management of heart failure symptoms are crucial.

In the investigation of the relationship between depression and cardiovascular diseases, the DII has been found to be positively correlated with CHF. According to a study, individuals with a higher DII score are more likely to develop heart failure, a finding that is particularly applicable to the adult population in the United States⁵³. Additionally, another study explored the relationship between the Mediterranean diet and heart failure, revealing that higher adherence to the Mediterranean diet is negatively associated with the severity of heart failure, suggesting that dietary interventions may be an effective approach in the treatment of heart failure⁵⁴. Differences in the incidence and severity of heart failure also exist among various racial and ethnic groups. For example, one study found that Hispanic patients have higher heart failure hospitalization rates compared to non-Hispanic white patients, which may be related to socioeconomic status and access to healthcare⁵⁵. Moreover,

another study indicated that although Hispanic patients have similar quality of heart failure treatment and care compared to non-Hispanic white patients, they have higher survival rates during hospitalization, which may reflect certain advantages in medical interventions⁵⁶.

The strength of our study lies in its utilization of the U.S. national database, which offers precise and substantial sample data. Our study provides groundwork for early prevention and treatment of congestive heart failure in depressed patients. This insight can aid medical practitioners in effectively mitigating congestive heart failure risk among depressed individuals through targeted dietary adjustments. Nonetheless, our study is not without limitations. As this was a cross-sectional study reliant on questionnaire data from the NHANES database, numerous confounding factors may influence the reliability of the results. Larger, more accurate prospective studies are imperative to delve deeper into the role of dietary habits in the depression-CHF relationship.

Conclusion

In summary, our study, which is based on NHANES database analysis, underscores the potential role of dietary habits fostering systemic chronic inflammation as a risk factor for congestive heart failure among individuals with depression, particularly those aged 60 years and above. Behavioral modifications hold promise for preventing and treating congestive heart failure⁵⁷. This study provides a foundation for future adjustments in dietary habits to more accurately reduce congestive heart failure risk among depressed patients and reveals a novel avenue for investigating the potential mechanisms underlying systemic chronic inflammation induced by dietary habits in disease relationships.

Data availability

All the data can be obtained from the open source platform provided in the article. The datasets used and analyzed in the current study are publicly available on the NHANES website (<https://wwwn.cdc.gov/nchs/nhanes/Default.aspx>).

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Author contributions

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Declarations

Competing interests

The authors declare no competing interests.

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