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The Dietary Inflammatory Index, Shiftwork, and Depression: Results from NHANES

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Abstract

Objectives—Abnormal physiology (e.g., inflammation), brought on by environmental exposures (e.g., diet or shiftwork [SW]), can affect numerous bodily systems, including the brain, and may be associated with depressive symptomatology. The study examined the associations between shiftwork and depressive symptoms and diet-related inflammation (estimated by the Dietary Inflammatory Index [DII]^{TM}) and depressive symptoms. Additionally, diet was examined as a mediator between shiftwork and depressive symptoms.

Methods—Data were obtained from the United States National Health and Nutrition Examination Survey (2005–2012). SW data were based on self-report. Dietary data were collected using 24-hour dietary recalls for DII calculation. Depressive symptoms were defined using a cut-point of 10 (moderate) on the Patient Health Questionnaire-9. Logistic regression was used to estimate odds ratios and 95% confidence intervals (95%CI) for depressive symptoms by SW and DII quartiles.

Results—DII scores were associated with depressive symptoms among women. Women in DII quartile 4 were 30% more likely to report depressive symptoms than women in quartile 1 (95%CI=1.00–1.68). There was no association between symptoms and SW when using a PHQ-9 cut-point of 10. When using a cut-point of 5 (mild depressive symptoms), those working any form of shiftwork were more likely to suffer from mild symptoms than day workers (odds ratio=1.22; 95%CI=1.04–1.43). There was some evidence for mediation by the DII between SW and depressive symptoms.

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Disclosure: Dr. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counseling and dietary intervention in clinical settings. Drs. Michael Wirth and Nitin Shivappa are employees of CHI.

Conclusions—Future longitudinal studies should examine effects of reductions in inflammation through diet on depressive symptoms, especially among shiftworkers, to elucidate the role of diet on depression among these groups.

Keywords

Dietary Inflammatory Index; shiftwork; depression; diet; PHQ-9; NHANES

Introduction

The prevalence of depressive symptoms or subsyndromal symptomatic depression was reported to range from around 3 to 16% depending on the population (Ayuso-Mateos, Nuevo, Verdes, Naidoo, & Chatterji, 2010; Judd, Rapaport, Paulus, & Brown, 1994; Rucci et al., 2003). Using data from 68 countries in the World Health Survey, it was found that experiencing depressive symptoms predicted health status (defined using metrics of vision, mobility, self-care, cognition, interpersonal activities, pain, discomfort, sleep and energy, and affect) in a similar manner as brief depression episodes or depression (Ayuso-Mateos, et al., 2010). Additionally, those experiencing subsyndromal symptomatic depression were more likely to develop major depression compared to those without depressive symptoms (Laborde-Lahoz et al., 2015).

These statistics are particularly disconcerting given that, based on the Global Burden of Disease study in 2010, the disability-adjusted life years (DALYs) for depressive disorders was 3.0% out of nearly 2.5 billion estimated total DALYs. The years of life lived with disability (YLD) for depressive disorders was estimated to be 9.3% of all YLDs (Ferrari et al., 2013). More concerning, on a global level, the World Health Organization (WHO) estimates that by 2030, depression will be the leading cause of DALYs globally and that 151 million people will suffer from depression (WHO, 2008).

Depression has been associated with physiological abnormalities that adversely can affect the brain, as well as other physiological systems (Yousofpour et al., 2015). These include processes such as increased production of pro-inflammatory cytokines, reduced insulin sensitivity, endothelial dysfunction, and elevated plasma homocysteine levels (Doyle et al., 2013; Lopresti, Hood, & Drummond, 2013; Poole, Dickens, & Steptoe, 2011). This is partially corroborated by the fact that depression has been associated with inflammation-related conditions such as cardiovascular disease (CVD), cancer, stroke, and diabetes (Berge & Riise, 2015; Kang et al., 2015; Reich, 2008; Yousofpour, et al., 2015).

Acute inflammation is a normal part of the immune response. However, repeated exposure to pro-inflammatory stimuli (e.g., tobacco use, obesity) can lead to a state of chronic inflammation, with diet being one of the strongest moderators of systemic inflammation (Lee, Lee, & Choue, 2013; Libby, 2007). Typically, diets high in fruits and vegetables, whole grains, and fish are associated with lower levels of systemic inflammation; whereas diets high in fats, protein, and simple carbohydrates are associated with higher levels of inflammation (Ahluwalia, Andreeva, Kesse-Guyot, & Hercberg, 2013). The Dietary Inflammatory Index (DII)^{TM} was developed to capture the inflammatory potential of one's diet (Shivappa, Steck, Hurley, Hussey, & Hebert, 2014). Previously, the DII was construct

validated against several different inflammatory markers including c-reactive protein (CRP), interleukin-6 (IL-6) and tumor necrosis factor (TNF) alpha (Shivappa, Steck, et al., 2014; Tabung et al., 2015; Wirth, Burch, Shivappa, Violanti, et al., 2014). Additionally, the DII has been associated with a variety of other conditions commonly associated with inflammation including cancer (Maisonneuve et al., 2015; Shivappa, Jackson, Bennett, & Hebert, 2015; Shivappa, Prizment, et al., 2014; Shivappa, Zucchetto, et al., 2015; Wirth, Shivappa, Steck, Hurley, & Hebert, 2015), CVD (Garcia-Arellano et al., 2015), telomere length(Garcia-Calzon et al., 2015), and mortality (Shivappa, Blair, et al., 2015). There was a 19% lower risk of depressive symptoms, defined using a cut-point of ≥10 on the Center for Epidemiologic Studies Depression-10 scale (CESD-10) among Australian women with more anti-inflammatory compared to pro-inflammatory diets (Shivappa, Schoenaker, Hebert, & Mishra, 2016). Using self-reported new depression diagnoses, there was a 47% increase in depression risk among those in quintile 5 (most pro-inflammatory) compared to quintile 1 (Sanchez-Villegas et al., 2015). The DII has yet to be examined with respect to depressive symptomatology among a representative US sample.

This analysis examined the relationship between the DII and depressive symptomatology using data from the US National Health and Nutrition Examination Survey (NHANES). In previous NHANES analyses, shift workers had more pro-inflammatory diets (i.e., higher DII scores) compared to their day-working counterparts (Wirth, Burch, Shivappa, Steck, et al., 2014). Interestingly, not only do shift workers (e.g., night or rotating shifts) have more pro-inflammatory diets, they also tend to experience more depression or depressive symptomatology than day workers (Bara & Arber, 2009; Kalmbach, Pillai, Cheng, Arnedt, & Drake, 2015; Kim et al., 2013). For example, among female nurses, those working any form of SW had a 52% greater risk of experiencing depressive symptomatology (odds ratio [OR]=1.52, 95% confidence interval [95%CI]=1.38–1.67) (Kim, et al., 2013).

Given that the DII and shiftwork (SW) have been associated with depression, the first objective of this study was to examine the associations between the DII and depression and SW and depression. Additionally, analyses examining DII scores and depression were stratified by gender. Previously, it was found that men have higher DII scores than women (Wirth, Shivappa, Hurley, & Hebert, 2016). Also, women experience more depressive symptoms than men (Grigoriadis & Robinson, 2007). Interestingly, it was recently shown that women experience greater inflammatory biomarker levels, depressive symptoms, and social disconnection when exposed to an inflammatory stimulus compared to men (Moieni et al., 2015). Additionally, DII scores have been shown to be associated with depression among females and not among males in the Whitehall II cohort study (T. Akbaraly et al., 2016). Considering that we previously showed that SW is associated with the DII and that the DII is associated with depressive symptoms, it is possible that the DII may lie on the causal pathway between SW and depressive symptoms. Therefore, the DII was examined as a potential mediator for the relationship between SW and depressive symptoms. Specifically, it was hypothesized that individuals with more pro-inflammatory diets (i.e., higher DII scores) or those participating in SW (e.g., non-daytime schedules) would be more likely to experience depressive symptomatology. Additionally, it was hypothesized that the DII acts as a mediator between SW and depressive symptoms.

Materials and Methods

Study Population

Information on US adults and children who participated in NHANES was collected in two-year cycles using a complex, multistage, probability cross-sectional design. This ensures selection of participants from various geographical locations and racial/ethnic groups. Initially, all participants have an in-home interview during which questionnaire data are obtained. This information covers a wide range of topics including demographic, socioeconomic, medical history, diet, and other lifestyle habits. Participants are then invited to visit a mobile examination center where biological samples are collected and clinical tests are performed. NHANES' methods and protocols are described in more detail elsewhere (CDC, 2013).

The total NHANES adult population for the 2005–2012 cycles was 22,692. Of these, 2,192 lacked dietary information; 1,620 lacked the Patient Health Questionnaire-9 (PHQ-9); and 5 reported caloric intake <100 kcal. The final sample size for all DII-based analyses was 18,875. Compared to those who were excluded, participants were more likely to be male (49% vs. 44%, $p<0.01$), non-Hispanic White (71% vs. 59%, $p<0.01$), married (65% vs. 59%), to have had at least some college education (60% vs. 53%, $p<0.01$), and be former smokers (25% vs. 21%). There were no other differences observed between those excluded and included for the measures listed in Table 1. SW information was not obtained for the 2011–2012 cycle. A total of 4,420 were missing information on SW because it was not reported. Another 7,021 reported not working or did not specify their shift type (sample size for SW analysis = 7,434). Informed consent was obtained from all participants and the study was approved by the National Center for Health Statistics Research and Ethics Review Board.

Depression Outcomes

The primary outcome of interest was depressive symptomatology, as measured by the PHQ-9. The PHQ-9 is a 9-item module from the full PHQ. The 9-questions are scored as 0–3; thus the minimum and maximum values are 0 and 27, respectively. Higher values represent more severe depressive symptomatology (Kroenke, Spitzer, & Williams, 2001). The PHQ-9 was criterion validated against mental health professional interviews and it was found that a score of 10 had a sensitivity and specificity of 88% in predicting major depression (Kroenke, et al., 2001). PHQ-9 scores were dichotomized using a cut-point of 10 for the main analyses. However, subsequent *post-hoc* sensitivity analyses also used cut-points of 5 and 15.

Dietary Inflammatory Index and Shiftwork Exposures

The development and validation of the DII has been described elsewhere (Shivappa, Steck, Hurley, Hussey, & Hebert, 2014; Shivappa, Steck, Hurley, Hussey, Ma, et al., 2014). In short, nearly 2,000 research articles, published between 1950 and 2010, examining the relationship between 45 different food parameters (mostly micro and macro nutrients) that were identified in the search and inflammation were reviewed to derive inflammatory effect scores. These scores were weighted based on study design. Additionally, DII calculation is

linked to a regionally representative world database. The world database contains standard means and deviations for the 45 food parameters from 11 populations around the world (Shivappa, Steck, Hurley, Hussey, & Hebert, 2014). NHANES used 24-hour dietary recalls (24HRs) to obtain dietary information. The DII food parameters available included carbohydrates; protein; fat; alcohol; fiber; cholesterol; saturated, monounsaturated, and polyunsaturated fatty acids; omega3 and omega6 polyunsaturated fatty acids; niacin; vitamins A, B1, B2, B6, B12, C, D, E; iron; magnesium; zinc; selenium; folic acid; beta carotene; and caffeine. A z-score was created for each food parameter by subtracting the world standard means from the participant's estimated intake, then dividing this by its standard deviation. This was then converted to a percentile and centered by doubling the value and subtracting 1. The product of the literature-derived inflammatory effect score and the centered percentile for each food parameter was summed across all food parameters to create the overall DII score. Higher (i.e., more positive) scores indicate more pro-inflammatory diets and negative values are more anti-inflammatory (Shivappa, Steck, Hurley, Hussey, & Hebert, 2014). To control for the effect of total energy intake, the DII was calculated per 1,000 calories of food consumed.

To assess SW, NHANES respondents who were "working at a job or business" or "with a job or business but not at work" were asked a follow-up question on the type of shift they typically work. Response options included: 1) regular daytime, 2) evening shifts, 3) night shifts, 4) rotating shifts, or 5) another schedule. Those responding "another schedule" were removed for lack of additional information. Night (2.5%) and evening (3.0%) shift workers were combined. Respondents who were unemployed (i.e., "looking for work" or "not working at a job or business") were excluded.

Covariates

Self-reported possible confounders included age; race; education; marital status; income; health insurance; perceived health; tobacco use; number of alcoholic drinks consumed per week; daily sleep duration; family smoking status; family history of heart disease; previous diagnoses of diabetes, cancer, arthritis, asthma, or any circulatory condition; BMI based on clinic-measured weight and height (kg/m^2]; waist circumference; and moderate-to-vigorous physical activity (MVPA, time in minutes). As a standard procedure, NHANES truncated age to 80 years for the 2007–2012 cycles and 85 years for the 2005–2006 cycle. To maintain consistency, age was truncated to 80 years for all cycles by the investigators.

Statistical Analyses

Survey design procedures in SAS® (version 9.4, Cary, NC) were used for all analyses. These procedures allow for control of the stratification and clustering employed by NHANES for sampling. For DII-based analyses, 8-year sampling weights were created by multiplying the two-year weights by one-fourth and six-year weights were created by multiplying by one-third for SW analyses. T-tests and chi-square tests compared population characteristics according to those self-reported depressive symptomatology status. Previously, using NHANES data, it was shown that shift workers have more pro-inflammatory diets (Wirth, Burch, Shivappa, Steck, et al., 2014). Considering that this population was slightly different due to exclusions, crude analyses examined the association between the DII and SW in the

current population using linear regression models. Also, crude associations between the DII and CRP were analyzed to confirm that the DII predicts inflammation in this specific NHANES population. All models were *a priori* adjusted for age and race. Initially, variables were entered into the full model if their p-value was <0.20 in bi-variable analyses (i.e., exposure + confounder). From the full model covariates were removed, one at a time, starting with the highest p-value. The final model consisted of the DII, age, race, and all other covariates that led to a 10% change in the OR of the DII; all statistically significant covariates also were retained (see Tables 2 and 3 for full list of adjustments).

Adjusted ORs and 95%CIs were estimated using logistic regression for PHQ-9 scores ≥10, which indicates moderate depression. Ancillary analyses examined PHQ-9 cut-points of 5 and 15 which indicate mild and moderate-severe depressive symptoms, respectively (Kroenke, et al., 2001). For all DII analyses, the primary comparison of interest was between DII quartiles 1 and 4. The p-value for trend was examined using the median values for each quartile. The DII also was analyzed as a continuous variable. For SW analyses, subjects working “night or evening shifts” and “rotating shifts” were compared to those on day shifts. They also were combined to compare any form of shiftwork (i.e., “any shiftwork”) to “day workers”.

To test potential mediation, we performed the Sobel test for mediation, which has been described in detail elsewhere (Sobel, 1990). Briefly, the three models needed for such an analysis included 1) depressive symptoms = SW, 2) DII = SW, and 3) depressive symptoms = SW + DII, with the DII being the mediator and SW the main exposure. All main analyses were stratified by sex.

Finally, it should be noted that 7,021 individuals reported not working at a job or did not provide information on their shift type. To further investigate the possibility of the healthy worker effect, a sensitivity analysis was conducted comparing depressive symptomatology between those not currently working and those working day shifts.

Results

Overall, there was approximately equal representation of males and females (49% vs. 51%, respectively). A majority of the population was European-American (71%), married or living with a partner (64%), had at least some college education (59%), had health insurance (81%), were middle-aged (mean age: 46.9, 95%CI: 46.2–47.6 years), and were overweight (mean BMI: 28.7, 95%CI: 28.5–28.9 kg/m^2, data not tabulated). The DII ranged from −5.62 to +4.82. Compared to those with no depressive symptomatology (i.e., PHQ-9 value <10), those with depressive symptoms were more likely to be female (64% vs. 50%); widowed, divorced or separated (30% vs. 17%); less than high-school educated (29% vs. 16%); perceive their health as poor or fair (51% vs. 14%); smoke (41% vs. 20%) and have a higher BMI (mean BMI: 30.1 vs. 28.6 kg/m^2, Table 1). Additionally, the mean (± standard error [SE]) DII was higher (i.e., more pro-inflammatory) among those with depressive symptomatology based on a PHQ-9 score of ≥10 (0.94 ± 0.06 vs. 0.45 ± 0.03, t value = 9.74, p<0.01). Of all working participants, most (83%) were day workers and 17% were night, evening, or rotating (night or evening = 9%; rotating = 8%) shift workers. As expected, the

mean DII was greater among shift workers compared to day workers (mean DII ± SE: 0.98 ± 0.06 vs. 0.60 ± 0.04, t value = −5.85, p<0.01, data not tabulated). Although not a primary aim of the current analysis, crude associations were examined between DII quartiles and CRP, which was only available for 2005–2010. Those in DII quartile 1 had statistically significantly lower CRP concentrations than those in DII quartile 4 (3.74 ± 0.13 vs. 4.44 ± 0.17 mg/L, t value = −3.73, p<0.01).

After adjustments for numerous factors (see footnotes of Table 2), there was no association between the DII and moderate depressive symptoms (DII quartile 4 compared to 1 OR=1.13, 95%CI=0.92–1.39). However, among women, a relationship was observed for moderate depressive symptoms among those in DII quartile 4 compared to 1 (OR=1.30, 95%CI=1.00–1.68). When analyzing the DII as a continuous variable, for every one-unit increase in DII scores, the odds of depressive symptoms increased by 5% (OR=1.05, 95%CI=1.00–1.10). The p-value for trend was not statistically significant for all subjects or males; however, there was an indication of a trend among women (p=0.056), although it did not quite achieve statistical significance. When PHQ-9 cut-points of 5 (mild) or 15 (moderate-severe) were examined, no associations were observed among men. Among women, odds of a PHQ-9 score of 5 (OR=1.26, 95%CI=1.03–1.54) or 15 (OR=1.68, 95%CI=1.08–2.63) were greater among those in DII quartile 4.

After adjustment for various factors listed in the footnotes of Table 3, no statistically significant differences in SW were observed among those with and without a PHQ-9 score of ≥10. Sensitivity analyses indicated that the same was true for a PHQ-9 cut-point of 15 (moderate-severe). However, for a cut-point of 5, indicating mild depressive symptoms, there was a statistically significantly increased odds that those with depressive symptoms worked night/evening or rotating shifts (OR=1.22, 95%CI=1.04–1.43), or only night/evening shifts (OR=1.29, 95%CI=1.06–1.58) compared to day workers (data not tabulated).

The Sobel test for mediation indicated that in this population the DII was a statistically significant mediator for the relationship between SW and depressive symptoms ($Z' = -3.81$, p<0.01) using all participants and a cut-point of 5 on the PHQ-9. In addition to mediation, effect modification was explored. However, the p-value for the hypothesized interaction between the DII and shiftwork was 0.90. When stratified by any form of shift work or day work, the OR for the DII quartile 4 compared to quartile 1 in predicting depressive symptoms was 1.39 (95%CI=0.91–2.12) for day workers and 1.88 (95%CI=0.94–3.74) for shiftworkers. An additional *post-hoc* analysis was performed to evaluate whether those not working were more likely to suffer from depressive symptoms compared to day shift workers, as this may provide evidence of a healthy worker effect. Those not working had an OR of 1.46 (95%CI=1.12–1.92) compared to day shift workers for a PHQ-9 score of ≥10.

Discussion

This study found that women in DII quartile 4 were more likely to experience depressive symptoms as measured by the PHQ-9 at cut-points of 5 (mild), 10 (moderate), and 15 (moderate-severe). This study also found that shift workers were more likely to experience depression symptoms at a cut-point of 5 (i.e., mild), but not 10 or 15 on the PHQ-9.

Using NHANES data, Dipnall and colleagues observed that, regardless of diabetes status, respondents reporting a more “healthy” diet had between 21% and 32% reduction in prevalence of depressive symptoms, as defined by the PHQ-9 (Dipnall et al., 2015). Other forms of prudent or “healthy” diets (e.g., Mediterranean) were associated with lower risk of depression or depressive symptomatology (Lai et al., 2014; Rienks, Dobson, & Mishra, 2013). Conversely, diets that are less healthy (e.g., Western) typically have been associated with a greater odds of depression (T. N. Akbaraly et al., 2009; Le Port et al., 2012). In general, these findings are in agreement with the current findings, at least those observed among women. It also is unclear as to why these results were restricted to females. It is possible that other factors contribute more to depressive symptoms among males than dietary sources of inflammation. The dietary indices or patterns in the literature cited above were not developed with a focus on inflammation. Healthier dietary patterns tend to be associated with lower levels of inflammation; whereas, more Westernized diets tend to be associated with higher levels of inflammation (Ahluwalia, et al., 2013; Zunszain, Hepgul, & Pariante, 2013). Lucas and colleagues created an inflammatory-based dietary pattern from Nurses’ Health Study data. There was a 41% (95%CI=1.22–1.63) increased risk of depression for the highest quintile compared to the lowest among women (Lucas et al., 2014).

Using data from the “Seguimiento Universidad de Narvarra” (SUN) project from Spain, Sanchez-Villegas and colleagues observed a hazard ratio of 1.47 (95%CI=1.17–1.85) in predicting depression among those in DII quintile 5 (most pro-inflammatory) compared to quintile 1 (Sanchez-Villegas, et al., 2015). Also, Shivappa and colleagues found that the lowest quartile of the DII was associated with a 19% reduction in risk of depressive symptoms as measured by the CESD-10 scale in Australian women (Shivappa, et al., 2016). Similar to the current findings with respect to sex differences, results from the Whitehall II study indicated that, among women only, for a one standard deviation increment in DII scores, odds of recurrent depressive symptoms increased by 66% (95%CI=1.30–2.12), as determined by a score of 16 or greater on the CESD. As in this study, there was no effect in men (T. Akbaraly, et al., 2016). The main differences with the current study is that these three other studies were longitudinal. Although the current analysis was cross-sectional, it is important to note the similarities among these studies, and the fact that the current study was based on a representative US sample.

Mechanisms through which diet may influence depression or depressive symptomatology include effects on neurotransmitters, oxidative stress, the hypothalamic-pituitary-adrenal (HPA) axis, and mitochondrial dysfunction (Lopresti, et al., 2013; Martinez-Gonzalez & Sanchez-Villegas, 2016). Diets associated with higher levels of inflammation (e.g., Western diets) were associated with higher levels of neuro-inflammation (Kanoski & Davidson, 2011). In recent years, a link between CVD, diabetes, metabolic syndrome, obesity, and depression has been suggested (Doyle, et al., 2013; Yousofpour, et al., 2015). These conditions are driven by numerous mechanisms (e.g., insulin resistance and aberrant endocrine and IL-6 responses to stress), some of which are affected by pro-inflammatory dietary choices, and are observed in those with depression (Lopresti, et al., 2013; McInnis et al., 2014).

Previous studies indicate that shiftwork may be associated with depression (Kalmbach, et al., 2015). For example, Bara and Arber observed that men working night shifts for more than 4 years were more likely to report mental ill health or depressive symptoms (OR=2.58, 95%CI=1.22–5.48), as measured by the General Health Questionnaire. Among women, "varied" shifts were associated with depressive symptoms (OR=4.17, 95%CI=1.45–11.98) (Bara & Arber, 2009). Similar to these studies, the current study found that shiftwork was associated with depressive symptoms on the PHQ-9, but only at a cut-point of 5.0, which indicates mild depressive symptoms. However, it is uncertain whether this score among shift workers reflects mild depressive symptoms or fatigue. Using the Korean NHANES, Kim and colleagues observed no association between SW and depressive feelings. However, the study by Kim et al., used only one question to ascertain depressive feelings (Kim, et al., 2013).

One disadvantage of SW from NHANES is that respondents are asked to report on current work status. This raises the potential issue of the healthy worker effect. From a theoretical standpoint, those who reported PHQ-9 scores in the moderate, moderate-severe, or severe categories may no longer be working or have changed occupations or shifts to alleviate symptoms. Although this cannot be confirmed using NHANES data, this hypothesis was partially corroborated by *post-hoc* analyses indicating that those not working had a 46% ($p<0.05$) greater risk of a PHQ-9 score ≥10 compared to day workers. However, it should be noted that these individuals might be experiencing depressive symptoms because they are out of work. Additionally, information on SW duration and modes or extent of SW adaptation were not available for analysis (Burch et al., 2009). Differences in depressive symptom ascertainment, study occupation or population, SW definition, and study design make comparisons to other studies difficult. However, previous literature indicates that common complaints among shiftworkers, include, increased stress, sleep and circadian disruption, and poor social functioning, which are all associated with depression or depressive symptoms (Germain & Kupfer, 2008; Hodgetts, Gallagher, Stow, Ferrier, & O'Brien, 2016).

It was hypothesized that the DII may act as a mediator between SW and depressive symptoms. For this to be true, SW must be associated with the DII as was observed previously (Wirth, Burch, Shivappa, Steck, et al., 2014). Given the difference in exclusion criteria of the current population and a new method of calculating DII scores per 1,000 calories consumed (termed the energy-density method), this relationship was re-examined. In the current population, mean DII values among shift workers were about 32% greater than those observed among day workers (i.e., more pro-inflammatory, $p<0.01$). The findings from this study suggest that the DII may act as a mediator in the relationship between SW and depressive symptoms. If this is confirmed in follow-up research, future intervention strategies to alleviate depression symptoms among shiftworkers or other individuals who experience circadian disruption might consider the incorporation of dietary factors. However, caution is warranted when interpreting results of the mediation analysis. The relationship between the DII and depression was moderate at best, and a PHQ-9 cut-off of 5 was used during the mediation analysis which only indicates mild depressive symptoms.

This study had numerous strengths. This analysis made use of the DII, a dietary tool designed specifically to assess inflammatory potential of the diet. Additionally, this is the

first examination of the relationship between the DII and depression in a national US sample. Similarly, to our knowledge, no previous study examined SW and depression using NHANES. By pooling multiple 2-year cycles together, this study had a large sample size, as well as access to a wide range of potential confounders. Despite its strengths, this study was subject to several limitations. Dietary information was based on one 24HR self-report which may not account for day-to-day variability in diet and may lead to imprecise estimates (Basiotis, Welsh, Cronin, Kelsay, & Mertz, 1987). However, use of the DII in NHANES has been reported previously (Wirth, Burch, Shivappa, Steck, et al., 2014). Additionally, social approval and desirability were previously found to bias dietary self-report (Hebert et al., 1997); these measures were not available for analysis. Also, information on timing of food consumption and patterns of food consumption were not available. Depressive symptoms were not clinically confirmed. However, the PHQ-9 is a validated questionnaire (Kroenke, et al., 2001). The self-reported SW information is limited within NHANES. For example, information on duration in years and frequency (e.g., how many night shifts per month) was not available. However, several publications have used measures of SW from NHANES to examine several outcomes including prostate specific antigen and metabolic syndrome (Flynn-Evans, Mucci, Stevens, & Lockley, 2013; Santhanam, Driscoll, Gress, & Khthir, 2014; Wirth, Burch, Shivappa, Steck, et al., 2014). It should be noted that differences in population characteristics were observed between those excluded due to lack of information compared to those included in the analyses. The cross-sectional nature of the study does not allow for inference on temporal ordering. For example, previous research indicates that depression may lead to changes in dietary patterns, including eating comfort foods, which tend be calorie-dense foods high in sugar and fat (Finch & Tomiyama, 2015). However, it should be noted that previous research indicates that the DII stays relatively constant over a period of 6 years (Tabung et al., 2016). Also, it was not possible to determine if anti-inflammatory diets directly led to a protective effect on depressive symptoms or if those eating more anti-inflammatory diets participate in other healthy behaviors that are protective of depressive symptoms. However, a range of measures were examined as potential confounders. An alternative interpretation is that those currently living with depressive symptoms have more pro-inflammatory diets. Given the amount of research indicating that diet and inflammation are associated with initiation or progression of mental health illness, it is possible that those living with depressive symptoms and eating a pro-inflammatory diet, possibly due to the depressive symptoms, may be putting themselves at increased risk for future mental illness.

In conclusion, this study found that women currently living with depressive symptoms, as measured by the PHQ-9, have more pro-inflammatory diets relative to those without depressive symptoms. Although there is a lack of temporal clarity due to the cross-sectional nature of this study, it does not change the fact that these individuals are consuming unhealthy, pro-inflammatory foods. Additionally, SW was associated with mild depressive symptoms. Dietary inflammatory potential was found to, at least partially, lie on the causal pathway between SW and depressive symptoms. Future longitudinal studies are needed to address these relationships in greater detail to help elucidate the role of diet in development and progression of depressive symptoms among shiftworkers or those experiencing similar risk profiles (i.e., those with sleep disorders).

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Bibliography

Ahluwalia N, Andreeva VA, Kesse-Guyot E, Hercberg S. Dietary patterns, inflammation and the metabolic syndrome. Diabetes Metab. 2013; 39(2):99–110. DOI: 10.1016/j.diabet.2012.08.007 [PubMed: 23062863]

Akbaraly T, Kerlau C, Wyart M, Chevallier N, Ndiaye L, Shivappa N, … Kivimaki M. Dietary inflammatory index and recurrence of depressive symptoms: Results from the Whitehall II Study. Clin Psychol Sci. 2016; 4(6):1125–1134. DOI: 10.1177/2167702616645777 [PubMed: 28070452]

Akbaraly TN, Brunner EJ, Ferrie JE, Marmot MG, Kivimaki M, Singh-Manoux A. Dietary pattern and depressive symptoms in middle age. British Journal of Psychiatry. 2009; 195(5):408–413. DOI: 10.1192/bjp.bp.108.058925 [PubMed: 19880930]

Ayuso-Mateos JL, Nuevo R, Verdes E, Naidoo N, Chatterji S. From depressive symptoms to depressive disorders: the relevance of thresholds. Br J Psychiatry. 2010; 196(5):365–371. DOI: 10.1192/bjp.bp.109.071191 [PubMed: 20435961]

Bara AC, Arber S. Working shifts and mental health--findings from the British Household Panel Survey (1995–2005). Scand J Work Environ Health. 2009; 35(5):361–367. [PubMed: 19688143]

Basiotis PP, Welsh SO, Cronin FJ, Kelsay JL, Mertz W. Number of Days of Food-Intake Records Required to Estimate Individual and Group Nutrient Intakes with Defined Confidence. Journal of Nutrition. 1987; 117(9):1638–1641. [PubMed: 3655942]

Berge LI, Riise T. Comorbidity between Type 2 Diabetes and Depression in the Adult Population: Directions of the Association and Its Possible Pathophysiological Mechanisms. International Journal of Endocrinology. 2015; Artn 164760. doi: 10.1155/2015/164760

Burch JB, Tom J, Zhai YS, Criswell L, Leo E, Ogoussan K. Shiftwork impacts and adaptation among health care workers. Occupational Medicine-Oxford. 2009; 59(3):159–166. DOI: 10.1093/occmed/kqp015

CDC. National Health and Nutrition Examinations Survey data. Hyattsville, MD: National Center for Health Statistics; 2013. Centers for Disease Control and Prevention.

Dipnall JF, Pasco JA, Meyer D, Berk M, Williams LJ, Dodd S, Jacka FN. The association between dietary patterns, diabetes and depression. Journal of Affective Disorders. 2015; 174:215–224. DOI: 10.1016/j.jad.2014.11.030 [PubMed: 25527991]

Doyle TA, de Groot M, Harris T, Schwartz F, Strotmeyer ES, Johnson KC, Kanaya A. Diabetes, depressive symptoms, and inflammation in older adults: results from the Health, Aging, and Body Composition Study. J Psychosom Res. 2013; 75(5):419–424. DOI: 10.1016/j.jpsychores.2013.08.006 [PubMed: 24182629]

Ferrari AJ, Charlson FJ, Norman RE, Patten SB, Freedman G, Murray CJ, … Whiteford HA. Burden of depressive disorders by country, sex, age, and year: findings from the global burden of disease study 2010. PLoS Med. 2013; 10(11):e1001547.doi: 10.1371/journal.pmed.1001547 [PubMed: 24223526]

Finch LE, Tomiyama AJ. Comfort eating, psychological stress, and depressive symptoms in young adult women. Appetite. 2015; 95:239–244. DOI: 10.1016/j.appet.2015.07.017 [PubMed: 26192221]

Flynn-Evans EE, Mucci L, Stevens RG, Lockley SW. Shiftwork and prostate-specific antigen in the National Health and Nutrition Examination Survey. J Natl Cancer Inst. 2013; 105(17):1292–1297. DOI: 10.1093/jnci/djt169 [PubMed: 23943864]

Garcia-Arellano A, Ramallal R, Ruiz-Canela M, Salas-Salvado J, Corella D, Shivappa N, … Predimed I. Dietary Inflammatory Index and Incidence of Cardiovascular Disease in the PREDIMED Study. Nutrients. 2015; 7(6):4124–4138. DOI: 10.3390/nu7064124 [PubMed: 26035241]

Garcia-Calzon S, Zalba G, Ruiz-Canela M, Shivappa N, Hebert JR, Martinez JA, … Marti A. Dietary inflammatory index and telomere length in subjects with a high cardiovascular disease risk from the PREDIMED-NAVARRA study: cross-sectional and longitudinal analyses over 5 y. Am J Clin Nutr. 2015; 102(4):897–904. DOI: 10.3945/ajcn.115.116863 [PubMed: 26354530]

Germain A, Kupfer DJ. Circadian rhythm disturbances in depression. Hum Psychopharmacol. 2008; 23(7):571–585. DOI: 10.1002/hup.964 [PubMed: 18680211]

Grigoriadis S, Robinson GE. Gender issues in depression. Ann Clin Psychiatry. 2007; 19(4):247–255. DOI: 10.1080/10401230701653294 [PubMed: 18058282]

Hebert JR, Ma Y, Clemow L, Ockene IS, Saperia G, Stanek EJ 3rd, … Ockene JK. Gender differences in social desirability and social approval bias in dietary self-report. Am J Epidemiol. 1997; 146(12):1046–1055. [PubMed: 9420529]

Hodgetts S, Gallagher P, Stow D, Ferrier IN, O'Brien JT. The impact and measurement of social dysfunction in late-life depression: an evaluation of current methods with a focus on wearable technology. Int J Geriatr Psychiatry. 2016; doi: 10.1002/gps.4632

Judd LL, Rapaport MH, Paulus MP, Brown JL. Subsyndromal symptomatic depression: a new mood disorder? J Clin Psychiatry. 1994; 55(Suppl):18–28.

Kalmbach DA, Pillai V, Cheng P, Arnedt JT, Drake CL. Shift work disorder, depression, and anxiety in the transition to rotating shifts: the role of sleep reactivity. Sleep Med. 2015; 16(12):1532–1538. DOI: 10.1016/j.sleep.2015.09.007 [PubMed: 26611952]

Kang HJ, Kim SY, Bae KY, Kim SW, Shin IS, Yoon JS, Kim JM. Comorbidity of depression with physical disorders: research and clinical implications. Chonnam Med J. 2015; 51(1):8–18. DOI: 10.4068/cmj.2015.51.1.8 [PubMed: 25914875]

Kanoski SE, Davidson TL. Western diet consumption and cognitive impairment: links to hippocampal dysfunction and obesity. Physiol Behav. 2011; 103(1):59–68. DOI: 10.1016/j.physbeh.2010.12.003 [PubMed: 21167850]

Kim I, Kim H, Lim S, Lee M, Bahk J, June KJ, … Chang WJ. Working hours and depressive symptomatology among full-time employees: Results from the fourth Korean National Health and Nutrition Examination Survey (2007–2009). Scandinavian Journal of Work Environment & Health. 2013; 39(5):515–520. DOI: 10.5271/sjweh.3356

Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001; 16(9):606–613. [PubMed: 11556941]

Laborde-Lahoz P, El-Gabalawy R, Kinley J, Kirwin PD, Sareen J, Pietrzak RH. Subsyndromal depression among older adults in the USA: prevalence, comorbidity, and risk for new-onset psychiatric disorders in late life. Int J Geriatr Psychiatry. 2015; 30(7):677–685. DOI: 10.1002/gps.4204 [PubMed: 25345806]

Lai JS, Hiles S, Bisquera A, Hure AJ, McEvoy M, Attia J. A systematic review and meta-analysis of dietary patterns and depression in community-dwelling adults. American Journal of Clinical Nutrition. 2014; 99(1):181–197. DOI: 10.3945/ajcn.113.069880 [PubMed: 24196402]

Le Port A, Gueguen A, Kesse-Guyot E, Melchior M, Lemogne C, Nabi H, … Czernichow S. Association between Dietary Patterns and Depressive Symptoms Over Time: A 10-Year Follow-Up Study of the GAZEL Cohort. Plos One. 2012; 7(12) ARTNe51593. doi: 10.1371/journal.pone.0051593

Lee H, Lee IS, Choue R. Obesity, inflammation and diet. Pediatr Gastroenterol Hepatol Nutr. 2013; 16(3):143–152. DOI: 10.5223/pghn.2013.16.3.143 [PubMed: 24224147]

Libby P. Inflammatory mechanisms: the molecular basis of inflammation and disease. Nutr Rev. 2007; 65(12 Pt 2):S140–146. [PubMed: 18240538]

Lopresti AL, Hood SD, Drummond PD. A review of lifestyle factors that contribute to important pathways associated with major depression: Diet, sleep and exercise. Journal of Affective Disorders. 2013; 148(1):12–27. DOI: 10.1016/j.jad.2013.01.014 [PubMed: 23415826]

Lucas M, Chocano-Bedoya P, Shulze MB, Mirzaei F, O'Reilly EJ, Okereke OI, … Ascherio A. Inflammatory dietary pattern and risk of depression among women. Brain Behavior and Immunity. 2014; 36:46–53. DOI: 10.1016/j.bbi.2013.09.014

Maisonneuve P, Shivappa N, Hebert JR, Bellomi M, Rampinelli C, Bertolotti R, … Gnagnarella P. Dietary inflammatory index and risk of lung cancer and other respiratory conditions among heavy smokers in the COSMOS screening study. Eur J Nutr. 2015; doi: 10.1007/s00394-015-0920-3

Martinez-Gonzalez MA, Sanchez-Villegas A. 2016; Food patterns and the prevention of depression. Proc Nutr Soc. :1–8. DOI: 10.1017/S0029665116000045 [PubMed: 26282529]

McInnis CM, Thoma MV, Gianferante D, Hanlin L, Chen X, Breines JG, … Rohleder N. Measures of adiposity predict interleukin-6 responses to repeated psychosocial stress. Brain Behav Immun. 2014; 42:33–40. DOI: 10.1016/j.bbi.2014.07.018 [PubMed: 25107874]

Moieni M, Irwin MR, Jevtic I, Olmstead R, Breen EC, Eisenberger NI. Sex differences in depressive and socioemotional responses to an inflammatory challenge: implications for sex differences in depression. Neuropsychopharmacology. 2015; 40(7):1709–1716. DOI: 10.1038/npp.2015.17 [PubMed: 25598426]

Poole L, Dickens C, Steptoe A. The puzzle of depression and acute coronary syndrome: reviewing the role of acute inflammation. J Psychosom Res. 2011; 71(2):61–68. DOI: 10.1016/j.jpsychores.2010.12.009 [PubMed: 21767684]

Reich M. Depression and cancer: recent data on clinical issues, research challenges and treatment approaches. Current Opinion in Oncology. 2008; 20(4):353–359. [PubMed: 18525327]

Rienks J, Dobson AJ, Mishra GD. Mediterranean dietary pattern and prevalence and incidence of depressive symptoms in mid-aged women: results from a large community-based prospective study. European Journal of Clinical Nutrition. 2013; 67(1):75–82. DOI: 10.1038/ejcn.2012.193 [PubMed: 23212131]

Rucci P, Gherardi S, Tansella M, Piccinelli M, Berardi D, Bisoffi G, … Pini S. Subthreshold psychiatric disorders in primary care: prevalence and associated characteristics. J Affect Disord. 2003; 76(1–3):171–181. [PubMed: 12943947]

Sanchez-Villegas A, Ruiz-Canela M, de la Fuente-Arrillaga C, Gea A, Shivappa N, Hebert JR, Martinez-Gonzalez MA. Dietary inflammatory index, cardiometabolic conditions and depression in the Seguimiento Universidad de Navarra cohort study. Br J Nutr. 2015; 114(9):1471–1479. DOI: 10.1017/S0007114515003074 [PubMed: 26344165]

Santhanam P, Driscoll HK, Gress TW, Khthir R. Metabolic disease and shift work: is there an association? An analysis of NHANES data for 2007–2008. Occup Environ Med. 2014; 71(9):661–662. DOI: 10.1136/oemed-2014-102352

Shivappa N, Blair CK, Prizment AE, Jacobs DR Jr, Steck SE, Hebert JR. Association between inflammatory potential of diet and mortality in the Iowa Women's Health study. Eur J Nutr. 2015; doi: 10.1007/s00394-015-0967-1

Shivappa N, Jackson MD, Bennett F, Hebert JR. Increased Dietary Inflammatory Index (DII) Is Associated With Increased Risk of Prostate Cancer in Jamaican Men. Nutr Cancer. 2015; 67(6):941–948. DOI: 10.1080/01635581.2015.1062117 [PubMed: 26226289]

Shivappa N, Prizment AE, Blair CK, Jacobs DR Jr, Steck SE, Hebert JR. Dietary inflammatory index and risk of colorectal cancer in the Iowa Women's Health Study. Cancer Epidemiol Biomarkers Prev. 2014; 23(11):2383–2392. DOI: 10.1158/1055-9965.EPI-14-0537 [PubMed: 25155761]

Shivappa N, Schoenaker DA, Hebert JR, Mishra GD. Association between inflammatory potential of diet and risk of depression in middle-aged women: the Australian Longitudinal Study on Women's Health. Br J Nutr. 2016; 116(6):1077–1086. DOI: 10.1017/S0007114516002853 [PubMed: 27498949]

Shivappa N, Steck SE, Hurley TG, Hussey JR, Hebert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr. 2014; 17(8):1689–1696. DOI: 10.1017/S1368980013002115 [PubMed: 23941862]

Shivappa N, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS, … Hebert JR. A population-based dietary inflammatory index predicts levels of C-reactive protein in the Seasonal Variation of Blood Cholesterol Study (SEASONS). Public Health Nutr. 2014; 17(8):1825–1833. DOI: 10.1017/S1368980013002565 [PubMed: 24107546]

Shivappa N, Zucchetto A, Montella M, Serraino D, Steck SE, La Vecchia C, Hebert JR. Inflammatory potential of diet and risk of colorectal cancer: a case-control study from Italy. Br J Nutr. 2015; 114(1):152–158. DOI: 10.1017/S0007114515001828 [PubMed: 26050563]

Sobel ME. Effect Analysis and Causation in Linear Structural Equation Models. Psychometrika. 1990; 55(3):495–515. DOI: 10.1007/Bf02294763

Tabung FK, Steck SE, Zhang J, Ma Y, Liese AD, Agalliu I, … Hebert JR. Construct validation of the dietary inflammatory index among postmenopausal women. Ann Epidemiol. 2015; 25(6):398–405. DOI: 10.1016/j.annepidem.2015.03.009 [PubMed: 25900255]

Tabung FK, Steck SE, Zhang J, Ma Y, Liese AD, Tylavsky FA, … Hebert JR. Longitudinal changes in the dietary inflammatory index: an assessment of the inflammatory potential of diet over time in postmenopausal women. Eur J Clin Nutr. 2016; doi: 10.1038/ejcn.2016.116

WHO. The Global Burden of Disease 2004 Update. World Health Organization; Geneva, Switzerland: 2008.

Wirth MD, Burch J, Shivappa N, Steck SE, Hurley TG, Vena JE, Hebert JR. Dietary inflammatory index scores differ by shift work status: NHANES 2005 to 2010. J Occup Environ Med. 2014; 56(2):145–148. DOI: 10.1097/JOM.0000000000000088 [PubMed: 24451608]

Wirth MD, Burch J, Shivappa N, Violanti JM, Burchfiel CM, Fekedulegn D, … Hebert JR. Association of a dietary inflammatory index with inflammatory indices and metabolic syndrome among police officers. J Occup Environ Med. 2014; 56(9):986–989. DOI: 10.1097/JOM.0000000000000213 [PubMed: 25046320]

Wirth MD, Shivappa N, Hurley TG, Hebert JR. Association between previously diagnosed circulatory conditions and a dietary inflammatory index. Nutr Res. 2016; 36(3):227–233. DOI: 10.1016/j.nutres.2015.11.016 [PubMed: 26923509]

Wirth MD, Shivappa N, Steck SE, Hurley TG, Hebert JR. The dietary inflammatory index is associated with colorectal cancer in the National Institutes of Health-American Association of Retired Persons Diet and Health Study. Br J Nutr. 2015; 113(11):1819–1827. DOI: 10.1017/S000711451500104X [PubMed: 25871645]

Yousofpour M, Kamalinejad M, Esfahani MM, Shams J, Tehrani HH, Bahrami M. Role of Heart and its Diseases in the Etiology of Depression According to Avicenna's Point of View and its Comparison with Views of Classic Medicine. Int J Prev Med. 2015; 6:49.doi: 10.4103/2008-7802.158178 [PubMed: 26124946]

Zunszain PA, Hepgul N, Pariante CM. Inflammation and depression. Curr Top Behav Neurosci. 2013; 14:135–151. DOI: 10.1007/7854_2012_211 [PubMed: 22553073]

Table 1

Population Characteristics by Depression Severity Categorization Based on the Depression Screener from the Patient Health Questionnaire, NHANES, 2005–2012.

Characteristic	Depressive Symptoms Present[1] (n=1,648)	No Depressive Symptoms (n=17,227)	p-value
Sex			<0.01
Male	595 (36%)	8727 (50%)	
Female	1053 (64%)	8500 (50%)	
Race			<0.01
Non-Hispanic White	716 (64%)	8145 (71%)	
Non-Hispanic Black	380 (15%)	3667 (11%)	
Mexican American	263 (8%)	2818 (8%)	
Other	289 (13%)	2597 (10%)	
Marital Status			<0.01
Married/Living with Partner	761 (49%)	10547 (65%)	
Widowed/Divorced/Separated	548 (30%)	3660 (17%)	
Never Married	339 (21%)	3010 (17%)	
Education			<0.01
<High School	639 (29%)	4386 (16%)	
Completed High School	400 (27%)	4012 (23%)	
Some College	462 (32%)	4920 (31%)	
≥College Degree	147 (12%)	3894 (29%)	
Perceived Health			<0.01
Excellent	61 (4%)	2546 (18%)	
Very Good	165 (12%)	4856 (34%)	
Good	481 (33%)	6318 (35%)	
Fair or Poor	941 (51%)	3500 (14%)	
Smoking Family Member			<0.01
Yes	546 (34%)	2686 (15%)	
No	1093 (66%)	14469 (85%)	
Smoking Status			<0.01
Current	650 (41%)	3431 (20%)	
Former	338 (20%)	4372 (25%)	
Never	660 (39%)	9417 (55%)	
Infection[2]			<0.01
Yes	651 (38%)	3891 (22%)	
No	987 (62%)	13298 (78%)	
Previous Cancer Diagnosis			0.02
Yes	179 (12%)	1583 (9%)	
No	1466 (88%)	15626 (91%)	
Previous Arthritis Diagnosis			<0.01
Yes	707 (39%)	4406 (24%)	
No	939 (61%)	12791 (76%)	

Characteristic	Depressive Symptoms Present[1] (n=1,648)	No Depressive Symptoms (n=17,227)	p-value
	Continuous Measures (Mean and 95% Confidence Interval)		
Age	45.7 (45.0–46.4)	47.0 (46.3–47.7)	<0.01
Sleep Duration (Hours)	6.3 (6.2–6.4)	6.9 (6.9 – 7.0)	<0.01
Body Mass Index (kg/m^2)	30.1 (29.6–30.7)	28.6 (28.4–28.8)	<0.01
Dietary Inflammatory Index	0.94 (0.83–1.05)	0.45 (0.38–0.52)	<0.01

Column percentages may not equal 100% due to rounding. Stratum numbers may not equal column totals due to missing data. All categorical variable p-values based on chi-square tests and all continuous p-values based on t-tests.

[1] A score ≥10 indicates at least moderate depressive symptomatology.

[2] Cold, stomach flu, or ear infection in the last 30 days.

Table 2

DII Quartile ORs (95%CI) for Presence of Depressive Symptomatology, NHANES, 2005–2012.

DII Quartile	Present[1]	Absent	Crude OR (95%CI)	Adjusted OR (95%CI)
All Subjects				
1	316 (17%)	4,465 (26%)	1.0 (Referent)	1.0 (Referent)
2	387 (22%)	4,472 (25%)	1.32 (1.08–1.59)	1.08 (0.87–1.33)
3	424 (27%)	4,244 (25%)	1.65 (1.31–2.08)	1.14 (0.87–1.49)
4	521 (33%)	4,046 (24%)	2.01 (1.69–2.39)	1.13 (0.92–1.39)
Females				
1	219 (19%)	2,575 (30%)	1.0 (Referent)	1.0 (Referent)
2	255 (23%)	2,234 (26%)	1.39 (1.09–1.78)	1.11 (0.85–1.45)
3	259 (25%)	1,897 (22%)	1.75 (1.40–2.19)	1.14 (0.88–1.47)
4	320 (32%)	1,794 (21%)	2.37 (1.91–2.95)	1.30 (1.00–1.68)
Males				
1	97 (13%)	1,890 (21%)	1.0 (Referent)	1.0 (Referent)
2	132 (21%)	2,238 (25%)	1.33 (0.92–1.92)	1.13 (0.77–1.65)
3	165 (32%)	2,347 (27%)	1.82 (1.19–2.79)	1.31 (0.81–2.13)
4	201 (34%)	2,252 (27%)	1.94 (1.37–2.75)	1.09 (0.73–1.63)

Column percentages may not equal 100% due to rounding. Column percentages based on weighted frequencies.

[1] A score ≥10 indicates at least moderate depressive symptomatology.

DII Quartile Ranges: 1 = −5.30 to −0.84, 2 = −0.83 to 0.73, 3 = 0.74 to 1.98, 4 = 1.99 to 4.82. **Adjustments**: race, education, marital status, perceived health, current infection status, family history of smoking, smoking status, past cancer diagnosis, arthritis, age, and average nightly sleep duration. **Abbreviations**: DII = Dietary Inflammatory Index.

Table 3

Shiftwork ORs (95%CI) for Presence of Depressive Symptomatology, NHANES, 2005–2012.

DII Quartile	Present[1]	Absent	Crude OR (95%CI)	Adjusted OR (95%CI)
All Subjects				
Day Shift	304 (78%)	5731 (83%)	1.0 (Referent)	1.0 (Referent)
Night or Evening	63 (14%)	694 (9%)	1.70 (1.23–2.37)	1.33 (0.90–1.96)
Rotating	43 (8%)	599 (8%)	1.07 (0.75–1.53)	0.90 (0.64–1.26)
Combined Night/Rotating	106 (22%)	1293 (17%)	1.40 (1.11–1.77)	1.13 (0.86–1.50)
Females				
Day Shift	193 (78%)	2628 (84%)	1.0 (Referent)	1.0 (Referent)
Night or Evening	37 (13%)	302 (8%)	1.70 (1.08–2.68)	1.24 (0.75–2.06)
Rotating	31 (9%)	275 (8%)	1.17 (0.72–1.91)	0.91 (0.59–1.39)
Combined Night/Rotating	68 (22%)	577 (16%)	1.44 (1.01–2.03)	1.08 (0.74–1.57)
Males				
Day Shift	111 (78%)	3103 (83%)	1.0 (Referent)	1.0 (Referent)
Night or Evening	26 (16%)	392 (9%)	1.83 (1.02–3.28)	1.41 (0.74–2.68)
Rotating	12 (6%)	324 (8%)	0.86 (0.46–1.61)	0.80 (0.43–1.49)
Combined Night/Rotating	38 (22%)	716 (17%)	1.40 (0.87–2.23)	1.17 (0.73–1.90)

Column percentages may not equal 100% due to rounding. Column percentages based on weighted frequencies.

[1] A score ≥10 indicates at least moderate depressive symptomatology.

Adjustments: marital status, perceived health, current infections, smoking status, family history of heart disease, past cancer diagnosis, average nightly sleep duration, BMI and waist circumference. Combined Night/Rotating is the combined categories of Night or Evening and Rotating.