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Associations between dietary quality, noise, and hearing: data from the National Health and Nutrition Examination Survey, 1999-2002

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Introduction

Noise-induced hearing loss (NIHL) is a global issue, and much of the burden is a function of occupational noise insult. According to one report, 16% of disabling hearing loss in adults worldwide is attributed to occupational noise, with 7 to 21% affected across different geographical regions (Nelson et al., 2005). In the USA, NIHL has long been one of the most common occupational diseases and one of the most reported occupational injuries (NIOSH, 2001). Nationally representative data document ongoing relationships of noise to hearing in the US population (Choi et al., 2012; Mahboubi et al., 2013). Because there are no international standards for audiometric surveillance and no uniform reporting criteria (Rabinowitz et al., 2012), it is difficult to precisely identify the number affected and the total economic impact. However, business cost models suggest NIHL carries a large financial cost (Tufts et al., 2010; Lahiri et al., 2011), and hearing loss is clearly an important public health issue (Stevens et al., 2011).

Given the significance of the issue of NIHL, multiple therapeutics have been assessed for potential protection of the human ear against noise insult (Attias et al., 1994; Attias et al., 2004; Quaranta et al., 2004; Kramer et al., 2006; Lin et al., 2010; Le Prell et al., 2011c; Quaranta et al., 2012). Other clinical investigations are underway (NCT00808470; NCT01345474; NCT01444846)¹. It is worth note that many of the above ongoing and completed studies assess molecules that would commonly be obtained from dietary sources. In clinical otoprotection trials to date, the agents have often been delivered at higher levels than would typically be achieved via dietary intake alone. These supplemented doses sharply contrast with dietary intake, and several prospective studies assessing the association of vitamin intake with hearing in the general population have not suggested benefit (Shargorodsky et al., 2010; Polanski & Cruz, 2013). However, multiple epidemiological studies suggest healthy eating habits which provide recommended nutrient intake amounts may confer some long-term benefit, with the specific nutrients and dietary components assessed varying across analyses (Gopinath et al., 2010b; Gopinath et al., 2011; Spankovich

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et al., 2011; Choi et al., 2013; Heine et al., 2013; Péneau et al., 2013; Spankovich & Le Prell, 2013).

Although a variety of data support the possibility that diet influences hearing status, review of the literature reveals mixed support regarding specific nutrients and the relationship between single nutrient intake and hearing (described in detail in Le Prell & Spankovich, 2013). Some of the nutrients suggested to play a role in human hearing are vitamins, including vitamins A, B (specifically B₂, B₉, and B₁₂), C, and E (Weijl et al., 2004; Durga et al., 2007; Michikawa et al., 2009; Gopinath et al., 2011; Spankovich et al., 2011; Choi et al., 2013). However, there are also studies that report no statistically significant relationships between these specific vitamins and hearing (Berner et al., 2000; Michikawa et al., 2009; Shargorodsky et al., 2010; Gopinath et al., 2011; Péneau et al., 2013). Some of the minerals suggested to play a role in hearing include magnesium (Mg) (Joachims et al., 1993; Attias et al., 1994; 2004; Spankovich et al., 2011; Choi et al., 2013) and selenium (Se) (Weijl et al., 2004; Chuang et al., 2007), although there are also studies reporting no statistically significant relationship between Mg and hearing (Walden et al., 2000). One of the key challenges for single-nutrient analyses is that diets include a complex combination of nutrients that have complex interactions. Macronutrient intake has also been suggested to be related to human hearing, with higher carbohydrate intake, fat intake, and cholesterol intake all being associated with negative hearing outcomes (Rosen & Olin, 1965; Rosen et al., 1970; Evans et al., 2006; Gopinath et al., 2010a). More recent studies assessing the relationship between dietary fat and hearing have built on the results, with more recent studies showing consumption of polyunsaturated fatty acids (the “healthier” fats) are associated with better hearing (Dullemeijer et al., 2010; Gopinath et al., 2010b; Spankovich et al., 2011). In another recent study, higher levels of retinol and vitamin B₁₂ had a statistically significant association with better hearing in women, as did increased dietary intake of meat, red meat, and organ meat by women (Péneau et al., 2013). The authors noted that red meat is a good source of retinol and B₁₂, although meat clearly provides protein and other macronutrients making specific relationships difficult to determine. In contrast to the outcomes in women, there was no observed relationship between meat intake and hearing outcomes in men, and an observed association between increased seafood and shellfish intake only approached statistical significance, with the obtained p-values being greater than 0.05 but less than 1.0 (Péneau et al., 2013). Taken together, the above data highlight the potential interactions among micro- and macro- nutrients and hearing, gender, and unknown environmental, genetic, or other individual risk factors.

Given the limitations of single nutrient analyses, we have used an alternative method to examine the relationship between diet and hearing, i.e. dietary pattern. The approach used here takes advantage of the Healthy Eating Index (HEI) as an overall dietary quality metric, and extends our previous research (Spankovich & Le Prell, 2013) and the field as a whole (for detailed review, see Le Prell & Spankovich, 2013) by now explicitly assessing the potential interaction between diet and noise on hearing. Previous efforts to assess the relationship between hearing status and diet have failed to examine interaction of noise exposure history as part of the analysis, or they have adjusted for the effects of noise as part of a multivariate model. The current analysis was motivated by the hypothesis that there may be an interaction between noise history and dietary quality, with an increased risk of

hearing loss in those with both poorer diets and greater noise exposure. The current analysis of the National Health and Nutrition Examination Survey (NHANES) database specifically assessed potential interactions between diet and noise with respect to threshold sensitivity.

Methods

The NHANES is an ongoing “rolling” cross-sectional survey of the civilian non-institutionalized population of the United States. Every two years, approximately 10,000 individuals are selected at random based on demographic distributions, yielding a population that is representative of the US population (for detailed discussion of the historic and current sampling procedures, see Johnson et al., 2014). Between 1999 and 2002, NHANES collected data on 21,004 individuals of all ages (9,965 in 1999-2000 and 11,039 in 2001-2002) (for further detail on NHANES sampling, data collection, and analysis methods, see Curtin et al., 2012). We extracted HEI data as a metric for dietary quality (as in Spankovich & Le Prell, 2013), and we extracted data on noise exposure (as in Mahboubi et al., 2013). We then assessed interactions between HEI scores, noise exposure, and threshold sensitivity after adjusting for age, sex, race/ethnicity, education, diabetes, hypertension, and smoking. Hearing metrics included a low-frequency pure-tone-average (LFPTA) at 0.5, 1, and 2 kHz, a high-frequency pure-tone-average (HFPTA) at 3, 4, 6, and 8 kHz.

Participants

The interaction between dietary quality, noise history, and hearing thresholds was explored using a cross-sectional analysis of 1999-2002 NHANES data. As per our previous report (Spankovich & Le Prell, 2013), from the total pool of 21,004 NHANES participants from the 1999-2000 and 2000-2002 cycles, there were 8,143 participants ages 20 to 69 years. Audiometric data were available for 3,853 participants from the two NHANES cycles (1999-2000: 1,807 participants; 2000-2002: 2,046 participants). Of the 3,853 participants with audiometric data, there were 2,176 participants included in the final analysis. As in Spankovich and Le Prell (2013), there were 1,487 participants excluded reasons including incomplete auditory data and/or missing dietary data (540 excluded), ear tubes, abnormal otoscopy, impacted cerumen, abnormal tympanometry (peak pressure -150 daPa; compliance 0.3 ml), and/or more than 10-dB difference between test and retest thresholds at 1 kHz (635 excluded), LFPTA or HFPTA thresholds > 3 standard deviations (SD) above the mean for their age group (186 excluded), or > 15 dB difference between ears on either LFPTA or HFPTA (126 excluded). In addition to the exclusions applied as in Spankovich and Le Prell (2013), the current analysis excluded an additional 190 participants that had reported noise exposure within the past 24-hours to reduce the likelihood that the hearing tests were not confounded by potential temporary threshold shift (TTS). Based on Cronbach's $\alpha = 0.80$ for right vs. left ear comparisons, the PTAs for right and left ears were averaged for all reported analyses.

Demographics and Variables

As part of the NHANES data collection, trained interviewers administer detailed questionnaires assessing various factors that may influence health outcomes. Across studies, one of the potential risk factors for hearing loss is sex, with males having worse thresholds

than females, although the influence of sex are difficult to interpret given sex-based differences in noise history (see Marlenga et al., 2012; Mahboubi et al., 2013). Other potentially important risk factors include race/ethnicity (Lin et al., 2012), education / socioeconomic status (Agrawal et al., 2008; Henderson et al., 2011; Zhan et al., 2011), noise exposure (Agrawal et al., 2009; Zhan et al., 2011; Mahboubi et al., 2013), smoking and second-hand smoke (Cruickshanks et al., 1998; Fabry et al., 2011), diabetes (Bainbridge et al., 2008), cardiovascular health (Agrawal et al., 2009; Nash et al., 2011), ototoxic drugs (Laurell et al., 1996), and there are some genes associated with vulnerability to hearing loss based on population studies (for reviews, see Liu & Yan, 2007; Uchida et al., 2011). The selection of modifying factors to include in these studies is a significant issue. For example, smoking has been associated with increased risk of hearing loss in several epidemiological studies (e.g. Cruickshanks et al., 1998; Agrawal et al., 2009; Gopinath et al., 2010). However, smoking is also associated with lower HEI scores (Guenther et al., 2008; Lutz et al., 2013). When smoking was considered in our statistical model without consideration of diet, there was a statistically significant contribution to the variance. When comparing smokers vs. nonsmokers, smokers had a statistically significant poorer HFPTA (Wald $F=5.30$, $df=1,29$; $p=0.029$) when adjusting for age, sex, race/ethnicity, education, diabetes, hypertension, and noise exposure variables. Smoking was included in model based on previous use in literature, however it did not significantly alter outcomes. However, when HEI score was adjusted in the same model the relationship was no longer statistically significant (Wald $F=2.75$, $df=1,29$; $p=0.108$). Similarly, retrospective analysis of data from the Nurses' Health study data revealed a statistically significant association between higher body mass index (BMI) and self-reported hearing loss in women (Curhan et al., 2013). However, when we assessed BMI as a potentially significant factor in preliminary analysis of the NHANES data, but this was not a significant factor and we did not include this as a factor in the final multivariate analysis. The final selection of factors included in the final analysis was based on preliminary tests of individual factor significance.

Here, race/ethnicity was grouped as non-Hispanic White (hereafter, white), non-Hispanic Black (hereafter, black), Mexican American, or Other. Education was coded as Less than High School, High School Diploma (including general equivalency diploma), or More than High School. Participants were asked if they were "Ever told by a doctor that they have diabetes" (Yes or No) "Ever told by a doctor that they have high blood pressure" (Yes or No), and smoking history was reported as having smoked at least 100 cigarettes in entire life and current smoker (Yes or No). In this study we adjusted for age, sex, race/ethnicity, education, cardiovascular health (diabetes, hypertension) and smoking. Despite the lack of a significant independent factor, smoking was included in the analysis based on previous associations in the literature. No adjustment was made for genetics (not available) or potential ototoxic drug use.

There were five topics about loud noise exposure sources included in the NHANES from 1999-2002, with loud noise defined in the NHANES questions as "so loud that you had to speak in a raised voice to be heard". The five areas include work related noise, military service, recreational firearm use, non-occupational exposure, and exposure in past 24 hours. The four variables: work exposure [ever exposed to loud noise at work for at least 3 months (Yes or No)], military service/veteran status (Yes or No), recreational firearm use [firearm

noise exposure at least once a month for a year (Yes or No)], and recreational noise exposure [loud noise other than firearm use outside of work at least once a month for a year (Yes or No)] were included in the model. We excluded all participants reporting loud noise/music within the past 24 hours (Yes or NO) as there was no opportunity to resolve the extent to which temporary noise-induced changes in hearing might mask true hearing status in those participants. Here, we considered the association of the first four individual noise variables as well as a composite noise exposure score based on the total number of reported noise sources, which had the potential to range from 0 to 4 based on the four questions in the NHANES. Although using dichotomous variables to score self-reported noise is not ideal, there has been some validation of the use of self-reported noisy profession over the life course in other studies (Davis et al., 2008). Reported use of hearing protection was not incorporated into the model.

Audiometric Testing

The audiological examination was performed in a mobile examination center (MEC) equipped with sound-isolated rooms by health technicians trained by a certified audiologist from the National Institute for Occupational Safety and Health (NIOSH). The examination consisted of an audiometric questionnaire, an otoscopic examination, tympanometry and pure-tone air-conduction threshold measures at 0.5, 1.0, 2.0, 3.0, 4.0, 6.0, and 8.0 kHz (National Health Examination Survey, 2001). Instrumentation included an audiometer (AD226; Interacoustics AS, Assens, Denmark) with standard headphones (TDH-39; Telephonics Corporation, Farmingdale, NY) and insert earphones (EarTone 3A; Etymotic Research, Elk Grove Village, IL) and an acoustic impedance tympanometer (Earscan, Micro Audiometric, Murphy, NC). Detailed descriptions of the audiometric testing procedures are available online (<http://www.cdc.gov/nchs/nhanes/au.pdf>). The primary analysis assessed LFPTA (0.5, 1, and 2 kHz) and HFPTA (3, 4, 6, and 8 kHz) as the dependent variable. After identifying robust interactions between noise, diet, and hearing, a final analysis assessed frequency specific relationships at 3, 4, 6, and 8 kHz.

Healthy Eating Index (HEI)

The HEI provides an overall assessment of type, quantity, and variety of foods, and compliance with US dietary recommendations (for additional detail, see United States Department of Agriculture, 1995). Dietary recall interviews were conducted during 1999-2002 NHANES MEC evaluations. An automated data collection system was used to obtain a 24-hour dietary recall for use in HEI calculation. The HEI data were obtained by downloading the data file for the HEI 1999-2002 from the USDA Center for Nutrition Policy and Promotion website (www.cnpp.usda.gov/HealthyEatingIndex-Archive.htm). The overall HEI score is the sum of 10 equally weighted components with a maximum HEI score of 100. For each of the five food-group components (meat, dairy, fruits, vegetables, and grains), individuals who consumed the recommended number of servings received a score of 10; scores decreased with less than recommended intake. Greater than recommended intake of the five food groups did not reduce the category score. Fat, saturated fat, cholesterol, and sodium were scored based on consumption of not more than the recommended maximum intake. If fat and saturated fat intake were 30% of total caloric intake or less (at or below the maximum recommended intake), a score of 10 was received for each; if cholesterol and

sodium intake were at or below maximum recommended intake values (300 mg cholesterol, 2,400 mg sodium), a score of 10 was received for each. Higher than recommended intake of fat, saturated fat, cholesterol, or sodium decreased scores. Finally, for dietary variety, a score of 10 was given if at least 16 different foods were consumed, decreasing to zero if 6 or less different foods were consumed. The primary analysis was based on the composite score. A secondary analysis was conducted within HEI sub-scale components to explore the association between specific dietary components and hearing.

Statistical Analysis

The USDA classifies HEI scores greater than 80 as “good” and scores less than 51 as “poor”. Using this rating scale, approximately 10% of the US population had “good” diets and approximately 16% had “poor” diets (Basiotis et al., 2002). Quintile-based analysis is a common method in nutritional epidemiology (Willett, 1990; Willett et al., 1997) and allows the potential associations of diet to be parsed within that 74% of the population falling into the “Needs Improvement” category. Our previous quintile-based analyses suggested a discrete difference in high-frequency thresholds for comparisons between the top 3 quintiles (better diets, with average HEI scores = 62) and the bottom 2 quintiles (poorer diets, with average HEI scores = 61) (Spankovich & Le Prell, 2013) and we therefore used a quintile based approach here as well, followed by analyses based on a similar dichotomy based on the top 3 quintiles versus the bottom two quintiles.

All analyses were performed in SPSS version 20. Data were entered into the Complex Samples Analysis incorporating 4-year sample weights. General linear models were performed to assess the association between dietary quality and hearing, noise exposure and hearing, and then the interaction between noise history and dietary quality on hearing. A final series of analyses assessed interactions within specific noise-exposed population subsets. The independent variables in the general linear model were HEI and/or noise exposure; the dependent variables were LFPTA and HFPTA. Covariates adjusted in the final models included: age, sex, race/ethnicity, education, diabetes, hypertension, and smoking. Wald F statistics were performed and $\alpha = 0.05$ was defined as statistically significant. There were no statistically significant differences between included and excluded participants with respect to sex, age, race/ethnicity, pure tone averages (LFPTA or HFPTA), percent reporting individual noise exposure variables, and mean healthy eating index (HEI).

Results

HEI scores varied across the population, ranging from 24 to 95 across participants. The majority of our sample, 73.4%, had intermediate scores (ranging from 51 to 80), which are classified as “needs improvement”, while 14.6% had “poor” scores and 12% had “good” scores. That distribution is consistent with other population-based evaluations of HEI scores (United States Department of Agriculture, 1995). The average HEI score was 63.11 (95% confidence interval = 62.37 - 63.85). Demographic data are provided in Table 1.

In the first set of analyses participants sorted into quintiles based on HEI (Figures 1A and 1B). There was a statistically significant negative association between HEI and HFPTA (Wald $F=5.365$, $df = 4,26$; $p=0.003$) but not HEI and LFPTA (Wald $F=2.021$, $df = 4,26$; p

=0.121), with higher (better) HEI was associated with lower (better) HFPTA. We then dichotomized dietary quality into “poorer diet” (bottom two quintiles; $n=863$, range = 24 to 61; mean = 50.91) and “better diet” (top 3 quintiles; $n=1313$, range = 61 to 95; mean = 71.82) groups; there was again a statistically significant negative association (Wald $F=22.438$, $df=1,29$; $p<0.0010$), with lower (better) HFPTA thresholds associated with higher (better) HEI scores (see Figure 1C). The final assessment examined the association between diet (dichotomized HEI score) and hearing within the specific frequencies included in the HFPTA (Figure 1D). There were statistically significant differences in pure-tone thresholds for participants in the two HEI categories (top 60% versus bottom 40%) at 3 kHz (Wald $F=22.453$, $df=1,29$; $p<0.001$), 4 kHz (Wald $F=42.712$, $df=1,29$; $p<0.001$), and 6 kHz (Wald $F=13.306$, $df=1,29$; $p=0.001$). The top HEI had better thresholds at individual frequencies compared to poorer HEI. There was not a statistically significant difference at 8 kHz (Wald $F=3.3543$, $df=1,29$; $p=0.077$). Specific lower frequency data was not analyzed due to absence of overall relationship (Figure 1B).

The second set of analyses examined the relationship between noise (number of reported sources) and HFPTA and LFPTA (see Figure 2A). The model adjusted for age, sex, race/ethnicity, education, diabetes, hypertension, and smoking, but not dietary quality. The number of reported noise sources ranged from 0 (no reported noise exposure) to 4 (all four queried noise sources reported as “yes”). As per Table 1, approximately 60.1% reported no to the 4 variables of noise exposure, and 40% answered “yes” to one of the four questions. Of the remaining subjects, 10.8% reported two sources of noise, 3.1% reported exposure to 3 noise sources, and 1% reported exposure to all 4 noise sources queried. See Table 2 for descriptive statistics for reported noise and diet.

When participants were sorted based on the number of reported noise sources (Figure 2A), there was a statistically significant positive association between number of self-reported noise sources and HFPTA (Wald $F=7.095$, $df=4,26$; $p=0.001$), where higher reported noise was associated with higher (poorer) HFPTA. No statistically significant relationship was seen for LFPTA (Wald $F=0.411$, $df=4,26$; $p=0.799$). We then dichotomized noise sources as “less noise” (0-1 sources) versus “more noise” (2-4 sources). There was a statistically significant relationship (Wald $F=10.885$, $df=1,29$; $p=0.003$), with lower (better) HFPTA thresholds associated with less noise (see Figure 2B). When we repeated this analysis within frequencies at 3, 4, 6, and 8 kHz, there were statistically significant differences in pure-tone thresholds for participants in the two noise categories (top 60% versus bottom 40%) at all four frequencies [3 kHz (Wald $F=5.658$, $df=1,29$; $p=0.024$), 4 kHz (Wald $F=9.395$, $df=1,29$; $p=0.005$), 6 kHz (Wald $F=13.895$, $df=1,29$; $p=0.001$), and 8 kHz (Wald $F=6.378$, $df=1,29$; $p=0.017$)] with no reliable differences at lower frequencies (0.5, 1, and 2 kHz, not shown).

The most important question of interest here was whether there is a statistically significant interaction between noise exposure, diet, and hearing, with dietary quality interacting with reported noise on higher-frequency hearing as suggested by adjusted descriptive data (see Table 2). The analysis was limited to HFPTA data given no statistically significant relationship between HEI and LFPTA (Figure 1A) and no statistically significant relationship between number of reported noise sources and LFPTA (Figure 2A). A statistically significant interaction was observed (Figure 3A); specifically, there was a

statistically significant interaction between HEI and number of noise sources reported, for HFPTA (Wald $F = 9.769$, $df = 3,27$; $p < 0.001$). The lower (poorer) the HEI and greater the number of noise sources the higher (poorer) the HFPTA. Within the subgroup reporting 0-1 noise sources ("Less noise") we found a statistically significant difference in HFPTA when the participants with HEI scores in the bottom 40% and the top 60% were compared (Wald $F = 7.549$, $df = 1,29$; $p = 0.010$). Similarly, when we completed comparisons within the subgroup reporting 2-4 noise sources ("More noise"), we detected a statistically significant difference between HFPTA when the participants with HEI scores in the bottom 40% and the top 60% of participants were compared (Wald $F = 10.751$, $df = 1,29$; $p = 0.003$). Comparisons within the participants with HEI scores in the top 60% of the population sample revealed no statistical difference in HFPTA when comparing participants with less noise vs. more noise (Wald $F = 0.970$, $df = 1,29$; $p = 0.333$). However, comparisons within the participants with HEI scores in the bottom 40% of the population sample revealed a robust, statistically significant difference in HFPTA for those reporting less noise vs. more noise (Wald $F = 12.193$, $df = 1,29$; $p = 0.002$).

In other words, participants that had diets that more closely met US Dietary Guidelines for Americans (higher HEI scores) had better hearing at higher frequencies (lower HFPTA at 3, 4, 6, and 8 kHz) regardless of the total amount of noise they were exposed to (0-1 vs 2-4 sources), but there were statistically significant threshold differences associated with noise in the participants that had poorer diets. The frequency specific thresholds are shown in Figure 3B and C for the four groups included in the interaction analysis shown in Figure 3A. There were statistically significant differences in HFPTA at all four frequencies in the poorer HEI group shown in Figure 3C [3 kHz (Wald $F = 6.782$, $df = 3,27$; $p < 0.001$), 4 kHz (Wald $F = 15.601$, $df = 3,27$; $p < 0.001$), 6 kHz (Wald $F = 6.619$, $df = 3,27$; $p = 0.002$), and 8 kHz (Wald $F = 3.320$, $df = 3,27$; $p = 0.035$].

The NHANES noise exposure data are self-reported and subject to recall bias, they are limited in ability to distinguish protected and unprotected exposures (use of earplugs/earmuffs), and they do not distinguish hazardous noise from non-hazardous noise (3 months of occupational noise is likely different from a career history including many years of noise exposure) (for discussion, see Mahboubi et al., 2013). Moreover, a single unprotected firearm discharge would be more hazardous than most unprotected recreation exposures. These shortcomings reduce the utility of an overall analysis based on number of noise sources, despite the robust interaction detected in this nationally representative sample. Therefore, we explored the association between noise source and hearing and then diet and hearing for participants exposed to different noise sources after adjusting for the variance of other sources of noise and demographic variables age, sex, race/ethnicity, education, diabetes, hypertension, and smoking (see Figure 4). Reported use of hearing protection was not included in this analysis.

Firearm Use Outside of Work

There was a statistically significant difference in HFPTA thresholds when those reporting firearm use were compared to others, after adjusting for other noise and demographic variables excluding diet (Figure 4A; Wald $F = 9.154$, $df = 1,29$; $p = 0.005$). The mean

HFPTA thresholds were approximately 4-dB worse in NHANES participants reporting use of firearms (Table 3). When we explored a potential relationship between diet and hearing in those participants reporting firearm use, mean thresholds were lower (better) among participants reporting better dietary quality compared to those with poorer diet quality (Wald $F = 4.911$, $df = 1,29$; $p = 0.035$). There was a statistically significant interaction (Wald $F = 16.6$, $df = 3,27$; $p < 0.001$), see Figure 4A and Table 4.

Military Personnel/Veteran Status

As observed for those reporting firearm use outside of work, there was a statistically significant difference in HFPTA thresholds when military personnel and those with veteran status were compared to others, after adjusting for other noise and demographic variables excluding diet (Figure 4B; Wald $F = 6.210$, $df = 1,29$; $p = 0.019$). The mean HFPTA thresholds were approximately 3-dB worse in NHANES participants reporting military service (Table 3). When we explored a potential relationship between diet and hearing in those participants reporting military service or veteran status, average thresholds were lower (better) among participants reporting better dietary quality compared to those with poorer diet quality (Wald $F = 6.510$, $df = 1,29$; $p = 0.016$). There was a statistically significant interaction (Wald $F = 10.789$, $df = 3,27$; $p < 0.001$), see Figure 4B and Table 4.

Occupational noise

The noise level and duration of insult for NHANES participants reporting occupational exposure to loud sound for more than 3 months are unknown, and there was no relationship between HFPTA threshold and self-reported workplace exposure when adjusting for other noise sources and demographic variables excluding diet (Table 3). However, when we explored a potential relationship between diet and hearing in those participants reporting occupational noise, average thresholds were lower (better) among participants reporting better dietary quality compared to those with poorer diet quality (Wald $F = 8.573$, $df = 1,29$; $p = 0.007$) and there was a statistically significant interaction (Wald $F = 7.927$, $df = 3,27$; $p < 0.001$), see Figure 4C and Table 4.

Exposure to Loud Sound Outside the Workplace

The noise level and duration of insult for NHANES participants reporting non-occupational exposure to loud sound is unknown, and there is not information available regarding the types of exposure participants experienced. The relationship between HFPTA threshold and self-reported non-workplace noise exposure approached the criterion of $\alpha = 0.05$ but did not meet the stated criterion when adjusting for other noise sources and demographic variables excluding diet (Table 3). When we explored a potential relationship between diet and hearing in those participants reporting non-occupational noise, average thresholds were lower (better) among participants reporting better dietary quality compared to those with poorer diet quality (Wald $F = 9.081$, $df = 1,29$; $p = 0.005$) and there was a statistically significant interaction (Wald $F = 9.609$, $df = 3,27$; $p < 0.001$), see Figure 4D and Table 4.

The primary analysis was based on the overall HEI score. However, the subscales are of interest. In post-hoc analyses, we explicitly considered the relationship between HFPTA and scores on the dietary subscales. The findings are summarized in Table 5. Statistically

significant interactions between each subscale, noise and HFPTA were found. However, only dietary variety showed a statistically significant relationship with HFPTA for both higher and lower levels of reported noise.

Discussion

In both animal and human literature, great variability in hearing damage susceptibility is seen with noise exposure, even in genetically homogenous animals with identical noise exposure (as discussed in Wang et al., 2002). Several factors may contribute to this variability including experimenter error (inconsistent exposure among animals, inaccurate physiological recording, and etc.), variable stress response, variable ischemic events, and possibly variation in diet (e.g. caloric intake) to name a few. This report describes a statistically significant interaction between noise exposure and dietary quality in their relationship to hearing status in participants with varied exposure to noise. The results of the cross-sectional analysis suggest a potential relationship in which hearing benefits associated with healthy diets may be greater in people with higher reported noise exposure. Put another way, the relationship between noise and hearing were seemingly amplified in the subset of the population with poorer dietary quality, compared to the relationship between noise and hearing in the population with better dietary quality. Within each exposure grouping, there were statistically significant interactions between noise and diet with respect to their relationship with HFPTA.

Associations for diet (Spankovich & Le Prell, 2013) and noise (Mahboubi et al., 2013) have been reported previously for NHANES participants, and the results in Figures 1 and 2 are consistent with previous reports, and provide new information in the form of frequency-specific analyses. We have demonstrated that dietary health, specifically the HEI, is a statistically significant independent factor related to high frequency hearing here and in a previous study (Spankovich and Le Prell, 2013). Based on this finding we suggest that future epidemiological analyses of factors associated with hearing loss consider adjustment for dietary health.

The association between noise and hearing varied as a function of insult (see Tables 3 and 4). The current analysis revealed robust (>5 dB) differences in HFPTA for individuals that are exposed to firearms discharge and those that are not. Firearm discharge produces very brief sounds with very high sound levels; damage to the inner ear can occur in a single exposure if hearing protection is not used correctly and consistently (Moon, 2007; Heupa et al., 2011; Moon et al., 2011; Saedi et al., 2013). Within firearm users, it is compelling that there was a statistically significant association between hearing and diet, with better HFPTA thresholds being associated with healthier diets, within this population that is at-risk for hearing loss. Analysis of the data from military personnel closely followed the results from civilians exposed to firearm noise in that there was a robust difference in HFPTA when military personnel were compared to non-military. Although exposure will vary with service assignment, personnel may be exposed to high-level continuous noise and/or impulse noise potentially related to firearm discharge, and NIHL is a known problem (for recent reviews, see Grantham, 2011; Yankaskas, 2013). Taken together, for both firearm exposures and

military personnel, there were robust interactions with noise appearing to be increased in those with poorer diets.

Given that impulse noise can produce an immediate mechanical injury to the organ of Corti (Henderson & Hamernik, 1986), it was somewhat surprising that the most robust interactions were observed in those that were exposed to firearms and military personnel. Healthy eating, or drugs or nutritional supplements, presumably cannot reduce the mechanical tearing and/or rupture of the reticular lamina that may be expected during impulse-related mechanical trauma. That being said, metabolic stress does not end when the noise insult is terminated. Instead, it can be an ongoing process, lasting multiple days post noise under at least some exposure conditions; clearly, hair cell lesions grow over the first 7-10 days post-exposure (Yamashita et al., 2004). Importantly, interventions that attenuate free radical production can reduce hearing loss and cell death even when the intervention begins post-noise (Yamashita et al., 2005).

There are multiple therapeutic agents that have been effective in narrowing the noise-lesioned area on the basilar membrane, suggesting that cells that are not immediately “torn apart” by noise can be rescued using agents that act on metabolic stress (for reviews, see Le Prell et al., 2007b; Le Prell & Bao, 2012). Pre-clinical studies have shown benefit for several molecules that can be obtained from normal healthy diets; some of these agents include beta-carotene, vitamins C and E, and magnesium (Le Prell et al., 2007a; Tamir et al., 2010; Le Prell et al., 2011a; Le Prell et al., 2011b), D-methionine (Campbell et al., 2007; Campbell et al., 2011; Clifford et al., 2011; Claussen et al., 2013; Lo et al., 2013; Rewerska et al., 2013), and N-acetylcysteine (Kopke et al., 2000; Kopke et al., 2005; Bielefeld et al., 2007; Kopke et al., 2007; Clifford et al., 2011). Ultimately, it may indeed be possible to prevent the secondary “spread” of damage that occurs with continued metabolic stress after noise stress. Thus, it is perhaps not that surprising that otoprotection studies using impulse noise in animals have been promising (Cassandro et al., 2003; Hight et al., 2003; Kopke et al., 2005; Coleman et al., 2007; Bielefeld et al., 2011; Gavriel et al., 2011; Xiong et al., 2013). Human trials assessing otoprotection after impulse noise have suffered from a variety of shortcomings including the lack of a placebo control against which recovery could be compared (Suckfuell et al., 2007) and variable changes in hearing in real-world trials, with TTS not consistently detected even in control subjects (Le Prell et al., 2011c; Lindblad et al., 2011). Variability has been a challenge in laboratory studies as well (Spankovich et al., 2013). In addition, human trials assessing otoprotection agents have in general not considered or adjusted for dietary health of participants. Assessment of dietary intake during military training has demonstrated that HEI scores improve in soldiers during basic combat training, which may alter susceptibility to NIHL during training possibly impacting additional benefit of otoprotection agents (Lutz et al., 2013). In other words if individuals already have a healthy diet, additional otoprotection strategies may be of limited value.

Worldwide, industries with high numbers of noise-exposed workers (and high rates of NIHL in workers) include agriculture, mining, construction, manufacturing, utilities, and transportation (Dasgupta et al., 2009; Kim, 2010; Kim et al., 2011; Vigeh et al., 2011; Chung et al., 2012; Kelly et al., 2012; Masilamani et al., 2012; Pingle, 2012; Singh et al., 2012; Belojevic, 2013; Cruz et al., 2013; Pawlaczyk-Luszczynska et al., 2013). Although

NIHL secondary to occupational noise is a known issue, within this NHANES cohort, there was not a statistically significant relationship between reported occupational noise (here defined as working in noise loud enough to need to raise voice to hear for at least 3 months) and hearing status. It is thus intriguing that there was nonetheless a statistically significant interaction between noise and dietary quality on HFPTA thresholds within the participants that reported exposure to occupational noise. National standards mandating use of hearing protection devices (HPDs: earplugs and earmuffs) in hazardous noise conditions vary across nations, with many nations being more conservative than the OSHA limits in the USA (Suter, 2007). The data presented here, in combination with our earlier report (Spankovich & Le Prell, 2013), suggest that workers should receive not only hearing conservation education, but also encouragement to adopt healthy eating strategies as part of annual worker safety training. The poorest HFPTA thresholds, after adjusting for age, sex, race/ethnicity, education, diabetes, hypertension, and smoking, were in workers that had the poorest dietary quality (bottom 40% of HEI scores), although the cross-sectional analysis cannot be used to infer causality. Regardless of whether there is in fact any benefit to the auditory system, guidance on the importance of healthy eating habits is consistent with public health advice that health professionals should provide consistent messages about nutrition (Nelson, 2013).

The final category of participants for which data were available is those that reported exposure to “noise outside the workplace”. At the time this data was collected, “noise outside the workplace” was explicitly defined as “loud noise outside of work, such as noise from power tools or loud music, for average of at least once a month for a year; loud noise meaning so loud that you had to speak in a raised voice to be heard.” There was a borderline significant association ($p=0.054$) between diet and noise on HFPTA in participants that self-reported noise exposure outside the workplace.

The major limitation of the study is the cross-sectional design. Though NHANES are comprehensive and nationally representative, cause and effect cannot be determined. In addition, we cannot exclude the possibility that missing and/or excluded data may introduce bias, or that other undetermined confounding variable(s) may exist. Finally, the reporting of dietary intake and noise exposure may be susceptible to over- and under-reporting.

Summary and Conclusions

Taken together, the observed associations are consistent with the possibility that healthier diets *may* be associated with some small but reliable benefit with respect to HFPTA in individuals that are exposed to noise of various types and kinds. In the occupational and non-occupational noise groups, there was approximately 3-dB difference in HFPTA thresholds when those with the top 60% of HEI scores were compared to those with the bottom 40% of HEI scores; the difference in mean HFPTA scores observed across the dichotomized HEI groups increased to approximately 5-dB for those that were exposed to firearms or served in the military.

With respect to hearing loss prevention, strategies that emphasize “engineering” solutions (turn it down) and “administrative” solutions (walk away) must continue, and education

about hearing conservation and/or hearing loss prevention should include guidance on when HPDs are needed and how to use them. In addition, we advocate that all health professionals encourage healthy eating strategies both for overall health, and also for potential benefits to hearing health.

While we do not advocate nutritional solutions, or supplements, in place of safe listening behaviors (including use of HPDs), these options may be more palatable for those that believe HPDs detract from the perceived quality of recreational activities, such as bar/club and/or concert attendance, where average measured sound levels commonly reach or exceed 100 dBA (Cabot et al., 1979; Gunderson et al., 1997; Smith et al., 2000; Serra et al., 2005; Opperman et al., 2006; Müller et al., 2010; Williams et al., 2010). We also note the possibility that for some individuals exposed to very high sound levels, there may be benefits related to combining HPD use with dietary choices. For some individuals that are exposed to high level sound, HPDs may not provide enough effective attenuation (based on conduction of sound through bone, see Berger, 2003). For others, HPDs may be rejected if they are perceived to detract from important situational awareness requirements (Casali et al., 2009; Patil & Breeze, 2011; Talcott et al., 2012). Hearing loss in populations that use firearms in environments where detection of low level sounds is important is likely to remain a challenge even though there is nearly universal agreement that HPD's are required as a primary defense against firearm noise insult. Best practice clearly dictates that engineering controls (to reduce noise at its source) and administrative controls (to reduce exposure time) be attempted first, with HPDs providing the next best strategy for protection when engineering and administrative controls are not feasible.

The data presented here and elsewhere regarding a statistically significant association between diet and hearing, and a statistically significant interaction between noise and diet on hearing, are consistent with the possibility that dietary quality may influence hearing status. However, questions remain as to what is the best diet to extend protection and what is the best method to measure diet quality. Further studies are needed to explore other indices of diet based on dietary guidelines (such as updated versions of the HEI [HEI-2005 and HEI-2010] and the Diet Quality Index), specialized diets [Dietary Approach to Stop Hypertension (DASH) Diet, Mediterranean Diet, and etc.] as well as food and nutrient-based indexes. Finally, we advise as otoprotection interventions progress to clinical trials and future epidemiological analyses consider risk factors for hearing loss; investigators should consider the possible influence of the subject's dietary health on outcomes.

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Figure 1.

1A) High frequency (HFPTA) and 1B) low frequency (LFPTA) pure-tone-average thresholds and healthy eating index quintiles. There was an overall statistically significant relationship between diet and HFPTA after adjusting for sex, race, age, education, diabetes, and smoking; as well as in pairwise comparison to the lowest quintile. Participants in the top 3 quintiles were statistically different from participants in the bottom quintile. LFPTA was not statistically related to HEI. 1C) HFPTA thresholds with participants dichotomized into healthier diets (top 60% of HEI scores) and poorer diets (bottom 40% of HEI scores). There was a statistically significant relationship between diet and HFPTA after adjusting for sex, race, age, education, diabetes, and smoking. 1D) Puretone thresholds with participants dichotomized into healthier diets (top 60% of HEI scores) and poorer diets (bottom 40% of HEI scores). There was a statistically significant relationship between diet and threshold at

3, 4, and 6 kHz after adjusting for sex, race, age, education, diabetes, and smoking. Data are Mean $\pm$ standard error (SE); asterisks indicate statistically significant differences in HFPTA for pair-wise group comparisons (p 's <0.05).

Figure 2.

2A) High frequency (HFPTA) and low frequency (LFPTA) pure-tone-average thresholds and the number of reported noise sources. There was a statistically significant relationship between number of noise sources and HFPTA after adjusting for sex, race, age, education, diabetes, and smoking; pairwise comparisons to lowest number (0) are shown in 2A. Participants reporting 3 sources of noise were statistically different from participants reporting 0 sources. LFPTA was not statistically related to noise sources. 2B) HFPTA thresholds with participants dichotomized into more noise (2-4 sources) and less noise (0-1 sources). There was a statistically significant relationship between noise and HFPTA after adjusting for sex, race, age, education, diabetes, and smoking. 2C) Pure-tone thresholds with participants dichotomized into less noise and more groups. There was a statistically

significant relationship between noise and threshold at 3, 4, 6, and 8 kHz after adjusting for sex, race, age, education, diabetes, and smoking. Data are Mean +/- standard error (SE); asterisks indicate statistically significant differences in HFPTA for pair-wise group comparisons (p 's <0.05).

Figure 3.

3A) There was a statistically significant interaction between number of noise sources (0-1 vs 2-4) and dietary quality (top 60% vs bottom 40%) with HFPTA after adjusting for sex, race, age, education, diabetes, and smoking ($p < 0.005$). 3B) Within the population with better diets there, were no statistically significant differences between those with less noise and those with more noise at any of the HFPTA frequencies tested. 3C) Within the population with poorer diets, there were statistically significant differences between those with less noise and those with more noise at all HFPTA frequencies tested. Asterisks indicate statistically significant differences in HFPTA for pair-wise group comparisons (p 's < 0.05). Additional descriptive data are provided in Table 2. Data are Mean $\pm$ standard error (SE).

Figure 4.

High frequency pure-tone-average (HFPTA) thresholds is influenced by dietary quality and noise. There was a statistically significant interaction between the reported noise source (4A: firearms; 4B: military service; 4C: occupational noise > 3 months; 4D: non-occupational noise) and dietary quality (top 60% vs bottom 40%) with HFPTA after adjusting for sex, race, age, education, diabetes, smoking and the other sources of noise reported. Outcomes of the pair-wise comparisons are shown in Tables 3 and 4. Data are Mean +/-standard error (SE).

Table 1

Descriptive data for demographics of NHANES 1999-2002 with sample weights applied.

Variable	Mean (SE)
Age	40.7 (0.33)
HFPTA	16.3 (0.43)
LFPTA	9.3 (0.21)
Average HEI	63.11 (0.36)
Average HEI, bottom 40%	50.91 (.33)
Average HEI, top 60%	71.82 (.23)
Variable	Count (%)
Male	n=847 (40.9%)
Female	n=1329 (59.1%)
Diabetes (Yes)	n=164 (5.6%)
Loud Job (Yes)	n=408 (21.6%)
Recreational Noise (Yes)	n=415 (22.6%)
Firearm noise (Yes)	n=110 (6%)
Veteran (Yes)	n=197 (9.9%)
Smoked 100 cigarettes (Yes)	n=970 (47.0%)
Hypertension (Yes)	n=501 (19.8%)
Race/Ethnicity	
White	n=998 (69.2%)
Black	n=413 (11.1%)
Mexican	n=547 (7.5%)
Other	n=218 (12.2%)
Education	
Less than High School	n=616 (17.3%)
High School or Equiv.	n=481 (24.0%)
More than High School	n=1077 (58.7%)
Number of Noise Sources	
0 Noise sources	n=1394 (60.1%)
1 Noise sources	n=521 (25.1%)
2 Noise sources	n=193 (10.8%)
3 Noise sources	n=55 (3.1%)
4 Noise sources	n=13 (1.0%)
0-1 Noise sources	n=1915(85.2%)
2-4 Noise sources	n=261 (14.8%)

*
SE = standard error

Table 2

Mean descriptive statistics, adjusted for age, sex, race/ethnicity, education, diabetes, hypertension, and smoking, sample weights applied

	Age (SE)	HEI (SE)	LFPTA (SE)	HFPTA (SE)
Lower noise, poorer diet	39.81 (0.59)	51.01 (0.41)	9.42 (0.35)	16.08 (0.52)
Lower noise, better diet	41.72 (0.47)	71.95 (0.26)	9.17 (0.22)	15.31 (0.49)
Higher noise, poorer diet	39.27 (1.22)	50.42 (0.60)	10.87 (0.79)	23.28 (1.83)
Higher noise, better diet	39.38 (0.99)	70.96 (0.61)	8.27 (0.61)	17.34 (1.34)

*
SE = standard error

Table 3

Relationship between HFPTA and noise source; adjusted for age, sex, race/ethnicity, education, diabetes, hypertension, and smoking, sample weights applied

	HFPTA Mean (SE)	95% CI	Wald F	p (yes vs no)
Firearm Noise				
Yes	21.62 (1.28)	19.01-24.24	9.154	0.005
No	17.69 (0.86)	15.94-19.45		
Veteran/Military				
Yes	21.25 (1.41)	18.36-24.14	6.210	0.019
No	18.06 (0.59)	16.85-19.27		
Loud Job (3months)				
Yes	19.51 (0.94)	17.59-21.42	0.282	0.600
No	19.80 (0.90)	17.97-21.6		
Loud Noise Outside Work				
Yes	20.46 (1.08)	18.26-22.67	4.027	0.054
No	18.85 (0.83)	17.15-20.55		

* SE = standard error, Bold = significant ($p < 0.05$)

Table 4

Relationship between HEI and HFPTA, within noise sources; adjusted for age, sex, race/ethnicity, education, diabetes, hypertension, smoking, and other noise sources, sample weights applied

	Better HEI Mean (SE)	95% CI	Poorer HEI Mean (SE)	95% CI	Wald F	p (better vs poorer HEI)	Wald F	Interaction between diet and noise?
Firearm Noise								
Yes	21.16 (1.21)	18.68-23.63	25.01 (1.51)	21.92-28.09	4.911	0.035	16.600	<0.001
No	15.16 (0.28)	14.58-15.74	16.87 (0.47)	15.92-17.83	14.505	0.001		
Veteran/Military								
Yes	23.53 (1.09)	21.29-25.29	28.80 (2.03)	24.64-32.96	6.510	0.016	10.789	<0.001
No	14.65 (0.27)	14.09-15.20	16.08 (0.43)	15.20-16.96	8.695	0.006		
Loud Job (3months)								
Yes	16.20 (0.64)	14.89-17.52	19.83 (1.02)	17.73-21.93	8.573	0.007	7.927	0.001
No	15.23 (0.29)	14.64-15.82	16.78 (0.50)	15.76-17.79	10.271	0.003		
Loud Noise Outside Work								
Yes	15.19 (0.77)	13.62-16.76	18.76 (1.14)	16.43-21.09	9.081	0.005	9.609	<0.001
No	15.57 (0.33)	14.89-16.24	16.97 (0.48)	15.99-17.95	7.506	0.010		

* SE = standard error, Bold = significant (p < 0.05)

Table 5

Relationship between HEI subscales and HFPTA, within noise sources; adjusted for age, sex, race/ethnicity, education, diabetes, hypertension, smoking, and other noise sources, sample weights applied

	Within Poorer bottom 40% of scores (SE)	95% CI	Within Better 60% of scores Mean (SE)	95% CI	Wald F	p (better vs poorer HEI)	Wald F	Interaction between diet and noise?
Fruits								
Lower Noise	15.93 (0.33)	15.25-16.62	15.27 (0.40)	14.45-16.09	2.315	0.139	4.797	0.008
Higher Noise	20.90 (1.00)	18.84-22.96	18.81 (1.46)	15.82-21.80	1.491	0.232		
Vegetables								
Lower Noise	16.02 (0.35)	15.32-16.73	15.25 (0.37)	14.50-16.00	3.951	0.056	3.633	0.025
Higher Noise	20.43 (1.21)	17.96-22.91	19.91 (1.00)	17.87-21.95	0.125	0.726		
Meats								
Lower Noise	15.63 (0.38)	14.85-16.41	15.63 (0.36)	14.89-16.37	0.000	0.996	3.396	0.032
Higher Noise	20.84 (1.36)	18.02-23.59	19.68 (1.08)	17.42-21.84	0.420	0.552		
Dairy								
Lower Noise	16.34 (0.44)	15.45-17.24	14.85 (0.35)	14.15-15.57	7.459	0.011	6.402	0.002
Higher Noise	20.71 (1.25)	18.13-23.30	19.78 (1.23)	17.23-22.32	0.249	0.621		
Grains								
Lower Noise	15.86 (0.41)	15.02-16.71	15.32 (0.30)	14.71-15.93	1.487	0.232	3.884	0.020
Higher Noise	20.71 (1.39)	17.87-23.52	19.59 (1.13)	17.25-21.95	0.330	0.570		
Variety								
Lower Noise	16.27 (0.41)	15.48-17.15	15.07 (0.35)	14.35-15.78	5.726	0.023	8.249	>0.001
Higher Noise	22.18 (1.30)	19.47-24.79	18.43 (1.19)	15.98-20.86	4.322	0.047		
Fats								
Lower Noise	15.87 (0.49)	14.84-16.90	15.48 (0.34)	14.78-16.17	0.461	0.502	5.523	0.004
Higher Noise	21.30 (1.16)	18.93-23.68	19.34 (0.98)	17.34-21.35	2.303	0.140		
Saturated Fats								
Lower Noise	15.45 (0.43)	14.57-16.33	15.71 (0.36)	15.01-16.49	0.322	0.575	7.620	0.001
Higher Noise	22.65 (1.28)	20.03-25.26	18.23 (0.95)	16.29-20.17	9.153	0.005		
Sodium								

	Within Poorer bottom 40% of scores (SE)	95% CI	Within Better 60% of scores Mean (SE)	95% CI	Wald F	p (better vs poorer HEI)	Wald F	Interaction between diet and noise?
Lower Noise	15.07 (0.33)	14.40-15.76	16.00 (0.33)	15.35-16.68	9.474	0.005	4.811	0.008
Higher Noise	20.31 (1.12)	18.02-22.59	19.94 (1.29)	17.33-22.58	0.049	0.827		
<i>Cholesterol</i>								
Lower Noise	15.61 (0.48)	14.62-16.58	15.63 (0.35)	14.92-16.32	0.002	0.965	3.648	0.025
Higher Noise	19.58 (1.93)	15.63-23.52	20.45 (0.87)	18.66-22.24	0.161	0.691		

* SE = standard error, Bold = significant (p < 0.05)