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Long-term aircraft noise exposure and risk of hypertension in postmenopausal women

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ABSTRACT

Background: Studies of the association between aircraft noise and hypertension are complicated by inadequate control for potential confounders and a lack of longitudinal assessments, and existing evidence is inconclusive.
Objectives: We evaluated the association between long-term aircraft noise exposure and risk of hypertension among post-menopausal women in the Women's Health Initiative Clinical Trials, an ongoing prospective U.S. cohort.
Methods: Day-night average (DNL) and night equivalent sound levels (L_{night}) were modeled for 90 U.S. airports from 1995 to 2010 in 5-year intervals using the Aviation Environmental Design Tool and linked to participant geocoded addresses from 1993 to 2010. Participants with modeled exposures ≥45 A-weighted decibels (dB [A]) were considered exposed, and those outside of 45 dB(A) who also did not live in close proximity to unmodeled airports were considered unexposed. Hypertension was defined as systolic/diastolic blood pressure ≥140/90 mmHg or inventoried/self-reported antihypertensive medication use. Using time-varying Cox proportional hazards models, we estimated hazard ratios (HRs) for incident hypertension when exposed to DNL or L_{night} ≥45 versus <45 dB(A), controlling for sociodemographic, behavioral, and environmental/contextual factors.
Results/discussion: There were 18,783 participants with non-missing DNL exposure and 14,443 with non-missing L_{night} exposure at risk of hypertension. In adjusted models, DNL and L_{night} ≥45 db(A) were associated with HRs of 1.00 (95% confidence interval [CI]: 0.93, 1.08) and 1.06 (95%CI: 0.91, 1.24), respectively. There was no evidence supporting a positive exposure-response relationship, and findings were robust in sensitivity analyses. Indications of elevated risk were seen among certain subgroups, such as those living in areas with lower population density ($HR_{interaction}$: 0.84; 95%CI: 0.72, 0.98) or nitrogen dioxide concentrations ($HR_{interaction}$: 0.82; 95% CI: 0.71, 0.95), which may indicate lower ambient/road traffic noise. Our findings do not suggest a relationship between aircraft noise and incident hypertension among older women in the U.S., though associations in lower ambient noise settings merit further investigation.

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1. Introduction

Noise is increasingly recognized as an important environmental stressor, especially among those living in urban communities or close to airports. Non-auditory effects associated with noise exposure, such as annoyance (Eze et al., 2018; Fidell et al., 2011; Miedema and Oudshoorn, 2001), cognitive impairment (Haines et al., 2001; Hygge et al., 2002; Irgens-Hansen et al., 2015; Jafari et al., 2019), sleep disturbance (Basner et al., 2011; Brink et al., 2019b; Douglas and Murphy, 2016), and adverse cardiovascular effects (Babisch, 2011; Correia et al., 2013; Héritier et al., 2017; Münzel et al., 2014; Osborne et al., 2020) have also been documented. Because hypertension is an established risk factor for cardiovascular disease and has been shown to be highly prevalent among certain populations such as older women (Wenger et al., 2018), understanding the relationship between noise and hypertension is of public health importance.

Community surveys have shown that aircraft noise may be a greater nuisance than other sources of noise (Brink et al., 2019a; Miedema and Oudshoorn, 2001; Wothge et al., 2017), and despite technological advancements leading to quieter aircraft, aircraft noise-related annoyance has substantially increased in affected communities (Guski et al., 2017). In fact, a recent, nationally representative survey found nearly two-thirds of people living near airports reported being highly annoyed at a day-night average sound level (DNL) above 65 A-weighted decibels (dB [A]), and 42% of respondents reported being highly annoyed by aircraft noise in general (Miller et al., 2021).

Noise is hypothesized to influence health outcomes through stress pathways, by activating the hypothalamic-pituitary-adrenocortical and sympathetic-adrenal-medullary axes with subsequent release of stress hormones (Babisch et al., 2001; Babisch, 2002; Selander et al., 2009), or through sleep disturbance pathways (Griefahn et al., 2000; Schmidt et al., 2013), by inducing vascular dysfunction. Links between aircraft noise and changes in blood pressure and hypertension via vascular inflammation and oxidative stress have been demonstrated in both human and animal studies (Münzel et al., 2017; Steven et al., 2020).

In spite of the biological plausibility, results from epidemiological studies of the relationship between aircraft noise and hypertension have been mixed. Some have reported that noise is associated with higher risk of hypertension (Dimakopoulou et al., 2017; Eriksson et al., 2010; Evrard et al., 2017; Jarup et al., 2008; Kim et al., 2021; Kourieh et al., 2022; Pyko et al., 2018), while others have found little evidence of an association (Carugno et al., 2018; Zeeb et al., 2017). These discrepancies may be related to differences in study design (e.g. cross-sectional vs. cohort), case definition (e.g. self-report of hypertension vs. blood pressure measurements), study population, noise exposure estimation protocols, and opportunities for residual confounding (Huang et al., 2015; van Kempen et al., 2018). There are also indications of sociodemographic disparities in noise exposure around U.S. airports (Simon et al., 2022) and in hypertension prevalence and incidence (Aggarwal et al., 2021; Claudel et al., 2018; Lackland, 2014) that warrant consideration in epidemiological studies.

Accordingly, we utilized modeled aircraft noise data from 90 U.S. airports to assess the longitudinal association between exposure to aircraft noise and incident hypertension in the Women's Health Initiative (WHI) Clinical Trials. Our cohort study evaluated relationships in a population at risk for hypertension using consistent estimation methods for noise exposure while controlling for potential confounding from individual and environmental risk factors to address limitations of prior studies.

2. Methods

2.1. Study population

The WHI is a large, national U.S. prospective cohort that enrolled 68,132 post-menopausal women ages 50–79 years into clinical trials at 40 clinical centers from 1993 to 1998 (Anderson et al., 2003). Participants were randomized into one or more overlapping Clinical Trials arms: 1) menopausal hormone therapy, 2) dietary modification, and/or 3) calcium/vitamin D supplementation. Participants were originally followed until the end of the main study in 2005, while a subset (82%) participated in the five-year Extension I study which continued follow-up until 2010. The WHI was reviewed and approved by the Fred Hutchinson Cancer Research Center (Fred Hutch) institutional review board (IRB) and the IRB for each WHI clinical center, and written informed consent was provided for each participant. As a part of cohort retention and follow-up activities, participant address information was confirmed during follow-up contacts and reviewed at least once a year.

Fig. 1 provides a flow chart outlining inclusion and exclusion criteria for participants in this current study. We excluded participants with baseline hypertension (n = 30,813), no follow-up data (n = 121), and missing noise exposure during follow-up (n = 746), yielding 36,542 participants at risk of hypertension. To limit exposure misclassification in the unexposed populations (i.e., exposure less than 45 dB [A]), we further excluded participants living outside of our 45 dB(A) noise contours but near other airports not included in the 90 airports for the entirety of our study period. We defined living near a non-study airport as living within a 22.2-mile radius of other civil and military airports; this radius represents the maximum noise contour boundary of any of the 90 modeled study airports. Exclusion of participants who lived outside of the 45 dB(A) contours of the study airports but within 22.2 miles of other airports resulted in 18,783 (27.6% of total Clinical Trials cohort) participants with DNL exposure estimates and 14,443 (21.2% of total Clinical Trials cohort) participants with night equivalent sound level (L_{night}) exposure estimates at risk for hypertension.

2.2. Noise assessment

Noise exposure contours were modeled by the U.S. Department of Transportation's John A. Volpe National Transportation Systems Center (Volpe) for 90 U.S. airports for 1995, 2000, 2005, and 2010 (Fig. 2). Detailed information on the generation of aircraft noise contours is provided by Kim et al. (2021). Briefly, noise contours were created using the Aviation Environmental Design Tool (AEDT), which was developed by the U.S. Federal Aviation Administration (FAA) using internationally accepted practices to estimate the contribution of aircraft to ambient noise (Ahearn et al., 2016). Aircraft operations data were sourced from Official Aviation Guide (OAG) for 1995 and the Enhanced Traffic Management System (ETMS) for 2000–2010. DNL and L_{night} were modeled in one dB(A) increments ranging from 45 (considered background) to 75 dB(A). A-weighting of decibels corrects for sound at frequencies that are unperceivable by the human ear (World Health Organization, 2018). DNL reflects noise exposure for an average 24-h period of the year that artificially weights (adds 10 dB [A]) to nighttime exposures (hours of 22:00 to 07:00) to account for sensitivity at lower ambient levels. L_{night} reflects noise exposure over nighttime hours. Geocoded participant addresses were assigned annually according to modeled 5-year noise exposure estimates, with intervening years (i.e., non-5-year intervals) weighted to the nearest available 5-year interval for all available participant locations and dates between 1993 and 2010, to estimate change in aircraft noise exposure over time.

2.3. Hypertension assessment

Clinical staff collected annual blood pressure measurements at WHI centers using standardized procedures (Anderson et al., 2003; Margolis et al., 2008). After a 5-min rest period at each annual visit, two measurements ≥30 s apart were taken from the right arms of participants using conventional mercury sphygmomanometers and averaged. Hypertension was defined as having any of the following: (1) systolic blood pressure (SBP) ≥140 mmHg, (2) diastolic blood pressure (DBP) ≥90 mmHg, (3) self-reported use of antihypertensive medication, or (4)

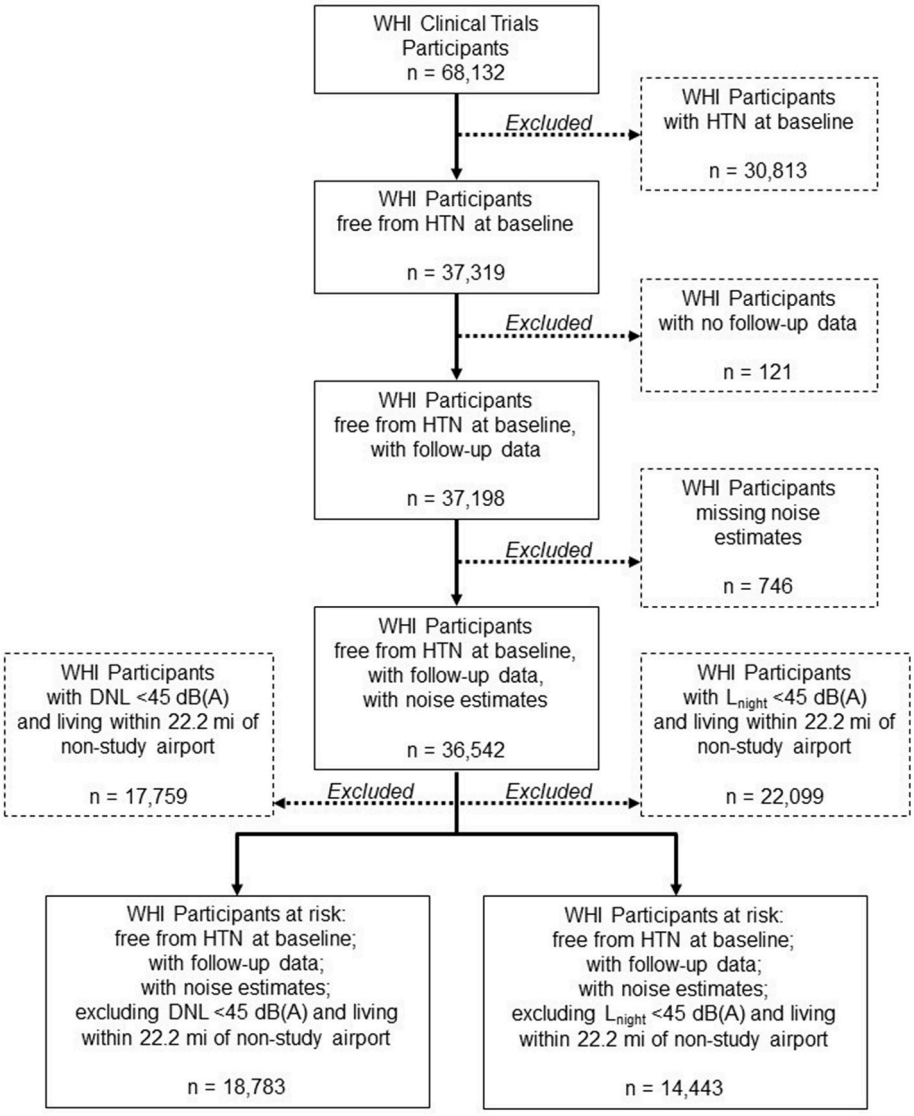

Fig. 1. WHI Clinical Trials participants included in the study.
Abbreviations: dB(A), A-weighted decibels; DNL, day-night average sound level; HTN, hypertension; L_{night}, nighttime average sound level; WHI, Women's Health Initiative.

inventoried use of antihypertensive medication. The time of hypertension event was defined as the date of the annual clinical visit during which blood pressure was measured or the date the mailed annual data collection form was completed. Antihypertensive medications were therapeutically classified as follows: angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, calcium channel blockers, diuretics, centrally acting antihypertensive agents, vasodilators, or any combination of these classes (Margolis et al., 2008). Incident hypertension was defined as the first occurrence of hypertension among those at risk through the end of Extension I study in 2010.

2.4. Covariates

Data were available on Clinical Trials arm (menopausal hormone therapy, dietary modification, calcium and vitamin D supplementation) involvement and unique WHI clinical center visited at baseline (n = 40), the latter of which was used to control for potential geospatial confounding. Participant-level demographic and behavioral data, obtained via self-administered questionnaires, included: age at baseline, race (White, Black/African-American, Asian/Pacific Islander, American Indian/Alaskan Native, Native Hawaiian/Other Pacific Islander, or more than one race); ethnicity (Hispanic/Latino or not Hispanic/Latino); education (college graduate or less than college degree); household income (<$20,000, $20,000-$50,000, or ≥$50,000 per year); insurance status (having any form of health insurance or uninsured); employment status (currently employed or unemployed); smoking status (never smoker [<100 cigarettes in lifetime], past smoker [≥100 cigarettes in lifetime but not currently smoking], or current smoker [≥100 cigarettes in lifetime and currently smoking]); and alcohol consumption (none or <1, 1–6, or ≥7 drinks per week).

Physical activity was available as total energy expenditure (metabolic equivalent of task [MET]-hours/week), which was based on the self-reported type, frequency, and duration of recreational physical activity. Body mass index (BMI) was calculated as weight (kilograms) divided by height (meters^2) as measured at WHI clinical centers during annual visits up to the end of the main study in 2005. Last-observed BMI measures were carried forward for those missing BMI measures after 2005 and participating through the Extension I study in 2010. Dietary sodium (g) was estimated using food frequency questionnaires provided at baseline with a random subsample of dietary modification participants providing data over follow-up (Patterson et al., 1999). Last-observed dietary sodium measurements were carried forward for

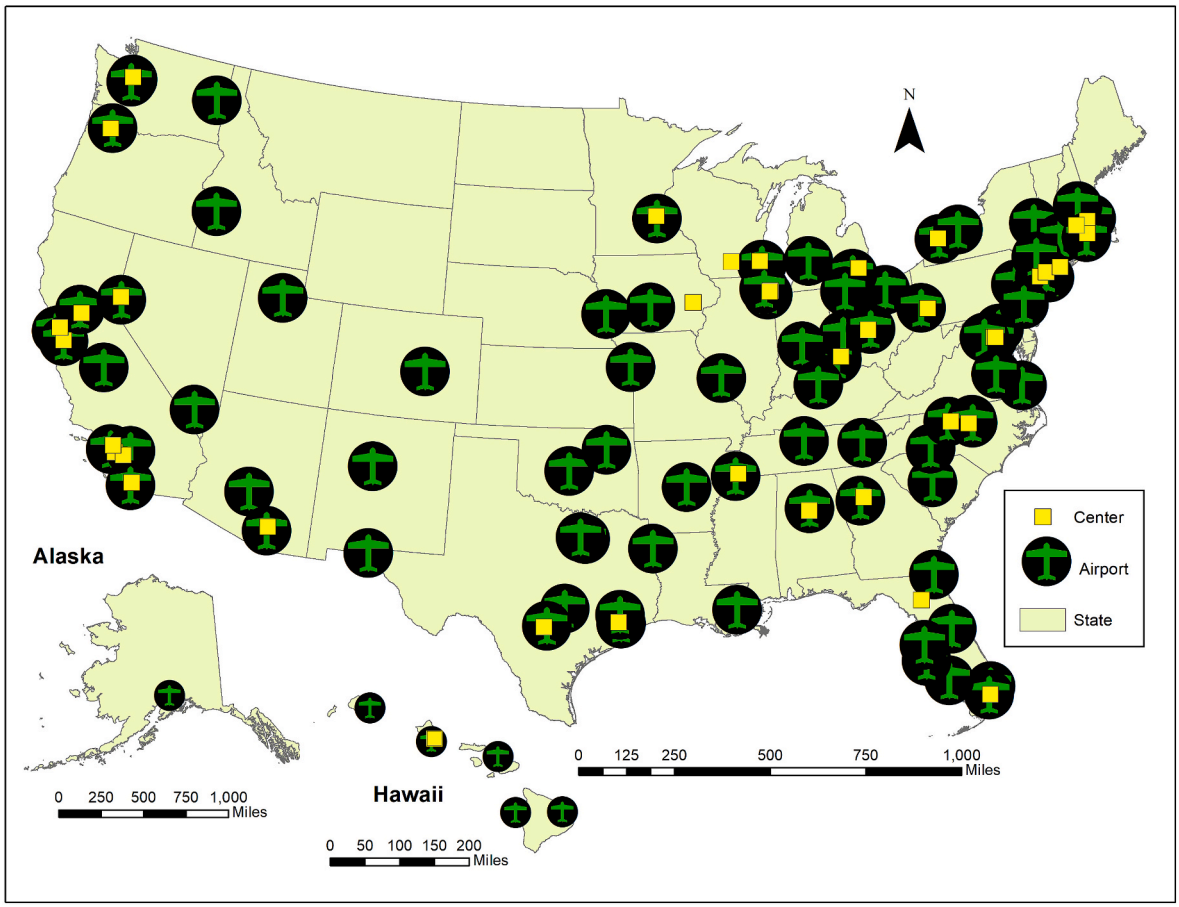

Fig. 2. U.S. airports (n = 90) included in the study and WHI clinical centers (n = 40).
Abbreviations: WHI, Women's Health Initiative.

missing visits.

We estimated environmental covariates through linkage of geocoded participant addresses over time (Whitsel et al., 2004). Neighborhood-level socioeconomic status was derived as a summation of z-transformed neighborhood measures of wealth/income, education and occupation. Details for their generation are described elsewhere (Roux et al., 2001). Increasing summary z-scores denote increasing neighborhood socioeconomic advantage. Air pollution measures were linked with geocoded participant addresses and included fine particulate matter ≤2.5 μm ($PM_{2.5}$, μg/m^3) and nitrogen dioxide (NO_2, parts per billion [ppb]). Ambient $PM_{2.5}$ and NO_2 estimates were generated and linked to geocoded participant addresses over follow-up using regionalized national universal kriging models (Sampson et al., 2013; Young et al., 2016). Population density (persons/mi^2) was estimated within 5.0 miles of geocoded participant addresses using U.S. Census 2000 and 2010 tract data (U.S. Census Bureau, 2000), as described by Dubowitz et al. (2012). Roadway proximity was estimated using major roads from Esri Data & Maps and detailed streets from the Esri StreetMap and North American Atlas products, as described by Hart et al. (2021). Roadway proximity was estimated as the distance (km) from geocoded participant addresses to primary roads with limited access or interstate highways (A1) and primary roads without limited access or U.S. and state highways (A2).

Age, $PM_{2.5}$, NO_2, population density, roadway proximity, dietary sodium, BMI, and physical activity were continuous linear variables, while neighborhood-level socioeconomic status was a continuous variable standardized around zero; all other variables were categorical. Time-varying factors included: noise exposure, smoking status, alcohol consumption, BMI, physical activity, neighborhood-level socioeconomic status, $PM_{2.5}$, NO_2, population density, roadway proximity and dietary sodium; all other covariates were fixed.

2.5. Statistical analyses

2.5.1. Main analysis

We tested for multicollinearity of continuous variables, indicated by tolerance values < 0.1, variance inflation factors >10, and comparing eigenvalues to condition indices (Schreiber-Gregory and Jackson, 2017).

We used time-varying Cox proportional hazards models (Powell and Bagnell, 2012) to estimate hazard ratios (HR) and 95% confidence intervals (95% CI) for the association between dichotomized exposure at 45 dB(A) and incident hypertension. Noise cut-points were based on World Health Organization Guideline (2018) Development Group recommendations for aircraft noise exposure and adverse health outcomes at 45 dB(A) day-evening-night sound levels (L_{den}). In secondary analyses we assessed aircraft noise exposure using increasing categories of DNL – 45–49, 50–54, 55–59, and ≥60 dB(A) – compared to DNL <45 dB(A), and L_{night} 45–49 and ≥ 50 compared to L_{night} <45 dB(A).

We used increasingly adjusted models to assess the relationships between noise exposures and incident hypertension. In Model 0, we adjusted for age, race, and ethnicity. In Model 1, we additionally adjusted for the following participant-level demographics and behaviors: Clinical Trials arm, education, employment, income, insurance, neighborhood-level socioeconomic status, WHI Center, smoking, and alcohol. In Model 2, we additionally adjusted for environmental contextual factors that may act as proxies for other sources of noise, such as urban settings or road traffic (Fecht et al., 2016; Stewart et al., 1999),

or air pollution; this model is Model 1 additionally adjusted for population density, roadway proximity and $PM_{2.5}$. Model 2 was selected as the main fully-adjusted model. Variable selection was informed by: a directed acyclic graph based on the current literature (Supplemental Fig. 1) created using DAGitty v3.0 (Textor et al., 2016), factoring in correlations (Supplemental Table 1), and effects on models when added individually (Supplemental Table 2).

2.5.2. Sensitivity analyses

We performed sensitivity analyses using Model 2 to test the robustness of our primary results and address potential exposure misclassification. First, we included dietary sodium intake as a model covariate since high sodium consumption is a risk factor for hypertension. However, studies have shown low confidence in using self-reported food frequency questionnaires estimates (Freedman et al., 2015; Huang et al., 2014). Second, to assess exposure misclassification among those living outside of the 45 dB(A) contours, we included and assigned those previously excluded participants that were living within 22.2 miles of other airports as exposed (where 22.2 miles is the maximum noise contour of any of the 90 airports). Third, to assess possible exposure misclassification by housing characteristics, we excluded participants living in areas with DNL ≥65 dB(A), as these residents meet the funding eligibility criteria for household noise mitigation measures (Tang, 2021). Fourth, to assess exposure misclassification due to time-activity patterns, we repeated our analysis in the subset of participants who reported being retired at baseline given their greater probability of being at home and exposed to aircraft noise throughout the day (Spalt et al., 2016).

2.5.3. Effect measure modification

We explored whether the association between aircraft noise exposure and incident hypertension varied across strata defined by age at baseline (below or above median of 61 years), BMI (≥30 kg/m^2, ≥20 and < 30 kg/m^2, or <20 kg/m^2), smoking status (current, former, or never

Table 1
Characteristics of WHI Clinical Trials participants at risk for hypertension at baseline.[a]

	DNL, dB(A)				L_{night}, dB(A)	
	Overall	Missing, %	<45	≥45	<45	≥45
N	18,783		11,213	7570	13,510	933
Age, yrs[b]	61.3 ± 6.8	0.0	61.4 ± 6.6	61.3 ± 7.0	61.4 ± 6.6	61.3 ± 7.1
BMI, kg/m^2[b]	27.7 ± 5.4	2.3	27.7 ± 5.3	27.8 ± 5.4	27.7 ± 5.3	28.2 ± 5.7
Dietary Sodium, g[b]	2.8 ± 1.3	4.3	2.8 ± 1.2	2.8 ± 1.3	2.8 ± 1.2	2.7 ± 1.4
Physical Activity, MET hrs/wk[b]	11.4 ± 13.2	9.4	11.4 ± 13.1	11.3 ± 13.3	11.4 ± 13.2	10.7 ± 13.1
nSES, z-score sum[b]	−0.2 ± 5.2	0.0	−0.5 ± 4.7	0.2 ± 5.7	−0.4 ± 4.9	−2.1 ± 5.5
Population Density, 1000 pop/mi^2[b]	3.9 ± 5.4	2.5	1.8 ± 2.4	6.9 ± 6.8	2.4 ± 3.4	6.6 ± 6.8
Roadway Proximity, km[b]	2.1 ± 2.8	0.0	2.7 ± 3.3	1.2 ± 1.2	2.5 ± 3.2	1.0 ± 0.9
$PM_{2.5}$, $\mu g/m^3$[b]	13.4 ± 2.8	2.2	12.6 ± 2.7	14.7 ± 2.5	12.9 ± 2.8	14.5 ± 2.7
NO_2, ppb[b]	16.3 ± 7.2	2.3	12.7 ± 5.3	21.6 ± 6.2	13.9 ± 6.0	21.4 ± 6.4
Clinical Trials Arm, %		0.0				
Calcium and Vitamin D	55.1		56.5	53.0	56.3	53.0
Dietary Modification	70.0		67.6	73.6	68.3	71.6
Menopausal Hormone Therapy	41.7		44.5	37.7	43.6	40.7
Race, %		1.4				
White	88.9		93.6	81.8	92.2	77.5
Black/African American	6.9		3.9	11.3	5.0	14.5
Asian/Pacific Islander	1.4		0.5	2.6	0.7	2.6
American Indian/Alaskan Native	0.3		0.3	0.2	0.3	0.6
Native Hawaiian/Other Pacific Islander	0.0		0.0	0.1	0.0	0.1
More than one race	1.2		1.0	1.6	1.0	1.7
Ethnicity, %		0.4				
Hispanic/Latino	3.8		1.5	7.1	1.7	10.3
Education, %		0.6				
College Graduate	39.2		37.2	42.2	37.9	36.9
Income USD, %		5.4				
≥$50,000	36.2		35.9	36.7	35.7	30.2
$20,000 to <$50,000	44.1		45.2	42.5	45.2	44.6
<$20,000	14.4		14.0	14.9	14.0	19.4
Any Insurance, %		0.7				
Yes	92.9		93.6	91.7	93.5	89.7
Currently Employed, %		12.7				
Yes	39.5		39.1	40.0	39.3	39.8
Currently Retired, %		12.7				
Yes	37.2		38.7	35.0	37.9	35.7
Smoking, %		1.1				
Current	9.1		8.6	9.7	9.0	9.1
Past	40.0		38.9	41.7	39.4	40.2
Never	49.9		51.5	47.4	50.5	49.2
Alcohol, %		0.5				
≥7 drinks/wk	10.6		10.5	10.7	10.6	8.0
1 to 6 drinks/wk	27.9		27.2	28.8	27.5	26.9
None or <1 drinks/wk	61.1		61.8	60.0	61.3	64.5
Hearing Loss, %		0.7				
Mild, Moderate, Severe	18.2		18.5	17.7	18.3	18.9

Abbreviations: BMI, body mass index; dB(A), A-weighted decibels; DNL, day-night average sound level; kg/m^2, kilograms per square meter; L_{night}, nighttime average sound level; m, meters; MET hrs/wk, metabolic equivalent hours per week; mi, mile; $\mu g/m^3$, micrograms per cubic meter; NO_2, nitrogen dioxide; nSES, neighborhood-level socioeconomic status; $PM_{2.5}$, fine particulate matter; pop/mi^2, population per square mile; ppb, parts per billion; USD, United States dollars; WHI, Women's Health Initiative.

[a] Overall values excluded participants living outside of <45 dB(A) contours and living within 22.2 mi of non-study airports.

[b] Values are mean plus/minus standard deviations.

Table 2
Hazard ratios and 95% confidence intervals for the association between aircraft noise exposure and incident hypertension among WHI Clinical Trials participants.

Model	Cases	PY	Cases	PY	HR (95%CI)
	DNL ≥45 dB(A)		DNL <45 dB(A)		DNL ≥45 vs. <45 dB(A)
Model 0[a]	2179	7735	3077	11,070	0.96 (0.91, 1.11)
Model 1[b]					1.04 (0.97, 1.11)
Model 2[c]					1.00 (0.93, 1.08)
	L_{night} ≥45 dB(A)		L_{night} <45 dB(A)		L_{night} ≥45 vs. <45 dB(A)
Model 0[a]	244	827	3653	13,046	0.96 (0.84, 1.09)
Model 1[b]					1.10 (0.94, 1.27)
Model 2[c]					1.06 (0.91, 1.24)

Abbreviations: CI, confidence interval; dB(A), A-weighted decibels; DNL, day-night average sound level; HR, hazard ratio; L_{night}, nighttime average sound level; nSES, neighborhood-level socioeconomic status; $PM_{2.5}$, fine particulate matter; PY, person-years; WHI, Women's Health Initiative.

[a] Model 0 is adjusted for age, race, and ethnicity.

[b] Model 1 is Model 0 additionally adjusted for Clinical Trials arm, education, income, insurance, employment, smoking, alcohol, nSES, and WHI Center.

[c] Model 2 is Model 1 additionally adjusted for population density, roadway proximity, and $PM_{2.5}$.

smoker), reported hearing loss at baseline (mild, moderate, severe, or none), population density (above or below median of 2250 persons/mi^2), NO_2 concentration (above or below median of 14.3 ppb), and $PM_{2.5}$ concentration (above or below median of 12.9 μg/m^3).

All analyses were conducted using Statistical Analysis System (SAS) 9.4 (Cary, NC).

2.6. Results

At baseline, exposed participants (DNL and L_{night} ≥45 dB [A]) were more likely than those unexposed to: live in more densely populated areas, live closer to major roadways, live in areas with higher levels of $PM_{2.5}$ and NO_2, be a race other than White, be of Hispanic or Latino ethnicity, have lower family household income, and lack health insurance (Table 1). There was a moderate positive correlation (r = 0.35) between DNL and L_{night} estimates (Supplemental Table 1).

For the DNL analysis, 44.9% (n = 8441) of participants developed incident hypertension over 92,418 person-years of follow-up. For the L_{night} analysis, 43.4% (n = 6262) developed incident hypertension over 68,780 person-years of follow-up. Both analyses had median follow-up times of 6.0 person-years (interquartile range: 3.0–8.0) with average hypertension incidence rates of 9.1 cases per 100 person-years. Of those exposed to DNL ≥45 dB(A), 42.3% (n = 3392) developed hypertension, and of those exposed to L_{night} ≥45 dB(A), 35.7% (n = 367) developed hypertension (DNL <45 dB [A] cases 46.9%; L_{night} <45 dB [A] cases 43.9%).

In the fully adjusted model (Model 2), we observed a HR of 1.00 (95% CI: 0.93, 1.08) for DNL and 1.06 (95% CI: 0.91, 1.24) for L_{night} in relation to incident hypertension (Table 2). We did not find a positive relationship between increasing categories of noise exposure and risk of hypertension, which presented with low precision from wide and overlapping confidence intervals (Fig. 3).

Including a large number of participants living near non-study airports and considering them to be exposed (Model S2) had a minimal effect on the DNL model but led to a shift towards the null in the L_{night} model (Supplemental Table 3). Fully adjusted results were robust to other sensitivity analyses.

In stratified analyses, the association between DNL estimates of aircraft noise and incident hypertension was similar across strata defined by age, BMI, smoking status, hearing loss, or $PM_{2.5}$ (Table 3). However, the associations between DNL estimates of aircraft noise and incident hypertension were more pronounced among participants living in areas with lower population density ($HR_{interaction}$: 0.84; 95% CI: 0.72, 0.98) or lower NO_2 levels ($HR_{interaction}$: 0.82; 95% CI: 0.71, 0.95). When instead considering the associations between L_{night} estimates of aircraft noise and incident hypertension, there were similar patterns with respect to population density and NO_2, along with higher estimates among those who had BMI ≥20 kg/m^2 ($HR_{interaction}$ ≥30 vs. <20 kg/m2: 1.50; 95% CI: 1.02.2.20; $HR_{interaction}$ ≥20 and <30 vs. <20 kg/m2: 1.61; 95% CI: 1.12, 2.31) or younger women who were less than 61 years old ($HR_{interaction}$: 0.73; 95% CI: 0.56, 0.95).

3. Discussion

In this prospective analysis of 18,783 post-menopausal women without hypertension, we found no association between aircraft noise exposure and increased risk of hypertension. Results were stable across a number of sensitivity analyses, including different estimates of aircraft noise and adjustment for various potential confounding factors. Likewise, we did not observe a positive relationship with increasing noise exposure. We did identify potential effect measure modification by population density and NO_2, suggesting that the association may be stronger in populations living in areas with lower ambient noise.

In a study applying the same noise exposure measurements used here to participants in the Nurses' Health Study and Nurses' Health Study II (NHS/NHS II), we found a similar small positive association between long-term aircraft noise exposure and risk of hypertension (HR: 1.03; 95% CI: 0.99, 1.07) at the DNL 45 dB(A) cut-point (Kim et al., 2021). Both studies used large, U.S.-based prospective female cohorts that were similar in age. Notable differences between this study and NHS/NHS II include participant age at recruitment, case definition (WHI: blood pressure measurements and antihypertensive medication use; NHS/NHS II: self-reported hypertension), and assessment of nighttime exposure.

It is instructive to compare our findings with studies that both found positive and null associations between aircraft noise and hypertension. For example, a recent cohort study of residents around three French airports by Kourieh et al. (2022) found that an L_{den} 10 dB(A) increase was associated with a greater incidence of hypertension (incidence rate ratio [IRR]: 1.36; 95% CI: 1.02, 1.82); the association was similar for L_{night} (IRR: 1.31; 95% CI 1.01, 1.71). Pyko et al. (2018) conducted a cohort study investigating residents around two major airports in Stockholm County (Sweden) and showed an increase in hypertension risk (HR: 1.24; 95% CI: 1.06, 1.45) when comparing ≥45 vs. <45 dB L_{den} five years preceding the event, and a positive exposure-response relationship. Unlike our study, the French and Stockholm County studies incorporated younger women and also men, which may explain in part our lower association. Women report less annoyance to aircraft noise compared to men, and annoyance may modify the relationship between noise and hypertension risk (Babisch et al., 2013; Baudin et al., 2020; Eriksson et al., 2010). Some studies have shown increased risk of hypertension in men, but not in women (Eriksson et al., 2010; Evrard et al., 2017). Noise annoyance has also been shown to have an inverted U-shaped pattern with age, where percentage of highly-annoyed persons peaks at 45 years of age and decreases thereafter (Van Gerven et al., 2009).

Our lack of a positive exposure-response relationship with increasing noise categories was consistent with several studies (Evrard et al., 2017; Zeeb et al., 2017). This raises questions about whether any observed associations are causal but could also point towards differential exposure error at different levels of ambient noise (i.e., if individuals with higher ambient noise took actions to reduce their personal exposures or became habituated to them over time (Fidell et al., 2013)).

Similar to other studies controlling for $PM_{2.5}$ and/or NO_2 when investigating aircraft noise exposure and risk of hypertension, our results did not appear to be confounded by air pollution (Evrard et al., 2015; Kim et al., 2021; Pyko et al., 2018). Still, the current state of understanding of the joint/confounding effects of noise and air pollution on hypertension remains inconclusive due to substantial variation in

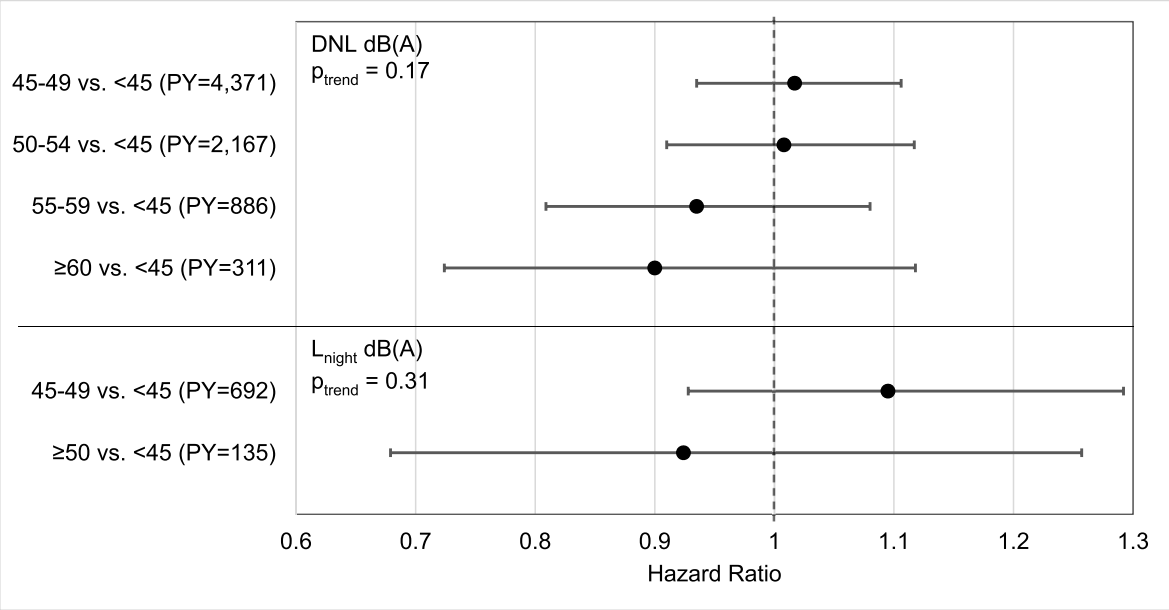

Fig. 3. Hazard ratios[a] and 95% confidence intervals for the association between ordinal aircraft noise exposure and incident hypertension among WHI Clinical Trials participants.

[a] Model is adjusted for age, race, ethnicity, Clinical Trials arm, education, income, insurance, employment, smoking, alcohol, nSES, WHI Center, population density, roadway proximity, and $PM_{2.5}$.

Abbreviations: dB(A), A-weighted decibels; DNL, day-night average sound level; L_{night}, nighttime average sound level; nSES, neighborhood-level socioeconomic status; $PM_{2.5}$, fine particulate matter; p_{trend}, p-value for trend; PY, person-years; WHI, Women's Health Initiative.

Table 3
Effect measure modification of the association between aircraft noise exposure and incident hypertension among WHI Clinical Trials participants.

Effect Modifier	DNL ≥45 dB(A)				Lnight ≥45 dB(A)			
	Cases	PY	HR (95% CI)[a]	*p*-int	Cases	PY	HR (95% CI)[a]	*p*-int
Age				0.759				0.020
≥61 years	1318	4535	1.02 (0.93, 1.11)		145	490	0.96 (0.79, 1.15)	
<61 years	861	3200	1.00 (0.90, 1.11)		99	337	1.31 (1.05, 1.64)	
BMI				0.235				0.033
≥30 kg/m^2	699	2297	1.04 (0.93, 1.16)		81	275	1.11 (0.87, 1.42)	
≥20 and < 30 kg/m^2	919	3463	0.94 (0.85, 1.04)		120	404	1.19 (0.97, 1.46)	
<20 kg/m^2	561	1976	1.04 (0.92, 1.17)		43	147	0.74 (0.54, 1.02)	
Smoking Status				0.645				0.307
Current Smoker	213	737	0.93 (0.77, 1.13)		24	77	1.13 (0.73, 1.75)	
Former Smoker	906	3233	0.99 (0.90, 1.10)		110	386	1.17 (0.95, 1.44)	
Never Smoker	1060	3764	1.02 (0.93, 1.12)		110	365	0.95 (0.76, 1.17)	
Hearing Loss				0.423				0.402
Mild, Moderate, Severe	436	1554	0.96 (0.84, 1.10)		52	185	0.96 (0.71, 1.28)	
None	1725	6113	1.01 (0.94, 1.10)		190	632	1.10 (0.93, 1.30)	
Population Density within 5 mi radius				0.026				0.364
≥2250 pop/mi^2	1827	6514	0.95 (0.86, 1.05)		206	712	1.02 (0.86, 1.22)	
<2250 pop/mi^2	352	1221	1.13 (1.00, 1.27)		38	115	1.21 (0.88, 1.68)	
NO_2				0.008				0.279
≥14.3 ppb	1805	6306	0.93 (0.84, 1.02)		207	693	1.00 (0.84, 1.19)	
<14.3 ppb	374	1429	1.13 (1.00, 1.28)		37	134	1.23 (0.87, 1.73)	
PM2.5				0.153				0.791
≥12.9 μg/m^3	1367	4518	1.00 (0.90, 1.09)		151	470	1.10 (0.91, 1.33)	
<12.9 μg/m^3	812	3216	1.10 (0.99, 1.21)		93	357	1.06 (0.83, 1.34)	

Abbreviations: BMI, body-mass index; CI, confidence interval; dB(A), A-weighted decibels; DNL, day-night average sound level; HR, hazard ratio; kg/m^2, kilograms per square meter; L_{night}, nighttime average sound level; mi, mile; NO_2, nitrogen dioxide; nSES, neighborhood-level socioeconomic status; p_{int}, p-value for interaction; $PM_{2.5}$, fine particulate matter; pop/mi^2, population per square mile; ppb, parts per billion; PY, person-years; WHI, Women's Health Initiative.

[a] Models are adjusted for age, race, ethnicity, Clinical Trials arm, education, income, insurance, employment, smoking, alcohol, nSES, WHI Center, population density, roadway proximity, and $PM_{2.5}$.

characteristics (e.g., meteorological conditions and dispersion, peak hours of exposure during the day) and potential collinearity with other confounders (e.g., lower socio-economic status, psychosocial stressors, or adverse lifestyle factors) that can often be linked with these pollutants (Münzel et al., 2020).

The environmental soundscape is a complex mixture of noise exposures, particularly when considering other sources of transportation or community noise around large urban centers. We found a stronger association with aircraft noise exposure for participants living in areas with less population density or lower NO_2 concentration, which may act as proxies for lower ambient or road traffic noise. For example, more densely populated areas may introduce noise from neighbors or social

gatherings (e.g., bars, restaurants), with the potential for those living in these areas to invest in sound insulation to mitigate their noisy surroundings, all of which may mask the effect of aircraft noise (Sørensen et al., 2021; Vienneau et al., 2019). Lim et al. (2008) found that community annoyance to equal levels of aircraft noise was higher in regions of low background compared to high background noise. In contrast, Pyko et al. (2018) found higher risk of hypertension among those exposed to both aircraft and road traffic noise ≥45 dB L_{den}. While multiple environmental stressors may mutually enhance the effect on hypertension risk, the investigators noted their finding may be due to chance as they did not find an individual effect for road traffic noise or for any other combinations with railway noise. Nevertheless, these environmental factors may be indicative of a soundscape with lesser background noise, thereby highlighting less competition from other noise sources and increased sensitivity to the effects of aircraft noise.

There were slight indications of higher risk of hypertension among those with higher BMI and nighttime noise exposure, but not DNL exposure, which may be related to the sleep disturbance pathway. Nevertheless, further research is warranted on the modifying role of cardiometabolic factors such as BMI on the aircraft noise-hypertension relationship, as our findings were subject to small sample sizes in the BMI subgroups and other studies have reported similar inconclusive results (Eriksson et al., 2014; Foraster et al., 2018).

In our study, participants had to first meet the strict WHI exclusion criteria (e.g. no competing risks, adherence, retention, safety criteria) (Hays et al., 2003). Subsequently, we required participants to have no history and be free of hypertension at the beginning of their follow-up thereby excluding nearly half of Clinical Trials participants. Therefore, our utilization of a cohort of older women means that participants had to survive free of hypertension for a longer time than studies with younger populations, resulting in survival bias that might attenuate our associations (Shaffer et al., 2021). Also, aircraft noise exposure contours have been decreasing up to at least 2010 (GAO, 2012), which may imply that hypertension risks from aviation noise could have manifested among those removed from our study population, leading to attenuated risk in an aging, prospective cohort. Therefore, factors attributed to their healthy survival as well as declines in overall exposure may contribute some bias and help explain the null or inverse relationships, as well as the elevated risk among younger participants, found in our study. Nevertheless, even a modest increase in risk of hypertension from aircraft noise may have a large impact on the development of adverse cardiovascular outcomes, such as coronary heart disease or stroke, given the prevalence of hypertension in the general population and its role as a major risk factor for CVD.

Our study presented additional limitations. First, we did not have many exposed participants at the higher decibel levels and for the nighttime noise metric, as the cohort was not originally designed to investigate the relationship between aircraft noise and health. Second, although the WHI study implemented effective retention and adherence strategies and procedures for the clinical trials, some participants may have skipped annual visits yet still have been included into our study population. Third, while AEDT is considered the accepted standard for modeling environmental noise, there is potential for exposure misclassification due to estimation of exposure using participant addresses and interpolation of data between 5-year intervals. Also, address-based estimates of ambient noise may not be representative of true personal exposure that is likely influenced by factors such as time-activity/mobility patterns, quality/construction of housing, room orientation, or window-opening behaviors (Kroesen et al., 2010; Lercher et al., 2000; Tao et al., 2020). This misclassification would likely be non-differential with bias towards the null. Fourth, we were unable to account for other sources of transportation or environmental noise that may be associated with incident hypertension. We used population density as a proxy for road traffic and urban noise, since several studies have shown associations between road traffic or urban noise and hypertension (Banerjee et al., 2014; Bluhm et al., 2007; D'Souza et al., 2021; Jarup et al., 2008). Fifth, the 90 airports we used were not randomly selected, but rather were included based on availability of operations data (FAA, 2021). Nevertheless, these airports capture the vast majority of U.S. passenger enplanements, and a wide variety of airport types. Lastly, our study population consisted of post-menopausal women, which may limit generalizability to men and younger women. These findings may highlight the higher prevalence of hypertension and lower control rates in men compared to women up to age 60 (Wenger et al., 2018). Additionally, there may be limited external validity to the U.S. population of post-menopausal women as randomization into WHI Clinical Trials may have resulted in a relatively healthier cohort. This could bias estimates towards the null and underestimate the true effect for this particular population.

Even so, our study has important strengths. Utilizing a large, prospective nationwide cohort like WHI, which representatively enrolled post-menopausal women and had widespread recruitment from 40 regional centers, allowed for a longitudinal assessment of the relationship between aircraft noise and adverse health outcomes. Additionally, we utilized aircraft noise exposure metrics that were consistently estimated using standardized methods at 90 U.S. airports over two decades. Our study was able to consider time-varying exposure alongside extensive data on personal, social, and environmental factors, and contributed some insight regarding the association between aircraft noise and health in the U.S.

4. Conclusion

After estimating aircraft noise exposure in multiple ways and using a series of analytical models, we did not observe an association between exposure to aircraft noise and risk of hypertension among post-menopausal women in the WHI Clinical Trials cohort. Our findings generally concur with similar studies assessing risk among older women. We observed indications of elevated risk among certain subpopulations, such as those who lived in areas with fewer other sources of ambient noise.

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Human studies approval

The WHI project was reviewed and approved by the Fred Hutchinson Cancer Research Center (Fred Hutch) IRB in accordance with the U.S. Department of Health and Human Services regulations at 45 CFR 46 (approval number: IR# 3467-EXT). Participants provided written informed consent to participate. Additional consent to review medical records was obtained through signed written consent. The Fred Hutchinson Cancer Research Center has an approved FWA on file with the Office for Human Research Protections (OHRP) under assurance number 0001920.

Credit author statement

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper:The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Dr. Wellenius has received consulting income from the Health Effects Institute (Boston, MA) and Google (Mountain View, CA).

Data availability

The authors do not have permission to share data.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2022.115037.

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