James Dahlgren Medical

Diplomate of the American Board of Internal Medicine
Occupational and Environmental Medicine; Toxicology;
341 Cobalt Way, suite 207, California 94085
Phone: 310-449-5525 Fax: 408-549-1057 email: dahlgren@ucla.edu

COMBAT JET NOISE FROM LANDING AND TAKING OFF AT WHIDBEY ISLAND-OLF COUPEVILLE IS PERMANENTLY INJURING THE EXPOSED POPULATION
2017

This report supplements my prior report and is being submitted on behalf of the Citizens of the Ebey's Reserve (COER) and myself. It provides a review of the Draft Environmental Impact Statement (DEIS) performed by Wyle Laboratories, a private company headquartered in the beltway around Washington DC. This private company is a huge international corporation that seems to do most of its work for the Department of Defense (DOD). It is not a scientific medical company, it is an engineering and logistic support company, not qualified to write a report on the health effects of aircraft noise on humans.

1. There are numerous failings in the Navy's DEIS. First, the personnel who authored this DEIS are not qualified to make judgments about the health effects of jet aircraft or PFOA. Here is the list of so-called "experts" responsible for the authorship of this Environmental Impact Statement:

Donald Wardell, *Biologist*
B.A. Environmental Studies, M.S. Natural Resources Sciences

James Welch, PMP, Military Operations
B.S. Math, Management, and Information Systems

Stephen McCabe, *Editor*
B.A. English, M.F.A. Creative Writing

Jenny Mogavero, GISP
B.S. Environmental/Physical Geography, M.A. Geography

Amber Lauzon, mapping
B.S. Geology, M.A. Geography

Danielle Thomas, *Graphic Designer*
A.A. Graphic Design, B.A. Psychology

Nicole Williams, *Word Processor*
A.S. Technical Studies, Certificate, MS Office

Patricia Mooney, *Word Processor*

Contractor, KBR Wyle
Brandon Robinette, Lead Engineer

Patrick Kester, Lead Engineer

As you can see from this list there's not a single doctoral level scientist, medical doctor, environmental engineer, industrial hygienist or other qualified person to comment on the issues in this case. The non-auditory health risks from noise were reviewed by Ben Sharp PhD. Internet information about Dr. Sharp is that he works at Wyle Laboratories and is an auditory engineer. He is not a medical trained person and is not qualified to comment on anything but the sound measurement issues. Reading his work in appendix A, it is clear that he thinks a statistically significant increase of 10%, 15% or 60% in death rates or risk of heart disease, hypertension and many other health problems that are outlined in the medical literature to occur from increased noise exposure is not important! His opinion is a shocking disregard for human life. The noise levels reported in almost all of the medical literature are for noise levels much lower than from naval operations at Whidbey island. Even more shocking is the fact that he failed to review all of the vast literature on the subject and reached an unsupportable conclusion that the literature on noise and non-auditory health effects is <u>not</u> settled science, but rather something so trivial that it should be ignored. The few articles he did review were glossed over in a cursory manner. The most comprehensive collection of studies performed by the World Health Organization (WHO) was not included in his review. There is a severe lack of qualification of all the Wyle Navy DEIS authors including Dr. Sharp. These facts plus the lack of accurate noise level calculations leads me to suggest that the Wyle Laboratories DEIS about Whidbey Island carries no weight whatsoever.

2. The issue in this case is impulse noise not noise sustained over long periods (1). The issue is that jet aircraft flying low over the residents creates unacceptable, acute, and unusually high levels of **<u>impulse</u>** noise (acute noise at over 100 dB(A). During the year, there are thousands of fights occurring during the day, evening and night causing serious and permanent injury to those within the DNL map outlined by the Wyle group (Appendix A). One adverse effect affecting every exposed citizen is "annoyance", which interferes with the quiet enjoyment of life. The issue of annoyance has been studied in many published papers in the peer reviewed literature (2-25). But more serious is the increased noise induced

damage to the hearing mechanism reducing hearing and causing tinnitus (i.e. cochlear injury) (26-33), disturbing sleep (34-49), raising blood pressure acutely and permanently (42, 43, 50-94), injuring the overall health (95), increasing cardiovascular diseases (47, 59, 61, 91, 92, 96-136), anxiety and other psychological problems (19, 24, 137-140), gastrointestinal disturbance (141-148), and reduced learning ability (especially in children) (24, 45, 65, 149-161). There are major injurious health effects arising from the frequent impulse noise events on Whidbey Island from the naval jets taking off, landing and flying over homes at low altitudes. The levels of impulse noise, low frequency infranoise[1] and vibration on the exposed residents on Whidbey Island are simply unacceptable. It is urgent that the noise situation be changed to protect the health as well as comfort of these citizens. As the following comment/report will show, the noise levels on Whidbey Island are a serious and real threat to the people exposed to these injurious levels of noise.

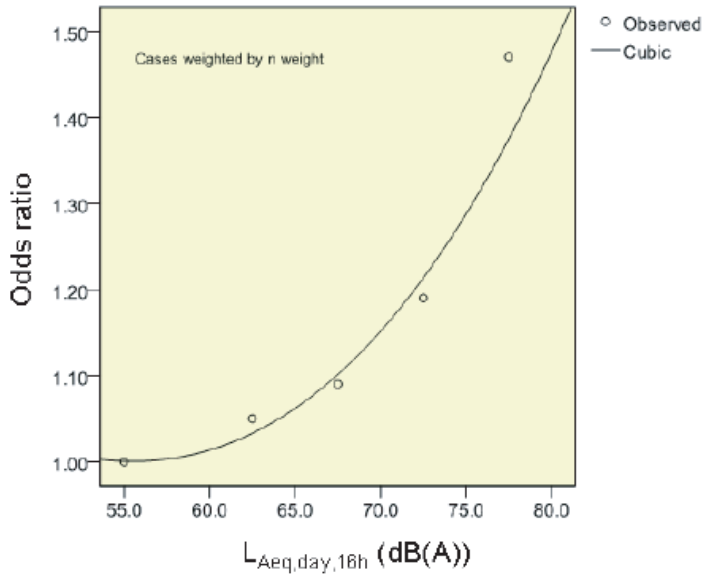
3. It is less desirable for the Wyle Laboratory personnel to take noise level estimates that they derived from their own proprietary sources rather than taking actual measurements of the sound levels at the Whidbey Island OLF. The COER expert in this litigation has measured the sound levels and they are higher than the Wyle Laboratory estimates. In every case the Wyle Laboratory estimated sound levels are below levels that have actually been measured. The second major failing in the Navy's case.
4. If we look at the Decibel Noise Level (DNL) maps provided in the DEIS (Appendix A) we see that large portions of the island are experiencing noise levels far above safe levels. According to the World Health Organization (WHO), the acceptable limit recommended by the scientists at WHO is a Leq_{16}[2] hour noise level at 60 dB(A)(136). The level at night, 11 PM to 6 AM, should be lower. There are newer studies presented below that indicate increased adverse health effects, mainly hypertension, if noise is Leq_{24} 50 dB(A) due to the presence of elevated blood pressure in people exposed to noise above that level. Here is a

1. [1] Infranoise- sound waves with frequencies below the lower limit of human audibility
   [2] Sound levels from 6 AM to 10 PM, sixteen hours.

quote from that 2011 monograph from the WHO: *"The pooled estimates and CIs are shown graphically in Fig. 2.1 (descriptive studies) and Fig. 2.2 (analytical studies). The descriptive (cross-sectional) studies (Fig. 2.1) cover the sound level range of Lday,16h from > 50 to 70 dB(A), while the cohort and case-control studies (Fig. 2.2) cover the range from ≤ 60 to 80 dB(A). The two curves together can serve as a basis for estimating the exposure–response relationship. From Fig. 2.1, it can be seen that below 60 dB(A) for Lday,16h no noticeable increase in myocardial infarction risk is to be detected. For noise levels greater than 60 dB(A), the myocardial infarction risk increases (Fig. 2.1 and 2.2)."*

5. Here is a graph from the WHO monograph with heart attack rates at Leq $_{16}$ 60 dB(A) with an increasing dose response as the Leq$_{16}$ rises.

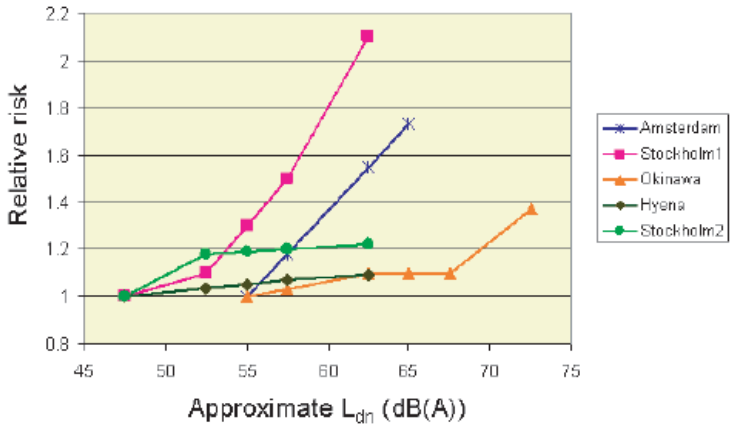
Fig. 2.3. Polynomial fit of the exposure-response relationship for road traffic noise and the incidence of myocardial infarction



Source: Babisch (21).

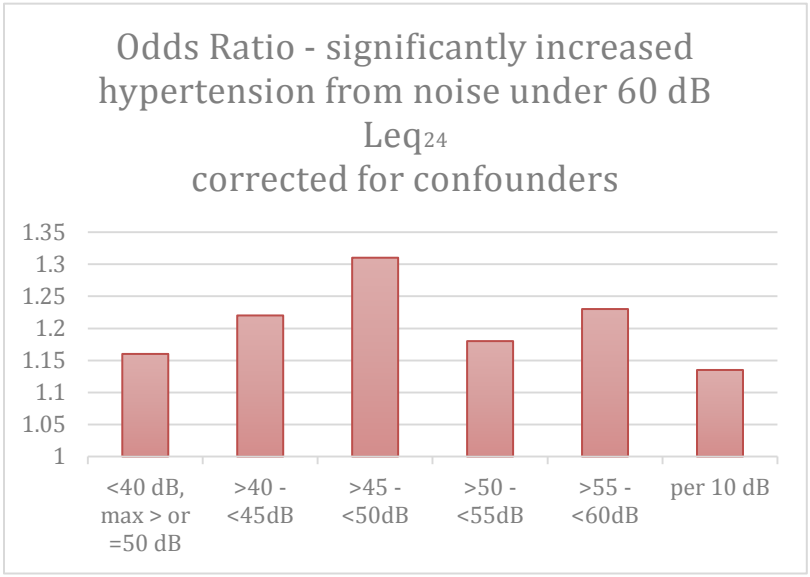
6. Hypertension is caused by excessive noise exposure; Ldn[3] is the noise metric in this graph. Here is a graph from the same WHO monograph.

Fig. 2.4. Association between aircraft noise and the prevalence or incidence of high blood pressure



Source: Babisch & Van Kamp (136).

7. Here is a graphic about significantly increased rates of hypertension caused from aircraft noise at Leq24 reported in 2016. The increased blood pressure occurs even under 40 dB if there are sound events during the night that exceed 50 dB such as an aircraft flying over the home of the subject. At 45 to 50 dB a 30% increase in blood pressure is seen (59).



[3] Ldn adds 10 dB for night measurements to allow for greater harm if the noise occurs while people are trying to sleep or are sleeping.

8. The DEIS from Wyle Laboratories was not reflective of an honest and thorough review of the scientific evidence that has been published over the last 53 years. The DEIS has a brief and inadequate summary of the extremely important non-auditory health effects of noise citing only a few studies and ignores the dozens of references that I have provided in this and prior reports. There is a large body of scientific evidence which is consistent and scientifically derived indicating severe and permanent health effects from aircraft noise and other noise sources as well. Studies of noise and health effects include aircraft, traffic and railroad as sources. It is good science to include these studies as well as the aircraft studies. The noise from all sources creates the same health effects. As you will see, the issue is noise regardless of the source of that noise.
9. For example, the Wyle authors used a PhD named Ben Sharp (no mention of the field in which he has a PhD) who reviewed a few articles on non-auditory effects of noise. Dr. Sharp quoted one study by Haralabidis 2008 (which suggested a statistically significant increase in BP from noise) but he failed to quote the methods, results and conclusion of this study. This study did 24-hour blood pressure monitoring of subjects sleeping. They then subjected the subjects while sleeping to sound pressure of 35 dB inside their home from aircraft noise. Dr. Sharp ridiculed the results by saying that they were not a large enough increase in blood pressure to be significant. Even though the changes from their baseline blood pressure was statistically significant. The authors of the study went on to note acute increases in blood pressure are associated with chronic high blood pressure and therefore the results were highly significant. The results of this study added weight to many other studies proving that even low level noise exposure can result in significant hypertension and subsequent cardiovascular disease. Here's a quote from that article in its conclusion(162): *"Within the HYENA project we found effects of long-term noise exposure on the prevalence of hypertension[18] and the acute effects reported here. Absence of short-term habituation to the cardiovascular effects of noise, especially those during sleep, found here and also reported before,[13,16,39] as well as evidence from studies on sleep-disorder which indicate that repeated arousals are associated with a sustained increase in daytime BP,[40] support*

a link between acute and long-term effects of noise exposure on hypertension[41,42] and cardiovascular disease,[43] in line with the general stress theory.[44]"

10. I would suggest that Dr. Sharp is completely and utterly incorrect in his view. Low-level noise events (i.e. >35 dB inside a house) do disturb sleep and that disturbance is very important to the health of the person. Note the maximums of noise in the Greek airport that would most closely approximate the very short term sound events (Whidbey events are louder) in Whidbey Island as found by JGL noise studies near the OLF and representative of higher levels of noise than estimated by the Wyle Laboratory EIS. Here is a graphic of the noise levels from Haralabidis et al:

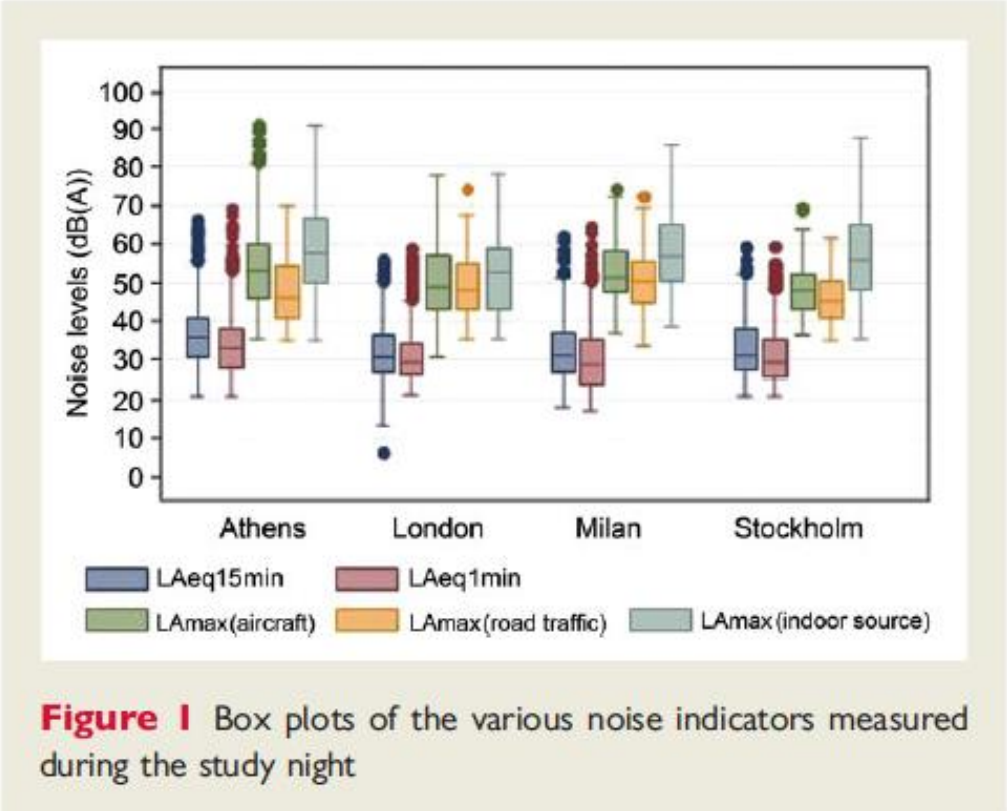


Figure 1 Box plots of the various noise indicators measured during the study night

11. The Haralabidis study indicates that the presence of aircraft noise creating a sudden, although slight, increase in sound pressure during sleep causes an adverse health effect which is not trivial as opined by Dr. Sharp. The finding of an event during the night that resulted in a spike from sound pressure >35 dB created a statistically significant rise of blood pressure of 6.2 mm systolic & 7.4 mm diastolic over 15 minute intervals when an aircraft event occurred.

12. A 2001 Swedish study reported a statistically significant increased prevalence of hypertension in people around an airport. The sound levels are far lower than in Whidbey Island. The maximum sound levels were above 72 dB(A) but in between sound levels were lower <55 dB(A). This like other studies indicate that the impulse noise adds to the stress response that startles the person, an unexpected rapidly rising relatively loud sound creates more stress and thus not only immediate increase in blood pressure but also permanent high blood pressure. Here is a table from that study:

Table 3 Prevalence of hypertension among residents with exposure to equal energy (FBN) and maximum (MNL) levels of aircraft noise, stratified by sex, age, and hearing loss

	n	*Hypertension*	*%*	*n*	*Hypertension*	*%*	*POR**	*95% CI**
	FBN <55 dBA			*FBN >55 dBA*				
Men	1291	199	15	64	14	22	1.7	0.9 to 3.3
Women	1530	196	13	74	13	18	1.4	0.8 to 2.8
<55 y	1975	166	8	96	10	10	1.2	0.6 to 2.5
≥56 y	846	229	27	42	17	40	1.9	1.0 to 3.7
Hearing loss	517	110	21	23	4	17	0.7	0.2 to 2.4
No hearing loss	2284	277	12	115	23	20	2.0	1.2 to 3.2
	MNL <72 dBA			*MNL >72 dBA*				
Men	1285	198	15	70	15	21	1.8	0.9 to 3.4
Women	1530	195	13	74	14	19	1.7	0.9 to 3.2
<55 y	1969	165	8	102	11	11	1.4	0.7 to 2.7
≥56 y	846	228	27	42	18	43	2.2	1.1 to 4.1
Hearing loss	517	109	21	23	5	22	1.1	0.4 to 3.3
No hearing loss	2279	276	12	120	24	20	2.1	1.3 to 3.4

*Prevalence odds ratio (95% CI) adjusted for age, sex, smoking, and education (except when stratified by sex).

13. For comparison to, the noise levels in these studies we can compare the noise measured by JGL at the Whidbey Island OLF. Sound attenuation inside a home from the flyover by the Growler jets varies depending on several factors. A reasonable sound attenuation inside buildings estimate would be between ~15 dB and ~25 dB according to the Wyle DEIS. The indoor noise levels rang from an average in the five locations, after attenuation, from 99 to 109 dB or 83 to 93 dB(A). This estimate does not take into account the greater impact of noise while sleeping, which would raise the levels impact by 10 dB(A). When a person is asleep they are more prone to blood pressure spikes than the same sound pressure event during waking hours. The Growler jets are in fact creating very high sound pressures, which are much higher than the levels at a civilian airport. Here is JGL data:

Table 1. Noise level statistics at each measurement location.

Statistic	Pos. 1	Pos. 2	Pos. 3	Pos. 4	Pos. 5
Maximum A-weighted Level (dBA)	119.2	113.4	115.7	114.3	81.1
Maximum Un-Weighted Peak Level (dB)	134.2	126.7	130.6	131.4	101.8
Session SEL (dBA)	128.5	124.5	122.7	127.7	92.1
Session Duration (minutes)	39	58	45	36	25
Total Jet Flyovers	35	43	26	28	8
Average SEL per Jet Flyover (dBA)	113.1	108.2	108.5	113.2	83.1

These sound measurements indicate a health damaging noise events for the neighbors to the Coupeville OLF thousands of times per year.

14. The Wyle Group used the occupational exposure limit of 90 dBA for an eight-hour exposure from OSHA as a reference. Wyle claims that this is level that causes permanent hearing injury. The OSHA level is not an appropriate metric and has no relevance to the neighborhood around the airfield. The use of hearing loss as the only permanent health related end point used by the Wyle group is wrong. A serious investigation of health effects from a noise exposure must take into consideration the more sensitive end points such blood pressure, learning impairment, sleeping, mental health, cardiovascular effects to name only a few documented health effects of noise below an environmental level of 90 dB. Hearing loss does occur and it occurs at lower than 90 dB in children and if there is an impulse noise above 90 dB. The main point is that hearing loss is not the most sensitive health end point impacted by noise, especially sudden, unexpected and sharply rising noise levels as we have in this case.
15. Vienneau et al. performed a meta-analysis that shows the safety oriented end point will result in noise levels far lower than 90 dB(A). The issue is non-auditory health effects, which have been shown to occur at lower levels than hearing loss (163):

"*We identified 10 studies on road and aircraft noise exposure conducted since the mid-1990s , providing a total of 12 risk estimates. Pooled relative risk for IHD was 1.06 (1.03–1.09) per 10dB increase in noise exposure with the linear exposure–response starting at 50 dB*". This is 50 dB metric is outside the

building and is not predicated on 40 years of exposure. People exposed to 90 dB(A) noise outside their home will have 6% increase or 24% of people will have hypertension because the rate of increase is for EACH 10 dB(A) increase in sound level.

16. There are several studies that link aircraft noise to increased occurrence of hypertension(58, 59, 61, 87, 123). Another study quoted by Dr. Sharp is Evrard published in 2015, which showed that there is a statistically significant increase in: Coronary Heart Disease – Odds Ratio 1.23; Cardiovascular Disease – Odds Ratio 1.18; Myocardial Infarction – Odds Ratio 1.31; for **each** 10 dB increase in the range of 42 to 64 dB. Dr. Sharp seems unaware of the fact that such findings are highly significant. Any occupational and environmental medical doctor would agree that these findings are highly significant and not trivial. Hypertension causes heart disease and strokes. It is not trivial or of no importance, especially when you realize that noise has many other undesirable adverse health effects.
17. Another study by Evrard et al in 2016 (87) notes the following data in a table:

Table 3 Effects estimates of various aircraft noise indicators* on hypertension and BP in men

	Hypertension		Diastolic BP		Systolic BP	
Indicator of exposure	OR† (95% CI)	p Value	Increase in mm Hg‡ (95% CI)	p Value	Increase in mm Hg‡ (95% CI)	p Value
L_{den} (dB(A))	**1.48 (1.00 to 1.97)**	**0.04**	**1.86 (0.40 to 3.30)**	**0.01**	**2.37 (0.16 to 4.59)**	**0.04**
$L_{Aeq,\ 16\ hours}$ (dB(A))	1.34 (0.90 to 1.79)	0.10	**1.51 (0.11 to 2.92)**	**0.03**	**2.19 (0.05 to 4.34)**	**0.05**
L_{night} (dB(A))	**1.34 (1.00 to 1.97)**	**0.04**	**1.67 (0.34 to 3.00)**	**0.01**	**2.17 (0.13 to 4.19)**	**0.04**

Bold values are statistically significant $p \leq 0.05$.
*Per 10 dB(A) increase.
†Adjusted for age, gender, BMI, physical activity, alcohol consumption and professional activity.
‡Adjusted for age, gender, BMI, physical activity, alcohol consumption, professional activity and hypertensive medication.
BMI, body mass index; BP, blood pressure.

This table makes the point that very low sound levels cause a significant increase in risk for hypertension from aircraft noise at a civilian airport where noise levels are much less than on Whidbey Island. The range of sound levels was from 42 to 64 dB(A). (Again the increase in blood presssure for each increment of 10 dB(A)). This data is similar to the other studies noted above and below.

18. In a study in Holland by Franssen, the same pattern emerges(42). Here are the noise levels around this civilian airport:

Table 2 Description of the aircraft noise exposure measures in the study population

Exposure measure	Range	Average	SD
L_{den}	41–76 dB(A)	51.3	3.1
$L_{Aeq,\ 22\text{-}23\ h}$	36–70 dB(A)	44.3	4.1
$L_{Aeq,\ 23\text{-}07\ h}$	32–65 dB(A)	37.9	4.0
Kosten units	0–64 Kosten units	17.3	6.8

Franssen published this table showing many health problems including hypertension and sleep disturbance with the sound levels much lower than experienced by residents of Whidbey Island;

Table 5 Odds ratios (OR) and 95% confidence intervals (CI) after multiple logistic regression of health indicators, in relation to various noise exposure measures per 10 dB(A) increase in noise levels, controlling for potential determinants

Health indicator	n_{total}	n_{effect}	Noise measure	OR	95% CI
Poor self-rated health (single question)	10412	1969	L_{den}	1.23	1.04 to 1.46
			$L_{Aeq,\ 23\text{-}07\ hrs}$	1.05	0.91 to 1.22
Poor self-rated health (VOEG score)	9887	1871	L_{den}	1.21	1.02 to 1.43
			$L_{Aeq,\ 23\text{-}07\ hrs}$	1.08	0.94 to 1.25
Medication for cardiovascular diseases/increased blood pressure	10105	1316	L_{den}	1.30	1.06 to 1.60
			$L_{Aeq,\ 23\text{-}07\ hrs}$	1.13	0.94 to 1.35
Prescribed sleep medication or sedatives	7240	516	L_{den}	1.25	0.93 to 1.68
			$L_{Aeq,\ 23\text{-}07\ hrs}$	0.91	0.70 to 1.18
			$L_{Aeq,\ 22\text{-}23\ hrs}$	1.26	0.99 to 1.60
Non-prescribed sleep medication or sedatives	7240	309	L_{den}	2.34	1.63 to 3.35
			$L_{Aeq,\ 23\text{-}07\ hrs}$	1.20	0.87 to 1.65
			$L_{Aeq,\ 22\text{-}23\ hrs}$	1.72	1.27 to 2.32
Frequent use of sleep medication or sedatives	7175	189	L_{den}	1.02	0.63 to 1.65
			$L_{Aeq,\ 23\text{-}07\ hrs}$	1.36	0.91 to 2.04
			$L_{Aeq,\ 22\text{-}23\ hrs}$	1.15	0.78 to 1.70

19. A later study by Eriksson in 2007 reported a statistically significant incidence of hypertension from aircraft noise (88). Here is a table from that study with results consistent with the findings of numerous other studies:

TABLE 3. Association Between Aircraft Noise Exposure and Cumulative Incidence of Hypertension Among Men in Stockholm* Following Exclusion of Those Smoking or Using Snuff Directly Preceding Blood Pressure Measurements

Noise Exposure	No.	No. with Hypertension	Crude RR	Crude (95% CI)	Adjusted† RR	Adjusted† (95% CI)
Energy-averaged aircraft noise level						
Continuous [per 5 dB(A)]			1.17	(1.07–1.29)	1.15	(1.05–1.25)
Dichotomous						
<50 dB(A)‡	1281	375	1.00		1.00	
≥50 dB(A)	301	117	1.33	(1.13–1.57)	1.29	(1.11–1.50)
Maximum aircraft noise level						
Continuous [per 3 dB(A)]			1.16	(1.06–1.27)	1.15	(1.06–1.25)
Dichotomous						
<70 dB(A)‡	1354	401	1.00		1.00	
≥70 dB(A)	228	91	1.35	(1.13–1.61)	1.32	(1.12–1.55)

*Based on subjects with complete data on exposure and confounding variables.
†Adjusted for age and BMI.
‡Reference category.

20. The HYENA project from the Haralabidis study (81, 162) investigated short-term changes of noise levels on blood pressure and heart rate during night-time sleep in subjects living near airports. Both blood pressure levels and heart rate increased with higher noise levels, independently of the noise source and of the sequence of the measurement during sleep time, which indicates absence of habituation during the study at night. The project also found effects of long-term noise exposure on the prevalence of hypertension and acute effects or arousals. There is a consistent absense of short-term habituation to cardiovascular effects of noise, as described in the HYENA project and and other studies (47, 48) as well as sleep-disorder evidence leading to increase in BP (164) . These authors indicate this supports a link between acute and long-term effects of noise on hypertension and cardiovascular disease(63, 88, 102)
21. The Wyle group consistently relies upon the "DNL." In 2011, they put together a 2011 report titled "Updating and Supplementing the Day-Night Average Sound Level (DNL). Discussed is the creation of DNL, including the reliance on the Schultz's curve, and described as "a dose-response curve that was easy to apply to quantify noise impact." Contradictory to its recent report on Whidbey, the Wyle group says *"While the Schultz curve is a valuable tool, it tends to give the impression that community noise impact is well represented by a single dose-response curve based on DNL. The curve is often treated as a black box, to be derived from social surveys and improved by curve fits of additional data. With so many data points in the current form, and the rather small differences with improved versions, it is questionable how much might be gained from continuing that kind of approach."* This means that including the peak levels is extremely important in supplementing the average. They go on to discuss, "annoyance is a multivariate function that includes many effects such as Speech interference; Sleep disturbance; Task interference; Impairment of classroom learning; Non-auditory health effects; and Aversive effects on emotion and tranquility." None of these items they previously considered important are properly researched or taken into account within the current DEIS. Similarly, the few literature citations are not

accurately reported in the Wyle 2016 draft concerning Whidbey Island DEIS for the US Navy.

22. Additionally, the Wyle group evaluated 628 social surveys on the response of residents to noise, 1943-2008. Wyle provides a number of insights and analyses in their study of surveys, the importance of surveys, and they provide recommendations to be included in future surveys. In referring to surveys, they make the observation, *"But those involve aircraft not typical of today's commercial fleet in the United States, may have been analyzed as much as possible, and/or are not structured such that models like Equations (2) or (3) can be fitted. If results are required that have not already been developed from prior surveys, then it is expected that a new survey that is designed for the candidate metrics would be required."* Whidbey Island is an obvious candidate for such a community survey conducted by a reputable epidemiologist and medical doctor, yet no community survey was performed within the context of the current DEIS.
23. None of the learning interference, speech intelligibility, non-auditory effects noted for school children noted the fact that the children live in the neighborhood and are in fact exposed outside of the school. The school children all live in residences within the contours of high sound pressures. An 8-hour school day is not the proper measurement for noise exposure of these children.
24. **The effects of noise on children and the susceptibility of children to health effects has been disregarded completely by the Wyle group.** A large review article reported an adverse effect on children from chronic noise exposure. The children had elevations of resting blood pressure, attentional deficiencies, and deficits in reading (165). Other effects include diminished task motivation, deficits in auditory disctractors, poorer memory when high information processing demands are present, and deficits in infant cognitive development. The attached Appendix B highlights the tables from this study showing the large number of studies and research on this subject. The effect on children is more distrubing than the effect on adults because the noise factor is reducing the child's ability to fulfill their full potential academically, not to mention adverse health effects that will require life-long and increased medical care.

25. Cohen in 1980 found that children from noisy schools have higher blood pressure and are more likely to give up on a task than children from quiet schools (166). Increase in time of exposure led to children being more distractible. He also found that prolonged noise exposure affects cognitive processes and there is a lack of adaptability in children shown in both blood pressure and cognitive processes. Cohen followed up tothis article in 1981 establishing the stability of these effects over time and the lack of adaptation to noise over extended periods of time (167).

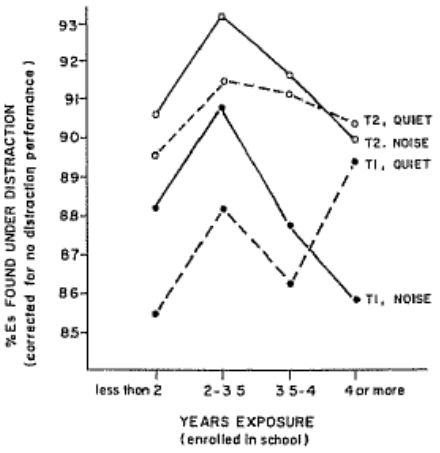


Figure 1. Distractibility at T1 and at T2 as a function of school noise level and duration of exposure (Each period on the number of year's of exposure coordinate represents one quarter (based on quartiles) of the sample. For example, 25% of the sample were enrolled in school for less than 2 years.)

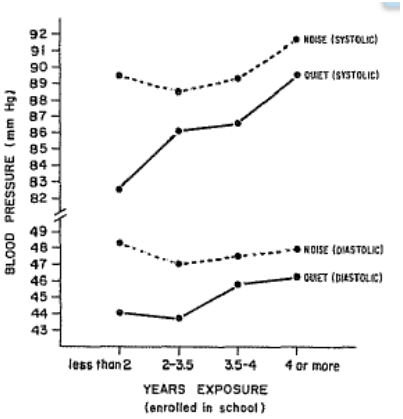


Figure 2. Systolic and diastolic blood pressure as a function of school noise level and duration of exposure. Each period on the number of years of exposure coordinate represents one quarter (based on quartiles) of the sample. For example, 25% of the sample were enrolled in school for less than 2 years. (From "Physiological, Motivational, and Cognitive Effects of Aircraft Noise on Children: Moving from the Laboratory to the Field" by Sheldon Cohen, Gary W. Evans, David S. Krantz, and Daniel Stokols, *American Psychologist*, 1980, *35*, 231–243. Copyright 1980 by the American Psychological Association. Reprinted by permission.)

26. Blood pressure and the effect of noise on annoyance, distractability and performance lasts. School children in noisy environments were poorer at solving test puzzles and tasks.Results from a nonauditory effects study show that children living in noisier areas of residential communities are subject to stress even if at modest levels (both noise and "stress")(168). The study examined multimethodological indices of stress among children living under 50 dB or above 60 dB but within ambient community noise levels. "Children residing in noisier areas of communities have marginally higher resting systolic blood pressure, greater heart rate reactivity to an acute stressor, and higher overnight cortisol levels indicative of modestly elevated physiological stress."

27. Psychophysiological activation, particularly blood pressure, is correlated with airport noise exposure among children. A number of articles describe how both acute and chronic exposure leads to blood pressure increases in children near source exposure (17, 114, 169, 170)
28. The DEIS fails to outline the harmful effects from the PFOAs and PFOs being used on site. PFOA and PFOS are highly toxic chemicals then have a long half-life in the body ranging from a few years to decades. These chemicals bio-accumulate, they are absorbed in the body at a rate faster than which is lost. The ratio between these chemicals in the water and the bio-concentration in people is 1 ug/L of PFOA in water leads to a 105 ug/L in their blood (171). Since PFOA has a much shorter half-life in the body, 3 years for PFOA versus 30 years for PFOS, the blood concentration of PFOS will be even higher from 1 ug/L in water (172). Using a water level of 0.070 ug/L of PFOA will create a level of 7.35 ug/L (ppb) in the blood of people using that water. This adds to the amount from other background sources. Since scientific research continues to find more adverse effects of PFOA at lower and lower doses, it is prudent take appropriate steps to protect the public health of people on Whidbey Island by reducing the exposure levels to as low as possible, preferably zero.
29. It is my understanding that the US Navy on Whidbey Island has contaminated water with PFOA and PFOS for both the municipal water supply and many private wells. Such contamination has or will result in adverse health effects in people using that water. It is my understanding that some of the water wells tested as high as 0.600 ug/L, which translates into 60 ug/L ppb of PFOA in the blood of people using that water. As you can see in the table below (Figure 4.11), these chemicals in the water give a very high level compared to background. The highest blood levels are for PFOS because it has such a long ½ life of 30 years.
30. As you can see from the attached list of articles in Appendix C, these chemicals, even at low-levels, causes alteration in many organ systems and can cause cancer, heart disease, thyroid dysfunction, birth defects and many other adverse health effects. The background level of PFOA blood level in individuals in the United States is about 7 ug/L (ppb) due to the fact that hundreds of consumer products

contain PFOA. The contamination on Whidbey Island Will greatly increase the level in the body of the individuals using that water for all purposes. The contamination creates a significant internal dose of toxic chemicals and adds to the adverse health effects from the usual toxic load of chemicals to which we are all exposed.

31. Is my understanding that removing the PFOA from water is extremely expensive and difficult, especially getting down to the extremely low levels in the parts of quadrillions. As with any cancer-causing agent, there is a risk even at the lowest possible level and therefore the ideal exposure level should be zero. As Russell Train the first head of the United States Environmental Protection Agency (USEPA) stated, "We live in a mine field of risk." All of the studies of PFOAs which there is so called "safe levels" are based on single chemical exposure studies and not the complex web of chemicals and other environmental stresses to which the citizens of Whidbey Island are exposed in addition to the PFOA and PFOS.
32. It is essential that the U.S. Navy stop using the PFOA and PFOS containing fire-fighting foam. It is also necessary that the U.S. Navy provide clean and safe domestic water to all of the inhabitants of Whidbey Island, not just bottled water.
33. It is also essential with U.S. Navy conduct blood tests on all of the residents of the Island. The exposed individuals must be provided medical care to detect any adverse health effects from the exposure. This medical monitoring program is needed for the rest of their lives so they can be diagnosed early and at the most optimal treatment time. This medical monitoring program would need to be administered by doctors who are educated about the adverse health effects of PFOA and PFOS. A similar program in West Virginia allowed many thousands of people to receive optimal medical care for their PFOA poisoning. The citizens of Whidbey Island are entitled to similar considerations.
34. The addition of exposure from the domestic water supply makes the levels much higher than background. Here is a table, also from De Witt 2015, showing much higher blood levels of these chemicals from the communities that have detectable

levels of PFOA and PFOS in their water: The table includes background levels of PFOA and PFOS (173):

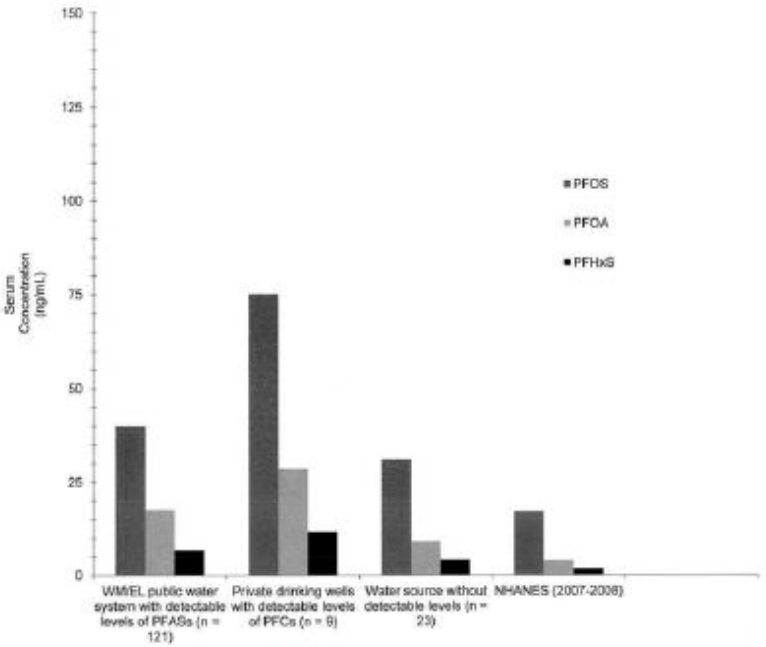
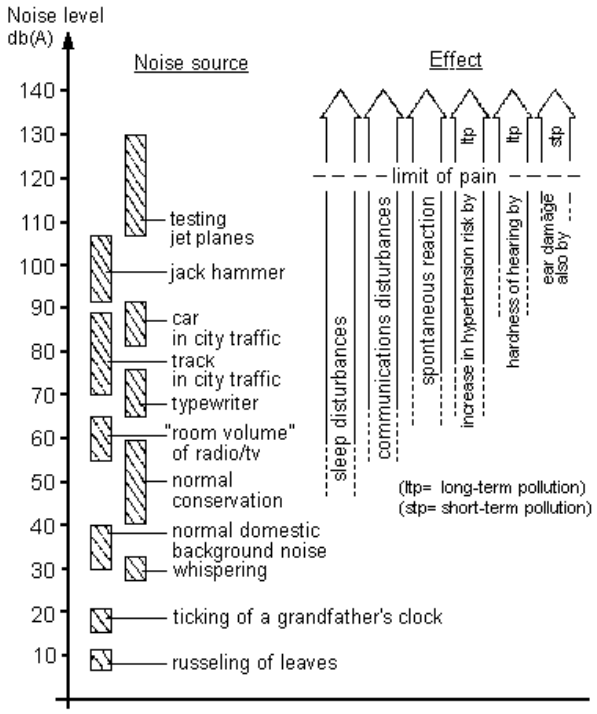


Fig. 4.11 Geometric serum PFOS, PFOA, and PFHxS concentrations in residents of affected communities through drinking water exposure near Decatur, AL compared to NHANES

The next section of this report adds additional peer reviewed scientific articles on the serious and well documented adverse health effects of noise. This report will also describe the health effects that have almost certainly already occurred from years of noise pollution from thousands of take offs and landings of the combat jets in Whidbey Island. The issue in this case is whether there is evidence that this noise pollution actually harms people. The Navy sponsored 2016 DEIS has suggested or states that there is no proof of harm to health from the jet noise. Or, if there is harm, it is negligible according to the Wyle Laboratory personnel.

1. Noise pollution is unwanted or harmful sound that intrudes upon human activity. Here is a graphic that describes where jet aircraft noise compares with other loud noise.



This figure illustrates levels of sound pressure or noise from different sources and explains the weighting of dB(A) (103)

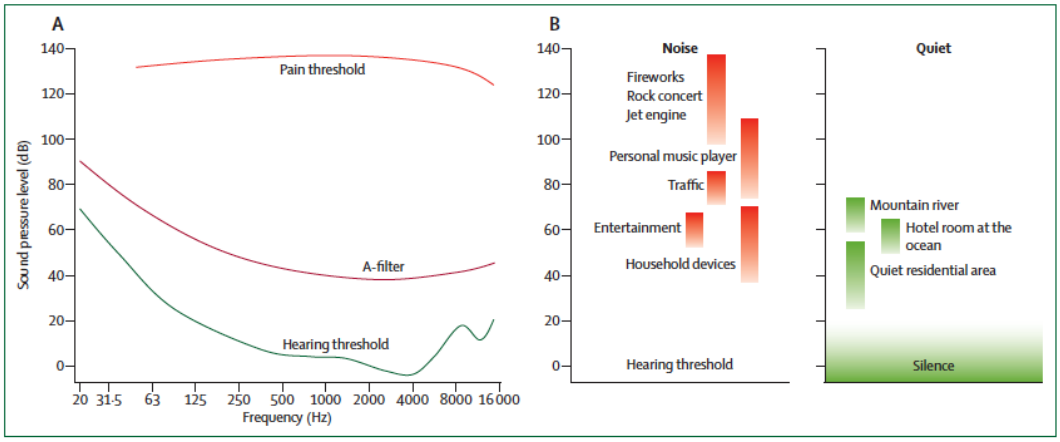
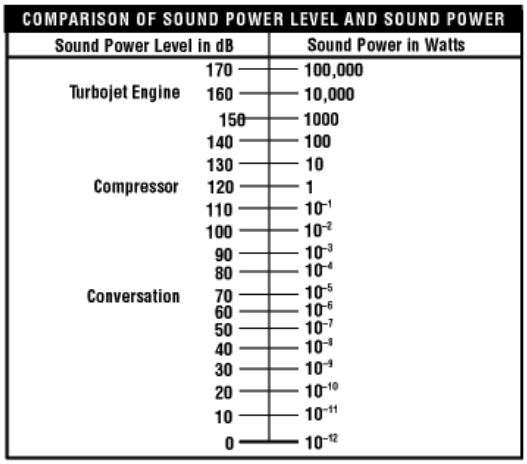


Figure 1: **Sound pressure levels**
(A) The sensitivity of the auditory system depends on sound frequency and sensitivity is highest between 2000 Hz and 5000 Hz (green line). The A-filter (dark red line) is a frequency-weighting of sound pressure levels that mimics the sensitivity of the auditory system (eg, low-frequency sounds contribute little to the A-weighted dB level). (B) A-weighted sound pressure levels for several environmental sounds, emphasising that whether or not a sound is perceived as noise depends largely on the context and the individual, and is only partly determined by its sound pressure levels. For example, spectators attending a rock concert might not perceive the music as noise, whereas residents in the vicinity of the venue might call it noise, even though sound pressure levels are much lower there than for inside.

2. Noise is measured in the amount of pressure occurring at different levels and is noted in decibels (dB) on log scale. An increase from 90 to 100 dB is not a 10% increase; it is a 10-fold increase in the pressure. There are two basic causative types of health effect impacts from noise pollution: (a) those arising from short term, but high intensity sound (impulse noise), and (b) those arising from longer term exposure to lower levels of sound. The first type manifest in close encounters with military jets landing and taking off such as we have at the Whidbey Island Navy OLF and Air Station(118). The Michalak study showed severe increases in blood pressure in elderly subjects from military jets flying low over residences. The study by Ising et al showed adverse effects on auditory function and blood pressure in children from low flying military jets (12, 114). Rapid acute changes in sound pressure/noise is more damaging than steady sound levels. Here is a

quote from Ising 1990: *"Our findings support the contention that auditory effects of MLAF (Military Low Aircraft Flight) noise are correlated with the Lmax rather than the Leq. In both the 75 m area and the 150 m area, the Leq values (68 dB(A) and 59 dB(A), respectively) were far below the minimum level for noise induced hearing loss (80 - 85 dB(A)). However, according to Spreng (1988), the difference in maximum noise levels (125 dB(A) and 112 dB(A), respectively could explain the area difference audiometric findings."*

3. Noise level exposure is strongly associated with permanent hypertension, heart attacks, anxiety, depression, gastrointestinal changes, and learning impairment. The association in epidemiological studies is not the only evidence that noise causes adverse health effects; there are animal and mechanistic studies that explain how noise pollution at the levels and circumstances present on Central Whidbey Island causes these health problems. The weight of the evidence provided shows that noise is causative of serious injuries.
4. Although noise pollution is *annoying, annoyance* is by no means the only adverse health effect. Decades of research have shown that the issue of noise pollution is a serious, disabling and even a life-threatening issue. The loud, short-term noise from the Navy jet flying over Whidbey Island is an issue of life and death. The noise exposure levels that have been documented from low flying combat jets in this case and in the literature, are in the range that is certain to injure some members of the exposed population, particularly the elderly and children.



5. Both JGL and AICUZ measured annual sound levels well above 70 dB in the COER neighborhood. In a Navy document[4] on environmental impact on the residents near Coupeville OLF it states, *"Residential land uses are normally considered incompatible with noise levels above 65 DNL."* Even more egregious, the Navy states there is no scientific evidence that has shown the noise pollution at OLF Coupeville from combat jets taking-off and landing to be hazardous to health.
6. In fact, the noise impact from combat jets, like the situation in this case, has been studied. The high-level noise exposure from a combat jet flying over a person has been shown in a scientific study to causes a significant increase in blood pressure and "shock" to the body with some individuals becoming acutely ill from the noise. If the noise rises and subsides quickly, such as occurs in this case when there are multiple jets flying one after the other, the blood pressures do not return to the pre-noise level and continues to climb higher and higher. This is shown in the graphic below from a published, peer reviewed study of combat jet noise by Michalak and colleagues (118).

[4] Page I-14, Final Environmental Assessment for the Transition of Expeditionary EA-6B Prowler Squadrons to EA-18G Growler at Naval Air StationWhidbey Island, Oak Harbor, Washington. October 2012.

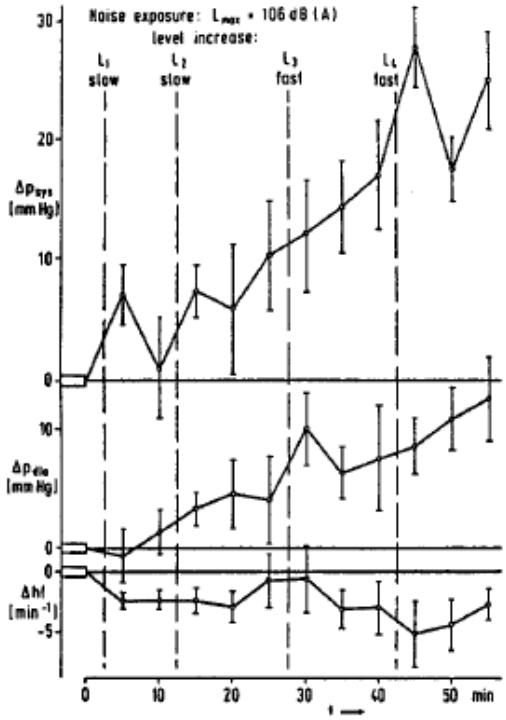


Fig. 3. Time response of blood pressure and heart rate changes after low-altitude flight noise exposure on Day 0 in subjects who had a maximum systolic blood pressure increase of more than 25 mm Hg. The differences for each person are related to the averaged initial values before the first noise exposure. The averaged values and standard errors of those differences ($n = 8$) are presented

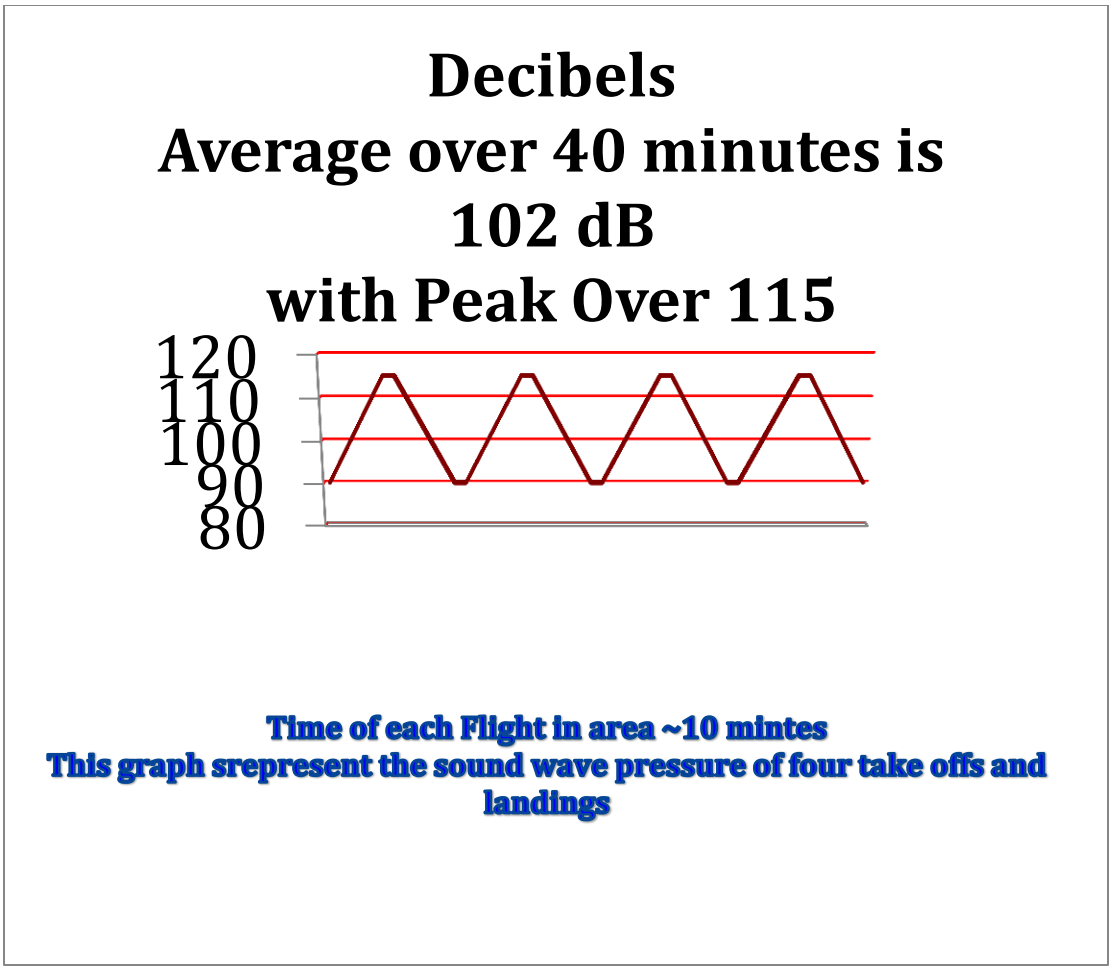
7. The Michalak study of combat jet noise documented that the people exposed to combat jet aircraft noise significantly raised their blood pressure and the brief noise exposure at these levels made some of the test subjects sick. The dB (A) levels used in this experiment were 106, 110 and 112 dBA. The graphic above used 106 dB and as you can see the blood pressure rises significantly at that level. The "shock" reaction and acute illness occurred when the noise level rose quickly, as occurs around the Coupeville OLF. When the noise rose quickly, by 30 dB over 0.4 seconds, as opposed to 4.0 seconds, 10 to 20% of the subjects experienced "shock" and sickness. Two study subjects had 40 mm rise in systolic blood pressure after four fly-overs at a maximum of 106 dBA. These authors noted that repeated exposures were additive, each exposure drove the BP higher, especially in the subjects who had an

initial higher BP response. The presence of these blood pressure elevations is most dangerous, especially if it occurs repetitively for months and years. Severe acute increases in blood pressure are itself dangerous. Acute elevations of blood pressure can trigger strokes and heart attacks. In this case the people near Coupeville OLF have been exposed for years. We do not know if there have been strokes or heart attacks triggered by these fly overs, but it is likely that such events have occurred. Michalak et al noted that elderly people are more sensitive to adverse effects from combat jet fly over noise. Wyle laboratories suggest that community surveys be conducted where there are noise pollution problems yet this has not been done for purposes of this DEIS.

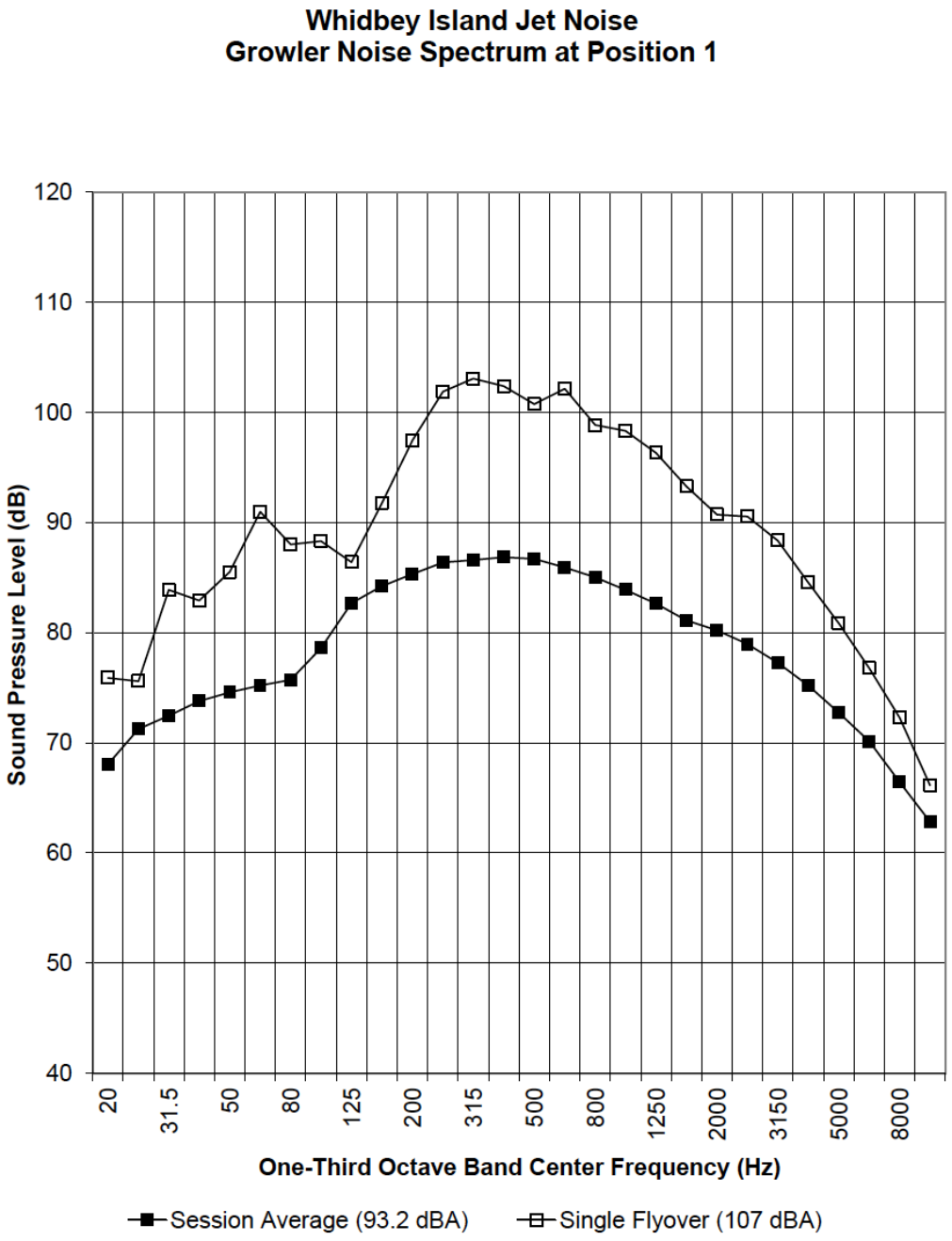
8. The development of noise-induced hypertension discussed below has occurred in community noise exposures of adults and children and in noise exposed workers. Several community studies have stressed that aircraft noise is more harmful than traffic noise. The noise patterns that have been studied in communities and in factories do show adverse effects, even when the noise is rising and falling as it does in Coupeville. It is the repeated stress reactions that lead to permanent hypertension. Noise induces an acute stress reaction, which over time becomes permanent.
9. The Michalak research examined people living in noisier versus quieter areas. They found that girls, ages 10 to 13 that lived in the noisier area compared to the quieter, reported higher blood pressure by an average of 9 mm systolic. Such elevation of blood pressure can lead to permanent hypertension.
10. In addition, the subjects of the combat jet experiment became sensitized to the jet noise pattern. Becoming sensitized or developing permanent conditioned response meant that when the test subjects heard the jet noise at a lower level intensity, they responded with a similar rise in blood pressure to the high level of noise. This

sensitization or conditioned response occurred even though the level of noise was not elevated to the point that it would have been predicted to cause the blood pressure to rise. This study is very relevant for the Whidbey Island population. The conditioned reflex means that when they hear the jet approaching the BP rises even before the jet noise reaches the subject.

11. Michalak's study refutes the DEIS study and the former base commander, Captain M. K. Nortier's, opinion that there is no evidence of health effects from the type of noise generated by the Navy's Growler jets flying, landing and taking off from Coupeville OLF.
12. The noise pattern at Central Whidbey Island has been measured and the noise levels are higher than the Michalak study. The noise measured at OLF Coupeville is illustrated by this graphic derived from JGL's study:



Here is the graph of the sound frequency pattern from the study of noise at OLF Coupeville area:



13. An exhaustive monograph by the World Health Organization (WHO) on the subject of adverse health consequences of auditory and non-auditory effects of noise writes in the abstract (103):

"Our understanding of molecular mechanisms involved in noise-induced hair-cell and nerve damage has substantially increased, and preventive and therapeutic drugs will probably become available within 10 years. Evidence of the non-auditory effects of environmental noise exposure on public health is growing. Observational and experimental studies have shown that noise exposure leads to annoyance, disturbs sleep and causes daytime sleepiness, affects patient outcomes and staff performance in hospitals, increases the occurrence of hypertension and cardiovascular disease, and impairs cognitive performance in schoolchildren."

World Health Organization (WHO) summarized the evidence of the non-auditory adverse health effects in these two paragraphs from page 16:

"Non-auditory health effects of noise have been studied in humans and animals for several decades, using laboratory and empirical methods. Biological reaction models have been derived, based on the general stress concept (17,23–30). Noise is a nonspecific stressor that arouses the autonomous nervous system and the endocrine system (9,11–14,31,32) (C. Maschke & K. Hecht, unpublished data, 2005). A neuro-endocrinological definition of stress is that it is a state that threatens homeostatic or adaptable systems in the body (16,33,34). Increased allostatic load is associated with various diseases, including ischaemic heart disease (35). The epidemiological reasoning is based on three facts. First, experimental studies in the laboratory have been carried out for a long time and revealed an increased vegetative and endocrine reactivity during periods of exposure (1,36–70). However, the question regarding long-term effects of chronic noise exposure cannot be answered from short-term experiments. Second, animal studies have shown manifest disorders in species exposed to high levels of noise for a long time (71–83). However, effects in humans and animals cannot be directly compared, particularly because two pathways may be relevant – the direct effect due to nervous innervation and the indirect effect due to the cognitive perception of the sound; the latter is certainly different in humans. Furthermore, noise

levels in animal studies were higher than in ambient situations. Third, occupational studies have shown health disorders in workers chronically exposed to noise for many years (20,84–98). However, noise levels were higher than in the ambient environment. Epidemiological research has therefore been carried out with respect to community noise levels to test the hypothesis and to quantify the risk.

Among other non-auditory health end-points, short-term changes in circulation, including blood pressure, heart rate, cardiac output and vasoconstriction, as well as stress hormones (epinephrine, norepinephrine and corticosteroids), have been studied in experimental settings for many years (32,99). Classical biological risk factors have been shown to be elevated in subjects that were exposed to high levels of noise (44,54,79,100–111).”

There are millions of disability lost life years from noise pollution as illustrated by this figure from the WHO study(103):

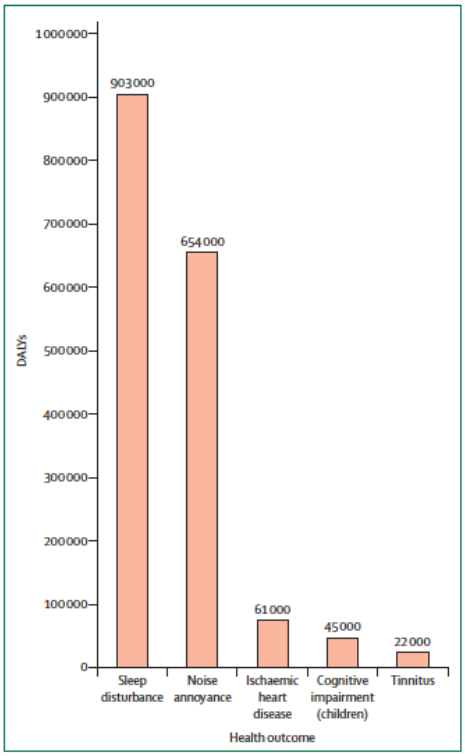


Figure 2: **DALYs attributed to environmental noise exposure in Europe.** According to WHO,[14] more than 1 million healthy life years (DALYs) are lost annually because of environmental noise exposure in European A-member states alone. Most of these DALYs can be attributed to noise-induced sleep disturbance and annoyance. DALYs=Disability-adjusted life years.

14. WHO utilized a very large database of studies and derives conclusions from hundreds of studies that note serious, adverse health effects from even modest elevations of noise levels. Most of the studies of interest have found injurious effects at sound levels far lower than those experienced by the residents of Whidbey Island. The residents experiencing noise pollution from the jets landing and taking off at OLF Coupeville on Whidbey Island are experiencing the adverse health effects that we would expect including: annoyance, hearing loss, sleep disturbance and cardiovascular problems. The WHO monograph illustrates the serious nature of what has happened and still is happening to the citizens living near the Coupeville landing field. The impact on the health of these people is certain to be devastating and has likely already increased morbidity and even shortened their lives.
15. The community of Central Whidbey Island is adversely impacted by the noise from combat jets landing and taking off from the Navy's practice airfield. In 1978 the US EPA published a monograph on noise pollution and recommended that community noise levels not exceed 70 decibels to prevent hearing loss (174). They included a graphic, which indicates that a community with significant noise pollution does react vigorously and justifiably if there are elevated noise levels. Here is the graphic from that monograph.

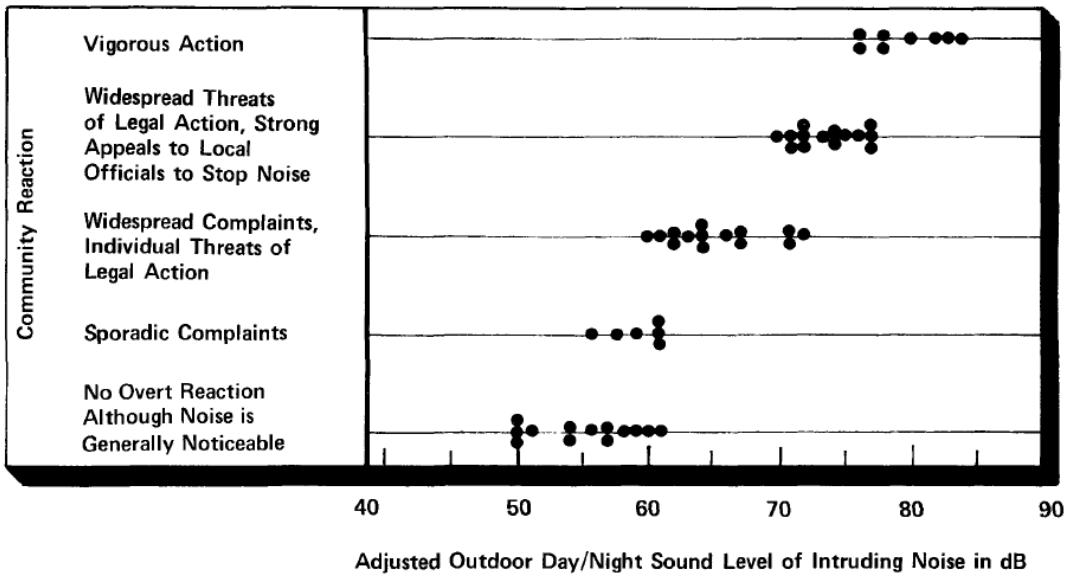


FIGURE 11. COMBINED DATA FROM COMMUNITY CASE STUDIES ADJUSTED FOR CONDITIONS OF EXPOSURE

This graphic suggests that until the community noise levels are less than 70 dB, the community will be up in arms. The JGL studies of the sound levels near the landing field are indicative of noise levels that results in vigorous community reaction.

16. A study of noise and whole body vibration [5] finds that the combination of noise and vibration is additive, causing more health problems than with noise alone (175). These authors also looked at the susceptibility of some people to be more impacted by noise and vibration. The people who were under chronic medical care by a doctor labeled as unhealthy had a greater adverse reaction to the noise and vibration than healthy people. Here is a table from that study.

[5] The residents near the Coupeville OLF experience whole body vibration and shaking of building caused by the Growler Jets.

TABLE 2

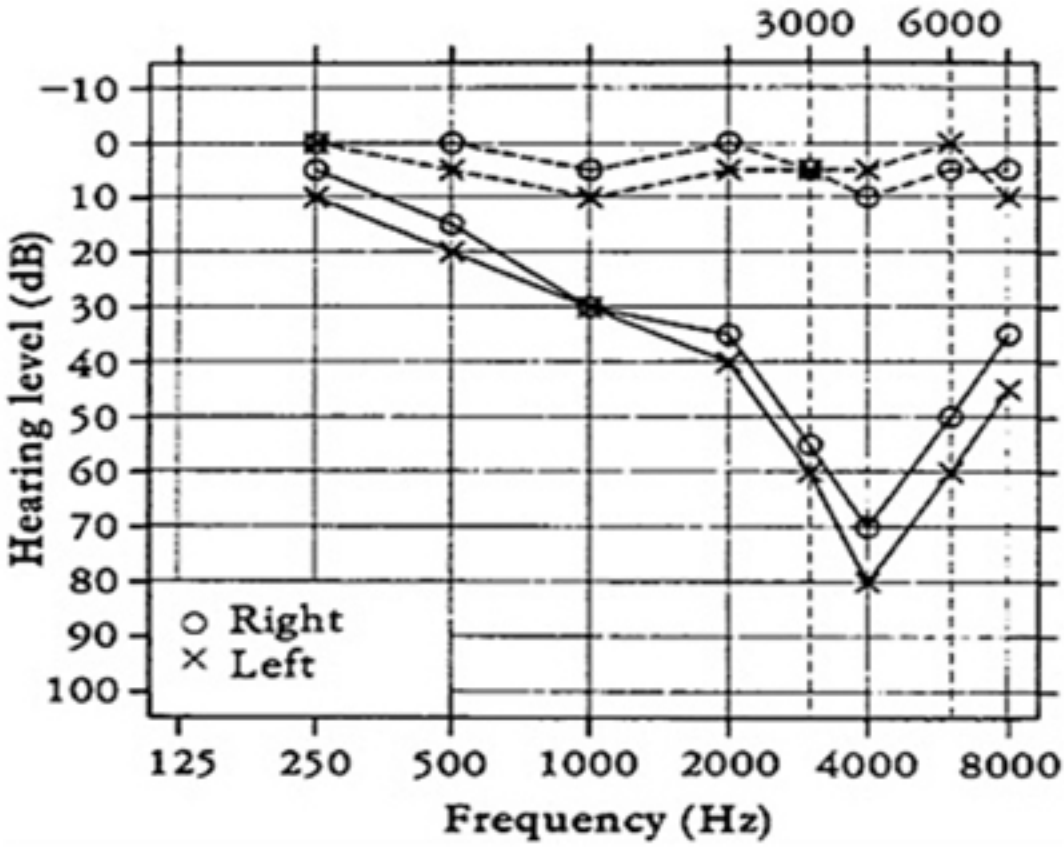
Average number of positive responses to 190 *questions by health status*

	Healthy	Unhealthy
Number of respondents	893	294
Average number of positive responses	29·6	42·6**

The difference in the average number is significant at the 1% level(**) compared to healthy respondents, according to the Student *t*-test (two-tailed).

By no means does this imply that the healthy people did not have adverse non-auditory responses to the noise and vibration energy. Rather, the noise and vibration significantly increases the harm to those already sick

17. There is evidence that the OLF Coupeville area residents have already developed noise induced hearing loss. A loss of hearing in the higher frequencies that is typical of noise induced hearing loss. Here is the pattern that we see in one of the residents who has been tested and we would see if we tested other residents, a drop of hearing at 4000 Hz.

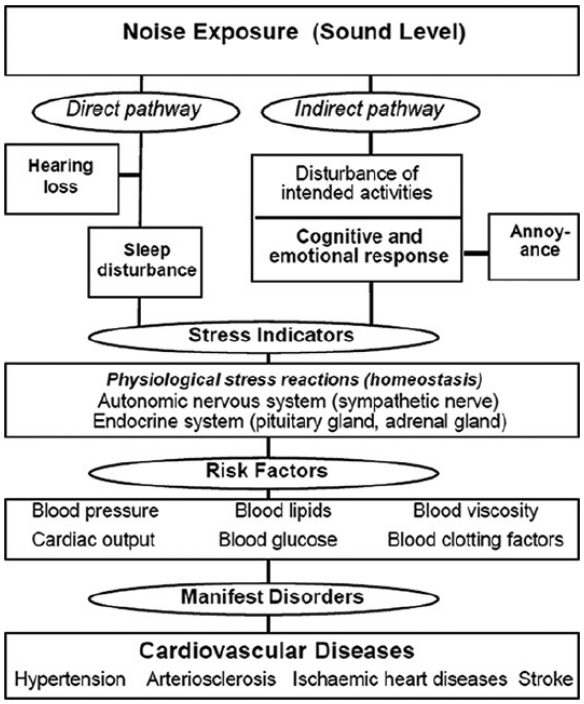


18. The 2011World Health Organization (WHO) published the monograph quoted above that reviewed the literature on adverse auditory and non-auditory health effects caused by noise. WHO concluded that in addition to hearing loss, non-auditory health effects were a serious public health and environmental health problem(136). WHO quoted numerous high quality studies to document the deadly effect of noise on cardiovascular health.
19. The study by Babisch and colleagues in Germany provided a meta-analysis of increase levels of noise causing cardiovascular problems, heart attacks and ischemic heart disease. Here is a graphic from a 2014 article that illustrates the pathways (101). Below is a table from Babisch showing an increased risk of ischemic heart disease and heart attacks (myocardial infarction) arising in the context of noise exposure levels far below the noise levels documented on Whidbey Island when the jets land and take off at OLF Coupeville.

Study-type (country)	Reference	Number of subjects	Number of cases	Response rate (%)	Covariates	Age (years)	Exposure assessment [dB (A)]	Health outcome
Caerphilly-CS (United Kingdom)	Babisch *et al.* 1993[29]	M: 2,512	M: 438	89	A, G, B, C, S, E, P, M, F, O	45-59	$L_{Aeq16\,h}$ ≤55->65	Prevalent IHD (clinical)
Speedwell-CS (United Kingdom)	Babisch *et al.* 1993[29]	M: 2,348	M: 340	92	A, G, B, C, S, P, F, O	46-63	$L_{Aeq16\,h}$ ≤55->65	Prevalent IHD (clinical)
Berlin I-hCC (Germany)-hCC	Babisch *et al.* 1994[26]	M: 243	M: 109	Cases: 89 Controls: 87	A, G, B, C, S	41-70	$L_{Aeq16\,h}$ ≤60->75	Incident MI (clinical)
Berlin II-pCC (Germany)	Babisch *et al.* 1994[26]	M: 4,035	M: 645	Cases: 91 Controls: 64	A, G, B, C, S, M, O	31-70	$L_{Aeq16\,h}$ ≤60->75	Incident MI (clinical)
Berlin II-CS (Germany)	Babisch *et al.* 1994[26]	M: 2,375	M: 206	64	A, G, B, C, S, M, O	31-70	$L_{Aeq16\,h}$ ≤60->75	Prevalent MI (self-reported)
Tokyo-CS (Japan)	Yoshida *et al.* 1997[30]	F: 3,950	F: 305	73	A, G	20-60	$L_{Aeq16\,h}$ ≤55->70	Prevalent CHD (self-reported)
Caerphilly-CO (United Kingdom)	Babisch *et al.* 1999[25]	M: 2,369	M: 281	Follow-up: 94	A, G, B, C, S, E, P, M, F, O	45-59	$L_{Aeq16\,h}$ ≤55-70	Incident MI (clinical)
Speedwell-CO (United Kingdom)	Babisch *et al.* 1999[25]	M: 2,330	M: 290	Follow-up: 78	A, G, B, C, S, P, F, O	46-63	$L_{Aeq16\,h}$ ≤55-70	Incident MI (clinical)
Berlin III-hCC (Germany)	Babisch *et al.* 2005[27]	M: 3,054 F: 1,061	M: 1.528 F: 354	Cases+controls: 84	A, G, B, C, S, M, F, O	20-69	$L_{Aeq16\,h}$ ≤60->70	Incident MI (clinical)
Netherlands-CO (The Netherlands)	Beelen *et al.* 2009[14]	M+F: 105,269	M+F: 3,089	Follow-up: high (death register)	A, G, C, S, O, Nb	55-69	$L_{Aeq,\,maxa}$ ≤50->65	Incident IHD (mortality)
Stockholm-pCC (Sweden)	Selander *et al.* 2009[13]	M+F: 3,518	M+F: 1,466	Cases: 72 controls: 70	A, G, C, B, S, P, N, O	45-70	$L_{Aeq24\,h}$ <50-≥60	Incident MI (clinical)
Stockholm/Gothenborg/ Malmo-CS (Sweden)	Eriksson *et al.* 2012[15]	M+F: 2,498	M+F: 161	59	A, G, C, S, N	18-80	L_{denr} <50-≥65	Prevalent CVD (self-reported)
Vancouver-CO (Canada)	Gan *et al.* 2012[16]	M: 189,713 F: 222,707	M+F: 3,095	Follow-up: high (death register)	A, G, C, O, Nb	45-85	L_{den} ≤58->70	Incident CHD (mortality)
Copenhagen/ Aarhus-CO (Denmark)	Sørensen *et al.* 2012[17]	M: 24,294 F: 26,319	M: 1,184 F: 416	Follow-up: 89	A, G, B, C, S, E, P, N, O	50-64	L_{den} ≤50->70	Incident MI (clinical)

[a]Maximum LAeq of weighted Lday, Levening, Lnight. [b]Studies provided adjusted and non-adjusted results regarding air pollution. M = Males, F = Females, MI = Myocardial infarction, CHD = Coronary heart disease, IHD = Ischaemic heart disease, CVD = Cardiovascular disease, CS = Cross-sectional, CO = Cohort, hCC = Hospital case-control, pCC = Population case-control, Covariates: A = Age, G = Gender, S = Smoking, B = Body mass index, P = Physical activity, C = Social class indicator, E = Alcohol intake, F = Family history of MI, M = Marital status, N = Air pollution, O = Other. Non-adjusted results were used in this meta-analysis

Another graphic from Babisch shows the multiple cardiovascular effects of noise:



20. The adverse effect of environmental traffic noise on cardiovascular health remains even when the impact from concomitant air pollution is controlled (90). There are numerous studies, analyzed by the weight of the evidence, that provide overwhelming evidence that noise exposure causes hypertension in both adults(50, 52, 53, 56, 57, 60, 62, 64, 66, 67, 75, 76, 80-82, 84-86, 88, 90, 93, 94, 97, 101, 103, 119, 176-184) and children(34, 65, 74, 103, 168). The duration and the dB level of the noise act together, the higher the exposure the shorter the duration of exposure that is needed. Noise induces a reaction in the body of immediate increase in many elements that raise blood pressure and other risk factors for cardiovascular damage, such as blood lipids. I include a bibliography of relevant articles that give a sense of the amount of information we have on this aspect of noise related personal injury (15, 35, 51-53, 56, 62, 67, 68, 84, 85, 92, 99, 103, 113, 119, 177, 180, 181, 184-188). Several studies document aircraft noise, specifically, as a cause of the adverse effects of noise.
21. One study of noise notes a dose response of noise and HBP. There is a large increase in hypertension prevalence as the sound pressure (SPL) increases (50). Here is a table from that study:

A dose response relation for noise induced hypertension

Table 2 Sound pressure level and prevalence of hypertension in female textile mill workers

SPL dB(A)	*No with hypertension*	*Total*	*Hypertensive prevalence (%)*
104	25	164	15·2
96	25	294	8·5
86–90	18	428	4·2
75–80	11	215	5·1
Total	79	1101	7·2

22. As with all diseases there is a genetic susceptibility factor. Not everyone exposed to noise develops clinically significant hypertension, cardiovascular disease, or other adverse effect. There is a well-described phenomenon of gene-environment interaction. An excellent prospective study followed hundreds of subjects exposed to noise over a 20-year period and measured their hypertensive susceptibility gene sub-types. A gene known to increase the risk of high blood pressure (HBP) is the angiotensin TT gene. In this study the presence of the TT subtype and noise exposure were synergistic for developing hypertension (82). The noise levels experienced by these subjects were less than the Central Whidney Island subjects. Duration of exposure in some subjects was similar. Contrary to The Navy's DEIS, it does not take 40 years of noise exposure at 90dB to cause hearing loss.
23. The effect of noise at night when people are trying to sleep occurs at very low level and there is growing evidence that night time noise is devastating to health. Here is a graphic from the WHO monograph on night time, **outside** noise:

Table 5.4
Effects of different levels of night noise on the population's health[2]

Average night noise level over a year $L_{night,outside}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{night,outside}$ of 30 dB is equivalent to the NOEL for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. $L_{night,outside}$ of 40 dB is equivalent to the LOAEL for night noise.
40 to 55 dB	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

24. If we were to study the Whidbey Island residents that have been exposed to the very high levels of aircraft noise at night we would find an increase in the prevalence and severity of hypertension and cardiovascular disease.
25. Noise-disturbed sleep is linked with multiple health effects. Sleep is a physiological state required for normal recuperation by the body and systems. Reduction and disruption are detrimental. In a clinical review of research and literature, Muzet finds sleep awakenings and sleep stage modifications that occur between 45 and 55 dB and above over the long-term can lead to detrimental health impacts (37). Partial sleep deprivation induces tiredness, increases a low vigilance state, and reduces both daytime performance and the overall quality of life (41). Sleep deprivation activates levels of stress known to be linked to hypertension, cardiovascular disease and other severe medical

problems.

26. The presence of increased noise especially aircraft noise pollution has been associated with learning problems in children (189). Here is a table showing this association.

Table 2. Multilevel Model Parameter Estimates for the Impact of Aircraft and Road Traffic Noise at School on Children's Cognitive Performance and Health Outcomes, United Kingdom RANCH Project, 2001–2003

Variable	Aircraft and Road Traffic Noise at School Adjusted for Sociodemographic Factors[a]							
	Original Sample ($n=960$)				Air Pollution Subsample ($n=719$)			
	No. of Participants	β[b]	95% CI	*P* Value	No. of Participants	β[b]	95% CI	*P* Value
Cognitive outcomes								
Reading comprehension	864				651			
Road traffic noise		−0.001	−0.014, 0.011	0.80		−0.002	−0.017, 0.013	0.77
Aircraft noise		−0.010	−0.020, 0.0005	0.06		−0.011	−0.022, 0.00021	0.05
Recognition memory	844				641			
Road traffic noise		−0.012	−0.046, 0.021	0.47		−0.012	−0.048, 0.023	0.50
Aircraft noise		−0.035*	−0.061, −0.009	0.01		−0.042*	−0.069, −0.016	<0.01
Information recall	837				638			
Road traffic noise		0.039	−0.030, 0.108	0.27		0.040	−0.014, 0.094	0.14
Aircraft noise		−0.025	−0.080, 0.028	0.35		−0.040	−0.082, 0.001	0.06
Conceptual recall	834				636			
Road traffic noise		−0.007	−0.008, 0.022	0.37		0.007	−0.007, 0.021	0.31
Aircraft noise		−0.011	−0.023, 0.001	<0.01		−0.015*	−0.025, −0.004	<0.01
Working memory	785				580			
Road traffic noise		0.038	−0.063, 0.142	0.45		0.036	−0.096, 0.167	0.60
Aircraft noise		−0.004	−0.063, 0.142	0.92		0.00077	−0.096, 0.097	0.99
Health outcomes								
Psychological distress	842				634			
Road traffic noise		−0.025	−0.084, 0.032	0.38		−0.030	−0.093, 0.033	0.35
Aircraft noise		−0.017	−0.064, 0.029	0.46		−0.023	−0.073, 0.026	0.36
Self-rated health	868				655			
Road traffic noise		0.0006	−0.024, 0.025	0.96		0.003	−0.024, 0.030	0.82
Aircraft noise		0.002	−0.018, 0.022	0.83		0.007	−0.015, 0.028	0.54
Systolic blood pressure	351				276			
Road traffic noise		−0.09	−0.25, 0.08	0.22		-0.092	−0.303, 0.118	0.39
Aircraft noise		0.02	−0.12, 0.15	0.77		0.024	−0.131, 0.179	0.76
Diastolic blood pressure	351				276			
Road traffic noise		0.02	−0.11, 0.15	0.76		0.042	−0.125, 0.211	0.61
Aircraft noise		0.01	−0.09, 0.12	0.83		0.019	−0.104, 0.144	0.75

27. The levels of noise that interfere with children's learning are far lower than the levels at central Whidbey Island. There are schools in the noise impacted area near OLF Coupeville. Those children are surely suffering from impaired learning ability due to the frequent loud noise impacting their schools. In the above study they review a number of studies that document serious noise induced impairment of children's cognitive function. The intermittent high and rapid increase and decrease in noise levels that are typical of aircraft noise cause more problems with learning than a continuous noise source (190). The

presence of an unexpected sound is more disruptive than a sound of the same level that is expected. The brain responds to an unexpected sound because we are hard wired to detect an unusual or unintended sound as possible danger. Here is a quote from Banbury et al. on this issue (191).

"When considering the functional character of the sense of hearing – as opposed to vision – one is struck by its omnidirectional nature and the fact that it has the capacity to receive information at almost all times, even in darkness or during sleep. To these features of hearing is added a superlative capacity to respond to change. Part of the evolutionary refinement of hearing has been its capacity to respond to sharp changes in energy, which might be associated with danger in the environment. Given these qualities, it comes as no surprise that hearing has been dubbed "the sentinel of the senses." This capacity to capture attention even while a person is otherwise engaged can be exploited usefully for the purpose of designing alarms. However, the same sentinel capacity carries with it the disadvantage that our attention will be captured by sounds with no relevance or significance, even when we are intent on concentrating on something else."

28. The non-auditory adverse health effects of sound include stomach ulcers and other GI problems(192). Here is a graphic from Jorge da Fonseca et al. study of rats exposed to low frequency noise (LFN) <200Hz. The sound pressure from the Growler jets shows the highest sound pressure (dB) at these lower Hz/frequencies (see JGL's study). The rats experienced severe damage to stomach tissue.

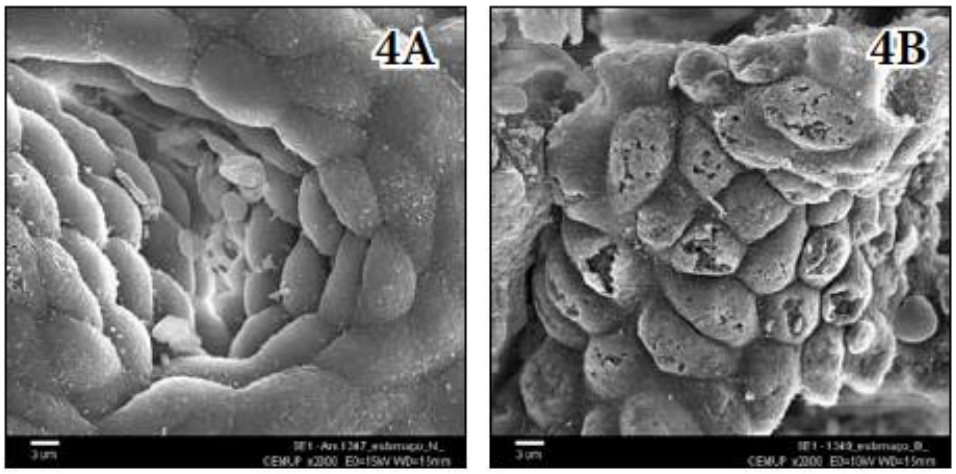


Fig. 4. *SEM micrographs of epithelial layer of rat stomach (x 2,000). Normal aspects from control rat (Fig. 4a) and massive cell death observed in noise-exposed rat (Fig. 4b).*

The effect on stomach tissue was due to direct impact of the sound waves on the tissue. The Growler jets have a pattern of noise frequency that includes sound wave frequency down to 10 Hz. The study of Low Frequency Noise (LFN) explains why a person feels the high level of sound in their gut. It is likely that the gastrointestinal system of the Whidbey residents is impacted adversely by the frequent loud noise especially the lower frequency sounds. Studies of people and dogs exposed to loud noise have altered stomach acid secretions and ulcers (193). Patients' with Crohn's disease have sensorineural hearing loss at 4000 Hz, the exact type of hearing loss caused by noise (143, 194-196). The authors of the Crohn's disease studies have not ascribed the sensorineural hearing loss to noise injury but rather to autoimmune damage to the auditory nerve. The pattern of hearing loss is only caused by noise exposure! The finding of gastrointestinal damage in the studies of noise-induced injury to the intestinal tract makes it more likely that the Crohn's disease is caused by noise exposure in susceptible people.

29. Low Frequency Noise and noise frequencies below audible ranges, i.e. infranoise, have received less attention than audible noise. However,

there is evidence that it adds to the risk, especially the risk of non-auditory effects such as gastrointestinal effects. One study found increased GI effects even though the subjects were wearing ear protection, presumably not experiencing threshold shifts in hearing. The subjects still felt the non-auditory effects of noise, experiencing GI symptoms including diarrhea (197).

30. Studies of non-auditory effects of noise pollution causing gastrointestinal problems include a number of animal and human studies that establish GI upset as a likely and common problem for the noise impacted residents of Whidbey Island (142, 144, 146, 147, 193, 198-204).
31. The residents of Whidbey Island are experiencing sudden, unexpected, incontrollable, unwanted loud sounds. The noise and vibration is intense enough to cause unacceptable interference in their lives. In addition to the serious physical effects caused by the jet noise, the citizens of the Coupeville OLF area are denied the quiet enjoyment of life.
32. The science quoted above indicates that there is solid uncontroverted evidence that health problems have occurred in the exposed population. If the flights continue more health damage will occur. My methodology to reach conclusions about the effect of noise and health is based on the weight of the evidence. There are nine considerations when determining causation, often referred to as the weight of the evidence (205). The nine "Hill viewpoints" are fulfilled in the case of noise and health impairment. The viewpoints are (1) strength of association i.e. increased relative risk or similar metric showing a higher than expected occurrence of disease or end-point of interest, (2) consistency i.e. the studies are generally in agreement, (3) specificity i.e. do the studies show the same effect in various populations, (4) temporality i.e. did the exposure occur before the outcome, (5) biological gradient i.e. is there a dose response, (6) plausibility i.e. the

cause and effect consistent with known biology, (7) coherence i.e. does the body of evidence make sense without major confounding (other effects that would occur with the cause of interest), (8) experiment i.e. do animal studies or laboratory simulations reflect a similar outcome and is there a mechanism that links the cause and effect, and (9) analogy i.e. does the cause have parallels from other cause and effect paradigms such as in this case other forms of stress causing similar outcomes. All of these factors do not need to be present to establish causation. In this case all of the elements are present, providing sufficient evidence for a conclusion that excessive noise causes the serious illnesses: auditory, cardiovascular, learning, psychiatric, neurological and gastrointestinal systems illnesses. The very high short term and repeated noise pollution present in and around OLF Coupeville on Whidbey Island from the jets landing and taking off is a certain cause of ill health. Noise pollution from the combat jet exercises must cease immediately to protect the health of the people living there.

James Dahlgren MD
February 16, 2017

Brief Biography

James Dahlgren MD is an board certified internist retired assistant professor from UCLA School of Medicine. His CV is attached in Appendix D. He has been in private practice of internal medicine with a sub-specialty in toxicology for forty-five years. He has studied and treated thousands of patients with toxic chemical injuries including numerous victims of toxic chemical poisoning including the subjects dramatized in the Erin Brockovich movie. Dr Dahlgren has been treating and evaluating people with exposure to noise and PFOAs includes evaluating patients with noise induced hearing loss and an increased prevalence of hypertension. One of these studies was presented at the American Public Health Convention. I studied the health effects of PFOA on hundreds of resisdents in Parkersberg, West Virginia. These subjects were exposed to PFOA and PFOS in their water supply. Some of the studies have been published in the peer reviewed medical literatur or at scientific conferences. Dr Dahlgren's work lead to a very large and important epidemiology study in the PFOA water contaminated zone that included 60,000 residents. Dr Dahlgren's clinical practice included thousands of subjects with a complex exposures to toxic and stressful events, resulting in adverse health effects.

References

1. Wunderli JM, Pieren R, Habermacher M, Vienneau D, Cajochen C, Probst-Hensch N, et al. Intermittency ratio: A metric reflecting short-term temporal variations of transportation noise exposure. Journal of exposure science & environmental epidemiology. 2016;26(6):575-85.
2. Zijlema WL, Morley DW, Stolk RP, Rosmalen JG. Noise and somatic symptoms: A role for personality traits? Int J Hyg Environ Health. 2015;218(6):543-9.
3. Westman Jc WJR. Noise and stress: A comprehensive approach. Environmental health perspectives. 1981;41:291-309.
4. Sung JH, Lee J, Park SJ, Sim CS. Relationship of Transportation Noise and Annoyance for Two Metropolitan Cities in Korea: Population Based Study. PLoS One. 2016;11(12):e0169035.
5. Shepherd D, Dirks K, Welch D, McBride D, Landon J. The Covariance between Air Pollution Annoyance and Noise Annoyance, and Its Relationship with Health-Related Quality of Life. Int J Environ Res Public Health. 2016;13(8):792.
6. Schultz TJ. Synthesis of social surveys on noise annoyance. The Journal of the Acoustical Society of America. 1978;64(2):377-405.
7. Rylander R. Noise, Stress, and Annoyance. Noise & Vibration Worldwide. 2006.
8. Rathsam J, Loubeau A, Klos J. Effects of indoor rattle sounds on annoyance caused by sonic booms. The Journal of the Acoustical Society of America. 2015;138(1):EL43-8.
9. Ragettli MS, Goudreau S, Plante C, Perron S, Fournier M, Smargiassi A. Annoyance from Road Traffic, Trains, Airplanes and from Total Environmental Noise Levels. Int J Environ Res Public Health. 2015;13(1).
10. Lekaviciute J, Argalasova-Sobotova L. Environmental noise and annoyance in adults: research in Central, Eastern and South-Eastern Europe and Newly Independent States. Noise Health. 2013;15(62):42-54.
11. Jakovljevic B, Paunovic K, Belojevic G. Road-traffic noise and factors influencing noise annoyance in an urban population. Environ Int. 2009;35(3):552-6.
12. Ising H, Rebentisch E, Poustka F, Curio I. Annoyance and health risk caused by military low-altitude flight noise. International archives of occupational and environmental health. 1990;62(5):357-63.
13. Guoqing D, Xiaoyi L, Xiang S, Zhengguang L, Qili L. Investigation of the relationship between aircraft noise and community annoyance in China. Noise Health. 2012;14(57):52-7.
14. Gille LA, Marquis-Favre C, Morel J. Testing of the European Union exposure-response relationships and annoyance equivalents model for annoyance due to transportation noises: The need of revised exposure-response relationships and annoyance equivalents model. Environ Int. 2016;94:83-94.
15. Fyhri A KR. Road traffic noise, sensitivity, annoyance and self-reported health--a structural equation model exercise. Environ Int. 2009;35(1:91-7.
16. Foraster M, Eze IC, Vienneau D, Brink M, Cajochen C, Caviezel S, et al. Long-term transportation noise annoyance is associated with subsequent lower levels of physical activity. Environ Int. 2016;91:341-9.

17. Evans GW, Hygge S, Bullinger M. Chronic noise and psychological stress. Psychological science. 1995;6(6):333-8.
18. Elmenhorst EM, Quehl J, Muller U, Basner M. Nocturnal air, road, and rail traffic noise and daytime cognitive performance and annoyance. The Journal of the Acoustical Society of America. 2014;135(1):213-22.
19. Beutel ME, Junger C, Klein EM, Wild P, Lackner K, Blettner M, et al. Noise Annoyance Is Associated with Depression and Anxiety in the General Population- The Contribution of Aircraft Noise. PLoS One. 2016;11(5):e0155357.
20. Bartels S, Marki F, Muller U. The influence of acoustical and non-acoustical factors on short-term annoyance due to aircraft noise in the field - The COSMA study. The Science of the total environment. 2015;538:834-43.
21. Babisch W, Pershagen G, Selander J, Houthuijs D, Breugelmans O, Cadum E, et al. Noise annoyance—A modifier of the association between noise level and cardiovascular health? Science of the total environment. 2013;452:50-7.
22. Babisch W, Houthuijs D, Pershagen G, Cadum E, Katsouyanni K, Velonakis M, et al. Annoyance due to aircraft noise has increased over the years--results of the HYENA study. Environ Int. 2009;35(8):1169-76.
23. Aparicio-Ramon DV, Morales Suarez-Varela MM, Garcia GA, Llopis Gonzale A, Ruano L, Sanchez AM, et al. Subjective annoyance caused by environmental noise. J Environ Pathol Toxicol Oncol 1993 Oct-Dec;12(4):237-43. 1993.
24. Alimohammadi I, Sandrock S, Gohari MR. The effects of low frequency noise on mental performance and annoyance. Environ Monit Assess. 2013;185(8):7043-51.
25. Abel SM. The extra-auditory effects of noise and annoyance: an overview of research. J Otolaryngol. 1990;19 Suppl 1:1-13.
26. Scheibe F, Haupt H, Ludwig C. Intensity-related changes in cochlear blood flow in the guinea pig during and following acoustic exposure. Eur Arch Otorhinolaryngol. 1993;250(5):281-5.
27. Scheibe F, Haupt H, Ludwig C. Intensity-dependent changes in oxygenation of cochlear perilymph during acoustic exposure. Hear Res. 1992;63(1-2):19-25.
28. Reinis S. Acute changes in animal inner ears due to simulated sonic booms. The Journal of the Acoustical Society of America. 1976;60(1):133-8.
29. Prieve BA, Yanz JL. Age-dependent changes in susceptibility to ototoxic hearing loss. Acta Otolaryngol (Stockh) 1984 Nov-Dec;98(5-6):428-38. 1984.
30. Ishida A, Matsui T, Yamamura K. The effects of low-frequency ultrasound on the inner ear: an electrophysiological study using the guinea pig cochlea. Eur Arch Otorhinolaryngol. 1993;250(1):22-6.
31. Huang EI, Durrant JD, Boston JR. Will diminishing cochlear delay affect speech perception in noise? Int J Audiol. 2015;54(8):562-7.
32. Henderson D, Hamernik RP, Sitler RW. Audiometric and histological correlates of exposure to 1-msec noise impulses in the chinchilla. The Journal of the Acoustical Society of America. 1974;56(4):1210-21.
33. Fechter Ld GCFSCJFJNKCDN-MAMPPB. Promotion of noise-induced cochlear injury by toluene and ethylbenzene in the rat. Toxicological sciences : an official journal of the Society of Toxicology. 2007;98(2):542-51.

34. Tiesler CM, Birk M, Thiering E, Kohlböck G, Koletzko S, Bauer C-P, et al. Exposure to road traffic noise and children's behavioural problems and sleep disturbance: Results from the GINIplus and LISAplus studies. Environmental research. 2013;123:1-8.
35. Stansfeld Sa MMP. Noise pollution: non-auditory effects on health. British Medical Bulletin. 2003;68:243-57.
36. Perron S, Tetreault LF, King N, Plante C, Smargiassi A. Review of the effect of aircraft noise on sleep disturbance in adults. Noise Health. 2012;14(57):58-67.
37. Muzet A. Environmental noise, sleep and health. Sleep Medicine Reviews. 2007;11:135-42.
38. Kwak KM, Ju YS, Kwon YJ, Chung YK, Kim BK, Kim H, et al. The effect of aircraft noise on sleep disturbance among the residents near a civilian airport: a cross-sectional study. Annals of occupational and environmental medicine. 2016;28(1):38.
39. Kim SJ, Chai SK, Lee KW, Park JB, Min KB, Kil HG, et al. Exposure-Response Relationship Between Aircraft Noise and Sleep Quality: A Community-based Cross-sectional Study. Osong public health and research perspectives. 2014;5(2):108-14.
40. Holt JB, Zhang X, Sizov N, Croft JB. Airport noise and self-reported sleep insufficiency, United States, 2008 and 2009. Prev Chronic Dis. 2015;12:E49.
41. Griefahn BaM, A. Noise-Induced Sleep Disturbances and their Effects on Health. Journal of Sound and Vibration. 1978;59(1):99-106.
42. Franssen EA, van Wiechen CM, Nagelkerke NJ, Lebret E. Aircraft noise around a large international airport and its impact on general health and medication use. Occupational and environmental medicine. 2004;61(5):405-13.
43. Floud S, Vigna-Taglianti F, Hansell A, Blangiardo M, Houthuijs D, Breugelmans O, et al. Medication use in relation to noise from aircraft and road traffic in six European countries: results of the HYENA study. Occupational and environmental medicine. 2011;68(7):518-24.
44. Fidell S, Tabachnick B, Mestre V, Fidell L. Aircraft noise-induced awakenings are more reasonably predicted from relative than from absolute sound exposure levels. The Journal of the Acoustical Society of America. 2013;134(5):3645-53.
45. Elmenhorst EM, Pennig S, Rolny V, Quehl J, Mueller U, Maass H, et al. Examining nocturnal railway noise and aircraft noise in the field: sleep, psychomotor performance, and annoyance. The Science of the total environment. 2012;424:48-56.
46. Dzhambov AM, Dimitrova DD, Mihaylova-Alakidi VK. Burden of Sleep Disturbance Due to Traffic Noise in Bulgaria. Folia Med (Plovdiv). 2015;57(3-4):264-9.
47. Di Nisi J, Muzet A, Ehrhart J, Libert JP. Comparison of cardiovascular responses to noise during waking and sleeping in humans. Sleep. 1990;13(2):108-20.
48. Carter NL. Transportation noise, sleep, and possible after-effects. Environ Int. 1996;22(1):105-16.
49. Basner M, Griefahn B, Berg M. Aircraft noise effects on sleep: mechanisms, mitigation and research needs. Noise Health. 2010;12(47):95-109.
50. Zhao YM, Zhang SZ, Selvin S, Spear RC. A dose response relation for noise induced hypertension. Br J Ind Med. 1991;48(3):179-84.
51. Yiming Z SZSSSRC. A dose response relation for noise induced hypertension. British Journal of Industrial Medicine. 1991;48:179-84.
52. Wu Tn KYCCPY. Study of noise exposure and high blood pressure in shipyard workers. American journal of industrial medicine. 1987;12:431-8.

53. van Kempen Emm KHBHCACBSBAMdHAEM. The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environ Health Perspectives. 2002;110(3):307-17.
54. Van Kempen E, Babisch W. The quantitative relationship between road traffic noise and hypertension: a meta-analysis. Journal of hypertension. 2012;30(6):1075-86.
55. Tomei F TEPBBTPAP. Study of some cardivascular parameters after chronic exposure to noise. International Journal of Cardiology. 1991;33:393-400.
56. Tomei F, De Sio S, Tomao E, Anzelmo V, Baccolo TP, Ciarrocca M, et al. Occupational exposure to noise and hypertension in pilots. Int J Environ Health Res. 2005;15(2):99-106.
57. Talbott EO, Gibson LB, Burks A, Engberg R, McHugh KP. Evidence for a dose-response relationship between occupational noise and blood pressure. Archives of environmental health. 1999;54(2):71-8.
58. Sorensen M. Aircraft noise exposure and hypertension. Occupational and environmental medicine. 2016.
59. Seidler A, Wagner M, Schubert M, Droge P, Romer K, Pons-Kuhnemann J, et al. Aircraft, road and railway traffic noise as risk factors for heart failure and hypertensive heart disease-A case-control study based on secondary data. Int J Hyg Environ Health. 2016;219(8):749-58.
60. Schulte W, Otten H. Results of a low-altitude flight noise study in Germany: long-term extraaural effects. Schriftenr Ver Wasser Boden Lufthyg. 1993;88:322-38.
61. Schmidt F, Kolle K, Kreuder K, Schnorbus B, Wild P, Hechtner M, et al. Nighttime aircraft noise impairs endothelial function and increases blood pressure in patients with or at high risk for coronary artery disease. Clin Res Cardiol. 2015;104(1):23-30.
62. Sbihi H DHWDPA. Hypertension in noise-exposed sawmill workers: a cohort study. Occupational and environmental medicine. 2008;65:643-6.
63. Rosenlund M, Berglind N, Pershagen G, Jarup L, Bluhm G. Increased prevalence of hypertension in a population exposed to aircraft noise. Occupational and environmental medicine. 2001;58(12):769-73.
64. Rhee MY, Kim HY, Roh SC, Kim HJ, Kwon HJ. The effects of chronic exposure to aircraft noise on the prevalence of hypertension. Hypertension research : official journal of the Japanese Society of Hypertension. 2008;31(4):641-7.
65. Paunovic K, Stansfeld S, Clark C, Belojevic G. Epidemiological studies on noise and blood pressure in children: Observations and suggestions. Environ Int. 2011;37(5):1030-41.
66. Pattenden S. Air traffic noise and hypertension in Stockholm County. Occupational and environmental medicine. 2001;58(12):761.
67. Ni Ch CZYZYZJWPJJLNWJLCKZZZZY. Associations of blood pressure and arterial compliance with occupational noise exposure in female workers of textile mill. Chinese Medical Journal. 2007;120(15):1309-13.
68. Neus H RHSW. Traffic noise and hypertension: an epidemiological study on the role of subjective reactions. International archives of occupational and environmental health. 1983;51:223-9.

69. Meline J, Van Hulst A, Thomas F, Chaix B. Road, rail, and air transportation noise in residential and workplace neighborhoods and blood pressure (RECORD Study). Noise Health. 2015;17(78):308-19.
70. Medoff HSBAM. Blood pressure in rats subjected to audiogenic stimulation. American Journal of Physiology. 1945;193:300-5.
71. McCann SMRABYEHSHA. Adrenalectomy and blood pressure of rats subjected to auditory stimulation. American Journal Physiology. 1948;155:128-31.
72. Matsui T, Uehara T, Miyakita T, Hiramatsu K, Yamamoto T. Dose-response relationship between hypertension and aircraft noise exposure around Kadena airfield in Okinawa. 2004.
73. Liu J, Xu M, Ding L, Zhang H, Pan L, Liu Q, et al. Prevalence of hypertension and noise-induced hearing loss in Chinese coal miners. Journal of Thoracic Disease. 2016;8(3):422-9.
74. Liu C, Fuertes E, Tiesler CM, Birk M, Babisch W, Bauer CP, et al. The associations between traffic-related air pollution and noise with blood pressure in children: results from the GINIplus and LISAplus studies. Int J Hyg Environ Health. 2014;217(4-5):499-505.
75. Leon Bluhm G BNNERM. Road traffic noise and hypertension. Occupational and environmental medicine. 2007;64(2):122-6.
76. Lee Jh KWYSRCNLCR. Cohort study for the effect of chronic noise exposure on blood pressure among male workers in Busan, Korea. American journal of industrial medicine. 2009.
77. Lang T, Fouriaud C, Jacquinet-Salord MC. Length of occupational noise exposure and blood pressure. Int Arch Occup Environ Health 1992;63(6):369-72. 1992.
78. Kavoussi N. The relationship between the length of exposure to noise and the incidence of hypertension at a silo in Terran. Med Lavoro. 1973;64(7-8):292-5.
79. Jonsson AHL. Prolonged exposure to a stressful stimulus (noise) as a cause of raised blood-pressure in man. The Lancet. 1977:86-7.
80. Jarup L, Dudley ML, Babisch W, Houthuijs D, Swart W, Pershagen G, et al. Hypertension and Exposure to Noise near Airports (HYENA): study design and noise exposure assessment. Environmental health perspectives. 2005;113(11):1473-8.
81. Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, et al. Hypertension and exposure to noise near airports: the HYENA study. Environmental health perspectives. 2008;116(3):329-33.
82. Hwang BF, Chang TY, Cheng KY, Liu CS. Gene-environment interaction between angiotensinogen and chronic exposure to occupational noise contribute to hypertension. Occupational and environmental medicine. 2012;69(4):236-42.
83. Huang D, Song X, Cui Q, Tian J, Wang Q, Yang K. Is there an association between aircraft noise exposure and the incidence of hypertension? A meta-analysis of 16784 participants. Noise Health. 2015;17(75):93-7.
84. Hessel Pa S-CGK. Occupational noise exposure and blood pressure: longitudinal and cross-sectional observations in a group of underground miners. Archives of environmental health. 1994;;49(2)::128-34.
85. Herbold M HHWKU. Effects of road traffic noise on prevalence of hypertension in men: results of the Luebeck Blood Pressure Study. Soz Praventivmed. 1989;;34(1)::19-23.

86. Haralabidis AS, Dimakopoulou K, Vigna-Taglianti F, Giampaolo M, Borgini A, Dudley ML, et al. Acute effects of night-time noise exposure on blood pressure in populations living near airports. Eur Heart J. 2008;29(5):658-64.
87. Evrard AS, Lefevre M, Champelovier P, Lambert J, Laumon B. Does aircraft noise exposure increase the risk of hypertension in the population living near airports in France? Occupational and environmental medicine. 2016.
88. Eriksson C, Rosenlund M, Pershagen G, Hilding A, Ostenson CG, Bluhm G. Aircraft noise and incidence of hypertension. Epidemiology (Cambridge, Mass). 2007;18(6):716-21.
89. Eriksson C, Bluhm G, Hilding A, Ostenson CG, Pershagen G. Aircraft noise and incidence of hypertension--gender specific effects. Environmental research. 2010;110(8):764-72.
90. de Kluizenaar Y, Gansevoort RT, Miedema HM, de Jong PE. Hypertension and road traffic noise exposure. Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine. 2007;49(5):484-92.
91. Charakida M, Deanfield JE. Nighttime aircraft noise exposure: flying towards arterial disease. Eur Heart J. 2013;34(45):3472-4.
92. Chang Ty STCLSYJRMCCC. Effects of occupational noise exposure on 24-hour ambulatory vascular properties in male workers. Environmental health perspectives. 2007;115(11):1660-4.
93. Chang TY, Liu CS, Huang KH, Chen RY, Lai JS, Bao BY. High-frequency hearing loss, occupational noise exposure and hypertension: a cross-sectional study in male workers. Environmental health : a global access science source. 2011;10:35.
94. Chang TY, Hwang BF, Liu CS, Chen RY, Wang VS, Bao BY, et al. Occupational noise exposure and incident hypertension in men: a prospective cohort study. American journal of epidemiology. 2013;177(8):818-25.
95. Turnovska T, Staykova J, Petkov T. Health assessment of populations living close to the airport of Bourgas, Bulgaria. Arhiv za Higijenu Rada I Toksikologiju/Archives of Industrial Hygiene and Toxicology. 2004;55(1):5-10.
96. Vienneau D, Perez L, Schindler C, Probst-Hensch N, Röösli M, editors. The relationship between traffic noise exposure and ischemic heart disease: a meta-analysis'. 42nd International Congress and Exposition on Noise Control Engineering; 2013.
97. Argalasova-Sobotova L, Lekaviciute J, Jeram S, Sevcikova L, Jurkovicova J. Environmental noise and cardiovascular disease in adults: research in Central, Eastern and South-Eastern Europe and Newly Independent States. Noise Health. 2013;15(62):22-31.
98. Babisch W. Stress hormones in the research on cardiovascular effects of noise. Noise Health. 2003;5(18)::1-11.
99. Babisch W. Transportation noise and cardiovascular risk: updated review and synthesis of epidemiological studies indicate that the evidence has increased. Noise Health. 2006;8(30)::1-29.
100. Babisch W. rOAD TRAFFIC NOISE AND CARDIOVASCULAR RISK. Noise & Health. 2008;10(38):27-33.
101. Babisch W. Updated exposure-response relationship between road traffic noise and coronary heart diseases: a meta-analysis. Noise Health. 2014;16(68):1-9.

102. Babisch W BBSMKNIH. Traffic Noise and Risk of Myocardial Infarction. Epidemiology (Cambridge, Mass). 2005;16(1):33-40.
103. Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, et al. Auditory and non-auditory effects of noise on health. Lancet. 2014;383(9925):1325-32.
104. Bluhm G, Eriksson C. Cardiovascular effects of environmental noise: research in Sweden. Noise Health. 2011;13(52):212-6.
105. Brenner H, Oberacker A, Kranig W, Buchwalsky R. A field study on the immediate effects of exposure to low-altitude flights on heart rate and arrhythmia in patients with cardiac diseases. International archives of occupational and environmental health. 1993;65(4):263-8.
106. Brunekreef B, Beelen R, Hoek G, Schouten L, Bausch-Goldbohm S, Fischer P, et al. Effects of long-term exposure to traffic-related air pollution on respiratory and cardiovascular mortality in the Netherlands: the NLCS-AIR study. Research report (Health Effects Institute). 2009(139):5-71; discussion 3-89.
107. Davies Hw TKKSMHMRHCDPA. Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology (Cambridge, Mass). 2005;16(1):25-32.
108. Evrard AS, Bouaoun L, Champelovier P, Lambert J, Laumon B. Does exposure to aircraft noise increase the mortality from cardiovascular disease in the population living in the vicinity of airports? Results of an ecological study in France. Noise Health. 2015;17(78):328-36.
109. Fruhstorfer B HH. Extra-auditory responses to long-term intermittent noise stimulation in humans. J Appl Physiol. 1980;49(6:985-93.
110. Hallback M, Jones JV, Bianchi G, Folkow B. Cardiovascular control in the Milan strain of spontaneously hypertensive rat (MHS) at "rest" and during acute mental "stress". Acta Physiol Scand. 1977;99(2):208-16.
111. Hansell AL, Blangiardo M, Fortunato L, Floud S, de Hoogh K, Fecht D, et al. Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. BMJ. 2013;347:f5432.
112. Harlan WR, Sharrett AR, Weill H, Turino GM, Borhani NO, Resnekov L. Impact of the environment on cardiovascular disease. Report of the American Heart Association Task Force on environment and the cardiovascular system. Circulation 1981 Jan;63(1):243A-246A. 1981.
113. Heinonen-Guzejev M VHSM-RHHKKMKJ. The association of noise sensitivity with coronary heart and cardiovascular mortality among Finnish adults. The Science of the total environment. 2007;372(2-3):406-12.
114. Ising H, Rebentisch E, Babisch W, Curio I, Sharp D, Baumgärtner H. Medically relevant effects of noise from military low-altitude flights—results of an interdisciplinary pilot study. Environ Int. 1990;16(4):411-23.
115. Kaltenbach M, Maschke C, Klinke R. Health consequences of aircraft noise. Deutsches Arzteblatt international. 2008;105(31-32):548-56.
116. Kmietowicz Z. Aircraft noise is linked to raised risk of cardiovascular disease. BMJ. 2013;347:f6082.
117. McNamee R BGDWMCN. Occupational noise exposure and ischaemic heart disease mortality. Occupational and environmental medicine. 2006;63:813-9.

118. Michalak R, Ising H, Rebentisch E. Acute circulatory effects of military low-altitude flight noise. International archives of occupational and environmental health. 1990;62(5):365-72.
119. Munzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. Eur Heart J. 2014;35(13):829-36.
120. Pearson T, Campbell MJ, Maheswaran R. Acute effects of aircraft noise on cardiovascular admissions - an interrupted time-series analysis of a six-day closure of London Heathrow Airport caused by volcanic ash. Spatial and spatio-temporal epidemiology. 2016;18:38-43.
121. Peters A, von Klot S, Heier M, Trentinaglia I, Hormann A, Wichmann HE, et al. Exposure to traffic and the onset of myocardial infarction. The New England journal of medicine. 2004;351(17):1721-30.
122. Ray Rl BJVEHH. Cardiovascular effects of noise during complex task performance. International Journal of Psychophysiology. 1984;1:335-40.
123. Schmidt FP, Basner M, Kroger G, Weck S, Schnorbus B, Muttray A, et al. Effect of nighttime aircraft noise exposure on endothelial function and stress hormone release in healthy adults. Eur Heart J. 2013;34(45):3508-14a.
124. Schneider A HRI-MAZWSGSRRRCJPMBOGWGPM. Changes in deceleration capacity of heart rate and heart rate variability induced by ambient air pollution in individuals with coronary artery disease. Part Fibre Toxicol. 2010;7:29.
125. Schwartz J, Litonjua A, Suh H, Verrier M, Zanobetti A, Syring M, et al. Traffic related pollution and heart rate variability in a panel of elderly subjects. Thorax. 2005;60(6):455-61.
126. Seidler A, Wagner M, Schubert M, Droge P, Pons-Kuhnemann J, Swart E, et al. Myocardial Infarction Risk Due to Aircraft, Road, and Rail Traffic Noise. Deutsches Arzteblatt international. 2016;113(24):407-14.
127. Singh Ap RRMBMRNHS. Effect of chronic and acute exposure to noise on phsiological functions in man. International archives of occupational and environmental health. 1982;50:169-74.
128. Stansfeld S. Airport noise and cardiovascular disease. BMJ. 2013;347:f5752.
129. Stansfeld S, Crombie R. Cardiovascular effects of environmental noise: research in the United Kingdom. Noise Health. 2011;13(52):229-33.
130. Taylor J. Noise: a new cardiovascular risk factor. Eur Heart J. 2014;35(13):821-2.
131. Tomei F, Tomao E, Papaleo B, Baccolo TP, Alfi P. Study of some cardiovascular parameters after chronic exposure to noise. Int J Cardiol. 1991;33(3):393-9.
132. Valenti VE, Guida HL, Frizzo AC, Cardoso AC, Vanderlei LC, Abreu LC. Auditory stimulation and cardiac autonomic regulation. Clinics (Sao Paulo). 2012;67(8):955-8.
133. Vera MN, Vila J, Godoy JF. Cardiovascular effects of traffic noise: the role of negative self-statements. Psychol Med. 1994;24(4):817-27.
134. Vienneau D, Perez L, Schindler C, Lieb C, Sommer H, Probst-Hensch N, et al. Years of life lost and morbidity cases attributable to transportation noise and air pollution: A comparative health risk assessment for Switzerland in 2010. Int J Hyg Environ Health. 2015;218(6):514-21.
135. W. B. Traffic noise and cardiovascular disease : Epidemiological review and synthesis. Noise & Health. 2000;2(8):9-32.

136. WHO. Burden of disease from environmental noise Quantification of healthy life years lost in Europe. Monographs WHO. 2011:1-126.
137. Hardoy MC, Carta MG, Marci AR, Carbone F, Cadeddu M, Kovess V, et al. Exposure to aircraft noise and risk of psychiatric disorders: the Elmas survey--aircraft noise and psychiatric disorders. Soc Psychiatry Psychiatr Epidemiol. 2005;40(1):24-6.
138. Haines MM, Stansfeld SA, Job RF, Berglund B, Head J. A follow-up study of effects of chronic aircraft noise exposure on child stress responses and cognition. International journal of epidemiology. 2001;30(4):839-45.
139. Dreger S, Meyer N, Fromme H, Bolte G, Study Group of the GMEc. Environmental noise and incident mental health problems: A prospective cohort study among school children in Germany. Environmental research. 2015;143(Pt A):49-54.
140. Chiovenda P, Pasqualetti P, Zappasodi F, Ercolani M, Milazzo D, Tomei G, et al. Environmental noise-exposed workers: event-related potentials, neuropsychological and mood assessment. Int J Psychophysiol. 2007;65(3):228-37.
141. Tanyel M, Lee KY, Chey WY, Chitrapu PR. Multistage enhancement of surface recordings of canine gastric electrical signals. Ann Biomed Eng. 1993;21(4):337-50.
142. Moslehi A, Nabavizadeh-Rafsanjani F, Keshavarz M, Rouhbakhsh N, Sotudeh M, Salimi E. Traffic noise exposure increases gastric acid secretion in rat. Acta medica Iranica. 2010;48(2):77-82.
143. Karmody CS, Valdez TA, Desai U, Blevins NH. Sensorineural hearing loss in patients with inflammatory bowel disease. Am J Otolaryngol. 2009;30(3):166-70.
144. Gue M, Fioramonti J, Frexinos J, Alvinerie M, Bueno L. Influence of acoustic stress by noise on gastrointestinal motility in dogs. Dig Dis Sci. 1987;32(12):1411-7.
145. Geerse GJ, van Gurp LC, Wiegant VM, Stam R. Individual reactivity to the open-field predicts the expression of cardiovascular and behavioural sensitisation to novel stress. Behav Brain Res. 2006;175(1):9-17.
146. Fonseca J, Martins-dos-Santos J, Oliveira P, Laranjeira N, Aguas A, Castelo-Branco N. Noise-induced gastric lesions: a light and electron microscopy study of the rat gastric wall exposed to low frequency noise. Arquivos de gastroenterologia. 2012;49(1):82-8.
147. Castle JS, Xing JH, Warner MR, Korsten MA. Environmental noise alters gastric myoelectrical activity: Effect of age. World J Gastroenterol. 2007;13(3):403-7.
148. Becker V, von Delius S, Bajbouj M, Karagianni A, Schmid RM, Meining A. Intravenous application of fluorescein for confocal laser scanning microscopy: evaluation of contrast dynamics and image quality with increasing injection-to-imaging time. Gastrointest Endosc. 2008;68(2):319-23.
149. Trimmel M, Atzlsdorfer J, Tupy N, Trimmel K. Effects of low intensity noise from aircraft or from neighbourhood on cognitive learning and electrophysiological stress responses. Int J Hyg Environ Health. 2012;215(6):547-54.
150. Chengzhi C, Yan T, Xuejun J, Xiang L, Youbin Q, Baijie T. Recovery of chronic noise exposure induced spatial learning and memory deficits in young male Sprague-Dawley rats. J Occup Health. 2011;53(3):157-63.
151. Clark C, Martin R, van Kempen E, Alfred T, Head J, Davies HW, et al. Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: the RANCH project. American journal of epidemiology. 2006;163(1):27-37.

152. Crombie R, Clark C, Stansfeld SA. Environmental noise exposure, early biological risk and mental health in nine to ten year old children: a cross-sectional field study. Environmental health : a global access science source. 2011;10:39.
153. Cui B, Wu M, She X. Effects of chronic noise exposure on spatial learning and memory of rats in relation to neurotransmitters and NMDAR2B alteration in the hippocampus. J Occup Health. 2009;51(2):152-8.
154. Klatte M, Bergstrom K, Lachmann T. Does noise affect learning? A short review on noise effects on cognitive performance in children. Front Psychol. 2013;4:578.
155. Kraus KS, Canlon B. Neuronal connectivity and interactions between the auditory and limbic systems. Effects of noise and tinnitus. Hear Res. 2012;288(1-2):34-46.
156. Krysa I. The effect of noise on learning and retention. Act Nerv Super (Praha). 1983;25(4):299-303.
157. Prior H. Effects of the acoustic environment on learning in rats. Physiol Behav. 2006;87(1):162-5.
158. Pujol S, Levain JP, Houot H, Petit R, Berthillier M, Defrance J, et al. Association between ambient noise exposure and school performance of children living in an urban area: a cross-sectional population-based study. Journal of urban health : bulletin of the New York Academy of Medicine. 2014;91(2):256-71.
159. Seabi J. An epidemiological prospective study of children's health and annoyance reactions to aircraft noise exposure in South Africa. Int J Environ Res Public Health. 2013;10(7):2760-77.
160. Seabi J, Cockcroft K, Goldschagg P, Greyling M. The impact of aircraft noise exposure on South African children's reading comprehension: the moderating effect of home language. Noise Health. 2012;14(60):244-52.
161. Stansfeld S, Clark C. Health Effects of Noise Exposure in Children. Current environmental health reports. 2015;2(2):171-8.
162. Haralabidis AS, Dimakopoulou K, Vigna-Taglianti F, Giampaolo M, Borgini A, Dudley M-L, et al. Acute effects of night-time noise exposure on blood pressure in populations living near airports. European heart journal. 2008;29(5):658-64.
163. Vienneau D, Schindler C, Perez L, Probst-Hensch N, Röösli M. The relationship between transportation noise exposure and ischemic heart disease: a meta-analysis. Environmental research. 2015;138:372-80.
164. Morrell MJ, Finn L, Kim H, Peppard PE, Badr MS, Young T. Sleep fragmentation, awake blood pressure, and sleep-disordered breathing in a population-based study. American journal of respiratory and critical care medicine. 2000;162(6):2091-6.
165. Lepore GEaS. Nonauditory Effects of Noise on Children: A Critical Review. Children's Environments. 1993;10(1):31-51.
166. Cohen S, Evans GW, Krantz DS, Stokols D. Physiological, motivational, and cognitive effects of aircraft noise on children: moving from the laboratory to the field. Am Psychol. 1980;35(3):231-43.
167. Cohen S, Evans GW, Krantz DS, Stokols D, Kelly S. Aircraft Noise and Children: Longitudal and Cross-Sectional Evidence on Adaptation to Noise and the Effectiveness of Noise Abatement. Journal of Personality and Social Psychology. 1981;40(2).

168. Evans GW, Lercher P, Meis M, Ising H, Kofler WW. Community noise exposure and stress in children. The Journal of the Acoustical Society of America. 2001;109(3):1023-7.
169. Regecova V, Kellerova E. Effects of urban noise pollution on blood pressure and heart rate in preschool children. J Hypertens. 1995;13(4):405-12.
170. Evans GW, Bullinger, M., and Hygge, S. Chronic noise and physiological response: A prospective, longitudinal study. Psychological science. 1998a;9:75-7.
171. Emmett EA, Shofer FS, Zhang H, Freeman D, Desai C, Shaw LM. Community exposure to perfluorooctanoate: relationships between serum concentrations and exposure sources. Journal of occupational and environmental medicine/American College of Occupational and Environmental Medicine. 2006;48(8):759.
172. Zhang Y, Beesoon S, Zhu L, Martin JW. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. Environmental science & technology. 2013;47(18):10619-27.
173. DeWitt JC. Toxicological effects of perfluoroalkyl and polyfluoroalkyl substances: Springer; 2015.
174. EPA U. Protective noise levels. 1978;550/9-79-100:1-28.
175. YAMANAKA K W-N, f. KOBAYASHI, S. KANADA, M. TANAHASHI, T. MURAMATSU AND S. YAMADA. CRITERIA FOR ACCEPTABLE LEVELS OF THE SHINKANSEN SUPER EXPRESS TRAIN NOISE AND VIBRATION IN RESIDENTIAL AREAS. Journal of Sottnd and Vibration. 1982;84(4):573-91.
176. Lee EY, Jerrett M, Ross Z, Coogan PF, Seto EY. Assessment of traffic-related noise in three cities in the United States. Environmental research. 2014;132C:182-9.
177. Babisch W, Wolf K, Petz M, Heinrich J, Cyrys J, Peters A. Associations between Traffic Noise, Particulate Air Pollution, Hypertension, and Isolated Systolic Hypertension in Adults: The KORA Study. Environmental health perspectives. 2014;122(5):492-8.
178. Babisch W, Swart W, Houthuijs D, Selander J, Bluhm G, Pershagen G, et al. Exposure modifiers of the relationships of transportation noise with high blood pressure and noise annoyance. The Journal of the Acoustical Society of America. 2012;132(6):3788-808.
179. Babisch W, Kamp I. Exposure-response relationship of the association between aircraft noise and the risk of hypertension. Noise Health. 2009;11(44):161-8.
180. Eriksson C RMPGHAOCGBG. Aircraft noise and incidence of hypertension. Epidemiology (Cambridge, Mass). 2005;18(6)::716-21.
181. Rosenlund M BNPGJLBG. Increased prevalence of hypertension in population exposed to aircraft noise. Occupational and environmental medicine. 2001;58:769-73.
182. Zhao Y, Zhang S, Selvin S, Spear RC. A dose-response relationship for occupational noise-induced hypertension. Schriftenr Ver Wasser Boden Lufthyg. 1993;88:189-207.
183. Johsson A, Hansson L. Prolonged exposure to a stressful stimulus (noise) as a cause of raised blood-pressure in man. Lancet. 1977;1(8002):86-7.
184. Ettema Jh ZRL. Health effects of exposure to noise, particularly aircraft noise. International Archives of Occupational Environmental Health. 1977;40:163-84.
185. Belojevic G S-TM. Prevalence of Arterial Hypertension and Myocardial Infarction in Relation to Subjective Ratings of Traffic Noise Exposure. Noise Health. 2002;4(16)::33-7.

186. Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C, Demers PA. Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology (Cambridge, Mass). 2005;16(1):25-32.
187. Jarup L BWHDPGKKCEDMLSPSISWBOBG. Hypertension and exposure to noise near airports: the HYENA study. Environmental health perspectives. 2008;116(3):329-33.
188. Passchier-Vermeer W PWF. Noise exposure and public health. Environ Health Perspectives. 2000;108(1):123-31.
189. Clark C, Crombie R, Head J, van Kamp I, van Kempen E, Stansfeld SA. Does traffic-related air pollution explain associations of aircraft and road traffic noise exposure on children's health and cognition? A secondary analysis of the United Kingdom sample from the RANCH project. American journal of epidemiology. 2012;176(4):327-37.
190. Hygge S. Classroom experiments on the effects of different noise sources and sound levels on long-term recall and recognition in children. Applied Cognitive Psychology. 2003;17(8):895-914.
191. Banbury SP, Macken WJ, Tremblay S, Jones DM. Auditory distraction and short-term memory: phenomena and practical implications. Hum Factors. 2001;43(1):12-29.
192. Da Fonseca J, Dos Santos JM, Branco NC, Alves-Pereira M, Grande N, Oliveira P, et al. Noise-induced gastric lesions: a light and scanning electron microscopy study of the alterations of the rat gastric mucosa induced by low frequency noise. Central European journal of public health. 2006;14(1).
193. Kim CY, Ryu JS, Hong SS. Effect of air-craft noise on gastric function. Yonsei medical journal. 1968;9(2):149-54.
194. Akbayir N, Calis AB, Alkim C, Sokmen HM, Erdem L, Ozbal A, et al. Sensorineural hearing loss in patients with inflammatory bowel disease: a subclinical extraintestinal manifestation. Dig Dis Sci. 2005;50(10):1938-45.
195. Kalyoncu D, Urganci N, Calis AB, Ozbal A. Sensorineural hearing loss in pediatric patients with inflammatory bowel disease. Dig Dis Sci. 2010;55(1):150-2.
196. Summers RW, Harker L. Ulcerative colitis and sensorineural hearing loss: is there a relationship? J Clin Gastroenterol. 1982;4(3):251-2.
197. Cohen S, Krantz DS, Evans GW, Stokols D. Cardiovascular and Behavioral Effects of Community Noise: Evidence from field studies of schoolchildren supports laboratory findings that high-intensity noise adversely affects physical health and psychological functioning. American Scientist. 1981:528-35.
198. Kasicka-Jonderko A, Jonderko K, Dolinski K, Dolinski M, Kaminska M, Szymszal M, et al. Extracirculatory effects of noise of various frequency spectra in humans--effect of pink and blue noise on gastric myoelectrical activity and gastrointestinal passage of nutrients. Journal of smooth muscle research = Nihon Heikatsukin Gakkai kikanshi. 2007;43(1):25-42.
199. da Fonseca J, dos Santos JM, Branco NC, Alves-Pereira M, Grande N, Oliveira P, et al. Noise-induced gastric lesions: a light and scanning electron microscopy study of the alterations of the rat gastric mucosa induced by low frequency noise. Cent Eur J Public Health. 2006;14(1):35-8.
200. Mintchev MP, Girard A, Bowes KL. Nonlinear adaptive noise compensation in electrogastrograms recorded from healthy dogs. IEEE Trans Biomed Eng. 2000;47(2):239-48.

201. Lercher P, Schmitzberger R, Kofler W. Perceived traffic air pollution, associated behavior and health in an alpine area. The Science of the total environment. 1995;169(1-3):71-4.
202. Bergmann JF, Caulin C, Geneve J, Simoneau G, Segrestaa JM. Effect of psychological stress on gastric potential difference in man. Agressologie: revue internationale de physio-biologie et de pharmacologie appliquees aux effets de l'agression. 1991;32(2):127-9.
203. Yamanouchi I, Fukuhara H, Shimura Y. The transmission of ambient noise and self-generated sound in the human body. Acta paediatrica Japonica; Overseas edition. 1990;32(6):615-24.
204. Mashchenko NP, Lipkan GN, Voitenko GN. [Use of plant and vitamin preparations to prevent stomach ulcers induced by immobilization, noise and vibration in rats]. Fiziologicheskii zhurnal. 1982;28(1):103-6.
205. Hill AB. THE ENVIRONMENT AND DISEASE: ASSOCIATION OR CAUSATION? Proceedings of the Royal Society of Medicine. 1965;58:295-300.