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Aviation Noise and Cardiovascular Health in the United States: a Review of the Evidence and Recommendations for Research Direction

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Abstract

Purpose of the Review—In the USA, there is mounting pressure on aviation operators and regulators to address concerns about community impacts of aircraft noise given increasing evidence of adverse health impacts, continuing community complaints, availability of cost-

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Compliance with Ethical Standards
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effective programs to reduce exposures to aircraft noise, and more stringent international policies. In the USA, regulation of civil aviation noise is the responsibility of the Federal Aviation Administration (FAA), which requires a "significant body of scientific support," particularly applicable to the USA, to inform health-based policy and regulatory decisions. However, there have been very few studies investigating the relationship between noise and health in the USA and limited studies across the globe characterizing the effects of aviation noise specifically on cardiovascular health. This review focuses on recent findings on the relationship between aircraft noise and cardiovascular outcomes and directions for future research.

Recent Findings—Epidemiological studies generally report statistically significant associations between aircraft noise and adverse cardiovascular outcomes, although with limited evidence within the USA. Sleep disturbance, associated with nighttime noise, has been shown to be a risk factor for cardiovascular disease given associations with inflammatory markers and metabolic changes. Given numerous cardiovascular markers, the most appropriate choices depend on the ultimate objectives of the individual studies.

Summary—Given the state of the literature, future research should leverage emerging tools to estimate aviation, railway, and road traffic noise and apply noise estimates to a range of epidemiological study designs and endpoints to inform causal interpretation and help determine potential intervention strategies.

Keywords

Cardiovascular disease; Cardiovascular health; Aircraft noise; Aviation noise; Transportation noise

Background and Context

The aviation industry in the USA continues to experience significant growth. In 2016, there were 19,536 airports (including 5136 public-use airports) servicing more than 932 million passengers in the USA and its territories [1, 2]. Growth in aviation has many economic and social benefits, but can also have negative impacts on environmental noise, air quality, and water quality in nearby communities, as well as broader impacts on energy usage and climate change. The Federal Aviation Administration (FAA) has been addressing these challenges through aircraft technology, alternative jet fuels, operational procedures, and policy measures [3]. The FAA's stated vision is "environmental protection that allows sustained aviation growth" with the guiding principles of limiting and reducing aviation impacts to levels that protect public health and welfare while ensuring energy availability and sustainability [3].

Historically, communities near airports have been primarily concerned with aircraft noise, particularly in metropolitan areas where there is high demand for aviation along with strong pressure to develop land near airports for residential and other incompatible uses [4]. The number of people exposed to significant aircraft noise—as federally defined by a Day-Night Average Sound Level (DNL) greater than 65 dB [4]—has decreased substantially over the past three decades primarily due to the transition to quieter aircraft and changes in operations. However, there are still community concerns about aircraft noise even from areas exposed to noise levels below DNL 65 dB [5].

Laws regarding aircraft noise date back to 1968 with the *Aircraft Noise Abatement Act* 49 U.S.C. 44715(1968). This act required FAA to establish noise standards in consultation with the Environmental Protection Agency (EPA) and to link the noise standards to aircraft certification. The regulation was updated in 1979 with the *Aviation Safety and Noise Abatement Act* and required FAA to publish a regulation on noise control and compatibility planning for airports. In 1990, Congress passed the *Airport Noise and Capacity Act* (*ANCA*). It mandated that the US air fleet convert to aircraft that met new noise certification standards that incorporated the most up-to-date safe and suitable noise reduction technologies into aircraft design leading to a reduction in environmental noise (14 CFR Part 36) [6].

The current definition of significant noise exposure at DNL greater than 65 dB is based on residential annoyance (FAA Order 1050.1F). However, there is now interest in examining not only annoyance, but also the potential for adverse health effects of noise. To date, there have been very few studies investigating the relationship between noise and health in the USA, and limited studies across the globe characterizing the effects of aviation noise specifically on cardiovascular health [7, 8, 9•, 10••, 11••, 12•]. Thus, there is an unmet need and opportunity to expand and strengthen the evidence base on aviation noise such that it is available for future decision-making.

Accordingly, on September 11, 2017, the US Department of Transportation's Volpe National Transportation Systems Center (Volpe) convened a workshop to review relevant topics and the most recent literature, discuss key questions, and determine next steps for research efforts on aviation noise and health in the USA. We briefly present our review of the literature on aircraft noise and cardiovascular outcomes over the last 5 years and summarize presentations on noise and health, noise and sleep, noise exposure metrics, and relevant cardiovascular endpoints. Finally, we outline conceptual research questions and recommendations for future noise-health research.

Noise Metrics

For the purposes of this review, noise is characterized as any unwanted sound and sound is defined as repetitive variations in air pressure (vibrations) that can be heard by the ear. Since any sound from aircraft is typically considered unwanted by residents, aircraft noise and sound can be used synonymously.

There are two types of metrics that can be derived from noise time histories. These can be referred to as psychoacoustic-based metrics and sound pressure level (SPL)-based metrics. Psychoacoustic-based metrics include loudness and metrics derived from it such as sharpness, fluctuation strength, roughness, and pitch strength [13]. These are used mainly to understand how sound is perceived or what drives particular responses to a sound. These metrics are typically used for basic research and not for regulations; consequently, studies relating these metrics to human responses are usually limited in scope. SPL-based metrics typically modify the noise levels by assigning weightings for different frequencies and duration. For example, the A-weighted SPL adjusts levels based on the frequency sensitivity of the human ear [14]. DNL is taken as a marker of a person's cumulative exposure over a

24-h period, expressed as the noise level for the average day of the year based on annual aircraft operations. DNL increases the A-weighted SPL by 10 dB for the hours between 10 PM and 7 AM to account for greater noise sensitivity and lower ambient noise levels during the night hours, before averaging these with the SPL for the rest of the day.

Although noise metrics can be measured directly using a sound level meter, typical human studies focus on hundreds or thousands of participants for exposure periods lasting months or years. In these cases, direct measurement is impractical and noise modeling is performed instead. The FAA's Aviation Environmental Design Tool (AEDT) [https://aedt.faa.gov/] provides a mature, validated method to compute noise exposure levels over a wide geographical area. Using flight tracks and aircraft operations data for a specific geographical location, AEDT produces noise exposure contours for a chosen metric, e.g., DNL.

There are several caveats to be aware of when relating DNL to community perception or potential adverse health effects. First, DNL does not differentiate noise sources with the same SPL, but dissimilar characteristics. For example, a tone (a sound evoking a strong sense of pitch) is typically considered more annoying than broadband noise, modulating sounds more annoying than steady state sounds, and sharp sounds more annoying than sounds with content across a broad spectrum. Thus, exposure summarized by DNL would give the same value for shrill, modulating, and tonal sources as for steady sources, well-balanced across the spectrum and lacking strong tones, provided that the aggregate A-weighted mean pressure was the same for both sources. Second, although DNL weighs more heavily noise exposure during nighttime hours, it still combines daytime and nighttime exposures. Thus, a study might fail to detect an association between residential DNL and sleep disturbances even if nighttime noise is indeed truly related to sleep health. This is an example of exposure misclassification of the etiologically relevant noise exposure. Other noise metrics that are potentially more suitable to specific settings are available (e.g., time above a certain threshold (TA) and sound equivalent level for night (LAeq Night)), but these are not currently considered in the regulatory setting.

Research on Aircraft Noise and Cardiovascular Health

Conceptual Models of Noise-Health Relationships

There are several models depicting the mechanisms by which noise may adversely affect health, including models illustrated by Swift [15], Munzel et al. [8, 16], and Babisch et al. [17]. As an example, Babisch et al. [17] distinguishes between "direct" and "indirect" pathways by which noise from any source may affect health. The "direct" pathway is characterized by activation of the central nervous system by the acoustic nerve and the "indirect" pathway is related to emotional and cognitive perception of sound leading to cortical activation and release of stress hormones [17, 18]. Activation of both pathways can affect autonomic nervous system control and endocrine systems resulting in dysregulation and eventual long-term health impacts [17]. This may explain why noise level and noise annoyance both relate to cardiovascular outcomes [17].

Review of the Literature on Aircraft Noise and Cardiovascular Outcomes from 2013 to 2017

As an input to the workshop, we performed a review of the literature published within the last 5 years (2013–2017) relating to the impact of environmental (community) aircraft noise exposures on markers of cardiovascular health. The literature review included searches of the PubMed®, EMBASE, Web of Science, EBSCO host, and the National Transportation Library databases with publication dates 2013–2017 and using the following search terms: aircraft noise + cardiovascular disease, heart disease, coronary heart disease, ischemic heart disease, hypertension, blood pressure, myocardial infarction, heart attack, stroke. The database searches were supplemented by general Google® searches as well as a review of the references cited in the relevant journal articles identified through the database searches. Our review included original journal articles published in the English language and excluded published letters and editorials that reviewed or critiqued a single research article.

The recent literature includes 17 articles [9•, 10••, 11••, 12•, 17, 19••, 20, 21••, 22–30], all but one of which were based in Europe (Table 1). In general, statistically significant associations were reported between aircraft noise exposure and a range of adverse cardiovascular outcomes, including the following: rates or risk of hospitalization or mortality from cardiovascular disease, coronary heart disease (CHD), myocardial infarction (MI), stroke, and heart failure [11••, 12•, 19••, 21••, 22–24]; higher blood pressure; higher rates or risk of hypertension and hypertensive heart disease; and vascular dysfunction [9•, 10••, 11••, 17, 20, 25, 27, 28]. Adverse cardiovascular outcomes were most often associated with nighttime aircraft noise exposure [10••, 11••, 19••, 20, 23, 27, 28] and more strongly associated in subgroups who were more highly exposed (noise level and duration of exposure) [9•, 11••, 21••, 23] or with risk factors for adverse cardiovascular outcomes [9•, 21••, 28, 31]. A minority of studies reported finding no association between aircraft noise and hypertension [9•] or no change in cardiovascular-related hospital admissions with an airport closure due to volcanic cloud [29].

Specific to the framework of the workshop, only one of the studies over the past 5 years was performed in the USA [22] [http://partner.mit.edu/]. This study included over six million individuals eligible to participate in the US national medical insurance (Medicare) program (≥ age 65 years) in 2009 residing in one of 2218 ZIP codes surrounding 89 airports. The study found 3.5% (95% confidence interval [CI] 0.15% to 7. 0%) higher rates of cardiovascular hospital admission per 10 dB higher DNL after controlling for potential confounders. Using these results, the authors estimated that 2.3% of all CVD hospitalizations in the study population were attributable to aircraft noise, while 6.8 and 4.2% were attributable to fine particulate matter and ozone, respectively. There are several limitations to this study including exposure assessment based on ZIP code of residence, limited data on individual-level risk factors (e.g., smoking, diet), lack of consideration for other potential sources of noise, and outcomes identified from administrative claims without further adjudication [22].

Many more recent studies have considered multiple sources of transportation noise and have found that the association between cardiovascular outcomes and road and railway noise are similar and often greater than for aircraft noise exposure [9•, 10••, 11••, 17, 19••, 21••, 23,

25]. These findings underscore the need for future studies to consider both the independent and joint effects of noise from multiple modes of transportation.

Noise, Sleep, and Cardiovascular Outcomes

Existing research suggests that (1) nighttime noise may disrupt sleep and (2) that disturbed sleep is a risk factor for cardiovascular disease. Thus, sleep may be an important causal intermediate between aviation noise exposure and increased cardiovascular risk. We next discuss each of these links in detail.

Sleep is a neurophysiological state when sympathetic tone normally decreases and parasympathetic tone increases, resulting in lowered blood pressure and heart rate. Sleep disturbances, however, can alter this normally cardio-protective effect.

Studies have investigated the relationship between environmental noise, and specifically aviation noise, and sleep disturbance [7]. Exposure to abrupt increases in noise may cause sudden shifts in sleep from deeper to lighter stages, or even to awakening, a phenomenon known as a cortical arousal. Noise may also contribute to short sleep duration, fragmented sleep, reduced slow-wave sleep (SWS), and variable sleep patterns [32, 33]. Chronic exposure to noise can also lead to psychological stress and insomnia, a disorder of conditioned hyperarousal and a risk factor for cardiovascular disease and mortality [34].

These sleep disturbances, individually or together, may impact a number of cardio-metabolic pathways that adversely affect cardiovascular health. Schmidt et al. [28] provides an illustration of the metabolic burden of sleep loss and Tobaldini [35] outlines some of the possible mechanisms. Arousals during sleep, which usually are accompanied by surges in sympathetic activation and acute blood pressure rises, have been identified as a strong predictor of daytime hypertension [36]. Sleep disturbances also are associated with markers of inflammation and metabolic changes such as higher cortisol levels or altered glucose tolerance [37–39], abnormal cortisol rhythms [40], abnormal heart rate variability [41], elevations in markers of systemic inflammation (e.g., IL1, IL6, TNFα, CRP) [42–44], activation of the immune system [45], abnormalities in appetite regulatory hormones such as lower leptin (appetite suppressant) and higher ghrelin (appetite stimulant) [46, 47], higher energy consumption (ingestion of calorie dense foods) [48], and visceral obesity [49].

These physiologic changes plausibly underlie the observed associations between chronic sleep disturbances and risk of cardiovascular disease. Sleep disturbance is associated with incident hypertension [50], incident diabetes [51], and atrial fibrillation [52]. A meta-analysis of over 153 studies, including over 5 million individuals, reported that curtailed sleep duration was associated with relative risks for mortality (1.12; 95% CI 1.08, 1.16), diabetes (1.37; 1.22, 1.53), hypertension (1.17; 1.09, 1.26), cardiovascular disease (1.16; 1.10, 1.23); coronary heart disease (1.26; 1.15, 1.38), and obesity (1.38; 1.25, 1.53) [53]. Adverse cardiovascular effects of sleep disturbances are also observed in children, with three cohort studies of children and adolescents demonstrating that low sleep efficiency is associated with a significant increase in elevated blood pressure, even after adjusting for other factors [54–56].

Additional considerations when studying sleep disturbance as a causal intermediate between noise and cardiovascular risk include accounting for potential confounding factors such as behavioral habits of modern life that lead to insufficient sleep; circadian rhythm disorders from jet lag, phase shifts, and shift work; and the presence of sleep disorders such as sleep apnea and periodic leg movement disorder (PLMD). There is also a need to consider vulnerable populations such as children, in whom early life influences can have a profound impact on future cardiovascular disease risk [57]. Low income and racial/ethnic minorities may also be predisposed to adverse effects of sleep disturbances on cardio-metabolic health [58]. This has particular public health implications as these groups also may be disproportionately exposed to sources of noise and have a high prevalence of short sleep.

Discussion of Relevant Markers of Cardiovascular Health

Following presentations on the above topics, workshop participants discussed at length what would be the “ideal” next study of aviation noise and cardiovascular health. In particular, we discussed the relative merits of various markers of cardiovascular health typically found in the literature (Table 2). We broadly categorize available markers as those related to the following: incident events, new diagnoses of established risk factors, markers of cardiovascular function, and markers of cardiovascular structure, acknowledging that not all endpoints fit neatly or exclusively into any given category.

In planning future studies of the potential impacts of aviation noise on cardiovascular health, we found that it was essential to be precise about the goals of such research as these goals will in turn inform study design and approach. Specifically, the overarching goal of understanding the impacts of aviation noise on cardiovascular health needs to be further refined so that the most appropriate research studies can be planned and executed. For example, studies aimed at improving estimates of the burden of disease attributable to specific changes in aviation noise levels (i.e., health impact assessments) typically dictate different approaches and settings versus studies aimed at improving our understanding of the underlying pathophysiological mechanisms by which aviation noise affects cardiovascular health.

Health impact assessments are often used to estimate the number of deaths or disease events averted or caused assuming one pattern of exposure versus an alternative, counterfactual exposure scenario. Health impact assessments have been used to quantify the burden of disease attributable to noise around airports in several European cities [26, 59]. Such assessments may also be used to inform the expected number of excess disease events following addition of a new runway at an airport or following implementation of new procedures aimed at reducing aviation noise exposures. Health impact assessments are sometimes limited by incomplete information on patterns of exposures or the relationship between exposure and outcomes in the population of interest. Thus, estimates of health impacts of aviation noise in the USA benefit from refined estimates of the distribution of exposures to aviation noise available through the AEDT or comparable models. Similarly, a more thorough evaluation of the effects of aviation noise on incident cardiovascular events such as MI or stroke, in specific populations and with special attention to identifying the

most vulnerable individuals, would permit more precise and informative health impact assessments directly related to policy questions of interest.

In contrast, studies designed to identify the physiologic mechanisms by which aviation noise may affect cardiovascular health are unlikely in the short-term to provide actionable insights that drive policy changes. For example, studies relating aviation noise to biochemical markers of stress or inflammation, or electrocardiographic markers of autonomic nervous system activation (e.g., heart rate variability), can provide valuable insights into the underlying mechanisms of disease, can help isolate the key features of exposure that elicit the observed health effects, or can aid identification of characteristics of individuals that may confer more or less susceptibility to these effects. Thus, results of studies of mechanisms of disease are, in aggregate, often used to provide biologic plausibility for observed associations with clinical disease endpoints, to refine the exposure metrics or populations of key interest, to develop conceptual models explaining how exposures are related to disease risk, to potentially identify intermediate targets amenable to interventions aimed at ameliorating the clinical impact of the exposure (e.g., use of anti-inflammatory or anti-hypertensive therapy in at risk groups), and to identify subclinical and clinical markers appropriate for monitoring the health effects of exposure.

In practice, many studies of aviation noise (and other environmental exposures) lie on a spectrum between these two goals, contributing to policy decisions and core knowledge in multiple ways. Some studies have reported an association between noise and measured blood pressure [20, 31, 60]. These studies may simultaneously provide insights about the mechanisms of disease (e.g., physiologic hemodynamic changes that may increase the risk of cardiovascular events in affected individuals), the exposure(s) of primary interest (e.g., separating out the effects of aviation noise from road traffic noise), and insights into the likely health impact of higher or lower future levels of exposure (given that blood pressure is an established and strong predictor of CVD risk). On the other hand, studies of heart rate variability or serum markers of systemic inflammation can provide novel insights into biologic mechanisms but provide relatively less information to enable the quantification of future risk of CVD. Studies of the impacts of aviation noise on sleep may inform both disease mechanism and inform interventions targeted at reducing exposures during the relevant time period.

Another important consideration when selecting the “ideal” cardiovascular health marker to measure is the time course of the effect under study. For instance, risk of cardiovascular events typically changes slowly over time while markers of vascular function can change virtually moment to moment; other markers fall somewhere in between these extremes. Thus, some markers of cardiovascular health will be better suited to studies targeted at evaluating the health impacts of short-term variation in exposure within individuals over time while other markers or endpoints will be better suited to evaluating differences in cardiovascular health across individuals with varying levels of long-term exposures. Blood pressure is an example of a marker that can provide insights about the impacts of both short-term (varying diurnally, across days, and across weeks) and long-term exposures.

In designing our ideal next study, workshop participants also considered the relative costs of these various markers of cardiovascular health, and the feasibility of measuring each marker in studies with sufficient sample size to detect associations of the expected magnitude. Many of the markers considered are time, equipment, or personnel intensive and thus challenging to assess well in large numbers of participants. For example, assessments of carotid intimal-medial thickness, flow-mediated dilation, overnight polysomnography, and ambulatory blood pressure monitoring have the potential to yield many novel insights, but are best suited to relatively smaller studies given their expense and participant or staff burden. On the other hand, as the cost of measurement of biochemical markers continues to drop, urinary or serum markers of cardiovascular health can often be measured in larger samples and/or repeatedly. Following participants for incident diagnoses or cardiovascular events can be scaled up even further, as demonstrated by several very large national cohort studies (e.g., the Women's Health Initiative (WHI), the Nurses' Health Studies (NHS), and the Health Professionals Follow-Up Study (HPFS)), among many others. The cost-effectiveness of proposed research is greatly increased by leveraging, where possible, existing data resources through collaboration with these and or other ongoing studies.

Although any of the above research designs can provide valuable evidence that improves our knowledge in this area, the ideal next study will be the one that is most closely aligned with the specific rather than broad policy questions or goals. If the policy question requires quantifying the number of cardiovascular deaths or events that might be prevented through introduction of new regulations, practices, or technologies, then new research that improves or facilitates detailed health impact assessments in the population of interest would be most responsive to these needs. Similarly, improved health impact assessments would allow regulators, airport operators, or community groups to identify the locations or populations where interventions to reduce exposure might have the greatest beneficial impacts on health. On the other hand, such studies will not typically provide the detail needed to establish biologic plausibility, inform conceptual models of exposure-disease relationships, or identify potential intermediate markers or targets for intervention.

Future Research Directions

Workshop participants made a number of recommendations for future research in this area. First, as noted above, we highlight the importance of clearly specifying the goals of the research program. Health impact assessments and the broad evidence needed to execute these well will likely have the greatest impact on policy in the short-term. In particular, in the short-term, key opportunities to improve the evidence base for policy decisions include the following: (a) improved estimates of exposure to noise, including identifying which metrics of aviation noise are most predictive of CVD risk, (b) more comprehensive assessment of the associations between aviation noise and risk of cardiovascular death, incident cardiovascular events, and new onset diagnoses of established cardiovascular risk factors, and (c) identification of subgroups of the population that may be at heightened risk. Evidence generated from these studies will enable government officials, airport operators, and community members to better understand the type and magnitude of health risks posed by aviation noise, how these estimates vary across location and people, and where interventions to reduce exposure might be most beneficial.

Over a longer-term, research studies are needed to fully elucidate the cascade of physiologic changes initiated by exposure to aviation noise and terminating in heightened risk of CVD morbidity and mortality. With this goal in mind, we suggest that studies evaluating clearly defined mechanistic pathways such as sleep disturbance, chronic heightened levels of stress, and changes in vascular functioning (among others) should be a priority. The evidence from this line of research will better establish biologic plausibility of the observed associations between aviation noise and CVD risk and provide key insights into future studies of specific cardiovascular event risk based on the presumed mechanisms of disease.

We further recommend that wherever possible investigators leverage existing data and research resources in well-characterized study populations, such as large, longitudinal cohorts with geocoded participant addresses, data on individual-level risk factors, and adjudicated cardiovascular events. Prime examples where collaboration may be particularly fruitful include prospective cohorts such as the Multi-Ethnic Study of Atherosclerosis (MESA), WHI, NHS, and the Sleep Heart Health Study, among many others. For example, as part of ongoing research within WHI, NHS, and HPFS, aircraft noise exposure is being modeled using AEDT in several metrics (DNL, Leq Night, Leq Day, TA 65 dB and TA 85 dB) at 5-year intervals between 1995 and 2015. These research efforts are investigating the effect of aviation noise on incident cardiovascular disease accounting for regional air pollution levels [https://ascent.aero/]. Performing analyses in the WHI, NHS, and HPFS cohorts using a common approach allows the association of aircraft noise to cardiovascular outcomes to be investigated in a very large and diverse population including both men and women. Beyond cohort studies, administrative claims data of hospitalizations among Medicare beneficiaries have already provided novel evidence [22], and there may be other administrative datasets that can be used for these purposes as well.

A related challenge in the USA is estimating exposure to road traffic noise, a likely confounder when examining associations between aviation noise and cardiovascular health. Unlike in the European Union, there is no legislative mandate for cities in the USA to systematically collect data on the key determinants of road or railway noise levels. Even basic markers of traffic volumes or fleet mix are not readily available in the USA on a national level. Although a number of studies suggest that residential proximity to major roadways in the USA is associated with adverse cardiovascular health, these studies have typically not been able to fully distinguish between the effects of traffic related air pollution versus traffic related noise [61–64].

Conclusions

There are growing concerns in the USA about aircraft noise. This is evident from increased community concerns about aircraft noise that have arisen in spite of a reduction in the number of people exposed to significant aircraft noise over the past decades. The limited evidence that is available today shows potential adverse impacts on cardiovascular health of aviation noise; however, the dataset is relatively small and the data have relatively large uncertainty. As such, there is an unmet need and opportunity to expand and strengthen the evidence base regarding the potential health impacts of aviation noise. This evidence base would be useful in informing decision-making regarding aviation noise in the USA. With

this need in mind, we call on the scientific community to leverage emerging tools to estimate aviation and road traffic noise to undertake a broad research agenda to estimate the potential adverse health effects of noise in the USA and more fully understand the causal mechanisms by which these putative effects occur as well as capturing the uncertainties in these impacts. The resulting evidence base will allow regulators and airport operators to ensure that continued aviation growth is accompanied by appropriate protections of the public health.

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References

Papers of particular interest, published recently, have been highlighted as:

• Of importance

•• Of major importance

1. U.S. Department of Transportation Bureau of Transportation Statistics. Airlines and airports. 2017. [Available from: https://www.bts.gov/topics/airlines-and-airports
2. U.S. Department of Transportation Federal Aviation Administration. Passenger boarding (enplanement) and all-cargo data for U.S. airports. 2017. [Available from: https://www.faa.gov/airports/planning_capacity/passenger_allcargo_stats/passenger/
3. U.S. Department of Transportaion Federal Avaiation Administration. Aviation environmental and energy policy statement. Fed Regist. 2012; 77(141):43137–43141.
4. Federal Aviation Administration. Aviation Abatement Policy. Fed Regist. 2000; 65(136):43802.
5. Eagan ME, Gardner R. National Research Council (U.S.) Transportation Research Board, Airport Cooperative Research Program, United States Federal Aviation Administration. Compilation of noise programs in areas outside DNL 65. Washington, D.C: Transp Res Board; 2009.
6. U.S. General Accounting Office, editor. Washington, DC: 2001. Aviation and the Environment: Transition to quieter aircrafts occured as planned but concerns about noise persists.
7. Basner M, Clark C, Hansell A, Hileman JI, Janssen S, Shepherd K, et al. Aviation noise impacts: state of the science. Noise Health. 2017; 19(87):41–50. [PubMed: 29192612]
8. Munzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. Eur Heart J. 2014; 35(13):829–36. [PubMed: 24616334]
9•. Zeeb H, Hegewald J, Schubert M, Wagner M, Droge P, Swart E, et al. Traffic noise and hypertension—results from a large case-control study. Environ Res. 2017; 157:110–7. Largest case-control study, to date, evaluating the association of traffic noise, including aircraft noise, with hypertension. Used health insurance claim data but had some individual-level information on age, sex, education and occupation. [PubMed: 28554004]
10••. Dimakopoulou K, Koutentakis K, Papageorgiou I, Kasdagli MI, Haralabidis AS, Sourtzi P, et al. Is aircraft noise exposure associated with cardiovascular disease and hypertension? Results from a cohort study in Athens, Greece. Occup Environ Med. 2017; 74(11):830–7. This study is one of two recent studies addressing the lack of prospective studies on aicraft noise and cardiovascular diesase. The study is relatively small given the outcomes, but was the first to investigate the effect on cardiac arrythmia. [PubMed: 28611191]
11••. 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. This large study investigates the associations between three sources of transportation noise and

relatively rarely investigated cardiovascular outcomes - heart failure and hypertensive heart disease. This study like Zeeb et al., 2017 uses health insurance claim data. [PubMed: 27667192]

12•. 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. This is one of a few studies relating aircraft noise with cardiovascular disease mortality; however it is an ecological study. [PubMed: 26356375]

13. Fastl H, Zwicker E. Psychoacoustics: facts and models. 3. Berlin; New York: Springer; 2007. 462xii

14. International Electrotechnical Commission. Electroacoustics— sound level meters—part 1: specifications. 2002 Contract No.: TC 29, ICS 17.140.50.

15. Swift H. A review of the literature related to potential health effects of aircraft noise: PARTNER Project 19 final report. Partnership for AiR Transportation Noise and Emission Reduction Center of Excellence, editor. 2010

16. Munzel T, Daiber A, Steven S, Tran LP, Ullmann E, Kossmann S, et al. Effects of noise on vascular function, oxidative stress, and inflammation: mechanistic insight from studies in mice. Eur Heart J. 2017; 38(37):2838–49. [PubMed: 28329261]

17. 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? Sci Total Environ. 2013; 452–453:50–7.

18. Babisch W. The noise/stress concept, risk assessment and research needs. Noise Health. 2002; 4(16):1–11.

19••. Heritier H, Vienneau D, Foraster M, Eze IC, Schaffner E, Thiesse L, et al. Transportation noise exposure and cardiovascular mortality: a nationwide cohort study from Switzerland. Eur J Epidemiol. 2017; 32(4):307–15. This very large nationwide study also adresses the lack of prospective studies evaluating the effect of aircraft noise on cardiovascular disease mortality. This study was able to show the individual effect of aircraft noise while accounting for the other two sources of transporation noise (rail and road). The study also introduced a novel metric, which quantified the contribution of individual noise events above background. [PubMed: 28280950]

20. 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? Occup Environ Med. 2017; 74(2):123–9. [PubMed: 27481872]

21••. 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. Dtsch Arztebl Int. 2016; 113(24):407–14. This study is related to Seidler A et al., 2016a. This large case-control study evaluates the relationship of myocardial infarction to exposure to three sources of transportation noise. [PubMed: 27380755]

22. Correia AW, Peters JL, Levy JI, Melly S, Dominici F. Residential exposure to aircraft noise and hospital admissions for cardiovascular diseases: multi-airport retrospective study. BMJ. 2013; 347:f5561. [PubMed: 24103538]

23. Floud S, Blangiardo M, Clark C, de Hoogh K, Babisch W, Houthuijs D, et al. Exposure to aircraft and road traffic noise and associations with heart disease and stroke in six European countries: across-sectional study. Environ Health. 2013; 12:89. [PubMed: 24131577]

24. 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. [PubMed: 24103537]

25. 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. [PubMed: 26356373]

26. 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. [PubMed: 26003939]

27. 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. [PubMed: 23821397]
28. 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. [PubMed: 25145323]
29. 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. Spat Spatiotemporal Epidemiol. 2016; 18:38–43. [PubMed: 27494958]
30. Stansfeld SA, Shipley M. Noise sensitivity and future risk of illness and mortality. Sci Total Environ. 2015; 520:114–9. [PubMed: 25804878]
31. Foraster M, Kunzli N, Aguilera I, Rivera M, Agis D, Vila J, et al. High blood pressure and long-term exposure to indoor noise and air pollution from road traffic. Environ Health Perspect. 2014; 122(11):1193–200. [PubMed: 25003348]
32. Basner M, Glatz C, Griefahn B, Penzel T, Samel A. Aircraft noise: effects on macro- and microstructure of sleep. Sleep Med. 2008; 9(4):382–7. [PubMed: 17870661]
33. Basner M, Griefahn B, Berg M. Aircraft noise effects on sleep: mechanisms, mitigation and research needs. Noise Health. 2010; 12(47):95–109. [PubMed: 20472955]
34. Javaheri S, Redline S. Insomnia and risk of cardiovascular disease. Chest. 2017; 152:435–44. [PubMed: 28153671]
35. Tobaldini E, Costantino G, Solbiati M, Cogliati C, Kara T, Nobili L, et al. Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. Neurosci Biobehav Rev. 2017; 74(Pt B): 321–9. [PubMed: 27397854]
36. Dean DA, Wang R, Jacobs DR, Duprez D, Punjabi NM, Zee PC, et al. A systematic assessment of the association of polysomnographic indices with blood pressure: the Multi-Ethnic Study of Atherosclerosis (MESA). Sleep. 2014
37. Tasali E, Leproult R, Ehrmann DA, Van Cauter E. Slow-wave sleep and the risk of type 2 diabetes in humans. Proc Natl Acad Sci U S A. 2008; 105(3):1044–9. [PubMed: 18172212]
38. Buxton OM, Cain SW, O'Connor SP, Porter JH, Duffy JF, Wang W, et al. Adverse metabolic consequences in humans of prolonged sleep restriction combined with circadian disruption. Sci Transl Med. 2012; 4(129):129ra43.
39. Leproult R, Copinschi G, Buxton O, Van Cauter E. Sleep loss results in an elevation of cortisol levels the next evening. Sleep. 1997; 20(10):865–70. [PubMed: 9415946]
40. Castro-Diehl C, Diez Roux AV, Redline S, Seeman T, Shrager SE, Shea S. Association of sleep duration and quality with alterations in the hypothalamic-pituitary adrenocortical axis: the Multi-Ethnic Study of Atherosclerosis (MESA). J Clin Endocrinol Metab. 2015; 100(8):3149–58. [PubMed: 26046965]
41. Castro-Diehl C, Diez Roux AV, Redline S, Seeman T, McKinley P, Sloan R, et al. Sleep duration and quality in relation to autonomic nervous system measures: the Multi-Ethnic Study of Atherosclerosis (MESA). Sleep. 2016; 39:1919–26. [PubMed: 27450685]
42. Mullington J, Hermann D, Holsboer F, Pollmacher T. Age-dependent suppression of nocturnal growth hormone levels during sleep deprivation. Neuroendocrinology. 1996; 64:233–41. [PubMed: 8875441]
43. Meier-Ewert HK, Ridker PM, Rifai N, Regan MM, Price NJ, Dinges DF, et al. Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. J Am Coll Cardiol. 2004; 18(43):678–83.
44. Mullington JM, Haack M, Toth M, Serrador JM, Meier-Ewert HK. Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. Prog Cardiovasc Dis. 2009; 51(4):294–302. [PubMed: 19110131]
45. Irwin MR, Wang M, Campomayor CO, Collado-Hidalgo A, Cole S. Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. Arch Intern Med. 2006; 166(16):1756–62. [PubMed: 16983055]
46. Spiegel K, Leproult R, L'Hermite-Baleriaux M, Copinschi G, Penev PD, Van Cauter E. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate

regulation, cortisol, and thyrotropin. J Clin Endocrinol Metab. 2004; 89(11):5762–71. [PubMed: 15531540]

47. Spiegel K, Leproult R, Tasali E, Penev P, Van Cauter E. Sleep curtailment results in decreased leptin levels and increased hunger and appetite. Sleep. 2003; 26:A174.
48. Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. Obesity. 2008; 16(3): 643–53. [PubMed: 18239586]
49. Patel SR, Blackwell T, Redline S, Ancoli-Israel S, Cauley JA, Hillier TA, et al. The association between sleep duration and obesity in older adults. Int J Obes. 2008; 32(12):1825–34.
50. Fung MM, Peters K, Redline S, Ziegler MG, Ancoli-Israel S, Barrett-Connor E, et al. Decreased slow wave sleep increases risk of developing hypertension in elderly men. Hypertension. 2011; 58(4):596–603. [PubMed: 21876072]
51. Anothaisintawee T, Reutrakul S, Van Cauter E, Thakkinstian A. Sleep disturbances compared to traditional risk factors for diabetes development: systematic review and meta-analysis. Sleep Med Rev. 2015; 30:11–24. [PubMed: 26687279]
52. Kwon Y, Gharib SA, Biggs ML, Jacobs DR Jr, Alonso A, Duprez D, et al. Association of sleep characteristics with atrial fibrillation: the Multi-Ethnic Study of Atherosclerosis. Thorax. 2015; 70:873–9. [PubMed: 25986436]
53. Itani O, Jike M, Watanabe N, Kaneita Y. Short sleep duration and health outcomes: a systematic review, meta-analysis, and meta-regression. Sleep Med. 2017; 32:246–56. [PubMed: 27743803]
54. Goodwin JL, Kaemingk KL, Fregosi RF, Rosen GM, Morgan WJ, Sherrill DL, et al. Clinical outcomes associated with sleep-disordered breathing in Caucasian and Hispanic children—the Tucson Children's Assessment of Sleep Apnea study (TuCASA). Sleep. 2003; 26(5):587–91. [PubMed: 12938812]
55. Bixler EO, Vgontzas AN, Lin HM, Liao D, Calhoun S, Fedok F, et al. Blood pressure associated with sleep-disordered breathing in a population sample of children. Hypertension. 2008; 52(5): 841–6. [PubMed: 18838624]
56. Javaheri S, Storfer-Isser A, Rosen CL, Redline S. Sleep quality and elevated blood pressure in adolescents. Circulation. 2008; 118(10):1034–40. [PubMed: 18711015]
57. Srinivasan SR, Myers L, Berenson GS. Predictability of childhood adiposity and insulin for developing insulin resistance syndrome (syndrome X) in young adulthood: the Bogalusa Heart Study. Diabetes. 2002; 51(1):204–9. [PubMed: 11756342]
58. Jackson CL, Redline S, Emmons KM. Sleep as a potential fundamental contributor to disparities in cardiovascular health. Annu Rev Public Health. 2015; 36:417–40. [PubMed: 25785893]
59. 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. Occup Environ Med. 2004; 61(5):405–13. [PubMed: 15090660]
60. 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. [PubMed: 18270210]
61. Ward-Caviness CK, Kraus WE, Blach C, Haynes CS, Dowdy E, Miranda ML, et al. Associations between residential proximity to traffic and vascular disease in a cardiac catheterization cohort. Arterioscler Thromb Vasc Biol. 2018; 38(1):275–82. [PubMed: 29191927]
62. Dorans KS, Wilker EH, Li W, Rice MB, Ljungman PL, Schwartz J, et al. Residential proximity to major roads, exposure to fine particulate matter, and coronary artery calcium: the Framingham Heart Study. Arterioscler Thromb Vasc Biol. 2016; 36(8):1679–85. [PubMed: 27312220]
63. Hart JE, Chiuve SE, Laden F, Albert CM. Roadway proximity and risk of sudden cardiac death in women. Circulation. 2014; 130(17):1474–82. [PubMed: 25332277]
64. Kirwa K, Eliot MN, Wang Y, Adams MA, Morgan CG, Kerr J, et al. Residential proximity to major roadways and prevalent hypertension among postmenopausal women: results from the Women's Health Initiative San Diego Cohort. J Am Heart Assoc. 2014; 3(5):e000727. [PubMed: 25274494]

Table 1

Studies of aircraft noise and cardiovascular disease (CVD) outcomes

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
Dimakopoulou et al. [10••]	HYENA Study-Athens (number [*n*] = 420)	Prospective	Athens, Greece/ 2004–2006	A-weighted sound equivalent level (LAeq) modeled with Integrated Noise Model (INM); also modeled road traffic noise	Incident hypertension, cardiac arrhythmia. myocardial infarction (MI), stroke, diabetes	Age, sex, body mass index (BMI), alcohol intake, education, exercise, smoking, salt intake	Aircraft noise, particularly nighttime noise, related to incident hypertension and prevalent and incident arrhythmia. Odds Ratio (OR) (95% Confidence Intervals (CI) per 10 dB increase in aircraft noise, L_{night}: • Hypertension: *2.63 (1.21–5.71)* • Arrhythmia: *2.09 (1.07–4.08)* • MI: 0.83 (0.31–2.20) • Stroke: 1.30 (0.32–5.31) • Diabetes: 1.09 (0.58–2.07)
Héritier et al. [19••]	Swiss National Cohort (> 30 years) (*n* = 4.4 million [mil])	Prospective	Switzerland/ 2000–2008	Day-evening-night equivalent level (L_{den}) estimated using FLULA2 and calculation of night intermittency ratio (IR); also modeled road traffic and rail noise	CVD mortality, ischemic heart disease (IHD). stroke, MI, heart failure (HF), blood pressure (BP) mortality	Sex, neighborhood socio-economic status (SES), marital status, education, language. nationality, nitrogen dioxide (NO_2)	Aircraft noise associated with mortality from MI, HF, and ischemic stroke. Hazard Ratio (HR) (95% CI per 10 dB increase in aircraft noise, L_{den}: • CVD: 0.994 (0.985–1.002) • BP: 1.012 (0.985–1.039) • IHD: 0.991 (0.978–1.003) • MI: *1.027 (1.006–1.049)* • HF: *1.056 (1.028–1.085)*

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
							• Stroke: 1.013 (0.993–1.033) • Hemorrhagic stroke: 0.991 (0.951–1.032) • Ischemic stroke: *1.074 (1.020–1.127)* At night, mid-range IR more harmful than continuous and highly intermittent noise. Relationship also seen with road and railway noise
Zeeb et al. [9•]	Residents near Frankfurt Airport (≥ 40 years) (cases = 137,577; controls = 355,591)	Case-control	Frankfurt, Germany/ 2005–2010	LAeq estimated using radar data; also modeled road traffic and rail noise	Hypertension	Age, sex, education. occupation, area-level poverty index	Significant associations found between transportation noise and hypertension only in cases with subsequent hypertensive heart disease (HHD) diagnosis. OR (95% CI) per 10 dB increase in aircraft noise, LAeq: • Hypertension (all cases): 0.997 (0.985–1.010) • Hypertension (cases with HHD): *1.139 (1.090–1.190)* Increased risk of hypertension also observed in cases with longer exposure duration
Evrard et al. [20]	DEBATS study (> 18 years) (*n* = 1244)	Cross-sectional	Paris, Toulousse. and Lyon. France/ 2013	LAeq estimated based on noise maps	Hypertension, BP	Age, sex, BMI, exercise, alcohol intake, occupation. hypertensive medication	Relationship found between nighttime aircraft noise and hypertension and BP in men. Daytime exposure associated with BP but not hypertension. Estimates (95% CI) per 10 dB increase in aircraft noise, L_{den}:

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
							• Hypertension: OR *1.48 (1.00–1.97)* • Diastolic BP: Beta (β): *1.86 (0.40–3.30)* • Systolic BP: β: *2.37 (0.16–4.59)* Estimates (95% CI) per 10 dB increase in aircraft noise, L_{night}: • Hypertension: OR *1.34 (1.00–1.97)* • Diastolic BP: β *1.67 (0.34–3.00)* • Systolic BP: β *2.17 (0.13–4.19)*
Pearson et al. [29]	Residents near London Heathrow Airport (*n* = 0.7 mil)	Interrupted time series	London, UK/ 2010	LAeq estimated based on noise contour of 55 dB	Cardiovascular hospital admissions	Compared to a control area selected using a 20-km buffer constructed around the 55 dB noise contour	No evidence of decreased hospital admission from CVD during 6-day airport closure due to volcanic ash cloud. • Relative Risk (RR) (95% CI): 0.97 (0.75–1.26).
Seidler et al. [21••]	Health insured in Rhine-Main area. Germany (≥ 40 years) (*n* = 104,145 cases and 654,172 controls)	Case-control	Rhine-Main area. Germany/ 2006–2010	LAeq modeled using radar data; also modeled road traffic and rail noise	HHD and HF	Age, sex, education. occupation, area-level poverty index	Aircraft noise associated with primarily HHD but also HF. OR (95% CI) per 10 dB increase in aircraft noise, LAeq: • HHD and HF: *1.016 (1.003–1.030)* Relationship also seen with road and railway noise

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
Seidler et al. [11••]	Health insured (as above) Germany (≥ 40 years) (*n* = 19,632 cases and 834,734 controls)	Case-control	Rhine-Main area. Germany/ 2006–2010	LAeq modeled using radar data; also modeled road traffic and rail noise	MI	Age, sex, education. occupation, area-level poverty index	Increased risk of MI found in relation to 24-h road traffic noise and railway noise, but not with aircraft noise. OR (95% CI) per 10 dB increase in aircraft noise, LAeq: • MI: 0.993 (0.966–1.020) However, significant relationship found between aircraft noise levels ≥ 60 dB and MI, in patients who subsequently died of ML OR (95% CI) per 10 dB increase in aircraft noise exposure. LAeq: • Fatal MI: *2.70 (1.08–6.74)*
Evrard et al. [12•]	Communities near French airports (*n* = 1.9 mil)	Ecological	Paris, Toulousse. and Lyon. France/ 2007–2010	L_{den} AEI (population-weighted or average energy index) modeled using INM.	Mortality from CVD, coronary heart disease (CHD), MI, and stroke	Age, sex, population density. lung cancer mortality, poverty index	Associations found between noise and CVD, CHD and MI mortality. Significant associations not attenuated by adjustment for air pollution (NO_2 and particulate matter (PM_{10})). Mortality Rate Ratio (MRR) (95% CI) per 10 dB increase in aircraft noise, L_{den}AEI: • CVD: *1.18 (1.11–1.25)* • CHD: *1.24 (1.12–1.36)* • MI: *1.28 (1.11–1.46)* • Stroke: 1.08 (0.97–1.21)

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
Méline et al. [25]	RECORD Study (30–79 years) (*n* = 7290)	Cross-sectional	Île-de-France region, France/ 2007–2008	L_{den} also modeled road traffic and rail noise	BP and hypertension		Total transportation noise and road traffic noise at workplace and workplace neighborhood were associated with BP, but not with hypertension. No association found with residential noise
Vienneau et al. [26]	Swiss population	Health impact assessment	Switzerland/ 2010	L_{den} Spatially resolved models; also modeled road traffic and rail noise	Mortality and morbidity for cardiovascular and respiratory conditions		Estimated 6000 years of life lost (YLL) from noise and 14,000 from air pollution. YLL for transportation dominated by road traffic air pollution, morbidity, and quality of life by noise
Schmidt et al. [28] (on-line 2014)	FLIGHT-RISK study (patients 30–75 years with or at high risk for CHD) *n* = 60	Laboratory	Not described	Leq Noise simulated and polygraphy recorded near German airport	Flow-mediated dilation (FMD), BP, as well as markers of hemodynamics, inflammation. neuro-hormones; also sleep quality	Age, sex, night sequence, noise sensitivity, attitude towards noise, cardiac rhythm	Nighttime noise related to reduced FMD and increased BP in patients with or at high risk for CHD, independent of annoyance FMD: from *9.6 ± 4.4 to 7.9 ± 3.7%* (*p* value < 0.001) Systolic BP: from *129.5 ± 16.5 to 133.6 ± 17.9 mmHg* (*p* value 0.03)
Stansfeld and Shipley [30]	Whitehall II Study of British Civil Servants (34–55 years) (*n* = 3630)		UK/1985–2009	Annoyance to all sources of noise assessed by questionnaire	CHD and stroke mortality and morbidity, non-fatal MI, angina; also mental health	Age, sex, employment grade, self-rated health. psychological distress	No association between noise sensitivity and CVD morbidity or mortality, except in people from lower employment grades where there was an association with angina. HR (95% CI) for association with noise sensitivity: • Mortality (all cause): 0.95 (0.79–1.14) • CHD mortality/ non-fatal MI: (1.03 (0.77–1.39) • Angina: 1.06 (0.89–1.25)

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
Babisch et al. [17]	HYENA Study (45–70 years) (*n* = 4861)	Cross-sectional	London, Berlin. Amsterdam. Stockholm. Milan, and Athens/ 2003–2005	LAeq modeled using INM and aircraft noise contour (ANCON [UK]); also measured road traffic noise	BP (hypertension)	Age, sex, BMI, alcohol intake, education, exercise, study area, road noise	The noise-hypertension relationship was modified by annoyance—higher in the more annoyed (interaction term *p* value = 0.048). OR (95% CI) per 10 dB increase in aircraft noise, L_{den}: • Hypertension (low annoyance): 0.944 (0.833–1.070) • Hypertension (moderate/ high annoyance): 1.095 (0.970–1.235) Indication that noise level is more strongly predictive than noise annoyance
Correia et al. [22]	Medicare enrollees surrounding 89 US airports (> 65 years) (*n* ~ 6 mil)	Retrospective study	USA/ 2009	Day-night average sound level (DNL) population-weighted average noise level	Hospital admission for cardiovascular diseases	Age, sex, race, area-level SES and demographics, air pollution ($PM_{2.5}$ and ozone)	Averaged across all airports and using a 90th percentile noise exposure metric, noise exposure associated with increased cardiovascular hospital admission rate. RR (95% CI) per 10 dB increase in aircraft noise exposure, DNL: • CVD hospital admission rate: *1.035 (1.002–1.070)*
Floud et al. [23]	HYENA Study (45–70 years) (*n* = 4712)	Cross-sectional	London, Berlin. Amsterdam. Stockholm. Milan, and Athens/ 2004–2006	LAeq using INM and ANCON Also measured road traffic noise and air pollution	Self-report of heart disease, stroke	Age, sex, BMI, education, ethnicity, other noise	Nighttime noise associated with heart disease and stroke in those living in the same location ≥ 20 years. Association persisted with further adjustment for air pollution in a subsample.

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
							OR (95% CI) per 10 dB increase in aircraft noise, L_{night}: • Heart disease and stroke: *1.25 (1.03–1.51)* Road noise also found to be associated with heart disease and stroke
Hansell et al. [24]	Residents in boroughs exposed to aircraft noise from Heathrow airport. (*n* ~ 3.6 mil)	Small area study	London. UK/2001–2005	Population-weighted average noise levels for daytime and nighttime	Hospital admission and mortality for stroke. CHD, and CVD classifications.	Controlled for: age, sex, ethnicity, SES. area-level lung cancer mortality	High noise levels associated with increased risks of stroke, CHD, and CVD for both hospital admissions and mortality. RR (95% CI) comparing highest to lowest nighttime aircraft noise exposure categories, L_{night}: • Stroke (hospital admissions): *1.29 (1.14–1.46)* • Stroke (mortality): *1.23 (1.02–1.49)* • CHD (hospital admissions): *1.12 (1.04–1.20)* • CHD (mortality): 1.11 (0.99–1.24) • CVD (hospital admissions): *1.09 (1.04–1.14)* • CVD (mortality):

Reference	Population	Study design	Place/years (years)	Noise metrics	CVD outcomes	Control variables	Key findings
							1.14 (1.03–1.26) Similar results observed for daytime aircraft noise
Schmidt et al. [27]	Healthy volunteers (mean 26 years) (*n* = 75)	Laboratory	Not described	LAeq Noise simulated and polygraphy recorded near German airport	FMD, BP, markers of hemodynamics, inflammation. neuro-hormones; also sleep quality		No direct relation of nighttime noise to FMD, but a monotonic dose-dependent FMD response. Evidence of priming effect —effect more evident when participants were first exposed to 30 then to 60 noise events. Also related to epinephrine levels and pulse transit time

Statistically significant in italics

Table 2

Examples of markers of cardiovascular health and potential biologic intermediates connecting aviation noise to cardiovascular health. Categories are not necessarily mutually exclusive and many markers could be interpreted to inform multiple aspects of cardiovascular health or mechanisms

New onset cardiovascular events	- Myocardial infarction - Stroke - Heart failure - Coronary artery revascularization - Cardiovascular death
New diagnosis of established risk factors for cardiovascular events	- Hypertension - Diabetes - Dyslipidemias - Atrial fibrillation - Renal disease
Markers of cardiovascular function	- Vascular function, resistance, and reactivity (e.g., blood pressure, pulse wave amplitude, flow-mediated dilation) - Electrocardiographic measures (e.g., heart rate, heart rate variability, ST-segment changes) - Left ventricular ejection fraction - Myocardial strain - Exercise capacity
Markers of cardiovascular structure	- Left ventricular mass - Cardiac hypertrophy - Carotid intimal-medial thickness (cIMT); carotid plaque - Coronary artery calcium