# Valuing Quiet

## An Economic Assessment of U.S. Environmental Noise as a Cardiovascular Health Hazard

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**Introduction:** Environmental noise pollution increases the risk for hearing loss, stress, sleep disruption, annoyance, and cardiovascular disease and has other adverse health impacts. Recent (2013) estimates suggest that more than 100 million Americans are exposed to unhealthy levels of noise. Given the pervasive nature and significant health effects of environmental noise pollution, the corresponding economic impacts may be substantial.

**Methods:** This 2014 economic assessment developed a new approach to estimate the impact of environmental noise on the prevalence and cost of key components of hypertension and cardiovascular disease in the U.S. By placing environmental noise in context with comparable environmental pollutants, this approach can inform public health law, planning, and policy. The effects of hypothetical national-scale changes in environmental noise levels on the prevalence and corresponding costs of hypertension and coronary heart disease were estimated, with the caveat that the national-level U.S. noise data our exposure estimates were derived from are >30 years old.

**Results:** The analyses suggested that a 5-dB noise reduction scenario would reduce the prevalence of hypertension by 1.4% and coronary heart disease by 1.8%. The annual economic benefit was estimated at $3.9 billion.

**Conclusions:** These findings suggest significant economic impacts from environmental noise-related cardiovascular disease. Given these initial findings, noise may deserve increased priority and research as an environmental health hazard.

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## Introduction

Environmental noise from road traffic, aircraft, construction and industrial activities, recreational activities, and other sources is a pervasive pollutant associated with a myriad health risks. Noise has detrimental health impacts even at relatively low exposure levels and among people not reporting noise annoyance.[1,2] Recent estimates indicate that more than 100 million Americans are exposed to levels of environmental noise that put them at risk for hearing loss,[3] stress, sleep disruption, annoyance, and cardiovascular disease.[1] A decrease in an annual equivalent continuous average ($L_{Aeq}$) environmental noise exposure level of 10 decibels (dB) decreases risk of cardiovascular disease by 7%–17%.[4] To put this in context, a decrease in noise levels of this magnitude is about twice the decrease in noise levels that occurred around airports after federal regulations in the 1970s. Cardiovascular disease is the top cause of mortality in the U.S.[5] and presents a substantial health-related economic burden. The ubiquitous nature of environmental noise in urban[6] and non-urban[7] areas of the U.S. affects an increasing number of Americans, but research on the economic toll resulting from cardiovascular disease has primarily been conducted in Europe.

Previous research evaluating the economic impacts of environmental noise has largely focused on transport-related noise and house prices.[8–11] This approach is limited because noise levels measured at a residence can differ substantially from individual noise exposures of residents. Also, perceptions of noise hazards, especially at

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low noise levels, are underestimated, and homebuyers may not fully perceive and value the potential health impacts of noise.[1,2] Finally, high relocation costs may mask people's true preferences.

This assessment seeks to expand upon our understanding of the economic ramifications of environmental noise pollution. We focus on two major categories of cardiovascular disease—coronary heart disease (CHD) and hypertension. A hypothetical noise reduction scenario of 5 dB at a population level is explored, applying published estimates of the relationship between environmental noise and cardiovascular disease, and estimating changes in prevalence and costs of cardiovascular disease. Given the cost of cardiovascular disease in the U.S., even small reductions in cardiovascular disease from reductions in environmental noise could produce significant economic benefits.

## Methods

### Prevalence and Costs of Cardiovascular Disease

Cardiovascular disease, which includes CHD and hypertension, is pervasive and costly, affecting 27% of Americans (>83 million) and accounting for 15% of healthcare expenditures ($324 billion) in 2010.[12,13] CHD, which includes myocardial infarction, affected 15.4 million Americans in 2010 and cost $96 billion in direct healthcare costs and >$81 billion in lost productivity.[13] Hypertension affected almost 78 million Americans in 2010[13] and cost $47.5 billion in treatment.[12] Some individuals are affected by both CHD and hypertension, which we accounted for in these analyses.

### Prevalence of Harmful Noise Exposure

Environmental impacts of noise are assessed with a sound level meter that measures decibel levels using the A-weighting filter, which approximates the sensitivity of the human ear to certain frequencies. Where localized exposure measurements are unavailable, these levels are estimated using noise exposure models. In 1974, the U.S. Environmental Protection Agency (EPA) recommended an average 24-hour exposure limit of 55 A-weighted dB (dBA) of environmental noise to protect the public from adverse effects on health and welfare in residential areas.[14] This limit is a day–night 24-hour average noise level ($L_{DN}$), with a 10-dB penalty applied during nighttime hours (11PM–7AM) to account for potential sleep disturbance. We based the scenario in this paper on $L_{DN}$ levels.[14] Other nations use different metrics, including the day–evening–night level ($L_{DEN}$), and the average over the 16 daytime hours ($L_{Aeq,16h}$) of 7AM–11PM. For context, 55 dBA is approximately as loud as a microwave oven at 1 to 2 ft, and a 24-hour $L_{DN}$ exposure of 55 dBA could result from 8 hours in a typical large office environment (55 dBA), a 1-hour lunch in a restaurant (60 dBA), a 1-hour commute by automobile (65 dBA), 6 hours doing miscellaneous activities at home (50 dBA), and 8 hours of sleep in a quiet bedroom (40 dBA).

A 1981 EPA report estimated that 46.2% of the population was exposed to ≥58 dBA $L_{DN}$ from environmental noise, and 13.9% were exposed to ≥65 dBA $L_{DN}$.[15] Though dated (and considerably different from recent WHO guidance),[16] a 1974 recommendation remains the most current EPA guidance on environmental noise, and the 1981 estimates are the most recent data on exposure above the recommended thresholds. These estimates were produced by applying models of 11 sources of environmental noise to the U.S. population. Among those exposed to ≥58 dBA $L_{DN}$, traffic or aircraft was the primary source of exposure for the vast majority, though rail, construction, and domestic appliances were also considered.[15]

We made the conservative assumption that the proportion of the U.S. population exposed to high levels of noise was the same in 2013 as estimated in 1981.[3] Assuming zero trend in noise levels is likely a considerably conservative assumption given increasing urbanization in the U.S.[17] In 2013, an estimated 46.2% of Americans (145.5 million) were exposed to ≥58 dBA $L_{DN}$, and 13.9% (43.8 million) were exposed to ≥65 dBA $L_{DN}$. In sum, high levels of environmental noise exposure (largely from transportation) are extremely common—conservatively, nearly half of all Americans are exposed to environmental noise above the outdated, but most recent, 1974 EPA-recommended level.

### Relationship Between Environmental Noise and Cardiovascular Disease

Potential mechanisms for noise to impact cardiovascular disease are described more fully by Hammer et al.[3] and comprehensively in Goines and Hagler.[1] Briefly, noise is an environmental stressor that impacts sleep, relaxation, and concentration and increases the risk of hypertension and CHD in the long term.[1,4] The impact of noise on cardiovascular disease varies based on the level of noise, duration of exposure, frequency spectrum, source, time of day, and other factors.[4] The exact mechanism(s) by which noise causes cardiovascular disease remains unclear, but appear to be mainly related to sleep, stress, and disruption of neuro-endocrine cycles.[4] Nevertheless, the weight of evidence of the effects of noise on cardiovascular disease (e.g., multiple studies of different types in different populations) is nevertheless substantial, and effect sizes have been shown to be similar across several studies.

Evaluations of the cardiovascular effects of environmental noise have utilized different noise measures (e.g., transport noise, occupational noise, self-reported noise annoyance) and different cardiovascular disease indicators (e.g., CHD, myocardial infarction, hypertension, stroke). Table 1 provides a summary of findings from a selection of primary studies and meta-analyses.

Noise is only one of many factors that impact the risk of CHD,[31] and even taking other lifestyle and environmental factors (such as air quality) into account, a majority of the studies found significant, positive relationships between environmental noise and CVD. As Basner and colleagues[4] summarize, the risk of hypertension or CHD increases by 7%–17% per 10 dB $L_{Aeq}$ increased noise exposure.

### Assessment of Economic Impact of Noise-Related Coronary Heart Disease and Hypertension

Drawing assumptions from the assessments above, the following model estimated the change in prevalence and cost of CHD and hypertension associated with a 5-dB $L_{DN}$ reduction in environmental noise exposure. Table 2 presents key assumptions for this model.

The 5-dB $L_{DN}$ reduction scenario corresponds to established elasticities that estimate how cardiovascular disease prevalence changes in relation to a 5- or 10-dB $L_{DN}$ change in noise exposure

**Table 1.** Selected Evidence on the Relationship Between Noise and Cardiovascular Disease

| Publication | Source of data | Noise source(s) | Cardiovascular disease metric | Relationship and metric(s) |
|---|---|---|---|---|
| Babisch (2014)[18] | Meta-analysis | Road traffic | Varying indicators of coronary heart disease | 10 dB $L_{DN}$ increase in noise exposure increases risk of CHD by 8% over a range of 52–77 dBA |
| Basner et al. (2013)[4] | Literature review | Road traffic<br>Air traffic | Hypertension<br>IHD | 10 dB $L_{Aeq}$ increase of environmental noise increases risk of hypertension or IHD 7%–17%. |
| Hansell et al. (2013)[19] | 3.6 million residents near Heathrow Airport, United Kingdom | Air traffic<br>Daytime and nighttime noise separate<br>Modeled noise exposure | Hospital admissions and mortality from:<br>Stroke<br>CHD<br>cardiovascular disease | Daytime aircraft noise resulted in statistically significant increased risk of hospital admission for stroke (1.24 RR); cardiovascular disease (1.14 RR); and CHD (1.21 RR); RRs were higher for nighttime noise, and similar impacts on mortality; Noise measure: daytime=51–63 dB $L_{Aeq}$, nighttime=50–55 dB $L_{Aeq}$ |
| Van Kempen and Babisch (2012)[20] | Meta-analysis | Road traffic | Hypertension | 5 dB $L_{Aeq,16h}$ increase in noise exposure increases risk of hypertension by 3.4% over a range of 45–75 dBA |
| Ndrepepa and Twardella (2012)[21] | Meta-analysis of 8 studies | Self-reported annoyance from road traffic | Hypertension<br>IHD | Noise annoyance significantly, positively associated with hypertension (1.16 pooled risk estimate). Noise annoyance significantly, positively associated with IHD (1.07 pooled risk estimate). |
| Sørensen et al. (2012)[22] | 57,053 residents of Copenhagen or Aarhus, Denmark | Road traffic<br>Modeled noise exposure | MI | 10 dB $L_{DEN}$ residential road traffic noise significantly associated with MI (1.12 incidence rate ratio) |
| Gan et al. (2012)[23] | Residents of Vancouver, Canada | Road traffic<br>Modeled noise exposure | CHD mortality | 10 dB(A) $L_{DEN}$ elevation in residential noise levels associated with risk of death from CHD (9% increase); other cardiovascular disease indicators also showed positive relationship with noise; stroke death showed positive relationship but not statistically significant |
| Gan et al. (2011)[24] | 6,307 participants in the U.S. NHANES survey | Self-reported occupational noise exposure | Angina pectoris<br>CHD<br>Hypertension<br>MI | Exposure to loud noise in the workplace significantly and positively associated with all cardiovascular disease measures: angina pectoris (2.91 OR); CHD (2.04 OR); hypertension (2.23 OR) |
| Babisch and van Kamp (2009)[25] | Meta-analysis | Air traffic | Hypertension | 10 dBA $L_{DN}$ air traffic noise significantly associated with increase in hypertension (1.13 RR) over the range 45–70 dBA |
| Babisch (2008)[26] | Meta-analysis of 7 studies | Road traffic | MI | Noise exposure levels above 60 dBA $L_{day,16h}$ significantly associated with increased risk of MI; for noise levels above 70 dBA $L_{day,16h}$, OR $>1.2$; no increased risk found at noise levels $<60$ dBA $L_{day,16h}$ |
| Babisch (2006)[27] | Literature review/meta-analysis | Air traffic<br>Road traffic<br>Rail traffic | IHD<br>Hypertension | Noise exposure levels less than 60 dBA $L_{day}$ were not associated with increased IHD; 65–70 dBA $L_{day}$ noise exposure was associated with increased risk of IHD (1.1–1.5 RR); 60–70 dBA $L_{day}$ noise exposure associated with increased risk of hypertension (1.4–2.1 RR), though earlier studies indicated a less clear relationship |

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**Table 1.** Selected Evidence on the Relationship Between Noise and Cardiovascular Disease *(continued)*

| Publication | Source of data | Noise source(s) | Cardiovascular disease metric | Relationship and metric(s) |
|---|---|---|---|---|
| Willich et al. (2006)[2] | 4,115 patients admitted to hospitals in Berlin | Self-reported environmental and occupational annoyance<br>Road traffic<br>Rail traffic<br>Modeled noise exposure | MI | Environmental noise annoyance slightly significantly positively associated with MI in women, and no association for men; environmental noise associated with increased risk of MI in men (1.46 OR) and women (3.36 OR); occupational noise associated with increased risk of MI in men (1.31 OR) but not in women |
| van Kempen and Staatsen (2005)[28] | Netherlands | Air traffic<br>Road traffic | Hypertension | Population-attributable risk of 0.06 for noise-induced hypertension (a maximum of 200,000 cases of hypertension in the Netherlands could be attributable to road traffic noise exposure) |
| van Kempen et al. (2002)[29] | Meta-analysis of more than 500 studies 1970–1999 in English, German, or Dutch | Occupational exposure<br>Road traffic<br>Rail traffic | Blood pressure | 5 dBA $L_{Aeq}$ occupational noise exposure significantly associated with increased hypertension (1.14 RR); 5 dBA $L_{Aeq}$ air traffic noise exposure significantly associated with increased hypertension (1.26 RR) (this based on just one study) |
| van Kempen et al. (2002)[29] | Meta-analysis of more than 500 studies 1970–1999 in English, German, or Dutch | Road traffic<br>Rail traffic | IHD | Community noise positively associated with MI, IHD, angina pectoris, use of cardiovascular medicines, but none of these measures were statistically significant |
| Passchier-Vermeer and Passchier (2000)[30] | Literature review | Mainly road traffic, one air traffic study | Hypertension<br>IHD | Sufficient evidence for a noise exposure–related effect above 70 dBA $L_{DN}$ |

CHD, coronary heart disease; IHD, ischemic heart disease; MI, myocardial infarction; NHANES, National Health and Nutrition Examination Survey; RR, relative risk.

(Table 1). A 5-dB reduction in annual $L_{DN}$ appears feasible based on demonstrated and ongoing reductions in aircraft noise following federal regulation,[33] and was modeled on similar regulatory approaches for air contaminants, which can include technology-forcing policies. A 5-dB reduction could be achieved through a multi-pronged intervention targeting noise sources that represent the greatest burden of noise exposure in urban areas, including low noise pavement and quiet tire design, traffic calming measures, noise barriers, changes in aircraft flight patterns, adoption of electrical vehicles, incorporation of available noise control technology into industrial and construction equipment, greater use of hearing protection in occupational and public settings, and other approaches.[34] No single one of these approaches could achieve a 5-dB $L_{DN}$ reduction at a population level, but an integrated strategy employing multiple approaches could do so. The cost would be substantial, but the changes would by necessity be phased in over a long period of time, and would provide ancillary benefits (e.g., improved quality of life and air pollution reduction).

The 5-dB $L_{DN}$ reduction was applied to the 145.5 million Americans exposed to 58 dBA $L_{DN}$, which we assumed was a conservative underestimate of those exposed to $\geq$55 dBA $L_{DN}$. It was also conservatively assumed that everyone in this group was exposed to the lowest noise level for the group (exactly 55 dBA $L_{DN}$), even though approximately 43 million in this group were estimated to have exposure levels of >65 dBA $L_{DN}$.

## Model 1: Coronary Heart Disease

The meta-analysis of Babisch et al.[4] on the relationship between road noise and CHD associated a 10-dB $L_{DN}$ increase in road traffic noise with an 8% increase in CHD. This measure is on the conservative side of the elasticities in Table 1. This effect size was selected because it covers all CHD, a more comprehensive measure than studies evaluating smaller categories of cardiovascular disease, such as myocardial infarction. The effect size is similar to the relationship between noise and CHD mortality found in Gan and colleagues[23] and the relationship between noise and hypertension found in van Kempen and Babisch.[20] This measure is also similar to or more conservative than that found in studies using different measures of noise (e.g., aircraft, rail, occupational, self-reported). The measure was halved to 4% as an estimate of the increase in CHD risk associated with a 5-dB $L_{DN}$ increase in environmental noise, to correspond with our hypothetical exposure reduction scenario.

By assigning the elevated relative risk (RR) of CHD of 1.04 to the 145.5 million Americans exposed to $\geq$55 dBA $L_{DN}$, the rate of CHD for those in the exposed group and those unexposed (Ru) was calculated using Equation 1:

$$CHD_c = (P_u \times R_u) + (P_e \times RR_e \times R_u) \quad (1)$$

where $CHD_c$ is the current number of cases of CHD (15.4 million, Table 2), $P_u$ is the population unexposed to high noise (169.5

**Table 2.** Key Assumptions

| Variable | Assumption | Source(s) | Notes |
|---|---|---|---|
| U.S. population 2013 | 315,000,000 | US Census Quickfacts[32] | |
| Number of Americans exposed to ≥55 dBA $L_{DN}$ of environmental noise | 145.5 million (46% of the total population) | Application of most recent EPA estimates[15] to current population | The EPA's 1981 exposure estimates are the most recent and best available information on the proportion of Americans exposed to high levels of environmental noise |
| Number of Americans exposed to ≥65 dBA $L_{DN}$ of environmental noise | 43.8 million (14% of the total population) | Application of most recent EPA estimates[14] to current population | The EPA's 1981 exposure estimates are the most recent and best available information on the proportion of Americans exposed to high levels of environmental noise |
| Relationship between environmental noise exposure and CHD | 1.04 RR per 5 dBA $L_{DN}$ | Babisch (2014)[18] | Babisch estimates 1.08 RR per 10 dBA $L_{DN}$, and we assume this estimate can be halved for our 5 dB LDN reduction scenario; we also assume that Babisch's estimates, which are for road noise only, can be applied to all environmental noise, as studies for other sources of environmental noise indicate similar relationships (Table 1) |
| Prevalence of CHD, U.S. | 15.4 million | Go et al. (2013)[12] | Figures are for 2010, estimated in 2013; no change assumed for 2013 |
| Population risk of CHD, U.S. | 4.89% | | 15.4 million Americans (Go et al. [2013][12]) of the 315 million population (U.S. Census[32]) |
| Direct cost of CHD, U.S. annual (health care, medications) | $96 billion | Lloyd-Jones et al. (2009)[13] | Figures are for 2009–2010, no change assumed for 2013 |
| Indirect cost of CHD, U.S. annual (lost productivity due to mortality/morbidity) | $81.1 billion | Lloyd-Jones et al. (2009)[13] | Figures are for 2009–2010, no change assumed for 2013 |
| Relationship between environmental noise exposure and hypertension | 1.034 RR per 5 dBA $L_{DN}$ | van Kempen and Babisch (2012)[20] | We assume that van Kempen and Babisch's estimated RR of 1.034 per 5 dBA $LA_{eq,16h}$ applies to 5 dB $L_{DN}$, because these measures are similar and tend to be highly correlated; we also assume that this estimate for road noise only can be applied to all environmental noise, as studies for other sources of environmental noise indicate similar relationships (Table 1) |
| Prevalence of hypertension, U.S. | 77.9 million | Go et al. (2013)[12] | Figures are for 2010, estimated in 2013; no change assumed for 2013 |
| Population risk of hypertension, U.S. | 24.7% | | 77.9 million Americans (Go et al. [2013][12]) of the 315 million population (U.S. Census[32]) |
| Direct cost of hypertension, U.S. annual (health care, medications) | $47.5 billion | Go et al. (2013)[12] | Figures are for 2009–2010, no change assumed for 2013 |
| Indirect cost of hypertension, U.S. annual (lost productivity due to mortality only) | $3.5 billion | Go et al. (2013)[12] | Figures are for 2009–2010, no change assumed for 2013 |

CHD, coronary heart disease; EPA, Environmental Protection Agency; RR, relative risk.

million), $R_u$ is the risk of CHD among $P_u$, $P_e$ is the population exposed to ≥55 dBA $L_{DN}$ (145.5 million), and $RR_e$ is the RR of CHD among $P_e$ (1.04). Solving Equation 1, the rate of CHD for those unexposed ($R_u$) is 4.8%, and for those exposed ($1.04*R_u$) is 4.99%.

In the assumed noise reduction scenario, the $P_e$ group experienced a 5-dB $L_{DN}$ reduction in noise exposure to 50 dBA $L_{DN}$, and the risk of CHD among this group reduced from 4.99% to 4.8%. The estimated number of cases of CHD in the exposure reduction scenario ($CHD_r$) was calculated using Equation 2:

$$CHD_r = (P_u \times R_u) + (P_e \times R_u) \quad (2)$$

where all variables are defined as in Equation 1.

**Table 3.** Model Results

| Model | Current situation | 5-dB reduction scenario estimate | Difference (current − reduction scenario) |
|---|---|---|---|
| Model 1: coronary heart disease | | | |
| Number of people exposed ≥55 dBA $L_{DN}$ | 145.5 million | 0 | −145.5 million |
| Number of affected individuals | 15.4 million | 15.1 million | −279,000 |
| Population risk (%) | 4.89 | 4.80 | −0.09 |
| Annual cost, direct ($) | 96 billion | 94.3 billion | −1.7 billion |
| Annual cost, indirect ($) | 81.1 billion | 79.6 billion | −1.5 billion |
| Model 2: hypertension | | | |
| Number of people exposed ≥ 55 dBA $L_{DN}$ | 145.5 million | 0 | −145.5 million |
| Number of affected individuals | 77.9 million | 76.7 million | −1.2 million |
| Population risk (%) | 24.7 | 24.3 | −0.4 |
| Annual cost, direct ($) | 47.5 billion | 46.8 billion | −684 million |
| Annual cost, indirect ($) | 3.5 billion | 3.4 billion | −50 million |

*Note:* Data sources and assumptions for "Current situation" explained in Table 2.

### Model 2: Hypertension

The meta-analysis of road traffic and hypertension conducted by Van Kempen and Babisch[20] estimated a RR of 1.034 for each additional 5 dB $LA_{eq,16h}$ of road noise exposure over a range of 45–75 dBA. This standard was applied to estimate the effect of environmental noise on hypertension in the U.S. Again, it was conservatively assumed that the 140 million Americans exposed to ≥55 dBA $L_{DN}$ were exposed at exactly 55 dBA $L_{DN}$. We also assumed that a 5-dB change in $LA_{eq,16h}$ is equivalent to a 5-dB change in $L_{DN}$ in our hypothetical exposure reduction scenario, as these measures are slightly different, but tend to be highly correlated.[18] The estimated RR of 1.034 per 5 dB $LA_{eq,16h}$ of noise exposure is conservative compared with other estimates in Table 1, namely Babisch and van Kamp,[25] which estimated an RR of hypertension of 1.13 per 10 dB $L_{DN}$, and Babisch,[27] which estimated an RR of hypertension of 1.4–2.1 per 10 dB increase in noise between 6AM and 10PM (the $L_{day}$ metric, nearly identical to the $L_{Aeq,16h}$).

Repeating the methodology from Model 1, the risks for hypertension among the exposed and unexposed population were calculated. The rate of hypertension for those unexposed ($R_u$) was 24.3%, and that for those exposed (1.034 × $R_u$) was 25.2% (Equation 1).

In the assumed noise reduction scenario, the noise-exposed group experienced a 5-dB $L_{DN}$ reduction in noise exposure to 50 dBA $L_{DN}$, and the risk of hypertension among this group reduced from 25.2% to 24.3% (Equation 2). Estimates of the number of hypertension cases in this scenario were generated.

### Sensitivity Analyses

Three analyses were performed: (1) ±20% the number of Americans exposed to high levels of noise; (2) ±20% RR; and (3) ±20% direct and indirect costs assumed for CHD and hypertension. Reductions in healthcare costs were estimated in proportion with reductions in prevalence of CHD and hypertension.

## Results

In the CHD model, the reduced exposure scenario ($CHD_r$) reduced CHD cases by 279,000 (1.8%), from 15.4 cases to 14.8 million cases (Table 3, Model 1). A corresponding 1.8% reduction in CHD costs would equate to annual savings of 1.8% of direct healthcare costs ($1.7 billion) and in 1.8% of indirect costs ($1.5 billion) from lost productivity.

The 5-dB $L_{DN}$ scenario reduced hypertension cases by 1.2 million (1.4%), from 77.9 million to 76.7 million (Table 3, Model 2). The associated 1.4% annual cost reduction equaled $684 million in direct healthcare costs and $50 million in indirect costs.

Table 4 provides a summary of the results of three sensitivity analyses. Because of the simplicity of the model, adjustments in each of three tests by 20% had similar impacts on the outcomes in terms of reduction in costs (roughly 20% reduction or increase in the overall outcome) and reduction of prevalence (roughly 20% reduction or increase).

## Discussion

This exploratory analysis evaluates the impact of environmental noise on two key components of cardiovascular health—CHD and hypertension. The results from Models 1 and 2 suggest that a 5-dB $L_{DN}$ reduction in environmental noise would reduce hypertension cases by 1.2 million and CHD cases by 279,000. The associated cost savings equal $2.4 billion annually in healthcare costs and $1.5 billion annually in productivity gains. Together, the estimated economic impact of the reduction scenario is more than $3.9 billion annually.

**Table 4.** Sensitivity Analyses

| Model | Reduction in prevalence | % reduction in prevalence | Reduction in direct and indirect costs (USD billions) | % reduction in direct and indirect costs |
|---|---|---|---|---|
| Model 1: coronary heart disease | | | | |
| Central estimate | 279,000 | 1.8 | 3.2 | 1.8 |
| +20%: U.S. population in the noise-exposed group | 334,000 | 2.2 | 3.8 | 2.2 |
| −20%: U.S. population in the noise-exposed group | 224,000 | 1.5 | 2.5 | 1.5 |
| +20%: risk ratio associated with exposed group | 334,000 | 2.2 | 3.8 | 2.2 |
| −20%: risk ratio associated with exposed group | 224,000 | 1.5 | 2.5 | 1.5 |
| +20%: direct and indirect costs | 279,000 | 1.8 | 3.9 | 1.8 |
| −20%: direct and indirect costs | 279,000 | 1.8 | 2.6 | 1.8 |
| Model 2: hypertension | | | | |
| Central estimate | 1.2 million | 1.4 | 0.7 | 1.4 |
| +20%: U.S. population in the noise-exposed group | 1.4 million | 1.7 | 0.9 | 1.7 |
| −20%: U.S. population in the noise-exposed group | 967,000 | 1.2 | 0.6 | 1.2 |
| +20%: risk ratio associated with exposed group | 1.4 million | 1.7 | 0.9 | 1.7 |
| −20%: risk ratio associated with exposed group | 967,000 | 1.2 | 0.6 | 1.2 |
| +20%: direct and indirect costs | 1.2 million | 1.4 | 0.9 | 1.4 |
| −20%: direct and indirect costs | 1.2 million | 1.4 | 0.6 | 1.4 |

This analysis underestimates the impact of environmental noise on cardiovascular disease in a number of ways. Impacts on only two significant components of cardiovascular disease, CHD and hypertension, have been estimated, and these account for less than half of the costs associated with cardiovascular disease.[12,13] With further research, the estimates could be applied to all cardiovascular disease, and the cost savings would likely be considerably larger.

These analyses represent the effect of noise exposure, and we believe that we have excluded confounding effects to the greatest extent possible. Many of the studies described in Table 1 account for demographic factors, other medical conditions, and other environmental factors (such as air quality) to attempt to isolate the impact of noise exposure on cardiovascular disease.

The threshold for the noise-exposed group was $\geq$55 dBA $L_{DN}$, though there is evidence in the literature that there may be important impacts at even lower levels of noise exposure.[16] Also, in the noise-exposed group, it was assumed that all individuals were exposed only at the minimal level for the group: 55 dBA $L_{DN}$.

Estimates for wider impacts, such as quality of life, were not included here, but would increase the benefit. For example, recent United Kingdom government guidance estimates that a decrease in environmental noise from 60 to 55 dBA $L_{Aeq,18hr}$ is worth £13 ($22) in quality of life from reduced myocardial infarction per noise-reduced household.[9]

Furthermore, these estimates are only illustrative of a portion of noise-related cardiovascular impacts, including only environmental noise exposure. Occupational noise exposure also has significant (and perhaps greater) cardiovascular impacts.[24,35]

Crude sensitivity analyses illustrate the impacts of changes in key model assumptions. Adjustments in each of the sensitivity tests by 20% have similar impacts on reduction in costs and prevalence (roughly 20% reduction or increase, except for reduction in cost, which has no impact on prevalence).

## Limitations

As a result of the paucity of current noise exposure estimates in the U.S., a number of assumptions were made in extrapolating from the 1981 EPA noise exposure

estimates.[15] Notably, assuming zero trend in noise levels is likely a conservative assumption, especially given increasing urbanization in the U.S.[17]

Another limitation of the analysis is that noise exposure metrics are often drawn from transportation research. Transportation is believed to be the primary source of environmental noise, and although different sources of noise can have different impacts on the listener, there is enough similarity in the documented cardiovascular disease/noise dose–response relationships that we elected to apply this transportation noise effect size to noise from any environmental source.

The use of $L_{DN}$ here is warranted given the demonstrated relationship between $L_{DN}$ and annoyance.[25] However, if a non–stress mediated pathway exists between noise exposure and cardiovascular disease, then the estimates presented here should be modified. Similarly, individual-level exposure variability due to recreational activities, occupational noise, listening to music, or other sources was not considered, and these activities may substantially increase noise exposure.[6,36]

## Conclusions

This study estimates that reducing environmental noise by 5 dB $L_{DN}$ would reduce hypertension cases by an estimated 1.2 million (1.4%) and CHD cases by 279,000 (1.8%). The associated cost savings and productivity gains are estimated to exceed $3.9 billion annually, demonstrating that environmental noise has significant economic ramifications.

This analysis is an important first step in estimating the economic and social costs of environmental noise exposure. Adding the benefits of reduced cardiovascular disease to cost–benefit analyses of proposed noise mitigation policies and investments (such as low-noise pavement, noise barriers, and active noise control) could appropriately enhance the economic valuation of these strategies, as the scale of the impacts estimated here make modest mitigation seem economically promising. This analysis also demonstrates that environmental noise exposures warrant further research and consideration in context with other environmental health priorities. Environmental noise exposure is a preventable risk factor for cardiovascular disease that is closely tied to community planning and government regulation, rather than personal risk factors, such as smoking, stress, and diet.[31,37]

Evidence is gathering on other non-auditory health impacts of noise, such as annoyance, sleep deprivation, childhood learning disruption, stress, and mental health,[1] and future estimates can address these impacts.

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## References

1. Goines L, Hagler L. Noise pollution: a modern plague. *South Med J*. 2007;100:287–294. http://dx.doi.org/10.1097/SMJ.0b013e3180318be5.
2. Willich SN, Wegscheider K, Stallmann M, Keil T. Noise burden and the risk of myocardial infarction. *Eur Heart J*. 2006;27:276–282. http://dx.doi.org/10.1093/eurheartj/ehi658.
3. Hammer MS, Swinburn TK, Neitzel RL. Environmental noise pollution in the United States: developing an effective public health response. *Environ Health Perspect*. 2014;122(2):115–119.
4. Basner M, Babisch W, Davis A, et al. Auditory and non-auditory effects of noise on health. *Lancet*. 2014;383:1325–1332. http://dx.doi.org/10.1016/S0140-6736(13)61613-X.
5. Heron M. Deaths: leading causes for 2010. *Natl Vital Stat Rep*. 2013;62:1–97.
6. Neitzel RL, Gershon RRM, McAlexander TP, Magda LA, Pearson JM. Exposures to transit and other sources of noise among New York City residents. *Environ Sci Technol*. 2012;46:500–508. http://dx.doi.org/10.1021/es2025406.
7. Flamme GA, Stephenson MR, Deiters K, et al. Typical noise exposure in daily life. *Int J Audiol*. 2012;51:S3–S11. http://dx.doi.org/10.3109/14992027.2011.635316.
8. DEFRA. *An Economic Valuation of Noise Pollution—Developing a Tool for Policy Appraisal*. London: DEFRA, 2008.
9. DEFRA. *Noise & Health—Valuing the Human Health Impacts of Environmental Noise Exposure*. London: DEFRA, 2010.
10. Cohen JP, Coughlin CC. Changing noise levels and housing prices near the Atlanta airport. *Growth Change*. 2009;40:287–313. http://dx.doi.org/10.1111/j.1468-2257.2009.00476.x.
11. Wilhelmsson M. The impact of traffic noise on the values of single-family houses. *J Environ Plan Manag*. 2000;43:799–815. http://dx.doi.org/10.1080/09640560020001692.
12. Go AS, Mozaffarian D, Roger VL, et al. Heart disease and stroke statistics—2013 update: a report from the American Heart Association. *Circulation*. 2013;127:e6–e245. http://dx.doi.org/10.1161/CIR.0b013e31828124ad.
13. Lloyd-Jones D, Adams RJ, Brown TM, et al. Heart disease and stroke statistics—2010 update: a report from the American Heart Association. *Circulation*. 2009;121:e46–e215. http://dx.doi.org/10.1161/circulationaha.109.192667.
14. U.S. Environmental Potection Agency. *Information on Levels of Environmental Noise Requisite to Protect Public Health and Welfare With an Adequate Margin of Safety*. Washington, DC: Environmental Potection Agency, Office of Noise Abatement and Control, 1974.
15. Simpson M, Bruce R. *Noise in America: Extent of the Noise Problem (Report 550/9-81-101)*. Washington, DC: Environmental Potection Agency, Office of Noise Abatement and Control, 1981.
16. WHO. *Night Noise Guidelines for Europe*. Copenhagen: WHO, 2009.
17. Garcia A. *Environmental Urban Noise*. Boston: WIT Press, 2001.
18. Babisch W. Updated exposure-response relationship between road traffic noise and coronary heart diseases: a meta-analysis. *Noise Health*. 2014;16:1–9. http://dx.doi.org/10.4103/1463-1741.127847.

19. Hansell AL, Blangiardo M, Fortunato L, et al. Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. *BMJ*. 2013;347:f5432. http://dx.doi.org/10.1136/bmj.f5432.
20. Van Kempen E, Babisch W. The quantitative relationship between road traffic noise and hypertension: a meta-analysis. *J Hypertens*. 2012;30: 1075–1086. http://dx.doi.org/10.1097/HJH.0b013e328352ac54.
21. Ndrepepa A, Twardella D. Relationship between noise annoyance from road traffic noise and cardiovascular diseases: a meta-analyses. *Noise Health*. 2011;13:251–253. http://dx.doi.org/10.4103/1463-1741.80163.
22. Sørensen M, Andersen ZJ, Nordsborg RB, et al. Road traffic noise and incident myocardial infarction: a prospective cohort study. *PLoS One*. 2012:7. http://dx.doi.org/10.1371/journal.pone.0039283.
23. Gan WQ, Davies HW, Koehoorn M, Brauer M. Association of long-term exposure to community noise and traffic-related air pollution with coronary heart disease mortality. *Am J Epidemiol*. 2012;175:898–906. http://dx.doi.org/10.1093/aje/kwr424.
24. Gan WQ, Davies HW, Demers P. Exposure to occupational noise and cardiovascular disease in the United States: the National Health and Nutrition Examination Survey 1999-2004. *Occup Environ Med*. 2011;68:183–190. http://dx.doi.org/10.1136/oem.2010.055269.
25. Babisch W, van Kamp I. Exposure-response relationship of the association between aircraft noise and the risk of hypertension. *Noise Health*. 2009;11:161–168. http://dx.doi.org/10.4103/1463-1741.53363.
26. Babisch W. Road traffic noise and cardiovascular risk. *Noise Health*. 2008;10:27–33. http://dx.doi.org/10.4103/1463-1741.39005.
27. Babisch W. Transportation noise and cardiovascular risk: updated review and synthesis of the epidemiological studies that indicate that the evidence has increased. *Noise Health*. 2006;8:1–29. http://dx.doi.org/10.4103/1463-1741.32464.
28. Kempen Eemm Staatsen B, van Kamp I. *Selection and Evaluation of Exposure-Effect Relationships for Health Impact Assessment in the Field of Noise and Health (RIVM Report 630400001/2005)*. Bilthoven, Netherlands: RIVM, 2005.
29. Van Kempen EEMM Kruize H, Boshuizen HC, Ameling CB, Statsen BAM. de Hollander AEM. The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. *Environ Health Perspect*. 2002;110:307–317. http://dx.doi.org/10.1289/ehp.02110307.
30. Passchier-Vermeer W, Passchier WF. Noise exposure and public health. *Environ Health Perspect*. 2000;108(suppl):123–131. http://dx.doi.org/10.2307/3454637.
31. Mozaffarian D, Wilson P, Kannel W. Contemporary reviews in cardiovascular medicine: beyond established and novel risk factors—lifestyle risk factors for cardiovascular disease. *Circulation*. 2008;117:3031–3038. http://dx.doi.org/10.1161/CIRCULATIONAHA.107.738732.
32. U.S. Census Bureau. U.S. Census: State and County QuickFacts. 2013. www.quickfacts.census.gov/qfd/states/00000.html.
33. Waitz I, Bernhard R, Hanson C. Challenges and promises in mitigating transportation noise. *Noise Eng*. 2007;37(3).
34. National Academy of Engineering. *Technology for a Quieter America*. Washington, DC: National Academies Press, 2010.
35. Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C, Demers PA. Occupational exposure to noise and mortality from acute myocardial infarction. *Epidemiology*. 2005;16(1):25–32. http://dx.doi.org/10.1097/01.ede.0000147121.13399.bf.
36. Neitzel R, Seixas N, Goldman B, Daniell W. Contributions of non-occupational activities to total noise exposure of construction workers. *Ann Occup Hyg*. 2004;48:463–473. http://dx.doi.org/10.1093/annhyg/meh041.
37. Perk J, Hambraeus K, Burell G, Carlsson R, Johansson P, Lisspers J. Study of Patient Information after percutaneous Coronary Intervention (SPICI): should prevention programmes become more effective? *EuroIntervention*. 2014. http://dx.doi.org/10.4244/EIJV10I11A223.