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Valuing Quiet: An economic assessment of US environmental noise as a cardiovascular health hazard

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Abstract

Introduction—Environmental noise pollution increases the risk for hearing loss, stress, sleep disruption, annoyance, cardiovascular disease, and has other adverse health impacts. Recent (2013) estimates suggest that over 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 significant.

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 US. 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 are estimated, with the caveat that the national-level US noise data our exposure estimates were derived from are >30 years old.

Results—The analyses suggest 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 is 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 of 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 >100 million Americans are exposed to levels of environmental noise that put them at risk for hearing loss,³ stress, sleep disruption, and annoyance, which over time increases risk of cardiovascular disease.¹ A 10 dB L_{Aeq} decrease in environmental noise exposure (about twice the decrease in noise around airports after federal regulations) decreases risk of cardiovascular disease 7–17%.⁴ Cardiovascular disease is the top cause of mortality in the United States (US)⁵ and presents a substantial health-related economic burden. The ubiquitous nature of environmental noise in urban⁶ and non-urban⁷ areas of the US 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 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 US, 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 (over 83 million) and accounting for 15% of health care 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 2010¹³ and cost \$47.5 billion in treatment.¹² Some individuals are affected by both CHD and hypertension, which we account for in this analyses).

Prevalence of harmful noise exposure

Environmental impacts of noise are assessed with a sound level meter that measures decibel levels (dB) 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 US Environmental Protection Agency (EPA) recommended an average 24-hour exposure limit of 55 A-weighted decibels (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 (11 PM – 7 AM) to account for potential sleep disturbance. We have 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_{AEQ16h}) 7 AM – 11 PM. For context, 55 dBA is approximately as loud a microwave oven, and a 24-hour L_{DN} exposure of 55 dBA could result from 8 hours in a typical large office environment (55 dBA), 1-hour lunch in a restaurant (60 dBA), 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} or greater from environmental noise, and 13.9% were exposed to 65 dBA L_{DN} .¹⁵ Though dated (and considerably different from recent World Health Organization (WHO) guidance),¹⁶ this 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 US population. Among those exposed to 58 dBA L_{DN} , traffic and/or aircraft was the source of exposure for the vast majority, while rail, construction, and domestic appliances were also considered.¹⁵

We make the conservative assumption that the proportion of the US population exposed to high levels of noise is 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 US.¹⁷ In 2013, an estimated 46.2% of Americans (145.5 million people) were exposed to 58 dBA L_{DN} or greater, and 13.9% (43.8 million people) were exposed to 65 dBA L_{DN} or greater. 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 al³ 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,⁴ but 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 (transport noise, occupational noise, self-reported noise annoyance) and different cardiovascular disease indicators (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 et al⁴ summarizes, the risk of hypertension or CHD increases 7–17% per 10 dB L_{Aeq} increased noise exposure.

Assessment of economic impact of noise-related CHD and hypertension

Drawing assumptions from the assessments above, the following model estimates 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). A 5 dB reduction in annual L_{DN} appears feasible based on demonstrated and ongoing reductions in aircraft noise following federal regulation,³³ and is modeled on similar regulatory approaches for air contaminants, which can include technology-forcing policies. A 5 dB reduction could be achieved through a multi-prong 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 assume is a conservative underestimate of those exposed to 55 dBA L_{DN} . It is 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 are estimated to have exposure levels of >65 dBA L_{DN} .

Model 1: Coronary Heart Disease—Babisch and colleagues' meta-analysis 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.⁴ This measure is on the conservative side of the elasticities in Table 1. The Babisch 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 et al²³ 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 (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 of CHD of 1.04 to the 145.5 million Americans exposed to 55 dBA L_{DN} or greater, the rate of CHD for those in the exposed group and those unexposed (R_u) was calculated using equation 1:

$$CHD_c = (P_u * R_u) + (P_e * RR_e * R_u) \quad (\text{Eq 1})$$

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 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 relative risk 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:

$$CHD_r = (P_u * R_u) + (P_e * R_u) \quad (\text{Eq 2})$$

where all variables are defined as in equation 1.

Model 2: Hypertension—Van Kempen and Babisch's meta-analysis of road traffic and hypertension estimated a relative risk of 1.034 for each additional 5 dB $LA_{eq}16hr$ of road noise exposure over a range of 45–75 dBA.²⁰ This standard is applied to estimate the effect of environmental noise on hypertension in the US. Again, it is conservatively assumed that the 140 million Americans exposed to 55 dBA L_{DN} are exposed at exactly 55 dBA L_{DN} . We also assume that a 5 dB change in $LA_{eq}16hr$ 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 relative risk of 1.034 per 5 dB $LA_{eq}16hr$ of noise exposure is conservative compared to other estimates in Table 1, namely Babisch and van Kamp,²⁵ which estimated a relative risk of hypertension of 1.13 per 10 dB L_{DN} , and Babisch,²⁷ which estimated a relative risk of hypertension of 1.4–2.1 per 10 dB L_{day} .

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) is 24.3%, and for those exposed ($1.034 * R_u$) is 25.2% (Eq 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% (Eq 2). Estimates of the number of hypertension cases in this scenario were generated.

Sensitivity analyses—Three analyses were performed: 1) $\pm 20\%$ the number of Americans exposed to high levels of noise; 2) $\pm 20\%$ relative risk, and; 3) $\pm 20\%$ 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

Model 1: Coronary Heart Disease

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.

Model 2: Hypertension

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.

Sensitivity Analyses

Table 4 provides a summary of the results of three sensitivity analyses performed. 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 health care costs and \$1.5 billion annually in productivity gains. Together, the estimated economic impact of the reduction scenario is over \$3.9 billion annually.

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 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 UK government guidance estimates 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 illustrated the impacts of changes in key model assumptions. Adjustments in each of the sensitivity tests by 20% had similar impacts on reduction in costs and prevalence (roughly 20% reduction or increase, except for reduction in cost which had no impact on prevalence).

Limitations

As a result of the paucity of current noise exposure estimates in the US, 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 US.¹⁷

Another limitation of the analysis is that noise exposure metrics are often drawn from transportation research. Transportation is believed to be the majority 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 the 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, etc. 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, 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 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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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 Air traffic	Hypertension Ischemic heart disease	10 dB L _{Aeq} increase of environmental noise increases risk of hypertension or ischemic heart disease (IHD) 7–17%.
Hansell et al (2013) ¹⁹	3.6 million residents near Heathrow Airport, UK	Air traffic Daytime and nighttime noise separate Modeled noise exposure	Hospital admissions and mortality from: Stroke CHD cardiovascular disease	Daytime aircraft noise resulted in statistically significant increased risk of hospital admission for stroke (1.24 relative risk), cardiovascular disease (1.14 RR) and CHD (1.21 RR). Relative risks 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 _{Aeq16hrs} increase in noise exposure increases risk of hypertension by 3.4% over a range of 45–75 dBA
Ndrepepa & Twardella (2012) ²¹	Meta-analysis of 8 studies	Self-reported annoyance from road traffic	Hypertension Ischemic heart disease	Noise annoyance significantly, positively associated with hypertension (1.16 pooled risk estimate). Noise annoyance significantly, positively associated with ischemic heart disease (1.07 pooled risk estimate).
Sørensen et al (2012) ²²	57,053 residents of Copenhagen or Aarhus, Denmark	Road traffic Modeled noise exposure	Myocardial infarction	10dB 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 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	6307 participants in the US NHANES survey	Self-reported occupational noise exposure	Angina pectoris CHD Hypertension Myocardial infarction	Exposure to loud noise in the workplace significantly and positively associated with all cardiovascular disease measures: angina pectoris (2.91 odds ratio), CHD (2.04 odds ratio), hypertension (2.23 odds ratio).
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 relative risk) over the range 45–70 dBA.

Publication	Source of data	Noise source(s)	Cardiovascular disease metric	Relationship and metric(s)
Babisch (2008) ²⁶	Meta-analysis of 7 studies	Road traffic	Myocardial infarction	Noise exposure levels above 60 dBA $L_{day, 16h}$ significantly associated with increased risk of MI. For noise levels above 70 dBA $L_{day, 16h}$ odds ratio >1.2. No increased risk found at noise levels below 60 dBA $L_{day, 16h}$.
Babisch (2006) ²⁷	Literature review/meta-analysis	Air traffic Road traffic Rail traffic	Ischemic heart disease 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 relative risk). 60–70 dBA L_{day} noise exposure associated with increased risk of hypertension (1.4 – 2.1 relative risk), though earlier studies indicated a less clear relationship.
van Kempen & Staatsen (2005) ²⁸	The Netherlands	Air traffic 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).
Willich et al (2006) ²	4115 patients admitted to hospitals in Berlin	Self-reported environmental and occupational annoyance Road traffic Rail traffic Modeled noise exposure	Myocardial infarction	Environmental noise annoyance slightly significantly positively associated with MI in women; no association for men. Environmental noise associated with increased risk of MI in men (1.46 odds ratio) and women (3.36 odds ratio). Occupational noise associated with increased risk of MI in men (1.31 odds ratio) but not in women.
van Kempen et al (2002) ²⁹	Meta-analysis of more than 500 studies 1970–1999 in English, German, or Dutch	Occupational exposure Road traffic Rail traffic	Blood pressure	5 dBA L_{Aeq} occupational noise exposure significantly associated with increased hypertension (1.14 relative risk). 5 dBA L_{Aeq} air traffic noise exposure significantly associated with increased hypertension (1.26 relative risk) (this based on just 1 study).
	Meta-analysis of more than 500 studies 1970–1999 in English, German, or Dutch	Road traffic Rail traffic	Ischemic heart disease	Community noise positively associated with MI, ischemic heart disease, angina pectoris, use of cardiovascular medicines, but none of these measures were statistically significant
Passchier-Vermeer & Passchier (2000) ³⁰	Literature review	Mainly road traffic, one air traffic study	Hypertension Ischemic heart disease	Sufficient evidence for a noise exposure related effect above 70 dBA L_{DN} .

Key Assumptions

Table 2

Variable	Assumption	Source(s)	Notes
US population 2013	315,000,000	US Census Quickfacts ³²	
Number of Americans exposed to 55 dBA L _{DN} or greater 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} or greater 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 relative risk per 5 dBA L _{DN}	Babisch 2014 ¹⁸	Babisch estimates 1.08 relative risk 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, US	15.4 million	Go et al 2013 ¹²	Figure for 2010, estimated in 2013, no change assumed for 2013.
Population risk of CHD, US	4.89%		15.4 million Americans (Go et al 2013 ¹²) of the 315 million population (US Census ³²)
Direct cost of CHD, US annual (health care, medications)	\$96 billion	Lloyd-Jones et al ¹³	Figures are for 2009–2010, no change assumed for 2013
Indirect cost of CHD, US annual (lost productivity due to mortality/morbidity)	\$81.1 billion	Lloyd-Jones et al ¹³	Figures are for 2009–2010, no change assumed for 2013
Relationship between environmental noise exposure and hypertension	1.034 relative risk per 5 dBA L _{DN}	van Kempen and Babisch 2012 ²⁰	We assume that van Kempen and Babisch's estimated relative risk of 1.034 per 5 dBA L _{A,eq} 16hr 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, US	77.9 million	Go et al ¹²	Figure for 2010, estimated in 2013, no change assumed for 2013
Population risk of hypertension, US	24.7%		77.9 million Americans (Go et al 2013 ¹²) of the 315 million population (US Census ³²)
Direct cost of hypertension, US annual (health care, medications)	\$47.5 billion	Go et al ¹²	Figures are for 2009–2010, no change assumed for 2013
Indirect cost of hypertension, US annual (lost productivity due to mortality only)	\$3.5 billion	Go et al ¹²	Figures are for 2009–2010, no change assumed for 2013

Table 3

Model Results

	Current situation	5 dB reduction scenario estimate	Difference (current – reduction scenario)
Model 1: Coronary Heart Disease			
N people exposed 55 dBA L _{DN}	145.5 million	0	–145.5 million
N affected individuals	15.4 million	15.1 million	– 279,000
Population risk	4.89%	4.80%	– 0.09 % points
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			
N people exposed 55 dBA L _{DN}	145.5 million	0	–145.5 million
N affected individuals	77.9 million	76.7 million	– 1.2 million
Population risk	24.7%	24.3%	– 0.4 % points
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.

Table 4

Sensitivity Analyses

	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% : US population in the noise exposed group	334,000	2.2%	3.8	2.2%
-20% : US 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% : US population in the noise exposed group	1.4 million	1.7%	0.9	1.7%
-20% : US 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%