NRDC
THE EARTH'S BEST DEFENSE

NATURAL RESOURCES DEFENSE COUNCIL

March 5, 2013

U.S. Coast Guard
Docket Management Facility (M-30)
U.S. Department of Transportation
West Building Ground Floor, Room W12-140
1200 New Jersey Avenue SE.
Washington, DC 20590-0001

Submitted via the Federal eRulemaking Portal: http://www.regulations.gov

Re: Comments on the Draft Environmental Assessment for the Bayonne Bridge Navigational Clearance Program, Docket # USCG-2012-1091

To whom it may concern:

These comments are written on behalf of the Natural Resources Defense Council ("NRDC"), and our over 565,000 members, 54,691 of whom live in New York and New Jersey. NRDC has a long history of working to improve air quality, particularly in communities impacted by freight transportation operations. We have commented on numerous environmental studies produced in connection with the National Environmental Policy Act ("NEPA"), including studies for port initiated projects. We thank you for the opportunity to provide these comments, and for your decision to extend the comment deadline and add a public hearing in response to requests made by community and environmental justice groups in Newark, New Jersey.

As you are aware, NEPA requires federal agencies to prepare an environmental impact statement ("EIS") for all "major Federal actions significantly affecting the quality of the human environment." 42 U.S.C. § 4332(C). The Coast Guard's issuance of a Section 9 permit pursuant to the Rivers and Harbors Act of 1899 is a "major Federal action" to which NEPA applies. The Coast Guard does not contest that the Port of NY/NJ's (the "Port") permit application triggers NEPA.

Prior to preparing an EIS, "federal agencies often must prepare an Environmental Assessment ('EA') when determining whether a project is one that will significantly affect the environment and require an EIS." *Fund for Animals v. Babbitt*, 89 F.3d 128, 130 (2nd Cir. 1996). An EA serves to help the lead agency determine if the federal action may significantly affect the environment. 40 C.F.R. § 1508.9. If the project may significantly affect the environment, then "[u]nder NEPA, federal agencies must prepare an Environmental Impact Statement ('EIS') assessing the

www.nrdc.org
1314 Second Street
Santa Monica, CA 90401
TEL 310 434-2300
FAX 310 434-2399
100% Postconsumer Recycled Paper
NEW YORK • WASHINGTON, DC • SAN FRANCISCO • BEIJING • CHICAGO

beneficial and adverse environmental impacts . . . that significantly affects the quality of the human environment." *Fund for Animals v. Babbitt*, 89 F.3d at 130.

Ultimately, NEPA has a dual purpose: it serves to inform decision making and disclose information to the public about how a federal action will affect the environment and public health. 40 C.F.R. § 1500.1(b)–(c); *Marsh v. Or. Natural Res. Council*, 490 U.S. 360, 371 (1989) ("NEPA ensures that the agency will not act on incomplete information, only to regret its decision after it is too late to correct."). Here, the Draft Environmental Assessment ("DEA") for the Bayonne Bridge Navigational Clearance Program (the "Project") fails in all of these respects. For instance:

1. The DEA's Induced Demand Analysis (Appendix I) concludes that the Project will induce only 0.7% of the Port's forecasted cargo volumes for 2035. This conclusion is contradicted by the Port's own statements and representations made in the DEA. The analysis is also based on unsupported assumptions. We believe the Project will account for as much as 34% of the expected cargo volumes at the Port in 2035.

2. The DEA relies on an inflated baseline that assumes previously projected levels of cargo volumes will occur with or without the Project. We believe that absent the project (no build scenario), the Port's cargo volumes would be 25% lower than what is projected for 2035.

3. The DEA's flawed baseline and Induced Demand Analysis infects the entire DEA by underestimating cargo throughput attributable to the Project as well as the environmental impacts associated with that cargo, including impacts to air quality, environmental justice communities, and cumulative impacts.

4. Even if the DEA's Induced Demand Analysis were correct, the DEA minimizes air quality impacts for environmental justice communities by making false assumptions about how cargo leaves the Port, including that the overwhelming majority of cargo moved west of the bridge moves by rail as opposed to diesel trucks.

5. The DEA fails to include a meaningful analysis of how the Project may create significant cumulative air quality and environmental justice impacts. The DEA inappropriately concludes that if the Project—in isolation—creates less than significant impacts that those impacts somehow cannot be cumulatively significant.

6. The DEA fails to take a hard look at how construction of the project has the potential to expose the residents of Bayonne, NJ and Staten Island,

New York to lead, PCBs, asbestos, arsenic, and radioactive waste. Construction of the Project will occur at or close to little league fields, parks, and a public school. The DEA reveals that the consultant hired to analyze the potential risk from hazardous contaminants was specifically instructed not to study risks created by adjacent sites or speak to local, state, or federal regulatory agencies about the potentially affected sites.

7. The DEA fails to take a hard look at how noise from the construction of the project, which will last for up to 20 months at any one location and occur in the residential communities of Bayonne, NJ and Staten Island, NY, will affect health and quality of life. Specifically, the DEA fails to report what the noise levels will be absent mitigation, and improperly avoids assessing the significance of those levels.

8. To the extent that the Coast Guard intends for mitigation to be adopted to reduce potentially significant impacts from hazardous contaminants and construction noise, the agency fails to provide the requisite analysis that the mitigation will in fact be effective at reducing significant impacts to less than significant levels.

9. The Coast Guard violated NEPA by failing to perform an EIS in the face of potentially significant impacts from the Project.

Below, we outline each of our concerns in greater detail. In support of this letter, we have sent, by federal express, copies of expert reports and other documents to support our arguments.[1]

I. THE DEA'S INDUCED DEMAND ANALYSIS IS FATALLY FLAWED

In assessing the environmental impact of the Project, the Coast Guard must consider all of the project's "[d]irect effects, which are caused by the action and occur at the same time and place" and "[i]ndirect effects, which are caused by the action and are later in time or farther removed in distance, but are still reasonably foreseeable." 40 C.F.R. § 1508.8. "Indirect effects may include growth inducing effects and other effects related to induced changes in the pattern of land use, population density or growth rate, and related effects on air and water and other natural systems, including ecosystems." *Id*.

[1] It was not reasonably possible to upload all of our supporting materials with our comments on the Federal eRulemaking Portal website http://www.regulations.gov. Thus, we are separately sending hard copies of attachments 1–97, including an index of the documents, which arrived before the March 5, 2013 comment deadline on March 4. The index to the documents sent via Fedex is also attached to this letter. Also attached herein is attachment 98.

We are very disappointed with the analysis conducted by the Coast Guard in Appendix I: Induced Demand Analysis, which concludes that the Project will induce only 0.7% of the forecasted cargo volumes at the Port. Not only are the conclusions within that analysis contradicted by the Port's own statements and representations made in the DEA, but the analysis itself is riddled with unsupported assumptions. As we discuss below, the DEA underestimates the cargo throughput attributable to the Project as well as any environmental impacts associated with that cargo. Stated differently, the DEA's faulty Induced Demand Analysis infects the entire DEA.

As discussed below, the DEA's Induced Demand Analysis is flawed in several significant ways. First, the analysis is contradicted by the Port's own statements and defies fundamental economic principles. Second, the analysis relies on an artificially inflated baseline (or no build alternative) that assumes that the Port will meet its cargo forecasts even if the bridged is not raised. By inflating the baseline, the DEA minimizes the cargo volumes attributable to the Project and obscures the intensity of the impact of the Project on environmental health. Third, the assumptions relied upon to conclude that the Project will result in only a 0.7% increase in cargo throughput are unsupported. Based on our analysis, the Project could generate as much as 34% more cargo at the Port in 2035.

A. The Port's Own Statements Demonstrate That The DEA Severely Underestimates The Growth Enabled By The Project

The Port has represented that raising the Bayonne Bridge is a critical infrastructure project that will enable the Port to remain competitive with other ports, capture business after the Panama Canal expansion is completed in 2015, and create new jobs. The Port has made these representations many times.

In its TIGER Grant Application, the Port based its request for $3 million for the Project on the assertion that:

> Increasing the air draft restriction of the Bayonne Bridge is crucial for maintaining and developing the regional economies of New York and New Jersey. The existing Bayonne air draft restriction may damage the economies of New York and New Jersey, as shipping companies will be encouraged to divert to ports capable of handling larger, economically efficient vessels.

TIGER Grant App. at 3. The Port goes on to conclude:

> Given existing Bayonne clearance restriction, the potential that post Panamax vessels will not be able to call at the Port of New York and New Jersey, and they could divert to ports outside of the region that are able to accommodate these vessels, may result in a loss of

> economic activity in the region. Improving the air draft restriction will ensure that New York and New Jersey remain capable of handling their shipping needs for years to come, by maintaining and expanding local business access to markets. Additionally . . . enabling larger vessels to reach the ports in Newark and Elizabeth will result in economies of scale with regards to shipping costs, thereby reducing shipping costs and providing a boost to the local economy . . .

Id. at 5.

The Port made similar representations in early 2012, when it asked the President to "fast track" the Project: "Raising the bridge roadway is crucial to maintaining the Port's position as the third largest port in the country. Moreover, the project has significant national impact on freight and goods movement since the [Port] accounts for 40% of east coast container imports." Letter from Patrick J. Foye, Executive Director, The Port Authority of NY & NJ, to the Honorable Janet Napolitano, Secretary, U.S. Department of Homeland Security, and the Honorable Ray LaHood, Secretary, U.S. Department of Transportation (March 23, 2013) from Port at 2.[2]

The DEA's purpose and need also supports the theory that the Project is necessary to secure projected cargo volumes. The DEA states:

> The project would also remove potential impediments to marine transport along the Kill Van Kull to adapt to changes in the shipping industry and ensure the long-term vitality and efficiency of the Port . . . . [L]osing these efficiencies and shipping cost reductions would make it more difficult for the Port to compete with other ports serving the margins of the Port's outer hinterland.

---

[2] *See also* Tori-Ann Cerbo, *Port Authority approves $25 million for planning of raising Bayonne Bridge's roadbed*, THE JERSEY JOURNAL, May 27, 2011, *available at* http://www.panynj.gov /bayonnebridge/pdf/052711Portroadbed.pdf; Steve Strunsky, *How a $1B lift will give Bayonne Bridge a boost*, STAR-LEDGER, Jan. 26, 2011, *available at* http://www.panynj.gov/bayonnebridge/pdf/ 012611howboost.pdf; Steve Strunsky, *Raising Bayonne Bridge makes way for new ships*, STAR-LEDGER, Dec. 30, 2010, *available at* http://www.panynj.gov/ bayonnebridge/pdf/123010raisingships.pdf; Peter Leach, *NY-NJ Port Expedites Bayonne Bridge Project*, JOURNAL OF COMMERCE, July 18, 2012, *available at* http://www.joc.com/maritimenews/international-freight-shipping/ny-nj-port-expedites-bayonne-bridgeproject_20120718.html; Joseph Bonney, *NY-NJ Port to Announce Bayonne Bridge Plan*, JOURNAL OF COMMERCE, Dec. 3, 2010, *available at* http://www.joc.com/maritime-news/ny-njport-announce-bayonne-bridge-plan_20101203.html.

DEA at 1-1, 1-2.

The Coast Guard's September 2011 NEPA Workplan for this Project echoed similar concerns: "The purpose of the project is to ensure the long-term vitality of the Port of New York and New Jersey . . . When larger ships are able to call on the Panama Canal, the height restriction at the Bayonne Bridge will limit the opportunity for the [Port] to attract shipping interests and to realize economic benefits for the region." Bayonne Bridge Navigation Clearance Project: NEPA Workplan (Sept. 2011) at 1-1, 1-5.

Further, in connection with the U.S. Army Corps of Engineers' data gathering for the Bayonne Bridge Air Draft Analysis, the Army Corps found that a failure to raise the bridge may affect decisions about which ports shippers use: "Eleven of the 15 carriers interviewed say that they may need to bypass the PONYNJ in the future if the Bayonne Bridge remains a restriction." U.S. Army Corps of Engineers, Bayonne Bridge Air Draft Analysis, at 26–27 (Sept. 2009) ("BBADA").

The Port's Consortium Port of New York and New Jersey Comprehensive Port Improvement Plan ("CPIP"), which is cited many times in the DEA, also states that the current air draft restriction is a "risk factor" that could affect the port's long term competitiveness. DEA at 18-10.

Common sense dictates that if the Port's "vitality" and "competitiveness" is at risk if the bridge is not raised, then, without the Project, the Port may not secure its projected cargo volumes and could in fact lose freight volumes at a significant rate. Yet, the baseline/no-build scenario in the DEA assumes the exact opposite. We discuss this more fully below.

B. The DEA Relies On An Inflated Baseline That Minimizes The Intensity Of The Project

An accurate baseline is necessary to determine the "intensity" of an action for purposes of determining "significance." *See Ctr. for Biological Diversity v. Provencio*, 2012 WL 966031, at *17 (D. Ariz. Jan. 23, 2012). If the lead agency fails to use the correct baseline, it may underestimate the effects of the Project, and incorrectly forego preparation of an EIS. The baseline is reported as conditions within the no build alternative. NEPA Workplan at 2–1 ("The No Action Alternative serves as a baseline for comparison to the other alternatives."). The baseline may not assume the existence of the Project.

> NEPA requires that an agency's alternatives analysis include a "no build" alternative. 40 C.F.R. § 1502.14(d). "Without [accurate baseline] data, an agency cannot carefully consider information about significant environment impacts . . . resulting in an arbitrary and capricious decision." *See N. Plains Res. Council, Inc. v. Surface*

> *Transp. Bd.*, 668 F.3d 1067, 1085 (9th Cir.2011). Accordingly, courts not infrequently find NEPA violations when an agency miscalculates the "no build" baseline or when the baseline assumes the existence of a proposed project. *See, e.g., Friends of Yosemite Valley v. Kempthorne*, 520 F.3d 1024, 1037–38 (9th Cir.2008); *N.C. Alliance for Transp. Reform, Inc. v. U.S. Dep't of Transp.*, 151 F.Supp.2d 661, 690 (M.D.N.C.2001).

N. Carolina Wildlife Fed. v. N. Carolina Dep't of Transp., 677 F.3d 596, 603 (4th Cir. 2012). The DEA violates NEPA because it relies on an inflated baseline, and thus, fails to accurately report the environmental effects of the "no build alternative" and Project as a whole. The DEA summarizes projected TEU volumes west of the bridge in 2020 and 2035 with and without the Project. DEA at 18-12. Total TEUs under the build and no build scenarios are equal in both forecast years. In other words, the baseline assumes that Port cargo volumes will be the same with or without the Project. The DEA rationalizes that this is the case because even if the bridge is not raised, shippers will continue to call at the Port at the same rate using vessels with lower TEU capacity and smaller keel to mast heights.

The DEA's rationalization is undermined by the Port's CPIP, which shows that design draft restrictions, which also limit the size of ships that can access the Port, can significantly reduce overall throughput at the Port, especially when other ports are undergoing simultaneous improvements.[3] Contrary to the assertions in the DEA, the CPIP supports the assertion that shippers desiring to use post-Panamax vessels are largely projected to call at ports that can accommodate them.

Specifically, as documented in our expert report authored by Sustainable Systems Research, LLC ("SSR Report"), we argue that channel depth restrictions are conceptually similar to the air draft restriction imposed by the bridge, and use data obtained from the DEA and CPIP to conclude that while the Port may continue to see an increase in cargo volumes if the bridge is not raised based in part on a shift to use of smaller vessels, those increases may not be as great as projected in the DEA. Sustainable Systems Research, Technical Memorandum: Review of the Bayonne Bridge Navigational Clearance Program Draft Environmental Assessment (March 1, 2013) ("SSR Report") at 15 *et seq*. Our analysis demonstrates that cargo volumes under the baseline scenario (no build conditions) could be 25% lower than forecast in the DEA for 2035, and that the Project could enable cargo volumes 1 to 2 orders of magnitude greater than what is reported in the DEA. *Id.* at 23. Specifically,

---

[3] The DEA highlights that "[p]orts all along the Eastern Seaboard are undertaking expansion and improvement projects to meet ongoing demand and in anticipation of greater reliance on Post-Panamax vessels." DEA 18-10 n.1 (listing improvement projects that benefit ports in Wilmington, Philadelphia, Miami, Savannah, Charleston, Baltimore, and Jacksonville).

cargo volumes west of the bridge in the build scenario could be 44% higher than in the no build/baseline scenario, which translates to 34% Port-wide if we conservatively assume that 20% of Port volumes move through terminals east of the bridge in 2035 and that those terminals experience no change in cargo volumes. *Id.*

C. The DEA's Induced Demand Analysis Is Unsupported

The DEA concludes that

> [The] total potential induced demand at the Port from the project would be approximately 92,400 TEUs, or 74,000 TEUs (80 percent) at terminals west of the Bayonne Bridge. This would be a minimal increase in cargo at the Port (less than one percent) from the 10.65 million TEUs estimated by the USACE's Bayonne Bridge Air Draft Analysis without the project, thereby having negligible impacts on global shipping patterns.

DEA at 18-16. In support of this conclusion, the DEA's Induced Demand Analysis relies on undefined "price elasticities," "costs," and "expected values," all of which feed into the DEA's conclusion that the Project results in only 0.7% additional cargo at the Port. However, the DEA never demonstrates why its chosen variables are suitable for computing cargo volumes from the Project. In other words, no discussion is provided whatsoever to explain, let alone, justify the minimal 0.7% increase in cargo the DEA attributes to the Project. Without such information, there is no way to verify (or support) the Coast Guard's Induced Demand Analysis.[4] Further, the DEA's Induced Demand Analysis appears to omit important cost information as well as the effect of competition from certain ports. SSR Report at 10–14. Accordingly, even if the Coast Guard rejects the SSR Report and the alternative induced growth numbers provided therein, it may not support the Induced Demand Analysis in the DEA.

The SSR Report starting at page 7 details flaws within the DEA's induced demand model.

---

[4] The Eastern Environmental Law Center asked for this data on at least 4 occasions. Moreover, it is surprising that the Coast Guard would release its Induced Demand Analysis without supporting data in light of the fact that commenters, including U.S. EPA, were skeptical of the Coast Guard's early position that the Project would not result in significant cargo throughput and criticized the agency's failure to conduct an induced growth analysis. U.S. Coast Guard, First Coast Guard District Bridge Program, Bayonne Bridge Navigational Clearance Program, Responses to Scoping Comments, NEPA Workplan, at 5, 12, 17, 26, 27, 28 (Feb. 2012).

D. The DEA's Reliance On An Inflated Baseline And Unsupported Induced Growth Analysis Infects The Entire DEA, Rendering The DEA Meaningless

Every chapter of the DEA (with the exception of construction) relies on the conclusion that the Project will only minimally affect cargo volumes. As a result, the DEA reports that the Project does not have the potential to create significant effects on e.g., air quality, traffic, cumulative effects, and environmental justice. If the DEA's Induced Demand Analysis is flawed, however, nearly every conclusion in the DEA becomes suspect. Additionally, if the Project will enable cargo on the order of one to two magnitudes greater than estimated in the DEA—as we predict—the Project will have potentially significant environmental impacts. This would require the Coast Guard to enlarge the geographic scope of its NEPA analysis, and reassess the Project's environmental effects.

For instance, the DEA is limited to an analysis of how construction of the Project will affect Bayonne, NJ and Staten Island, NY. The DEA did not examine how, for example, neighborhoods in Newark, NJ, which experience significant amounts of traffic from port-serving trucks, would be affected. The DEA presumably omitted Newark from its analysis based on its conclusion that the Project would only marginally increase cargo volumes and hence truck traffic in Newark.

Based on our induced growth estimates, however, peak truck trips leaving the Elizabeth Terminal would range from 174–739 trips/hour, while at Newark the estimated range is 40–168 peak truck trips/hour, and at Howland Hook it is 31–132 peak truck trips/hour. SSR Report Table 9 at 31.[5] These projected truck trips are significantly greater than the 1–2 additional trips per hour from each of these terminals that the DEA predicted would be generated by cargo operations west of the bridge. DEA at 18-17. This increase in truck trips will be accommodated by an increase in air pollution impacts, as well as potentially significant cumulative and environmental justice impacts.

II. THE DEA'S ANALYSIS OF AIR QUALITY IMPACTS IS INADEQUATE AND REVEALS POTENTIALLY SIGNIFICANT IMPACTS

Below, we articulate the public health consequences of exposure to diesel exhaust and why those impacts are of particular concern in the context of the Project at issue. We also illustrate how the DEA underestimates how much air pollution will be generated by the Project, individually and in connection with other past, present and reasonably foreseeable projects, and creates environmental justice impacts. We

[5] These figures assume the use of different mode split assumptions than those utilized in the DEA. As discussed in the SSR Report pages 24–27 and *infra*, the DEA fails to accurately account for how cargo leaves the Port by, for example, assuming that 80% of cargo west of the bridge leaves by rail rather than by truck.

conclude by urging the Coast Guard to revise its air quality, cumulative impacts, and environmental justice impacts analysis, in addition to preparing a health risk assessment for the Project. We also request that a health impact assessment be prepared by an independent consultant.

A. Port Operations Generate Diesel Exhaust, Which Adversely Affects Public Health

Most of the equipment used to transport freight, including trucks, trains, ships, and cranes, are powered by diesel engines. These engines emit fine particulate matter ("PM"), nitrogen oxides ("NOx"), and volatile organic compounds ("VOCs"), along with many other toxic air pollutants. Numerous studies have documented a wide range of adverse health impacts from exposure to PM, including increased rates of respiratory illness and asthma, cardiovascular disease, heart attacks, strokes, emergency room visits, and premature death.[6] Exposure to PM has also been linked to birth defects, low birth weights, and premature births.[7] Emerging studies have shown a potential connection between exposure to fine PM and diabetes, cognitive decline, and other serious impacts to the brain.[8]

---

[6] *See* Nino Künzli et al., *Ambient Air Pollution and Atherosclerosis in Los Angeles*, 113 ENVTL. HEALTH PERSP. 201, 201–06 (2005); Kristin A. Miller et al., *Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women*, 356 NEW ENG. J. MED. 447, 447–58 (2007); B. Hoffman et al., *Residential Exposure to Traffic Is Associated With Coronary Atherosclerosis*, 116 CIRCULATION 489 (2007); C. Arden Pope III et al., *Ischemic Heart Disease Events Triggered by Short-Term Exposure to Fine Particulate Air Pollution*, 114 CIRCULATION 2443 (2006); Joel Schwartz et al., *Particulate Air Pollution and Hospital Emergency Room Visits for Asthma in Seattle*, 147 AM. J. RESPIRATORY AND CRITICAL CARE MED. 826, 826–31 (1993); Michael Jerrett et al., *Spatial Analysis of Air Pollution and Mortality in Los Angeles*, 16 EPIDEMIOLOGY 727, 727–36 (2005); Hazrije Mustafic et al, *Main Air Pollutants and Myocardial Infarction: A Systematic Review and Meta-Analysis*, 307 J. AM. MED. ASS'N 713 (2012); Gregory A. Wellenius et al, *Ambient Air Pollution and the Risk of Acute Ischemic Stroke*, 172 ARCHIVES INTERNAL MED. 229 (2012).

[7] *See* Beate Ritz et al., *Air Pollution and Infant Death in Southern California, 1989–2000*, 118 PEDIATRICS 493, 493–502 (2000); Michelle Wilhelm & Beate Ritz, *Residential Proximity to Traffic and Adverse Birth Outcomes in Los Angeles County, California, 1994–1996*, 111 ENVTL HEALTH PERSP. 207, 207–16 (2003); Michelle Wilhelm & Beate Ritz, *Local Variations in CO and Particulate Air Pollution and Adverse Birth Outcomes in Los Angeles County, California, USA*, 113 ENVTL. HEALTH PERSP. 1212, 1212–21 (2005).

[8] *See* Heather E. Volk et al., *Residential Proximity to Freeways and Autism in the CHARGE Study*, 119 ENVTL. HEALTH PERSP. 873 (2011); Zorana J. Anderson et al.,

Of special concern is the soot in diesel exhaust—diesel PM,[9] which is especially toxic not only because of its very small size, but also because the soot particles contain roughly forty different toxic air contaminants, fifteen of which are recognized carcinogens.[10] Recently, the International Agency for Research on Cancer ("IARC"), a branch of the World Health Organization, concluded that diesel engine exhaust is "carcinogenic to humans."[11] This identification of diesel exhaust as a human carcinogen, coming from the world's foremost authority on dangerous carcinogens, follows IARC's 1988 finding that diesel exhaust was a "probable" human carcinogen.

Diabetes Incidence and Long-Term Exposure to Air Pollution: A Cohort Study, 35 DIABETES CARE 92 (2011); Lilian Calderón-Garcidueñas et al., *Neuroinflammation, Hyperphosphorylated Tau, Diffuse Amyloid Plaques, and Down-Regulation of the Cellular Prion Protein in Air Pollution Exposed Children and Young Adults*, 28 J. Alzheimer's Disease 93 (2012); Jennifer Weuve et al., *Exposure to Particulate Air Pollution and Cognitive Decline in Older Women*, 172 ARCHIVES INTERNAL MED. 219 (2012).

[9] Diesel PM is generally more harmful than non-diesel forms of particulate matter. *See* U.S. EPA, Diesel Particulate Matter, http://www.epa.gov/region1/eco/airtox/diesel.html (last visited Feb. 27, 2013); State of New Jersey, Department of Environmental Protection, Bureau of Mobile Sources, Health Concerns of Diesel, http://www.state.nj.us/dep/stopthesoot/dieselhealthconcerns.htm (last visited Feb. 27, 2013); Krivoshto, et al., Journal of the American Board of Family Medicine, The Toxicity of Diesel Exhaust: Implications for Primary Care, vol. 21, no. 1, at 55–62 (Jan–Feb. 2008), *available at* http://www.jabfm.org/content/21/1/55.full.

[10] Diesel exhaust contains the following toxic constituents: acetaldehyde, acrolein, aniline, antimony compounds, arsenic, benzene, beryllium compounds, biphenyl, bis[2-ethylhexyl]phthalate, 1,3-butadiene, cadmium, chlorine, chlorobenzene, chromium compounds, cobalt compounds, cresol isomers, cyanide compounds, dioxins and dibenzofurans, dibutylphthalate, ethyl benzene, formaldehyde, hexane, inorganic lead, manganese compounds, mercury compounds, methanol, methyl ethyl ketone, naphthalene, nickel, 4-nitrobiphenyl, phenol, phosphorus, POM including PAHs and their derivatives, propionaldehyde, selenium compounds, styrene, toluene, and xylenes. *See Air: Health Effects of Diesel Exhaust*, Cal. Off. Envtl Health Hazard Assessment, http://www.oehha.ca.gov/public_info/facts/dieselfacts.html (last visited March 4, 2013); *The Report on Diesel Exhaust*, Cal. Air Res. Bd., http://www.arb.ca.gov/toxics/dieseltac/de-fnds.htm (last visited March 4, 2013).

[11] Press Release, International Agency for Research on Cancer, World Health Organization, IARC: Diesel Engine Exhaust Carcinogenic (June 12, 2012), *available at* http://press.iarc.fr/ pr213_E.pdf.

The State of California identifies diesel PM as a carcinogen. The State made that determination in 1998 based on dozens of human epidemiological studies showing that long-term occupational exposure to diesel exhaust can be associated with a 40 percent increase in the relative risk of lung cancer.[12] The U.S. Environmental Protection Agency ("EPA") currently characterizes diesel exhaust as a "likely" carcinogen,[13] and has sought to regulate the pollution from diesel engines based on its health effects.

Dozens of studies have shown high risks of lung cancer in occupations with high diesel exposures, including rail workers, truck drivers, and miners. Not only are the risks of lung cancer approaching that of heavy smokers for the very highest exposed workers, but the elevated risks of lung cancer apply to the general population in areas with high levels of diesel PM (e.g. communities near freeways and busy freight corridors).[14] Indeed, recent studies have documented the health hazards of

---

[12] California Air Resources Board, Identification of Particulate Emissions from Diesel-Fueled Engines as a Toxic Air Contaminant, http://www.arb.ca.gov/regact/diesltac/diesltac.htm (last visited March 4, 2013); California Air Resources Board, Initial Statement of Reasons for Rulemaking: Staff Report (Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant) (June 1998), *available at* http://www.arb.ca.gov/toxics/dieseltac/staffrpt.pdf; *Chemicals Known to the State to Cause Cancer or Reproductive Toxicity*, Cal. Off. Envtl. Health Hazard Assessment (Feb, 17, 2012), *available at* http://www.oehha.ca.gov/ prop65/prop65_list/files/P65single021712.pdf.

[13] Assessment and Standards Division, U.S. Envtl. Protection Agency, Regulatory Impact Analysis: Control of Emissions of Air Pollution from Locomotive Engines and Marine Compression Ignition Engines Less than 30 Liters Per Cylinder 2–49 (2008), *available at* http://nepis.epa.gov/Exe/ZyNET.exe/P10024CN.TXT?ZyActionD=ZyDocument&Client=EPA&Index=2006+Thru+2010&Docs=&Query=&Time=&EndTime=&SearchMethod=1&TocRestrict=n&Toc=&TocEntry=&QField=&QFieldYear=&QFieldMonth=&QFieldDay=&IntQFieldOp=0&ExtQFieldOp=0&XmlQuery=&File=D%3A\zyfiles\Index%20Data\06thru10\Txt\00000005\P10024CN.txt&User=ANONYMOUS&Password=anonymous&SortMethod=h|-&MaximumDocuments=1&FuzzyDegree=0&ImageQuality=r75g8/r75g8/x150y150g16/i425&Display=p|f&DefSeekPage=x&SearchBack=ZyActionL&Back=ZyActionS&BackDesc=Results%20page&MaximumPages=1&ZyEntry=1&SeekPage=x&ZyPURL.

[14] *See* Debra T. Silverman et al., *The Diesel Exhaust in Miners Study: A Nested Case–Control Study of Lung Cancer and Diesel Exhaust*, 104 J. NAT'L CANCER INST. 1 (2012).

living near busy roadways heavily traveled by diesel trucks.[15] Such studies illustrate that communities near busy roadways used by Port-serving diesel trucks are at risk for significant health impacts. SSR Report pages 41–53 and Appendix B detail how a number of communities northwest of the bridge, including Ironbound, South Ward, and Elizabeth are adversely affected by Port operations, including diesel exhaust from port-serving trucks, ocean going vessels, and cargo handling equipment.

NOx also has a toxic effect on human airways, leading to inflammation, asthmatic reactions, and worsening of allergies and asthma symptoms.[16] In addition, NOx reacts with VOCs in the sunlight to form ozone—also known as smog. This layer of brown haze contributes to decreased lung function, increased respiratory symptoms, asthma, emergency room visits, hospital admissions, and premature deaths.[17] Ozone can also cause irreversible changes in lung structure, eventually leading to chronic respiratory illnesses, such as emphysema and chronic bronchitis.[18]

---

[15] Nino Kunzli et al., *Ambient Air Pollution and the Progression of Atherosclerosis in Adults*, PLOS ONE, vol. 5, issue 2 (Feb 2010), *available at* http://www.plosone.org/article/related/info%3Adoi%2F10.1371%2Fjournal.pone.0009096;jsessionid= 46505FD078D22F6B9543D5BB5C4BDF0D; W James Gauderman et al., *Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study*, THE LANCET (Jan. 26, 2007); Press release, University of Southern California, USC study shows living near a highway affects lung development in children (Jan. 25, 2007); Living Close to Freeways Decreases Lung Development, AAP Grand Rounds, Vol 18, No. 6 (Dec. 2007), *available at* http://aapgrandrounds.aappublications.org/content/18/6/67.extract; Laura Perez et al., *Global Goods Movement and the Local Burden of Childhood Asthma in Southern California*, AMERICAN JOURNAL OF PUBLIC HEALTH, Vol. 99, No. S3 (2009), *available at* http://ajph.aphapublications.org/doi/full/10.2105/AJPH.2008.154955?prevSearch=mcconnell&searchHistoryKey=.

[16] *See* R.J. Davies et al., *Allergen-Irritant Interaction and the Role of Corticosteroids*, 52 ALLERGY 59, 59–65 (1997); R.J. Davies et al., *Why is Allergy Increasing?—Environmental Factors*, 28 CLINICAL & EXPERIMENTAL ALLERGY 8, 8–14 (1998).

[17] *See* U.S. Envtl. Protection Agency, Provisional Assessment of Recent Studies on Health and Ecological Effects of Ozone Exposure (2009).

[18] *See* J.E. Hodgkin et al., *COPD Prevalence in Nonsmokers in High and Low Photochemical Air Pollution Areas*, 86 CHEST 830, 830–38 (1984); David E. Abbey et al., *Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population*, 48 ARCHIVES OF ENVTL. HEALTH 33, 33–46 (1993).

The point of this discussion is that the health effects of exposure to diesel exhaust can be deadly. The Coast Guard must ensure that it fully considers how the Project will affect communities near the Port.

B. The DEA Underestimates How The Project Will Affect Air Quality And Health

The DEA predicts a 0.7% increase in cargo from the Project and deems this insignificant in relation to projected cargo in 2035. The DEA then translates this additional cargo into truck and rail trips leaving the Port. It concludes that induced cargo from the Project will only result in a total of 54 truck trips per day west of the bridge, which translates to 5.4 truck trips per hour, or an average of 1 to 2 trucks per hour from each of the three terminals west of the bridge. DEA at 18-17.

Even if we assume that the DEA's 0.7% induced cargo rate were correct, DEA's analysis relies on a number of oversimplifications about "mode splits" that results in minimizing the air quality and public health effect of increased truck traffic on communities northwest of the bridge, including Newark. For example, and as discussed in SSR Report at 24–27:

- The DEA oddly assumes that only 20% of induced freight traffic will travel by truck, while the remaining 80% will travel by rail. DEA at 18-15. By way of example, CPIP indicates that 85% of container cargo leaving the Port is transported by truck, while only 14% leaves by rail and 1% by barge. CPIP at 107.

- The DEA assumes that the volume of cargo traveling west of the bridge is constant over time at 80%. DEA at 18-15–18-16. This contradicts historical trends indicating that cargo growth west of the bridge is greater than the rest of the Port.

- The DEA assumes that the induced freight volumes traveling west of the bridge are divided equally between all three terminals: Howland Hook, Elizabeth, and Newark. This is not credible given that Port Newark/Elizabeth is the busiest cargo facility at the Port. DEA 1-3.

These and other assumptions work together to minimize the effect induced cargo will have on communities impacted by port-trucking operations. Indeed, if more realistic and refined assumptions are made about how cargo leaves the Port, truck trips could be 10 times larger at the Port of Elizabeth. SSR Report Table 7 at 29. Simply put, once more realistic and refined assumptions are applied, one can predict higher levels of truck traffic in communities northwest of the bridge. These levels are even larger when our induced growth analysis is applied. For instance, in 2035:

- Using DEA assumptions, a total of 54 truck trips are predicted to occur from all three terminals west of the bridge. Using SSR mode splits and DEA cargo volumes, the estimate ranges from 63–268 truck trips/day.

- Using DEA mode splits and SSR cargo volumes, the estimate is 2,340 truck trips/day.

- Using SSR mode split assumptions and SSR cargo volumes, the estimate ranges from 2,450–10,390 truck trips/day.

SSR Report at 28. Accordingly, the SSR Report indicates a potential for more truck traffic in communities west of the bridge than reported in the DEA. This could result in great environmental and public health impacts for communities such as Newark.

The SSR Report goes on to predict the additional emissions that could result for various cargo volume increase scenarios and mode splits for trucks, cargo handling equipment, marine vessels, and rail. These projections are provided in Table 11, page 37. Notably, these emissions projections do not include emissions from ship auxiliary engines or rail. Further, because we have chosen to focus on localized effects of the increased cargo volumes, emissions from trucks are only estimated for on terminal activities and for truck travel for a distance of 1.5 miles from the Port, and only along routes that are in port communities. Should the Coast Guard revise its analysis, it should ensure that all Port sources of emissions are included, and that both a regional and local analysis of air quality impacts is provided.[19] Additionally, the revised analysis should include a health risk assessment ("HRA") that reports the cancer and non-cancer health risks from air pollution. The HRA should include cancer risks associated with exposure to diesel PM. We have provided guidance documents on how such an HRA should be conducted.[20]

---

[19] Indeed, while we have omitted certain emission sources from our estimates, such sources, e.g., emissions from ship auxiliary engines and rail, are large sources of Port pollution and should be included in any revised analysis. *See* The Port Authority of New York and New Jersey, Port Commerce Department, 2008 Multi-Facility Emissions Inventory of Cargo Handling Equipment, Heavy-Duty Diesel Vehicles, Railroad Locomotives and Commercial Marine Vessels (Dec. 2010), *available at* http://www.panynj.gov/about/pdf/portwide-ei-report-2008.pdf; *see also, e.g,* Port of Long Beach, Air Emissions Inventory – 2011 (July 2012), *available at* http://www.polb.com/ civica/filebank/blobdload.asp?BlobID=10194; Port of Los Angeles, Air Emissions Inventory – 2011 (July 2012), *available at* http://www.portoflosangeles.org/pdf/ 2011_Air_Emissions_Inventory.pdf.

[20] California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxics Hot Spots Program Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health

C. The DEA Underestimates Cumulative Air Quality Impacts And Environmental Justice Impacts From The Project

The air quality impacts from the Project are discussed immediately above and in greater detail in the SSR Report pages 33–40. These impacts, however, cannot be viewed in isolation. They must be viewed in the appropriate context, which includes understanding how communities impacted by port-generated air pollution are *also* impacted by other pollution sources in the area, *and* are "environmental justice" communities.

"Cumulative impact" is the impact on the environment which results from the incremental impact of the action when added to other past, present, and reasonably foreseeable future actions regardless of what agency (Federal or non-Federal) or person undertakes such other actions. Cumulative impacts can result from individually minor but collectively significant actions taking place over a period of time. 40 C.F.R. § 1508.7.

As discussed in the SSR Report, the New Jersey Department of Environmental Protection and the National-Scale Air Toxics Assessments report the environmental health conditions for communities in Newark as creating unacceptable levels of increased cancer risk. SSR Report at 44–48. Accordingly, while Port-generated air pollution is a problem for all communities that breathe diesel exhaust from port-serving trucks, ships, trains, and cargo handling equipment, it is an acute, long-standing problem for a number of communities near the Port.

Further, the public health impacts created by exposure to port-generated air pollution is an environmental justice issue. In 2004, EPA studied communities near 47 marine ports and 37 rail yards in the U.S. to better understand how populations near these facilities are exposed to air pollution.[21] EPA found that over 13 million people that live in the vicinity of these freight transportation facilities are exposed to elevated levels of diesel PM.[22] Of these 13 million people, 3.5 million are children, and a disproportionate number of these individuals are low-income persons of color.[23] Accordingly, while the nation's freight transportation system creates economic benefits, it also generates significant air pollution and associated

---

Risk Assessments (August 2003), *available at* http://www.oehha.ca.gov/air/hot_spots/HRAguidefinal.html; California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, A Guide to Health Risk Assessment, *available at* http://oehha.ca.gov/risk/layperson/index.html; National Research Council of the National Academies, Science and Decisions: Advancing Risk Assessment (2009), *available at* http://www.nap.edu/openbook.php?record_id=12209&page=R1.

[21] Assessment and Standards Division, *supra* note 14, at 2–57.

[22] *Id.*

[23] *Id.*

public health impacts for an increasing number of individuals in this country. This phenomenon is detailed in a report drafted by the National Environmental Justice Advisory Council ("NEJAC"), which is a federal advisory committee to EPA.[24]

This phenomenon is also detailed—on a local level—in SSR Report, Appendix B, where we outline how low-income communities of color are disproportionately affected by Port operations and other pollution sources west of the bridge. Within these communities, there are also large populations of children and elderly. Children and the elderly are considered "vulnerable populations" when exposed to air pollution because their bodies are more susceptible to air pollution than healthy, young adults.

In accordance with NEPA, any pollution attributable to the Project must be added to the existing levels of pollution already experienced by impacted communities, as well as air pollution from reasonably foreseeable operations in the area. *N. Plains Resource Council v. Surface Transp. Bd.*, 668 F.3d 1067, 1076 (9th Cir. 2011).

However, completely absent in the DEA is any hard data on the existing or future air pollution levels in communities that could be impacted by the Project. The DEA includes one brief paragraph on the "cumulative effects" within the "operational period." DEA at 18-20. That paragraph concludes: "Since the project has been determined to have no direct or indirect effect on regional traffic capacity or vehicle miles traveled (VMT), and no substantial effect on volume of port activity or overall maritime trade patterns, it would have no cumulative effect in combination with other projects." *Id*.

The DEA's cumulative impacts discussion is hardly adequate given that the entire purpose of a cumulative impact analysis is to determine if an individually *in*significant impact results in a collectively significant effect when other actions are considered. 40 C.F. R. § 1508.7. Simply put, the DEA violates NEPA by failing to perform any cumulative impact analysis at all.

Moreover, given that the DEA underestimates the effect of port-generated air pollution on local communities by (1) using unrealistic mode split assumptions and (2) underestimating cargo growth attributable to the Project, the Coast Guard's error is even more egregious.

Given the environmental justice and cumulative air quality impacts experienced by local communities west of the bridge (*see* SSR Report Appendix B), we recommend

[24] National Environmental Justice Advisory Council, Reducing Air Emissions Associated With Goods Movement: Working Towards Environmental Justice (Nov. 2009), *available at* http://www.epa.gov/compliance/ej/resources/publications/nejac/2009-goodsmovement.pdf.

that a Health Impact Assessment ("HIA") be prepared.[25] The HIA should include an assessment of baseline health conditions, and analyze the impacts on health from the Project. The HIA should be conducted with input from the Port, Coast Guard, and community but be prepared by an independent consultant agreeable to the communities impacted by the Project. The HIA should be conducted in addition to the previously requested HRA, which should include a discussion of the cancer and non-cancer health risks attributable to the air pollution from the Project in addition to other past, current, and future air pollution sources that affect local and regional air quality.

III. MITIGATION TO REDUCE THE PROJECT'S AIR QUALITY IMPACTS IS AVAILABLE

In the presence of potentially significant impacts, the lead agency is obligated to identify means to mitigate the Project's adverse impacts. *See, e.g.*, 40 C.F.R. § 1502.16. The Council on Environmental Quality ("CEQ") explains this requirement:

> All relevant, reasonable mitigation measures that could improve the project are to be identified, even if they are outside the jurisdiction of the lead agency or the cooperating agencies . . . . This will serve to alert agencies or officials who can implement these extra measures, and will encourage them to do so.

CEQ, Forty Most Asked Questions Concerning CEQ's NEPA Regulations, 46 Fed. Reg. 18026-01, 18031–32 (1981).

Thus, the Coast Guide should revise its analysis to include a discussion of mitigation measures available to reduce the Project's environmental impacts. This must include mitigation that the agency may not have authority to implement.

We are aware that U.S. EPA and the Eastern Environmental Law Center have identified mitigation measures that can be undertaken to reduce the Project's impacts, particularly with respect to an increase in diesel emissions. *See, e.g.*, Letter from Grace Musumeci, Chief, Environmental Review Section, U.S. Environmental Protection Agency, Region 2, to Gary Kassof, Bridge Program Manager, First Coast Guard District, re: Bayonne Bridge Navigation Clearance Project NEPA Workplan (Dec. 7, 2011). We urge the Coast Guard to consider these measures and foster paths for their adoptions.

We also provide a copy of NRDC's "Clean Cargo" guide, which includes a compendium of measures that can be adopted to reduce community exposure to

[25] For more information on HIAs see: www.who.int/hia/about/en/ and www.humanimpact.org/.

diesel pollution from the freight transportation industry.[26] In this guide, we discuss measures such as shoreside power for ships, use of cleaner marine fuels for ships, modernization of diesel trucks and cargo handling equipment, as well as air filtration systems, vegetative buffers, and other measures that have been adopted to reduce tailpipe emissions and community exposure. We urge the Coast Guide to consider the measures included in this guide.

IV. THE DEA'S ANALYSIS OF HAZARDOUS CONTAMINANTS IS INADEQUATE AND REVEALS POTENTIALLY SIGNIFICANT IMPACTS

The DEA fails to take a hard look at how construction of the project has the potential to expose the residents of Bayonne, NJ and Staten Island, New York to lead, PCBs, asbestos, arsenic, and radioactive waste. Construction of the Project will entail demolition of existing structures, excavation and removal of some existing soil for off-site disposal, and dewatering of groundwater will likely also be required in some locations. DEA at 16-74. Such construction activities have the potential to expose workers and residents to hazardous contaminants that the DEA acknowledges may exist at the site and adjacent cites. *See id*.

This is particularly concerning given that there are two parks in Bayonne that the DEA reports will be closed during construction, DEA at 16-12, 16-21, and there is a playground and public school located in close proximity to one of the Project's construction work zones. *Id.* at 16-20. These areas, by the DEA's own admission, will be affected by construction in some way—creating a potential risk for children if hazardous contaminants beneath the soil or above are disturbed.

As documented more fully in the attached report by CA Rich Environmental Specialists ("CA Rich Report"), the DEA's analysis of the potential risk from hazardous contaminants is full of data gaps, and is below the standard of care for Phase I assessments and for what should have been done given the complexity of the Project and data available about the contaminants at or near the construction sites. As a result, the investigation performed fails to fulfill the purpose of an EA, that is, to perform sufficient analysis to determine if there is a potential for significant effects. *See Fund for Animals v. Babbitt*, 89 F.3d at 130. What is particularly disturbing about the DEA's analysis is that it appears that relevant information *was* readily available to the consultant who authored Appendix G that would have helped define potential risks from construction, but the Port precluded the consultant from accessing that data by instructing the consultant not to, e.g.,

---

[26] NRDC, Clean Cargo: A Guide to Reducing Diesel Air Pollution from the Freight Industry in your Community clean cargo guide, *available at* http://www.nrdc.org/air/diesel-exhaust/files/clean-cargo-toolkit.pdf; *see also* Port of Los Angeles & Port of Long Beach, San Pedro Bay Ports Clean Air Action Plan: 2010 Update, *available at* http://www.cleanairactionplan.org/civica/filebank/blobdload.asp?BlobID=2485.

speak to regulatory agencies, investigate adjacent sites, conduct interviews with property managers, among other things. *See, e.g.*, DEA at 14-3, Appendix G at 4, 5, 9, 13–15, 19–20.

Instead of performing an adequate investigation to assess the risk from hazardous contaminants, the DEA simply declares that "[d]etailed procedures" would be incorporated into construction documents, "[p]reventative measures would be undertaken," and "[a]ll work would be performed in accordance with applicable local, state, and federal requirements." DEA at 16-74. The DEA then concludes, that

> Following construction of the project, there would be no significant potential for continued exposure. In order to prevent such exposure pathways and doses, the project would include appropriate health and safety and investigative/remedial measures (conducted in consultation with the appropriate regulatory authorities).

DEA 16-74. There are several problems with these statements. First, they appear to concede that there may be significant impacts from exposure to hazardous contaminants *during* construction. If there is a potential for significant impacts, then an EIS is required. *Fund for Animals v. Babbitt*, 89 F.3d at 130. Second, the existence of "detailed procedures" and "applicable local, state, and federal regulations" does not excuse the Coast Guard's failure to take a hard look at the potential risks created by construction now; indeed, this is the very purpose of the EA. *N. Plains Resource Council v. Surface Transp. Bd.*, 668 F.3d at 1083. Third, assuming that mitigation exists to reduce any potentially significant impacts to exposure to hazardous contaminants, the DEA lacks an analysis that these measures will in fact work and reduce potentially significant levels to insignificance.

Indeed, while an agency may forgo preparation of an EIS in the face of potentially significant impacts by adopting mitigation measures, such mitigation must be sufficient to reduce potentially significant impacts to insignificant levels. *Greater Yellowstone Coal. v. Flowers*, 359 F.3d 1257, 1276 (10th Cir. 2004); *Nat'l Audubon Soc. v. Hoffman*, 132 F.3d 7, 17 (2nd Cir. 1997). Further, "mitigation measures [must] be supported by substantial evidence . . . to avoid creating a temptation for federal agencies to rely on mitigation proposals" to avoid an EIS. *Id.*

In determining the sufficiency of the record for a "mitigated FONSI," the Ninth Circuit has stated that a "perfunctory description or mere listing of mitigation measures, without supporting analytical data, is insufficient to support a finding of no significant impact." *Nat'l Parks v. Babbitt*, 241 F.3d 722, 734–35 (9th Cir. 2001), abrogated on other grounds by *Monsanto v. Geertson Seed Farms*, 130 S.Ct. 2743, 2757 (2010) (internal citations omitted) (EA mitigation program inadequate where agency, among other things, failed to analyze how long it would take to

reduce air pollution created by the project or "how great a reduction might ultimately be accomplished").

In summary, the DEA's analysis of hazardous contaminants is woefully inadequate. The DEA itself concedes potentially significant risks during construction, which warrants an EIS. Finally, the Coast Guard cannot rely on a mitigated FONSI to avoid preparation of an EIS given the absence of any analysis that mitigation will in fact reduce impacts to less than significant levels. In short, the Coast Guard's approach to hazardous contaminants is to act first and study later. This violates NEPA.

V. THE DEA'S ANALYSIS OF CONSTRUCTION NOISE IS INADEQUATE AND REVEALS POTENTIALLY SIGNIFICANT IMPACTS

The DEA's construction noise analysis is also deficient. First, the DEA does not disclose what the pre-mitigation noise levels from construction of the Project would be even though it appears that the Coast Guard quantified them.[27] NEPA requires that the lead agency provide the data on which it bases its analysis. Indeed, such information is needed to understand whether mitigations are needed and which mitigations are appropriate.

Second, it appears that the construction of the Project will, in fact, create a significant amount of noise. While the DEA argues that there "are no federal or state regulations which definitively define what constitutes a construction noise impact," the operational noise chapter of the DEA outlines standards that the Table 16-43 appears to violate. DEA at 16-69, 16-70. Specifically, the DEA provides the following thresholds in its operational noise chapter:

- From the New York City CEQR Technical Manual: an increase of 3–5 dBA or more at sensitive receptors over a No Build condition ranging from 60 to 62 dBA $L_{eq(1)}$, and an increase of 3 dBA at night (10 pm to 7 am), are significant. DEA at 13-6–13-7.

[27] The DEA explains that the Federal Highway Administration Road Construction Noise Model (RCNM 1.1) "was used to predict noise levels due to stationary highway construction operations," and page 16-67, Table 16-41, contains a table entitled "Selected Construction Equipment Noise Reference Levels and Usage Factors from RCNM 1.1" that lists some of the kinds of equipment and noise levels included in the model. DEA at 16-66–16-67. The DEA, however, does not disclose the results of the noise model, or in other words, what the overall noise impacts would be from the construction of the Project. On page 16-70, Table 16-43, the DEA contains a table entitled: "Construction Noise Analysis Results." Curiously, however, the noise levels reported in this table "assume implementation" of mitigation measures. This accordingly also fails to disclose what the noise impacts—without mitigation—would be from construction of the Project.

- From the Federal Highway Administration: a substantial noise increase is defined as an increase of 6 dBA. DEA at 13-4.

Table 16-43 reports $L_{eq(1)}$ increases ranging as high as 13.1 dBA at night, and a number of day and night increases above 8 dBA. The table reports a number of receptors that will experience day or night time noise levels above 70 dBA. By way of reference, the sound of a highway or train traffic at 15 meters is 70 dBA. DEA at 13-2. The DEA reports that the construction will occur for as many as 20 months in particular locations, and for 45 months overall. DEA at 16-71. Further, the DEA explains that "an increase in noise level of 10 decibels is considered . . . as a doubling in noise level," and Table 16-43 reports that some areas will see an increase of 10.2, 12.1, 13.1, and a few close to 10—8.5 and 9.2. Moreover, considering the three factors that the DEA asserts "should be considered when determining whether construction-related activities would result in a noise impact"—magnitude of the noise, magnitude of the increase in noise, and duration of the increased noise levels—the noise impacts seem to be significant. *See* DEA at 16-69. The DEA's own data and analysis contradicts the DEA's repeated assertions that the construction noise levels are "relatively modest." *See* DEA at 16-70, 16-71.

Additionally, the DEA appears to admit that noise impacts will be significant—and need to be mitigated—without coming right out and stating it. The DEA explains that "[t]hese increases are likely to be noisy and intrusive to some residences and users of public facilities and institution[s]," and that PANYNJ will even set up a program to provide storm windows, air conditioning units, and alternative ventilation, presumably in addition to the other mitigation options. DEA at 16-71. Mitigation of this type would only be initiated if impacts were deemed significant.

Third, the DEA explains that analysis of the expected noise levels and commitment to mitigations will occur *after* the Project would be approved. The DEA explains that "[p]rior to the start of any work the Contractor is to perform a noise analysis based on their anticipated construction activities and submit for approval a Noise Mitigation Plan that will adhere to the noise criteria indicated in the contract documents." DEA at 16-68. This turns NEPA on its head. NEPA requires this analysis to occur *before* the project is approved, not *after*. Further, the DEA also does not explain what the "noise criteria indicated in the contract documents" would be. This is a critical omission, preventing decision makers and the public from understanding what noise levels the contractors are going to be required to stay within. The last paragraph of the construction noise analysis states that contractors will be required to "utilize construction equipment and path controls which in combination do not produce L_{max} noise levels at 50 feet which would exceed 85 dBA during weekday daytime hours (i.e., between 7 AM and 6 PM), and which produce L_{max} noise levels at 50 feet which would be no more than 8 dBA above existing noise levels during nighttime and weekend work periods." DEA at 16-72. It is not clear if this is the "noise criteria" that will be in the "contract

documents." If so, this would allow for a significant amount of noise, especially if there are multiple contractors operating simultaneously. Also troublesome is that 8 dBA is a lower noise level than what Table 16-43 projects for some locations, even though Table 16-43 "assume[s] implementation" of "path controls." *See* DEA at 16-70.

Fourth, the DEA lists "examples of the types of measures that may be utilized" for noise mitigation but does not provide any information, analysis, or evidence about the measures' effectiveness or substantiate that the mitigation options are sufficient to reduce potentially significant noise levels. *Nat'l Audubon Soc. v. Hoffman*, 132 F.3d at 17. As part of demonstrating the effectiveness of any proposed mitigation, the Coast Guard should ensure that there is adequate enforcement and regular monitoring of the mitigation measures.

Finally, the DEA does not contain any discussion on how noise affects public health and quality of life. Without such a discussion, it is impossible for decision makers and the public to understand the real-life impacts of Project-generated noise.

Scientific evidence in the public health literature firmly establishes the relationship between exposure to noise and health impacts. Table 1 below shows classifications of effects that were deemed to have "sufficient evidence" in understanding the effects of noise on health in a 1994 review—nearly 20 years ago. Some of these effects have now been seen at even lower noise levels.

Table 1.

Observation Threshold – Environmental Noise (that is, not occupational)			
	Metric	*Value DB(A)*	*Where measured*
Hypertension	L_{dn}	70	Indoors
Ischemic heart disease	$L_{Aeq.24h}$	70	Outdoors
Annoyance	L_{dn}	42 (30 for impulse noise)	Indoors
Performance, school children	$L_{Aeq.8th}$	70	Outdoors
Sleep disturbances, including:			
sleep patterns,	$L_{Aep.night}$	<60	Outdoors
awakening,	SEL	55	Indoors
sleep quality,	$L_{Aep.night}$	40	Outdoors
heart rate, and	SEL	40	Indoors
mood next day	$L_{Aep.night}$	<60	Outdoors

Some significant additions to the literature since that time are included in the attachments to this letter, including some emerging results about noise and health.

Noise creates annoyance in those exposed and annoyance levels have been well-established at noise higher than 42 A weighted decibels or dB(A), with environmental impulse noise levels above 30dB causing annoyance. Annoyance is a psychosocial effect of exposure to noise, including a sense of resentment, discomfort, displeasure or offense.[28] Annoyance is related to several stress-related health effects associated with noise, including elevated blood pressure (hypertension) and circulatory disease. (See studies under cardiovascular disease). One study from Sweden suggests that even if a quiet side of a house has a noise level under 45dB, the sound levels from road traffic noise on the other side of the house should not exceed 60dB to protect against annoyances and adverse health effects.[29] The Swedish authors conclude that: "A very good sound environment that promotes health and well-being is one where sound levels from road traffic noise in residential areas are below LAeq;24h = 45 dB."[30]

Noise and vibration exposures are linked to sleep disturbance and its resultant impacts. Sleep is important for regeneration of the body, and disturbed sleep can have health consequences. Disturbance of sleep from noise has been shown to begin in the 40–60 dB(A) range. A study in Finland found that nighttime traffic noise levels above 55 dB were associated with insomnia symptoms, including not being able to fall asleep, waking up during the night, waking up too early in the morning, and nonrestorative sleep, but that for individuals who exhibited traits of anxiety, noise levels above 50dB were linked to insomnia.[31] Even if construction of the Bayonne Bridge and roadway occurs only during the day, babies and young children nap during the day and night-shift workers have to sleep during the day, so there could be sleep impacts. Vibration from the Bayonne Bridge construction and use might be likened to low frequency nocturnal vibration from freight trains, which has been shown to impact sleep, with impacts increasing with greater vibration amplitude.[32] The freight train study results "suggest that individuals living near to railway lines and thus subjected to the accompanying noise and vibration exposure are at risk for having their sleep impaired, and that this may lead to reduced

---

[28] Passchier-Vermeer W, Passchier WF. Noise exposure and public health. Environmental health perspectives 2000;108 Suppl 1:123-31.
[29] Ohrstrom E, Skanberg A, Svensson H, Gidlof-Gunnarsson A. Effect of road traffic noise and the benefit of access to quietness. Journal of Sound and Vibration 2006;295:40-59.
[30] *Id.*
[31] Halonen JI, Vahtera J, Stansfeld S, et al. Associations between nighttime traffic noise and sleep: the Finnish public sector study. Environ Health Perspect 2012;120:1391-6.
[32] Smith MG, Croy I, Ogren M, Persson Waye K. On the influence of freight trains on humans: a laboratory investigation of the impact of nocturnal low frequency vibration and noise on sleep and heart rate. PLoS One 2013;8:e55829.

concentration and daytime functioning in the short term and impaired health in the long term."[33]

Noise exposure is linked to stress-related health effects, including an increase in blood pressure, stroke, and cardiovascular disease. An association between road traffic noise and hypertension was found in a 2007 study of residents living near roadways in Sweden.[34] This study described risk factors as high among those living in older homes without triple glazed windows and with bedroom windows facing the street.[35] Other studies have shown that long-term traffic exposure, especially at night, increases the risk of heart attacks and other cardiovascular diseases.[36] Studies have shown these impacts especially related to exposure to train, aircraft, and highway noise. Studies of children show that road traffic noise at home is a stressor that can affect children's blood pressure, with the authors again noting that children go to bed earlier and also sleep during the day, so that daytime noise matters to this group.[37] Studies of men in Berlin exposed to road noise levels higher than 70 dB(A) during the day showed a 30% higher risk of heart attack (MI) than those not exposed.[38] A study published in 2012 found a dose-dependent higher risk for MI in those exposed to long-term residential road traffic noise. That is, the higher the noise exposure, the higher the risk for MI.[39] The authors note that noise exposure has been linked to metabolic and endocrine function and has been shown to impair the immune system. Finally, a 2011 study showed that "exposure to residential road traffic noise was associated with a higher risk for stroke among people older than 64.5 years of age."[40]

---

[33] *Id.*
[34] Leon Bluhm G, Berglind N, Nordling E, Rosenlund M. Road traffic noise and hypertension. Occup Environ Med 2007;64:122-6.
[35] *Id.*
[36] Babisch W, Beule B, Schust M, Kersten N, Ising H. Traffic noise and risk of myocardial infarction. Epidemiology 2005;16:33-40; Sorensen M, Andersen ZJ, Nordsborg RB, et al. Road traffic noise and incident myocardial infarction: a prospective cohort study. PLoS One 2012;7:e39283; Selander J, Nilsson ME, Bluhm G, et al. Long-term exposure to road traffic noise and myocardial infarction. Epidemiology (Cambridge, Mass) 2009;20:272-9.
[37] Babisch W, Neuhauser H, Thamm M, Seiwert M. Blood pressure of 8-14 year old children in relation to traffic noise at home--results of the German Environmental Survey for Children (GerES IV). Sci Total Environ 2009;407:5839-43.
[38] Babisch W, Beule B, Schust M, Kersten N, Ising H. Traffic noise and risk of myocardial infarction. Epidemiology 2005;16:33-40.
[39] Sorensen M, Andersen ZJ, Nordsborg RB, et al. Road traffic noise and incident myocardial infarction: a prospective cohort study. PLoS One 2012;7:e39283.
[40] Sorensen M, ZJ A, al. e. Road traffic noise and stroke: a prospective cohort study. European Heart Journal 2011;32:737-44.

Noise exposure is linked to decreased school performance, cognitive impairment, and decreased academic achievement in children. The World Health Organization recommends that indoor levels of noise not be above 30–45dB, depending on the measurement method.[41] There is significant evidence that school age children exposed to high levels of traffic noise during a school day will be at increased risk of attention span, concentration, and remembering, and reading ability deficits.[42] According to a review by Passchier-Vermeer, there is overwhelming evidence from laboratory experiments that the presence of uncontrollable noise can significantly impair cognitive performance, with the authors also noting that studies show that "schoolchildren exposed to high levels of traffic noise show impairments in performing cognitive tasks."[43]

A 2013 Danish study investigated whether long-term exposure to residential road traffic noise is associated with an increased risk of Type 2 diabetes (using nitrogen oxides to control for air pollution). They concluded that exposure to urban noise may adversely influence health, because of the association they found between road traffic noise and a higher risk of diabetes. They note that other studies have linked sleep problems with increased morning glucose levels and decreased insulin levels and that hormones responsible for appetite regulation are affected by sleep problems.[44]

In the situation of the Bayonne Bridge project, nearby residents will be exposed to both air pollution and noise during construction of the Bridge and roadway realignment. A recent study by Beelen found that background black smoke (a marker for diesel exhaust) concentrations, traffic intensity on the nearest road, and traffic noise above 65 dB(A) were associated with specific cardiovascular causes of death. Associations with traffic noise held up for heart failure mortality after adjustment for background black smoke and traffic intensity on the nearest road but not for other cardiovascular endpoints.[45] The authors note that: "As this is the first study that reported the effects of long-term exposure to air pollution, traffic

---

[41] Smith MG, Croy I, Ogren M, Persson Waye K. On the influence of freight trains on humans: a laboratory investigation of the impact of nocturnal low frequency vibration and noise on sleep and heart rate. PLoS One 2013;8:e55829.
[42] Passchier-Vermeer W, Passchier WF. Noise exposure and public health. Environmental health perspectives 2000;108 Suppl 1:123-31.
[43] Passchier-Vermeer W, Passchier WF. Noise exposure and public health. Environ Health Perspect 2000;108 Suppl 1:123-31.
[44] Sorensen M, Andersen ZJ, Nordsborg RB, et al. Long-term exposure to road traffic noise and incident diabetes: a cohort study. Environ Health Perspect 2013;121:217-22.
[45] Beelen R, Hoek G, Houthuijs D, et al. The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. Occup Environ Med 2009;66:243-50.

intensity and traffic noise on mortality together in one study, further studies are required to confirm or refute our findings." Nonetheless, this report shows that exposure to near-roadway air pollution was linked to CV mortality and that noise exposure might also play an independent role, especially for certain types of CV mortality.[46] Both of these exposures—particulate air pollution and noise—are relevant for the Bayonne Bridge project.

In summary, the Coast Guard's analysis as to whether potentially significant impacts exist with respect noise is at best, cursory, and at worst, deceptive. Moreover, to the extent a potential for significant effects exists, the DEA hardly satisfies NEPA's requirements for a mitigated FONSI.

VI. NEPA REQUIRES THE COAST GUARD TO PREPARE AN EIS FOR THIS PROJECT

As described above, if the project may significantly affect the environment, then "[u]nder NEPA, federal agencies must prepare an Environmental Impact Statement ('EIS') assessing the beneficial and adverse environmental impacts . . . that significantly affects the quality of the human environment." *Fund for Animals v. Babbitt*, 89 F.3d at 130. In determining the "significance" of an impact, lead agencies are required to look at both the context of the action and its intensity. 40 C.F.R. § 1508.27. "Context" means that the significance of an action must be analyzed from several perspectives, such as "the affected region, the affected interests, and the locality." 40 C.F.R. § 1508.27(a). "Intensity" "refers to the severity of impact." *Id*. § 1508.27(b).

In assessing "intensity," the CEQ regulations direct agencies to consider ten factors, including the degree to which the proposed action affects public health, *id*. § 1508.27(b)(2); unique characteristics of the geographic area such as proximity to park lands, wetlands, prime farmlands, or ecologically critical areas, *id*. § 1508.27(b)(3); the degree to which the effects on the quality of the human environment are likely to be highly controversial, *id*. § 1508.27(b)(4); the degree to which the possible effects on the human environment are highly uncertain or involve unique or unknown risks, *id*. § 1508.27(b)(5); whether the action is related to other actions with individually insignificant but cumulatively significant impacts; *id*. § 1508(b)(7); and whether the action threatens a violation of Federal, State, or local law or requirements imposed for the protection of the environment, *id*. § 1508.27(b)(10). The presence of any one of the ten factors enumerated in § 1508.27(b) may trigger the duty to prepare an EIS. *Ocean Advocates v. U.S. Army Corps of Eng'rs*, 402 F.3d 846, 865 (9th Cir. 2005).

A number of the factors described above are present, which indicate a potential for significant effects and the necessity of an EIS. In terms of air quality:

---

[46] *Id.*

- The Project will most certainly affect public health. 40 C.F.R. § 1508.27(b)(2). This is certainly the case if our alternative baseline and induced growth estimates are utilized. But even if they are not, a 0.7% growth in cargo volumes could still adversely affect public health, particularly if realistic mode splits are utilized.

- The impacts of this Project will be disproportionately born by environmental justice communities, and may be significant when combined with other past, present, and future emissions. *Id.* § 1508.27(b)(7).

- To the extent the Coast Guard is concerned about the speculative or uncertain nature of the Project's ability to induce growth, that position bolsters the need to prepare an EIS. *Id.* § 1508.27(b)(5).

- This Project is controversial. A project is "controversial" and may require an EIS if there is substantial dispute as to the size, nature, or effect of the action. 40 C.F.R. § 1508.27(b)(4); *Pub. Citizen v. Dep't of Transp.*, 316 F.3d 1002, 1024 (9th Cir. 2003), *rev'd on other grounds by Dep't of Transp. v. Pub. Citizen*, 540 U.S. 1088 (2003). Our comments, combined with EPA's criticisms, which have yet to be resolved, indicate a substantial dispute about the effect of this project, particularly as to cargo growth.

With respect to release of hazardous contaminants during construction and construction noise, similar arguments can be made. For example, there is a potential for impacts to public health, 40 C.F.R. § 1508.27(b)(2), and such impacts will disproportionately affect environmental justice communities in Bayonne, New Jersey and Staten Island, New York and may result in cumulatively significant impacts. *Id.* § 1508.27(b)(7). Moreover, the DEA itself seems to concede potentially significant effects for both resource categories. *See supra* Sections IV & V.

Notably, in 2010, the Port specifically sought, through a TIGER Grant Application, millions in federal funds to prepare an EIS. Tiger Grant App. at 1. There is no evidence to suggest that the Port and Coast Guard should have deviated from the Port's original plan.

VII. CONCLUSION

The DEA fails to take the requisite "hard look" in order to determine if the Project may result in potentially significant effects. Further, errors in the DEA render the document legally indefensible, and preclude the Coast Guard from relying on it to render a FONSI. The Coast Guard must conduct the analysis NEPA requires, and in

light of the magnitude of potential impacts described above, prepare that analysis in an EIS.

As part of the EIS, the Coast Guard should:

1. Utilize an accurate baseline or "no build alternative" that is consistent with the Port's public statements about the need for this Project, and that takes into consideration the methodologies utilized in the CPIP and the SSR Report.

2. Revise its Induced Demand Analysis in light of the CPIP and SSR Report.

3. Revise its projection of how much air pollution will be created by the Project.

4. Revise its projection of how much air pollution will be created by the Project in connection with other past, present, and reasonably foreseeable projects in the area.

5. Analyze how the Project will affect regional and localized air pollution levels given the concerns raised above (e.g., about the DEA's mode splits and induced demand analysis).

6. Assess the health consequences that may result from exposure to that pollution, individually and in combination with other past, present, and foreseeable future pollution sources in the area. As part of that analysis, prepare a HRA for the Project, which reports the cancer and non-cancer health risks from air pollution. The HRA should include cancer risks associated with exposure to diesel PM. In addition, a HIA should be prepared that includes an assessment of baseline health conditions, and an analysis of the impacts on health from the Project. The HIA should be conducted with input from the Port, Coast Guard, and community but be prepared by an independent consultant agreeable to the communities impacted by the Project.

7. Revise its environmental justice analysis to account for errors in the DEA's induced growth, mode shift, and air quality and cumulative impact discussions.

8. Perform a complete assessment of hazardous contaminants in the construction area, which, for instance, fills the data gaps identified in the CA Rich Report.

9. Revise its construction noise section so that pre-mitigation noise levels are reported, and provide the public with thresholds that can be used to evaluate the significance of those levels.

10. Assuming potentially significant environmental impacts exist, discuss all available mitigation measures to reduce those impacts, including an analysis of the effectiveness of each mitigation measure. Mitigation measures should be fully enforceable (e.g., made a condition of the Coast Guard's permit) and provide for adequate monitoring to ensure the mitigation is completed and effective.

Thank you again for the opportunity to provide public comments. Please feel free to contact me if you have any questions. I can be reached at (310) 424-2300 or mlinperrella@nrdc.org.

Sincerely,

Melissa Lin Perrella
Senior Attorney
Natural Resources Defense Council

Enclosures:

- Index of Attachments to NDC's Comments on the Draft Environmental Assessment for the Bayonne Bridge Navigational Clearance Program
- G. Leon Bluhm et al., *Road traffic noise and hypertension,* OCCUP ENVIRON MED, vol. 64, p. 122 (2007).

Index of Attachments to NRDC's Comments on the Draft Environmental Assessment for the Bayonne Bridge Navigational Clearance Program

Description	Doc. #
TIGER II Planning Grant Application, Surface Transportation Infrastructure Discretionary Grant Application Package, Opportunity Number DTOS59-10-RA-TIGER2, Completion ID TIGER2-11, Application Filing Name: Bayonne Bridge Navigational Clearance (Aug. 2010).	1
Letter from Patrick J. Foye, Executive Director, The Port Authority of NY & NJ, to the Honorable Janet Napolitano, Secretary, U.S. Department of Homeland Security and the Honorable Ray LaHood, Secretary, U.S. Department of Transportation, (March 23, 2012) (with a cover letter from Kathy Ray, FOIA Officer, U.S. Department of Transportation, to William J. Schulte, Eastern Environmental Law Center (March 30, 2012)).	2
Toni-Ann Cerbo, *Port Authority approves $25 million for planning of raising Bayonne Bridge's roadbed*, THE JERSEY JOURNAL, May 27, 2011, *available at* http://www.panynj.gov/bayonnebridge/pdf/052711Portroadbed.pdf.	3
Steve Strunsky, *How a $1B lift will give Bayonne Bridge a boost*, STAR-LEDGER, Jan. 26, 2011, *available at* http://www.panynj.gov/bayonnebridge/pdf/012611howboost.pdf.	4
Steve Strunsky, *Raising Bayonne Bridge makes way for new ships*, STAR-LEDGER, Dec 30, 2010, *available at* http://www.panynj.gov/bayonnebridge/pdf/123010raisingships.pdf.	5
Peter Leach, *NY-NJ Port Expedites Bayonne Bridge Project*, JOURNAL OF COMMERCE, July 18, 2012, *available at* http://www.joc.com/maritime-news/international-freight-shipping/ny-nj-port-expedites-bayonne-bridge-project_20120718.html.	6
Joseph Bonney, *NY-NJ Port to Announce Bayonne Bridge Plan*, JOURNAL OF COMMERCE, Dec. 3, 2010, *available at* http://www.joc.com/maritime-news/ny-nj-port-announce-bayonne-bridge-plan_20101203.html.	7
Bayonne Bridge Navigation Clearance Project: NEPA Workplan (Sept. 2011), *available at* http://cleanandsafeports.org/wp-content/uploads/2012/07/2011-09-30-NEPA-Work-PlanFINAL1.pdf.	8
U.S. Army Corps of Engineers, Bayonne Bridge Air Draft Analysis (Sept. 2009), *available at* http://www.nysanet.org/documents/Bayonne-Bridge-Air-Draft-Analysis.pdf.	9
CPIP Consortium, Port of New York & New Jersey, Comprehensive Port Improvement Plan, Volume 1: The Plan (Sept. 2005) & Volume 2: Toolkit (Aug. 2005), *available at* http://www.panynj.gov/about/pdf/cpip/V1_Plan.pdf and http://www.panynj.gov/about/port-initiatives.html.	10
Sustainable Systems Research, Technical Memorandum: Review of the Bayonne Bridge Navigational Clearance Program Draft Environmental Assessment (March 1, 2013).	11
U.S. Coast Guard, First Coast Guard District Bridge Program, Bayonne Bridge Navigational Clearance Program, Responses to Scoping Comments, NEPA Workplan (Feb. 2012).	12

The Port Authority of NY & NJ, Port Authority Marine Container Terminals Truck Origin-Destination Survey 2005 (Nov. 2005, revised Feb. 2006).	13
State of New Jersey, Department of Environmental Protection, Estimated Air Quality Impacts on Surrounding Communities of PM2.5 and SO_2 Emissions Resulting From Maritime Operations at Elizabeth Port Authority Marine Terminal and Port Newark (Oct. 9, 2009).	14
State of New Jersey, Department of Environmental Protection, Estimated Air Quality Impacts on Surrounding Communities of PM2.5 and SO_2 Emissions Resulting from Maritime Operations at the Elizabeth Port Authority Marine Terminal and Port Newark: Phase 2 Future Impacts (2015) (Aug. 16, 2011).	15
The Port Authority of New York and New Jersey, Port Commerce Department, 2008 Multi-Facility Emissions Inventory of Cargo Handling Equipment, Heavy-Duty Diesel Vehicles, Railroad Locomotives and Commercial Marine Vessels (Dec. 2010), *available at* http://www.panynj.gov/about/pdf/portwide-ei-report-2008.pdf.	16
Documents received in response to a FOIA request to the U.S. EPA.	17
North American Port Container Traffic 2011(1), *available at* http://aapa.files.cms-plus.com/Statistics/North%20AMERICAN%20PORT%20CONTAINER%20TRAFFIC%202011.pdf.	18
Port Authority of NY & NJ, Port Redevelopment, Inland Access, http://www.panynj.gov/port/inland-access.html.	19
Port of New York and New Jersey, Services, New York Container Terminal, Port Newark Container Terminal, Maher Terminal, APM Terminals, *available at* http://www.panynj.gov/port/containerized-cargo.html.	20
Port of Los Angeles, Southern California International Gateway Project, Draft Environmental Impact Report (Sept. 2012) (excerpts), *available at* http://www.portoflosangeles.org/EIR/SCIG/DEIR/deir_scig.asp.	21
U.S. EPA, MOBILE6 Vehicle Emission Modeling Software, http://www.epa.gov/otaq/m6.htm.	22
Letter from Frederick M. Sellars, Vice President, Arcadis, to Alan Dresser & Yiling Zhang, Bureau of Technical Services, Division of Air Quality, New Jersey Department of Environmental Protection, re: Protocol for Cumulative Impact Modeling for NO_2 1-hour Average Impacts; PI# 08857, BOP110001; Newark, Essex County, NJ (Feb. 23, 2012) (incl. Attachment A: Cumulative Impact Modeling Source Inventory).	23
Wexler, A, and K. Pinkerton, Toxicity of Source-Oriented Ambient Submicron Particulate Matter, Contract Number 06-0331 (May 2012), Prepared for the California Air Resources Board, the California Environmental Protection Agency, and the Electric Power Research Institute, *available at* http://www.arb.ca.gov/research/apr/past/06-331.pdf.	24
U.S. EPA, Technology Transfer Network Air Toxics, 2005 Assessment Results *available at* http://www.epa.gov/ttn/atw/nata2005/tables.html.	25
State of New Jersey, Department of Environmental Protection, Air Toxics in New Jersey, Diesel Emissions, http://www.nj.gov/dep/airtoxics/diesemis.htm.	26

California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxics Hot Spots Program Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (August 2003), *available at* http://www.oehha.ca.gov/air/hot_spots/HRAguidefinal.html.	27
California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, A Guide to Health Risk Assessment, *available at* http://oehha.ca.gov/risk/layperson/index.html.	28
National Research Council of the National Academies, Science and Decisions: Advancing Risk Assessment (2009), *available at* http://www.nap.edu/openbook.php?record_id=12209&page=R1.	29
Nino Künzli et al., *Ambient Air Pollution and Atherosclerosis in Los Angeles*, 113 ENVTL. HEALTH PERSP. 201, 201–06 (2005).	30
Kristin A. Miller et al., *Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women*, 356 NEW ENG. J. MED. 447, 447–58 (2007).	31
B. Hoffman et al., *Residential Exposure to Traffic Is Associated With Coronary Atherosclerosis*, 116 CIRCULATION 489 (2007).	32
C. Arden Pope III et al., *Ischemic Heart Disease Events Triggered by Short-Term Exposure to Fine Particulate Air Pollution*, 114 CIRCULATION 2443 (2006).	33
Joel Schwartz et al., *Particulate Air Pollution and Hospital Emergency Room Visits for Asthma in Seattle*, 147 AM. J. RESPIRATORY AND CRITICAL CARE MED. 826, 826–31 (1993).	34
Michael Jerrett et al., *Spatial Analysis of Air Pollution and Mortality in Los Angeles*, 16 EPIDEMIOLOGY 727, 727–36 (2005).	35
Hazrije Mustafic et al., *Main Air Pollutants and Myocardial Infarction: A Systematic Review and Meta-Analysis*, 307 J. AM. MED. ASS'N 713 (2012).	36
Gregory A. Wellenius et al., *Ambient Air Pollution and the Risk of Acute Ischemic Stroke*, 172 ARCHIVES INTERNAL MED. 229 (2012).	37
Beate Ritz et al., *Air Pollution and Infant Death in Southern California, 1989–2000*, 118 PEDIATRICS 493, 493–502 (2000).	38
Michelle Wilhelm & Beate Ritz, *Residential Proximity to Traffic and Adverse Birth Outcomes in Los Angeles County, California, 1994–1996*, 111 ENVTL HEALTH PERSP. 207, 207–16 (2003).	39
Michelle Wilhelm & Beate Ritz, *Local Variations in CO and Particulate Air Pollution and Adverse Birth Outcomes in Los Angeles County, California, USA*, 113 ENVTL. HEALTH PERSP. 1212, 1212–21 (2005).	40
Heather E. Volk et al., *Residential Proximity to Freeways and Autism in the CHARGE Study*, 119 ENVTL. HEALTH PERSP. 873 (2011).	41
Zorana J. Anderson et al., *Diabetes Incidence and Long-Term Exposure to Air Pollution: A Cohort Study*, 35 DIABETES CARE 92 (2011).	42
Lilian Calderón-Garcidueñas et al., *Neuroinflammation, Hyperphosphorylated Tau, Diffuse Amyloid Plaques, and Down-Regulation of the Cellular Prion Protein in Air Pollution Exposed Children and Young Adults*, 28 J. Alzheimer's Disease 93 (2012).	43
Jennifer Weuve et al., *Exposure to Particulate Air Pollution and Cognitive Decline in Older Women*, 172 ARCHIVES INTERNAL MED. 219 (2012).	44

U.S. EPA, Region 1: EPA New England, Diesel Particulate Matter, http://www.epa.gov/region1/eco/airtox/diesel.html.	45
State of New Jersey, Department of Environmental Protection, Bureau of Mobile Sources, Health Concerns of Diesel, http://www.state.nj.us/dep/stopthesoot/dieselhealthconcerns.htm.	46
Irina N. Krivoshto et al., *The Toxicity of Diesel Exhaust: Implications for Primary Care*, 21 JOURNAL OF THE AMERICAN BOARD OF FAMILY MEDICINE 55–62 (Jan.–Feb. 2008), *available at* http://www.jabfm.org/content/21/1/55.full.	47
Cal/EPA, Office of Environmental Health Hazard Assessment and The American Lung Association of California, Health Effects of Diesel Exhaust, http://www.oehha.ca.gov/public_info/facts/dieselfacts.html.	48
California Air Resources Board, The Report on Diesel Exhaust, http://www.arb.ca.gov/toxics/dieseltac/de-fnds.htm.	49
Press Release, International Agency for Research on Cancer, World Health Organization, IARC: Diesel Engine Exhaust Carcinogenic (June 12, 2012), *available at* http://press.iarc.fr/pr213_E.pdf.	50
California Air Resources Board, Rulemaking Identification of Particulate Emissions from Diesel-Fueled Engines as a Toxic Air Contaminant, http://www.arb.ca.gov/regact/diesltac/diesltac.htm.	51
California Air Resources Board, Initial Statement of Reasons for Rulemaking: Staff Report (Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant) (June 1998), *available at* http://www.arb.ca.gov/toxics/dieseltac/staffrpt.pdf.	52
State of California, Environmental Protection Agency, Chemicals Known to the State to Cause Cancer or Reproductive Toxicity (Feb. 17, 2012), *available at* http://www.oehha.ca.gov/prop65/prop65_list/files/P65single021712.pdf.	53
U.S. Environmental Protection Agency, Regulatory Impact Analysis: Control of Emissions of Air Pollution from Locomotive Engines and Marine Compression Ignition Engines Less than 30 Liters Per Cylinder (May 2008), *available at* http://nepis.epa.gov/Exe/ZyNET.exe/P10024CN.TXT?ZyActionD=ZyDocument&Client=EPA&Index=2006+Thru+2010&Docs=&Query=&Time=&EndTime=&SearchMethod=1&TocRestrict=n&Toc=&TocEntry=&QField=&QFieldYear=&QFieldMonth=&QFieldDay=&IntQFieldOp=0&ExtQFieldOp=0&XmlQuery=&File=D%3A\zyfiles\Index%20Data\06thru10\Txt\00000005\P10024CN.txt&User=ANONYMOUS&Password=anonymous&SortMethod=h|-&MaximumDocuments=1&FuzzyDegree=0&ImageQuality=r75g8/r75g8/x150y150g16/i425&Display=p|f&DefSeekPage=x&SearchBack=ZyActionL&Back=ZyActionS&BackDesc=Results%20page&MaximumPages=1&ZyEntry=1&SeekPage=x&ZyPURL.	54
Debra T. Silverman et al., *The Diesel Exhaust in Miners Study: A Nested Case–Control Study of Lung Cancer and Diesel Exhaust*, 104 J. NAT'L CANCER INST. 1 (2012).	55
Nino Kunzli et al., *Ambient Air Pollution and the Progression of Atherosclerosis in Adults*, PLOS ONE, vol. 5, issue 2 (Feb 2010), *available at* http://www.plosone.org/article/related/info%3Adoi%2F10.1371%2Fjournal.pone.0009096;jsessionid=46505FD078D22F6B9543D5BB5C4BDF0D.	56

W James Gauderman et al., *Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study*, THE LANCET (Jan. 26, 2007).	57
Press release, University of Southern California, USC study shows living near a highway affects lung development in children (Jan. 25, 2007); Living Close to Freeways Decreases Lung Development, AAP Grand Rounds, Vol 18, No. 6 (Dec 2007), *available at* http://aapgrandrounds.aappublications.org/content/18/6/67.extract.	58
Laura Perez et al., *Global Goods Movement and the Local Burden of Childhood Asthma in Southern California*, AMERICAN JOURNAL OF PUBLIC HEALTH, Vol. 99, No. S3 (2009), *available at* http://ajph.aphapublications.org/doi/full/10.2105/AJPH.2008.154955?prevSearch=mcconnell&searchHistoryKey=.	59
RJ Davies et al., *Allergen-irritant interaction and the role of corticosteroids*, 52 ALLERGY 59, 59–65 (1997).	60
R.J. Davies et al., *Why is Allergy Increasing?—environmental factors*, 28 CLINICAL & EXPERIMENTAL ALLERGY 8, 8–14 (1998).	61
U.S. EPA, Provisional Assessment of Recent Studies on Health and Ecological Effects of Ozone Exposure (Sept. 2009).	62
J E Hodgkin et al., *COPD prevalence in nonsmokers in high and low photochemical air pollution areas*, 86 CHEST 830, 830–38 (1984).	63
David E. Abbey et al., *Long-term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population*, 48 ARCHIVES OF ENVTL. HEALTH 33, 33–46 (1993).	64
Port of Long Beach, Air Emissions Inventory – 2011 (July 2012), *available at* http://www.polb.com/civica/filebank/blobdload.asp?BlobID=10194.	65
Port of Los Angeles, Air Emissions Inventory – 2011 (July 2012), *available at* http://www.portoflosangeles.org/pdf/2011_Air_Emissions_Inventory.pdf.	66
National Environmental Justice Advisory Council, Reducing Air Emissions Associated With Goods Movement: Working Towards Environmental Justice (Nov. 2009), *available at* http://www.epa.gov/compliance/ej/resources/publications/nejac/2009-goods-movement.pdf.	67
NRDC, Clean Cargo: A Guide to Reducing Diesel Air Pollution from the Freight Industry in your Community clean cargo guide, *available at* http://www.nrdc.org/air/diesel-exhaust/files/clean-cargo-toolkit.pdf.	68
Port of Los Angeles & Port of Long Beach, San Pedro Bay Ports Clean Air Action Plan: 2010 Update, *available at* http://www.cleanairactionplan.org/civica/filebank/blobdload.asp?BlobID=2485.	69
CA Rich Consultants, Draft Environmental Assessment Review, Bayonne Bridge Navigational Clearance Program (Feb. 2013).	70
ASTM International, Standard Practice for Environmental Site Assessments: Phase I Environmental Site Assessment Process, Designation: E 1527-05.	71
New York State Department of Environmental Conservation, DEC Program Policy, DER-10/Technical Guidance for Site Investigation and Remediation (May 3, 2010), *available at* http://www.dec.ny.gov/docs/remediation_hudson_pdf/der10.pdf.	72

U.S. Department of Labor, Occupational Safety and Health Guideline for Uranium and Insoluble Compounds, *available at* http://www.osha.gov/SLTC/healthguidelines/uraniuminsolublecompounds/recognition.html.	73
New York City, Mayor's Office of Environmental Coordination, City Environmental Quality Review Technical Manual, Chapters 12, 19 & 22, Revisions (May 2010, July 2012), *available at* http://www.nyc.gov/html/oec/html/ceqr/technical_manual_2012.shtml.	74
Rob Beelen et al., *The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study*, OCCUP. ENVIRON. MED. (Nov. 18, 2008).	75
U.S. Department of Transportation, Federal Railroad Administration, General Health Effects of Transportation Noise (June 2002).	76
Mette Sorensen et al., *Road traffic noise and stroke: a prospective cohort study*, EUROPEAN HEART JOURNAL (Jan 2011).	77
Willy Passchier-Vermeer et al., *Noise Exposure and Public Health*, ENVIRONMENTAL HEALTH PERSPECTIVES, vol. 108, supplement 1 (March 2000).	78
Jenny Selander et al., *Long-Term Exposure to Road Traffic Noise and Myocardial Infarction*, EPIDEMIOLOGY vol. 2, no. 2 (March 2009).	79
Wolfgang Babisch et al., *Blood pressure of 8-14 year old children in relation to traffic noise at home – Results of the German Environmental Survey for Children (GerES IV)*, SCIENCE OF THE TOTAL ENVIRONMENT 407, 5839 (2009).	80
Wolfgang Babisch et al., *Traffic Noise and Risk of Myocardial Infarction*, EPIDEMIOLOGY vol. 16, no. 1 (Jan. 2005).	81
E. Ohrstrom et al., *Effects of road traffic noise and the benefit of access to quietness*, JOURNAL OF SOUND AND VIBRATION 295, 40 (2006).	82
Payam Dadvand et al., Maternal Exposure to Particulate Air Pollution and Term Birth Weight: A Multi-Country Evaluation of Effect and Heterogeneity, ENV. HEALTH PERSPECTIVES (Feb. 6, 2013), *available at* http://ehp.niehs.nih.gov/2013/03/1205575/.	83
Letter from Grace Musumeci, Chief, Environmental Review Section, U.S. Environmental Protection Agency, Region 2, to Gary Kassof, Bridge Program Manager, First Coast Guard District, re: Bayonne Bridge Navigation Clearance Project NEPA Workplan (Dec. 7, 2011).	84
Letter from John Formosa, Major Project Manager, U.S. Department of Transportation, Federal Highway Administration, to Mr. Gary Kassof, Bridge Program Manager, U.S. Coast Guard First Coast Guard District (Nov. 22, 2011).	85
Letter from Bill Baroni, Deputy Executive Director, The Port of New York and New Jersey, to Garry Kassof, Bridge Program Manager, US Coast Guard First Coast Guard District, re: Port Authority of New York and New Jersey, Bayonne Bridge Navigational Clearance Program (BBNCP), Application for a USCG Bridge Permit (Section 9), (July 28, 2011).	86
Letter from Michael Davies, P.E., Acting Division Administrator, U.S. Department of Transportation, Federal Highway Administration, to Christopher Ward, Executive Director, Port Authority of New York and New Jersey (May 5, 2011).	87

U.S. Army Corps of Engineers, IWR White Paper: The Implications of Panama Canal Expansion to U.S. Ports and Coastal Navigation Economic Analysis.	88
Martha Matsuoka et al., Global Trade Impacts: Addressing the Health, Social and Environmental Consequences of Moving International Freight Through Our Communities (Executive Summary) (Feb. 2011), *available at* http://www.uepi.oxy.edu/wp-content/uploads/2013/01/Pub-ES_Global_Trade_Impacts.pdf.	89
Port of Long Beach, Pier S Marine Terminal & Back Channel Improvements Project Draft Environmental Impact Statement (DEIS)/Draft Environmental Impact Report (DEIR) (Sept. 2011), *available at* http://www.polb.com/environment/docs.asp (excerpts).	90
The Star-Ledger Continuous News Desk, *Diesel fumes are found to cause cancer, N.J. residents at high risk* (June 13, 2012), http://www.nj.com/news/index.ssf/2012/06/diesel_fumes_are_found_to_caus.html	91
State of New Jersey, Department of Environmental Protection, Preparing a Diesel Risk Assessment, http://www.state.nj.us/dep/airtoxics/diesrisk.htm.	92
Mette Sorensen et al., *Road Traffic Noise and Incident Myocardial Infarction: A Prospective Cohort Study*, PLoS ONE, vol. 7, issue 6 (June 2012).	93
Michael G. Smith et al., *On the Influence of Freight Trains on Humans: A Laboratory Investigation of the Impact of Nocturnal Low Frequency Vibration and Noise on Sleep and Heart Rate*, PLoS One, vol. 8, issue 2 (Feb. 2013).	94
Mette Sorensen et al., *Long-Term Exposure to Road Traffic Noise and Incident Diabetes: A Cohort Study*, Env. Health Perspectives, vol. 121, no. 2 (Feb. 2013).	95
Kenneth I. Hume, *Noise Pollution: A Ubiquitous Unrecognized Disruptor of Sleep?*, SLEEP, vol. 34, no. 1 (2011).	96
Jaana I. Halonen et al., *Associations between Nighttime Traffic Noise and Sleep: The Finnish Public Sector Study*, ENV. HEALTH PERSPECTIVES, vol. 120, no. 10 (Oct. 2012).	97
G. Leon Bluhm et al., *Road traffic noise and hypertension*, OCCUP. ENVIRON. MED. (2007).	98

ATTACHMENT 98

ORIGINAL ARTICLE

Road traffic noise and hypertension

Gösta Leon Bluhm, Niklas Berglind, Emma Nordling, Mats Rosenlund

Occup Environ Med 2007;**64**:122–126. doi: 10.1136/oem.2005.025866

See end of article for authors' affiliations

Correspondence to: Dr G Bluhm, Department of Occupational and Environmental Health, Stockholm County Council, Norrbacka 3rd Floor, Karolinska University Hospital, SE-171 76 Stockholm, Sweden; gosta.bluhm@ki.se

Accepted 6 October 2006
Published Online First 19 October 2006

Background: It has been suggested that noise exposure increases the risk of hypertension. Road traffic is the dominant source of community noise exposure.
Objective: To study the association between exposure to residential road traffic noise and hypertension in an urban municipality.
Methods: The study population comprised randomly selected subjects aged 19–80 years. A postal questionnaire provided information on individual characteristics, including diagnosis of hypertension. The response rate was 77%, resulting in a study population of 667 subjects. The outdoor equivalent traffic noise level (Leq 24 h) at the residence of each individual was determined using noise-dispersion models and manual noise assessments. The individual noise exposure was classified in units of 5 dB(A), from <45 dB(A) to >65 dB(A).
Results: The odds ratio (OR) for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% confidence interval (CI) 1.06 to 1.80) per 5 dB(A) increase in noise exposure. The association seemed stronger among women (OR 1.71; 95% CI 1.17 to 2.50) and among those who had lived at the address for >10 years (OR 1.93; 95% CI 1.29 to 2.83). Analyses of categorical exposure variables suggested an exposure–response relationship. The strongest association between exposure to traffic noise and hypertension was found among those with the least expected misclassification of true individual exposure, as indicated by not having triple-glazed windows, living in an old house and having the bedroom window facing a street (OR 2.47; 95% CI 1.38 to 4.43).
Conclusion: The results of our study suggest an association between exposure to residential road traffic noise and hypertension.

Noise acts as a ubiquitous stress-mediating factor in the physical environment. General annoyance, disturbances in psychosocial well-being and reduction in sleep quality are commonly reported effects of noise exposure.[1 2] An increased risk of non-auditory physiological effects due to noise, such as hypertension and ischaemic heart disease, have also been suggested.[3–8] Most previous studies have been performed in occupational settings with high noise levels.[5 6 8–10] Community noise is less well studied.

Road traffic is the dominating source of community noise in the urban environment. Few studies have investigated an association between exposure to road traffic noise and hypertension, and the results are conflicting.[3 4 11] Studies in this field have low precision and validity problems, including crude exposure assessments, selection bias and limited control of important confounding factors. Exposure has usually been assessed either from subjective reports or without consideration of important factors that may influence the individual exposure level—for example, window type, bedroom window orientation and type of residence.

The suggested biological mechanism for an association between exposure to community noise and hypertension is that noise induces stress by disturbing sleep and interfering with relaxation and concentration and many other cognitive effects that activate the sympathetic nervous system and the endocrine system.[12] The primary physiological effects of noise exposure are vegetative reactions such as increase in blood pressure, heart rate and finger pulse amplitude, cardiac arrhythmia, and changes in respiration and body movements.[13] Therefore, a hypothesis has emerged that stress due to persistent exposure to environmental noise could result in permanent vascular changes, with increased blood pressure and ischaemic heart disease as potential outcomes.[14–16]

Our objective was to study a possible association between exposure to residential road traffic noise and hypertension among adults in an urban municipality. To better characterise individual noise exposure, we aimed at investigating factors that may influence the true exposure level, such as window type, bedroom window orientation and type of residence.

METHODS

Study population

The study was performed in a municipality with 55 000 inhabitants located 15 km north of Stockholm City. A questionnaire designed for a countywide investigation of health effects related to various environmental factors was distributed in April 1997 to 1000 individuals aged 19–80 years living in the municipality.[17] A stratified random sampling procedure was applied to ensure a sufficient number of subjects exposed to traffic noise, consisting of two strata with 500 residents in each. The noise-exposed group was drawn from those living within 100 m on each side of the highway, main roads or the railway. The other sample was drawn from the remaining parts of the municipality. Statistics Sweden performed the sampling by combining the National Population Register (containing background information for the study population) with the Real Estate Register (containing geographical coordinates for the residence of each individual). The response rate was 77% in both samples. This study focused on exposure to road traffic noise; thus, subjects who were residing close to the railway (n = 91) were not included. One subject who had removed the identification sticker from the returned questionnaire was excluded, as we did not have the address of that subject. In total, the study comprised 667 subjects.

Questionnaire

The survey included 87 questions and was mainly focused on prevalence of allergic diseases and environmental risk factors of regional importance. Information on educational level, employment status, general living conditions, and smoking habits was

Table 1 Number of study subjects in different noise exposure classes according to method of exposure assessment

Noise exposure	Classified by dispersion model: National Road Administration	Sollentuna municipality	Manually classified	Total
⩽45 dB(A)	0	0	125	125
45–50 dB(A)	0	0	120	120
50–55 dB(A)	96	0	209	305
55–60 dB(A)	20	25	47	92
60–65 dB(A)	2	9	12	23
>65 dB(A)	2	0	0	2
Total	120	34	513	667

provided. Data on annoyance from traffic noise and sleep disturbance due to noise were also collected. Hypertension was defined as a positive answer to the question ''Have you been diagnosed with hypertension by a physician during the past five years?''. Individual information on background factors such as age, sex and ethnic background was obtained from the National Population Register.

Exposure assessments

The exposure to outdoor residential road traffic noise during 1997 was assessed for all subjects in A-weighted average sound pressure levels (dB(A)) and expressed as the annual mean 24-h equivalent noise level (Leq 24 h). The individuals were classified into exposure categories of 5 dBA, from ⩽45 dB(A) to >65 dB(A), according to the noise level at their residence (table 1). For the six-lane highway that intersects the municipality, the Swedish National Road Administration calculated the noise propagation using a validated Nordic prediction model for road traffic noise.[18] This dispersion model covered the addresses of 120 subjects. The Sollentuna Environment and Health Protection Administration applied a similar prediction model to classify exposure around other major roads in the area in 55–60 and 60–65 dB(A) exposure categories. This model covered another 34 subjects. For residences not covered by any of these models (n = 513), the noise exposure was manually classified into groups of 5 dB(A) by an environmental health officer with extensive knowledge of the local traffic noise propagation and blinded to the outcome status. Important parameters for all assessment methods were traffic flow, geographical location and existing noise measurements.

Table 2 presents the characteristics of the study subjects, including living conditions and selected lifestyle factors.

Statistical analysis

The association between exposure to road traffic noise and hypertension was investigated using logistic regression, and is presented as odds ratios (OR) with 95% confidence intervals (95% CI). In most analyses, exposure to road traffic noise was used as a continuous variable and the results are presented per 5 dB(A) increase in noise exposure. As the exposure measure was determined in 5 dB(A) wide classes, the continuous variable used the class middle for everyone in that class. The top and bottom classes were open, and the subjects in these classes were given a value of 2.5 dB(A) from the nearest class boundary. In the analysis using a categorical exposure variable, subjects exposed to noise levels of 60–65 dB(A) and >65 dB(A) were merged with those exposed to 55–60 dB(A) owing to small numbers in the top categories of exposure. The final multiple logistic regression model included age as a linear term,

Table 2 Background characteristics of study subjects in Sollentuna, Sweden 1997

	n (%)	Mean (SD)
Age (years)		**48 (16)**
Sex		
Male	310 (46)	
Female	357 (54)	
Smoking		
Never smokers	341 (52)	
Former smokers	187 (28)	
Number of cigarettes		14 (8)
Current smokers	131 (20)	
Number of cigarettes		13 (8)
Occupation		
Within the working force*	465 (73)	
Retired	100 (15)	
Otherwise outside the working force†	74 (12)	
Type of residence		
Apartment	278 (42)	
Single family house	384 (58)	
Duration of residence (years)		
<1	75 (11)	
1–10	293 (44)	
>10	297 (45)	
Window type		
Triple glazing	418 (65)	
Double glazing	228 (35)	
Building year		
Up to 1975	423 (66)	
After 1975	215 (34)	
Bedroom window		
Facing the street	331 (51)	
Facing other	318 (49)	

*Employed, self-employed, on parental leave, student.
†Homemakers, unemployed, on disability or sick leave.

an indicator variable for house type, occupation in three levels, smoking status and amount smoked for former and current smokers. Model selection was based on the evaluation of the influence of each covariate on the effect estimate of exposure to road traffic noise on hypertension. Only those with complete data on all covariates were included in the multiple logistic regression analyses. To explore the potential modification of the effect of noise exposure, an interaction term between the covariate and the noise variable was included in the model, and p values for the interaction term are presented. All statistical analyses were performed with Stata V.8.2.

RESULTS

Altogether, 80 (13%) subjects in the whole study population were diagnosed with hypertension. The OR for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% CI 1.06 to 1.80) per 5 dB(A) increase in exposure to road traffic noise. Analyses using categorical exposure variables suggested an exposure–response relationship between road traffic noise and hypertension (table 3). Analyses of potential modification of the effect of road traffic noise on hypertension by other factors showed a stronger association among those who had lived at their residence for >10 years (OR 1.93; 95% CI 1.29 to 2.83), those who lived in a house built before 1976 (OR 1.83; 95% CI 1.29 to 2.61) and those who had their bedroom windows facing the street (OR 1.82; 95% CI 1.22 to 2.70; fig 1). A stronger effect was also suggested for those living in single-family houses (OR 1.74; 95% CI 1.20 to 2.51) and those who did not have triple-glazed windows (OR 1.66; 95% CI 1.17 to 2.34). There was some indication that the effect was stronger among female subjects (OR 1.71; 95% CI 1.17 to 2.50), although the sex difference was not significant.

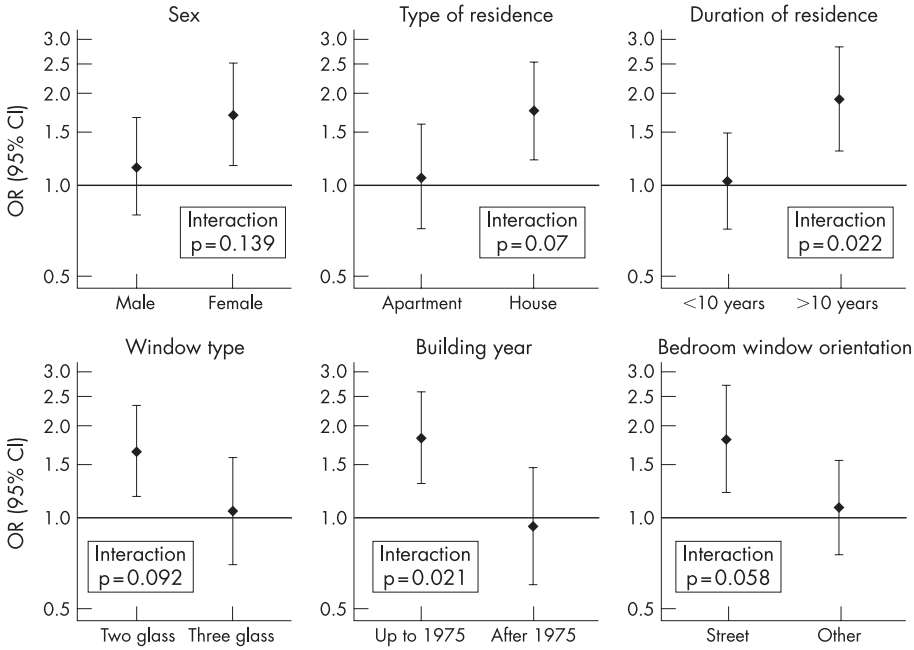

Figure 1 Odds ratios (OR) and 95% confidence intervals (CI) for hypertension associated with a 5 dB(A) increase in exposure to road traffic noise by sex, type of residence, duration of residence, window type, building year and bedroom window orientation. Odds ratios were adjusted for age, type of residence, occupation, smoking status and number of cigarettes.

To better characterise individual exposure, we used information on those residential factors that are likely to influence the true individual exposure level. The indicators of misclassification of true exposure were houses built after 1975 (when new Swedish regulations for building constructions including higher standards for thermal isolation were settled), houses equipped with triple-glazing (that reduce indoor noise levels) and bedroom windows not directly facing the street (that result in reduced exposure to night-time noise). The results indicate stronger associations with decreasing misclassification of the true individual noise exposure, up to an OR of 2.47 (95% CI 1.38 to 4.43) in the group where we expect least exposure misclassification (table 4).

Several variables exerted confounding on the association between exposure to traffic noise and hypertension, and were thus adjusted for in the analyses (age, residence type, occupational level and smoking). Many other potential confounders were also evaluated—for example, education and hearing loss, but these factors did not influence the effect estimate (fig 2). Excluding those who had lived at their residence for <1 year did not affect the results. In addition, there was virtually no difference in effect estimates using either of the different methods for noise exposure assessment separately, suggesting that these methods assessed exposure equally well.

DISCUSSION

We found an association between exposure to road traffic noise and hypertension. Other studies have reported an association between hypertension and occupational noise exposure[5 6 8] or exposure to aircraft noise.[19–21] There is a lack of previous epidemiological data linking exposure to road traffic noise and hypertension, although a few studies have suggested some association.[3 4] In addition, a recent study reported an increased risk of myocardial infarction among men associated with long-term exposure to road traffic noise.[7]

The results of our study point to a linear exposure–response relationship between road traffic noise and hypertension at lower noise levels compared with previous reports. In a study of noise exposure and annoyance or sleep disturbances in the same study population, we found a distinct exposure–response relationship.[22] These findings indicate that our method of determining individual exposure to road traffic noise is reasonably valid in terms of perception of noise exposure. In addition, we combined two ways of exposure assessment, using calculated dispersion models and manual classification, and both produced virtually the same results.

We evaluated confounding from many factors including smoking and occupational status, but residual confounding may still be present, especially from noise at other locations, for example, at work. In addition, some factors that may act as risk

Table 3 Association between exposure to road traffic noise and hypertension (n = 608)

	n	Number with hypertension (%)	OR (95% CI)*
Continuous (per 5 dB(A) increase)			1.38 (1.06 to 1.80)
Category (db(A))			
⩽45	115	6 (5)	1.00 (reference)
45–50	105	13 (12)	1.74 (0.60 to 5.01)
50–55	281	39 (14)	2.07 (0.82 to 5.24)
>55	107	22 (21)	3.47 (1.27 to 9.43)

Adjusted for age, type of residence, occupational status, smoking status and number of cigarettes.

Table 4 The association between exposure to road traffic noise per 5 dB(A) and hypertension according to the number of indicators of potential exposure misclassification (n = 559)

Indicators of exposure misclassification*	n	OR (95% CI)†
3 of 3	67	0.83 (0.43 to 1.60)
2 of 3	120	0.98 (0.56 to 1.73)
1 of 3	221	1.47 (0.93 to 2.33)
0 of 3	151	2.47 (1.38 to 4.43)

*Triple-glazed windows, building built after 1975; bedroom window not directly facing the street.
†Adjusted for age, type of residence, occupational status, smoking status and number of cigarettes.

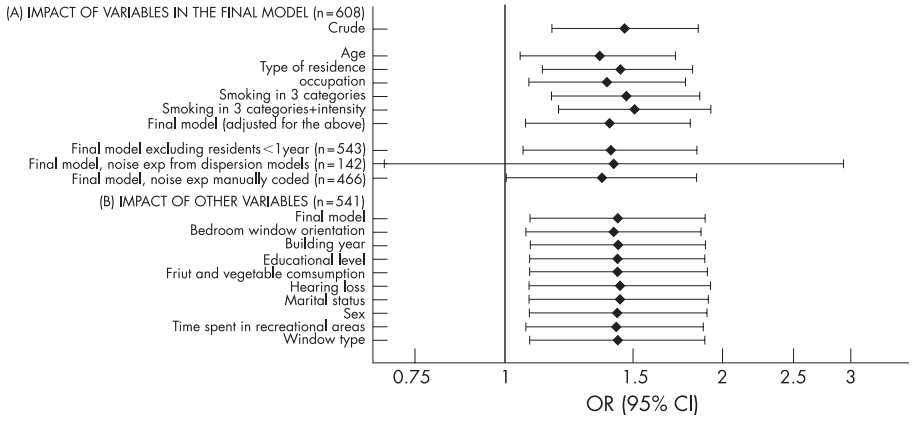

Figure 2 Odds ratios (OR) and 95% confidence intervals (CI) for hypertension associated with a 5 dB(A) increase in exposure to road traffic noise. (A) The individual and total confounding effect on the point estimate from the variables included in the final model; (B) the additional confounding effect from variables not included in the final model.

factors for hypertension were not recorded—for example, alcohol consumption and physical inactivity—although a strong association with exposure to residential road traffic noise seem unlikely, making bias due to confounding from such factors less likely.

Taking hearing loss into account did not seem to change the results. A positive relationship between hearing loss and hypertension has previously been found.[23] However, the scientific support for an association between hearing loss and hypertension is weak and somewhat speculative, and the interrelationship has to be interpreted with caution.

Disease outcome in this study was based on self-reported diagnosis of hypertension, which might be a source of bias. However, it has been reported that self-administered questionnaires may have good accuracy to confirm hypertension.[24] As exposure was assessed objectively by geographical dispersion models or by an operator blinded to disease status, the data on exposure and outcome were collected independently, making differential misclassification of exposure or disease less likely. In addition, the high response rates reduce the possibility that the results were strongly influenced by selection bias.

Although our study was cross-sectional, we had access to crude data on duration of residence in the categories <1 year, 1–10 year and >10 years. Stratification on that variable indicated an association primarily among those who had lived at the address for at least 10 years, suggesting that least misclassification of true individual exposure in that group or that 10 years of exposure might be needed to exert an effect. As non-differential misclassification of exposure is important to consider, we especially focused on several other factors that are likely to affect the individual exposure to road traffic noise. These include triple-glazed windows that have a noise-isolating effect, modern buildings that are better isolated and bedroom windows that do not directly face the street. All these factors are supposed to result in lower night-time exposure levels. Indeed, when none of these factors were present—that is, where we assume that the modelled exposure level better reflects the true individual exposure level—the association was particularly strong (OR 2.47; 95% CI 1.38 to 4.43).

The association seemed stronger among women than among men. Although there may be biological reasons—for example, the use of hormonal contraceptives that could explain such differences—it may also be due to chance or different patterns in misclassification of exposure. The relationship was also stronger among those living in single-family houses than among those living in apartments. These findings could partly be due to differences in building construction. Three glass windows were present in 50% of the apartments compared with 27% of the single-family houses.

In conclusion, our results suggest an association between residential exposure to road traffic noise and hypertension. This implies that road traffic noise may be a risk factor for cardiovascular disease.

ACKNOWLEDGEMENTS

We thank Dan Sjöberg at the Sollentuna Environmental and Health Protection Administration for invaluable help in assembling noise data and estimating noise exposure.

Authors' affiliations

Gösta Leon Bluhm, Unit of Environmental Epidemiology, Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden
Niklas Berglind, Emma Nordling, Mats Rosenlund, Department of Occupational and Environmental Health, Stockholm County Council, Stockholm, Sweden

Funding: Stockholm County Council and Sollentuna Municipality provided funding for the questionnaire survey and noise exposure assessment. These authorities had no role in the conduct of the study, interpretation of the results or the decision to send the study for publication.

Competing interests: None declared.

An ethics committee was consulted, but a formal approval was judged unnecessary for this study.

REFERENCES

1 **Öhrström E**. Psycho-social effects of traffic noise exposure. *J Sound Vibr* 1991;**151**:513–17.
2 **Griefahn B**, Scheumer-Kohrs A, Scheumer R, *et al*. Physiological, subjective and behavioural responses during sleep to noise from rail and road traffic. *Noise Health* 2000;**3**:59–71.
3 **Knipschild P**, Salle H. Road traffic noise and cardiovascular disease, a population study in the Netherlands. *Int Arch Occup Environ Health* 1979;**44**:55–9.
4 **Herbold M**, Hense HW, Keil U. Effects of road traffic noise on prevalence of hypertension in men: results of the Lubeck blood pressure study. *Soz Praventivmed* 1989;**34**:19–23.
5 **Fogari A**, Zoppi A, Vanasia G, *et al*. Occupational noise exposure and blood pressure. *J Hypertens* 1994;**12**:475–9.
6 **Powazka E**, Pawlas K, Zahorska-Markiewicz B, *et al*. A cross-sectional study of occupational noise exposure and blood pressure in steelworkers. *Noise Health* 2002;**17**:15–22.
7 **Babisch W**, Beule B, Schust M, *et al*. Traffic noise and risk of myocardial infarction. *Epidemiology* 2005;**16**:33–40.
8 **Davies HW**, Teschke K, Kennedy SM, *et al*. Occupational exposure to noise and mortality from acute myocardial infarction. *Epidemiology* 2005;**16**:25–32.
9 **Hirai A**, Takata M, Mikawa M, *et al*. Prolonged exposure to industrial noise causes hearing loss but not high blood pressure: a study of 2124 factory laborers in Japan. *J Hypertens* 1991;**9**:1069–73.
10 **Hessel PA**. Occupational noise exposure and blood pressure: longitudinal and cross-sectional observations in a group of underground miners. *Arch Environ Health* 1994;**49**:128–34.
11 **Babisch W**, Ising H, Gallacher JEJ, *et al*. Traffic noise and cardiovascular risk: the Caerphilly study, first phase. Outdoor noise levels and risk factors. *Arch Environ Health* 1988;**43**:407–14.

12 **Babisch W**, Fromme H, Beyer A, *et al*. Increased catecholamine levels in urine in subjects exposed to road traffic noise. The role of stress hormones in noise research. *Environ Int* 2001;**26**:475–81.
13 **Berglund B**, Lindvall T. Community noise. Stockholm. Archives of the Center for Sensory Research. Stockholm: University and Karolinska Institutet, 1995.
14 **Lundberg U**. Coping with stress: neuroendocrine reactions and implications for health. *Noise Health* 1999;**4**:67–74.
15 **Babisch W**. Traffic noise and cardiovascular disease: epidemiological review and synthesis. *Noise Health* 2000;**8**:9–32.
16 **Stansfeld S**, Haines M, Brown B. Noise and health in the urban environment. *Rev Environ Health* 2000;**15**:43–82.
17 **Miljömedicinska enheten (Deparment of Environmental Health)**. *Miljöhälsorapport 1998. Om samband mellan miljö och hälsa i Stockholms län (Environmental health report about relations between environment and health in Stockholm County)* (In Swedish). Stockholm: Stockholm County Council, 1998.
18 **Swedish Environmental Protection Agency**. Swedish National Road Administration. Nordiska Ministerrådet. Vägtrafikbuller. Nordisk beräkningsmodell, reviderad 1996 (Road traffic noise. Nordic prediction model, revised 1996). Report 4653 (in Swedish). Stockholm: Naturvårdsverkets förlag, 1999.
19 **Knipschild P**, Oudshoorn N VII. Medical effects of aircraft noise. Drug survey. *Int Arch Occup Environ Health* 1977;**40**:197–200.
20 **Rosenlund M**, Berglind N, Pershagen G, *et al*. Increased prevalence of hypertension in a population exposed to aircraft noise. *Occup Environ Med* 2001;**58**:769–73.
21 **Franssen EAM**, van Wiechen CMAG, Nagelkerke NJD, *et al*. Aircraft noise around a large international airport and its impact on general health and medication use. *Occup Environ Med* 2004;**61**:405–13.
22 **Bluhm G**, Nordling E, Berglind N. Road traffic noise and annoyance—an increasing environmental health problem. *Noise Health* 2004;**6**:43–9.
23 **Talbott EO**, Findlay RC, Kuller LH, *et al*. Noise-induced hearing loss: a possible marker for high blood pressure in older noise-exposed populations. *J Occup Med* 1990;**32**:690–7.
24 **Okura Y**, Urban LH, Mahoney DW, *et al*. Agreement between self-report questionnaires and medical data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure. *J Clin Epidemiol* 2004;**57**:1096–103.

Road traffic noise and hypertension

Gösta Leon Bluhm, Niklas Berglind, Emma Nordling, et al.

Occup Environ Med 2007 64: 122-126 originally published online October 19, 2006
doi: 10.1136/oem.2005.025866