

**U.S Coast Hearing on Draft FONSI for Ambassador Bridge Enhancement
Project
Testimony
March 17, 2009**

My name is Rashida Tlaib and I am a resident serving this district as State Representative. As State Representative of the host community for the Ambassador Bridge Enhancement Project, I am submitting the following comments in opposition to the issuance of a Finding of No Significant Impact (FONSI).

Southwest Detroit is home to one of the most diverse communities in Michigan with a vibrant and growing business district in Detroit. More importantly it is the only part of Detroit that is growing in population. This is true even though the community has faced tremendous challenges that many communities would not survive—we host the Detroit Salt Mine, Marathon Oil Refinery, three freeways dividing up our neighborhoods, the largest rail yard in the state and the Ambassador Bridge and 30% of our residents live below poverty, while air quality in Southwest Detroit is among the worst in the City.

The National Environmental Policy Act (NEPA) provides for a comprehensive process mandated by federal law to ensure the conduct of all federal agencies is protecting the human health and environment of the public. An Environmental Assessment, a lower tier process, was completed for this massive project and the findings failed to recognize that there is a significant impact on Southwest Detroit and a full Environmental Impact Study is needed.

My residents deserve much more than a short cut EA process. It is apparent from the sheer number of residents attending this hearing concerned about their human environment. A project of this size, a controversial one at that, must undertake a full Environmental Impact Study with sufficient evaluation, notice and comment process.

The Detroit International Bridge Company (DIBC) claims that bridge traffic has significantly decreased, so there is time for a full and comprehensive Environmental Impact Study that is transparent and open for my community members to comment. It will also provide enough time to take a closer look at the impact on their human health, their children's health and their property values. A simple EA does not balance different kinds of positive and negative environmental effects, one against the other, nor does it weigh environmental impacts against a project's other objectives. A proposal to twin the Ambassador Bridge will have significant impacts on the quality of the human environment in Southwest Detroit, so a full EIS is required.

The issuance of a "Finding of No Significant Impact" (FONSI) in twinning the Ambassador Bridge is concerning due to a number of issues: 1) the fact that the EA did not include the whole, but only segment of the project, which makes it flawed, 2) the nearby Gateway Project changes undermine the EA in allowing bridge traffic into my neighborhoods, 3) there are a number of

disputes on the usage and ownership of surrounding property, 4) it is uncertain that the current Ambassador Bridge will not remain open, 5) lack of independent analysis and consultation with relevant agencies, and 6) the fact that a bi-national study between Canada and U.S. rejected the Twin Span of the Ambassador Bridge, recognizing the impact on air quality.

1) The Environmental Assessment completed on the project is not inclusive of the whole project.

There is a clear issue of segmentation that is reflected by the fact that the EA does not include the entire plan for the border crossing. The EA does not include the usage of Fort Street, city owned roads, impact on Riverside Park, and the massive design changes to the Gateway Project. It is clear by the construction on the ground and the discrepancy in previous statements by DIBC that the EA clearly does not reflect DIBC's intent for the border crossing. According to a letter by DIBC addressed to Michigan Department of Transportation (MDOT), it states the need to expand the Ambassador Bridge's operations "in order to be able to efficiently process international traffic." (May 17, 2006 DIBC letter) There is no indication that the State and/or MDOT will allow any permanent usage of Fort Street. There is a deliberate process of segmentation, which spoils the EA in this project.

2) The fact that the nearby Gateway Project's EA has been undermined by the recent DIBC actions to allow bridge traffic into our neighborhoods.

DIBC design changes to the Gateway Project, which DIBC claims to be an integral part of the Enhancement Project is jeopardizing the "purpose and need" of the project. According to a letter from the Michigan Department of Transportation (MDOT), DIBC proposed five changes to the original Gateway Project that violate the purpose and need of the project. This includes the usage of: 1) 23rd Street, 2) Fort Street, 3) West Grand Boulevard, 4) I-75 Service Drive and 5) easement onto a private parcel. The irreparable harm on the community from these design changes and how it relates to the Enhancement Project must be considered under the NEPA process. The construction taking place outside of the Gateway EA prejudices the outcome of the NEPA decision-making process.

3) There are a number of controversies with regard to the Detroit International Bridge Company's right to construct the Bridge when there is dispute on ownership and usage of surrounding property.

There have been significant changes in the existing land use during the Gateway Project and the possibility building piers on Riverside Park that raises serious doubt that property has been properly acquired for this project. Under the Bridge Administration Manual, any Bridge Program issues or actions that are, or have the potential of becoming controversial, or involve or may involve litigation, shall be forwarded to the Commandant (G-OPT) for information and decision regarding action to be taken on such issues or actions. Such projects/issues may involve controversy between federal, state and local agencies, disagreements with the bridge owners or applicants, controversy with environmental and navigational entities, political interest, precedent setting, environmental impact statements, etc.

There has been litigation between the City of Detroit and DIBC regarding the use of Riverside Park. The current City of Detroit administration has no intention of selling Riverside Park and according to a June 27, 2007 memo from the City of Detroit, "building permits would be required to construct the new span, supports and related or accessory structures." According to the City of Detroit, DIBC has not applied for such permits for the Twin Span.

Recently, DIBC stated in written response to the Michigan Strategic Fund for the Enhancement Project that "all property has been acquired to allow for the construction of both phases." (MSF, May 25, 2007)

In addition, it is clear that changes to roadways are being permanently altered to implement the project. The final EA on ABEP states that "no changes to the plazas or the local roadways will be made as a result of the proposed project." (ABEP Section 3.2.4.) In a response to questions, it stated that "no local roadways will be permanently altered to implement the project." Did the USCG confirm that with the City of Detroit and the State of Michigan?

4) Was the Environmental Assessment completed in anticipation of ten lanes of International Bridge Traffic?

DIBC has not committed to closing down the current Ambassador Bridge if the so called "replacement bridge" is built. There have been no clear commitments that this Twin Span will retire the existing Ambassador Bridge. DIBC has flip-flopped on this issue and there continues to be a lack of transparency in understanding the future plan. If a condition is placed on DIBC to close the existing bridge, who will enforce and how?

The DIBC's intention is to use both bridges which amount to ten lanes of international bridge traffic going into my community on a daily basis. An EA was not conducted with this as a consideration.

5) The Environmental Assessment process lacked independent analysis and consultation with the City of Detroit, Michigan Department of Environmental Quality and the Environmental Protection Agency.

According to federal law, if an agency permits an applicant to prepare an environmental assessment, the agency must make its own evaluation of the environmental issues and take responsibility for the scope and content of the environmental assessment. DIBC contracted with a consultant in Florida to conduct the EA on this massive project. What consultation was done with other government agencies? In 2007, the Environmental Protection Agency (EPA) criticized the limited scope of the environmental study being conducted on this project.

6) A bi-national study rejected the twin span proposal recognizing the impact on air quality as a major factor.

The host community of the Ambassador Bridge exhibits one of Detroit's highest rates of persistent asthma for children. This childhood asthma prevalence reflects the already stained environment in Southwest Detroit's neighborhoods.

The DRIC study criticized the twin spanning based on its impact on air quality and yet this Draft FONSI claims there will be no impact on the local environment. The study ranked it *14th in Regional Mobility and almost last in the Air Quality and Protecting Cultural Resources areas.* (DRIC Evaluation of Illustrative Alternatives, Vol. 1 S-41) It was ranked *35 out of 37 options based on impact on Air Quality.* (Table A-3, DRIC Evaluation of Illustrative Alternatives, vol. 1).

Southwest Detroit has endured displacement and anguish as a result of the power that the Bridge Company has over local, state and federal agencies. My community doesn't have millions of dollars to hire its own firm to conduct an EA, but my residents have a better meter: their own personal stories of how their community, life and enjoyment of property has been impacted by the lack of transparency by DIBC and the continued changes to already agreed upon projects. Alone the controversial long history of DIBC's failed commitments and backdoor dealings should trigger a closer look at the adverse impacts.

If the FONSI stands, the USCG will have neglected its duties under NEPA and their own Bridge Administrative Manual. The federal government has a responsibility to the public to protect them from irreparable harm. This is the legislative intent of NEPA. The EA conducted on this project is questionable. My community members and I don't have the money or resources to understand how the USCG process worked in this instance and we certainly do not have the luxury of any short cuts. My community doesn't rely on DIBC. We depend on our own government to protect us from private companies such as this from cutting corners and manipulating the system so they can make a profit off our great City of Detroit. There are too

many questions raised in this project and the only ones who seem to have the answers are DIBC's attorneys.

I want to publicly thank Congressman John Dingell for requesting this hearing and allowing my residents concerns to be heard.

Full

ENVIRONMENTAL
STUDY
NEEDED



PLEASE

SAVE

OUR

LIVES!

UNITED STATES COAST GUARD
PUBLIC MEETING

COMMENT FORM

AMBASSADOR BRIDGE ENHANCEMENT PROJECT

In the space below, please provide your comment regarding the Ambassador Bridge Enhancement Project. All comments received will be posted to <http://www.regulations.gov> under the Coast Guard docket number USCG-2009-0093.

Please submit to a Coast Guard official before the end of this public meeting.

DO

THE

Big
part

thing
for

PLEASE SAVE
OUR NEIGHBORHOOD.
I have Asthma
and SEVERE Lung
PROBLEMS. I'M ON
a FIXED INCOME
and cannot AFFORD
TO MOVE. THE SMOKE
FROM THE BRIDGE
AFFECTS OUR
NEIGHBORHOOD, OUR
HEALTH, OUR
CHURCHES, ST. ANNE,

Holy Redeemer
Gessiah, I
DESTROY our QUALITY
OF LIFE. The
Pollution is Killing
us. I am not speaking
FOR MYSELF but all
who LIVE in this
AFFECTED AREA.
MY FATHER
BELONGED to the
COAST GUARD and
proudly DEFENDED
the Great Lakes
and RIVERS. IF HE
WAS a WIFE HE
WOULD "Fight
the good Fight
and ~~stop~~ stop the
Embassad of Bridge.
From the existing
plan to set up
expansion of the
BRIDGE. Am M. Boutt 313-
242-2521

EX Representative / speaker
STEVE TOBOCMAN

Ambassador Bridge Enhancement Project (ABEP)
U.S. Coast Guard Environmental Analysis (EA) Hearing
March 17, 2009

Comments of Steve Tobocman

1. Introduction:

- Homeowner for 7 years 1032 Vinewood; 1 block from W. Grand Blvd. entrance
- Asthma sufferer (inhaler within six months of moving to new home)
- State Representative for 6 years, most recently House Majority Floor Leader
- Community advocate on transportation projects and issue dating back to 2000. Extensive involvement with DIBC, MDOT, ABEP, DIFT, DRIC, Gateway, DRTP, etc.

2. This EA Is Second-Rate Treatment: Experience Attending First DIBC Hearing on ABEP. *[Insert Steve's story about first hearing.]*

3. Factual Inaccuracies Suggest USCG Cannot Grant this EA

- Coast Guard Handbook 4-3 states: *"Federal approval is not granted when there is doubt of the right of the builder to construct and utilize a bridge. (Canadian approvals?). Bridge permit applications shall not be accepted for processing if upon receipt it is evident that the bridge project is under an existing injunction or any other legal obstruction, of if there are any doubt concerning the applicant's property rights."*
- New information on Riverside Park should stop this process.
- DIBC does not possess air rights over Fort Street, a state route. This should stop process.
- Draft FONSI, Section IV, pp. 7-8, *"The ABEP is not expected to require residential or commercial relocations, alter approved traffic projections, route traffic onto local roadways or neighborhoods . . ."* Gateway is now seeking to take part of Fort Street, close 23rd Street and otherwise impact local roadways and neighborhoods, making this inaccurate.
- Even the air and noise analysis suggests there can be no modification of Gateway without a new EA or EIS under NEPA. Appendix A, p. 202 *"Any such modification to the plaza would have to be evaluated under a separate proposal and would require a separate environmental study."* Air analysis done on closing existing span, so please restrict permit to replacement bridge.

4. Segmentation Problems with this EA

- I believe this EA is a deliberate attempt to segment the process on the part of DIBC. There are several examples of necessary components for, and, in fact, construction of the ABEP that are contained in the Gateway.
- The FONSI is clear that according to Section V, p. 10 "*The Coast Guard . . . views the ABEP as a natural extension of the Gateway Project . . .*"
- Construction of the "Dukes of Hazzard" Bridge to Nowhere that DIBC has constructed (under the Gateway) is evidence that it considers this one project. It is building ABEP under Gateway.
- PAB application to MSF discusses Phase One and Phase Two of one project they seek federal tax-exempt bond financing for.

5. Changing Nature, Some Say Changing Story and Inconsistencies of DIBC, Make the EA Invalid

**Sean Mann, Hubbard Farms resident,
Testimony for Coast Guard hearing on Draft FONSI for Ambassador Bridge
Enhancement Project
March 17, 2009**

My name is Sean Mann and I am a homeowner in Hubbard Farms. I am providing testimony tonight because I firmly believe that this Draft Finding of No Significant Impact for the Ambassador Bridge Enhancement Project is inaccurate, technically flawed and leaves too many questions unanswered.

Property Rights

The first question that the Draft FONSI raises in my mind is, where is the Ambassador Bridge's twin span going to be located?

The cover of the Draft Environmental Assessment contains an illustration of the proposed twin span that would be situated just to the west of the existing span with the towers for the cable-stayed span being situated in the City of Detroit's Riverside Park. Similar drawings and illustrations can be found in Appendix B of the Final Environmental Assessment.

While trying to secure Private Activity Bonds, the Detroit International Bridge Project in a May 25, 2007 letter to the board of the Michigan Strategic Fund to answer questions posed at a previous MSF meeting stated that they had all the necessary land for their project:

"All property has been acquired to allow for the construction of both phases of the project."

My problem with this is the fact that the DIBC has not acquired the property or proper easements from the City of Detroit for the use of Riverside Park where the towers would be situated.

A February 19, 2009 memo by the City of Detroit's City Planning Commission indicates the current mayoral administration "has no plans to sell or lease the subject portion of the Riverside Park to the DIBC." In fact the City of Detroit took legal action in the summer of 2008 to evict the DIBC from Riverside Park, who occupied a significant portion of the park after September 11, 2001 under the guise of national security concerns but have been utilizing it for storage.

Section 1.7 of the Final Environmental Assessment recognizes the fact that the DIBC has not obtained the land or easements that are owned by the City of Detroit:

"Currently, negotiations are in progress for property rights to parcels owned by the City of Detroit, i.e. bridge support piers location. The completion of these negotiations is subject to determination of whether the property will be owned by DIBC or whether an easement will be granted. All property or rights will be acquired before construction is begun."

B

The City of Detroit's resistance to sell or lease Riverside Park to the DIBC and the Final EA's recognition that the DIBC has not acquired all needed properties is significant and brings into question the entire application for a bridge permit because of existing Coast Guard policies. In the Coast Guard's Bridge Administration Manual (COMDTINST M16590.5C) on pages 4-3 and 4-4 under the section titled "Bridge Permits" the guidelines states:

"Federal approval is not granted when there is doubt of the right of the builder to construct and utilize a bridge. Bridge permit applications shall not be accepted for processing if upon receipt it is evident that the bridge project is under an existing injunction or any other legal obstruction, or if there are any doubts concerning the applicant's property rights."

Based on this guideline it is not clear to me how the Coast Guard could have accepted the application for the bridge permit let alone proceeded to the point of a Draft FONSI when the applicant has not acquired the necessary property and is currently involved in a lawsuit with the current owner over the property. The city's lawsuit against the bridge company has been adjourned until May 2009 and certainly the issuance of a permit should be delayed until this legal matter has been resolved.

Riverside Park

I take issue with the Final EA's assertion that Riverside park will not be affected by the ABEP. Or as Section 3.2.5 of the Final EA puts it.

The Proposed Project will not directly impact Riverside Park. Pier construction will be located to avoid the areas of the park that are accessible to the public.

The ABEP would permanently affect a portion of Riverside Park by placing towers in the park and having a six lane structure running immediately above it. It is unclear in the Final EA whether the Coast Guard made any effort to contact Homeland Security officials to determine if the DIBC was ever asked or granted permission to fence off a portion of Riverside Park?

Until the City's lawsuit against the DIBC is resolved and the right of the DIBC to fence off a section of the park is determined it is overly presumptive to assume any portion of the park will be permanently inaccessible to the public and thus not affected by the new span.

Furthermore, why should we believe that more of the park won't be fenced off to 'protect' the new towers as the DIBC has already done with the existing structures without any formal approval or guidance from the Department of Homeland Security?

Canadian Approval

Canadian approval has not yet been granted for the ABEP. Until the appropriate permits have been issued by Canadian authorities the ultimate feasibility of the project is far from certain. That being the case I would hope that the Coast Guard would follow their previous mentioned guideline that "Federal approval is not granted when there is doubt of the right of the builder to construct and utilize a bridge" until Canadian authorities have issued the necessary permits that would allow the project to move forward on both sides of the border.

Construction of ABEP

I am deeply concerned by the construction that the DIBC has undertaken next to the existing span. It is quite apparent that the DIBC has already commenced construction of the second span of the Ambassador Bridge as proposed in the ABEP before obtaining appropriate permits and finalizing the requirements of the National Environmental Protection Act (NEPA).

In the Environmental Assessment for the Gateway Project (ABGP) clearly show that the Gateway Project in regards to the ramp for the span, ends well before it approaches Fort Street. Similarly the drawings provided by the DIBC for the ABEP in Appendix B of the Final EA show that the project begins at a similar spot significantly north of Fort Street. However any layman can see by driving down Fort Street that there are now piers for a new span that are immediately adjacent to Fort Street. These piers were not part of the Ambassador Bridge Gateway Project Agreement between the Michigan Department of Transportation (MDOT) and the DIBC and based on the letter from a MDOT official to the DIBC, dated December 23, 2008, they are in conflict with the ABGP plans and were not approved by or even proposed to MDOT before their construction. Has the Coast Guard been in contact with MDOT since December 2008 in regards to these piers and their compliance with the contractual agreements pertaining to the ABGP?

Based on the location of the piers and the drawings for the proposed span for the ABEP it is a logical conclusion that the DIBC has commenced construction of their twin span before the proper federal permits have been issued.

These actions bring in to doubt not only the credibility of the DIBC but the execution of NEPA process in regards to the ABEP. In a January 29, 2009 Coast Guard letter from Captain Shultz to Congressman Dingell, it was indicated that the Coast Guard was going to conduct a site visit the last week of January 2009 and that if the DIBC has commenced "ABEP construction prior to the USCG completing NEPA . . . the USCG will initiate civil penalties against DIBC."

Did the USCG conduct this site visit with MDOT and Federal Highway Administration (FHWA) officials and did it determine that indeed the piers were part of the ABEP

project? If so, has the USCG initiated the process for levying civil penalties against the DIBC?

Gateway Plaza alterations

The commencement of the second span is only one recent development that brings into doubt this Draft FONSI. Over the past couple months the DIBC has redesigned their portion of the ABGP and commenced construction in a manner that is believed to be permanently redirecting truck traffic onto local streets, which would negate the Purpose and Need of the EA for the ABGP.

In December 9, 2008 the DIBC submitted altered plans for the Gateway Plaza to MDOT. A December 23, 2008 letter from an MDOT official to the DIBC indicated a series of concerns pertaining to the new plans, including the "expanded use of West Fort Street without DEPARTMENT approval. The DEPARTMENT cannot permit this DIBC proposal on the use of West Fort Street as it is a major change to the purpose and need of the Gateway Project."

However it has become apparent that the DIBC has begun construction that will permanently alter their portion of the Gateway Plaza. A February 9, 2009 letter from an MDOT official to the DIBC states that the DIBC has been "constructing the plaza under a new design that includes expanding the plaza into M-85 (a state trunkline), 23rd street and West Grand Boulevard (city streets) without coordination or approval by either MDOT or the city . . . these actions represent blatant disregard on the part of DIBC of the agreements for construction of the Gateway Project, and place in jeopardy nearly \$145 million of federal funds . . ."

It would appear the gas pump that is being constructed in the plaza is too close to Fort Street to allow space for the agreed upon truck ramp without having to annex one or two lanes of Fort Street, which would violate the ABGP's Purpose and Need to remove truck traffic from local streets. The ABEP Final EA states itself that "The Gateway Project will provide more direct access to the interstate system and remove all truck traffic from Fort Street." (ABEP Section 3.2.4)

While this Draft FONSI is limited in its scope, changes to the Gateway Project are significant due to the degree that the ABEP's Final EA is dependent upon the findings of the ABGP EA.

The Coast Guard evaluation of the ABEP indicates that the primary impacts to neighborhoods in the vicinity of the Ambassador/Gateway Corridor were implemented through the Gateway Project, which resulted in an EA/FONSI for NEPA. The only portion of the ABEP that is outside of the approved Gateway (and ABEP) study area extends eastward from the eastern limit of the plaza to the shoreline of Detroit River. Appendix A, pg 81

"The Coast Guard considers the application of the Gateway Project EA/FONSI as pertinent documentation for the ABEP, and as such the Gateway EA/FONSI is incorporated by reference into the overall evaluation of the project by the Coast Guard in accordance with 40 CFR 1506." (ABEP Draft FONSI, Section IV)

"The ABEP is not expected to require residential or commercial relocations, alter approved traffic projections, route traffic onto local roadways or neighborhoods, or cause any significant impacts in the ABEP project area or the areas analyzed and approved for the Gateway Project." (ABEP Draft FONSI, Section V,)

The location of the recently constructed gas pumps and the aforementioned piers that are outside the ABGP agreement bring in to doubt the following comments in the Final EA:

No local roadways will be permanently altered to implement the project.
(Appendix A, response to question on pg 277)

No changes to the plazas or the local roadways will be made as a result of the Proposed Project. (ABEP Section 3.2.4)

I would like to know what consultation the USCG has made with MDOT over the three months to confirm that these statements are still applicable.

Segmentation

All the prior arguments lead to the biggest flaw with the Draft FONSI and the application as a whole. This application represents a continued piecemeal approach to the NEPA process by the DIBC in terms of its short-term and long-term plans for the established Ambassador Bridge corridor.

According to the FHWA website on the NEPA process, the scale of the environmental assessments for project should reflect not just the short term construction but the long term impact on the corridor. In terms of deciding the local termini for the EA:

" . . . related improvements within a transportation facility should be evaluated as one project, rather than selecting termini based on what is programmed as short range improvements. Construction may then be "staged," or programmed for shorter sections or discrete construction elements as funding permits."¹

The FHWA website goes on to warn about the problem of "segmentation."

"Project sponsors should also be aware of the problem of "segmentation."
Segmentation may occur when a transportation need extends throughout an entire

¹ <http://www.environment.fhwa.dot.gov/projdev/tdmtermini.asp>

corridor, but project sponsors discuss the environmental issues and transportation need of only a segment of the corridor."

The scope of the Final EA is limited to the span and the immediate area between Fort Street and the Canadian border. As mentioned before, the environmental impact on the local streets and surrounding neighborhoods was covered by the ABGP EA. However the ABGP EA does not reflect the aforementioned changes made to the Gateway Plaza or the DIBC's other plans for existing Ambassador Bridge corridor.

The DIBC has openly stated that it intends to expand its exiting plaza in a manner that would cause the redirection of a state trunkline road. A May 17, 2006 DIBC letter asking MDOT for staff to review expansion of the plaza expressed their intentions for the corridor stated:

"I am writing to request your commitment to participate in the necessary process needed for the relocation of M-85, Fort Street in the area around the Ambassador Bridge. The relocation of this major corridor is needed for many reasons . . . along with allowing for the ability to expand and relocate the existing Ambassador Bridge operations in order to able to efficiently process international traffic."

Only an environmental study that reflects and examines the twin span as proposed in the ABEP, as well as the recent changes to the Gateway Plaza and its impact on local roads and the DIBC's plans to redirect Fort Street can truly meet the environmental needs of the this community and regions by assessing the entire impact on the existing corridor

Ultimately, this Final EA and Draft FONSI represent the culmination of what appears to the be a technically flawed environment process that is too limited in its scope and fails to recognize persistent questions over property rights as well as current changes on the ground that have the potential to permanently impact this vital corridor and the surrounding communities. I would ask that the USCG take notice of the crowd here tonight and reflect on its own NEPA Implementing Procedures and Policy for Considering Environmental Impacts, COMDTINST M16475.1 and determine a that a full Environmental Impact Statement is warranted due to the obvious "potential for controversy in terms of public opinion."

Only a full Environmental Impact Statement would allow proper community input as well as the necessary time and context to address the many unsolved questions surrounding the Ambassador Bridge Expansion Project.

I'm Dr. Bollig-Fischer

For 12 years I've studied human health and disease

5 years researching the causes and treatments for cancer

- Currently primary usage for the ambassador bridge includes high diesel truck volumes
- A higher capacity span foreshadows increased diesel truck traffic in the future, concentrated at this single location when the economy and U.S./Canadian trade rebounds
- An increase in truck traffic will cause an equal rise in diesel emissions
- Ample Epidemiological and mechanistic evidence show that diesel emissions cause asthma and lung cancer.
- Levels for diesel emissions in Detroit are already at high levels that cause disease **and so Any** increase in diesel emissions will cause a concordant rise in lung diseases here
- **For perspective** In High DE polluted areas residents on average were found to live 10 years fewer than people living elsewhere
- **Also** According to research done in 2008 at the Oakland California Port, where measures are being taken to **reduce** diesel emissions by 85% in a decade, we know that neighborhoods near and adjacent to the ambassador bridge experience the effects of diesel emissions: this includes downtown, the riverfront, corktown, greektown, Mexican town, southwest Detroit
- **Based** on the research outlined here, the issuance of no finding of significant impact is premature and called into question. A full environmental study should be undergone by experienced, unbiased researchers to determine the current and projected impact of the ambassador bridge spans.
- **Now**, People and investors living and working in these named border communities show commitments to recycling, green industries, parks, and historical preservation.
- DIBC's vision for Detroit is contrary to the livability and economic vision supported by the local neighborhoods and is antagonistic to the revitalization of downtown Detroit. As evidenced by DIBC's methods to build unmitigated concrete expanses a mile from downtown Detroit, and their methods that closed off large parts of the public riverside park from the public.
- The ambassador bridge has served Detroit well for nearly 100 years. **But** times have changed— in 2009 and for the next 100 years – it needs to be seriously considered that a location near downtown Detroit and in the middle of a vibrant community is the **wrong place** for a new higher capacity bridge, a trucking industry epicenter and the life-threatening pollution that it creates.



**UNITED STATES COAST GUARD
PUBLIC MEETING**

COMMENT FORM

AMBASSADOR BRIDGE ENHANCEMENT PROJECT

In the space below, please provide your comment regarding the Ambassador Bridge Enhancement Project. All comments received will be posted to <http://www.regulations.gov> under the Coast Guard docket number USCG-2009-0093.

Please submit to a Coast Guard official before the end of this public meeting.

The statement (and 2 attachments)
of Reginald L. McGhee is
clipped to this page.

**Public Hearing on the Issuance of a "Finding of No Significant Impact" Permit
March 17, 2009**

**Statement of Reginald L. McGhee
Detroit resident**

Good evening. My name is Reginald McGhee and I am resident of Detroit. Currently I reside in Lafayette Park, approximately two miles north of this hearing. For nearly 10 years I was a resident of this community, on West Grand Boulevard near W. Vernor.

I wanted to enter into the record this evening two documents. One is a 2005 article from the *Science Daily* magazine, entitled "Researchers Show How Air Pollution Can Cause Heart Disease." The essence of the article describes how in a well-designed study, scientists at the New York University School of Medicine provided compelling evidence that long term exposure of air pollution – even at levels within federal standards – causes heart disease.

The second document is a statement by the *American Heart Association* entitled, "Air Pollution and Cardiovascular Disease." This document is a detailed scientific review of existing literature by five doctors, of how the gases, liquids and particulate matter - comprising what we call air pollution - places human populations at consistent and increased risk for cardiovascular disease and events.

Obviously, the building of another span of the Ambassador Bridge to provide for increased truck traffic in this area will lead to an increase in air pollution. There can be no sensible, responsible and legal denial of this fact. Trucks burn diesel fuel. Diesel fuel contains gases, liquids and particulate matter. All of those by-products cause heart disease.

Whether in transit on the bridge, or while idling while parked, increased truck traffic will contribute to a marked increase in air pollution. This will have a serious impact on the health and quality of life of the residents of this area. To suggest otherwise suggests conscious negligence on the part of those who would own and seek to build the bridge, those who own and operate the trucking companies, and those public officials responsible for the regulation of the bridge operation and public health of the residents, of both countries affected.

To suggest that there would be "no significant impact" on the area residents by the construction of another span of the bridge, to facilitate increased truck traffic, is irresponsible and negligent behavior.

All further construction on a new bridge span should be halted until an Environmental Impact Study can be conducted. That study should include the impact of increased air pollution on the residents of nearby communities as part of its focus.

Thank you.

Web address:

<http://www.sciencedaily.com/releases/2005/12/051230085740.htm>

Researchers Show How Air Pollution Can Cause Heart Disease

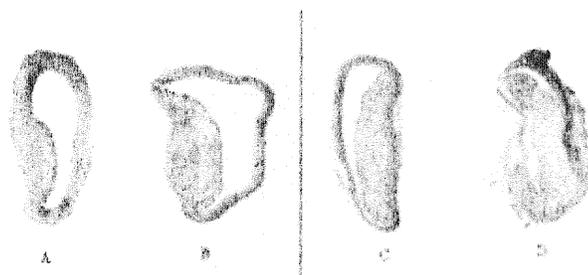
ScienceDaily (Dec. 30, 2005) — New York University School of Medicine researchers provide some of the most compelling evidence yet that long-term exposure to air pollution--even at levels within federal standards--causes heart disease. Previous studies have linked air pollution to cardiovascular disease but until now it was poorly understood how pollution damaged the body's blood vessels.

In a well-designed mouse study, where animals breathed air as polluted as the air in New York City, the researchers pinpointed specific mechanisms and showed that air pollution can be particularly damaging when coupled with a high-fat diet, according to new research published in the December 21 issue of JAMA.

"We established a causal link between air pollution and atherosclerosis," says Lung Chi Chen, Ph.D., Associate Professor of Environmental Medicine at NYU School of Medicine and a lead author of the study. Atherosclerosis--the hardening, narrowing, and clogging of the arteries--is an important component of cardiovascular disease.

The study, done in collaboration with the Mount Sinai School of Medicine and University of Michigan, looked at the effects of airborne particles measuring less than 2.5 microns, referred to as PM2.5, the size linked most strongly with cardiovascular disease. The emissions arise primarily from power plants and vehicle exhaust. The US Environmental Protection Agency (EPA) has regulated PM2.5 since 1997, limiting each person's average exposure per year to no more than 15 micrograms per cubic meter. These tiny particles of dust, soot, and smoke lead to an estimated 60,000 premature deaths every year in the United States.

Dr. Chen and his colleagues divided 28 mice, which were genetically prone to developing cardiovascular disease, into two groups eating either normal or high-fat diets. For the next six months, half of the mice in each feeding group breathed doses of either particle-free filtered air or concentrated air containing PM2.5 at levels that averaged out to 15.2 micrograms per cubic meter. This amount is within the range of annual EPA limits and equivalent to air quality in urban areas such as New York City.



Among mice fed normal diets, those exposed to polluted air had more plaque clogging their arteries (A) than those that breathed filtered air (B). But in mice that ate high fat diets, the impact of PM2.5 was even more pronounced: those who breathed air pollution (C) had nearly double the amount of plaque as those who inhaled filtered air (D).

C

The researchers then conducted an array of tests to measure whether the PM2.5 exposure had any impact on the mice's cardiovascular health. Overall, mice who breathed polluted air fared worse than those inhaling filtered air. But when coupled with a high-fat diet, the impact of PM2.5 exposure was even more dramatic. The results added up to a clear cause and effect relationship between PM2.5 exposure and atherosclerosis, according to the study.

On the whole, mice breathing polluted air had far more plaque than those breathing filtered air. In cross sections taken from the largest artery in the body--the aorta--mice on normal diets and exposed to PM2.5 had arteries 19.2 percent filled with plaque, the fatty deposits that clog arteries. The arteries of those breathing particle-free air were 13.2 percent obstructed. Among high-fat diet mice, those exposed to PM2.5 had arteries that were 41.5 percent obstructed by plaque, whereas the arteries of the pollution-free mice were 26.2 percent clogged. In both normal and high-fat diet mice, PM2.5 exposure increased cholesterol levels, which are thought to exacerbate plaque buildup.

Though findings for increased plaque among mice eating normal diets were not statistically significant, Dr. Chen believes that future research on larger numbers of animals will solidify the trend. "Even with the low-fat diet, there's still something there. So that is something to think about," he says. He suspects that PM2.5 exposure could also greatly affect even people who do not eat high-fat diets.

Mice exposed to PM2.5 also appeared prone to developing high blood pressure, another element of cardiovascular disease, because their arteries had become less elastic. To measure tension in the arteries, the researchers tested how the neurotransmitters serotonin and acetylcholine affected the aortic arches of PM2.5-exposed mice differently than those of controls. The arteries taken from exposed mice were less elastic than the control group, constricting more in the presence of serotonin and relaxing less in response to acetylcholine. Once again, the mice fed high-fat diets suffered the most pronounced effects from breathing polluted air.

Finally, the researchers also examined various measures of vascular inflammation, which is involved in atherosclerosis on a number of levels. In the aortas of PM2.5-exposed mice, for example, they found increased levels of macrophages, immune cells that are an important ingredient in plaque deposits and also active participants in a biochemical pathway related to inflammation. The study revealed several signs that this pathway was more active, strengthening the connection between airborne particles and cardiovascular disease.

###

The authors of the new study are: Morton Lippmann, Lung Chi Chen, and Ximei Jin of the NYU School of Medicine's Nelson Institute of Environmental Medicine, based in Tuxedo, New York; Qinghua Sun, Alex Natanzon, Juan-Gilberto S. Aguinaldo, Zahi A. Fayad, Valentin Fuster, and Sayjay Rajagopalan of the Mount Sinai School of Medicine, New York; and Robert D. Brook and Damon Duquaine of University of Michigan, Ann Arbor. The study was funded by the EPA and the National Institute of Environmental Health Sciences.

Adapted from materials provided by [New York University Medical Center and School of Medicine](#).

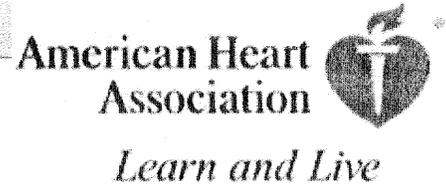
Email or share this story:  [BOOKMARK](#) 

Need to cite this story in your essay, paper, or report? Use one of the following formats:

APA

MLA

New York University Medical Center and School of Medicine (2005, December 30). Researchers Show How Air Pollution Can Cause Heart Disease. *ScienceDaily*. Retrieved February 25, 2009, from <http://www.sciencedaily.com/releases/2005/12/051230085740.htm>



Circulation

Search: [Advanced Search](#)

[Circulation Home](#) [Subscriptions](#) [Archives](#) [Feedback](#) [Authors](#) [Help](#) [AHA Journals Home](#)

Circulation. 2004;109:2655-2671 [« Previous Article | Table of Contents | Next Article »](#)
doi: 10.1161/01.CIR.0000128587.30041.C8



(Circulation. 2004;109:2655-2671.)
© 2004 American Heart Association, Inc.

AHA Scientific Statement

Air Pollution and Cardiovascular Disease

A Statement for Healthcare Professionals From the Expert Panel on Population and Prevention Science of the American Heart Association

Robert D. Brook, MD; Barry Franklin, PhD, Chair;
Wayne Cascio, MD; Yuling Hong, MD, PhD; George Howard, PhD;
Michael Lipsett, MD; Russell Luepker, MD;
Murray Mittleman, MD, ScD; Jonathan Samet, MD;
Sidney C. Smith, Jr, MD; Ira Tager, MD

This Article

Free upon publication

- ▶ [Abstract](#) **FREE**
- ▶ [Full Text \(PDF\)](#)
- ▶ [Alert me when this article is cited](#)
- ▶ [Alert me if a correction is posted](#)
- ▶ [Citation Map](#)

Services

- ▶ [Email this article to a friend](#)
- ▶ [Similar articles in this journal](#)
- ▶ [Similar articles in PubMed](#)
- ▶ [Alert me to new issues of the journal](#)
- ▶ [Download to citation manager](#)
- ▶ [Request Permissions](#)

Citing Articles

- ▶ [Citing Articles via HighWire](#)
- ▶ [Citing Articles via Google Scholar](#)

Google Scholar

- ▶ [Articles by Brook, R. D.](#)
- ▶ [Articles by Tager, I.](#)
- ▶ [Search for Related Content](#)

PubMed

- ▶ [PubMed Citation](#)
- ▶ [Articles by Brook, R. D.](#)
- ▶ [Articles by Tager, I.](#)
- ▶ [Pubmed/NCBI databases](#)
 - [Compound via MeSH](#)
 - [Substance via MeSH](#)
- ▶ [Medline Plus Health Information](#)

- [Air Pollution](#)

Hazardous Substances DB

- [CARBON MONOXIDE](#)

- [OZONE](#)

Related Collections

- ▶ [Other Ethics and Policy](#)

- ▶ [Risk Factors](#)

- ▶ [Epidemiology](#)

▶ **Abstract**

Air pollution is a heterogeneous, complex mixture of gases, liquids, and particulate matter. Epidemiological studies have demonstrated a consistent increased risk for cardiovascular events in relation to both short- and long-term exposure to present-day concentrations of ambient particulate matter. Several plausible mechanistic pathways have been described, including enhanced coagulation/thrombosis, a propensity for arrhythmias, acute arterial vasoconstriction, systemic inflammatory responses, and the chronic promotion of atherosclerosis. The purpose of this statement is to provide healthcare professionals and regulatory agencies with a comprehensive review of the literature on air pollution and cardiovascular disease. In addition, the implications of these findings in relation to public health and regulatory policies are addressed. Practical recommendations for healthcare providers and their patients are outlined. In the final section, suggestions for future research are made to address a number of remaining scientific questions.

- ▲ [Top](#)
- [Abstract](#)
- ▼ [Introduction](#)
- ▼ [Ambient Air Pollutants](#)
- ▼ [Epidemiology of Ambient Air...](#)
- ▼ [Potential Biological Mechanisms](#)
- ▼ [Summary](#)
- ▼ [Potential for Prevention/Public...](#)
- ▼ [Conclusions and Recommendations](#)
- ▼ [References](#)

Key Words: AHA Scientific Statements • air pollution • cardiovascular diseases • respiration

▶ **Introduction**

Recently, the American Heart Association (AHA) published "Guidelines for Primary Prevention of Cardiovascular Disease and Stroke" as an aid to healthcare professionals and their patients without established coronary artery disease or other atherosclerotic diseases.¹ The statement was intended to complement the AHA/American College of Cardiology (ACC) "Guidelines for Preventing Heart Attack and Death in Patients with Atherosclerotic Cardiovascular Disease."² Both sets of recommendations emphasized multifactorial interventions, especially more intensive measures/goals to modify individual cardiovascular risk factors with diet, drugs, exercise, weight management, complete smoking cessation, and avoidance of secondhand smoke (SHS), or combinations thereof.

- ▲ [Top](#)
- ▲ [Abstract](#)
- [Introduction](#)
- ▼ [Ambient Air Pollutants](#)
- ▼ [Epidemiology of Ambient Air...](#)
- ▼ [Potential Biological Mechanisms](#)
- ▼ [Summary](#)
- ▼ [Potential for Prevention/Public...](#)
- ▼ [Conclusions and Recommendations](#)
- ▼ [References](#)

Over the last decade, however, a growing body of epidemiological and clinical evidence has led to a heightened concern about the potential deleterious effects of ambient air pollution on health and its relation to heart disease and stroke. Of special interest are several environmental air pollutants that include carbon monoxide, oxides of nitrogen, sulfur dioxide, ozone, lead, and particulate matter ("thoracic particles" [PM_{10}] $<10 \mu m$ in aerodynamic diameter, "fine particles" [$PM_{2.5}$] $<2.5 \mu m$, and "coarse particles" [PM_{10} to 2.5]). These pollutants are associated with increased hospitalization³ and mortality due to cardiovascular disease,⁴⁻⁶ especially in persons with congestive heart failure, frequent arrhythmias, or both.⁷ The well-established causal associations between active and passive smoking with heart disease and stroke support the plausibility of an adverse effect of PM on the cardiovascular system.

The most recent analysis of the National Mortality and Morbidity Air Pollution Study (NMMAPS), based on data from 90 of the largest cities in the United States, estimated that daily total and cardiopulmonary mortality increased in the short term by 0.21% (± 0.06 standard error [SE]) and 0.31% (± 0.09 SE), respectively, for each $10\text{-}\mu g/m^3$ increase in PM_{10} (measured over a 24-hour period).⁸ To give some context to a 24-hour PM_{10} increment of $10 \mu g/m^3$, the US Environmental Protection Agency (EPA) reported a range of maximum city-specific 24-hour PM_{10} concentrations from 26 to $534 \mu g/m^3$.⁹ Data from the American Cancer Society (ACS) cohort⁵ estimated that for each $10\text{-}\mu g/m^3$ increase in annual average exposure to $PM_{2.5}$, long-term all-cause, cardiopulmonary, and lung cancer mortality were increased by approximately 4%, 6%, and 8%, respectively. On the basis of a job exposure matrix, Gustavsson et al.¹⁰ reported increasing risks of myocardial infarction among ≈ 3000 Swedish workers with increasing cumulative exposure to products from nonvehicular combustion processes.

To evaluate whether high concentrations of ambient particles can trigger the onset of acute myocardial infarction (AMI), Peters and associates,¹¹ using a case-crossover approach, interviewed 772 patients with AMI as part of the Determinants of Myocardial Infarction Onset Study. Elevated concentrations of $PM_{2.5}$ were associated with a transient risk of AMI onset during 2 separate time periods (within 2 hours and 1 day after exposure). On the other hand, investigators in Seattle, Wash, did not find an association between high levels of PM_{10} and the occurrence of primary cardiac arrest that occurred outside of the hospital in presumably healthy adults¹² or in subjects with known underlying heart disease.¹³ A recent report by Suwa et al.¹⁴ provides experimental evidence to support the hypothesis that these epidemiological data truly reflect the deleterious effects of particulate pollution on the cardiovascular system. Compared with their control counterparts, hyperlipidemic rabbits exposed to PM_{10} showed more advanced coronary lesions, increased plaque size, more extensive atherosclerosis in the aorta, and an increase in the volume fraction of lesions composed of lipids (ie, plaques more likely to rupture).¹⁵ Other contemporary studies suggest that possible links between acute and/or chronic exposure to PM and cardiovascular events may be related to increases in heart rate and blood pressure, fibrinogen, and blood coagulation factors; arterial vasoconstriction; inflammatory mediators (eg, C-reactive protein [CRP]); endothelial injury/dysfunction; and decreases in

heart rate variability (HRV).¹⁶ Consequences of these effects may include myocardial ischemia (manifested as significant ST-segment depression during exercise testing,¹⁷ angina pectoris, or both), malignant ventricular arrhythmias,¹⁸ increased plaque vulnerability, and enhanced potential for acute thrombosis triggering acute coronary syndromes. Further support that these changes can be attributed to air pollution comes from studies of the effects of SHS, which is the single largest contributor to indoor PM¹⁹ when a smoker is present. Exposure to SHS increases platelet activation,²⁰ causes rapid deterioration in endothelial function,^{21,22} promotes atherosclerotic plaque development,²³ and abets infarct expansion in experimental animals.²⁴ Because exposure to the SHS of just 1 cigarette per day accelerates the progression of atherosclerosis,²⁵ it is plausible that even low doses of air pollution could have negative effects on coronary morphology and circulation.

Collectively, these and other studies (described herein) suggest that air pollution may accelerate the development of coronary atherosclerosis and worsen its sequelae. Some of these effects may occur over time, as with acceleration of the progression of atherosclerosis, or rather abruptly, as with the triggering of an arrhythmia or myocardial infarction by acute inflammatory responses, altered platelet adhesiveness, or perhaps vascular endothelial dysfunction. This AHA scientific statement provides healthcare professionals and regulatory agencies with a comprehensive review of the relationship between air pollution and cardiovascular disease. A brief description of the different types of air pollutants is provided first for background. In the remaining sections, the focus of this statement is on PM, with occasional references to the health effects of other pollutants, alone or in combination. The link between SHS and heart disease is outlined next, which provides a relevant model for the cardiovascular effects of air pollution. In the following sections, many of the pertinent epidemiological studies and the potential pathophysiological mechanisms underlying the increased risk of cardiovascular events due to PM are discussed. In the summary and conclusion sections, the implications of these data regarding public health policy and unanswered (future) research questions are addressed.

▶ Ambient Air Pollutants

A brief description of several individual air pollutants is provided first for background. A complete discussion is beyond the scope of this statement, and interested readers may find a more comprehensive review on this subject elsewhere.²⁶

Particulate Matter

Airborne PM consists of a heterogeneous mixture of solid and liquid particles suspended in air, continually varying in size and chemical composition in space and time (Figure 1). Primary particles are emitted directly into the atmosphere, such as diesel soot, whereas secondary particles are created through physicochemical transformation of gases, such as nitrate and sulfate formation from gaseous nitric acid and sulfur dioxide (SO₂), respectively. The

▲ Top
▲ Abstract
▲ Introduction
• Ambient Air Pollutants
▼ Epidemiology of Ambient Air...
▼ Potential Biological Mechanisms
▼ Summary
▼ Potential for Prevention/Public...
▼ Conclusions and Recommendations
▼ References

numerous natural and anthropogenic sources of PM include motor vehicle emissions, tire fragmentation and resuspension of road dust, power generation and other industrial combustion, smelting and other metal processing, agriculture, construction and demolition activities, residential wood burning, windblown soil, pollens and molds, forest fires and combustion of agricultural debris, volcanic emissions, and sea spray. Although there are thousands of chemicals that have been detected in PM in different locations, some of the more common constituents include nitrates, sulfates, elemental and organic carbon, organic compounds (eg, polycyclic aromatic hydrocarbons), biological compounds (eg, endotoxin, cell fragments), and a variety of metals (eg, iron, copper, nickel, zinc, and vanadium).

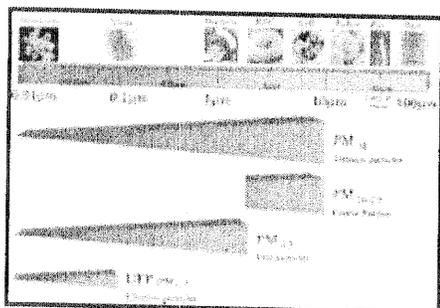


Figure 1. Particulate matter air pollution size distribution

View larger version (38K):
[\[in this window\]](#)
[\[in a new window\]](#)

Largely because of the complex nature of PM, it has been measured and regulated based primarily on mass within defined size ranges. In 1987, the regulatory focus shifted from total suspended particles to particles that could readily penetrate and deposit in the tracheobronchial tree, or PM_{10} (PM with a median aerodynamic diameter of $<10 \mu m$). In 1997, the US EPA promulgated 24-hour and annual average standards for $PM_{2.5}$ (PM with median aerodynamic diameter $<2.5 \mu m$), comprising the size fraction that can reach the small airways and alveoli. The existing federal PM_{10} standards were retained, however, to address health effects that could be related to the "coarse fraction" ($PM_{10 \text{ to } 2.5}$). Currently, a separate $PM_{10 \text{ to } 2.5}$ standard is under consideration. In general, $PM_{2.5}$ originates mostly from combustion sources and includes primary and secondary particles, whereas the coarse fraction derives predominantly from natural sources, especially crustal material (including windblown soil) and grinding processes. Important bioaerosols (eg, endotoxin, pollen grains, and fungal spores) are found mostly in the coarse fraction (and larger particles), although both endotoxin (an essential component of the cell wall of Gram-negative bacteria) and the antigenic protein content of pollen grains can also adsorb onto the surface of fine PM. Generally, larger particles demonstrate a greater fractional deposition in the extrathoracic and upper tracheobronchial regions, whereas smaller particles (eg, $PM_{2.5}$) show greater deposition in the deep lung. Although $PM_{2.5}$ generally behaves as a regional pollutant, there can be considerable small-scale spatial variability due to point source

emissions (eg, a smelter) or features such as street canyons in large cities. In addition, prevailing wind patterns can affect human exposures.

More recently, considerable research attention has been devoted to ultrafine particles (UFPs) <100 nm (0.1 μm) in diameter, which result from combustion processes. UFPs tend to be short-lived, because they agglomerate and coalesce into larger particles. However, they demonstrate very high deposition in human alveoli,²⁷ account for a major portion of the actual numbers of particles within PM, and have a high surface area-to-mass ratio, potentially leading to enhanced biological toxicity. UFPs may even be able to pass directly into the circulatory system, which could allow them to be disseminated systemically.²⁸⁻³⁰

Nitrogen Oxides

Nitrogen oxides are reactive substances commonly understood to encompass nitric oxide (NO), nitrogen dioxide (NO₂), nitrogen trioxide, nitrogen tetroxide (N₂O₄), and di-nitrogen pentoxide (N₂O₅). These compounds are referred to collectively as "NO_x." Gaseous nitric acid (HNO₃), a major source of particulate nitrate, is formed when NO₂ reacts with hydroxyl radicals during the day and when N₂O₅ reacts with water vapor at night.³¹ Other members of the larger family of nitrogen oxides include nitrous acid, nitrous oxide, peroxyacetyl nitrate (responsible for some of the irritant effects of photochemical smog), nitrites, nitroso compounds, and other nitrogen-containing acids. Most toxicological and epidemiological research has focused on NO₂, because of the fact that (1) NO₂ is one of the regulated air pollutants for which standards are available worldwide; (2) NO from vehicular exhaust and power plants is largely converted to NO₂; and (3) NO₂ plays a primary role in the formation of tropospheric ozone (O₃).

The main anthropogenic source of NO_x in ambient air is fossil fuel combustion in motor vehicles and industrial processes, particularly in power generation. High-temperature combustion results in the oxidation of atmospheric N₂, first to NO and then to NO₂. Motor vehicle emissions near busy streets can result in high local NO_x concentrations. The typical diurnal NO_x pattern consists of a low background concentration, with morning and late afternoon peaks resulting from rush-hour traffic. Nitrogen in fossil fuels such as coal can be oxidized to NO₂ under oxygen-rich combustion conditions. NO₂ and NO are both formed naturally as a result of bacterial metabolism of nitrogenous compounds and, to a lesser extent, from fires, volcanoes, and fixation by lightning. The generation of tropospheric ozone and other photochemical oxidants is initiated with photolysis of NO₂, whereas NO acts as an ozone scavenger.³²

Significant human exposure can occur in nonoccupational indoor settings.^{33,34} Gas-burning appliances, such as unvented furnaces and stoves, are the principal sources of indoor NO_x, although kerosene space heaters and tobacco smoke may also play a role.^{35,36} In urban areas, infiltration of ambient NO₂ from vehicular emissions may also influence indoor exposures.³³ Several reports have also documented toxic concentrations of NO₂ in ice-skating rinks.^{37,38}

Carbon Monoxide

Carbon monoxide (CO) is a nearly ubiquitous product of incomplete combustion of carbon-containing fuels. Outdoor sources include motor vehicles; engines on motorboats, lawnmowers, chain saws, and other devices that require fossil fuel combustion; residential wood burning; improperly adjusted gas-burning and oil appliances; coal combustion; and tobacco smoking.^{39,40} In urban areas, the contributions of diesel and stationary source combustion are relatively small in relation to gasoline-powered engines.⁴¹

CO is an odorless, colorless, and tasteless gas that binds to hemoglobin with an affinity 250 times that of oxygen, thereby interfering with the systemic delivery of oxygen to tissues. In addition, binding of CO to hemoglobin causes an allosteric change in the conformation of the oxyhemoglobin complex that increases the oxygen affinity of the remaining binding sites and interferes with the release of O₂ at the tissue level. In addition, CO binds to cytochrome oxidase, exacerbates cellular hypoxia, and binds to other extravascular proteins that include myoglobin, cytochrome P-450, catalase, and peroxidases.⁴² Given current ambient CO concentrations in the United States, it is likely that in most circumstances, this pollutant serves more as an indicator of combustion-related pollution than as a direct toxicant. However, in some situations (eg, insufficiently ventilated parking structures), CO could attain concentrations sufficient to lead to physiologically meaningful increases in carboxyhemoglobin in persons with significant atherosclerotic disease or other cardiac conditions.

Sulfur Dioxide

Sulfur dioxide (SO₂) is a highly irritating, colorless, soluble gas with a pungent odor and taste. In contact with water, it forms sulfurous acid, which accounts for its strong irritant effects on eyes, mucous membranes, and skin.⁴³ SO₂ is efficiently scrubbed from inhaled air in the upper airway.

In the absence of human activities, background concentrations of ambient SO₂ are very low, in the range of 1 ppb. In ambient air, the principal sources of SO₂ include combustion of sulfur-containing fuels, especially in power plants and diesel engines (prior to the reformulation of diesel fuels), and roasting of metal sulfide ores. Sulfur dioxide is oxidized to sulfur trioxide, which, because of its strong affinity for water, can be rapidly hydrated to form sulfuric acid.⁴⁴ Elevated levels of SO₂ have been associated with widespread illness in several 20th century air pollution catastrophes; however, much of the morbidity and mortality in these episodes may have been due to its role in the formation of particulate sulfates. In nonoccupational settings, SO₂ is generally found at substantially lower concentrations indoors than outside; however, the use of kerosene space heaters can generate significant indoor concentrations.³⁵

Ozone

Ozone (O₃) is a highly reactive, colorless-to-bluish gas with a characteristic odor associated with electrical discharges.⁴³ Low-level exposure is ubiquitous, because O₃ is formed by natural processes and by human activities. Ozone is formed in the stratosphere by the action of solar radiation on molecular oxygen (O₂). Because stratospheric O₃ prevents high-energy UV radiation from penetrating the atmosphere, many

terrestrial life forms would be unable to survive without this O₃ "shield."

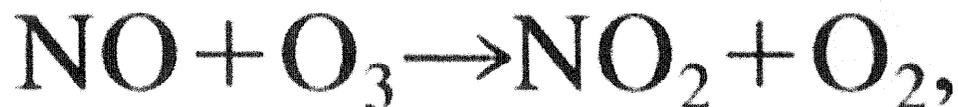
O₃ has been recognized since the 1950s as the principal component of photochemical smog. In the troposphere, it is formed by the action of solar UV radiation on nitrogen oxides and reactive hydrocarbons, both of which are emitted by motor vehicles and many industrial sources. The reaction sequence involves photolysis of NO₂ into NO and oxygen atoms. The latter react with molecular oxygen to form O₃. NO, however, rapidly scavenges O₃, regenerating NO₂ and O₂, as summarized below: ☐



☐



☐



where M represents any nearby molecule that can absorb the energy of the reaction. Under steady-state conditions, there is little accumulation of O₃. However, many reactive organic compounds can facilitate the oxidation of NO to NO₂ by alternative mechanisms. These reactions reduce NO scavenging, which allows O₃ concentrations to increase.⁴⁵

Photochemical O₃ formation tends to be greatest on warm, sunny days. The typical profile of tropospheric O₃ formation in populated areas is characterized by a broad peak that lasts from the late morning until the late afternoon or early evening. However, large-scale transport may result in elevated O₃ concentrations that extend over thousands of square miles to include rural areas far removed from the precursor sources. Thermal inversion height, wind speed and direction, addition of other O₃ precursors along an air mass trajectory, and other factors affect the temporal O₃ patterns downwind, so that peak concentrations may occur anytime from noon until late in the evening.⁴⁶ Human activities are major sources of O₃ precursors, although the latter are also generated by nonanthropogenic processes that include the intrusion of stratospheric O₃, the action of lightning on molecular oxygen, and chemical reactions involving naturally occurring nitrogen oxides and organic compounds, such as biogenic methane and other volatile organic

compounds.⁴⁵

Secondhand Smoke

The national awareness of SHS as a major risk factor for chronic disease was substantially heightened with the 1986 Report of the Surgeon General, "The Health Consequences of Involuntary Smoking,"⁴⁷ and the 1992 EPA report on the respiratory health effects of SHS.⁴⁸ The primary focus of the Surgeon General's report was on lung cancer and other respiratory diseases,⁴⁷ although evidence linking passive smoking to heart disease was just beginning to emerge.⁴⁹ Similarly, the EPA report focused primarily on the impact of SHS on lung cancer and asthma⁵⁰ by reporting that an estimated annual 3000 lung cancer deaths could be attributed to SHS.⁴⁸

There is also clear evidence of an association of SHS and cardiovascular diseases. Several reports, including 2 recent separate meta-analyses of 17 and 18 individual studies, assessed the association of SHS with heart disease.^{20,51-53} Both estimated that nonsmoking spouses of smoking partners experience an approximately 25% (95% confidence interval [CI] 17% to 32%) increased risk of heart disease.^{52,53} A review of 6 studies examining the association between SHS in the workplace and cardiovascular disease noted that although none of the studies individually reached the level of a significant association ($P < 0.05$), there was a positive association in 5 of the 6 studies and a significant exposure-response relation between the intensity of SHS (measured by the number of cigarettes smoked by coworkers) and coronary risk in 2 of 3 studies that examined the trend.⁵⁴

It is likely that SHS acts to increase cardiac risk through both chronic (atherogenic) and acute pathways. A review of the association of SHS and subclinical measures of atherosclerosis reported a consistent association of significantly increased intima-medial thickness of the carotid artery in 8 reports.⁵⁵ In addition, exposure to SHS has been shown experimentally to have effects on endothelium-dependent arterial dilatation^{21,22} and coronary flow reserve in humans.⁵⁶

Initial reports on an association between SHS and stroke support the observed association with heart disease. Two case-control studies have shown significantly increased odds ratios of clinical stroke with exposure to SHS: 1.72 (95% CI 1.07 to 2.77) and 2.59 (95% CI 1.51 to 4.47) for exposure to a spouse smoking 1 to 20 cigarettes a day and ≥ 21 cigarettes per day,⁵⁷ respectively, and 1.82 (95% CI 1.34 to 2.49) for SHS exposure at home or work for at least 1 year during the decade preceding the event.⁵⁸ There was a 6% (95% CI 0.64 to 1.75) increased odds of silent cerebral infarction in a population-based cohort study of nonsmokers exposed to SHS in relation to those not exposed; however, this difference did not reach statistical significance.⁵⁹

The impact of SHS on heart disease and stroke is also supported by the remarkable "natural experiment" observed when Helena, Mont, banned public smoking beginning June 5, 2002. During the 6-month period of the ban, admissions to the local hospital for acute myocardial infarction dropped by 40%, a decline that was not observed in any of the other hospitals from surrounding communities.⁶⁰ The study investigators

hypothesized that the reduction in hospital admissions was due to acute coagulation changes associated with reduced SHS exposure.

A recent review of the studies concluded that it is unlikely that experimental biases can invalidate the general conclusions of the epidemiological studies.⁶¹ Moreover, the review cites the strong experimental evidence that provides a biological basis for the observed associations between SHS exposure and increased risk of cardiovascular disease.⁶¹

The importance of SHS as a public health issue comes from the high prevalence of SHS exposure in the general population. On the basis of the most recent data from the National Health Interview Survey, the prevalence of active cigarette smoking was 21.4% (95% CI 20.2% to 22.5%) in 2003.⁶² NHANES III (1988–1991) found that ≈90% of tobacco nonusers have detectable levels of serum cotinine,⁶³ which suggests that as much as 65% of the entire population are nonsmokers exposed to SHS. This arises primarily from exposures at home or at the workplace. Between 1988 and 1991, among adult nonsmokers, 37% reported living in a home with at least 1 smoker or being exposed to SHS at work.⁶³ Forty-three percent of children lived in a home with at least 1 smoker, higher than the percentage observed among adults, which is due in part to children's opportunity to be exposed to SHS by either a smoking mother or father.⁶³ More recent reports available at the CDC website suggest that there may be substantial decreased exposure to SHS over the interval from 1991–1994 to 1999–2000, with decreased levels of cotinine in children (–58%), adolescents (–55%), and adults (–75%).^{63a}

Exposure to SHS in workplace settings is also surprisingly high. Nineteen states participating in the Centers for Disease Control and Prevention's Behavioral Risk Factor Surveillance System included questions related to working in a smoke-free workplace during 1999. The survey reports a prevalence range of exposure to cigarette smoke at work from 18% (District of Columbia) to 38% (Mississippi).⁶⁴ Some other studies reported even higher rates.^{65,66}

SHS exposure is associated with well-established increases in relative risks for circulatory diseases. It directly affects, at some level, perhaps as much as two thirds of the population, with some suggestion that the proportion exposed may be decreasing. Although the increased risk for cardiovascular diseases associated with SHS exposure (relative risk [RR] 1.25) has not drawn attention to it as a "major" cardiovascular risk factor, the high prevalence of exposure does make it a major public health issue. Given past patterns of exposure, it has been estimated that between 35 000 and 65 000 ischemic heart disease deaths result from SHS annually.^{67,68} This is an order of magnitude greater than the estimated 3000 annual deaths from lung cancer. As such, SHS "pollution" represents a substantial public health burden, ranking it as one of the most important preventable causes of cardiovascular death.

► Epidemiology of Ambient Air Pollution and Cardiovascular Disease

An association between high levels of anthropogenic air pollutants and human illnesses has been known for more than half a century. A few episodes of markedly increased mortality rates during extreme elevations in urban pollution, such as in the Meuse Valley, Belgium, in December 1930⁶⁹ and during the London fog incident of 1952,⁷⁰ sparked the initial epidemiological research. As a result, a several-decades-long effort to reduce air pollution ensued and culminated in the Clean Air Act legislation of 1970. Despite improvements in air quality over the past few decades, associations between current ambient pollution levels and excess morbidity and mortality have been consistently detected.⁷¹⁻⁷⁶

▲ Top
▲ Abstract
▲ Introduction
▲ Ambient Air Pollutants
▪ Epidemiology of Ambient Air...
▼ Potential Biological Mechanisms
▼ Summary
▼ Potential for Prevention/Public...
▼ Conclusions and Recommendations
▼ References

There are several hundred published epidemiological studies linking air pollution with human illnesses. A number of extensive reviews on this topic are available.⁷⁷⁻⁷⁹ Although many pollutants may cause disease individually or in combination (eg, O₃, SO₂, and NO₂),⁸⁰ over the past decade, PM has become a major focus of research. During the past 15 years, the magnitude of evidence and number of studies linking air pollution to cardiovascular diseases has grown substantially.^{77,78}

In broad terms, studies can be separated into those that have investigated the health effects of acute or chronic air pollution exposure. Observations related to the adverse health effects of short-term exposure are more numerous. In these studies, population-wide changes in acute outcomes (mortality, symptomatology, hospitalizations, and healthcare visits) are linked to short-term variations in ambient pollutant concentrations, most frequently through the use of population-based time-series analysis. More recently, case-crossover designs have been added to the analytical repertoire. Publications on the health effects of long-term exposure are few. These studies have involved analysis of data (eg, total mortality and in some circumstances cardiovascular events) from a few large cohorts from multiple geographic locations that differ in the average chronic ambient concentrations and mixtures of air pollutants.

Long-Term Health Effects Studies

The first large, prospective cohort study that demonstrated an adverse health impact of long-term air pollution exposure was the Harvard Six Cities study by Dockery et al.⁸¹ This study demonstrated that chronic exposure to air pollutants is independently related to cardiovascular mortality. In a cohort of 8111 adults with 14 to 16 years of follow-up, the adjusted overall mortality rate ratio for the most-polluted city versus the least-polluted city was 1.26 (95% CI 1.08 to 1.47). Further adjustment for a variety of individual-level risk factors that included tobacco smoking, gender, body mass index, educational attainment, occupational exposures, hypertension, and diabetes did not significantly alter the relationship. Cardiovascular deaths accounted for the largest single category of the increased mortality. Of the 1401 validated deaths, 646 were due to cardiovascular causes (International Classification of Diseases, 9th Revision [ICD-9] codes 400 to 440). The risk ratios for lung cancer and overall cardiopulmonary mortality

were increased by similar magnitudes. Among air pollutants, elevations of PM_{2.5} and sulfates showed the strongest associations with disease.

These findings were complemented by similar observations from the first analysis of air pollution in relation to mortality in the ACS Cancer Prevention II study population.⁸² Recently, a follow-up of the original ACS cohort by Pope et al,⁵ based on additional subject mortality and ambient pollutant data, has provided the largest study of the long-term health effects of air pollution. In approximately 500 000 adults who resided in all 50 states, chronic exposure to multiple air pollutants was linked to mortality statistics for a 16-year period. The ACS follow-up study increased the degree of control for confounding variables, such as diet and gaseous copollutants. The primary results showed that each 10- $\mu\text{g}/\text{m}^3$ increase in annual PM_{2.5} mean concentration, based on a number of different averaging periods, was associated with increases in all-cause, cardiopulmonary, and lung cancer mortality of 4%, 6%, and 8%, respectively. The relationship between PM_{2.5} and adverse health effects was linear and without a discernible lower "safe" threshold. Mortality was most strongly associated with PM_{2.5}, sulfate particles, and SO₂. There also appeared to be an association between cardiopulmonary mortality and summertime O₃, when based on mean summer O₃ levels from 1982 to 1998. Educational level was a modifier of the risks estimated for PM-associated mortality. The increased risks were limited to those with no more than a high school education. This suggests that 1 or more unaccounted-for factors, such as intraurban geographic location or socioeconomic status, may be important determinants of health risk.

Hoek et al⁸³ confirmed the importance of within-city residential variations as a risk factor for mortality due to air pollution. In a cohort of 5000 adults followed up for 8 years, exposure to traffic-related air pollutants was more highly related to mortality than were citywide background levels. Of the various pollutant metrics, an indicator variable for living near a major road was most strongly associated with cardiopulmonary mortality in this cohort (RR 1.95, 95% CI 1.09 to 3.52). This study suggests that an individual's exposure to the "toxic" components of air pollution may vary as much within a single city as across different cities. Furthermore, it demonstrates that emissions from motor vehicles, a common source of urban air pollution, may be associated with an increased risk of mortality.

Until recently, the specific causes of the increased cardiovascular mortality due to long-term air pollution exposure have remained unclear. In an analysis of the ACS study published this year, the investigators reported PM-mortality associations with the specific cause of death.⁴ A statistically robust association between PM_{2.5} and overall cardiovascular mortality was confirmed for a 10- $\mu\text{g}/\text{m}^3$ increase in long-term exposure (RR 1.12, 95% CI 1.08 to 1.15). The single largest increase in risk was for ischemic heart disease (RR 1.18, 95% CI 1.14 to 1.23), which also accounted for the largest proportion of deaths. In addition, the risk for arrhythmia, heart failure, or cardiac arrest mortality was also increased (RR 1.13, 95% CI 1.05 to 1.21). There was no evidence for excess mortality in the entire cohort due to other reasons (eg, aortic aneurysms, stroke, diabetes, hypertensive disease, or any respiratory illness). These findings suggest that air pollution promotes both ischemic and nonischemic cardiovascular events.

Short-Term Health Effects Studies

The Six Cities⁸¹ and ACS^{4,5,82} studies provide strong evidence for the occurrence of adverse cardiovascular effects due to long-term air pollution exposure. However, many more studies have focused on short-term relationships between pollution exposure and adverse outcomes. The acute effects of air pollution are generally investigated by time-series analyses of changes in health outcomes (eg, mortality) in relation to day-to-day variations in ambient air pollution concentrations. The 2 largest studies to date are the NMMAPS in the United States^{6,8} and the Air Pollution and Health: a European Approach (APHEA-2) project.⁸⁴ These studies produced remarkably consistent results.

The NMMAPS observed outcomes in 50 million people in the 20 largest cities in the United States. Average mortality rates were independently associated with particle concentrations the day before death. Each $10\text{-}\mu\text{g}/\text{m}^3$ elevation in PM_{10} was associated with an increase of 0.21% (± 0.06 SE) and 0.31% (± 0.09 SE) for daily all-cause and cardiopulmonary mortality, respectively.⁸

The APHEA-2 study demonstrated slightly more robust associations between adverse health outcomes and air pollution.⁸⁴ For 43 million people in 29 European cities, the estimated increase in daily mortality was 0.6% (95% CI 0.4% to 0.8%) for each $10\text{-}\mu\text{g}/\text{m}^3$ increase in PM_{10} . Cardiovascular deaths were increased by 0.69% (95% CI 0.31% to 1.08%).⁸⁵ APHEA-2 based calculations on average particle concentrations the day of and 1 day before observed health outcomes (a 2-day exposure time window). The NMMAPS investigators reported no differences among various lag time periods from 0 to 2 days and therefore based their estimates solely on the prior 24-hour period (1-day lag). The size of the observed health effect in a study is known to vary slightly depending on exposure metric and lag periods used in analyses. This may have contributed to the stronger associations found in the European study. Additional analyses of the APHEA-2 mortality data, based on lag periods up to 40 days, found that the risk of adverse health effects associated with air pollution more than doubled (eg, 1.97% increase in cardiovascular mortality [95% CI 1.38% to 2.55%] per $10\text{-}\mu\text{g}/\text{m}^3$ elevation in PM_{10}).⁸⁵ This finding indicated that the increase in cardiopulmonary mortality was not simply the result of "harvesting" (also called mortality displacement, which refers to the advancement of death by no more than a few days for severely ill individuals). Analyses of data from other locations also have indicated that the increased risks cannot be explained solely by harvesting and that longer lags are associated with higher relative risks of cardiopulmonary mortality.⁸⁶⁻⁸⁸ The higher relative risks demonstrated by using this statistical modeling may reflect the accumulation of both acute and subacute health effects over the longer lag periods.

APHEA-2 found that cities with higher levels of the copollutant NO_2 exhibited larger associations between changes in PM concentrations and mortality. In the United States, this modifying effect of NO_2 was not demonstrated. The APHEA-2 investigators speculated that this might reflect a higher proportion of NO_2 that is derived from diesel exhaust in Europe. Outcome differences by geographical region were also noted in both studies. In Europe, cities with warmer climates demonstrated stronger mortality associations with air

pollutants. The NMMAPS study in the United States reported stronger relationships in the Northeast than the Southeast. It is plausible that differences in the underlying susceptibility of the populations, time spent outdoors, commute times, and ambient meteorology, as well as differences in the overall ambient pollutant mixtures, underlie the observed regional variability in risk estimates for both studies.

Hundreds of smaller, short-term studies have been published over the last few decades on the effects of acute air pollution exposure, as reviewed by Brunekreef and Holgate⁷⁷ and Pope.⁷⁸ Typically, short-term mortality rates^{73,88} hospital admissions,⁸⁹⁻⁹¹ emergency room visits,^{92,93} and symptom exacerbations^{94,95} have been shown to increase in relation to variations in air pollution levels (time-series studies). Observations from across North America^{96,97} and Europe^{3,98} have demonstrated higher rates of hospitalizations for all cardiovascular causes. Direct associations have also been identified with respect to incidence of ischemic heart disease, arrhythmias, and heart failure.⁹⁸ A pooled analysis of hospital admissions studies showed significant increases in admission rates of 0.8% and 0.7% for heart failure and ischemic heart disease, respectively, for each 10- $\mu\text{g}/\text{m}^3$ elevation in PM_{10} .⁹⁹ More focused investigations have demonstrated elevated risks for AMI,¹¹ implantable cardioverter defibrillator discharges,¹⁸ and myocardial ischemia during stress testing.¹⁷ Extreme elevations in air pollution have also been associated with increased blood pressure during a prolonged air stagnation episode in Europe.¹⁰⁰ Finally, recent studies from Seoul, South Korea¹⁰¹ and Taiwan¹⁰² have reported higher incidences of ischemic strokes in direct relation to changes in ambient particle concentrations. In summary, these findings imply that short-term elevations in ambient particle levels are capable of evoking cardiac arrhythmias, worsening heart failure, and triggering acute atherosclerotic/ischemic cardiovascular complications.

Populations at Elevated Risk

It is now reasonably well established that both short-term and chronic air pollution exposures are related to cardiovascular diseases. Whether there are specific individuals or subsets of patients at increased relative risk is less well documented. A few observations have suggested that the elderly^{5,81,84,103} and those with less than a high school education (low socioeconomic status) may be particularly susceptible populations.⁵ Whether increased age per se or the high prevalence of underlying cardiovascular disease and other risk factors explains the enhanced risk observed in elderly populations is unclear. The presence of preexisting chronic lung disease, coronary heart disease, and heart failure may also elevate short-term cardiovascular mortality risk.¹⁰³ Most recently, publications from 2 separate groups provide evidence that the acute risk for cardiovascular events in patients with diabetes mellitus may be 2-fold higher than for nondiabetics.^{104,105} It is plausible that both diabetes itself and the high incidence of concomitant cardiovascular disease may explain this relationship. There is no convincing evidence that gender, race, and other preexisting coronary risk factors (eg, obesity, dyslipidemia, and hypertension) increase the risk of cardiovascular events due to air pollution.

The effect of tobacco smoking on the cardiovascular risk due to long-term air pollution exposure was recently investigated in the large ACS study.⁴ The risks for specific cardiovascular causes of death for each

10- $\mu\text{g}/\text{m}^3$ increase in the long-term exposure to $\text{PM}_{2.5}$ were stratified by smoking status. Ischemic heart disease mortality was consistently elevated, with RRs of 1.22 (95% CI 1.14 to 1.29), 1.15 (95% CI 1.07 to 1.23), and 1.16 (95% CI 1.07 to 1.27) for never-smokers, former smokers, and current smokers, respectively. Interestingly, smoking status clearly affected the risk associated with several other causes of death. The risk due to arrhythmias, heart failure, and cardiac arrest was not significantly elevated for those who never smoked (RR 1.04 [95% CI 0.95 to 1.15]). However, among former and current smokers, the risk increased substantially, with RRs of 1.14 (95% CI 1.00 to 1.29) and 1.31 (95% CI 1.12 to 2.19), respectively. Mortality risk due to hypertensive disease was also only increased in patients who actively smoked (RR 1.57, 95% CI 1.12 to 2.19). These findings show that smoking, while not affecting the PM-associated risk from ischemic heart disease, appeared to interact with air pollution to increase the risk of death from other circulatory diseases.

Environmental Pollution and Congenital Heart Disease

Malformations of the cardiovascular system are among the more frequently occurring congenital defects. The incidence of congenital heart disease is estimated at 8.1 per 1000 births.¹⁰⁶ The actual rate may be underestimated, because some cases may be lethal in utero and result in spontaneous abortion and stillbirths. Congenital heart disease is associated with genetic defects, infections (eg, rubella), radiation, medications, and environmental exposures.¹⁰⁶⁻¹¹¹ Recent data from Eastern Europe in areas with high levels of industrial pollution suggest the possibility of increased heart defects.¹¹²⁻¹¹⁴ More recently, a study of birth records in Los Angeles, Calif, found that odds ratios (ORs) for cardiac ventricular septal defects increased in a dose-response fashion with increasing carbon monoxide exposure (OR for second quartile 1.62 [95% CI 1.05 to 2.48], OR for third quartile 2.09 [95% CI 1.19 to 3.67], and OR for fourth quartile 2.95 [95% CI 1.44 to 6.05]).¹¹⁵ Also observed were valvular, aortic, and truncal defects associated with O_3 levels. PM and other measured air pollutants showed no association. Although some animal data also suggest a relationship between air pollutants and congenital cardiac defects, these epidemiological data can only be considered suggestive at this time.

Significance of Epidemiological Findings

Epidemiological research has served to drive major governmental regulations and thus has been the subject of intense scrutiny. Two of the largest studies of the health effects of long-term air pollution exposure^{81,82} that have served as the basis for the setting of annual average $\text{PM}_{2.5}$ standards have undergone complete reanalyses by independent investigators to ensure reproducibility.¹¹⁶ The reanalyses validated the quality of the data and replicated the original results without any substantial alteration in findings. Although exposure to ambient air pollution poses smaller relative risks for incident cardiovascular disease than obesity or tobacco smoking, because it is ubiquitous, the absolute number of people affected is enormous, and exposure occurs over an entire lifetime. The adverse effects on the public health are clearly not limited to a harvesting effect as described earlier. Rather, Pope⁷⁸ has estimated an average loss of life expectancy directly related to chronic air pollution exposure from between 1.8 and 3.1 years for those living in the most polluted cities in the United States. Cardiovascular causes account for the majority (69%) of the overall

excess in morbidity and mortality.⁷⁸

Present Controversies

Given the continuous spatial and temporal variability of air pollution, combined with individuals' movements through numerous microenvironments every day, it is not surprising that exposure assessment in air pollution studies has always been subject to varying degrees of measurement error. Generally, in the epidemiological studies, an individual subject's exposure level has been estimated from citywide pollution measurement averages obtained from at most a few central locations. Past studies have also been limited by the fact that the numerous gaseous and particulate pollutants tend to covary in time and space. This has limited the ability to confidently link health outcomes with any given pollutant, although this may be an unrealistic goal given the complexity of the ambient pollutant mixture and the potential for combined toxic effects from many different combinations of constituents. Use of new tools such as geographic information systems and personal monitoring devices and better measures of the full toxic air pollution mix (eg, individual chemical and physical constituents or measurements of total oxidant capacity) may provide more refined estimates of the adverse health effects that can be related to specific (mixtures of) components.

Despite many past limitations, there has been a strong consistency in the findings among the array of assorted studies. A reasonable argument can now be made that the "real" effects are likely to be even stronger than previously estimated. Indeed, a recent study suggests a more robust linkage. In a recent "natural experiment," Clancy et al¹¹⁷ were able to demonstrate a decrease in health effects after the intentional lowering of air pollutant levels. These investigators compared 72 months of mortality data before and after a ban on burning coal within the city of Dublin, Ireland, went into effect. Nontraumatic deaths decreased by 5.7% (95% CI 4.0% to 7.0%) and cardiovascular mortality by 10.3% (95% CI 8.0% to 13.0%). The authors estimated that the ban resulted in 243 fewer cardiovascular deaths per year. The decrease in the mortality rate in this "natural experiment" is more than twice what would be predicted by the short-term time-series analyses.

► Potential Biological Mechanisms

The putative biological mechanisms linking air pollution to heart disease involve direct effects of pollutants on the cardiovascular system, blood, and lung receptors, and/or indirect effects mediated through pulmonary oxidative stress and inflammatory responses. Direct effects may occur via agents that readily cross the pulmonary epithelium into the circulation, such as gases, and possibly UFPs²⁸⁻³⁰ along with soluble constituents of PM_{2.5} (eg, transition metals). In addition, activation of pulmonary neural reflexes secondary to PM interactions with lung receptors may play a role. Ensuing alterations in autonomic tone, under appropriate circumstances, might contribute to the instability of a vascular plaque or initiate cardiac arrhythmias. These

- ▲ [Top](#)
- ▲ [Abstract](#)
- ▲ [Introduction](#)
- ▲ [Ambient Air Pollutants](#)
- ▲ [Epidemiology of Ambient Air...](#)
- [Potential Biological Mechanisms](#)
- ▼ [Summary](#)
- ▼ [Potential for Prevention/Public...](#)
- ▼ [Conclusions and Recommendations](#)
- ▼ [References](#)

direct effects of air pollution represent a plausible explanation for the occurrence of rapid (within a few hours) cardiovascular responses, such as increased myocardial infarctions.¹¹ Less acute (several hours to days) and chronic indirect effects may occur via pulmonary oxidative stress/inflammation induced by inhaled pollutants. This subsequently may contribute to a systemic inflammatory state, which may in turn be capable of activating hemostatic pathways, impairing vascular function, and accelerating atherosclerosis. A general scheme illustrating potential mechanisms of the effects of PM on the cardiovascular system is shown in [Figure 2](#).

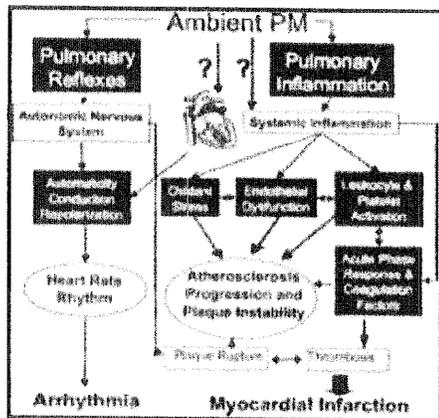


Figure 2. Possible biological mechanisms linking PM with cardiovascular disease.

[View larger version \(54K\):](#)
[\[in this window\]](#)
[\[in a new window\]](#)

Pulmonary and Systemic Oxidative Stress and Inflammation

Inhalation of air pollutants induces pulmonary oxidative stress and inflammation.¹¹⁸ Exposure of human lungs to concentrated PM¹¹⁹ and O₃¹²⁰ produces an inflammatory response consistent with *in vivo* animal models¹²¹ and *in vitro* cellular models.^{122–125} The presence of soluble transition metals in PM enhances the inflammatory responses¹²⁶ via increased oxidative stress.^{127,128} However, lung inflammation may also occur via direct UFP effects independent of transition metals or soluble components.¹²⁹ Similarly, O₃ mediates a pulmonary inflammatory response via oxidative stress^{118,130} and an impairment in lung function that can be attenuated by antioxidants.¹³¹

Oxidative stress occurs after exposure to ultrafine carbon black and diesel exhaust particles (DEPs),^{129,132} ambient PM_{2.5},¹³³ and cigarette smoke. A recent *in vivo* experiment using *in situ* chemiluminescence demonstrated the rapid occurrence of oxidative stress even in tissues beyond the lung. After exposure to concentrated ambient PM_{2.5} for 2 hours, there was a doubling in reactive oxygen species generation in the hearts and lungs of rats.¹³⁴ This may occur in response to a variety of transition metals¹³⁴ or free radical components known to exist within PM_{2.5} as a result of atmospheric chemical reactions.¹³⁵ Personal

exposure to ambient concentrations of PM_{2.5} is also associated with increased levels of markers of lipid and protein oxidation in human blood.¹³³ Pulmonary inflammation results at least in part because of the increased production of free radicals.¹¹⁸ Oxidative stress activates specific transcription factors, including nuclear factor- κ B and activator protein-1, which upregulate the expression of genes for cytokines, chemokines, and other proinflammatory mediators.¹³² DEPs or organic extracts of DEPs can, through oxidant effects on mitochondria, induce apoptosis or necrosis of macrophages and respiratory epithelial cells, possibly decreasing the host defenses to respiratory infection or increasing airway reactivity.¹³⁶

Endotoxin, usually associated with coarse particles, has also been shown to induce proinflammatory cytokine production^{137,138}; increase lung inflammation, airway responsiveness, and systemic immune cell populations; and decrease lung function.^{139,140} Endotoxin has been found to account for in vitro cytotoxicity and cytokine production related to PM_{2.5} and coarse PM exposures^{141,142}; however, its role in the overall toxicity of ambient PM remains to be clarified.

The intrapulmonary responses elicited by PM may also be due in part to neurogenic inflammation. Sensory neurons in contact with irritant particles (eg, within the conducting airways) can be stimulated to release neuropeptides (eg, substance P, calcitonin gene related peptide, and neurokinin A), which can initiate airway inflammatory events, including release of cytokines, vasodilation, and mucus secretion. Neuropeptides act on a variety of cell types within the lung, including epithelial and smooth muscle cells (resulting in modulation of inflammation and increased airway responsiveness), as well as immune cells (polymorphonuclear leukocytes [PMNs], lymphocytes, eosinophils, and others), thus amplifying the inflammatory response. Recent in vitro experiments indicate that specific irritant (capsaicin or vanilloid) receptors on neurons mediate PM-related neurogenic inflammation, as evidenced by responses to particles originating from diverse sources.¹⁴³

Several controlled-exposure studies demonstrate that inhalation of particles^{119,144,145} and O₃¹²⁰ evokes both a pulmonary and a systemic inflammatory response in humans. One hour of exposure to very high concentrations of diesel exhaust has been shown to induce an inflammatory reaction in the lungs of healthy adults. This response included increased numbers of PMNs, T- and B-lymphocytes, mast cells, and inflammatory mediators.¹⁴⁴ One of these studies showed an increase in adhesion molecules that facilitate the passage of inflammatory cells from the circulation into the airways. In the blood, platelets and PMNs increased, which suggests that exposure to DEPs stimulated the bone marrow to release these cells into the circulation.

Exposure to DEP has been shown to increase intra-airway transcription of mRNA for interleukin (IL)-8 (a protein that attracts PMNs to sites of injury)¹⁴⁶ and increased production of IL-8 and growth-regulating oncogene- α (GRO)-promoting airway inflammation. Exposure of healthy adults to concentrated ambient particles (CAP) for 2 hours can increase airway inflammation without concomitant lung injury.¹¹⁹ Nevertheless, plasma fibrinogen was elevated by the CAP exposure relative to filtered air. This controlled

exposure study suggests that exposure to ambient particles in healthy humans can result in a mild pulmonary inflammatory response and increased blood factors that effect coagulation, even without lung damage. A similar human study also demonstrated an increase in blood fibrinogen after short-term exposure to CAP.¹⁴⁷ However, no other changes in inflammatory mediators or other coagulation factors were found.

Additional studies support the existence of a systemic inflammatory response beyond the lungs after air pollution exposure. In humans, exposure to forest fire smoke (measured as PM₁₀ and SO₂) at levels that did not result in changes in lung function nevertheless resulted in stimulation of bone marrow to release immature PMNs into the circulation.¹⁴⁸

In an animal experiment, rabbits that received 5 mg of PM₁₀ intrapharyngeally twice per week for 3 weeks exhibited increased production of PMNs in the bone marrow and accelerated release into the circulation.¹⁴⁹ The PM₁₀ exposure resulted in diffuse inflammation of the lungs, with particles present in alveolar macrophages, lung epithelial cells, and airway walls. The effects on PMN production in bone marrow and release of immature cells into the blood were associated with the numbers of particles ingested by alveolar macrophages.

Effects of Inflammation, Oxidative Stress, and Alterations in Blood-Borne Factors on the Cardiovascular System

Changes in the composition of the blood may result from air pollution exposure, with potentially serious effects on individuals with cardiovascular disease. Nearly a decade ago, Seaton et al¹⁵⁰ proposed a general hypothesis that exposure to inhaled particles induces alveolar inflammation, leading to exacerbation of preexisting lung disease, increased blood coagulability, and an associated increased risk of cardiovascular events. Subsequently, evidence supporting this hypothesis has slowly been accumulating. As described above, several studies of controlled exposures to particles demonstrate increases in both cellular and biochemical markers of pulmonary and systemic inflammation.

Exposure to PM increases fibrinogen,^{119,147} a key component in blood coagulation and platelet thrombosis and a major determinant of blood viscosity. Blood viscosity has been associated with severity of cardiovascular disease¹⁵¹ and has been found to increase in association with increased levels of ambient total suspended particles and SO₂.¹⁵² Fibrinogen is also well established as an important independent risk factor for myocardial infarction and stroke. Epidemiological data suggest potential effects of particulate air pollution on blood coagulation.¹⁵²⁻¹⁵⁴ Of note is that the strength of plasma level of fibrinogen to predict cardiac events and death in middle-aged men is modified by the presence of other inflammation-sensitive proteins,¹⁵⁵ which suggests that inflammation has a significant role in the determination of cardiovascular risk. In addition, enhanced platelet aggregation may further promote acute thrombosis formation after exposure to diesel exhaust¹⁵⁶ and UFPs.¹⁵⁷ The mechanisms responsible for platelet activation and fibrinogen elevation remain to be fully elucidated. Nevertheless, these findings support the notion that air pollution can acutely increase the risk of thrombosis, thus promoting ischemic events.

Increased concentrations of IL-6 are associated with an increased risk of cardiovascular events^{158,159} and mortality.¹⁶⁰ Serum IL-6, IL-1 β , and granulocyte macrophage colony-stimulating factor are increased in healthy male subjects after exposures to increased air pollution due to forest fires and are increased in vitro with exposure of human lung macrophages to urban PM₁₀.¹⁶¹ IL-6 is directly involved in regulation of the synthesis of C-reactive protein in the liver. CRP is a sensitive indicator of infection, injury, and inflammation and is linked to increased risk of cardiovascular disease.^{162,163} CRP concentration has been shown to be positively associated with exposure to total suspended particles¹⁶⁴ and PM₁₀.¹⁵³

The mechanisms by which CRP increases the risk of cardiovascular events is the subject of intense research. One possibility is that CRP impairs endothelial vasoreactivity in individuals with preexisting coronary artery disease.¹⁶⁵ In addition, CRP may contribute directly to the development and progression of atherosclerosis via a number of mechanisms that involve enhanced formation of foam cells, recruitment of monocytes into the arterial wall, stimulation of prothrombotic tissue factors, decreased NO synthase activity, and expression of adhesion molecules.¹⁶⁶ Inflammation (proinflammatory cytokines, CRP, and components of innate immunity) plays a significant role in the genesis of atherosclerosis and in plaque instability.¹⁶⁷ It is possible, therefore, that air pollution-mediated systemic inflammation both promotes atherosclerosis formation over the long term¹⁴ and instigates acute plaque instability and sudden cardiovascular events in the short term. Indeed, in hyperlipidemic rabbits exposed to PM, the progression of coronary atherosclerosis and extracellular lipid pools increased after 4 weeks.¹⁴ The degree of plaque formation correlated with the number of alveolar macrophages that phagocytosed PM. These effects are likely to be superimposed on the effects of age, hypertension, hyperlipidemia, diabetes, and other conditions associated with underlying inflammation.

Alterations in vascular tone due to air pollution exposure have also been demonstrated. The inhalation of high urban levels of CAP and ozone for 2 hours caused conduit arterial vasoconstriction in healthy adults.¹⁶⁸ Similarly, small pulmonary arteries were shown to constrict after short-term exposure to CAP in rats.¹⁶⁹ It is possible that the acute systemic inflammation and oxidative stress^{133,134} are responsible for triggering endothelial dysfunction leading to vasoconstriction.¹⁷⁰ In support of this hypothesis, impaired arterial endothelial relaxation and decreased NO formation have been shown to occur in vessels exposed to DEPs owing to excess reactive oxygen species generation.¹⁷¹ Alternatively, increased production of endothelins may play a role in the acute vasoconstriction.¹⁷²

At present, the precise mechanisms underlying the rapid alterations in vascular tone remain to be resolved. However, a few published reports support the relevance of these findings by demonstrating an effect of air pollution on cardiovascular hemodynamics.^{100,172} Ambient air pollution increases blood pressure in cardiac rehabilitation patients¹⁷³ and in adults with lung disease.¹⁷⁴ Indeed, arterial vasoconstriction is a likely explanation for the findings of the ULTRA study (The Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air). Ambient levels of PM 2 days before submaximal exercise testing were significantly associated with increased ST-segment depression during the test.¹⁷ This finding suggests that

air pollution exposure conveys a greater susceptibility to myocardial ischemia, as demonstrated in an experimental study of dogs exposed to CAP.¹⁷⁵ These results also offer insight regarding the relationship between exposure to PM and the timing of AMI.¹¹ Significant associations were identified between symptom onset and both acute (levels within 2 hours before symptoms) and subacute (previous-day average concentration) exposures to PM_{2.5}. Sudden arterial vasoconstriction (and/or possibly endothelial dysfunction) could conceivably instigate acute coronary syndromes by triggering plaque instability or by decreasing myocardial perfusion in patients with existing atherosclerosis.

Disturbances of the Cardiac Autonomic Nervous System

Mortality associated with air pollution might be further explained, at least in part, by alterations in the autonomic input to the heart. HRV, resting heart rate, and blood pressure are modulated by a balance between the 2 determinants of autonomic tone (the sympathetic and parasympathetic nervous systems). Decreased HRV predicts an increased risk of cardiovascular morbidity and mortality in the elderly and those with significant heart disease.¹⁷⁶ This is generally determined by analyses of time (eg, standard deviation of normal RR intervals [SDNN]) and frequency domains (eg, low frequency/high frequency ratio by power spectral analysis, reflecting autonomic balance) measured during 24 hours of electrocardiography. Because overall HRV (SDNN) decreases in response to ambient PM exposure,¹⁷⁷⁻¹⁸¹ decreased parasympathetic input to the heart may provide an important mechanistic link between air pollution and cardiovascular mortality by promoting fatal tachyarrhythmias. A recent study of controlled exposure to CAP provides further support to the notion that PM is capable of reducing HRV.¹⁸² In general, the decrease of HRV occurs rapidly and is inversely proportional to the increase in the concentration of PM. However, in one study,¹⁷⁷ short-term measures of parasympathetic tone (r-MSSD) were increased in a group of individuals with preexisting heart disease. It is conceivable that in certain populations, air pollution-mediated bradyarrhythmias may also contribute to sudden death.

The relevance that these observed short-term changes in HRV have in relation to the worsening of cardiovascular outcomes and the triggering of significant arrhythmias over the long term remains unclear. However, some evidence suggests that PM exposure does promote clinically meaningful changes in cardiac electrophysiology. The incidence of cardiac arrhythmias has been associated with exposure to PM_{2.5} in high-risk individuals (eg, individuals having an implanted cardioverter defibrillator). In 100 patients monitored for ≈3 years, NO₂ and CO concentrations were most strongly related to implanted cardioverter defibrillator discharges, whereas particulate black carbon showed a slightly lesser degree of association with such events.¹⁸ Although this study is limited by the small number of high-risk patients and by the lack of individual clinical data beyond implanted cardioverter defibrillator discharges, it does suggest a potential for adverse effects of PM and gaseous pollutants on cardiac autonomic balance. A recent mortality time-series study⁹⁸ further supports this observation. The risks of mortality from threatening arrhythmias were shown to increase in relation to 7-day mean levels of black smoke and PM₁₀.

These clinical observations are consistent with several studies reporting the induction of cardiac

arrhythmias in compromised animals (ie, with pulmonary and systemic hypertension) exposed to PM.¹⁸³ Dogs exposed to CAP for 6 hours on 3 consecutive days showed increases in low- and high-frequency HRV, as well as an elevated low frequency/high frequency ratio.¹⁸⁴ In addition, exposure to residual oil fly ash, a component of PM, decreased HRV and increased arrhythmia frequency in a myocardial infarction model of rats.¹⁸⁵ These animal exposure findings support the notion that air pollution is capable of altering autonomic balance in a manner that favors significant tachyarrhythmias. The underlying mechanisms responsible remain unclear but may involve activation of pulmonary neural reflex arcs, direct effects of pollutants on cardiac ion channels, or consequences of the heightened systemic inflammatory state.

► Summary

Air pollution consists of a complex mixture of compounds in gaseous (eg, NO₂, SO₂, CO, O₃), liquid, and solid phases. PM itself is a heterogeneous mixture of suspended particles that vary in chemical composition and size, ranging from clusters of molecules (with diameters of several nanometers) to coarse PM (up to 10 μm and beyond). Among the many natural and anthropogenic sources of air pollution, the combustion of fossil fuels is a major contributor in urban and industrialized societies.

- ▲ [Top](#)
- ▲ [Abstract](#)
- ▲ [Introduction](#)
- ▲ [Ambient Air Pollutants](#)
- ▲ [Epidemiology of Ambient Air...](#)
- ▲ [Potential Biological Mechanisms](#)
- [Summary](#)
- ▼ [Potential for Prevention/Public...](#)
- ▼ [Conclusions and Recommendations](#)
- ▼ [References](#)

Numerous epidemiological studies conducted worldwide have demonstrated consistent associations between short-term elevations in PM and increases in daily cardiovascular morbidity and mortality. Several studies have also reported adverse cardiovascular outcomes in relation to long-term PM exposure. Elderly patients, those with underlying coronary or pulmonary disease, lower socioeconomic populations, and diabetics may be at particularly increased risk. At present, the constituent PM responsible for mediating these effects, along with the roles of the various gaseous copollutants, remain to be clarified.

Experimental evidence has revealed plausible biological mechanisms whereby PM has the potential to cause and exacerbate cardiovascular disease. One pathway involves the initiation of pulmonary and systemic oxidative stress and inflammation by components within PM. Subsequently, a cascade of physiological responses may follow that are capable of instigating cardiovascular events. These include alterations in blood rheology that favor thrombosis, cardiac dysrhythmias, acute vascular dysfunction, plaque instability, and the long-term development of atherosclerosis. Additional pathways may also be involved, such as changes in autonomic balance via lung neural reflex arcs and/or by PM (or certain components) reaching the circulation and beyond.

► Potential for Prevention/Public Policy

▲ Top

The increase in relative risk for cardiovascular disease due to air pollution for an individual is small compared with the impact of the established cardiovascular risk factors. However, because of the enormous number of people affected, even conservative risk estimates translate into a substantial increase in total mortality within the population. The impact on cardiovascular disease therefore represents a serious public health problem. The latest draft of the US EPA Air

Quality Criteria for Particulate Matter has confirmed the presence of an apparent linear dose-response relationship between PM and adverse events.¹⁸⁶ Data from all North American studies demonstrate that this curve is without a discernible threshold below which PM concentrations pose no health risk to the general population.¹⁸⁶ At present-day levels, $\approx 40\,000$ deaths per year in Austria, France, and Switzerland combined have been attributed to PM.¹⁸⁷ Estimates based on time-series studies suggest that ≈ 5000 excess deaths per year in Canada¹⁸⁸ and 6000 cardiovascular events in the United Kingdom¹⁸⁹ can be attributed to poor air quality. Approximately 1 in 50 myocardial infarctions were thought to be triggered by outdoor air pollution in a London, England, study.³ On a global scale, the World Health Organization has estimated that 800 000 deaths occur per year and 7.9 million disability-adjusted life-years are lost annually due to PM exposure.^{189a}

Given the burden of epidemiological evidence, the US EPA updated the National Ambient Air Quality Standards in 1997 to specifically include PM_{2.5} (Table).¹⁹⁰ The most current estimates by the EPA suggest that attainment of these standards would reduce total mortality by 23 000 deaths annually and cardiovascular hospital admissions by 42 000 per year in the United States.¹⁹¹ Nevertheless, 19% of all US counties with air-quality monitoring systems are presently not meeting these standards.¹⁹² This percentage is substantially greater in regions such as the industrial Midwest (41%) and southern California (60%). In light of these data, there is a clear potential to improve the national public health and to substantially reduce cardiovascular morbidity and mortality by reducing PM levels to current EPA standards. The potential cardiovascular health effect of reducing the gaseous copollutants remains less certain.

- ▲ [Top](#)
- ▲ [Abstract](#)
- ▲ [Introduction](#)
- ▲ [Ambient Air Pollutants](#)
- ▲ [Epidemiology of Ambient Air...](#)
- ▲ [Potential Biological Mechanisms](#)
- ▲ [Summary](#)
- [Potential for Prevention/Public...](#)
- ▼ [Conclusions and Recommendations](#)
- ▼ [References](#)

View this table: Current US EPA National Ambient Air Quality Standards for PM (1997 NAAQS)
[\[in this window\]](#)
[\[in a new window\]](#)

► Conclusions and Recommendations

Most^{3-8,81} but not all^{12,13} studies have found positive associations between several different air pollutants and adverse health outcomes.

- ▲ [Top](#)
- ▲ [Abstract](#)
- ▲ [Introduction](#)
- ▲ [Ambient Air Pollutants](#)

The results of observational studies are influenced by numerous factors, including characteristics of the air pollution, the population studied, and methodological issues, such as control of relevant confounders. The lack of complete uniformity is not surprising given that numerous variables (atmospheric conditions, geographic locations, cohort characteristics, sample sizes, exposure estimates, and statistical modeling) can affect the results. Our understanding of the relevant biological mechanisms involved also remains incomplete. Nevertheless, the existing body of evidence is adequately consistent, coherent, and plausible enough to draw several conclusions.¹⁹³ At the very least, short-term exposure to elevated PM significantly contributes to increased acute cardiovascular mortality, particularly in certain at-risk subsets of the population. Hospital admissions for several cardiovascular and pulmonary diseases acutely increase in response to higher ambient PM concentrations. The evidence further implicates prolonged exposure to elevated levels of PM in reducing overall life expectancy on the order of a few years.

▲	<u>AMBIENT AIR POLLUTION</u>
▲	<u>Epidemiology of Ambient Air...</u>
▲	<u>Potential Biological Mechanisms</u>
▲	<u>Summary</u>
▲	<u>Potential for Prevention/Public...</u>
▪	Conclusions and Recommendations
▼	References

On the basis of these conclusions and the potential to improve the public health, the AHA writing group supports the promulgation and implementation of regulations to expedite the attainment of the existing National Ambient Air Quality Standards. Moreover, because a number of studies have demonstrated associations between particulate air pollution and adverse cardiovascular effects even when levels of ambient PM_{2.5} were within current standards,^{5,11} even more stringent standards for PM_{2.5} should be strongly considered by the EPA. Additional approaches to reduce the burden of disease related to air pollution should be highlighted. The levels of O₃ and PM in many US cities are published daily by the EPA, along with a health alert system that reflects recommended changes in activity. This information is also available and updated daily on the EPA AIRNow web site (<http://www.epa.gov/airnow>). The AHA supports these recommendations as guidelines for activity restriction for persons with known heart disease (or with an "at-risk" profile by Framingham or another scoring system), and pulmonary disease, the elderly, and those with diabetes mellitus. A concerted effort should be made to educate healthcare providers and at-risk patients alike about this source of information and about the potential health hazards of elevated air pollution levels. The AHA should also actively work to educate the public and public policy makers about the effects of air pollution on cardiovascular disease by featured presentations at the annual Scientific Sessions, AHA-sponsored public education activities, and advocacy.

In October 2003, the EPA expanded its Air Quality Index program to include information on particle pollution, or fine particles. Next-day forecasts and real-time air quality information about particle pollution are available on the AIRNow Web site for more than 150 cities across the country. Air quality forecasts and reports for particle pollution are available in the local media (newspapers, television, and radio) in these cities and in the national media. The EPA is working to increase the number of state and local air quality agencies that forecast and report real-time particle pollution levels. It is expected that within a few years, this program will mirror the O₃ program in terms of geographic coverage and availability to the public. The AIRNow Web site also contains information about the health effects of particle pollution, and the EPA is

developing a section with information and tools for healthcare professionals.

Although there is a strong case that air pollution increases the risk of cardiovascular disease, we recognize the need to address a number of remaining scientific questions. Both the US EPA and the National Institutes of Health have increased the priority of research funding in an effort to overcome these shortcomings, and a committee of the National Research Council set out a long-range research agenda in 1998.¹⁹⁴ A workshop sponsored by these and other agencies was convened in August 2002 to foster multidisciplinary research on the cardiovascular effects of PM. The AHA writing group supports these measures and recognizes several important areas for future research^{80,192}:

1. Improve our understanding of the underlying biological mechanisms.

- Better describe the basic mechanisms (mediators, cell signaling, pathways) involved in altering HRV (autonomic tone), vascular function, and atherogenesis.
- Increased use of relevant animal models of exposure when investigating cardiovascular outcomes (eg, inhalation chambers at meaningful CAP concentrations). Experiments using intrapulmonary installation provide insight into basic mechanisms; however, the ability to extrapolate findings to humans is limited owing to the route of administration of extraordinarily high quantities of PM.
- Determine the pathophysiological relevance of the many pathways that may contribute to the development of both acute and chronic disease.
- Demonstrate reproducibility of the relevant potential mechanisms under a variety of pollutant regimens and subject risk profiles.
- Demonstrate the occurrence of such cardiovascular end points at environmentally relevant concentrations of ambient pollutants.
- Determine causal pathways, which may become targets for future means of preventive strategies
- Determine whether long-term exposure to PM at environmentally relevant concentrations promotes the genesis/progression of atherosclerosis in humans.

2. Identification of the differential toxicity of various constituents and sources of air pollution, including:

- Specific chemical and biological constituents of PM (eg, metals, carbon, polycyclic aromatic hydrocarbons, endotoxin).
- The role of different PM size fractions, including UFPs (<0.1 μm) and the coarse fraction (PM₁₀ to 2.5).
- The effects of gaseous copollutants alone or in combination with PM.

3. Epidemiological investigations designed to address some of the limitations of prior reports, including studies that involve the following:

- Better characterization of the populations of individuals at high risk related to short-term elevations in PM (eg, comorbidities).
- Improvement of exposure estimates and metrics (eg, use of personal monitoring systems).
- Examination of the relationships between traffic emissions and adverse cardiac effects.



**UNITED STATES COAST GUARD
PUBLIC MEETING**

COMMENT FORM

AMBASSADOR BRIDGE ENHANCEMENT PROJECT

In the space below, please provide your comment regarding the Ambassador Bridge Enhancement Project. All comments received will be posted to <http://www.regulations.gov> under the Coast Guard docket number USCG-2009-0093.

Please submit to a Coast Guard official before the end of this public meeting.

Our asthma rate is three times normal. These are children who will have this disease all their lives. They will be people with disabilities. Find a way to clean the air before you go any further.

The fact that hundreds of people are proven the inadequacy of communication prior to the final decision

F

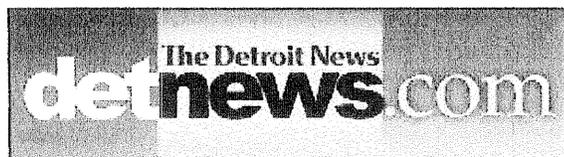
Please place the two (2) articles
into the record.

They are from January 2009. One
is from U.S. Det and the second
a follow-up from the Detroit news.

Thomas F. DOMBROSKI

Resident SW Detroit

Precinct 15 delegate 55-13



Thursday, January 15, 2009

Detroit

Second bridge to Canada wins fed approval

State can begin right-of-way acquisition and building plans after receiving final environmental clearance.

Tom Greenwood / The Detroit News

The U.S. Department of Transportation announced Wednesday that it has approved an environmental impact review of plans to build a second bridge across the Detroit River to Canada.

The decision represents the final environmental clearance on the U.S. side for the Detroit River International Crossing study project and will allow Michigan to begin right-of-way acquisition and construction planning for the new span.

A similar review of an environmental study on the Canadian side conducted earlier this year by Ontario and Transport Canada is nearing completion.

"This is a big milestone in the process," said Bill Shreck, director of communications for the Michigan Department of Transportation.

"It's an important component of the border system we need to make Michigan competitive in the 21st century."

Although no decision has yet been made, officials in Michigan are leaning toward locating the American end of the bridge in the Downriver area, specifically in Del Ray, near Zug Island.

In July 2008, Oakland County Executive L. Brooks Patterson and Windsor Mayor Eddie Francis endorsed building the bridge at that location.

Critics have slammed the idea of a second bridge, calling the \$1 billion project too expensive and unnecessary given the current economic situation.

Others are dismayed that the construction of a second bridge at that site forces the demolition

of up to 414 homes in Del Ray and would uproot as many as 56 businesses that employ nearly 1,000 people.

The Detroit International Bridge Co., which owns the Ambassador Bridge, has proposed building a second, privately owned bridge between Detroit and Windsor.

A call to the Detroit International Bridge Co. was not returned.

You can reach Tom Greenwood at (313) 222-2023.

Find this article at:

<http://www.detroitnews.com/apps/pbcs.dll/article?AID=/20090115/METRO01/901150379>

Check the box to include the list of links referenced in the article.

© Copyright 2008 The Detroit News. All rights reserved.



[FHWA Home](#) | [Feedback](#)



**REFOCUS.
REFORM.
RENEW.**
A New Transportation Agenda
IN AMERICA

**U.S. Department of Transportation
Office of Public Affairs
Washington, D.C.
www.dot.gov/affairs/briefing.htm**

News

FHWA 01-09
Wednesday, January 14, 2009

Contact: Doug Hecox
Tel.: (202) 366-0660

USDOT Signs 'Record of Decision' for Detroit River International Crossing

WASHINGTON - Plans to build a second border crossing between Michigan and Ontario have received the necessary environmental approvals from the U.S. Department of Transportation.

The "record of decision" (ROD), signed today by U.S. officials, represents the Detroit River International Crossing's (DRIC) final environmental clearance and allows Michigan to begin right-of-way acquisition and construction planning for the bridge.

If completed, the project - including a plaza where tolls and U.S. border inspection activities will occur, and an interchange connecting it to I-75 - would span nearly seven miles. Under current estimates, the new crossing is expected to be open to traffic in 2013.

Environmental review for the U.S. side of the project, which concluded with today's ROD signing, began March 24, 2003, with the publication of a Notice of Intent in the Federal Register. A similar review of environmental impacts on the river's Canadian side conducted earlier this year by Ontario and Transport Canada is nearing completion.

The process to complete DRIC's federal environmental documentation lasted less than four years, about half the time needed for similar projects of this size, and included more than 100 meetings and public hearings.

###

FHWA Press Room

[FHWA Home](#) | [Feedback](#)

© FHWA

United States Department of Transportation - **Federal Highway Administration**